

**Case Report** 

# Left Atrial Decompression on Extracorporeal Membrane Oxygenation of a Neonate with Fulminant Enteroviral Myocarditis

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#### Abstract

Initiation of extracorporeal membrane oxygenation may increase left ventricular volume, pressure and wall stress. Left atrial decompression may reduce ventricular wall stress and filling pressures, thus improving coronary perfusion. This may contribute to ventricular functional recovery.

The pediatric experience consists mainly of postoperative congenital heart disease patients. Scarce reports exist about neonates on extracorporeal membrane oxygenation due to other acute cardiac causes. We present a neonate with fulminant myocarditis with severe pulmonary edema upon extracorporeal membrane oxygenation initiation, which improved almost instantly after atrial decompression.

**Keywords:** Extracorporeal memebrane oxygenation; Lung edema; Atrial septostomy; Myocarditis

### Introduction

Extracorporeal membrane oxygenation (ECMO) is the primary mechanical circulatory support for pediatric patients with severe acute cardiac and/or pulmonary failure. However, in a failing heart, initiation of ECMO may increase left ventricular volume, pressure and wall stress, and may even result in myocardial ischemia [1]. Left atrial decompression may therefore be effective in reducing left ventricular wall stress, reducing left ventricular filling pressures and improving coronary perfusion [2]. This may increase the probability of left ventricular functional recovery.

Left atrial decompression of patients on ECMO has been shown to be both an effective and safe procedure [1-5]. However, the pediatric experience reported in the literature consists mainly of postoperative congenital heart disease (CHD) patients and cardiomyopathies [1-5]. Scarce reports exist about non-CHD neonates on ECMO [6,7]. We present a neonate with acute fulminant enteroviral myocarditis with severe pulmonary edema upon ECMO initiation.

## **Case Presentation**

An 11 day old neonate was admitted with symptoms of progressive congestive heart failure, including decreased appetite and malaise. In the emergency room she was found to be hypothermic and in shock with poor perfusion and tachycardia. She was intubated and treated with intravenous fluids and adrenaline. Echocardiography demonstrated normal heart morphology, with reduced segmental left ventricular contraction and moderate mitral regurgitation. Her troponin level was 15,000 ng/mL and brain natriuretic peptide level was 5,000 pg/mL. She was diagnosed with fulminant myocarditis with cardiogenic shock. Despite massive supportive treatment with vasoactive amines and mechanical ventilation her hemodynamic status and oxygenation index worsened, blood pressures were low, there was no urinary output, and severe lactic acidosis, therefore ECMO therapy was initiated.

Echocardiography while on ECMO showed a severe global and segmental myocardial dysfunction, predominantly posterior and inferior wall dyskinesis, fractional shortening of 13%, and free mitral regurgitation. ECMO flow was maintained at 400-500 mL/min. On chest x-ray severe pulmonary edema was noted. Polymerase chain reaction was positive for enterovirus. The pulmonary edema was considered to result from increased Left atrial pressure due to left ventricular dysfunction and free mitral regurgitation. It has been suggested that decompression of the left atrium by atrial septostomy may improve the pulmonary edema. In addition, creating a venting mechanism of the left atrium may increase the myocardial perfusion pressure.

The percutaneous atrial septostomy was performed bedside on day 2 with echo control, with no complications. Chest x-ray performed 3 hours later demonstrated marked improvement with clearing of the lung fields. The infant was weaned from ECMO on day 7. Repeat echocardiograms demonstrated improved myocardial function and residual moderate mitral regurgitation. The patient continued with furosemide and captopril. Due to feeding problems, presumably caused by bowel ischemia and reduced motility, a nasogastric tube and later a jejunostomy were installed. On the latest follow-up visit at the age of 6 months she is well-developed and nourished, shows no signs of congestive heart failure with captopril alone, and on echocardiogram there is mild segmental left ventricular wall motion abnormality (normal 32% fractional shortening) and mild to moderate

mitral regurgitation. The atrial septal defect is trivial and does not require interventional closure. The jejunostomy was successfully closed and she has no further feeding difficulties.

## Discussion

The majority of enteroviral infections are mild, self-limiting febrile diseases. Neonates may acquire enteroviral infections either vertically during pregnancy or delivery, or postnatally by close contact with infected caretakers. Myocarditis is an uncommon complication of enteroviral infection in neonates and may lead to devastating results. In our case the acute disease was fulminant and unresponsive to conservative measures, requiring the use of ECMO. Balloon atrial septostomy was performed to relieve left atrial pressure and the subsequent pulmonary edema, with a favorable result.

Pulmonary edema is a serious complication of ECMO support because it might delay successful weaning from ECMO even after cardiac function has recovered [2]. Left atrial decompression of patients on ECMO has been shown to be both effective and safe procedure. There is no consensus with regard to the timing of left atrial decompression; therefore it should be considered when lung edema is seen accompanied by left ventricular dysfunction. Reported complications included bleeding, hemolysis, atrial perforation, air in the circuit and central nervous system bleeding [2].

There are few reports describing non-CHD neonates on ECMO and the response of their myocardium to ECMO physiology. The largest series of left heart decompression in pediatric patients on ECMO are those of Hacking et al. and Boscamp et al. [2,3]. In these articles some of the patients were on ECMO because of myocarditis, but no age characteristics were given for the myocarditis patients group. Kotani et al., Callahan et al. and Abraham et al. reported cardiac catheterizations on ECMO respectively, but none of the patients was diagnosed with acute myocarditis [1,4,5].

We found scarce evidence for the safety and efficacy of left heart decompression in neonatal myocarditis patients on ECMO. Cofer et al. described 2 neonates with enteroviral myocarditis; one survived but remained with left ventricular dysfunction, the second died of widespread myocardial necrosis [6]. Koenig et al. reported 3 neonates with myocarditis (2 were enterovirus positive), of whom 2 survived [7]. Kim et al. published a successful atrial septostomy in a toddler with severe myocarditis and pulmonary edema on ECMO, but the age group is different from our case [8]. Our patient's pulmonary edema improved almost instantly after left atrial decompression, paving the way for successful weaning from ECMO. Although the general left ventricular dysfunction has markedly improved, there are still significant wall motion abnormalities of the infero-posterior segments.

# Conclusion

Little has been published regarding performing left atrial decompression on neonates with acute fulminant myocarditis. We described the clinical course, pathophysiology and management of this procedure in our patient. Our patient's lung edema promptly improved following the catheterization and further progress continued over the following days. Other case reports from more than 20 years ago showed mixed results. Larger series may pour light on the outcomes of left heart decompression in these infants.

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