Cardiovascular Revascularization Medicine 20 (2019) 588–593

Contents lists available at ScienceDirect

Cardiovascular Revascularization Medicine

Pre-Operative Cardiovascular Testing and Post-Renal Transplant Clinical Outcomes

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ARTICLE INFO

Article history:
Received 11 April 2019
Accepted 12 April 2019

Keywords:
Kidney transplant
Functional testing
Cardiovascular outcomes

ABSTRACT

Background: Cardiovascular disease, a major contributor to morbidity and mortality in chronic kidney disease and kidney transplant patients, is closely evaluated before kidney transplant. We aimed to characterize pre-transplant cardiac testing practices and post-transplant cardiac outcomes at a single academic center.
Methods: This was a retrospective, single-center analysis of consecutive adults receiving first renal transplant from 1/1/2016 to 6/31/2017. Data included demographics, medical history, and medications. Pre-transplant workup included echocardiograms, cardiac stress testing, coronary computed tomography, left heart catheterization (LHC), and any revascularization. Outcomes included all-cause mortality, cardiac mortality, myocardial infarction (MI), and myocardial injury.
Results: Our analysis included 235 patients with mean follow-up of 1.6 ± 0.53 years. Of these, 219 (93%) patients had non-invasive functional testing before transplant, with 198 normal and 21 abnormal. The most common modalities were dobutamine stress echocardiogram (88) and pharmacological myocardial perfusion imaging (60). Twenty-four (10%) patients had an LHC, including 14 abnormal studies, and 10 who subsequently underwent successful revascularization. There were 3 deaths, 2 that were cardiac-specific. There were no ST-elevation MIs and 1 Type I non-ST-elevation MI (NSTEMI), occurring 2 days after transplant. Of those patients with a 30-day post-operative troponin, 30 (13%) patients had an elevation due to a type II NSTEMI or myocardial injury.
Conclusions: Non-invasive functional testing is common prior to renal transplantation, with most being normal. Few patients are revascularized before transplantation. Perioperative death and acute coronary syndrome are rare, but troponin elevations due to type II NSTEMI and myocardial injury are common.

1. Introduction

Chronic kidney disease (CKD) and end-stage renal disease (ESRD) are closely linked to coronary artery disease (CAD). Kidney disease is an independent risk factor for cardiovascular disease, [1] and CAD is the leading cause of death in wait-listed kidney transplant candidates [2]. Cardiovascular disease is, therefore, closely evaluated during pre-transplant workup. Current guidelines recommend noninvasive cardiac stress testing on the basis of risk factors, with subsequent revascularization if appropriate [3]. However, there are no guidelines regarding the specific type or timing of testing, and the prognostic impact of cardiac testing remains unclear. This study aims to characterize pre-transplantation cardiac testing practices and to report post-transplant cardiac outcomes in patients receiving renal allografts.

2. Materials and methods

This is a single-center, retrospective cohort study. We included consecutive patients undergoing first renal transplantation at our institution between 1/1/2016 and 6/31/2017. Patients under age 18, receiving multi-visceral transplant (including small bowel, pancreas, and/or liver), or status post-prior kidney transplantation were excluded (Fig. 1). Electronic medical records were reviewed for demographic and

Abbreviations: ACE, angiotensin-converting enzyme; ACS, acute coronary syndrome; ARB, angiotensin receptor blocker; CABG, coronary artery bypass graft; CAD, coronary artery disease; CCB, calcium channel blocker; CK-MB, creatine kinase-MB; CKD, chronic kidney disease; CT, computed tomography; DSE, dobutamine stress echo; ECG, electrocardiogram; ESRD, end-stage renal disease; LHC, left heart catheterization; MI, myocardial infarction; MPI, myocardial perfusion imaging; MRA, mineralocorticoid receptor antagonist; NSTEMI, non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction; TTE, transthoracic echocardiogram.

