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# A Case of Fulminant Myocarditis Successfully Treated with Percutaneous Cardiopulmonary Support (PCPS)

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**Abstract:** A 36-year-old woman with fulminant myocarditis, cardiogenic shock and disseminated intravascular coagulation was successfully treated by percutaneous cardiopulmonary support (PCPS) and an intra-aortic balloon pump (IABP). She made a gradual recovery and could be successfully weaned from the PCPS after support periods of 68 hours without complications. Her left ventricular function was has since been maintained for 20 months. Fulminant myocarditis requires aggressive therapy for rescue, and PCPS is an effective treatment modality for avoiding ischemia of important organs while also achieving a sufficient level of cardiopulmonary assist.

**Key words: Fulminant myocarditis, PCPS, Case**

## Introduction

In most patients, myocarditis is asymptomatic and recovery is uneventful. However, a small number of myocarditis patients demonstrate acute and critical symptoms ranging from heart failure, atrial and ventricular arrhythmias, conduction disturbances, systemic emboli, cardiogenic shock and death.[1] In the acute setting, a viremic patient with hepatorenal dysfunction is rarely indicated for cardiac transplantation.[2] The potential for a complete recovery was recently recognized in young fulminant myocarditis patients who were maintained by ventricular support with extra corporeal blood pumps.[3][4] Many devices, such as the pneumatic left ventricular assist device (LVAD) and percutaneous cardiopulmonary support (PCPS) are currently available for mechanical support.[5] In this case report, we describe a case of early myocardial recovery during support with PCPS.

## Case Report

A 36-year-old female was admitted in the late night on August 19, 2001, because of a high fever (39℃), general fatigue, palpitation and effort dyspnea for 3 days. An electrocardiogram showed slow ventricular tachycardia (Fig. 1A) and a transient complete A-V block. A chest X-ray revealed a cardiothoracic ratio of 56% and slight lung congestion. Laboratory studies revealed a white blood cell count of 3,600/mm$^3$ and C-reactive protein of 3.9 mg/dL. Serum aspartate aminotransferase (AST) was 199 IU/L (normal range, ＜33 IU/L), lactate dehydrogenase (LDH) was 1,531 IU/L (normal range, 260－485 IU/L), creatine kinase (CK) was 841 IU/L (normal range, 47－195 IU/L), troponin-T was 7.29 ng/mL (normal range, ＜0.1 ng/mL) and myosin light chain I (MLCI) was 38 ng/mL (normal range, ＜2.5

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ng/mL). The alanine aminotransferase (ALT) (24 IU/L) and gamma－glutamyltransferase (γ－GTP) (23 IU/L) levels were normal. Two－dimensional echocardiography showed cardiac dilatation with diffuse left－ventricular hypokinesia including severe hypokinetic septal and anterior left－ventricular wall. After hospitalization, the physical findings showed a body temperature 39.2℃, blood pressure 94/60 mmHg with sinus rhythm (85/min), and the heart sounds were normal. During the first 6 hours, her consciousness was clear, and her blood pressure was stable. On the second day of hospitalization, she had marked bradycardia by complete A－V block (Fig. 1B), she was gradually losing consciousness, and finally she went into cardiac shock (blood pressure 60－70 mmHg). Temporary pacemaker was inserted, but her blood pressure could not control with these treatments. Laboratory data showed elevated AST (16,140 IU/L), LDH (42,600 IU/L), and CK (923 IU/L) levels. She developed ischemic hepatitis— the ALT level peaked at 6,910 U/L (normal range, ＜35 U/L), total bilirubin, 3.9 mg/dL and low cholinesterase level, 150 IU/L (normal range, 220–470 U/L) ; and disseminated intravascular coagulation — international normalised ratio (INR), 3.4 (normal range, 1.0－1.2) ; activated partial thromboplastin time (APPT), 54 s (normal range, 24–38 s) ;

