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Acute Kidney Injury and Mortality following Ventricular Assist Device Implantation

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Key Words

Acute kidney injury · Cardiac surgery · Mortality · Ventricular assist device · RIFLE criteria

Abstract

Background: Ventricular assist devices (VADs) are increasingly common, and their surgical implantation predisposes patients to an increased risk of acute kidney injury (AKI). We sought to evaluate the incidence, risk factors and short- and long-term all-cause mortality of patients with AKI following VAD implantation. ***Methods:*** We identified all patients who underwent VAD implantation at the University of Chicago between January 1, 2008, and January 31, 2012. We evaluated the incidence of AKI, defined as a ≥50% increase in serum creatinine over the first 7 postoperative days (RIFLE Risk-Creatinine). A logistic regression model was used to identify risk factors for the development of AKI, and a Cox proportional hazards model was used to examine factors associated with 30-day and 365-day all-cause mortality. ***Results:*** A total of 157 eligible patients had VAD implantations with 44 (28%) developing postimplantation AKI. In a multivariate analysis, only diabetes mellitus [odds ratio = 2.25 (1.03–4.94), p = 0.04] was identified as a significant predictor of postoperative AKI. Using a multivariable model censored for heart transplantation, only AKI [hazard ratio, HR = 3.01 (1.15–7.92), p = 0.03] and cardiopulmonary bypass time [HR = 1.01 (1.001–1.02), p = 0.02] were independent predictors of 30-day mortality. Preoperative body mass index [HR = 0.95 (0.90–0.99), p = 0.03], preoperative diabetes mellitus [HR = 1.89 (1.07–3.35), p = 0.03] and postimplantation AKI [HR = 1.85 (1.06–3.21), p = 0.03] independently predicted 365-day mortality. ***Conclusion:*** AKI is common following VAD implantation and is an independent predictor of 30-day and 1-year all-cause mortality.

Introduction

Acute kidney injury (AKI) is common following traditional cardiac surgery (coronary artery bypass grafting and/or valve replacement) and is an independent risk factor for postoperative mortality [1, 2]. Additionally, AKI has been associated with higher patient morbidity, longer length of stay and higher costs [1, 3–5]. Multiple preoperative risk stratification systems have been developed to predict the development of AKI following traditional car-

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diac surgery; however, their role and application in the setting of ventricular assist device (VAD) implantation is unclear [6–9].

Since the Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure (REMATCH) trial [10], there has been an increased number of VADs being implanted, with nearly 6,000 VADs being implanted since June 2006 [11]. The first-generation VADs were pulsatile and presumably more 'physiologic'; however, they were associated with higher infection and thrombosis rates, lower durability and even lower cardiac transplant rates in comparison to continuous flow devices [12]. This has led to a transition away from pulsatile flow devices to continuous flow devices which now make up for >90% implants in the United States [13]. While the increasing numbers of implants have improved our understanding of the impact of AKI during the perioperative period, its etiology, predictors and short- and long-term consequences require further investigation. Previous studies have been difficult to interpret, as many have not used consensus definitions of AKI [14–19]. Utilizing consensus definitions of AKI, we performed a single-center, retrospective, longitudinal cohort study to evaluate the incidence of AKI following VAD implantation, identify risk factors as well as assess its impact on 30- and 365-day survival.

Methods

The Institutional Review Board of The University of Chicago approved this study. We performed a single-center retrospective chart review of all patients who underwent a VAD placement at our institution from January 1, 2008, through January 31, 2012. Patients with end-stage renal disease, those who were already on renal replacement therapy (RRT) for AKI prior to VAD implantation and those who died intraoperatively were excluded.

Observations and Measurements

Demographic, biochemical and clinical profiles were obtained from the INTERMACS national registry, Society of Thoracic Surgeons (STS) database and the University of Chicago electronic medical record (EMR) [20]. We collected preoperative data on age, race, sex, body mass index (BMI), baseline renal function (estimated glomerular filtration rate, eGFR, using MDRD) [21], past medical history, hemodynamic numbers, intraoperative data including cardiopulmonary bypass time and receipt of blood products, postoperative data including length of stay, postoperative complications (e.g. sepsis, thromboembolic events), need for RRT and in-hospital mortality. Baseline creatinine was defined as the listing of creatinine data obtained from the INTERMACS registry; if INTERMACS did not contain a preoperative serum creatinine, then the last serum creatinine in the EMR prior to device implantation was used. We collected information about the indication for VAD implantation (bridge to transplant or destination therapy) as well as the type of device implanted.