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https://doi.org/10.1016/j.carrev.2019.04.017
1553-8389/

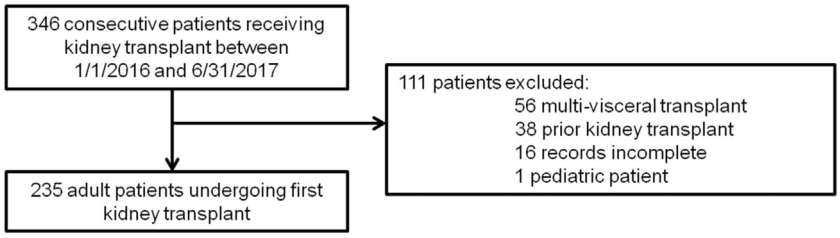

Fig. 1. Study inclusion criteria. Study enrollment by inclusion and exclusion criteria. A total of 346 patients underwent kidney transplant during our study period with 235 meeting inclusion criteria and 111 patients with exclusion criteria. Multi-visceral transplant included kidney-pancreas, kidney-pancreas-small bowel and kidney-liver transplants.

medical data, including medical history, medications, pre-transplant cardiac testing, and post-transplant clinical outcomes. This study proceeded with approval from the institutional review board.

Demographics included age, sex, and race. Past medical history included diabetes mellitus, hypertension, stable CAD, prior myocardial infarction (MI), cerebrovascular accident (CVA), current and prior tobacco use, years on dialysis, and type of dialysis. Patients were reviewed for use of antiplatelet, HMG-CoA reductase inhibitors (statins), insulin, beta-blockers (BB), angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARB), calcium channel blockers, mineralocorticoid receptor antagonists (MRA), and diuretics for a 6-month period before transplant.

Data regarding pre-transplant cardiac evaluation included resting transthoracic echocardiogram (TTE) and cardiac stress testing, the latter encompassing exercise stress echocardiogram, dobutamine stress echocardiogram (DSE), exercise myocardial perfusion imaging (MPI), pharmacologic MPI, and cardiac computed tomography (CT). If further coronary evaluation was performed, the left heart catheterization (LHC) was recorded, along with the revascularization of coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI). Post-transplant clinical outcomes included ST-elevation MI (STEMI), Type 1 non-ST-elevation MI (NSTEMI), all-cause mortality, and cardiac death. Type I NSTEMI was defined using the 4th universal definition of MI and injury, which is defined as detection of a rise and/or fall in troponin with at least 1 value above the 99th percentile and with at least 1 of the following: symptoms of acute myocardial ischemia; new ischemic electrocardiographic changes; development of pathological Q waves; imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with ischemic etiology; and identification of a coronary thrombus by angiography, including intracoronary imaging or by autopsy [4].

We used the 4th universal definition of MI and injury, which defines myocardial injury as troponin elevation above the 99th percentile, and further defines type II MI as acute myocardial injury with evidence of myocardial supply/demand mismatch, including ischemic symptoms, ischemic electrocardiogram (ECG) changes, pathologic Q waves, or imaging evidence of new ischemia. To identify Type II NSTEMI and myocardial injury, we captured any cardiac enzymes (troponins and creatine kinase-MB (CK-MB)) drawn in the postoperative period, and their overall peak. For patients with elevated troponins, we searched the chart for evidence of acute ischemia-related symptoms, ECG changes, or cardiac imaging findings, as well as the clinical judgment and diagnosis of the care team, if documented. Finally, overall survival and myocardial injury-free survival curves were generated using the Kaplan-Meier method [4].

3. Results

In total, 235 patients met inclusion criteria for our analysis. The mean length of follow-up was 1.63 years (SD 0.53). Our population had a mean age at transplantation of 53 ± 12.3 years, with a tendency toward male sex (56.6%) and African-American ethnicity (63.4%). The most common co-morbidity was hypertension (88.1%), and the most common pre-transplant medication class was beta blockers (59.6%). The most common etiology of CKD/ESRD was hypertension (36.7%) and diabetes mellitus (26.2%). Further baseline characteristics, pre-transplant medications, and etiology of CKD/ESRD are delineated in Tables 1 and 2.

TTEs were performed in 212 patients (90.2%) during pre-transplant workup (Table 3). The left ventricular (LV) systolic function was normal in 199 (93.8%) patients with a mean LV ejection fraction of 60.9% (Table 3). Thirteen (6.2%) patients had an abnormal study, with only 4 (1.9%) patients having moderate or severe LV dysfunction.

Table 1
Demographics and baseline characteristics.
Characteristics of the study population including baseline demographics, past medical history, years on dialysis and type of dialysis, donor type, and medications the patient was on within 6 months of transplant.