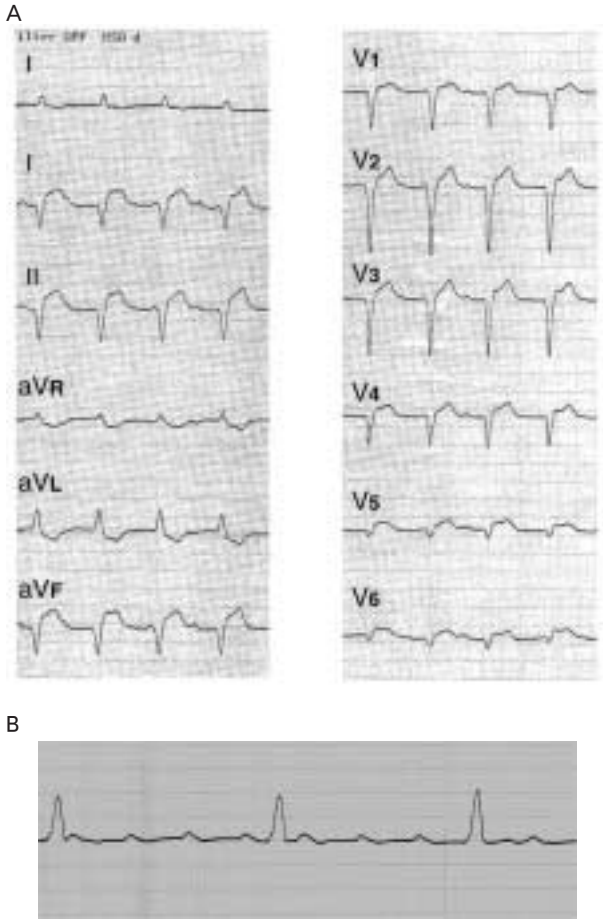


**Fig. 1.** ECG at hospital admission (A), a complete A–V block (B)

fibrin degradation products (FDP), >80 μg/mL (normal range, <5 μg/mL), platelet count, $5.1\times10^4/\mu L$ (normal range, $12.7–35.6\times10^4/\mu L$) ; fibrinogen level, 140 mg/dL (normal range, 150–350 mg/dL). She had normal coronary arteries on catheterization, but she demonstrated global hypokinesis with an ejection fraction of 30% on an echocardiogram.

Because of disseminated intravascular coagulation, an endomyocardial biopsy was not performed. Over the next two hours, despite the insertion of an intra-aortic balloon pump, she had a low mean arterial pressure of 62 mmHg. As measured by a Swan-Ganz catheter, a cardiac index (CI) of $2.0\ L\cdot min^{-1}\cdot m^{-2}$ and pulmonary wedge pressure (PCWP) of 22 mmHg were observed. A worsening of respiratory distress necessitated intubation and ventilation to maintain adequate oxygen saturation. Fulminant myocarditis was diagnosed based on the clinical findings including the laboratory data, electrocardiogram, chest X-ray and echocardiography findings. Because of a lack of an improvement, cannulas (15-Fr and 19-Fr) were inserted from the left femoral artery and right femoral vein, respectively, and PCPS was initiated at a flow rate of 3 L/min. On the second day after PCPS, the ejection fraction, as measured by echocardiography, decreased to 10% (Fig. 2A). After the institution of PCPS, her condition stabilized. She made a gradual recovery and could be successfully weaned from the system after support peri-

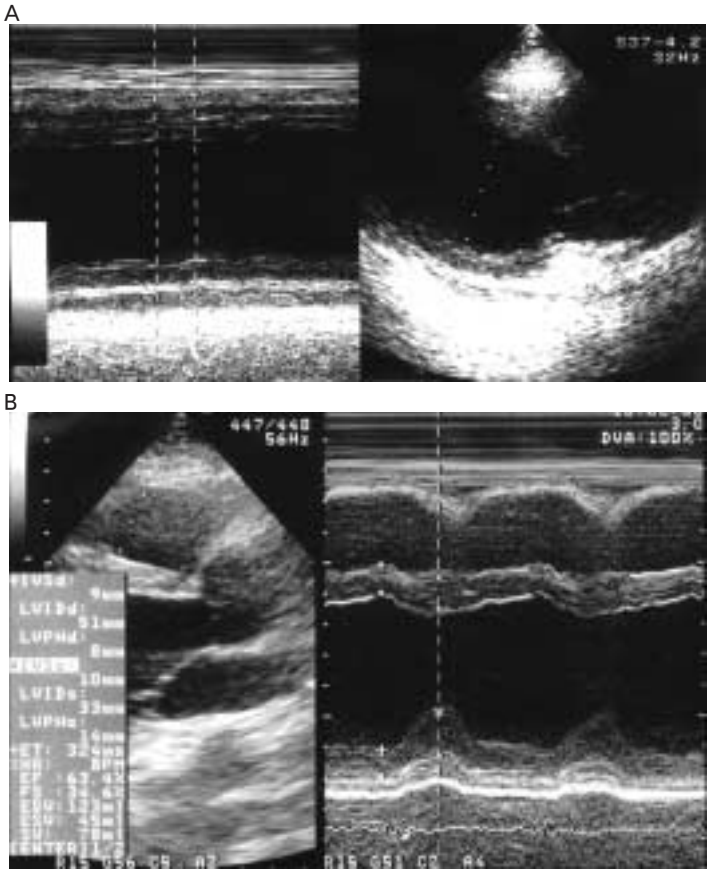


**Fig. 2.** M-mode and two-dimensional echocardiograms pre-treatment (A) and post-treatment (B) are shown. The left ventricular wall motion recovered following percutaneous cardiopulmonary support.

ods of 68 hours without complications. The liver function and coagulation both returned to the normal range (Fig. 3). The cardiac symptoms on the 7th day was classified as New York Heart Association class I, with both the cardiothoracic ratio and left ventricular ejection fraction showing near-normal values. Repeat echocardiography performed 10 days after admission showed a marked improvement in left ventricular systolic contraction (ejection fraction 63%), which was now globally only mildly reduced (Fig. 2B). The paired-sera titration of antibodies to virus disclosed no significant increase in titer. She was discharged on September 29, 2001, under oral medication with 5 mg of carvedilol and 4 mg of candesartan per day. She has returned to normal life and has been followed up every other month at the out-patient clinic of Fukuoka University Hospital. At 20 months after the event, her left ventricular ejection fraction was 60% and end-diastric left ventricular dimension was 52 mm, as measured by echocardiography, and she has since had no signs of heart failure.