Outcomes

The primary outcome of AKI was defined as a 50% rise in serum creatinine over the preoperative baseline during the first 7 postoperative days as per the creatinine-based RIFLE (Risk of renal dysfunction, Injury to the kidney, Failure or Loss of kidney function, and End-stage renal disease; AKI assessed over 7 days) Risk criteria [22]. The Acute Kidney Injury Network (AKIN; AKI assessed over 48 h) creatinine criteria [23, 24] and urine output criteria were utilized for secondary analyses. All-cause mortality was monitored over 1 year after implantation, with data reported at 30 and 365 days.

Statistical Methods

Qualitative data were recorded in a categorical fashion, and quantitative covariates were measured as continuous variables. Differences between characteristics were analyzed either by an unpaired t test or the Wilcoxon signed-rank test as appropriate. Categorical variables were assessed by χ^2 analysis or Fisher's exact test and were reported with their corresponding odds ratio (OR) and 95% confidence intervals (CI). Continuous variables are represented with mean and their 95% CI values and hazard ratio (HR) when pertaining to survival data.

To assess determinants of AKI, we first performed a univariate logistic regression analysis. Significant predictors ($p < 0.10$) were then included in a multivariate model.

Survival data were analyzed using Cox proportional hazards modeling. All survival data were censored for cardiac transplantation. We analyzed survival at 30 and 365 days. Significant variables ($p < 0.10$) were then included in a multivariate model. All statistical tests were two-sided with α set at 0.05 for statistical significance. STATA 11.2 (StataCorp LP, College Station, Tex., USA) was used for all data analysis.

Results

Baseline Characteristics of Patients with AKI

We identified 168 patients who underwent VAD implantation at our institution during the study period. Eleven subjects were excluded from the analysis, with 8 patients having previous end-stage renal disease, 2 requiring preimplant RRT for AKI and 1 subject with intraoperative mortality. In the final cohort of 157, a total of 44 (28%) patients developed AKI. Table 1 demonstrates the pre-, intra- and postoperative characteristics of those with and without AKI. Prior to implantation, there was no difference in age, baseline renal function (serum creatinine and eGFR), New York Heart Association (NYHA) CHF class, preoperative hemodynamics, INTERMACS score, the presence of cardiogenic shock or inotrope use in those with and without AKI. However, the patients with AKI tended to have more diabetes and cerebrovas-

Table 1. Pre-, intra- and postoperative characteristics of patients with and without AKI

