

# **Postoperative Management of Lung TPL**



## **Kyeongman Jeon**

Organization
Current Position

Sungkyunkwan University School of Medicine, Samsung Medical Center

Professor

#### **Educational background**

2005-2011 Ph.D., Sungkyunkwan University 2001-2003 M.A., Sungkyunkwan University 1993-1999 B.A., Dongguk University

#### **Professional experience**

2010-Present Professor, Samsung Medical Center, Sungkyunkwan University School of Medicine

Lung transplantation for patients with various end-stage lung diseases has become an increasingly standard therapy. The first lung transplantation in Korea was performed in 1996, and the numbers have steadily increased with approximately 150 lung transplantations reported annually since 2019. However, the number of patients transplanted from intensive care units (ICUs) with mechanical respiratory support has steadily increased based on the allocation system with medical urgency, technical improvements, and reports of beneficial outcomes. Therefore, the need for intensive care before and after lung transplantations has increased in recent years, along with the multidisciplinary team approach. Furthermore, critical care issues are involved in the early postoperative complication management, as well as the safe bridging of candidates by mechanical respiratory support.

The spectrum of early postoperative complications after lung transplantation varies, including primary graft dysfunction, arrhythmias, infections, non-pulmonary organ failure, and anastomotic and pleural complications, as well as postoperative bleeding. Therefore, prevention and adequate management of these complications in the early postoperative period may have significant impact on lung graft function and clinical outcome. Some complications may be preventable or easy to recover from if managed appropriately. However, management strategies are less clear since robust evidence from systematic evaluation or clinical trials is not fully available in many situations. In addition, other basic issues contribute to increased caring difficulty for recipients in the ICU, including mechanical ventilation (MV) support and hemodynamic management. The current practice in the ventilation and hemodynamic management is mainly based on the results of observational studies, underlying pathophysiology, and expert opinions as high-quality evidence-based guidelines are not yet available. Therefore, the practical post-transplant intensive care management aspects, especially ventilation and hemodynamic management during early postoperative period in the ICU, will be addressed.

### Postoperative Monitoring in the ICU

Patients typically arrive in the ICU with a pulmonary artery catheter (PAC) in addition to venous and arterial lines in place, chest tubes to drain pleural spaces, and an indwelling bladder catheter in place to fully monitor gas exchange, hemodynamics, amount of mediastinal and pleural drainage, and urinary output. ICU monitoring is similar to intraoperative monitoring. Monitoring in the ICU is essential because it provides information on the patient's clinical status, diagnostic assessment of complications, and future management plans, while monitoring in the operating room is designed to assess acute change of vital functions resulting from response to medication and surgical manipulation. Therefore, arterial blood gas analyses should be performed regularly for early detection of gas exchange abnormalities and acid-base imbalance. This will guide the appropriate adjustments for respiratory and metabolic supports.

The PAC is routinely used from the operating room even though there is a paucity of data on it's use in the postoperative



lung transplantation period. One of the most common indications for the PAC use is severe pulmonary hypertension given its high reliability in measuring beat—to beat pulmonary artery pressure. Moreover, the PAC can determine the right ventricle (RV) function, which might be associated with lung transplantation prognosis. However, several studies have suggested that RV function normalizes in adult patients after lung transplantation, even in patients with severe preoperative RV dysfunction. Therefore, there is no need to monitor the pulmonary hypertension or RV function postoperatively unless there are other problems that affect the pulmonary artery pressure. Furthermore, cardiac function could be evaluated with bedside echocardiography, including RV function. Therefore, PAC itself has no potential for benefit unless it guides therapies that improve patient outcomes in lung transplantation.

#### **Management of Mechanical Ventilation**

The goals of MV following lung transplantation are to promote graft function, maintain adequate gas exchange, and prevent ventilator-induced lung injury. There are no large, multicenter trials to guide MV management after lung transplantation despite the critical role of MV in lung transplantation, and there are only a few studies addressing appropriate MV after lung transplantation. This demonstrated wide variation in practices. Nonetheless, the currently applied lung protective MV strategies in lung transplantation have been extrapolated from the practice guideline on MV patients with acute respiratory distress syndrome (ARDS) since experimental data suggests that all lung transplantation recipients are at risk of ventilator induced lung injury. In addition, the lung protective ventilation benefits extend to surgical patients at risk for ARDS. Interestingly, however, a recent survey addressing MV practice after lung transplantation has shown that many of the reported practices did not conform to consensus guidelines on ARDS management.

