Pulmonary Artery Systolic Pressure Measured Intraoperatively by Right Heart Catheterization Is a Predictor of Kidney Transplant Recipient Survival

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Background: The effect of pulmonary artery systolic pressure (PASP) measured by Swan-Ganz right heart catheter (SG-RHC) on kidney transplant recipient survival has not been previously studied. The objective of this study was to assess the relationships between PASP measured via SG-RHC, done intraoperatively at the time of anesthesia at the beginning of kidney transplant surgery, and patient survival. Multiple comorbidities, time on dialysis before the transplantation, and graft function were also analyzed in our study.

Material/Methods: This was a retrospective cohort study using data from all consecutive patients undergoing kidney transplant between January 1, 2005 and December 31, 2009 at Tampa General Hospital. Kidney transplant recipients were divided into 2 groups: Group 1 with PASP <35 mmHg and group 2 with PASP ≥35 mmHg. Patients and graft survival data, time on dialysis before transplant, and comorbidities were compared between the 2 groups.

Results: Only 363 patients were found to have a documented PASP measurement at the time of anesthesia induction for the transplant surgery, and were included in the specific analysis of our study. Patients with PASP ≥35 mmHg showed a significant decrease in survival in comparison to patients having PASP values <35 mmHg (HR 1.88; 95% CI 1.012 to 3.47, P=0.04). There was a significant positive correlation between time on dialysis and PASP (rho 0.20; 95% CI 0.09 to 0.30, p<0.001), as well as a significant difference in median time on dialysis between PASP <35 vs. PASP ≥35 (22 vs. 29 months, p=0.004). There were no significant differences in graft failure between the 2 PASP groups (HR 0.34; 95% CI 0.12 to 1.01, P=0.05).

Conclusions: Patients with PASP ≥35 mmHg, measured intraoperatively by SG-RHC, showed significantly shorter survival in comparison to patients having PASP values <35 mmHg. This result suggests the need for a randomized controlled trial to address the importance of post-transplant pulmonary hypertension management in patient survival.

MeSH Keywords: Arterial Pressure • Catheterization, Swan-Ganz • Graft Survival • Kidney Transplantation • Patient Outcome Assessment

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Elevated pulmonary artery systolic pressure (PASP), defining pulmonary hypertension (PH), is rare in the general population but is common among patients with end-stage renal disease (ESRD), and has been shown to be associated with higher mortality rates [1–3]. Despite the significant effect of elevated pulmonary pressures on ESRD patient survival, limited attention has been given to the progression/regression of elevated PASP after kidney transplant [4–10]. Screening for pulmonary pressure is being performed using echocardiogram (ECHO) on ESRD patients undergoing kidney transplant evaluation. The ECHO results give an estimate of PASP, which in turn is used to predict patient survival [4,11]. The limited available studies reported that pre-transplant pulmonary hypertensive is associated with reduced patient survival and early graft dysfunction after kidney transplantation when measured by ECHO [4,12]. However, due to the low sensitivity of the ECHO measurements, the results remain unreliable and prone to false-positive results in ESRD patients [13–15]. Therefore, ESRD patients should not be diagnosed as having PH until the PASP is measured and confirmed by RHC.

Although studies have investigated the predictors of kidney transplant outcomes, the impact of PASP measured by RHC on graft function and survival after kidney transplantation has not been studied. We hypothesize that PASP, when measured by Swan-Ganz right heart catheter (SG-RHC) in kidney transplant patients, is a reliable predictor of kidney transplant recipient outcomes.

Accordingly, the objective of this study was to assess PASP using SG-RHC at the time of surgery, along with other variables such as pre-transplant comorbidities, to investigate relationships between PASP assessed via RHC and patient and graft survival in kidney transplant patients.

**Material and Methods**

**Study design and patients**

A retrospective cohort study was conducted at Tampa General Hospital (TGH) after receiving approval from the Institutional Review Board. Data were collected on all adult kidney transplant patients who had a documented PASP measurement in the anesthesia records from January 1, 2005 to December 31, 2009. Only 363 patients had documented PASP measurement and were included in our study-specific data collection and analysis. Data collection included demographic information, pre-transplant comorbidities, and pulmonary systolic arterial pressure measured by SG-RHC, as well as graft and patient survival. Graft failure was defined as return to dialysis and/or receiving a second kidney transplant.