	No AKI (n = 113)	AKI (n = 44)	p value
a *Baseline clinical characteristics of VAD patients by AKI status*			
Operative age, years	58.3±12.7	56.4±12.7	0.4
Weight, kg	89.2±23.3	84.3±25.3	0.25
Race			
Caucasian	68 (60.2)	23 (52.3)	0.39
African-American	36 (31.9)	18 (40.9)	
Asian	4 (3.5)	0 (0)	
Unknown	5 (4.4)	3 (6.8)	
Males	89 (78.8)	34 (77.3)	0.84
BMI	28.9±6.3	28.4±7.3	0.62
Baseline serum creatinine, mg/dl	1.67±1.01	1.58±0.71	0.57
Baseline eGFR (MDRD), ml/min	49.3±26.7	50.4±22.1	0.82
Preoperative hematocrit	34.2±6.2	36.5±5.3	0.05
HbA1c	6.9±1.2	6.9±1.2	0.84
Diabetes	35 (31.0)	23 (52.3)	0.01
PVD	7 (6.2)	3 (6.8)	0.85
Hypertension	48 (42.5)	23 (52.2)	0.27
CVD	9 (8.0)	9 (20.5)	0.03
Hyperlipidemia	61 (54)	21 (47.7)	0.48
CHF	101 (89.4)	43 (97.7)	0.09
Ejection fraction	17.7±7.6	16.5±7.9	0.57
Cardiogenic shock	34 (30.1)	16 (36.36)	0.68
VAD device			
HeartMate II	88 (77.9)	35 (79.6)	
HeartWare	19 (16.8)	5 (11.4)	
BiVAD	3 (2.7)	1 (2.2)	0.48
PVAD/RVAD	0 (0)	1 (2.2)	
HeartMate XVE	3 (2.6)	2 (4.6)	
Goal of implant			
Bridge to transplant	54 (47.8)	21 (48.8)	0.90
Destination therapy	59 (52.2)	22 (51.2)	
NYHA classification (n =133)			
Class I	1 (1.1)	1 (2.6)	0.58
Class II	1 (1.1)	1 (2.6)	
Class III	9 (9.4)	4 (10.5)	
Class IV	84 (88.4)	32 (84.2)	
INTERMACS score (n = 147)			
1	18 (17)	11 (26.82)	
2	46 (43.39)	14 (34.14)	0.63
3	25 (23.58)	10 (24.39)	
4	15 (14.15)	6 (14.63)	
≥5	2 (1.88)	0	
Preoperative hemodynamics (n = 111)			
RAP, mm Hg	13.7±6.6	14.5±7.5	0.60
RVSP, mm Hg	53.3±14.8	51±12.7	0.45
PASP, mm Hg	54.3±15.1	51.6±13.9	0.40
PADP, mm Hg	26.9±8.5	26.5±7.2	0.84
PCWP, mm Hg	25.9±8.7	23.6±10.2	0.25
Cardiac output, l/min	3.78±1.7	3.60±1.4	0.62
Cardiac index, l/min/m^2	1.84±0.6	1.82±0.7	0.87

Table 1 (continued)

	No AKI (n = 113)	AKI (n = 44)	p value
b *Intra- and postoperative outcomes in patients with and without AKI following VAD implantation*			
Intraoperative outcomes			
CPB time, min	131.0±36.5	133.9±49.4	0.69
Patients who received intraoperative blood products	88 (77.9)	26 (59.1)	0.02
Patients who received intraoperative PRBC	61 (54.0)	24 (54.6)	0.94
Units of intraoperative PRBC used	1 (0–3)	1 (0–6)	0.45
Postoperative outcomes			
Net fluid balance at 7 days, liters	–6.65±5.62	–4.06±5.78	0.02
ICU length of stay, days	6.4±7.7	11.2±11.0	0.006
Total length of stay, days	29.2±59.4	34.5±46.1	0.60
Pneumonia	4 (3.5)	4 (9.1)	0.16
Reoperation for bleeding	11 (9.7)	3 (6.8)	0.57
Gastrointestinal bleeding	6 (5.3)	3 (6.8)	0.71
Blood product transfusion	70 (68.6)	23 (57.5)	0.21
Units of PRBC transfused	3 (2–6)	7 (4–16)	0.002
In-hospital mortality	9 (8.0)	14 (31.8)	<0.001
30-day mortality	8 (7.1)	11 (25.0)	0.002
RRT within first 30 days	2 (1.8)	9 (20.5)	<0.001
Postoperative sepsis (including driveline during implantation hospitalization)	7 (6.19)	6 (13.64)	0.13
Postoperative ischemic stroke (during implantation hospitalization)	1 (0.88)	2 (4.55)	0.13

Data are presented as n (%), mean ± SD or median (interquartile range), as appropriate. RAP = Right atrial pressures; RVSP = right ventricle systolic pressure; PASP = pulmonary artery systolic pressure; PADP = pulmonary artery diastolic pressure; PCWP = pulmonary capillary wedge pressure; PVAD/RVAD = paracorporeal ventricular/right ventricular assist device; CPB = cardiopulmonary bypass; CHF = congestive heart failure; PVD = peripheral vascular disease; RRT = renal replacement therapy; PRBC = packed red blood cell.

