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Pre-Transplant Left Ventricular Geometry and Major Adverse Cardiovascular Events After Kidney Transplantation

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Background: Preventing major adverse cardiovascular events (MACE) after kidney transplantation motivates pre-transplant cardiac evaluation that includes two-dimensional transthoracic echocardiography (TTE). The relationship of relative wall thickness (RWT) to left ventricular mass index (LVMI) in predicting post-transplant MACE is unclear.

Material/Methods: In this multi-ethnic Canadian single-center cohort study, we identified 1063 adults undergoing pre-transplant TTE within 1 year pre-transplant and with minimum 6 months of post-kidney transplant follow-up for MACE, defined as a composite of coronary revascularization, myocardial infarction, stroke, and cardiac death. Left ventricular hypertrophy (LVH, >131 g/m² in men and >100 g/m² in women) and increased RWT (>0.45) were *a priori* used to define normal (no LVH, normal RWT), concentric remodeling (no LVH, increased RWT), eccentric hypertrophy (LVH, normal RWT), and concentric hypertrophy (LVH, increased RWT).

Results: There were 134 MACE over 3577 patient-years of post-transplant follow-up. Both LVH (HR 1.58, p=0.022) and high RWT (HR 1.44, p=0.041) predicted MACE in multivariate survival regression analysis independently of common pre-transplant MACE risk factors. Concentric remodeling, concentric hypertrophy, and eccentric hypertrophy all increased the risk for MACE (4.44, 5.05, and 5.55 events per 100 patient-years, respectively) versus normal echocardiography (2.71 events per 100 patient-years, all p<0.05 for difference). In Cox interactive regression analysis, LVMI and RWT were independently associated with MACE (p=0.015, p=0.025) and significantly interacted (p=0.008).

Conclusions: LV geometric parameters beyond LVH alone can assist post-transplant prognostication in kidney transplant candidates.

MeSH Keywords: Cardiovascular Diseases • Echocardiography • Hypertrophy, Left Ventricular • Patient Outcome Assessment

Abbreviations: CH – concentric hypertrophy; CKD – chronic kidney disease; CR – concentric remodeling; EH – eccentric hypertrophy; DBP – diastolic blood pressure; EDV – end-diastolic volume; ESKD – end-stage kidney disease; ESV – end-systolic volume; KT – kidney transplantation; KTR – kidney transplant recipients; LVEF – left ventricular ejection fraction; LVH – left ventricular hypertrophy; LVIDd – LV end-diastolic dimension; LVIDs – LV end-systolic dimension; LVMI – left ventricular mass index; LVPWtd – LV diastolic posterior wall thickness; MACE – major adverse cardiac events; MI – myocardial infarction; p-y – patient-years; RWT – relative wall thickness; SBP – systolic blood pressure; SWTd – diastolic septal wall thickness; TTE – transthoracic echocardiography

Full-text PDF: <https://www.annalsoftransplantation.com/abstract/index/idArt/913649>

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Background

Cardiovascular disease (CVD) is the leading cause of death among kidney transplant recipients (KTR) [1,2]; therefore, a pre-transplant evaluation for prevalent CVD is commonly performed [3]. However, kidney transplant (KT) candidates are often asymptomatic despite constituting a population with a significant CVD burden [4]. More involved cardiac evaluation such as stress testing and invasive coronary angiography is expensive or may lead to further kidney damage [5]. Two-dimensional transthoracic echocardiography (TTE) provides non-invasive and accurate evaluation of cardiac function, especially for detecting left ventricular hypertrophy (LVH), a function of left-ventricular mass index (LVMI), and a significant independent risk factor for major adverse cardiovascular events (MACE) [6]. TTE is a popular pre-transplant assessment tool for screening purposes. However, it remains unclear whether information obtained from pre-operative echocardiography apart from LVH is significantly correlated with post-kidney transplant cardiac outcomes. There is mixed evidence for positive and negative cardiac remodeling post-transplant, as risk factors for volume and pressure overload compound after transplantation [6,7]. TTE provides several measurements beyond LVMI, which although less often considered in TTE-based cardiac risk assessment may still predict MACE.

