

# Successful Management of Fulminant Myocarditis with Left Ventricular Assist Device: Report of a Severe Case

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**We report a 65-year-old man with fulminant myocarditis undergoing percutaneous cardiopulmonary support (PCPS) and left ventricular assist device (LVAD). PCPS and intra-aortic balloon pumping was initially introduced for cardiogenic shock in the emergency department. We switched to LVAD because cardiac function did not recover despite PCPS for 5 days. Cardiac function then gradually improved, and the device was successfully weaned after 11 days of the LVAD support. He was discharged on postoperative day 63 with no complications. We here report the appropriate timing of LVAD application for fulminant myocarditis. (Ann Thorac Cardiovasc Surg 2010; 16: 48–51)**

**Key words:** fulminant myocarditis, left ventricular assist device, percutaneous cardiopulmonary support

## Introduction

Fulminant myocarditis, which is commonly developed by virus infection with severe clinical presentations, is characterized as an inflammatory disease with rapid cardiac deterioration and marked myocardial inflammation.<sup>1)</sup> Patients who develop cardiogenic shock or fatal arrhythmia often require mechanical circulatory support. In most cases, however, multiple organ failure (MOF) gradually develops, and the patient ultimately dies. We here report a successful survival case of fulminant myocarditis by left ventricular assist device (LVAD).

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## Case Report

A 65-year-old man visited a general hospital with a 10-day history of chest pain and high fever. He was transferred to our emergency department with suspected myocarditis or cardiac infarction because an electrocardiogram revealed sinus tachycardia with ST-segment elevation in leads V2–6, and a laboratory examination revealed an elevation of cardiac enzymes. On physical examination, his blood pressure, heart rate, and body temperature were 95/57 mmHg, 105 beats/min, and 37.2°C. A laboratory examination revealed the following data: white blood cell count 8,700/μl; C-reactive protein 3.7 mg/dl; creatinine kinase 1,770 IU/l; creatinine kinase MB 142 IU/l; and brain natriuretic peptide 527 pg/ml. The neutralizing antibody titer for parainfluenza showed a 160-fold increase. Chest X-rays showed acute pulmonary edema (Fig. 1). Echocardiography revealed global severe left ventricular dysfunction with ejection fraction of 27%. Emergency coronary angiography was performed with assistance by an intra-aortic balloon pumping (IABP), but it showed no coronary stenosis. The patient's hemodynamic and respiratory status had been deteriorated despite 24 hours of support by the IABP and high-dose inotropes.



**Fig. 1.** Chest X-ray showed pulmonary congestion on admission.



**Fig. 2.** Chest X-ray showed that severe pulmonary congestion was still apparent just before LVAD placement.

Hemodynamic data included a systemic blood pressure of 78/40 mmHg, a heart rate of 118 beats/min, a pulmonary artery pressure of 28/15 mmHg, and a cardiac index of 1.4 L/min/m<sup>2</sup>. The patient required ventilator support, and we also instituted percutaneous cardiopulmonary support (PCPS). But cardiac function did not improve despite an intravenous immunoglobulin therapy (5 g/day) and mechanical circulatory support for 5 days. Severe pulmonary congestion was still apparent on the chest X-ray, and continuous hemodialysis was also required for his poor urine volume (Fig. 2). Lastly, echocardiography demonstrated global akinesis of the left ventriculum and ejection fraction of less than 5%. Hemolysis, which was caused by the PCPS, had been getting worse. The patient was complicated by MOF that contained cardiac failure, respiratory failure, and renal failure; furthermore, hepatic insufficiency was gradually developing, and total bilirubin level rose to 4.0 mg/dl. We decided to switch the PCPS to LVAD (Toyobo Co., Ltd., Osaka, Japan) after 132 hours under assistance by the PCPS. The LVAD was implanted from the left atrium to the ascending aorta under sternotomy. Over the following 2 days, the circulation was supported by PCPS, which was continued because of pulmonary dysfunction in addition to the LVAD support. The pump flow was controlled initially at 1.5 L/min as pulmonary congestion was gradually improving, and the PCPS was successfully

weaned on postoperative day 2. However, total bilirubin level had risen to 28.2 mg/dl, and pneumonia, resulting from methicillin-resistant *Staphylococcus aureus* and *Pseudomonas aeruginosa*, had developed. On the other hand, his hemodynamic status had been stabilized by LVAD support. Echocardiography on postoperative day 9 showed normal left ventricular size and an improvement in left ventricular function with full device support. The LVAD was then removed after 11 days. After the LVAD explantation, his cardiac index was 3.0 L/min/m<sup>2</sup>. The mechanical ventilator was also successfully weaned on postoperative day 32. Postoperative echocardiography showed improvement of his left ventricular function, and ejection fraction was 42%. Total bilirubin level dropped to 1.4 mg/dl, and brain natriuretic peptide decreased to 75 pg/ml. He went home on postoperative day 63 and has been well in the out-patient clinic (Fig. 3).

