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Case Report

Lung Reperfusion Injury after Congenital Cardiac Surgery in Pediatric Patients: Comparing Outcomes with and without Veno-Arterial ECMO Support

*Alvionita Nur Fitriana[1], Fajar Perdhana[1,2]**

[1]Study Program of Anesthesiology and Intensive Care, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia.

[2]Department of Anesthesiology and Reanimation, Dr Soetomo General Academic Hospital,, Surabaya, Indonesia.

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ABSTRACT

Lung reperfusion injury (LRI) is a rare but severe complication following pediatric cardiac surgery with cardiopulmonary bypass in patients with complex congenital heart disease. We report two cases of postoperative LRI with different clinical outcomes. The first patient, a 9-year-old female with tetralogy of Fallot, developed severe hypoxemic respiratory failure and ARDS on postoperative day 2 and recovered after timely veno-arterial extracorporeal membrane oxygenation (VA-ECMO). The second patient, a 14-month-old male with situs ambiguus, right isomerism, and double outlet right ventricle, progressed to ARDS and multiorgan failure despite maximal conventional therapy and died on postoperative day 10 without ECMO support. These cases emphasize the importance of early recognition of LRI and timely ECMO initiation in selected patients with refractory respiratory failure.

Introduction

Lung reperfusion injury (LRI) is a form of ischemia-reperfusion injury that occurs after restoration of blood flow to pulmonary tissue previously subjected to hypoperfusion or ischemia. It is characterized by increased vascular permeability, alveolar edema, impaired gas exchange, and often progresses to acute respiratory distress syndrome (ARDS), with reported mortality rates exceeding 30-50% in severe cases [1-2]. The pathophysiology involves complement activation, neutrophil infiltration, reactive oxygen species generation, and inflammatory cytokine release (TNF-α, IL-6, and IL-8), which collectively damage the pulmonary endothelium and increase alveolar-capillary permeability [3-4].

Postperfusion lung syndrome, the clinical manifestation of LRI, is well-established after pulmonary thromboendarterectomy and lung transplantation. However, LRI also occurs after repair of complex congenital heart disease with prolonged cardiopulmonary bypass (CPB) times, especially in patients with pulmonary hypertension, systemic-to-pulmonary collaterals, and critical pulmonary stenosis requiring surgical relief and sudden pulmonary reperfusion [5]. In the pediatric population, published reports of LRI outside the transplant setting remain limited, particularly comparing outcomes of ECMO versus conventional management [6]. Extracorporeal membrane oxygenation (ECMO) has become an essential modality for acute respiratory or cardiocirculatory failure refractory to conventional management in pediatric cardiac surgical patients [7]. Veno-arterial ECMO (VA-ECMO) provides both respiratory and hemodynamic support and allows

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*Corresponding author.
E-mail address: dr.fajarperdhana@gmail.com
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lung rest strategies by minimizing ventilator pressures and tidal volumes. Recent literature suggests optimal outcomes when ECMO is initiated early, ideally within the first 7 days of mechanical ventilation, before multiorgan failure develops [6].

This case report documents two pediatric patients with LRI following surgical correction of complex congenital heart disease—one managed with VA-ECMO with successful recovery and one managed conservatively without ECMO, resulting in fatal multiorgan failure. We aim to highlight the clinical spectrum of LRI, decision-making regarding ECMO candidacy, and prognostic implications in the pediatric setting.

Case Report

Case 1 – Tetralogy of Fallot with MAPCA

A 9-year-old female (19 kg, 115 cm) with tetralogy of Fallot and complex cardiac anatomy presented with progressive exertional dyspnea and cyanosis since age 2 (baseline SpO2 73%). She underwent total correction with relief of critical pulmonary stenosis, resulting in an improved McGoon ratio (1.8), no residual ventricular septal defect, and preserved ventricular function. On POD 2, she developed severe respiratory failure with ARDS, requiring veno-arterial extracorporeal membrane oxygenation and a lung-protective strategy after failing conventional management. Chest radiography on POD 2 showed alveolar pulmonary edema, bilateral pleural effusions, and a right-sided hydropneumothorax (Figure 1a). Adjunctive therapies included inhaled nitric oxide, diuretic infusion, low-dose hydrocortisone, broad-spectrum antibiotics, and optimized sedation. She improved on ECMO and was decannulated by POD 5. By POD 9, ARDS had resolved and her SpO_2 was 98%. A follow-up chest radiograph obtained on POD 9, after ECMO decannulation, is shown in Figure 1b. She was positioned for successful extubation and is anticipated to have good long-term pulmonary and cardiac recovery. On POD 11, the patient was extubated and showed very good improvement. The patient was then discharged from the hospital on POD 21.

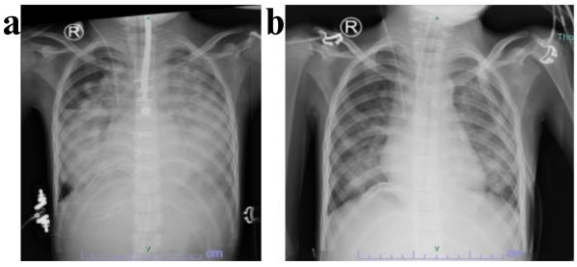


Figure 1- (a) Chest X-ray showed alveolar lung edema with bilateral pleural effusion and fluidopneumothorax dextra on POD 2 (b) after decannulation from ECMO on POD 9.