	N = 235 (percentage or SD)
Follow up (years)	
Mean	1.63 ± 0.53
Range	0.08–2.71
Age (years)	
Mean	53 ± 12.3
Range	19–77
Gender	
Male	133 (56.6)
Female	102 (43.4)
Race	
African-American	149 (63.4)
Caucasian	45 (19.1)
Asian	6 (2.6)
Other	35 (14.9)
Ethnicity	
Non-Hispanic	209 (88.9)
Hispanic	26 (11.1)
Diabetes	76 (32.3)
Hypertension	207 (88.1)
Hyperlipidemia	124 (52.8)
Stable coronary artery disease	18 (7.7)
Prior myocardial infarction	13 (5.5)
Prior stroke	18 (7.7)
Tobacco use	
Current	12 (5.1)
Prior	68 (28.9)
Dialysis	182 (77.4)
Years on dialysis	5 (2.1)
Hemodialysis	160 (68.1)
Peritoneal dialysis	38 (16.2)
Donor type	
Deceased donor kidney	149 (63.4)
Live donor kidney	56 (23.8)
Live donor kidney-national kidney registry	26 (11.1)
Live donor kidney-paired kidney exchange	4 (1.7)
Zero mismatch deceased donor kidney	0 (0.0)
Medications	
Aspirin	83 (35.3)
Statin	103 (43.8)
Insulin	46 (19.6)
Beta blocker	140 (59.6)
ACE inhibitor or aldosterone receptor blocker	102 (43.4)
Calcium channel blocker	105 (44.7)
Aldosterone antagonist	5 (2.1)
Diuretic	36 (15.3)

Table 2
Etiology of chronic kidney disease and end-stage renal disease.
Distribution of the etiologies for ESRD for the patients included in our study. Patients had multiple etiologies attributed to their kidney disease so total is higher.

Etiology	#	%
Hypertension	94	36.7%
Diabetes mellitus (type 1 and 2)	67	26.2%
Unknown, despite work-up	22	8.6%
Polycystic kidney disease	20	7.8%
Systemic lupus erythematosus	8	3.1%
IgA nephropathy	8	3.1%
Focal segmental glomerulosclerosis	8	3.1%
HIV	5	2.0%
Lithium toxicity	4	1.6%
Congenital	3	1.2%
Granulomatosis with polyangitis	3	1.2%
Obstructive uropathy	2	0.8%
Membranous glomerulonephritis	1	0.4%
Chronic interstitial nephritis	1	0.4%
Anti-glomerular basement membrane disease	1	0.4%
Nephrotic syndrome	1	0.4%
Pauci immune glomerulonephritis	1	0.4%
Post-infectious glomerulonephritis	1	0.4%
Medical cystic disease	1	0.4%
Bilateral nephrectomy	1	0.4%
Thin basement membrane disease	1	0.4%
Hemolytic uremic syndrome	1	0.4%
ANCA vasculitis	1	0.4%
Acute interstitial nephritis	1	0.4%
Total	256[a]	100.0%

[a] 21 patients had multiple etiologies attributed to their kidney disease so total is 256 and not 235.

In addition, only 1 (0.5%) patient had moderate or severe right ventricular dysfunction. Six studies (2.9%) showed wall motion abnormalities (Table 3). Pulmonary artery systolic pressure (PASP) was reported in 107 (51.2%) TTEs, with a mean pressure of 32.72 mmHg (Table 3).

During our index period, 221 patients (94.0%) had pre-transplant cardiac stress testing of any kind. Of those patients, 121 (51.5%) underwent stress echocardiogram, 98 underwent MPI (41.7%), and 2 underwent exercise stress ECG (0.9%) (Table 4). Among the 219 (93.2%) patients with stress imaging, 198 (90.4%) had normal studies. Overall, 200 (85.1%) had a normal workup, while 24 (10.2%) had an abnormal workup (Table 4). There were 12 (5.1%) patients with no stress test performed or stress test not found. DSE was the most common pre-transplant cardiac stress modality performed at our center, representing 89 (37.9%) patients. LHC occurred in 24 patients (10.2%), with 14 (6.0%) showing obstructive coronary disease (Table 4). Among patients with LHC, 10 were normal, 3 had 1-vessel disease, 5 had 2-vessel disease, 6 had 3-vessel disease (Table 5), and none had left main disease. All patients undergoing LHC had a prior stress test, including 15 with MPI and 8 with stress echo (Table 6), of which 10 had normal and 13 had abnormal results. Ten patients (4.2%) ultimately underwent revascularization before transplant, with 5 (2.1%) undergoing CABG, 4 (1.7%) undergoing PCI, and 1 (0.4%) undergoing CABG and PCI (Table 5).

