A case of fulminant myocarditis with full recovery after a 38-h sustained asystole

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Abstract

A 69-year-old man was diagnosed with fulminant myocarditis with circulatory collapse. His cardiac rhythm deteriorated to asystole on the second day; however circulatory status was maintained through extracorporeal membrane oxygenation and intra-aortic balloon pumping. After 38h-lasting asystole, heart resumed beating. He was discharged without neurological deficits on day 25.

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Key clinical message

Even if cardiac rhythm deteriorated to asystole in the clinical course of fulminant myocarditis, cardiac function may recover, and the patient may be discharged without brain damage, if circulation could be maintained by appropriate mechanical cardiac supports.

Abstract

A 69-year-old man was diagnosed with fulminant myocarditis with circulatory collapse. His cardiac rhythm deteriorated to asystole on the second day; however, circulatory status was maintained through extracorporeal membrane oxygenation and intra-aortic balloon pumping. After 38h-lasting asystole, his heart resumed beating. He was discharged without neurological deficits on day 25.

Key Words

Asystole, Cardiac arrest, Extracorporeal membrane oxygenation, Fulminant myocarditis, Intra-aortic balloon pumping

Introduction

Fulminant myocarditis is an uncommon, but severe cardiac inflammatory disease that can be fatal. The disease is characterized by a severe and sudden onset marked by a rapid progressive deterioration that can occur within 2 or 3 days. The use of extracorporeal membrane oxygenation (ECMO) is common; therefore, its application in fulminant myocarditis has also increased. Several case reports indicated that mechanical circulation support (MCS) devices, including ECMO, which provides full circulatory support with time for the heart to recover. However, to our knowledge, there are no reports of a patient with fulminant myocarditis who subsequently recovered cardiac function after long-term asystole.

Case Presentation

A 69-year-old man with no history of cardiac disease was transported to a previous hospital with a fever of 39°C and fainting. An electrocardiogram (ECG) showed ST-segment elevation and elevated myocardial desensitization enzymes. Echocardiography revealed severe diffuse hypokinesis and pericardial effusion. Additionally, remarkable stenosis was not observed during coronary artery angiography. He was admitted to the previous hospital with suspected myocarditis. Three days after admission, the level of consciousness decreased, hepatobiliary enzymes increased, renal function worsened, and blood pressure decreased, thereafter he was transferred to our hospital.

On physical examination at our emergency department, his Glasgow coma scale was eyes: 4, verbal: 4, motor: 6; blood pressure (BP): 112/90 mmHg; heart rate (HR): 132 beats per minute (bpm), regular with no catecholamine support; respirations: 27 breaths per minute, oxygen saturation: 96% with a 4 L oral mask, and temperature was 37.2°C. The laboratory data at the time of arrival are shown in Table 1. Overall, white blood cells, C-reactive protein (CRP), and myocardial enzymes were prominently elevated. Troponin I levels were above 2000 ng/L. A 12-lead ECG showed ST-segment elevation in all guides (Figure 1). After admission to the intensive care unit, considering the possibility of a bacterial infection, relevant treatment was initiated. On day two, his HR increased to 180 bpm and his systolic BP dropped to 60 mmHg Echocardiography revealed significant decrease in ejection fraction to approximately 10%. The patient then fell into pulseless electrical activity; cardiopulmonary resuscitation occurred immediately, and the patient was resuscitated after administration of 1 mg of adrenaline. Since circulatory dynamics remained unstable, veno-arterial ECMO (V-A ECMO) and intra-aortic balloon pumping (IABP) were introduced. Intravenous high-dose methylprednisolone therapy (1000 mg for 3 days) and immunoglobulin therapy (0.5 kg/kg for 2 days) were also initiated. On day four ECG showed asystole; however, systolic BP was maintained at 80 mmHg under V-A ECMO with a flow of approximately 3 L/min and IABP with an internal trigger mode. Circulatory dynamics were maintained, therefore, intensive care was continued. After 38 h of asystole, electrical activity revived on ECG with a HR of 50-60 bpm (Figure 2). He responded to a call, suggesting cerebral function was maintained to some level. Cardiac function gradually improved, and the patient was weaned off V-A ECMO on day 14. The cause of fulminant myocarditis in this case was unclear, as we did not perform magnetic resonance imaging or myocardial biopsy at our hospital. Laboratory data that screened for causative viruses also showed no significant findings. On day 15, echocardiography showed an improved ejection fraction of approximately 60%. The patient was weaned from the IABP and extubated on day 18. After extubation, consciousness continued without any obvious higher functional impairment. On day 25, the patient was transferred to another hospital.

