Published in final edited form as:

J Surg Res. 2013 August ; 183(2): 916–921. doi:10.1016/j.jss.2013.02.008.

Influence of hemodialysis on clinical outcomes after lung transplantation

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Abstract

Background—Chronic renal failure after lung transplantation is associated with significant morbidity. However, the significance of acute kidney injury (AKI) after lung transplantation remains unclear and poorly studied. We hypothesized that hemodialysis (HD)-dependent AKI after lung transplantation is associated with significant mortality.

Materials and methods—We performed a retrospective review of all patients undergoing lung transplantation from July 1991 to July 2009 at our institution. Recipients with AKI (creatinine > 3 mg/dL) were identified. We compared recipients without AKI *versus* recipients with and without HD-dependent AKI. Kaplan-Meier survival curves were compared by log rank test.

Results—Of 352 lung transplant recipients reviewed at our institution, 17 developed non-HD-dependent AKI (5%) and 16 developed HD-dependent AKI (4.6%). Cardiopulmonary bypass was significantly higher in patients with HD-dependent AKI. None of the recipients who required HD had recovery of renal function. The 30-day mortality was significantly greater in recipients requiring HD (63% *versus* 0%; $P < 0.0001$). One-year mortality after transplantation was significantly increased in recipients with HD-dependent AKI compared with those with non-HD-dependent AKI (87.5% *versus* 17.6%; $P < 0.001$).

Conclusions—Hemodialysis is associated with mortality after lung transplantation. Fortunately, AKI that does not progress to HD commonly resolves and has a better overall survival. Avoidance, if possible, of cardiopulmonary bypass may attenuate the incidence of AKI. Aggressive measures to identify and treat early postoperative renal dysfunction and prevent progression to HD may improve outcomes after lung transplantation.

Keywords

Transplantation; Lung; Dialysis; Acute kidney injury; Outcomes; Mortality

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1. Introduction

Lung transplantation is the best treatment for end-stage lung disease. However, medical complications after transplantation continue to cause significant morbidity and mortality for recipients [1]. Multiple studies have demonstrated a natural decline in renal function in lung transplant recipients over time [2–4]. Renal dysfunction is a major long-term complication after transplantation [1,5].

However, the significance of early postoperative acute kidney injury, especially hemodialysis (HD)-dependent kidney injury, remains unclear. There is a 25% incidence of acute kidney injury (AKI) after non-renal solid organ transplantation, which is associated with increased mortality, length of stay, and cost [6]. Canver and colleagues [7] reported that postoperative HD in heart transplant recipients was associated with a 44% mortality rate.

In this study, we investigated the incidence of AKI and its impact on outcomes after lung transplantation. We hypothesized that HD-dependent AKI is associated with significant mortality after lung transplantation.

2. Materials and methods

2.1. Data source and patient population

We obtained approval for this investigation from the Human Investigation Committee of the University of Virginia Health System, including a waiver of the need to obtain patient consent. All patients undergoing lung transplantation at our institution were prospectively entered into the Lung Transplant Research database.

We performed a retrospective review of all patients undergoing lung transplantation from July 1991 to July 2009. Of the 352 recipients identified, 319 recipients did not have AKI, whereas 33 recipients had postoperative AKI. We stratified recipients into three groups: recipients without AKI (No AKI), recipients with non-HD-dependent AKI (ND-AKI), and recipients with HD-dependent AKI (DD-AKI). We excluded from the study all patients with incomplete data for preoperative and postoperative renal function. Recipients undergoing simultaneous heart and lung transplantation were also excluded from the study.

2.2. Variables examined and outcomes measured

We defined kidney injury or dysfunction as a creatinine >3 mg/dL during the first 5 postoperative days. Recipients with AKI were subsequently stratified into the two groups, DD-AKI and ND-AKI, based on the requirement of HD during the acute postoperative period.

We based indications for hemodialysis on standard criteria and at the recommendation of the consulting nephrologist [8]. In this patient population, the most common indications for HD included volume overload, electrolyte abnormalities, and acid–base disarray. Our techniques for cardiopulmonary bypass and extracorporeal membrane oxygenation (ECMO) have largely remained unchanged over time and do not differ between different patient populations. We use arteriovenous cardiopulmonary bypass and ECMO; in the operating

room, we use the chest as our access, and postoperatively, the groin. This is standard at our institution. Earlier institution of ECMO in patients with primary graft dysfunction is the only change that has occurred in use of ECMO over time at our institution.