Table 2. Prevalence of AKI following VAD implantation across varied definitions

RIFLE SCr Risk	44 (28.0)
RIFLE SCr Injury	22 (14.0)
RIFLE SCr Failure	6 (3.8)
AKIN SCr stage 1	67 (42.7)
AKIN SCr stage 2	13 (8.3)
AKIN SCr stage 3	7 (4.46)
AKIN/RIFLE urine output stage 1	119 (75.8)
AKIN/RIFLE urine output stage 2	113 (72.0)
AKIN/RIFLE urine output stage 3	24 (15.3)

Data are presented as n (%). SCr = Serum creatinine.

cular disease (CVD) and had higher preoperative hematocrits (table 1a). Intraoperatively, there was no difference in cardiopulmonary bypass time, need for packed red blood cell (PRBC) transfusion or units of PRBC transfused between the two groups. Postoperatively, there was no difference in complications including bleeding, cardiac tamponade, prolonged ventilation, postoperative sepsis, ischemic strokes (symptoms lasting longer than 24 h) or pneumonia; however, those with AKI had longer ICU stays and a higher in-hospital mortality (table 1b). Both groups had a net negative fluid balance by day 7. However, patients with AKI had a significantly less net negative fluid balance by day 7 in comparison to the no AKI group (p = 0.02).

Table 2 demonstrates the varied AKI event rates in the complete cohort of 157 subjects, utilizing the RIFLE and AKIN criteria. The incidence varied greatly depending on the AKI criteria (creatinine, urine output, table 2).

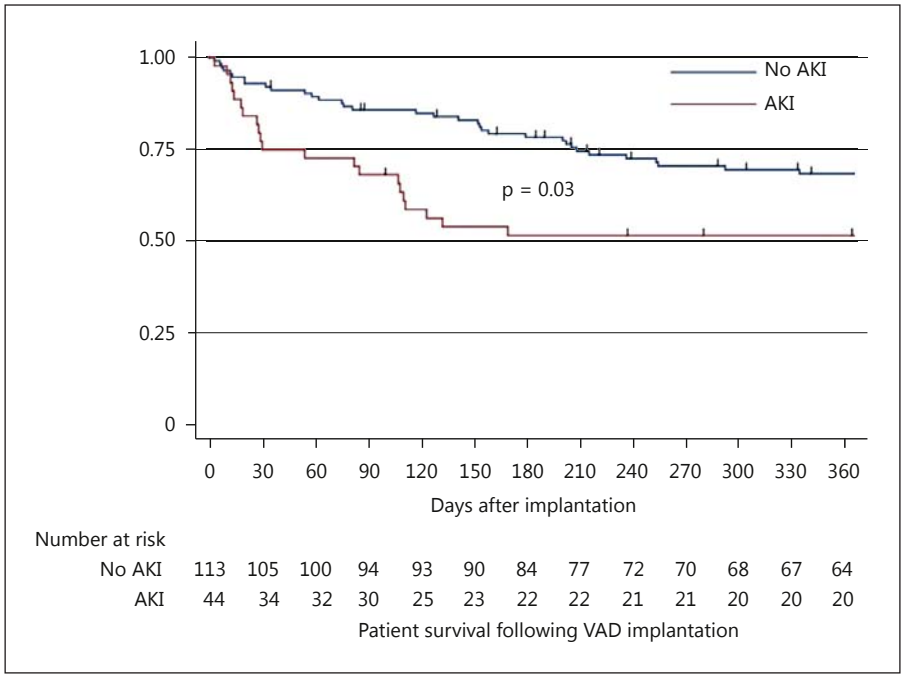

Fig. 1. One-year survival curves for patients with and without AKI. The hash marks denote subject censoring. At 365 days, 21 (48%) of the patients with AKI had died compared to 34 (30.1%) of those without AKI (p = 0.03).

Predictors of AKI following VAD Implantation

Utilizing univariate logistic regression modeling, diabetes and prior CVD were identified as significant predictors of the development of AKI (table 3). However, on a multivariate analysis, only diabetes [OR = 2.27 (1.03–4.97), p = 0.04] was identified as a significant predictor of postoperative AKI with prior cerebrovascular disease and preoperative hematocrit demonstrating a trend towards significance (table 3).