**Pulmonary artery systolic pressure (PASP)**

Based on previous publications showing PASP ≥35 mmHg to be significantly correlated with increased mortality in patients with chronic kidney disease/ESRD, we divided our study population into 2 groups: group 1 with PASP <35 mmHg and group 2 with PASP ≥35 mmHg [16–19].

**Swan-Ganz right heart catheterization**

Right heart catheterization prior to kidney transplantation was performed via the internal jugular vein using a Swan-Ganz (SG) catheter. Using a SG catheter intraoperatively is a standard anesthesia protocol in the kidney transplant program at Tampa General Hospital. Pulmonary artery systolic pressure was recorded before graft revascularization.

**Time on dialysis before transplant**

The date of starting dialysis before transplant was determined from the kidney transplant program candidate list at Tampa General Hospital. Type of dialysis (hemodialysis vs. peritoneal dialysis) was not obtainable because the list of active transplant candidates did not include accurate and updated data on dialysis modality. The overall estimated percentage of peritoneal dialysis in our community is less than 5% of the dialysis population.

**Graft dysfunction and survival**

Graft loss and patient survival were determined based on patient follow-up documentation in the medical records and reporting to the United Network for Organ Sharing (UNOS). The cause of death was difficult to obtain accurately due to the reports being submitted to UNOS by multiple private practices.

**Statistical analysis**

Kaplan-Meier survival and failure estimates were used to determine patient survival and graft loss, respectively. Hazard ratios with 95% confidence intervals (CIs) were calculated using the Cox proportional hazards model. Kaplan-Meier patient survival/graft failure curves by PASP with log-rank P values were also calculated. Outliers were verified to ensure that data entry errors did not occur. For univariate normally distributed continuous data, we used the t test. For skewed data we used the Wilcoxon rank-sum test. Spearman’s rank correlation coefficient was used to study the association between continuous variables. The chi-square test was used for categorical variables unless any of the expected cell counts were found to be less than 5, in which case Fisher’s exact test was used instead. All statistical analyses were performed using STATA®/MP 13.1®

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Results

Patient demographics

Of the 363 patients studied, 168 in group 1 vs. 112 in group 2 received grafts from deceased donors (p=0.048). Group 1 had more male patients than group 2 (135 patients vs. 92 patients, respectively) (p=0.047). Whites accounted for 60.3%, African Americans 23.5%, and Hispanics 11.7% of the total patient population, with no significant differences between the 2 groups (p=0.652). The average age at time of transplant was 52 years, with no significant difference between the 2 groups (p=0.146) (Table 1).

Comorbidities and delay graft function (DGF)

The incidence of DGF, defined by requiring dialysis in the first week after transplant, was higher in group 2 (26 out of 131 patients in group 2 vs. 21 out of 228 patients in group 1, p=0.004) (Table 1). A total of 212 patients were diagnosed with hypertension in group 1 vs. 127 in group 2 (p=0.22). In group 1, 79 patients were diagnosed with diabetes mellitus vs. 64 patients in group 2 (p=0.01). The estimated cardiac function (ejection fraction – EF), determined by ECHO performed within 1 year before the transplant, showed no difference between the 2 groups, with EF of 60.3% in group 1 vs. 59.1% in group 2 (p=0.27). There were more patients with smoking history in group 1 vs. 2, but the difference was not significant (Table 1).

Survival and graft loss

Survival and graft failure of the 363 patients included in the study were analyzed via Kaplan-Meier estimation using PASP values as the variable of interest. Based on the cut-off value of PASP of 35, patients with PASP ≥35 mmHg showed a significant decrease in survival in comparison to patients with PASP value of <35 mmHg (HR 1.88; 95% CI 1.012 to 3.47, P=0.04) (Figure 1). When adjusted for comorbidities, including histories of hypertension, diabetes, smoking, coronary artery disease, and heart failure using multivariate Cox regression analysis, the group with PASP ≥35 mmHg still showed a significant decrease in survival (HR 1.98; 95% CI 1.042 to 3.74, P=0.037). Graft failure assessed by Kaplan-Meier analysis showed graft failure probabilities for both PASP groups were increased at similar rates (deceased subjects were included in this analysis) (HR 1.98; 95% CI 1.042 to 3.74, P=0.037). Graft failure assessed by Kaplan-Meier analysis showed graft failure probabilities for both PASP groups were increased at similar rates (deceased subjects were included in this analysis) (HR 1.98; 95% CI 1.042 to 3.74, P=0.037). Graft failure assessed by Kaplan-Meier analysis showed graft failure probabilities for both PASP groups were increased at similar rates (deceased subjects were included in this analysis) (HR 1.98; 95% CI 1.042 to 3.74, P=0.037). Graft failure assessed by Kaplan-Meier analysis showed graft failure probabilities for both PASP groups were increased at similar rates (deceased subjects were included in this analysis) (HR 1.98; 95% CI 1.042 to 3.74, P=0.037).

