Tricuspid Regurgitation and Kidney Transplant Recipient Outcomes

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Rationale & Objective: Kidney function can be adversely affected by significant tricuspid regurgitation (TR) owing to effects on cardiac output and systemic venous congestion. However, the impact of significant TR on short- and long-term kidney function following a kidney transplant remains uncertain.

Study Design: Retrospective observational cohort.

Setting & Participants: Kidney transplant recipients from a single center between 2016 and 2019.

Exposure: Significant TR, defined by at least moderate regurgitation, on echocardiogram before kidney transplantation.

Outcomes: Primary end points included the estimated glomerular filtration rate (eGFR) at the following 3 time points: 2 weeks, 3 months, and 1 year after transplantation. Secondary end points included major adverse cardiac events including nonfatal myocardial infarction, all-cause mortality, and hospitalization owing to cardiovascular disease.

Analytical Approach: Propensity score matching was performed in 1:3 ratio between patients treated with significant TR and controls, within a caliper 0.05 standard deviation of the propensity score, to analyze for the primary end point.

Results: Among 557 kidney transplant recipients, 26 (5%) exhibited significant TR pretransplantation. According to propensity score matching analysis, with 1:3 ratio between 24 patients with significant TR and 72 controls, the presence of significant TR was associated with a lower eGFR post-transplantation. Specifically, the mean eGFR was 41.2 mL/min/1.73 m^2 compared to 53.3 mL/min/1.73 m^2 at 2 weeks ($P < 0.01$), 50.0 mL/min/1.73 m^2 versus 60.3 mL/min/1.73 m^2 at 3 months ($P < 0.01$), and 49.4 mL/min/1.73 m^2 versus 61.2 mL/min/1.73 m^2 at 1 year ($P < 0.01$). Delayed graft function was observed in 41.7% of the patients with significant TR compared to 12.5% of those without significant TR ($P < 0.01$). No patients with significant TR required dialysis after 1 year. 1-year major adverse cardiac events were nonsignificantly higher among patients with significant TR (20.8% vs 8.1%; $P = 0.16$).

Limitations: Retrospective design and relatively small TR population.

Conclusions: The presence of significant TR among kidney transplant recipients was associated with a lower eGFR at 2 weeks, 3 months, and 1 year following transplant, although all remained dialysis independent at 1 year.

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Significant tricuspid regurgitation (significant TR), defined as at least moderate regurgitation based on echocardiography criteria,[1,2] is a robust marker of poor prognosis regardless of etiology.[3-5] Its presence is associated with increased morbidity and mortality, marked by a low cardiac output state and systemic venous congestion of the systemic veins. Both of these manifestations of significant TR may act synergistically to compromise kidney function.[6,7] Additionally, in the setting of a reduced glomerular filtration rate (GFR), volume overload and cardiac afterload may increase, further worsening the valvular regurgitation resulting in cardiorenal syndrome.[8,9]

Significant TR is relatively common among patients receiving hemodialysis, with reported incidence of up to 13.9%.[10] Incidence may very as the estimation of TR severity using echocardiography can be biased by the patient's volume state, the systemic blood pressure, and the heart rhythm at the time of the study. Kidney transplantation is the preferred treatment for kidney failure patients regardless of their TR status. However, the presence and persistence of significant TR may increase the risk of poor kidney function in the allograft. Hence, we aimed to assess the short- and long-term risk of significant TR in a cohort of patients with transplanted kidneys.

METHODS

Study Samples and Design

The study group was selected from a retrospective registry of patients who underwent kidney transplantation between 2016 and 2019 at Rabin Medical Center, a tertiary medical center in Israel. The data on the patients in this registry included clinical, laboratory, and cardiac echocardiography details and were updated regularly during the routine follow-up.

Study patients included adults older than 18 years. Patients who had concomitant heart or liver transplantation were excluded. For each patient, a full medical history was obtained including information regarding patient demographics (age and gender), medical history (diabetes, hypertension, smoking, chronic obstructive pulmonary disease, peripheral vascular disease, cerebrovascular accidents, and lipid profile), duration of dialysis until

PLAIN-LANGUAGE SUMMARY

Significant tricuspid regurgitation (TR) is associated with increased mortality rates and kidney failure, but its impact on kidney transplant recipients is poorly investigated. We examined how significant TR diagnosed pretransplantation affects kidney function within the first posttransplant year in a retrospective cohort study. Among 24 patients with significant TR, there was a consistent pattern of lower kidney function at 2 weeks, 3 months, and 1 year following transplantation, compared to 72 matched controls based on a propensity score. Results were statistically significant at all time points within the first year after transplant. These findings suggest that selected individuals with significant TR are able to undergo successful kidney transplantation, although with worse kidney function following transplantation.

transplantation, the presence of an arteriovenous fistula, and the transplantation date and allograft source (living vs deceased).