TTE provides data on relative wall thickness (RWT), which along with LVMI can be used to classify different left ventricular geometrical patterns [8]. For example, normal RWT and increased LVMI characterize a geometry known as eccentric hypertrophy. In studies of the general population, abnormal LV geometries relate adversely to prognosis, including carrying a higher incidence of MACE [9–12]. However, such data are scarce in KTR, particularly with respect to which pre-transplant LV geometries represent an increased risk for post-transplant MACE. As an inexpensive and widely performed pre-transplant screening procedure, any additional information that TTE provides might assist in customizing future pre- and post-transplant screening algorithms.

Material and Methods

Study sample and follow-up

St. Michael's Hospital (SMH) is an urban university-affiliated tertiary-care medical-surgical center that actively follows over 1700 prevalent KTR and performs approximately 130 adult single-organ kidney transplants annually. Post-transplant clinic visits typically occur weekly to month 1, biweekly to month 3, monthly to month 6, quarterly to month 12, and twice annually or annually thereafter. At each clinic visit, trained personnel record anthropometry and resting blood pressure (BP).

Ethnicity is recorded based on self-report. Laboratory testing is performed as close to each clinic visit as possible, with additional testing performed between visits according to a separate, more frequent schedule. The eGFR is calculated by the Modification of Diet in Renal Disease-7 equation [13]. As part of a standard protocol of evaluating KT candidates, a resting TTE and some form of cardiac stress testing is performed prior to transplantation. KT candidates who are found to demonstrate abnormalities by TTE or other tests are then referred to a cardiologist, after which these candidates may proceed to coronary angiography or other testing to help inform the decision about proceeding to kidney transplantation.

For this study, all KTR followed at SMH who underwent TTE within one year pre-transplant and were transplanted between October 1, 2006 and December 1, 2015 were first identified from the clinical electronic database. We then conducted an analysis of this population as of May 31, 2016 to ensure that all these patients had a minimum of 6 months potential post-transplant follow-up. No other exclusion criteria were applied for the study. The primary aim of this retrospective cohort study was to assess associations between parameters of pre-transplant LV geometry and post-transplant MACE. The secondary aim was to specifically assess the impact of an increased RWT or LVH on post-transplant MACE. Institutional research ethics board approval (REB10-204) was obtained for the study.

Patient assessment and data collection

MACE are routinely captured by transcriptions from local and citywide hospital databases as part of routine care, and supplemented by patient and family physician interviews where necessary. MACE was defined as the composite incidence of coronary revascularization, myocardial infarction, stroke, and cardiac death. Post-transplant MACE events were reviewed by at least 2 investigators.

TTE parameters were obtained from the transcribed reports of regional institutions that used standard M mode and two-dimensional images. For each TTE examination, left atrial diameter, diastolic septal wall thickness (SWTd), diastolic LV posterior wall thickness (LVPWTd), LV end-diastolic dimension (LVIDd), LV end-systolic dimension (LVIDs), end-systolic volume (ESV), and end-diastolic volume (EDV) were determined according to American Society of Echocardiography recommendations [14]. LVMI, RWT, and LV ejection fraction (LVEF) were calculated according to the same recommendations.

Patients were classified as having LVH if they had an LVMI greater than 100 g/m² in women and greater than 131 g/m² in men [15]. RWT, calculated using the formula $2 \times \text{LVPWTd} / \text{LVIDd}$, was considered increased when greater than 0.45 [16]. These divisions of LVMI and RWT were used to create 4 classifications

of LV geometry: normal (no LVH and normal RWT), concentric remodeling (no LVH and increased RWT), eccentric hypertrophy (LVH and normal RWT), and concentric hypertrophy (LVH and increased RWT). The interaction between these dichotomized parameters was also evaluated.

Since this study was a retrospective review of clinic data pertaining to a prevalent KTR population, individual informed consent was not obtained. The study was performed in accordance with the 2000 Declaration of Helsinki and the 2008 Declaration of Istanbul.

Statistical analysis

All analyses were conducted with R, version 3.4.0 (the R Foundation for Statistical Computing). All data are reported as mean \pm SD, unless otherwise stated. Two-tailed *p* values below 0.05 were taken to imply statistical significance. All missing values were handled by exclusion from relevant analysis without imputation. Comparisons were made using the *t* test, Fisher exact test, chi-square test, competing risks analysis, or Cox proportional hazards analysis, as appropriate. Gray's modified chi-square statistic was used to test for differences between groups in competing risks analysis. Normality and skewness were assessed visually or with skewness tests for all subgroups, supplemented with the Shapiro-Wilk normality test for small-*n* subgroups. The proportionality of hazards assumption was tested with Schoenfeld residuals-based methods in R [17]. Selected covariates for multiple Cox regression models were based on biological plausibility, using variables determined at or prior to transplant, since risk was measured from transplantation [18].