Case 2 – DORV Fallot Type with Complex Anatomy

A 14-month-old male (6 kg, 70 cm) with complex congenital heart disease presented with cyanosis since 6 months of age and failure to thrive (baseline SpO2 72%). Double outlet right ventricle repair was performed via median sternotomy with cardiopulmonary bypass. Post-bypass transesophageal echocardiography confirmed successful correction with mild pulmonary stenosis, mild pulmonary regurgitation, and a small residual ventricular septal defect. The patient remained relatively stable on low-dose inotropic support and FiO2 0.5 during postoperative days (POD) 0–1. However, beginning on POD 2, progressive respiratory deterioration developed, characterized by increasing oxygen requirements, bilateral alveolar infiltrates, and worsening oxygenation (Figure 2). By POD 4, the criteria for acute respiratory distress syndrome were met. Key complications that followed included the following:

(1) acute kidney injury (serum creatinine >0.6 mg/dL despite renal replacement therapy); (2) hepatic dysfunction (transaminases >250 U/L); (3) coagulopathy (INR >2.5, platelets $<50 \times 10^3/\mu L$); (4) presumed sepsis by POD 5–6; (5) vasopressor-resistant shock with rising lactate and profound acidosis; and (6) asystolic cardiac arrest on POD 10. Despite maximal organ support—including high-dose inotropes/vasopressors, aggressive fluid management, broad-spectrum antibiotics, stress-dose corticosteroids, and peritoneal dialysis—multiorgan failure ensued. Thirty minutes of cardiopulmonary resuscitation failed to achieve a return of spontaneous circulation. Death was declared.

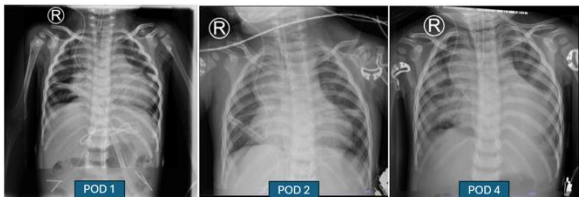


Figure 2- Serial chest X-ray on a 14-month-old male with complex congenital heart disease

Discussion

Lung reperfusion injury (LRI) in pediatric heart surgery is a specific injury that occurs when blood flow is restored to lungs that had previously been poorly perfused. This sudden increase in blood flow can damage the inner lining of blood vessels, make lung tissue leakier, and cause lung swelling. In both cases discussed, the problem came from blockages caused by birth defects: poor blood flow in central lung arteries due to severe narrowing and special blood vessel connections. When surgeons removed the blockage and restored normal blood flow, the lungs suddenly received more oxygen-rich blood, triggering this kind of injury [8].

At the molecular level, reperfusion triggers oxidative stress, inflammation, and breakdown of the vascular barrier via interconnected pathways. Rapid re-oxygenation generates reactive oxygen species, including the superoxide anion, the hydroxyl radical, and hydrogen peroxide. These overwhelm the body's natural antioxidant defenses and cause damage to cell membranes, proteins, and DNA. At the same time, activation of complement (C3a, C5a), cell receptors, and damage signals draws neutrophils, macrophages, and lymphocytes into the lung tissue, worsening the inflammation [4,9]. These cells release inflammatory mediators such as TNF-α, IL-6, and IL-8, as well as enzymes that degrade tissue. As a result, cell-to-cell connections are damaged, the blood vessel barrier fails, and protein-rich fluid leaks into the air sacs. The condition appears on scans as fluid in both lungs and severe hypoxemia, which matches ARDS. Activation of the regulation pathway and mitochondrial dysfunction further amplify tissue injury and impair recovery. Tissue factor exposure and platelet activation promote the formation of microthrombi within the pulmonary microvasculature, exacerbating ventilation–perfusion mismatch and increasing right ventricular afterload; simultaneously, reperfusion-induced aberrant calcium influx into mitochondria opens the mitochondrial permeability transition pore, uncoupling oxidative phosphorylation, depleting cellular energy stores, and precipitating apoptosis in both pulmonary endothelial and myocardial cells. These mechanisms explain the acute respiratory and hemodynamic decompensation observed in both patients, the development of refractory hypoxemia and right heart strain, and the critical need for advanced supportive interventions [4,9-10].

Clinically, LRI must be distinguished from pneumonia, transfusion-related acute lung injury, cardiogenic pulmonary edema, and isolated pulmonary hypertensive crisis. Management strategies and prognostic implications differ substantially. In both cases, early temporal relation to cardiopulmonary bypass, a characteristic radiographic pattern of bilateral alveolar edema without a cardiogenic cause, preserved left ventricular systolic function, and limited early microbiological evidence supported LRI as the main process [2,11]. Case 1 was a 9-year-old with an adequate body size, older age, and fulminant but isolated respiratory failure. He responded well to early VA-ECMO, with radiologic resolution and good recovery. Case 2 was a 14-month-old with extreme anatomical complexity, very small body size, pre-existing pulmonary compromise, and progressive organ dysfunction. Despite maximal management, he experienced refractory shock and died from organ failure. These cases show that, while the pathophysiology of LRI was similar, patient age, body size, disease severity, and the timing of organ involvement strongly influenced outcomes and determined the benefit of rescue therapies such as ECMO.

Conclusion

Two cases demonstrate severe lung reperfusion injury following complex congenital cardiac surgery, each resulting in a different outcome. The first case concerns a 9-year-old patient with tetralogy of Fallot who developed fulminant acute respiratory distress syndrome (ARDS) on postoperative day 2 and achieved full recovery after early initiation of veno-arterial extracorporeal membrane oxygenation (ECMO). The second case involves a 14-month-old patient with a double outlet right ventricle who progressed to ARDS with multiorgan failure despite maximal therapy and ultimately died from refractory shock. These cases underscore that high-risk anatomy with chronically hypoperfused pulmonary arteries increases susceptibility to lung reperfusion injury. Rapid recognition, implementation of lung-protective ventilation, and timely initiation of ECMO before the onset of multiorgan dysfunction are critical for survival. Advanced age, greater body size, and limited systemic involvement are associated with improved outcomes.

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