The TR status was determined in an echocardiography study within the year before the transplantation. TR severity was mainly determined using visual and semi-quantitative measures. A large, wide jet with the vena contracta width equal or greater than 0.4 cm combined with a dense, parabolic, or triangular regurgitant jet on a continuous wave doppler, along with the intermediate of a large hepatic flow convergence zone, were used for the diagnosis of significant TR.[1,2] Further data collected from this study included heart chambers' dimensions, the estimated left ventricular ejection fraction, the right ventricular function, other valves' function, and the estimated systolic pulmonary pressure.

The study protocol and data collection were approved by Rabin Medical Center's human research committee, who deemed the need for consent unnecessary (approval ID: RMC-515-19).

Study End Points

The primary end point was the estimated GFR (eGFR) calculated by the Chronic Kidney Disease Epidemiology Collaboration equation[11] at the following 3 time points within the first year after the transplantation: 10-20 days (2 weeks), 60-120 days (3 months), and 335-395 days (1 year). If estimated more than once at each period, we averaged the eGFR.

The secondary end points were overall mortality and major advanced cardiac events at 1 month and at 1 year, including nonfatal myocardial infarction, all-cause mortality, and hospitalization owing to cardiovascular disease following the transplantation. Myocardial infarction was defined according to the fourth universal definition of myocardial infarction type 1 or type 2.[12]

Data regarding mortality were retrieved from the Israel Ministry of Health's database. In-hospital and clinical events were retrospectively collected in the institutional database. If necessary, we collected data pertinent to the study from other electronic databases. All adverse events were confirmed separately by 2 researchers (KS and TS).

Statistical Analysis

Continuous data were summarized as means and standard deviations or medians and interquartile ranges and were compared using t tests. Categorical variables were presented as frequencies and were compared using χ^2 or Fisher exact tests. The normality of variable distributions was assessed using the Kolmogorov-Smirnov test. For the primary analysis, we compiled a cohort of propensity score–matched patients with 1:3 ratio between patients treated with significant TR and controls, within a caliper 0.05 standard deviation of the propensity score. Propensity score matching was performed using a "closest neighbor, greedy" algorithm, attempting to match patients with TR with control patients with the closest propensity score. The propensity score was derived from a multivariable logistic regression model that included significant TR, considered as the independent (outcome) variable, and all baseline clinical characteristics as covariates. The propensity score–matched cohort was analyzed for the main outcome. Binary logistic regression analyses were performed to identify independent predictors of the primary end point. Covariates for the model were chosen according to their known association with significant TR and outcomes, and included age at transplantation, gender, diabetes, duration of dialysis before the surgery, the presence of active arteriovenous fistulas, estimated left ventricular ejection fraction, the presence of moderate or severe mitral regurgitation, systolic pulmonary pressure, and the source of the donor organ. Effect sizes were presented as odds ratios and 95% confidence intervals. A P value of <0.05 was considered statistically significant. (All statistical analysis were performed with the IBM SPSS statistics V.29 software (IBM).

RESULTS

Six hundred four (604) patients underwent kidney transplantation between 2016 and 2019. Of them, 47 patients were excluded owing to the following reasons: 27 owing to concomitant liver transplantation and 20 owing to insufficient echocardiography data. Out of the final 557 patients, 26 patients had significant TR in the pretransplant echocardiography study. All of them had functional TR. The baseline characteristics and the echocardiographic characteristics of the entire cohort are summarized in Tables S1 and S2. Following propensity and adjustment for confounders, 24 significant TR patients were matched with 72 control patients in 1:3 ratio and were subsequently compared for outcomes. The clinical characteristics of these matched groups are depicted in Table 1. The mean

Table 1. Baseline Characteristics of Patients Following Propensity Matching

Variable	PS-Matched Significant TR; n = 24	PS-Matched Controls; n = 72	*P* Value	Standardized Difference
Age (mean, y)	47.9 ± 19	48.2 ± 15.8	0.9	0.02
Duration of dialysis (mean, mo)	65.1 ± 30.6	64.2 ± 35.1	0.4	0.05
AV fistula (%)	16 (66.7)	47 (65.2)	0.5	0.04
Living donor (%)	8 (33.3)	27 (37.5)	0.5	0.05
Diabetes (%)	18 (75.0)	55 (76.4)	1	−0.001
Hypertension (%)	22 (91.7)	65 (90.2)	0.4	0.05
Known IHD (%)	7 (29.2)	22 (30.6)	0.8	−0.003
Estimated EF (mean, %)	55 ± 7.5	55.3 ± 7.6	0.7	0.01
Right ventricular dysfunction (%)	3 (12.5)	7 (9.7)	0.1	0.03
Estimated PASP (mean, mmHg)	45.4 ± 12.8	42.9 ± 13.2	0.2	0.02
Moderate/severe mitral regurgitation (%)	12 (50.0)	35 (48.6)	0.3	0.05

