



# Extracorporeal Membrane Oxygenation Support on Acute Fulminant Myocarditis Associated with Parainfluenza Infection: A Case Report

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## Abstract

Acute fulminant myocarditis (AFM) is a clinical condition that can lead to sudden onset of rapidly progressing cardiogenic shock with significant arrhythmia and possible cardiac arrest. Mechanical circulation support has been reported to provide effective cardiac support in patients with AFM. In this article, we aimed to present the case of a 14-year-old girl with AFM who experienced a cardiogenic shock and resistant ventricular tachycardia unresponsive to medical treatment; however, she could be successfully treated with veno-arterial extracorporeal membrane oxygenation and discharged without neurological sequelae from the hospital.

**Keywords:** Extracorporeal membrane oxygenation, cardiogenic shock, myocarditis, parainfluenza, ventricular tachycardia

## INTRODUCTION

Acute fulminant myocarditis (AFM) is a clinical condition that can lead to sudden onset of rapidly progressing cardiogenic shock with significant arrhythmia and possible cardiac arrest. In patients with AFM who are unresponsive to medical treatment, mechanical circulation support can provide effective cardiopulmonary support (1-3).

In this article, we have presented the case of a 14-year-old girl with AFM who experienced cardiogenic shock and resistant ventricular tachycardia (VT) unresponsive to medical treatment, which was successfully treated with veno-arterial (VA) extracorporeal membrane oxygenation (ECMO).

## CASE PRESENTATION

A previously healthy 14-year-old girl was admitted to the emergency service with the complaint of upper respiratory tract infection for a week. She then developed complaints of

shortness of breath, fatigue, cough, and palpitations the next day. On physical examination, the patient's general condition seemed poor. She showed dyspnea and tachypnea, and had 87% oxygen saturation, 40/min respiratory rate, 190-bpm heart rate, and 90/50 mmHg blood pressure, with subcostal and intercostal retractions. On auscultation, bilateral breath sounds were decreased in the lower zones and extensive crepitation rales were present. On postero-anterior chest X-ray, bilateral blunting of the costophrenic and cardiophrenic angles as well as paracardiac infiltration was observed (Figure 1).

The patient's blood test examination results were as follows: White blood cells: 13,700/ $\mu$ L, hemoglobin level: 11.7 g/dL, platelet count: 133,000/ $\mu$ L, sodium level: 130 mmol/L, potassium level: 4.8 mmol/L, calcium: 8.2 mg/dL, lactate dehydrogenase: 878 U/L, aspartate aminotransferase (AST): 471 U/L, alanine aminotransferase (ALT): 696 U/L, C-reactive protein: 9.1 mg/L, urea: 46 mg/dL, creatinine level: 0.76 mg/dL, prothrombin time: 25.9 s (9.6-14.1), activated partial thromboplastin time:



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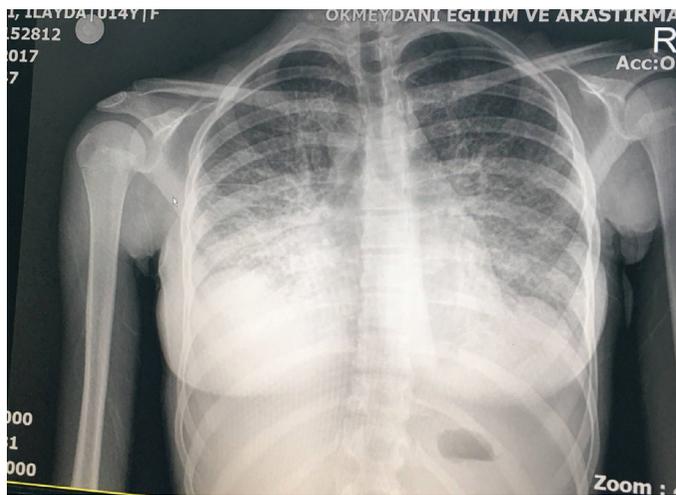
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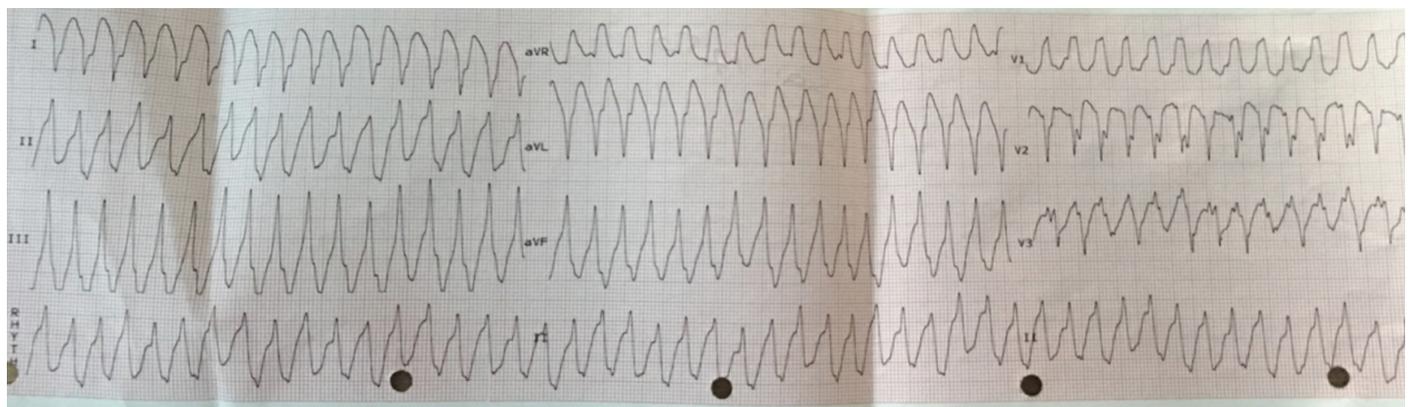
33.4 s (19-33), international normalized ratio: 2.32 (0.8-1.2), in the venous blood gas pH: 7.42, pCO<sub>2</sub>: 33.5, HCO<sub>3</sub>: 21.4, base excess: 2.29, lactate level: 1.6 mmol/L (the values between brackets refer to normal ranges). The patient was admitted with the diagnosis of pneumonia and pleural effusion. Due to complaints of chest pain and hypoxemia and VT observed in electrocardiography (Figure 2), the patient was transferred to the pediatric intensive care unit (PICU). Consent was obtained from the patient.