Table 1. Clinical and demographic factors by PH ≥35 vs. PH <35.

| Outcome (n=363) | PH <35 (n=230) | PH ≥35 (n=133) | P-value |
|----------------|----------------|----------------|--------|
| Donor/recipient Deceased | 168 (73%) | 112 (84%) | 0.048 |
| Sex | | | 0.047 |
| Male | 135 (58%) | 92 (69%) | |
| Race | | | 0.652 |
| White | 147 (69.35%) | 82 (61.6%) | |
| Hispanic | 27 (11.6%) | 15 (11.2%) | |
| Black | 44 (18.9%) | 32 (24%) | |
| Asian | 9 (3.9%) | 4 (3.0%) | |
| Other | 3 (1.3%) | 0 | |
| Age at transplant | 51.8±12.6 | 53.8±12.2 | 0.146 |
| Delayed graft function | | | 0.004 |
| No | 207 (90%) | 105 (78.9%) | |
| Yes | 23 (10%) | 28 (21%) | |
| History of hypertension | | | 0.227 |
| Yes | 212 (92%) | 127 (95.4%) | |
| History of diabetes | | | 0.01 |
| Yes | 79 (34.3%) | 64 (48.1%) | |
| History of smoking | | | 0.308 |
| Yes | 110 (47.8%) | 57 (42.8%) | |
| EF% (n=310) | 60.3±7.6 | 59.1±8.5 | 0.27 |
Figure 1. Kaplan-Meier survival estimates of transplant recipients by PASP (≥35 vs. <35). Log-rank P=0.04.

Figure 2. Kaplan-Meier survival estimates of graft failure curves by PASP (≥35 vs. <35). Log-rank P=0.05 (includes deceased).

Figure 3. Multivariate Cox regression analysis for the comorbidities (HTN, CAD, CHF, and history of smoking) shows PASP as a significant factor for long-term survival.

Figure 4. The graph is a scatter plot of months on dialysis vs. PASP levels. The fitted line suggests a strong linear correlation (rho 0.20; 95% CI 0.09 to 0.30, p<0.001).
Kidney transplantation is the treatment of choice for selected ESRD patients [20]. Graft and patient survival after kidney transplantation have improved over the past decade. Death-censored graft survival has improved, while death with a functioning organ has increased over the same period. This is likely a reflection of better immunosuppression use, but with the persistence of complex comorbidities in transplant recipients [21]. Cardiovascular disease remains the most common cause of death with graft function after transplant, and accounts for 30% of related deaths, with the highest rates early after transplant [22]. Therefore, there has been a focus on ischemic heart disease, heart failure, and arrhythmias in these patients. In contrast, data on pulmonary hypertension as a cardiovascular risk factor in kidney transplant recipients is limited. The results of our analyses show for the first time that pre-transplant elevated PASP is correlates with patient survival after kidney transplantation. This association appears to be independent of other variables tested, including pre-transplant hypertension, heart failure, and history of smoking, but not history of diabetes mellitus. Previous studies addressed the impact of diabetes as a major cardiovascular risk factor on post-transplant patients and graft survival [23–25]. However, Baek et al. assessed clinical outcomes after living donor kidney transplantation in patients with diabetes compared to non-diabetic patients. The incidence of acute rejection, death-censored graft failure, non-death-censored graft failure, and mortality were not significantly different between patients with and without diabetes after living donor kidney transplantation [26]. Nevertheless, we are aware of this limitation in our study, and a well-designed prospective clinical trial is needed to assure accurate randomization and minimize the effect of such clinical variables on the clinical outcome.