## Discussion

Fulminant myocarditis can be the cause of rapid cardiac decompensation which is resistant to maximal medical therapy. Successful weaning from left ventricular mechanical support is relatively rare in fulminant myocarditis.[6)7)] However, successful results with ventricular support systems have also been recorded recently in individuals with fulminant myocarditis.[4)8)9)]

We herein reported the case of a 36-year-old woman with fulminant myocarditis who was successfully weaned from a PCPS with almost a full recovery of her myocardial function. Our patient presented with acute palpitation, features of shock and ECG changes strongly suggestive of acute myocardial disease, in addition to an increased CPK and gross LV systolic dysfunction. Serial frequent ECGs, good quality echocardiograms, antiviral antibody titres both in acute and convalescent sera, and in suspected cases an endomyocardial biopsy, are essential for the proper diagnosis and management of this disease. However, in this case, the antiviral antibody titres of the major virus related to myocarditis did not increase more than four-fold compared to the control. We could not perform a myocardial biopsy in this case. Febrile onset, clinical, ECG and ventricular function recovery in a few weeks with normal haemogram and biochemical parameters were

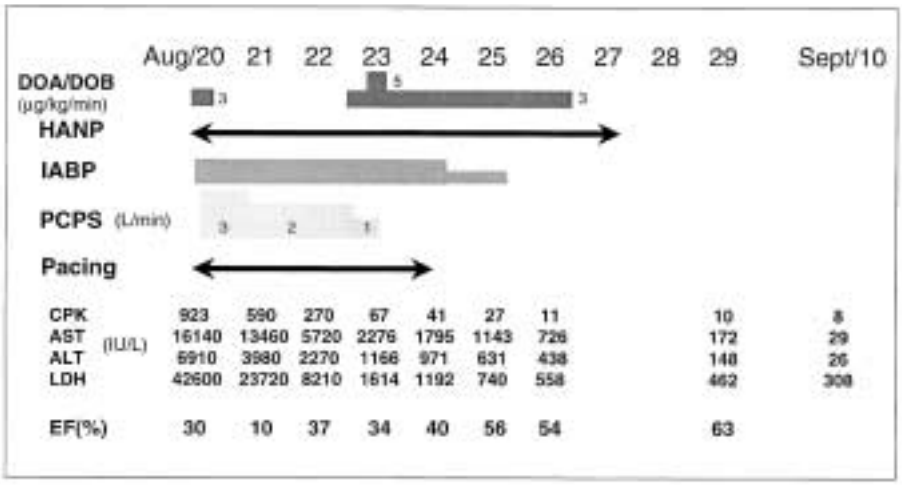


**Fig. 3.** Clinical course of the patient.

DOA, dopamine ; DOB, dobutamine ; HANP, human atrial natriuretic peptide ; EF, left ventricular ejection fraction

highly suggestive of myocarditis in the convalescent period. The diagnosis of myocarditis was entertained only when the coronary function was normal and the cardiac function was recovered. Similar cases of myocarditis in young adults with ECG changes masquerading acute myocardial infarction have also been previously reported.[10] A young adult presenting with chest pain and ECG changes stimulating acute myocardial infarction is likely to be afflicted with myocarditis.

Many devices, such as LVAD and PCPS are currently available for mechanical support. The proper selection of patients for a given therapy has a major impact on the clinical outcome.[4] The timely use of cardiopulmonary devices, before the onset of multiple or irreversible end-organ failure, is essential. The use of short-term devices is indicated if myocardial recovery is anticipated, as in cases of fluminant myocarditis.

A progression from viral myocarditis to dilated cardiomyopathy has long been hypothesized, but the actual extent of this progression remains uncertain. However, a causal link between viral myocarditis and dilated cardiomyopathy has recently become more evident than before owing to tremendous advances in the molecular analyses of autopsy and endomyocardial biopsy specimens. The beneficial effects of alpha 1-adrenergic blocking agents, carteolol, verapamil, and ACE inhibitors have been clinically and experimentally demonstrated in the treatment of viral myocarditis and dilated cardiomyopathy.[11] Fulminant myocarditis causes a distinct onset of illness and severe hemodynamic compromise. In contrast, acute myocarditis has an indistinct presentation, less severe hemodynamic compromise and a greater likelihood of progression to dilated cardiomyopathy.[12]

Patients with cardiogenic shock secondary to acute myocarditis in its fulminant presentation are surprisingly able to recover and thereby achieve a normal cardiac function. Based on the above findings, aggressive approach regarding the use of mechanical support is therefore considered to be strongly justified in the treatment of such cases.

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