We examined patient demographic characteristics, preoperative risk factors, operative features, and postoperative outcomes. Since 1998, our institution calculated oxygenation index (OI) to determine the severity of reperfusion injury (RI). Oxygenation index is defined as $OI = [\text{Percentage inspired oxygen}] \times [\text{Mean airway pressure}] / [\text{Partial pressure of oxygen}]$. Oxygenation index as an indicator for severity of lung injury was developed by critical care physicians and has been used to stratify patients in the Acute Respiratory Distress Syndrome Network trials [9]. We collected the raw data of FIO_2 , PaO_2 , and mean airway pressure for most recipients. We used OI in this study to document the severity of RI, where RI was defined as an $OI > 7$. The primary end points in this study were short-term (30-d) and long-term (1-y) survival after lung transplantation in recipients with and without DD-AKI, compared with recipients without AKI.

2.3. Statistical analysis

We compared patient demographics, preoperative risk factors, operative features, and postoperative outcomes in lung recipients with no AKI, DD-AKI, and ND-AKI using univariate analysis. We compared categorical variables using chi-square or Fisher's exact test where appropriate. Continuous variables were compared using analysis of variance with Tukey's correction. Categorical data are reported as frequencies and percentages; we used a statistical significance of $P < 0.05$.

We analyzed Kaplan-Meier survival curves after transplantation for recipients without AKI, with HD-dependent AKI, and with non-HD-dependent AKI. We used the log rank test to compare the influence of HD on survival. Data manipulation and analysis were performed with SAS version 9.1.3 software (SAS Institute, Inc., Cary, NC).

3. Results

Between 1991 and 2009, 352 patients underwent lung transplantation at our institution. Of this cohort, 33 recipients had postoperative AKI (10%). Of the patients with AKI, 48% (16 patients) required HD. When comparing all groups (No AKI, DD-AKI, and ND-AKI), all recipients were similar for preoperative and operative features. There were no significant differences in preoperative creatinine, age, diabetes, or primary diagnosis (Table 1). Single and double lung transplants were performed with equal frequency in all study groups, with equivalent ischemic times. When comparing only recipients with kidney injury, ND-AKI *versus* DD-AKI, recipients requiring HD had significantly higher use of cardiopulmonary bypass during transplantation (43.8% *versus* 11.8%; $P = 0.04$). However there was no significant difference in the use of cardiopulmonary bypass compared with recipients without AKI ($P = 0.18$).

Postoperative complications in recipients with DD-AKI and ND-AKI were largely similar (Table 2). Recipients with and without HD had equivalent episodes of pneumonia, stroke, acute respiratory distress syndrome (ARDS), and gastrointestinal events. However,

recipients with DD-AKI required significantly more ECMO after transplantation compared with recipients with ND-AKI (56.3% *versus* 5.9%; $P = 0.002$). There was no significant difference in primary graft dysfunction as measured by OI among the three groups ($P = 0.35$). When comparing all three groups, postoperative complications were significantly less in recipients without AKI. Recipients without AKI had significantly less pneumonia, ARDS, arrhythmias, stroke, and gastrointestinal events (Table 2).

No recipients who required HD had recovery of renal function. As shown in Table 3, 30-d mortality was significantly greater in recipients requiring HD compared with those who did not progress to HD (62.5% *versus* 0%; $P < 0.0001$). One-year mortality after transplantation was significantly increased in recipients with HD-dependent AKI compared with those with non-HD-dependent AKI (87.5% *versus* 17.6%; $P < 0.001$). As demonstrated by Kaplan-Meier survival curves at 30 d (Fig. 1) and 1 y (Fig. 2), recipients with AKI, especially those requiring HD, had significantly decreased survival compared with those who did not develop AKI ($P < 0.0001$).