Abbreviations: AV, arteriovenous; EF, ejection fraction; IHD, ischemic heart disease; PASP, pulmonary artery systolic pressure; PS, propensity score; TR, tricuspid regurgitation.

age, the duration of dialysis, the presence of arteriovenous fistulas, and the rates of diabetes, essential hypertension, and ischemic heart disease were similar in both groups.

Echocardiographic characteristics are detailed in Table 2. The rate of right ventricular dysfunction, pulmonary artery systolic pressure, and the presence of moderate or severe mitral regurgitation were similar in both groups.

The eGFR after transplantation in patients with significant TR was lower during the first year of follow-up. In the first 2 weeks after the transplantation, the mean eGFR was 41.2 mL/min/1.73 m^2 in patients with significant TR compared to 53.3 mL/min/1.73 m^2 in nonsignificant TR patients ($P < 0.01$). Results were consistent at 3 months (50.0 mL/min/1.73 m^2 vs 60.3 mL/min/1.73 m^2; $P < 0.01$) and at 1 year (49.4 mL/min/1.73 m^2 vs 61.2 mL/min/1.73 m^2; $P < 0.01$) (Fig 1).

Delayed graft function was observed in 10 (41.7%) of the patients with significant TR compared to 9 (12.5%) of those without significant TR ($P < 0.01$). None of the patients with significant TR had an eGFR lower than 15 mL/min/1.73 m^2 after 1 year, whereas 2 (2.8%) of the patients in the nonsignificant TR group had.

Binary logistic regression analysis identified significant TR as a predictor of the eGFR lower than 60 mL/min/1.73 m^2 in patients with significant TR at 2 weeks and 3 months after transplantation (odds ratio, 1.29; 95% confidence interval, 1.03-1.55; $P = 0.03$ and odds ratio, 1.30; 95% confidence interval, 1.05-1.56; $P = 0.04$, respectively) (Tables 3 and 4). Results were similar at 1 year (odds ratio, 1.25; 95% confidence interval, 0.98-1.43; $P = 0.08$) (Table 5).

The occurrence of major adverse cardiac events at 1 year was observed to be higher in patients with significant TR compared to the matched controls with nonsignificant TR, although the difference did not reach statistical significance (20.8% vs 18.1%; $P = 0.16$). Overall mortality at 1 year was comparable regardless of the presence of significant TR (Table 6).

Data regarding the TR grade postkidney transplant were available for 16 of the 26 patients with pretransplant significant TR. Among these, the TR grade has remained significant in 11 patients (68.8%), whereas it became mild in 3 patients and resolved in 2 patients.

DISCUSSION

Among kidney transplant recipients with moderate or severe TR, the eGFR was lower at 2 weeks, 3 months, and 1

Table 2. Echocardiographic Characteristics of Patients Following Propensity Matching

Variable	PS-Matched Significant TR; n = 24	PS-Matched Controls; n = 72	*P* Value
Estimated EF (mean, %)	53.0 ± 7.2	58.4 ± 7.1	0.03
LVEDD (mean, cm)	4.9 ± 0.5	4.9 ± 0.6	0.7
LVESD (mean, cm)	3.4 ± 0.6	3.2 ± 0.6	0.5
IVS (mean, cm)	1.1 ± 0.2	1.1 ± 0.2	0.6
LVPW (mean, cm)	1.1 ± 0.3	1.1 ± 0.2	0.3
LA diameter (mean, cm)	4.5 ± 0.9	4.3 ± 0.7	0.1
LA area (mean, cm^2)	25.6 ± 4.3	21.6 ± 6.2	0.1
Right ventricular dysfunction (%)	3 (12.5)	7 (9.7)	0.1
Right ventricular enlargement (%)	10 (41.7)	8 (11.1)	<0.01
Estimated PASP (mean, mmHg)	45.4 ± 12.8	42.9 ± 13.2	0.2
Moderate/severe mitral regurgitation (%)	12 (50.0)	35 (48.6)	0.3
Mean aortic valve gradients (mean, mmHg)	4.2 ± 3.2	6.6 ± 6.1	0.8

Abbreviations: EF, ejection fraction; IVS, interventricular septum; LA, left atrium; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LVPW, left ventricular posterior wall; PASP, pulmonary artery systolic pressure; PS, propensity score; TR, tricuspid regurgitation.