AKI and All-Cause Mortality: 30 and 365 Days

At 30 days, 11 (26%) of those with AKI had died compared to 8 (7%) without AKI (p = 0.002). At 365 days, 21 (48%) of those with AKI had died compared to 34 (30%) without AKI (p = 0.03). Figure 1 demonstrates the Kaplan-Meier survival curves for those with and without AKI, with data censored for cardiac transplantation.

In a univariate model to identify predictors of 30- and 365-day mortality, several variables were determined to be significantly associated with patient survival (tables 4, 5). Using a multivariable model, AKI [HR = 3.01 (1.15–7.91), p = 0.03] and longer bypass time [HR = 1.01 (1.001–1.02), p = 0.02] were independent predictors of a higher 30-day mortality (table 4), while AKI [HR = 1.85 (1.06–3.22), p = 0.03] and diabetes mellitus [HR = 1.9 (1.07–3.35), p = 0.03] were associated with higher mortality at 365 days (table 5). Higher preoperative BMIs were protective, and were associated with lower mortality at 365 days [HR = 0.95 (0.90–0.99), p = 0.03].

Discussion

Current literature on incidence of AKI in patients with VAD implantation and its outcomes are limited. Prior studies have defined AKI based on the need for RRT, while others have used nonstandardized definitions [14–19, 25–28]. Conducting a single-center retrospective cohort study utilizing internationally accepted consensus definitions, we identified the incidence of AKI as 28% (n = 44) with a high portion of these patients receiving RRT (n = 9; 5.7% of the total cohort). Additionally, our study demonstrates that AKI is associated with short- and long-term mortality following VAD implantation [29].

Diabetes has been shown to increase the risk of AKI following traditional cardiac surgery [6, 7]. In a multivariate analysis, we identified diabetes as the sole predictor of AKI after VAD implantation. Previous studies in VADs had identified age, baseline eGFR and perfusion time as other risk factors for the development of post-implant AKI [30, 31]. However, similar to the study by Borgi et al. [32], we were unable to demonstrate perfusion

Table 3. Predictors of AKI following VAD implantation

	OR (95% CI)	p value
Univariate predictors		
Age	0.99 (0.96–1.01)	0.4
Male sex	0.91 (0.39–2.12)	0.84
African-American race	1.48 (0.72–3.04)	0.29
Weight	0.99 (0.98–1.01)	0.25
BMI	0.99 (0.93–1.04)	0.62
Preoperative eGFR	1.00 (0.99–1.02)	0.82
Creatinine	0.89 (0.59–1.34)	0.57
Hypertension	1.48 (0.73–2.98)	0.27
CHF	5.1 (0.64–40.5)	0.12
Diabetes	2.44 (1.20–4.98)	0.01
Prior CVD	3.12 (1.09–8.07)	0.03
Peripheral vascular disease	1.1 (0.27–4.49)	0.88
Preoperative hematocrit	1.07 (0.99–1.13)	0.05
Cardiopulmonary bypass perfusion time	1.00 (0.99–1.01)	0.69
Intraoperative PRBC use	1.02 (0.51–2.06)	0.95
Right atrial pressure	1.02 (0.95–1.08)	0.6
Pulmonary capillary wedge pressure	0.98 (0.94–1.02)	0.40
Cardiac output	0.95 (0.73–1.25)	0.76
Multivariate predictors		
Diabetes	2.27 (1.03–4.97)	0.04
Prior CVD	2.84 (0.95–8.6)	0.06
Preoperative hematocrit	1.06 (0.99–1.13)	0.06

CVD = Cerebrovascular disease; PRBC = packed red blood cells.