In this study, the use of postoperative ECMO was significantly higher in recipients with HD-dependent AKI compared with those with non-HD-dependent AKI (Tables 1 and 2). We institute ECMO for lung transplant recipients with grade 3 primary graft dysfunction or ischemia-reperfusion injury; however, this depends on the surgeon as well. More recently, we have favored earlier institution of ECMO when severe primary graft dysfunction is evident, because it has been demonstrated that ECMO instituted earlier and for shorter times improves mortality [10]. Extracorporeal membrane oxygenation is known to be associated with the development of AKI, and occurs in up to 58% of patients requiring ECMO. Furthermore, ECMO can carry significant morbidity and mortality for patients [11]. Therefore, there is a potential for HD-dependent AKI to act as a surrogate for ECMO when the outcome of mortality is studied. To eliminate this potential confounder, we performed a subanalysis of the data. We excluded from the study the 10 patients who required ECMO in the DD-AKI and ND-AKI groups. In this subanalysis, the need for HD was again significantly associated with mortality, and recipients requiring HD had decreased survival compared with those who did not require HD ($P < 0.001$).

Another potential confounding factor relates to the relatively long study period (18 y). Over that period, there were changes in donor selection criteria, postoperative immunosuppressive regimens, and postoperative management strategies. To address these issues, we compared donor demographics in the AKI and non-AKI groups to see whether recipients developing AKI came from sicker donors. There were no significant differences in donor characteristics between the AKI and non-AKI groups (Table 4). Next, we divided the cohort into patients transplanted before and after the year 2000. We chose this date because it marked the time when our institution moved away from using cyclosporine for induction therapy, and it represented a chronological midpoint within our study period. Analysis of these two groups demonstrated a trend toward increased HD-dependent and non-HD-dependent AKI within the pre-2000 group, but this increase was not statistically significant ($P = 0.12$ and 0.44 , respectively) (Table 5). In addition, the pre-2000 DD-AKI group had a significantly higher 30-d mortality (55% [$n = 5$] *versus* 0% [$n = 0$]; $P = 0.029$) and 1-y mortality (78% [$n = 7$] *versus* 13% [$n = 1$]; $P = 0.015$) compared with the pre-2000 ND-AKI group. This

relationship was maintained in the post-2000 group for 30-d mortality (71% [$n = 5$] *versus* 0% [$n = 0$]; $P = 0.005$) and 1-ymortality (100% [$n = 7$] *versus* 22% [$n = 2$]; $P = 0.003$), and corroborates our mortality analysis over the entire study period.

4. Discussion

Renal dysfunction after lung transplantation has largely been described as chronic renal failure, with up to 25% of recipients with renal injury at 1 y [5] and up to 16% of recipients ultimately progressing to end-stage renal disease [2,4,5,12]. Clearly, chronic renal failure is associated with significant morbidity and mortality for recipients. Despite the extensive literature on chronic renal failure after transplantation, the importance of AKI in the postoperative period has not been well investigated.

In this study, lung transplant recipients had a 10% incidence of postoperative AKI, of which 5% had non-HD-dependent kidney injury and 4.6% had HD-dependent kidney injury. Hemodialysis was significantly associated with mortality after lung transplantation, with a 63% 30-d hospital mortality rate ($P < 0.0001$). There was a significant decrease in survival in recipients with AKI that required HD. Only 12% of recipients were alive at 1 y after transplantation ($P < 0.001$). Similar to our findings, Rocha and colleagues [13] reported a 70% in-hospital mortality rate for lung transplant recipients requiring HD and a significant reduction in survival in recipients requiring HD.

Postoperative complications after transplantation were not significantly increased in AKI recipients who required HD, compared with those that did not require HD (Table 2). This similarity between AKI recipients with and without HD may lead one to conclude that HD may be the true cause of the dismal 12% 1-y survival. In other words, the poor survival after HD is not the result of a complicated surgical procedure or another phenomenon causing increased mortality, such as sepsis or cardiovascular disease. In congruence with our findings, Rocha and colleagues [13] found no significant difference in mechanical ventilation or length of hospital stay in recipients with and without HD-dependent AKI.