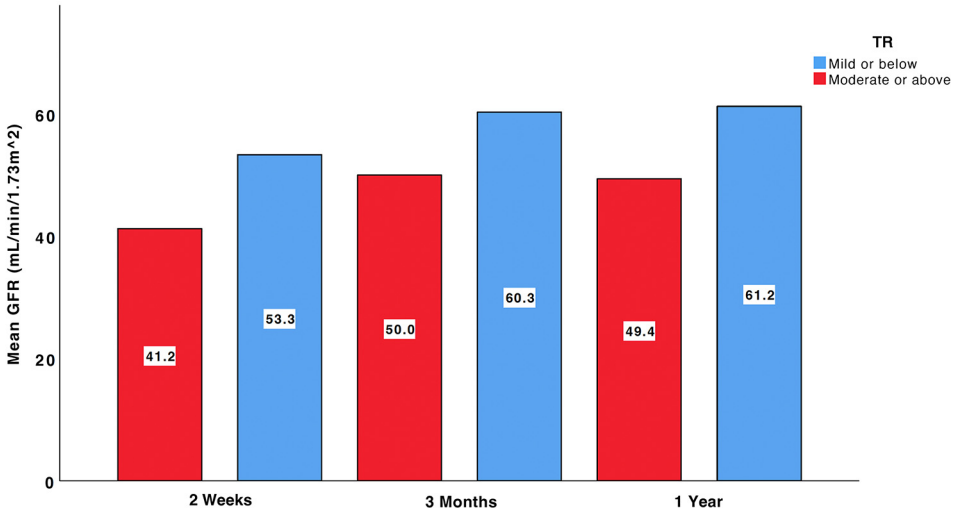

Figure 1. The mean eGFR at 3 intervals after kidney transplantation in patients with significant tricuspid regurgitation (significant TR) compared to matched controls with up to mild TR. Abbreviation: eGFR, estimated glomerular filtration rate.

year following transplant than among matched controls without significant TR. Critically, all patients with TR remained dialysis independent at 1 year and several had improvement in or resolution of TR. These observations merit attention as the lower eGFR may reflect a detrimental effect of significant TR on the transplanted kidney.[6,13] Previous studies have shown that the characteristic hemodynamics of significant TR reduces renal blood flow and increases renal interstitial hydrostatic pressure, thus augmenting kidney dysfunction.[10,14]

The eGFR at 3 months and 1 year after kidney transplantation is predictive of the long-term survival of the transplanted organ.[15-17] Thus, the association between the significant TR and the decline in the 3-month and 1-year posttransplant kidney function identified pretransplant significant TR as a prognostic factor for poorer long-term transplant success. Given this, identification of significant TR should be pursued in kidney transplant candidates, including younger candidates, for whom we wish to have sustained allograft function. We suggest a close follow-up for these patients, and considerations of early interventions, to reduce right-sided volume overload, including early arteriovenous fistula closure and valvular interventions.

Table 3. Binary Logistic Regression Analysis of an eGFR Lower Than 60 mL/min/1.73 m^2 at 2 Weeks Following Propensity Matching

	Odds Ratio	95% Confidence Interval	*P* Value
Age (per y)	1.02	0.99-1.06	0.1
Female gender	1.22	0.88-1.64	0.4
Diabetes	1.41	1.06-1.77	0.03
Estimated EF (per %)	1.04	0.91-1.21	0.3
Duration of dialysis (per mo)	1.08	0.91-1.26	0.1
AV fistula	1.10	0.90-1.32	0.4
Deceased donor	1.04	0.52-1.58	0.5
Moderate/severe mitral regurgitation	1.04	0.59-1.53	0.2
Significant tricuspid regurgitation	1.29	1.03-1.55	0.03

Abbreviations: AV, arteriovenous; EF, ejection fraction; eGFR, estimated glomerular filtration rate.

Shimada et al[18] showed in a rat model that kidney congestion reduces renal blood flow, reduces the GFR, and increases renal interstitial hydrostatic pressure. In addition, the organ congestion in this model was associated with injury to the tubulointerstitial and glomerular tissue and extracellular matrix expansion. The pathophysiology of this kidney injury was related to pericyte detachment in the congested kidney owing to receptors' upregulation of factors affecting pericyte-myofibroblast transition, such as transgelin and platelet-derived growth factors. Furthermore, the compression of peritubular capillaries, tissue hypoxia, and physical stress congregated to pericyte detachment, augmenting the extracellular matrix expansion and further tubular injury.