Results

Pre-transplant patient characteristics across LV geometries

A total of 1063 patients with pre-transplant TTE were identified on initial screening, corresponding to 86.8% of all patients transplanted during the study period. Of these, 469 (44%) patients were classified as normal, 340 (32%) as concentric remodeling (CR), 147 (14%) as concentric hypertrophy (CH), and 107 (10%) as eccentric hypertrophy (EH). Baseline transplant characteristics are shown and compared in Table 1. Recipients of a live donor transplant were overrepresented in the normal LV geometry group, and less represented among those with CH (42.9% normal vs. 24.5% CH, $p < 0.001$). Males were less likely to have LVH. With respect to other demographic parameters, no significant differences arose between groups. There was no statistically significant difference in pre-transplant MACE in the various hypertrophied groups. However, hypertension and diabetes as the primary cause of ESKD were each associated with abnormal LV geometry, particularly CH.

Early post-transplant patient characteristics and long-term graft outcomes

Table 2 outlines patient characteristics at 3 months post-transplant. Those with CH tended to have a lower BMI (24.7 ± 5.0 in CH vs. 26.44 ± 5.2 in normal, $p = 0.006$). Beta-blockers and angiotensin-II blockers were prescribed more in patients with CH. There were no significant differences in renal function or microalbuminuria, with GFR estimated by the Modification of Diet in Renal Disease formula. There was no difference in early graft function among the various groups. However, the cumulative incidence of graft failure was significantly higher with all abnormal LV geometries versus normal geometry (ranging from 19.7 to 22.4% for abnormal geometry vs. 12.1% for normal geometry, $p = 0.004$).

MACE incidence post-transplant

Figure 1 depicts the differences in post-transplant MACE incidence expressed as the number of events per 100 patient-years. A higher MACE incidence was associated with the presence of any abnormal LV geometry. Differences between CR, CH, or EH and normal all reached statistical significance ($p < 0.05$).

MACE survival differences tested by the modified chi-square method are illustrated through cumulative incidence curves as shown in Figure 2 in which individual incidence curves for each geometry are shown. In competing risks analysis with non-cardiac death, MACE incidence for any non-normal LV geometry was significantly greater (15% vs. 10% at 1500 days post-transplant, $p = 0.003$). Dichotomizing by $RWT > 0.45$, MACE incidence was significantly higher in those with increased RWT than with normal RWT (15% vs. 11% at 1500 days, $p = 0.046$). Dichotomizing by LVH, those with LVH pre-transplant were at higher risk for both MACE and competing all-cause mortality (14% vs. 10% at 1500 days, $p = 0.030$ for MACE, 3% vs. 5% at 1500 days, $p = 0.008$ for all-cause mortality).

Table 3 provides univariate Cox regression results for all measured echocardiogram parameters to MACE incidence. Both increased RWT and LVH dichotomization associated adversely with MACE-free survival (RWT: 1.416 (95% confidence interval 1.01–1.99) $p = 0.044$; LVH: 1.521 (95% confidence interval 1.06–2.18) $p = 0.026$). When RWT and LVMI were considered as continuous parameters, both continued to associate significantly with MACE (RWT: 8.72 (1.82–41.9) $p = 0.014$; LVH: 1.01 (1.00–1.01) $p < 0.001$). Other echocardiographic parameters correlating strongly with increased MACE risk were left atrial diameter, right ventricular systolic pressure, LVPWTd, and SWTd (all $p < 0.01$).

Table 1. Baseline patient or transplant characteristics across LV geometry types.