During our follow-up period, there were 3 patients with mortality of any cause. Two patients had cardiac death, including 1 patient with a type I NSTEMI on post-operative day 2 followed by cardiac death 90 days after transplant, and another patient with cardiac death at 34 days post-transplant (Table 7). Of the 200 patients who had a normal pre-operative ischemic workup, 27 (13.5%) had death, NSTEMI, or myocardial injury (Table 8). Baseline characteristics and workup for patients with cardiac death or MI are delineated in Table 9.

In our study, 47 patients (20.0%) had at least 1 troponin checked in the postoperative period (Table 10). Of these, 16 were undetectable and 31 were elevated, including 6 patients who were determined to have type II NSTEMI after review of clinical information. The average post-transplant troponin was 3.55 ng/mL, with a range of 0.016 to 104.0, and average CK-MB was 3.58 ng/mL, with a range of 0.7 to 13.0 (Table 10). At 1-year follow-up, overall survival was >99%, and myocardial injury-free survival was 85% (Figs. 2, 3).

Table 3
Pre-transplant transthoracic echocardiogram (TTE) characteristics.
TTE results prior to transplant with special attention to left ventricular function, pulmonary artery systolic pressure, right ventricular function and wall motion abnormalities. If patients had multiple TTEs prior to transplant, the study closest to their date of transplant was used.

	N = 235 (percentage or SD)
Patients with pre-transplant TTE	212 (90.2)
LV function (%)	
EF mean ± SD	60.87 ± 6.734
Normal	199 (84.7)
Mild dysfunction	9 (3.8)
Moderate dysfunction	4 (1.7)
Severe dysfunction	0
Pulmonary artery systolic pressure (mmHg)	
PASP Reported (N)	133 (56.6)
Mean ± SD	32.08 ± 11.69
RV function	
Normal	207 (88.1)
Mild dysfunction	2 (0.9)
Moderate dysfunction	1 (0.4)
Severe dysfunction	0
Wall motion abnormalities	6 (2.6)

Table 4
Pre-transplant ischemic workup.
Pre-transplant ischemic work-up and result broken down by type and modality (i.e. exercise, vasodilator, dobutamine). Note, columns do not add up to 235 as some patients had unknown pre-transplant ischemic work-up and many with abnormal initial work-up also had left heart catheterization.

Workup	Total (n = 235 patients)	Normal	Abnormal
Stress echo	121 (51.5)	116 (49.4)	5 (2.1)
Exercise	30 (12.8)	30 (12.8)	0
Vasodilator	2 (0.9)	2 (0.9)	0
Dobutamine	89 (37.9)	84 (35.7)	5 (2.1)
Myocardial perfusion imaging	98 (41.7)	82 (34.9)	16 (6.8)
Exercise	27 (11.5)	22 (9.4)	5 (2.1)
Vasodilator	60 (25.5)	52 (22.1)	8 (3.4)
Dobutamine	2 (0.9)	1 (0.4)	1 (0.4)
Unknown stress modality	9 (3.8)	7 (3.0)	2 (0.9)
Exercise ECG	2 (0.9)	2 (0.9)	0
Coronary CT	2 (0.9)	1 (0.4)	1 (0.4)
Left heart catheterization	24 (10.2)	10 (4.3)	14 (6.0)

4. Discussion

Our study shows that cardiac testing remains a ubiquitous practice in pre-transplant evaluation, with an ongoing low rate of adverse cardiac outcomes for those patients who are ultimately deemed suitable for kidney transplant. At our center, 90.2% of kidney transplant recipients underwent pre-transplant cardiac evaluation, including at least a resting TTE. A total of 93.2% had stress images, >90% of which were normal. LHC was relatively rare given the high rate of normal studies. We report low all-cause mortality, cardiac-specific mortality, and MI in the post-transplant period. We also demonstrated that among patients in whom cardiac enzymes were checked in the post-transplantation period, low-level troponin elevation without clinical evidence of ischemia is common. The overall importance and impact of this elevation are unknown. Overall survival at 1 year was 99%. Of the 3 patients who died, only 1 had an abnormal pre-transplant cardiac evaluation. This occurred despite pre-transplant revascularization, including CABG and subsequent PCI.