Table 4. HRs for 30-day all-cause mortality following VAD implantation

	HR (95% CI)	p value
Univariate analysis		
Age	1.04 (0.99–1.09)	0.07
Male sex	1.50 (0.44–5.16)	0.52
African-American race	0.34 (0.10–1.17)	0.09
Preoperative eGFR	0.99 (0.98–1.02)	0.77
AKI	3.74 (1.50–9.30)	0.005
BMI	0.92 (0.84–1.00)	0.06
Diabetes	2.47 (0.99–6.15)	0.051
CHF	0.42 (0.12–1.44)	0.17
Hypertension	0.89 (0.36–2.21)	0.80
Cardiogenic shock	0.96 (0.37–2.54)	0.95
CVD	2.25 (0.75–6.79)	0.15
Cardiopulmonary bypass perfusion time	1.01 (1.00–1.02)	<0.001
Preoperative hematocrit	1.00 (0.92–1.10)	0.87
Intraoperative PRBC use	3.42 (1.13–10.32)	0.03
Right atrial pressure	0.98 (0.90–1.07)	0.70
Pulmonary capillary wedge pressure	1.02 (0.97–1.13)	0.49
Cardiac output	1.04 (0.74–1.45)	0.81
Multivariate analysis		
Age	1.01 (0.97–1.07)	0.56
African-American race	0.50 (0.13–1.99)	0.33
AKI	3.01 (1.15–7.91)	0.03
BMI	0.92 (0.84–1.00)	0.07
Diabetes	2.76 (0.98–7.72)	0.054
Cardiopulmonary bypass perfusion time	1.01 (1.001–1.02)	0.02
Intraoperative PRBC use	2.34 (0.75–7.31)	0.14

CVD = Cerebrovascular disease; PRBC = packed red blood cells.

time as a risk factor for AKI. Our inability to replicate differences in baseline eGFR may stem from our utilization of the standardized creatinine-based definition of AKI rather than more subjective definitions [14–19].

In our study, predictors of 30-day mortality included AKI and perfusion time. Our findings mirror those of Borgi et al. [32], a smaller single-center study (n = 100), and demonstrate that post-VAD AKI is also a predictor of 1-year mortality. After our adjusted analyses, these findings further strengthen the preexisting literature demonstrating the long-term consequences of AKI in a variety of clinical settings [33, 34]. Additionally, we observed that patients with a higher preoperative BMI had lower 1-year mortality; corroborating the findings of Butler et al. [35] who demonstrated that in univariate analyses the 180- and 365-day survival after VAD implantation was significantly higher among patients with higher BMIs. However, unlike our study, this effect was no longer significant following multivariate analysis.

Age, baseline eGFR, gender and African-American race have been previously reported to have an impact on AKI and mortality. However, we did not identify these to be significant AKI risk factors (tables 4, 5). The recently published fifth INTERMACS report identified age to be a predictor of mortality, although the actuarial survival of patients older than 70 years was reported to be only modestly inferior to those older than age 50 [13]. Similarly, we were unable to identify baseline eGFR as a predictor for short- and long-term survival. Even after stratifying preimplant eGFR to <60 and ≥60 ml/min, we found no significant difference in long-term mortality (data not shown). This was contrary to the study done by Sandner et al. [36], which reported worse survival for the group with eGFR <60 ml/min. Our inability to demonstrate

Table 5. HRs for 365-day all-cause mortality following VAD implantation

	HR (95% CI)	p value
Univariate Cox proportional hazards		
Age	1.00 (0.99–1.03)	0.53
Male sex	1.12 (0.58–2.19)	0.72
African-American race	0.99 (0.56–1.72)	0.96
Preoperative eGFR	0.99 (0.98–1.00)	0.29
AKI	2.01 (1.16–3.48)	0.01
BMI	0.96 (0.92–1.00)	0.08
Diabetes	1.66 (0.97–2.84)	0.06
CHF	0.63 (0.27–1.48)	0.29
Hypertension	1.26 (0.74–2.16)	0.31
Cardiogenic shock	1.06 (0.60–1.88)	0.81
CVD	1.25 (0.57–2.77)	0.58
Cardiopulmonary bypass perfusion time	1.00 (1.00–1.01)	0.04
Preoperative hematocrit	1.00 (0.95–1.05)	0.99
Intraoperative PRBC use	1.25 (0.73–2.16)	0.40
Right atrial pressure	0.99 (0.95–1.04)	0.86
Pulmonary capillary wedge pressure	1.00 (0.97–1.04)	0.92
Cardiac output	1.02 (0.85–1.23)	0.80
Multivariate Cox proportional hazards		
AKI	1.85 (1.06–3.22)	0.03
BMI	0.95 (0.90–0.99)	0.03
Diabetes	1.90 (1.07–3.35)	0.02
Cardiopulmonary bypass perfusion time	1.00 (0.99–1.01)	0.07