Characteristic	Normal (n=469)	Con. rem. (n=340)	Con. hyp. (n=147)	Ecc. hyp. (n=107)	P
Demography data					
Avg. f/u from tx. (years)	3.65±2.44	3.49±2.36	3.98±2.58	3.69±2.6	0.247
Age at tx. (years)	52.5±13.0	52.9±12.9	51.8±13.4	51.3±14.	0.656
Male (N, % male)	291, 62.1%	235, 69.1%	71, 48.3%	48, 44.9%	<0.001
Ethnicity (N, % white)	104, 22.2%	69, 20.29%	25, 17.01%	18, 16.8%	0.423
Smoking (N, % smoking)	173, 36.8%	126, 37.0%	53, 36.0%	37, 34.5%	0.968
Pre-tx MACE	54, 11.5%	49, 14.4%	23, 15.7%	18, 16.8%	0.333
ESKD cause (N, %)					
Hypertension	44, 9.4%	43, 12.7%	30, 20.4%	12, 11.2%	0.005
Polycystic KD	63, 13.4%	29, 8.5%	12, 8.2%	9, 8.4%	0.073
Diabetes Mellitus	79, 16.8%	78, 22.9%	35, 23.8%	14, 13.1%	0.026
Glomerulonephritis	163, 34.8%	100, 29.4%	42, 28.6%	33, 30.8%	0.316
Unknown/other	141, 30.1%	104, 30.6%	41, 27.9%	43, 40.2%	0.164
Allograft Data					
Hemodialysis	206, 43.9%	165, 48.5%	77, 52.4%	59, 55.1%	0.039
Not dialysed	68, 14.5%	34, 10.0%	6, 4.1%	5, 4.7%	<0.001
Avg. dialysis vintage	4.50±4.0	4.64±3.7	6.70±4.8	5.05±3.8	<0.001
Second + transplant	24, 5.1%	19, 5.6%	12, 8.2%	5, 4.7%	0.534
Live donor (N, % live)	201, 42.9%	108, 31.8%	36, 24.5%	33, 30.8%	<0.001

MACE defined as vascular surgery, MI, stroke, or cardiac-related death. Comparisons made with 2x4 contingency table analysis for dichotomous factors, or ANOVA for continuous variables.

Multiple regression for MACE incidence

Multivariate regression analyses associating RWT and LVH/LVMI with MACE incidence are shown in Table 4. Both RWT and LVH were independent of typical risk pre-transplant predictors of post-transplant CVD. Male sex, pre-transplant MACE, live donation, and smoking status were included in the model. Both LVH and LVMI associated significantly with MACE incidence (HR 1.58 (1.07–2.35) and 1.01 (1.00–1.01), respectively, both $p < 0.05$). RWT was significantly associated as a continuous, and dichotomized, parameter (continuous: HR 6.72 (1.27–35.7) $p = 0.025$; dichotomous: HR 1.44 (1.02–2.01) $p = 0.041$). When considering geometric classifications, any non-normal cardiac geometry was also independently associated with MACE (HR 1.60 (1.09–2.37), $p = 0.018$). LVMI and RWT significantly interacted ($p = 0.008$).

Discussion

In this retrospective cohort study of a large, multi-ethnic KTR population, we suggest a possible predictive value to common pre-transplant echocardiographic parameters in the incidence of post-transplant MACE. The continuous variables LVMI and RWT and the categorical variable LVH were associated with post-transplant MACE, but these associations are unsurprisingly not fully independent of prior MACE, and may be mediated through causal mechanisms involving diabetes and systolic blood pressure. Nonetheless, identifying these pre-transplant echocardiographic abnormalities may assist in better focusing efforts on controlling these other long-term cardiovascular risk factors in KT candidates both before and after transplantation. RWT independently predicts cardiovascular events in patients with both hypertension and diabetes [19], and after myocardial infarction [20]. Specific classes of antihypertensive agents may affect RWT and LVMI selectively [21]. These associations and

Table 2. Three-month and long-term post-transplant patient characteristics across LV geometries.