The American College of Cardiology/American Heart Association guidelines recommend consideration of pre-transplant cardiac testing in patients with multiple risk factors common to both CAD and CKD, including hypertension, diabetes, and dyslipidemia, but stop short of

Table 5
Pre-transplant coronary evaluation and revascularization.
Pre-transplant revascularization broken down by extent of vessel disease and modality of revascularization.

	Diagnostic modality		Revascularization		
	Coronary CT	Left heart catheterization	CABG only	PCI only	CABG and PCI
Normal	1 (0.4)	10 (4.3)	0	0	0
1-vessel disease	0	3 (1.3)	0	1 (0.4)	0
2-vessel disease	1 (0.4)	5 (2.1)	1 (0.4)	3 (1.3)	0
3-vessel disease	0	6 (2.6)	4 (1.7)	0	1 (0.4)
Left main disease	0	0	0	0	0
Total	2 (0.9)	24 (10.2)	5 (2.1)	4 (1.7)	1 (0.4)

Table 6
Pre-transplant stress results in patients subsequently undergoing left heart catheterization.
Results of stress testing in patients with left heart catheterization.

Left heart catheterization	Stress result	Stress modality		Total
		Stress echo	MPI	
Normal	Normal	3	4	7
	Abnormal	1	1	2
Abnormal	Normal	1	2	3
	Abnormal	3	8	11
Total		8	15	23

Table 8
Post-transplant outcomes.
Reflects all outcomes within the study population during the follow-up period. Outcomes are separated by <30 days, >30 days to 1 year, and >1 year.

Outcome	Total	30 days	>30 days to 1 year	>1 year
Death (all-cause)	3 (1.3%)	0	2	1
Cardiac death	2 (0.9%)	0	2	0
Non-cardiac	1 (0.4%)	0	0	1
STEMI	0	0	0	0
NSTEMI type 1	1 (0.4%)	1	0	0
NSTEMI type 2	6 (2.6%)	5	0	1
Myocardial injury	27 (11.5%)	25	1	1

detailing specific recommendations for modality or revascularization [3]. This is appropriate and, in clinical practice, testing is determined on a case-by-case basis, weighing factors such as blood pressure, body habitus, and exercise tolerance. Previous studies of cardiac testing in potential transplant recipients, including DSE and MPI, have suggested that, overall, invasive and noninvasive cardiac testing predicts outcomes poorly, and a substantial number of patients have adverse cardiac events despite negative pre-transplant testing [5]. The utility of abnormal testing results is not clarified by our data, as very few patients had coronary intervention on the basis of their pre-transplant testing, and post-transplant coronary ischemia is rare regardless of pre-transplant testing results.

There are several limitations to this study. Our study is retrospective and limited to one institution. The small number and low incidence of outcomes in our study mitigated our ability to detect differences in risk of adverse outcomes on the basis of pre-transplant cardiac testing results. However, the low mortality and acute coronary syndrome (ACS) in our study likely reflect the transplant selection process, in which optimized medical risk factors, willingness to follow up, and adequate social support improve likelihood of transplantation. Although our follow-up period precluded evaluation of long-term post-transplant outcomes, we were able to accomplish our main focus of investigating perioperative outcomes. We were unable to capture patients on the transplant waitlist, those who were undergoing risk stratification, and those determined to have prohibitive risk. Without investigating all patients tested, it is difficult to make conclusions about the advisability of cardiac testing. However, a selection bias for healthy patients with well-managed comorbidities is not only expected, it is fundamental to the very nature of the transplant population. We believe our data are still valuable in showing that in transplanted patients, cardiac death and MI are rare regardless of abnormal ischemic testing or pre-transplant revascularization.

The clinical relevance of elevated cardiac enzymes in the post-operative setting after kidney transplant is an area that should be explored further. Previous studies have demonstrated that among patients undergoing noncardiac surgery, peak post-operative troponin level within the first 3 days after surgery was significantly associated with 30-day mortality [6]. In addition, elevated post-operative troponin without an ischemic feature was associated with 30-day mortality. It is important to note that in our study, post-operative cardiac enzyme testing was not standardized and was at the discretion of the primary provider. Still, we believe that our study adds to the literature by demonstrating low mortality despite the relative frequency of post-operative troponin elevation. In fact, our study likely underestimates the prevalence of troponin elevation following transplantation, which would theoretically result in higher-than-expected mortality if there is a strong relationship. Moreover, the lack of universal troponin testing,

Table 7
Incidence of pre-transplant ischemic workup and post-transplant outcomes.
Post-transplant outcomes separated by normal, abnormal or unknown transplant ischemic work-up. Outcomes included death (cardiac and non-cardiac), myocardial infarction (STEMI, Type I NSTEMI, and Type II NSTEMI) and myocardial injury using the 4th universal definition.