Discussion

Here, we report a case of fulminant myocarditis that recovered with no neurological damage at all after 38-h asystole. To our knowledge, this is the first reported case of fulminant myocarditis that recovered from long-term asystole. Cardiac function in fulminant myocarditis is often reversible and improves after overcoming the acute phase, although patients who suffered cardiac arrest have a poor prognosis, even with ECMO or IABP. There is also no established consensus on the reversibility of myocardial function, except in specific

environments, such as cardiac surgery under cardiopulmonary bypass. This case suggested that there is a possibility of subsequent improvement, even after prolonged asystole.

In fulminant myocarditis, temporary cardiopulmonary support is an important treatment approach as cardiac function can be restored following the acute phase. An observational study assessing fulminant myocarditis found that ECMO was equivalent to ventricular assist devices (VAD) and easier to introduce. Moreover, in fulminant myocarditis complicated by malignant ventricular arrhythmias, left VAD is unlikely to provide sufficient hemodynamic support when the right ventricle does not work effectively, whereas ECMO effectively bypasses biventricular failure. ECMO is the first treatment option for catastrophic myocarditis owing to low invasiveness, mobility for bedside implementation, and utility during cardiopulmonary resuscitation in cardiac arrests.

The concurrent use of devices such as VA-ECMO with IABP or Impella, rather than VA-ECMO alone, in patients with severe heart failure and cardiogenic shock may reduce the left ventricle load, minimize myocardial injury, and improve clinical outcomes. In severe cardiac dysfunction, the aortic valve does not open even when circulation is adequately secured by retrograde perfusion with VA-ECMO, and left ventricular thrombosis is a concern. The combination of IABP and V-A ECMO may reduce the risk of left ventricular thrombus by maintaining physiological antegrade blood flow [9]. However, despite the theoretical advantage, the effectiveness of the combined use of IABP and V-A ECMO, is inconclusive. Several observational studies have shown conflicting results, and there are currently no randomized trials[10]. In this case, the risk of ventricular thrombosis was significantly high when circulation was supported by V-A ECMO only, as no antegrade blood flow was generated during asystole. Therefore, additional use of IABP might have been effective for preventing intraventricular thrombosis and reducing light ventricular load in the present case, which might result in the recovery of cardiac function. Regrettably, the current cause of fulminant myocarditis was not identified; however, this case illustrates that cardiac function might be restored even after prolonged asystole with adequate hemodynamic support.

Conclusion

We encountered a case of fulminant myocarditis that recovered with no neurological damage at all after a 38-h asystole. Even if a fulminant myocarditis patient develops asystole on ECG, there might be a possibility of subsequent improvement.

Authors' Contributions

T.A.: Data and information collection; manuscript preparation, manuscript review, and literature review.

A.E.: Manuscript preparation, manuscript review, and literature review.

H.S. and K.S.: Data and information collection.

K.M., and Y.O.: Conception of the study; preparation of the manuscript draft.

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Conflict of interest

The authors have no conflict of interest to declare.

Data availability statement

All data generated during this study can be accessed through direct communication with the corresponding author and through the agreement of all research team members.

Ethical approval

The manuscript was approved by the Ethics Committee of Tsuchiura Kyodo General Hospital and Matsudo General Hospital.

Consent

Written informed consent was obtained from the patient to publish this report.

References

Table

Table 1: Laboratory data from the emergency room

Complete Blood Count Date	Complete Blood Count Date	Complete Blood Count Date	Biochemistry Date	Biochem
WBC	13800	/μl	T-Bil	0.74
Seg	87.2	%	AST	1315
Eo	1.2	%	ALT	484
Baso	0.2	%	LDH	1993
Mono	3.1	%	Γ-GTP	101
Lymph	8.3	%	CPK	8116
RBC	498	$\times 10^4/\mu l$	CK-MB	141
Hb	15.0	g/dl	Troponin T	>2000
Hct	42.9	%	BNP	562.2
PLT	6.1	$\times 10^4/\mu l$	BUN	48.4
			Cre	2.82
			Na	128
			Cl	97
			K	4.1
			Ca	7.6
			Tp	5.2
			Alb	4.1
			CRP	0.18

White blood cell : WBC, Segment cell : seg, Eosinophil : Eo, Basophil : Baso, Monocyte : Mono, lymphocytes : Lymph, Red blood cell : RBC, Hemoglobin : Hb, Hematocrit : Hct, Platelet : PLT, Total-Bilirubin : T-Bil, Direct- Bilirubin, Aspartate aminotransferase : AST, Alanine aminotransferase : ALT, lactate dehydrogenase : LDH, γ-Glutamyl transpeptidase : Γ-GTP, Creatine phosphokinase : CPK, Blood urea nitrogen : BUN, Creatinine : Cre, Total protein: TP, Albumin : Alb, C-reactive protein : CRP, Prothrombin Time : PT, Prothrombin Time-International Normalized Ratio : PT-INR, Activated partial thromboplastin time : APTT, Base excess : B.E