Characteristic	Normal (n=469)	Con. rem. (n=340)	Con. hyp. (n=147)	Ecc. hyp. (n=107)	P
Physical measurements					
SBP (mmHg)	129.4±41.3	130.1±47.0	128.5±44.0	131.2±48.0	0.958
DBP (mmHg)	79.7±25.6	79.1±29.2	77.3±26.7	78.4±28.8	0.834
Height (cm)	168.±56.4	170.±64.0	166.2±58.0	165.7±60.0	0.929
Weight (kg)	75.6±28.1	76.9±31.7	69.0±26.2	72.6±29.6	0.062
BMI (kg/m ²)	26.44±5.2	26.5±5.00	24.72±5.0	26.3±5.55	0.006
Laboratory measurements					
Fasting glucose (mmol/L)	6.09±2.87	6.88±3.67	6.27±3.06	6.56±3.22	0.012
Random glucose (mmol/L)	6.58±3.50	7.28±4.18	6.63±3.15	7.10±3.91	0.073
HbA1c (% glycated Hb)	0.06±0.02	0.06±0.02	0.06±0.02	0.06±0.02	1.000
Total cholesterol (mmol/L)	4.44±1.81	4.50±2.07	4.55±1.78	4.34±1.76	0.840
HDL-cholesterol (mmol/L)	1.32±0.59	1.34±0.66	1.34±0.61	1.31±0.62	0.960
LDL-cholesterol (mmol/L)	2.36±1.24	2.42±1.41	2.49±1.24	2.23±1.14	0.502
Triglycerides (mmol/L)	1.82±1.50	1.70±1.06	1.73±1.10	1.78±0.98	0.661
C-reactive protein (mg/L)	5.20±10.7	7.77±21.1	5.42±10.3	4.87±8.43	0.098
Renal function/outcomes					
Serum creatinine (micromol/L)	128.±73.1	134.±80.8	120.3±58.0	121.1±67.0	0.245
Estimated GFR (mdrd)	54.6±24.0	54.0±26.6	53.7±23.5	55.1±25.9	0.968
MACR (mcg/L)-to-(mmol/L)	11.5±33.7	17.4±59.4	8.06±12.7	13.9±47.9	0.157
24 h urine prot. (mmol/L)	0.52±0.42	0.74±0.81	0.61±0.55	0.95±0.80	0.002
Graft failure incidence	57, 12.1%	69, 20.3%	29, 19.7%	24, 22.4%	0.004
MACE (per 100p-y)	43, 2.66	47, 4.31	26, 4.86	18, 5.34	<0.001
Medications (N, %)					
Statin	224, 47.8%	174, 51.2%	67, 45.6%	50, 46.7%	0.634
ACE inhibitor	54, 11.5%	26, 7.7%	11, 7.5%	7, 6.5%	0.145
Beta-blocker	163, 34.8%	142, 41.8%	74, 50.3%	48, 44.9%	0.004
Angiotensin II blocker	57, 12.2%	54, 15.9%	31, 21.1%	19, 17.8%	0.028

MACE defined as post-transplant MI, stroke, angioplasty/CABG, other vascular surgery, or cardiac-related death. Comparisons made with 2×4 chi-square analysis for dichotomous factors, or ANOVA for continuous factors. Graft failure and MACE are long-term outcomes.

interventions have not been evaluated in ESKD patients or in KT candidates. The present study highlights that post-transplant cardiac prognosis with TTE can be further informed by LV geometry beyond LV hypertrophy alone. Increased RWT in isolation (concentric remodeling) can pose an equivalent hazard as concentric or eccentric hypertrophy, for example, and

neither concentric nor eccentric hypertrophy geometries are associated with a greater hazard than the other. Absence of LVH does not imply absence of cardiovascular risk.

This study also provides evidence for the validity of the risk classification criteria typically used in the CKD population

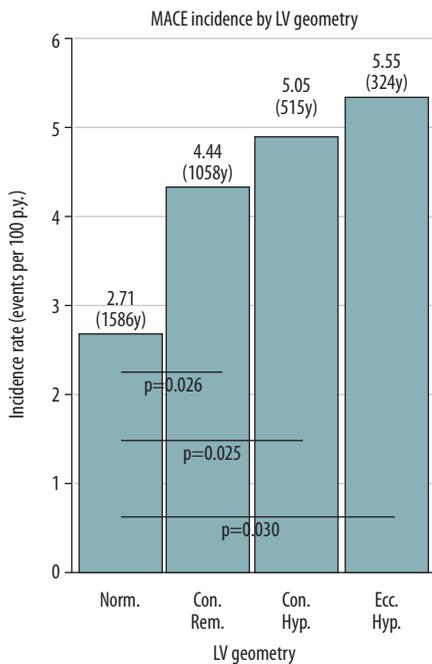


Figure 1. Incidence rate of MACE within pre-transplant LV geometry groups. Units expressed in events per 100 patient-years at risk. Incidence rate is shown for each group over each bar. Significance was assessed via *t* test with comparison to normal geometry. Incidence rate differences assessed using an expanded definition of MACE (any cardiac event), including new-onset diagnosed coronary artery disease, congestive heart failure, or atrial fibrillation within tracked events.