In a recent publication, George and colleagues [14] retrospectively reviewed the United Network for Organ Sharing database for lung transplant recipients requiring HD. Their analysis revealed that 5.5% of lung transplant recipients required HD postoperatively, similar to our incidence of 4.6%. Patients requiring HD had increased mortality at 30 d (24%) and 1 y (64%), compared with non-HD patients. Similar to our study, there was an association between postoperative ECMO and HD-dependent AKI. In contrast to our study, they identified preoperative factors such as diabetes and poor renal function as associated with the need for HD postoperatively. Interestingly, they demonstrated that patients at low-volume centers (<18 lung transplants per year) with HD had increased mortality compared with high-volume centers. Of note, our institution performs 25–50 lung transplants per year.

In our study, cardiopulmonary bypass during transplantation was significantly associated with HD after lung transplantation (Table 1). Recipients with HD-dependent AKI had significantly greater use of cardiopulmonary bypass intraoperatively, compared with recipients without HD-dependent AKI. At our institution, cardiopulmonary bypass is not routinely used for lung transplantation. It is implemented only when believed to improve

outcomes for the recipient. Cardiopulmonary bypass is typically used in patients with primary pulmonary hypertension, with pulmonary artery pressures, or instability after the first lung transplant in a sequential double-lung transplant. However, we actively aim to stay away from cardiopulmonary bypass if it is possible and safe for the patient. Cardiac surgery with cardiopulmonary bypass is the second most common cause of all renal injury in critically ill patients [15]. The use and duration of cardiopulmonary bypass has been shown to be independently associated with the development of AKI [16,17]. Avoiding cardiopulmonary bypass if possible may help attenuate the incidence of AKI and, most important, the need for postoperative HD.

Fortunately, AKI in lung transplant recipients who did not progress to HD commonly resolved, and those recipients had better overall survival (Table 3). Aggressive measures to identify and treat early postoperative renal dysfunction are imperative. In their review, Bloom and Doyle [18] suggested that the common causes of AKI after lung transplantation are similar to those causes after any surgery. These common causes include volume contraction, acute tubular necrosis, calcineurin inhibitors (cyclosporine and tacrolimus), atheroembolism, and acute interstitial nephritis [18]. Calcineurin inhibitor nephrotoxicity has been significantly associated with renal dysfunction after lung transplantation, although its effects are reversible [1,2,4,12,18]. Calcineurin inhibitor use during the immediate postoperative period causes renal vasoconstriction and reduces renal perfusion [19–21]. In our institution, we routinely use induction therapy with an interleukin-2 (IL-2) receptor blocker (currently basiliximab), and initiate tacrolimus around postoperative day 2. Similar to renal-sparing protocols with other solid organs (e.g., heart, liver), we delay initiation of calcineurin inhibitor treatment for up to 5 d when there is postoperative renal dysfunction. Before using of IL-2 receptor blockers, patients were given intravenous calcineurin inhibitors (cyclosporine) until they were able to tolerate oral medications. Our institution stopped using cyclosporine for induction therapy in 2000. The use of thymoglobulin and then subsequently IL-2 receptor blockers has allowed for this reduction in early calcineurin inhibitor use in the hope of reducing associated nephrotoxicity.

Our subgroup analysis of patients before and after our switch away from cyclosporine demonstrated a higher incidence of AKI and HD-dependent AKI before the year 2000, but this difference was subgroup analysis allowed us to compare broadly differences in intensive care and donor selection criteria over the course of this study. Although limited by our small sample sizes, this analysis suggests that the differences in management over our study period did not have a statistically significant effect on AKI incidence and patient mortality related to AKI.

Other treatable causes of postoperative renal dysfunction are hypotension, hypoperfusion, and aggressive diuresis [1]. Lung transplant recipients commonly undergo diuresis during the postoperative period to treat pulmonary edema after transplantation. However, excessive diuresis leads to a decrease in circulating volume, which can lead to renal hypoperfusion. Therefore, postoperative diuresis must be used judiciously and implemented on an individual basis.

4.1. Limitations

This study is inherently limited by its retrospective nature and evaluation of a single institution. It is also limited by its inclusion of lung transplant recipients over an 18-y period. We attempted to address the changes in practice patterns over the study period by performing the subgroup analysis for before and after the year 2000, as discussed above. However, the statistical strength of this analysis could be questioned because of the small sample sizes within these subgroups.