Transthoracic echocardiography (TTE) demonstrated large heart cavities in the left portion (left ventricular end-diastolic diameter: 54 mm), decreased systolic ejection fraction [(EF) 34%], and moderate insufficiency in the mitral valve (Figure 3).

The patient's condition was considered as AFM because of the rapid deterioration. Amiodarone was intravenously (IV) administered with a loading dose of 5 mg/kg twice for VT. However, due to continued VT, lidocaine (1 mg/kg/10 min) was administered as a loading dose and 25 µg/kg/min IV infusion as a maintenance dose. The blood test results revealed creatine



**Figure 1.** Patient's chest X-Ray



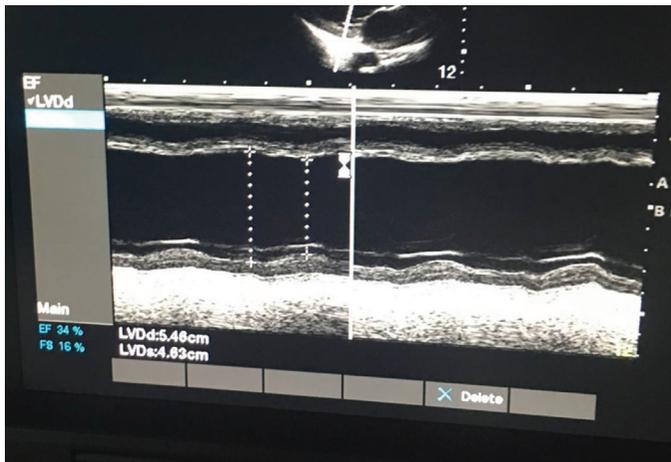
**Figure 2.** Patient's electrocardiogram with ventricular tachycardia

kinase myocardial band level of 6.5 U/L (0.6-6.3) and troponin level of 0.87 ng/mL (0.01-0.04).

After 12 h of admission to the PICU, the patient's blood tests were repeated, and the results were as follows: AST: 1,803 U/L, ALT: 1,679 U/L, urea: 63 mg/dL, albumin: 0.61 g/dL, venous blood gas pH: 7.38, pCO<sub>2</sub>: 30.1, HCO<sub>3</sub>: 19.5, BE: -6.5, and lactate: 11.3. The patient was connected to a mechanical ventilator with endotracheal intubation, and due to hypoperfusion, IV milrinone (0.5 mcg/kg/min) and low-dose IV noradrenalin (0.06 mcg/kg/min) were initiated as inotropic therapy; amiodarone dose was increased to 15 mg/kg/day; and lidocaine dose was also increased to 50 mcg/kg/min for resistant VT. Meanwhile, 1 g/kg IV immunoglobulin, 1 g pulse steroid, and 1 g/kg of 20% human albumin were also administered.

After 2 h, the patient's condition worsened. Systemic hypoperfusion (with metabolic acidosis, hypoxemia, and lactate elevation), hypotension (80/45 mmHg), and decreased EF in TTE (EF 10%) were observed. Because of the resistant cardiogenic shock and VT, the patient was connected to a VA-ECMO system. The cannulation sites were femoral artery with 18-F cannulae and jugular vein with 24-F cannulae. Then, the frequency of VT decreased. TTE was performed daily. EF was then gradually increased (on the 8<sup>th</sup> day of ECMO EF was 45%). VA-ECMO support was discontinued after 188 h. The tracheal aspirate sample was tested using multilocus polymerase chain reaction, and the sample was positive for parainfluenza virus. However, because of the hemodynamic stability and increased EF (to 45%), ribavirin was not administered. No complications were observed related to ECMO.

After the discontinuation of ECMO, VT was continued, and flecainide was administered instead of lidocaine, because of the 7-day maximum dose restriction. Beta blocker was also added to the regime. After 2 days, the patient was extubated, supported with a continuous positive airway pressure, and no additional oxygen was provided.