In relation to delayed graft function (DGF) incidence, we acknowledge that the definition of DGF is not consistent in the literature [27]. However, it has been shown that DGF is associated with inferior patient and kidney allograft survival [28]. We defined DGF when patients require dialysis within the first 7 days after transplant, regardless of the indication. Our results showed a significant possible association between PASP and DGF. The higher incidence of DGF in group 2 (with PASP ≥35 mmHg) could be explained by previous findings addressing the effect of elevated pulmonary pressure on glomerular filtration rate in native kidneys. The potential mechanism is that elevated pulmonary artery pressure leads to increased venous congestion and neuro-hormonal activation, which then leads to a negative glomerular filtration pressure and ultimately decreases the GFR [29–31]. Other factors affecting DGF were not analyzed in this study, which is a limitation.

Among the variables evaluated in our study, time on dialysis was a predictor of an elevated PASP, which is consistent with previous studies [32,33]. The pathogenesis of elevated pulmonary artery systolic pressure in dialysis patients is not clear. Beigi et al. showed that temporary arteriovenous (AVF) access closure and successful kidney transplantation causes a significant fall in cardiac output and pulmonary artery systolic pressure, indicating the possibility that excessive pulmonary blood flow is involved in the pathogenesis of the disease [34]. Also, Nakhouli et al. reported a significant decline in PASP in 11 dialysis patients with successful transplantation and in 8 dialysis patients with temporary closure of their Arterial-Venous Fistula (AVF) access [32]. The ideal approach for AVF management in patients without adverse symptoms after kidney transplantation is a subject of debate. In current clinical practice, the AVF is often neglected if the patient with a functional kidney allograft is not reporting any symptoms related to their AVF. However, in some practices, routine closure is performed [35]. It is remarkable that despite the scale of care for AVF-related complications for patients on hemodialysis, not a single remark is made about AVF care after transplantation in any of the current vascular access guidelines [36,37]. The results of our study provide additional evidence supporting the importance of AVF management after transplantation, as well as the importance of considering AVF as one of the factors affecting cardiovascular risks in determining patient outcome after transplant.

In terms of patient survival, we report, for the first time, a decreased survival probability for patients with PASP ≥35 mmHg as measured intraoperatively by right heart catheterization. However, this association is not a definitive demonstration of causality, as the study was retrospective in nature. Placing SG-RHC intraoperatively in preparation for kidney transplantation is not considered a standard of care. This practice has been implemented by the anesthesiologists at our program for many years and we are not making any recommendation for such implementation. The decision to use the SG-RHC is not based on any selective data about the patients in terms of cardiovascular comorbidities. This imposes a limitation to our study since no clinical indications were considered and no appropriate measurements were taken, especially the pulmonary capillary wedge pressure, to determine the volume status. However, we were able to use important data collected by SG-RHC, which is considered the criterion standard, to diagnose elevated PASP and relate that to overall kidney transplant recipient outcome. All previously published studies that correlated elevated pulmonary pressures to transplant recipient outcome used data from a non-invasive ECHO to measure pulmonary pressures. Unfortunately, the ECHO might be less accurate when assessing pulmonary pressures among ESRD patients [38,39]. Pulmonary capillary wedge pressure was not measured or documented in our patients records to determine if the elevated PASP is due to primary pulmonary hypertension or volume overload. However, the mere fact of the elevation of PASP, which is correlated with poor survival, could...
signify that any of the above etiologies might be a factor influencing the results. Future studies may shed more light on the effect of these etiologies, distinctly or jointly, on survival.

The results of the present study support current guidelines [40] of regular screening of dialysis patients with potential for kidney transplant with echocardiography. We suggest considering RHC for patients with elevated estimated PASP detected by ECHO. We also suggest that more attention is needed for aggressive follow-up after transplantation for patients diagnosed with elevated pulmonary pressures, as well as considering a multidisciplinary team approach including cardiology clinic, pulmonary clinic, and vascular access clinic, in addition to transplant nephrology clinic. We believe such approach would improve long-term patient and graft survival.

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Conclusions

The study findings show for the first time that patients who receive kidney transplant with PASP ≥35 mmHg, measured intraoperatively by SG-RHC, have significantly shorter survival than patients with PASP values <35 mmHg. ESRD patients might need to be evaluated with RHC as part of their kidney transplant work-up if they are found to have elevated estimated pulmonary pressure measured by ECHO.

Conflict of interest

None.
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