when assessing the importance of an increased RWT or LVMI. Transplant candidates with RWT over 0.45 or LVMI of 100–130 (depending on sex) may be at additively heightened risk for post-transplant MACE. Hence, the cutoffs for risk classification used in CKD and the general population appear to apply reasonably to pre-transplant candidates, in whom echocardiographic data are routinely obtained. Increased RWT and LVMI may situate along the causal pathway of other prognosticators of post-transplant cardiac health, such as pre-transplant MACE, diabetes, and uncontrolled hypertension. Although an association between echocardiography and MACE risk is well-documented in the general and dialysis/non-dialysis CKD populations [9,10,22], evidence is scarce in the CKD-KT candidate population [23]. To our knowledge, only one study regarding pre-transplant TTE and post-transplant MACE has been conducted, with a focus on wall motion abnormality in dobutamine stress echocardiography rather than resting LV geometry [24].

Concerns regarding kidney transplant candidate survival and allograft success drive extensive screening for cardiovascular disease prior to transplant. Such screening must be employed judiciously however, since pre-transplant tests and procedures are labor-intensive and expensive [23]. Echocardiography has previously been shown prospectively to exclude more invasive testing such as coronary angiography [21,22]. In other studies [24–27], TTE and TTE-based multi-factor predictive models achieve high sensitivity and specificity for post-transplant MACE incidence, and in special subpopulations such as insulin-dependent diabetic KT candidates [28]. Many KT candidates have TTE performed as part of their dialysis initiation screening without screening for coronary artery disease *per se*. Increasing the interpretability and quantity of information

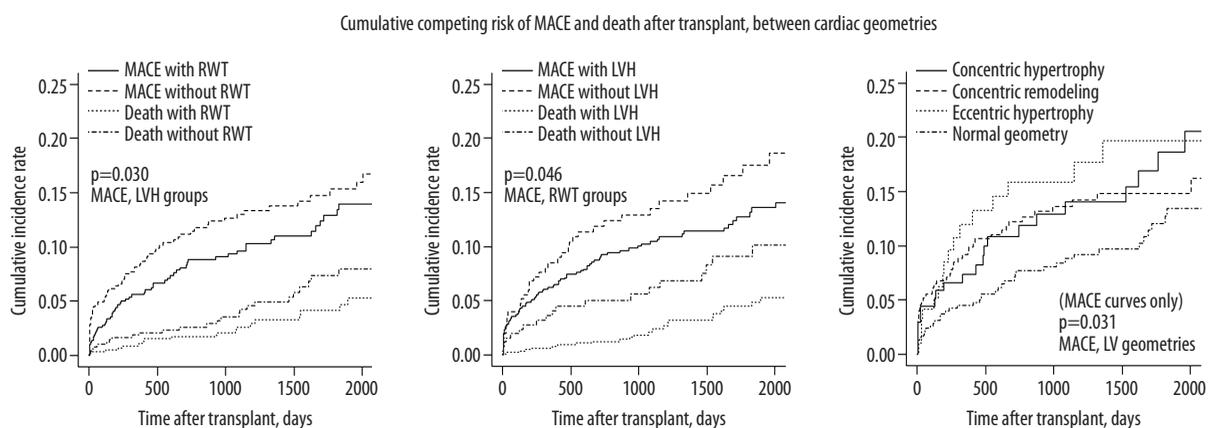


Figure 2. Cumulative incidence of MACE across strata of LV geometrical parameters. All-cause mortality included as a competing risk for all tests, and cumulative all-cause mortality incidence is plotted in the LVH and RWT panels. P values indicate the significance of difference between groups for MACE incidence only. Death incidence curves for panel 3 were excluded to improve clarity.

Table 3. Univariate Cox proportional hazards regressions for MACE incidence.

Variable	HR (95% CI)	P
LV geometry parameters		
“Increased” RWT (>0.45)	1.416 (1.01–1.99)	0.044
LVH (LVMI >100 or >131)	1.521 (1.06–2.18)	0.026
Other echocardiogram data		
RV syst. pressure (mmHg)	1.024 (1.01–1.04)	0.007
Left atrial diameter (cm)	1.564 (1.21–2.03)	<0.001
LVIDd (cm)	1.086 (0.82–1.42)	0.550
LVIDs (cm)	1.097 (0.84–1.43)	0.498
LVPWtd (cm)	3.996 (1.68–9.50)	0.002
SWTd (cm)	5.412 (2.63–11.0)	<0.001
End systolic volume (cm ³)	1.002 (1.00–1.01)	0.539
End diastolic volume (cm ³)	1.002 (1.00–1.01)	0.466
LV ejection fraction (%)	0.894 (0.26–3.12)	0.861
Relative wall thickness	8.715 (1.82–41.9)	0.007
LV mass index (g/m ²)	1.008 (1.00–1.01)	<0.001