Although low tidal ventilation (usually 6 mL/kg of predicted body weight) has been the preferred strategy even in lung transplantation, recipient characteristics from a survey on MV following lung transplantation most commonly determine tidal volume. Titrating tidal volume to donor-predicted body weight rather than recipient-predicted body weight reduces the risk of delivering insufficient or excessive tidal volume in size-mismatched allografts. However, undersized allografts might receive relatively higher tidal volumes compared with oversized allografts based on the donor-predicted body weights. Therefore, adjustments to the adequate tidal volume should be made based upon gas exchange over the next several hours following initial low tidal volume ventilation.

Weaning from MV is usually completed within 72 h and extubation performed in the ICU in non-complicated patients after lung transplantation. The median MV duration after lung transplantation is 2 to 3 days. MV are usually intentionally weaned in slow manner in patients with a high risk of severe graft dysfunction or inadequate gas exchange. Lung allografts have disrupted nerve supply as a donor harvesting consequence. Weak cough, poor respiratory mechanics caused by deconditioning, and inadequate pain control lead to inability to clear airway secretions. Early tracheostomy should be considered when weaning from MV is longer than 1 week.

In patients with severe graft dysfunction, MV may be insufficient to provide adequate gas exchange and high ventilator settings may be harmful to the allograft. ECMO support could be efficient and a rescue therapy for this critical presentation. It is generally accepted that early ECMO institution leads to improved salvage rates. Several case series have shown that recipients with refractory graft dysfunction requiring ECMO experienced long-term survival similar to that reported in patients not supported with ECMO. These data support ECMO use for severe graft dysfunction management, particularly to correct refractory hypoxemia and to reduce additional damage from MV to the already injured graft. The high incidence of complications, such as bleeding, vascular injury, and neurologic deficits, has been one of the major concerns when using ECMO in the postoperative period of lung transplantation, although the incidence of such complications has dramatically decreased in recent years.

#### **Management of Hemodynamics**

The initial hemodynamic management goal following lung transplantation is to maintain adequate organ perfusion, which is monitored by measuring lactate, urine output, and mixed venous oxygen saturation, if available. However, transplanted lungs have varying degrees of pulmonary edema as a result of increased vascular permeability and disrupted lymphatic drainage. In addition, increasing cardiac output with inotropes with or without vasopressors may also contribute to pulmonary edema by increasing the amount of flow through the lung allografts. Therefore, individualized management is required to maintain adequate perfusion pressure balance with the lowest possible cardiac output to reduce the exacerbating pulmonary edema risk. Postoperative volume status management is actually an area of considerable heterogeneity in practices. Dedicated protocol implementation that maintain specific hemodynamic targets has been shown to be associated with reduced graft dysfunction severity. In addition, the use of fluid restriction strategies was found to be unrelated to increased vasopressor use or renal function deterioration. These data suggest the potential benefits of more aggressive diuresis and fluid restrictions in the early postoperative period of lung transplantation.



Systemic inflammatory response syndrome from surgical insult with low systemic vascular resistance, medication-induced hypotension, hemorrhage, tamponade, and heart failure are generally the cause of hypotension, which is common in the immediate postoperative setting. Management should be causally determined including volume management and transfusions, vasopressor or inotrope application, bleeding diatheses correction, and surgical revision. Patients with a low systemic vascular resistance may need additional vasopressor treatment, with norepinephrine and vasopressin being the preferred agents. Vasopressin does not increase pulmonary vascular resistance since there is a lack of V1 receptors in the pulmonary arteries. Several studies have demonstrated that vasopressin can effectively ameliorate systemic hypotension without increasing pulmonary vascular resistance. Norepinephrine has been shown not only to slightly increase pulmonary vascular resistance but also to improve RV function through its inotropic effects. This may indicate an advantage of norepinephrine in patients with impaired RV function requiring vasopressors, but it has arrhythmia risk. On the other hand, the vasopressin addition to catecholamine vasopressors was significantly associated with a lower atrial fibrillation risk. Therefore, the choice of inotropes and vasopressors in postoperative lung transplantation care should take into consideration their effects on systemic and pulmonary vascular resistance and should be individualized based on patient response.

Pulmonary edema risk is higher with a transient diastolic dysfunction of the left ventricle (LV), which becomes incapable of handling a normal preload in the early postoperative period in patients with significant pulmonary hypertension before lung transplantation. The small and "deconditioned" LV of patients with severe pulmonary hypertension are prone to developing diastolic dysfunction when exposed to a normal or high preload after transplantation, resulting in elevated left-sided filling pressures and pulmonary edema. Bridging this period with veno-arterial ECMO has been described for the postoperative management of recipients with severe pulmonary hypertension to specifically address these issues and allow time to recovery of the "deconditioned" LV, which can take several days.