We also recognize that there are methods of defining renal failure other than creatinine clearance. However, the main purpose of this study was not to compare recipients with and without renal failure, but instead to look at the impact of HD. Therefore, we believe that the use of creatinine clearance was appropriate. Finally, in this study, the use of ECMO was significantly higher in recipients with HD-dependent AKI compared with those with non-HD-dependent AKI (Table 2). Therefore, ECMO can act as a potential confounder. To address this potential bias, we performed a subanalysis removing all recipients requiring ECMO. In this subanalysis, our findings were corroborated. After removing all recipients requiring ECMO, HD was still significantly associated with mortality, and recipients requiring HD had decreased survival compared with recipients who did not require HD ($P < 0.001$).

Based on our study and corroborated by George and colleagues [14], HD-dependent AKI is associated with mortality after lung transplantation. Fortunately, AKI that does not progress to HD commonly resolves and has a better overall survival. Aggressive measures to identify and treat early postoperative renal dysfunction and prevent progression to HD may improve outcomes after lung transplantation.

Acknowledgments

This project was supported by Award T32HL007849 (P.I. Irving L. Kron) from the National Heart, Lung, and Blood Institute, and Award 5T32AI078875-02 (P.I. Robert G. Sawyer) from the National Institutes of Health. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Heart, Lung, and Blood Institute or the National Institutes of Health.

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Fig. 1.
Kaplan-Meier 30-d survival curve for lung transplant recipients with HD-dependent AKI versus non-HD AKI after lung transplantation. (Color version of figure is available online.)

Fig. 2.
Kaplan-Meier 1-y survival curve for lung transplant recipients with HD-dependent AKI
versus non-HD AKI after lung transplantation. (Color version of figure is available online.)

Table 1

Demographics and operative features for patients with and without AKI after lung transplantation.

	ND-AKI (n = 17)	DD-AKI (n = 16)	P value*	No AKI (n = 319)	P value [†]
Age at operation, y (mean ± SE)	47.5 ± 3.9	52.4 ± 2.6	0.30	52.0 ± 2.7	0.36
Gender (male)	8 (50%)	11 (64.7%)	0.75	170 (53.1%)	0.62
Race			0.17		0.43
White	13 (76.5%)	15 (93.8%)		273 (90.1%)	
African-American	4 (23.5%)	1 (6.3%)		30 (9.9%)	
Diabetes	3 (17.7%)	1 (6.3%)	0.60		
Preoperative creatinine (mean ± SE)	1.2 ± 0.14	0.95 ± 0.12	0.09	0.92 ± 0.02	0.09
Primary diagnosis			0.46		0.49
Chronic obstructive pulmonary disorder	7 (41.2%)	4 (28.6%)		159 (49.7%)	
Cystic fibrosis	1 (5.9%)	3 (21.4%)		24 (7.5%)	
Pulmonary fibrosis	4 (23.5%)	2 (12.5%)		47 (14.7%)	
Pulmonary hypertension	0	1 (7.1%)		11 (3.4%)	
Alpha-1 anti-trypsin deficiency	1 (5.9%)	0		16 (5.0%)	
Other	4 (23.5%)	6 (42.9%)		63 (19.7%)	
Transplant type					
Single	14 (82.0%)	10 (62.5%)	0.36	230 (71.8%)	0.15
Double	3 (17.6%)	6 (37.5%)	0.50	90 (28.1%)	0.50
Induction therapy	10 (58.8%)	5 (31.3%)	0.11	114 (35.6%)	0.14
Cardiopulmonary bypass	2 (11.8%)	7 (43.8%)	0.04	47 (14.7%)	0.18
Right ischemic time, min (mean ± SE)	282.4 ± 17.9	125 ± 39.5	0.94	268.9 ± 7.2	0.78
Left ischemic time, min (mean ± SE)	261.8 ± 15.5	239.3 ± 28.2	0.51	263.9 ± 6.9	0.55

DD-AKI = dialysis dependent acute kidney injury; ND-AKI = non-dialysis dependent acute kidney injury.

SE = standard error.

*ND-AKI versus DD-AKI.

[†]ND-AKI versus DD-AKI versus No AKI.

Table 2

In-hospital postoperative complications for patients with and without AKI after lung transplantation.