	Total	Death		Myocardial Infarction (30 days)			Myocardial injury (30 days)
		Cardiac	Non-cardiac	STEMI	Type 1 NSTEMI	Type 2 NSTEMI	
Normal pre-transplant ischemic workup	200 (85.1)	0	1 (0.4)	0	0	4 (1.7)	22 (9.4)
Abnormal pre-transplant ischemic workup	23 (9.8)	1 (0.4)	0	0	1 (0.4)	1 (0.4)	2 (0.9)
No or unknown pre-transplant ischemic workup	12 (5.1)	1 (0.4)	0	0	0	0	1 (0.4)
Any revascularization	10 (4.3)	0	0	0	0	0	0
CABG	5 (2.1)	0	0	0	0	0	0
PCI	4 (1.7)	0	0	0	0	0	0
CABG + PCI	1 (0.4)	1 (0.4)	0	0	1 (0.4)	0	0
Total	235[a]	2[a] (0.9%)	1 (0.4%)	0	1[a] (0.4%)	5 (2.1%)	25 (10.6%)

[a] This reflects the sum of the first 3 rows, which includes all patients, rather than all rows, because revascularization and workup are not mutually exclusive. The non-summative total reflects overlap between revascularization and pre-transplant workup.

Table 9
Characteristics of patients with adverse outcomes.
Pre-transplant baseline demographics, past medical history, medications and pre-operative risk for patients with adverse outcomes within 1 year post-transplant. RCRI reflects risk of major cardiac event and Gupta reflects risk of myocardial infarction or cardiac arrest, intraoperatively or up to 30 days post-operatively. RCRI = Revised Cardiac Risk Index.

Patient	Outcome	Demographics			History				Pre-operative risk scores		Pre-operative cardiac workup		
		Age	Gender	Race	PMH	Years on dialysis	Type of dialysis	Medications	RCRI[a]	Gupta[b]	Type	Findings	Intervention
Patient 1	Cardiac Death, 1 Year	69	Male	African American	Diabetes Mellitus, Hypertension, Hyperlipidemia, CVA	5	HD	Aspirin, Statin, Insulin, Beta Blocker	Class IV (11%)	1.7%	None found	N/A	None
Patient 2	Cardiac Death, 1 Year NSTEMI Type I, 30 Days	62	Male	African American	Hypertension, Hyperlipidemia, Prior MI, CABG (LIMA to LAD, SVG to LCx)	6	HD	Aspirin, Statin, Beta Blocker, ACE/ARB	Class IV (11%)	1.5%	Exercise MPI	Small, moderate apical defect	DES to Diagonal Branch Occlusion
Patient 3	NSTEMI Type II, 30 Days	50	Male	African American	Diabetes Mellitus, Hypertension, Stable CAD	7	PD	Aspirin, Statin, Insulin, Beta Blocker, ACE/ARB	Class IV (11%)	1.2%	Exercise MPI	Medium sized, severe inferior defect	None
Patient 4	NSTEMI Type II, 30 Days	51	Male	Other	Hypertension	1	HD	Aspirin, Statin, Beta Blocker	Class II (6.6%)	1.2%	Dobutamine Echo	Normal	None
Patient 5	NSTEMI Type II, 30 Days	75	Female	African American	Hypertension, Prior Tobacco Use	4	HD	Statin, Beta Blocker, ACE/ARB, CCB, Aldo antagonist	Class II (6.6%)	1.9%	Exercise MPI	Normal	None
Patient 6	NSTEMI Type II, 30 Days	64	Male	African American	Diabetes Mellitus, Hypertension, Prior Tobacco Use	5	HD	Beta Blocker, ACE/ARB	Class II (6.6%)	2.9%	Vasodilator MPI	Normal	None
Patient 7	NSTEMI Type II, 30 Days	57	Male	African American	Hypertension, Prior MI, CVA, Current Tobacco Use	1	HD	Aspirin, Statin, Beta Blocker, ACE/ARB, Diuretic	Class III (11%)	1.3%	Exercise Echo	Normal	None
Patient 9	NSTEMI Type II, 1 year	52	Male	African American	Hypertension	7	HD	Beta Blocker, ACE/ARB, CCB	Class II (6.6%)	1.2%	Vasodilator MPI	Normal	None

[a] RCRI = Revised Cardiac Risk Index. Risk of major cardiac event.
[b] Risk of myocardial infarction or cardiac arrest, intraoperatively or up to 30 days post-operatively.