## Discussion

The clinical presentation of myocarditis ranges widely from asymptomatic electrocardiogram abnormality to fatal heart failure. The histopathological characteristics of myocarditis are mentioned in the “Dallas classification system.” According to that classification, an inflammatory infiltrate and myocyte damage are both observed in



**Fig. 3.** Chest X-ray at discharge.

myocardium.<sup>2)</sup> Fulminant myocarditis is especially featured by very extensive inflammatory infiltrates and numerous foci of myocyte necrosis. The initial symptom of fulminant myocarditis is an extremely severe condition that affects morbidity and mortality. However, even such patients have a likelihood that the cardiac functions can recover through appropriate treatment. Some reports suggest the potential therapeutic efficacy of intravenous immunoglobulin therapy for fulminant myocarditis.<sup>3,4)</sup> Abe et al. reported the suppression of serum cytokines in those who were treated with intravenous immunoglobulin therapy and PCPS, and they survived without complications.<sup>5)</sup> We also carried out intravenous immunoglobulin therapy for this case, but it might not be effective. It is necessary to assess the clinical efficacy of intravenous immunoglobulin therapy in fulminant myocarditis in more cases.

Patients with fulminant myocarditis usually suffer from cardiogenic shock that requires mechanical circulatory support. In Japan, IABP and PCPS are commonly used as the first choices of mechanical circulatory support because most institutions can provide these devices. Patients complicated by cardiac crises should be immediately saved by mechanical circulatory support. PCPS is beneficial to these patients because cardiologists are accustomed to treatment by PCPS, and they can quickly set it up without

a cardiovascular surgeon. PCPS can be considered as the first-line treatment of mechanical support for fulminant myocarditis.<sup>6)</sup> It is important to manage PCPS to reduce the afterload of the left ventricle. The elevated left ventricular end diastolic pressure may lead to a deterioration of pulmonary function and complicate the course of PCPS treatment. IABP and left atrial venting may work to reduce left ventricular afterload. If that is impossible, it is important to find the best balance between flow support and afterload.<sup>7)</sup> But the continuance of PCPS makes many problems that include hemolysis, circulatory disturbances of the legs, or MOF. Further, a membrane oxygenator cannot maintain performance for a long time and patients cannot be rehabilitated with PCPS. Thus PCPS is not suitable for long-term support until cardiac function improves.

We determined to switch to LVAD support from PCPS for 5 days; nevertheless, MOF developed. The introduction of LVAD was late, and it led the patient to pneumonia and hepatic insufficiency, which requires a long hospital stay. Aoyama et al.<sup>8)</sup> reported that the important factors concerning the prognosis of fulminant myocarditis were the severity and grade of cardiac and renal function, the adjusted support flow rate, the prevention of circulatory disturbances of the legs, and MOF associated with PCPS. Most of these problems are resolved by LVAD, which has stronger circulatory support than PCPS. However, it is difficult to treat MOF, especially hepatic insufficiency even though cardiorespiratory failure and renal failure can be supported by LVAD and hemodialysis. Peek et al.<sup>9)</sup> reported that patients whose bilirubin level rose to a high value during PCPS had a high mortality. On the other hand, in patients with LVAD support, pre-operative direct and indirect bilirubin levels are identified as the most important predictors of survival.<sup>10)</sup> PCPS should be changed to LVAD support before the bilirubin level comes up. Accordingly, we should have switched to LVAD on the 3rd day after PCPS implantation, before the total bilirubin level increased to 3.0 mg/dl, signaling hepatic insufficiency. We previously reported the strategy of PCPS, that LVAD should be introduced according to end-organ function and the expected support period if native cardiac function does not recover for 2 or 3 days despite PCPS.<sup>11)</sup> When patients often need respiratory support because of pulmonary dysfunction just after the switch to LVAD, we can use some devices, such as a veno-veno extracorporeal membrane oxygenation and a right ventricular assist system with extracorporeal membrane oxygenation other than PCPS.<sup>12)</sup> In this we elected to

continue PCPS in addition to LVAD for several days until the patient's respiratory status improved. LVAD maintained a sufficient flow with PCPS flow of 1.5 L/min. Some studies suggest the beneficial effects of LVAD for fulminant myocarditis, and a period of weeks to months of LVAD support in these reports was necessary for cardiac recovery.<sup>13-16</sup> If cardiac function does not recover, LVAD can allow a bridge to heart transplantation. Reiss et al. reported that seven patients with fulminant myocarditis were treated with ventricular assist devices, and four of them could be successfully bridged to heart transplantation after a mean support time of 163 days.<sup>17</sup>

We have described that a patient survived the acute phase crisis of fulminant myocarditis with LVAD and was discharged with no complications. The initial presentations of fulminant myocarditis are fatal; however, an appropriate application of mechanical circulatory support has supplied a good outcome. LVAD should be introduced before MOF associated with longer PCPS develops.