	ND-AKI (<i>n</i> = 17)	DD-AKI (<i>n</i> = 16)	<i>P</i> value [*]	No AKI (<i>n</i> = 319)	<i>P</i> value [†]
ECMO	1 (5.9%)	9 (56.3%)	0.002	14 (4.3%)	<0.0001 [§]
Pneumonia	10 (58.8%)	6 (37.5%)	0.30	39 (12.2%)	<0.0001 [*]
Oxygenation index	6.8 ± 2.2	141.4 ± 87.1	0.40	70.2 ± 16.2	0.35
Pleural effusion	2 (11.8%)	1 (6.3%)	1	24 (7.5%)	0.79
Adult respiratory distress syndrome	5 (29.4%)	5 (31.3%)	1	20 (6.3%)	<0.0001
Myocardial infarction	0	1 (6.3%)	0.49	2 (0.6%)	0.05
Arrhythmias	6 (35.3%)	4 (25.0%)	0.71	30 (9.4%)	0.001
Stroke	1 (5.9%)	2 (12.5%)	0.60	4 (1.3%)	0.004
Gastrointestinal event	5 (29.4%)	3 (18.8%)	0.69	19 (5.9%)	0.0004

DD-AKI = dialysis dependent acute kidney injury; ND-AKI = non-dialysis dependent acute kidney injury.

^{*} ND-AKI *versus* DD-AKI.

[†] ND-AKI *versus* DD-AKI *versus* No AKI.

[§] When adjusted for ECMO (all ECMO recipients were removed), there was a continued significant difference in mortality.

Table 3

Short-term and long-term mortality after lung transplantation.

	ND-AKI (<i>n</i> = 17)	DD-AKI (<i>n</i> = 16)	<i>P</i> value [*]	No AKI (<i>n</i> = 319)	<i>P</i> value [†]
30-d mortality	0	10 (62.5%)	<0.0001	13 (4.1%)	<0.0001
1-y mortality	3 (17.6%)	14 (87.5%)	<0.001	42 (13.1%)	<0.0001

DD-AKI = dialysis dependent acute kidney injury; ND-AKI = non-dialysis dependent acute kidney injury.

^{*} ND-AKI versus DD-AKI.

[†] ND-AKI versus DD-AKI versus No AKI.

Table 4

Donor patient demographics for lung transplant recipients with and without AKI after transplantation.

	ND-AKI (<i>n</i> = 17)	DD-AKI (<i>n</i> = 16)	No AKI (<i>n</i> = 319)	<i>P</i> value*
Donor gender (male)	9 (60%)	9 (64.3 %)	206 (70.3%)	0.91
Donor age (mean)	38	30.8	30.8	0.18
Donor cytomegalovirus positive	8 (57.1%)	6 (42.9%)	162 (53.6%)	0.70
Donor death				0.28
Cerebral vascular injury/Intracerebral hemorrhage	4 (30.8%)	5 (38.5%)	93 (33.8%)	
Gunshot wound	1 (7.7%)	3 (23.1%)	63 (22.9%)	
Motor vehicle accident	0	0	1 (0.4%)	
Suicide	0	1 (7.7%)	15 (5.5%)	
Trauma	6 (46.1%)	3 (23.1%)	95 (23.1%)	
Other	2 (15.4%)	1 (7.7%)	8 (2.9%)	
Donor smoking	3 (17.5%)	3 (18.8%)	49 (15.3%)	0.71
Preservation solution				0.41
Euro-Collins	7 (53.9%)	4 (50%)	76 (31%)	
Other	2 (15.2%)	0	15 (61%)	
Perfadex (Vitrolife, Sweden)	3 (23.1%)	4 (50%)	134 (54.7%)	
University of Wisconsin	1 (7.8%)	0	20 (8.2%)	

DD-AKI = dialysis dependent acute kidney injury; ND-AKI = non-dialysis dependent acute kidney injury.

* ND-AKI versus DD-AKI versus No AKI.

Table 5

Acute kidney injury in lung transplant patients before and after the year 2000.

	Before 2000 (n = 131)	After 2000 (n = 222)	P value
ND-AKI	8 (6.1%)	9 (4.1%)	0.44
DD-AKI	9 (6.9%)	7 (3.2%)	0.12
No AKI	114 (87%)	206 (92.8%)	0.09

DD-AKI = dialysis dependent acute kidney injury; ND-AKI = non-dialysis dependent acute kidney injury.