CVD = Cerebrovascular disease; PRBC = packed red blood cells.

eGFR as a risk factor may stem from the lower eGFR values in our cohort than those previously published (49.7 ml/min). Traditionally, African-Americans have been noted to have worse outcomes after cardiac surgeries [37, 38], but our study as well as two recently published studies showed no difference in outcomes in patients who had a VAD placed [39, 40]. Women have also been reported to be at higher risk for mortality after traditional cardiothoracic surgery, but we did not appreciate that in any of our analyses [6, 41, 42].

Haase et al. [43] demonstrated no difference in diagnosing AKI following traditional cardiac surgery when comparing the RIFLE and AKIN criteria. However, unlike our cohort, in their prospective observational study, less than 25% of all patients had an eGFR less than 60 ml/min. Our study demonstrated clear differences between the AKIN and RIFLE criteria-defined AKI, with over 67 (42.7%) subjects developing AKIN serum creatinine-based stage 1, compared to 28% for RIFLE Risk. In a secondary analysis, the use of AKIN stage 1 (creatinine criteria) was not associated with a significant risk of mortality at 30 [HR = 1.88 (0.76–4.67, p = 0.17)] or 365 [HR = 1.41 (0.83–2.41, p = 0.2)] days. This analysis suggests the use of a 0.3 mg/dl increase in serum creatinine to identify post-VAD AKI may be inferior to RIFLE Risk (50% increase) from a prognostic perspective. Similarly, despite the high proportion of patients with AKI defined by urine output (n = 119, 75.8%, stage 1 <0.5 ml/kg/h for 6 h; table 2), we did not note any statistically significant impact on 30- and 365-day survival (data not shown). A recent study [44] in a general ICU cohort has raised the question whether the urine output criteria for defining AKI is too liberal. Similar to our observation, the authors were unable to show AKI, as defined by urine output criteria, to have a significant impact on 1-year mortality. However, these findings need to be validated in larger multicenter prospective trials.

Questions remain about the effects of pulsatile versus continuous flow devices on renal function. While pulsatile flow is physiologic and theoretically better, based on the limited published human data, renal outcomes have not differed across the two modalities [25]. Perhaps counterintuitively, continuous flow devices have been associated with lower device-related complications and improved outcomes [28, 45, 46]. The further investigation of this issue has proven difficult, with greater than 90% of US VAD implantations being continuous flow devices [13].

Our study has several strengths. To our knowledge, this is the largest cohort study of VAD patients to employ a consensus definition of AKI, including analyzing AKI end points defined by urine output. Additionally, compared to previously published papers, we have a significant number of African-American patients as well as destination therapy VAD patients. Both of these subpopulations are steadily increasing [11]. Our study suffers from all the inherent limitations of a single-center retrospective review. However, these same single-center factors allowed us to have excellent patient follow-up and complete, demographic, postoperative serum creatinine and urine output data. Additional strengths include utilization of our single-center EMR and access to INTERMACS to validate the true preimplantation baseline creatinine/eGFR. Included in our limitations are the low numbers of patients who developed severe AKI (RIFLE I or F), and this limits our ability to make any conclusions about severe AKI.

In conclusion, we identified the incidence of AKI following VAD implantation to be 28%, and those who de-

veloped AKI had increased risk for both short- and long-term mortality. Further investigations are needed to tease out the differences between the risks of AKI following traditional cardiac surgery versus VAD implantations. Additionally, given the high AKI event rates and the mounting evidence linking AKI to mortality following VAD implantation, the use of biomarkers to identify patients at risk may have a role. Larger prospective multicenter trials are needed to develop a risk stratification system to identify patients at risk for developing post-VAD implantation AKI.

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