RWT=2*LVPWd/LVIDd, unitless ratio of cm/cm. LVH diagnosis based on an LVMI>100 g/m² in women and >131 g/m² in men.

that can be derived from TTE will further improve the ability of TTE and similar non-invasive echocardiography to potentially preclude other tests, if such tests are not specifically indicated. TTE provides more information than just the detection of LVH and the presence of regional wall motion abnormalities, whose finding usually dictates further cardiac investigation. Prospective clinical trials of intensive pre-transplant screening are currently lacking; recruitment to future trials of CVD prevention in KT candidates and KT recipients may be better informed by the enrichment with subjects who demonstrate select abnormalities on screening echocardiography.

This retrospective cohort study is inherently constrained by non-centralized echocardiographic evaluations, although its pragmatic applicability to other transplant institutions is also enhanced by the large sample size, making any center-based effect unlikely. Interventions were not controlled between groups, although medication profiles tended to not differ significantly between groups. Furthermore, the time between TTE and transplant varied, reaching up to 1 year prior to transplant. Depending on proximity to last dialysis, echocardiographic measurements of cardiac chamber sizes (although not necessarily wall thickness) may have been influenced by patient blood volume [21]. As this was primarily an echocardiographic investigation, we did not have information on electrocardiographic abnormalities and had limited information about pre-transplant or post-transplant biochemical and pharmacological parameters. We studied only patients who were successfully transplanted, not those who were delisted or died while waiting for a transplant. Lastly, the correlations in this study do not imply

Table 4. Multivariate Cox proportional hazards regressions for MACE incidence, comparing RWT, LVMI, and LVH type to common predictors of post-transplant MACE.

Variable	HR (95% CI)	P	Variable	HR (95% CI)	P
Non-normal LV	1.60 (1.09–2.37)	0.018	RWT (cm/cm)	6.72 (1.27–35.7)	0.025
Male	1.40 (0.91–1.13)	0.122	Male	1.37 (0.89–2.09)	0.147
Live donor	0.74 (0.50–1.11)	0.144	Live donor	0.75 (0.50–1.12)	0.154
Smoking	1.57 (0.07–2.29)	0.020	Smoking	1.57 (1.08–2.30)	0.019
Prior MACE	2.29 (1.49–3.52)	0.001	Prior MACE	2.29 (1.49–3.51)	0.002
LVH	1.58 (1.07–2.35)	0.022	LVMI (g/m²)	1.01 (1.00–1.01)	0.015
Male	1.47 (0.96–2.27)	0.079	Male	1.27 (0.82–1.95)	0.279
Live donor	0.74 (0.50–1.11)	0.144	Live donor	0.74 (0.50–1.11)	0.150
Smoking	1.50 (0.92–2.19)	0.039	Smoking	1.47 (1.01–2.16)	0.047
Prior MACE	2.31 (1.50–3.55)	0.001	Prior MACE	2.29 (1.49–3.53)	0.002

All variables listed except “LVMI” and “RWT” are dichotomous (0=no, 1=yes). Normal LV refers to RWT<0.45 and no LVH. RWT=2*LVPWd/LVIDd.

causation, but simply identify a subset of KT candidates for further study. The main strength of the study, however, is the size and multi-ethnicity of its cohort with minimal exclusions to better represent the largest possible proportion of KT candidates receiving a transplant, as well as a long post-transplant follow-up of these candidates for the occurrence of MACE.

Conclusions

TTE in KT candidates beyond LVH alone can better inform the risk of post-transplant MACE. Additional information on LV

geometry, including RWT, LVMI, and LVH patterns, improves the assessment of that risk and may serve to more effectively address other CVD risk factors in the pre-transplant ESKD population. Considering abnormal LV geometrical parameters, including concentric remodeling or eccentric hypertrophy, as being at risk for MACE besides those with LVH, may also help identify potential KT candidates to enrich recruitment to prospective clinical trials of CVD prevention after kidney transplantation.

Conflict of interest

None.

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