Changes in the eGFR associated with chronic significant TR are well known in native kidneys.[13,14,19] In this study, we describe a state in which a normal kidney was abruptly exposed to the hemodynamics of significant TR. The model of acute kidney injury in heart failure can be an applicable model to explain the malfunction of a normal transplanted kidney when set to function under a significant TR state. Acute kidney injury is traditionally explained by the decline of the forward cardiac output and mean atrial pressure leading to reduced kidney perfusion. However, Chen et al[20] suggested that increased central venous congestion, as seen in severe cardiac dysfunction, increases the kidneys' afterload, hence contributing to the

Table 4. Binary Logistic Regression Analysis of an eGFR Lower Than 60 mL/min/1.73 m^2 at 3 Months

	Odds Ratio	95% Confidence Interval	*P* Value
Age (per y)	1.03	1.01-1.05	0.03
Female gender	1.28	0.81-1.71	0.3
Diabetes	1.43	1.11-1.77	0.02
Estimated EF (per %)	1.04	0.90-1.20	0.6
Duration of dialysis (per mo)	1.13	0.76-1.55	0.8
AV fistula	1.11	0.98-1.23	0.3
Deceased donor	1.09	0.68-1.49	0.5
Moderate/severe mitral regurgitation	1.09	0.77-1.42	0.3
Significant tricuspid regurgitation	1.30	1.05-1.56	0.04

Abbreviations: AV, arteriovenous; EF, ejection fraction; eGFR, estimated glomerular filtration rate.

evolution of acute kidney injury even without volume overload. The consistent pattern of a lower eGFR in patients with significant TR at all time points within the first year after transplant may reflect the acute decompensation of the transplanted organ followed by the long-term impact of the valvular disease. The trend toward the higher incidence of major adverse cardiac events at 1 year supports this potential harmful impact.

Our findings highlight the need to diagnose significant TR in candidates for kidney transplantation. Further longer-term studies are needed to determine long-term outcomes in transplant recipients with significant TR as well as to investigate whether any interventions, including valve repair, are indicated to improve outcomes.

The study's retrospective design and the small size of the significant TR cohort are the main limitations. In addition, the study lacks echocardiographic and hemodynamic data relevant to determining the preload and afterload state in each patient and at each one of the time intervals when the eGFR was calculated. Furthermore, we cannot provide data regarding follow-up echocardiography studies after kidney transplantation for all patients. Thus, we cannot exclude other factors causing the decline in eGFR after 1 year. The presented data support the assumption that the significant TR state has not been resolved with kidney transplantation in most of the cases. This assumption may also be supported by the follow-up study by Prihadi et al.[21]

In conclusion, the presence of significant TR in patients who had kidney transplantation was associated with a lower eGFR at 2 weeks, 3 months, and 1 year posttransplant.

Table 5. Binary Logistic Regression Analysis of an eGFR Lower Than 60 mL/min/1.73 m^2 at 1 Year

	Odds Ratio	95% Confidence Interval	*P* Value
Age (per y)	1.05	1.02-1.09	0.02
Female gender	1.04	0.55-1.57	0.7
Diabetes	1.38	1.08-1.69	0.02
Estimated EF (per %)	1.08	0.88-1.39	0.5
Duration of dialysis (per mo)	1.12	1.02-1.22	0.03
AV fistula	1.04	0.81-1.27	0.5
Deceased donor	1.03	0.78-1.28	0.2
Moderate/severe mitral regurgitation	1.06	0.88-1.25	0.3
Significant tricuspid regurgitation	1.25	0.98-1.27	0.08

Abbreviations: AV, arteriovenous; EF, ejection fraction; eGFR, estimated glomerular filtration rate.

Table 6. Incidence of Mortality and MACE After Kidney Transplantation Following Propensity Matching

	PS-Matched Significant TR; n = 24	PS-Matched Controls; n = 72	*P* Value
MACE at 1 mo (%)	3 (12.5)	8 (11.1)	0.3
MACE at 1 y (%)	5 (20.8)	13 (18.1)	0.2
Mortality at 1 mo (%)	0 (0.0)	1 (1.4)	0.2
Mortality at 1 y (%)	0 (0.0)	2 (2.8)	0.1

Abbreviations: MACE, major adverse cardiac events; PS, propensity score; TR, tricuspid regurgitation.

SUPPLEMENTARY MATERIAL

Supplementary File (PDF)

Table S1. Baseline characteristics of patients according to the presence of significant tricuspid regurgitation in the total cohort.

Table S2. Echocardiographic characteristics of patients according to the presence of significant tricuspid regurgitation in the total cohort.

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