**Figure 3.** Patient's transthoracic echocardiogram

The frequency and duration of arrhythmia was decreased gradually and finally stopped. TTE before discharging from the hospital revealed that the left heart cavities were enlarged (left ventricular end-diastolic volume: 54 mm), left ventricular EF was 47%, and moderate mitral regurgitation with central origin was present. The patient was discharged from the hospital with no neurological sequelae, and triple antiarrhythmic drugs, enalapril, and furosemide were prescribed.

## DISCUSSION

The clinical condition of pediatric patients with AFM can worsen rapidly and unpredictably within hours of first presentation. AFM involves acute illness with hemodynamic derangement and ventricular arrhythmias due to a severe inflammatory process, and patients require the support of cardiac pump function and/or the urgent management of serious arrhythmias (4).

The selection of an appropriate therapy for ventricular arrhythmias focused on the prevention of sudden cardiac death and associated medical conditions. The management of arrhythmia may include appropriate antiarrhythmic therapy with drugs, implantable devices, ablation, or surgery. A beta blocker and amiodarone were recommended for recurrent polymorphic VT. IV lidocaine may be considered for the treatment of recurrent sustained VT or ventricular fibrillation (VF) in patients not responding to beta-blockers or amiodarone. Electrical cardioversion or defibrillation is recommended in patients with sustained and haemodynamically intolerated VT or VF. Urgent coronary angiography is indicated in these patients for revascularization when considering myocardial ischemia. Radiofrequency catheter ablation with the implantation of a left ventricular assist device/implantable cardioverter defibrillator

should be considered in patients with recurrent VT or VF not responding to revascularization and optimal medical treatment. When the conventional therapy of medication, implantable devices, and ablation fails to convert refractory VT, leading to cardiogenic shock, ECMO can be performed for maintaining the hemodynamic stability and end-organ perfusion until further decision or recovery (5,6). In our case, ECMO was considered as the mechanical support for AFM because of sustained ventricular tachyarrhythmia unresponsive to medical treatment and infeasible radiofrequency catheter ablation. However, the outcomes after ECMO for cardiac insufficiency in children are associated with underlying diseases (7). In a meta-analysis about the clinical outcomes in pediatric patients hospitalized with AFM and treated with ECMO, the survival rates were reported to be between 53.8% and 83.3% (8). Li et al. (9) reported the case of a 9-year-old girl with AFM, who rapidly worsened into pulseless VT and refractory cardiac arrest despite prolonged cardiopulmonary resuscitation for nearly 180 min and multiple antiarrhythmic therapy. They performed ECMO, and the girl recovered with intact cardiac and neurocognitive functions after 221 h of ECMO treatment. Similarly, in our report, after 188 h of ECMO treatment, our patient successfully recovered without cardiac and neurocognitive damage.

Recent reports with larger cohorts indicated that AFM is associated with overall worse outcomes that included lower left ventricular EF at the last follow-up, higher in-hospital mortality, and increased rates of cardiac transplantation (8-11). Mechanical circulation support can be considered at the first line of treatment. The disease has a high mortality unless provided with effective and on-time intervention with mechanical circulation support. The survival rate of patients with mechanical circulation support can rise up to 80%. Especially, in case of cardiogenic shock and resistant VT, mechanical circulation support is a good option for reducing the mortality rate and increasing the healing rate of myocardial muscles. As alternatives to mechanical circulation support, intra-aortic balloon pump, percutaneous cardiopulmonary support system, and ventricular assist device can be used. However, in children, ECMO is used more frequently because of its ease of use, low invasiveness, and more effective outcomes (3,12,13).

Viral infections are frequently the preliminary etiology of AFM. However, parainfluenza-related myocarditis has been less frequently reported (14,15). Parainfluenza virus was detected in the tracheal aspiration fluid of our patient. Ribavirin can be used as an antiviral agent (14-16). However, due to the fact that our patient's renal function was insufficient and EF increased to 40%,

ribavirin was not used. In addition, there are only limited studies available on the use of ribavirin in respiratory tract infections caused by parainfluenza virus. However, studies on whether IV immunoglobulin is effective or not has been reported in the past (15-17).

Our patient was administered pulse methylprednisolone with pentaglobulin. Corticosteroids reduce the amount of T lymphocytes, which are the mediators of ongoing heart damage in AFM through lysis. With a reduced number of T lymphocytes, cardiac ventricular function has been reported to improve (15-17).

## CONCLUSION

Treatment of cardiogenic shock and resistant VT related to AFM should involve a multidisciplinary approach, such as mechanical circulation support, antiarrhythmic drugs, inotropic drugs, immunoglobulin, steroids, and other supportive treatments.

### Ethics

**Informed Consent:** Consent was obtained from the patient.

**Peer-review:** Externally peer-reviewed.

### Authorship Contributions

Concept: A.İ., F.A., Design: A.İ., F.A., Data Collection or Processing: M.A., F.A., Analysis or Interpretation: M.A., F.A., Literature Search: M.A., Writing: M.A.

**Conflict of Interest:** No conflict of interest was declared by the authors.

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