## References

- Lieberman EB, Hutchins GM, Herskowitz A, Rose NR, Baughman KL. Clinicopathologic description of myocarditis. *J Am Coll Cardiol* 1991; **18**: 1617-26.
- Aretz HT, Billingham ME, Edwards WD, Factor SM, Fallon JT, et al. Myocarditis. A histopathologic definition and classification. *Am J Cardiovasc Pathol* 1987; **1**: 3-14.
- Takeda Y, Yasuda S, Miyazaki S, Daikoku S, Nakatani S, et al. High-dose immunoglobulin G therapy for fulminant myocarditis. *Jpn Circ J* 1998; **62**: 871-2.
- Shioji K, Matsuura Y, Iwase T, Kitaguchi S, Nakamura H, et al. Successful immunoglobulin treatment for fulminant myocarditis and serial analysis of serum thioredoxin: a case report. *Circ J* 2002; **66**: 977-80.
- Abe S, Okura Y, Hoyano M, Kazama R, Watanabe S, et al. Plasma concentrations of cytokines and neurohumoral factors in a case of fulminant myocarditis successfully treated with intravenous immunoglobulin and percutaneous cardiopulmonary support. *Circ J* 2004; **68**: 1223-6.
- Chen YS, Yu HY, Huang SC, Chiu KM, Lin TY, et al. Experience and result of extracorporeal membrane oxygenation in treating fulminant myocarditis with shock: what mechanical support should be considered first? *J Heart Lung Transplant* 2005; **24**: 81-7.
- Tokunaga S, Morita S, Masuda M, Tomita Y, Nishida T, et al. How to cope with the pitfalls of extracorporeal membrane oxygenation support: case report of a girl with fulminant myocarditis. *J Artif Organs* 2007; **10**: 115-7.
- Aoyama N, Izumi T, Hiramori K, Isobe M, Kawana M, et al. National survey of fulminant myocarditis in Japan: therapeutic guidelines and long-term prognosis of using percutaneous cardiopulmonary support for fulminant myocarditis (special report from a scientific committee). *Circ J* 2002; **66**: 133-44.
- Peek GJ, Killer HM, Sosnowski MA, Firmin RK. Modular extracorporeal life support for multiorgan failure patients. *Liver* 2002; **22** (Suppl 2): 69-71.
- Reinhartz O, Farrar DJ, Hershon JH, Avery GJ Jr, Haeusslein EA, et al. Importance of preoperative liver function as a predictor of survival in patients supported with Thoratec ventricular assist devices as a bridge to transplantation. *J Thorac Cardiovasc Surg* 1998; **116**: 633-40.
- Hata M, Shiono M, Orime Y, Yagi SY, Yamamoto T, et al. Strategy of circulatory support with percutaneous cardiopulmonary support. *Artif Organs* 2000; **24**: 636-9.
- Gojo S, Kyo S, Sato H, Nishimura M, Asakura T, et al. Successful LVAS and RVAS-ECMO support in a patient with fulminant myocarditis who failed to recover from ventricular fibrillation with PCPS and IABP. *J Thorac Cardiovasc Surg* 2003; **126**: 885-6.
- Rockman HA, Adamson RM, Dembitsky WP, Bonar JW, Jaski BE. Acute fulminant myocarditis: long-term follow-up after circulatory support with left ventricular assist device. *Am Heart J* 1991; **121** (3 Pt 1): 922-6.
- Maybaum S, Stockwell P, Naka Y, Catanese K, Flannery M, et al. Assessment of myocardial recovery in a patient with acute myocarditis supported with a left ventricular assist device: a case report. *J Heart Lung Transplant* 2003; **22**: 202-9.
- Leprince P, Combes A, Bonnet N, Ouattara A, Luyt CE, et al. Circulatory support for fulminant myocarditis: consideration for implantation, weaning and explantation. *Eur J Cardiothorac Surg* 2003; **24**: 399-403.
- Holman WL, Bourge RC, Kirklin JK. Case report: circulatory support for seventy days with resolution of acute heart failure. *J Thorac Cardiovasc Surg* 1991; **102**: 932-4.
- Reiss N, El-Banayosy A, Arusoglu L, Blanz U, Bairaktaris A, et al. Acute fulminant myocarditis in children and adolescents: the role of mechanical circulatory assist. *ASAIO J* 2006; **52**: 211-4.