Table 10
Post-transplant cardiac enzyme testing.
Cardiac enzyme levels and timing for patients who had cardiac enzymes drawn post-transplant.

	N = 235 (percentage or SD)
Troponin within 30 days of transplant	47 (20%)
Peak troponin <0.015 ng/mL	16 (34%)
Detectable peak troponin	31 (66%)
Mean (ng/mL) ± SD	3.55 ± 18.6
Range (ng/mL)	0.016–104.0
Time to troponin (days ± SD)	5.3 ± 6.08
Range (days)	0–23
CK-MB within 30 days of transplant	15 (6%)
Peak CK-MB undetectable	3 (7%)
Detectable peak CK-MB	12 (26%)
Mean (ng/mL) ± SD	3.58 ± 3.55
Range (ng/mL)	0.7–13
Time to CK-MB (days ± SD)	3.6 ± 4.45
Range (days)	0–14

while mitigating the accuracy of defining the prevalence of myocardial injury, reflects current clinical practice, and we believe our results remain valid. The universal measurement of pre- and post-transplant troponins may be beneficial and deserves future research.

In conclusion, at our center, non-invasive cardiac testing is common before kidney transplant. The most common modalities are DSE and pharmacological MPI. Pre-transplant LHC and subsequent revascularization are rare. For patients who undergo transplantation, perioperative death and ACS are rare. However, myocardial injury is relatively common among patients in whom troponins are checked. There is no clear pattern of pre-cardiac testing results or revascularization in patients who develop post-transplant major adverse cardiac outcomes. Further research is needed to define the utility and best implementation of pre-transplant cardiac testing, with the consideration of a more standardized practice of monitoring troponin levels pre- and post-kidney transplant.

Declarations of interest

Ron Waksman – Advisory Board: Abbott Vascular, Amgen, Boston Scientific, Medtronic, Philips Volcano, Pi-Cardia Ltd., Cardioset; Consultant: Abbott Vascular, Amgen, Biosensors, Biotronik, Boston Scientific, Medtronic, Philips Volcano, Pi-Cardia Ltd., Cardioset; Grant Support: Abbott Vascular, AstraZeneca, Biosensors, Biotronik, Boston Scientific, Chiesi; Speakers Bureau: AstraZeneca, Chiesi; Investor: MedAlliance.

Toby Rogers – Consultant: Medtronic; Proctor: Edwards Lifesciences.

All other authors – None.

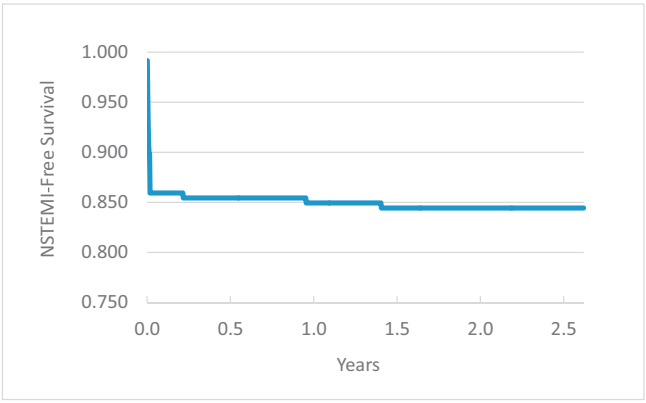

Fig. 3. Kaplan-Meier myocardial injury-free survival. Post-transplant myocardial injury-free survival during our follow-up period. Myocardial injury-free survival was defined as time until first occurrence of STEMI, Type I NSTEMI, Type II NSTEMI, and myocardial injury. We found a total of 34 patients with elevated troponin following transplantation. One was determined to be due to ACS (Type I NSTEMI), while the remaining 33 were determined to be due to Type II NSTEMI or myocardial injury.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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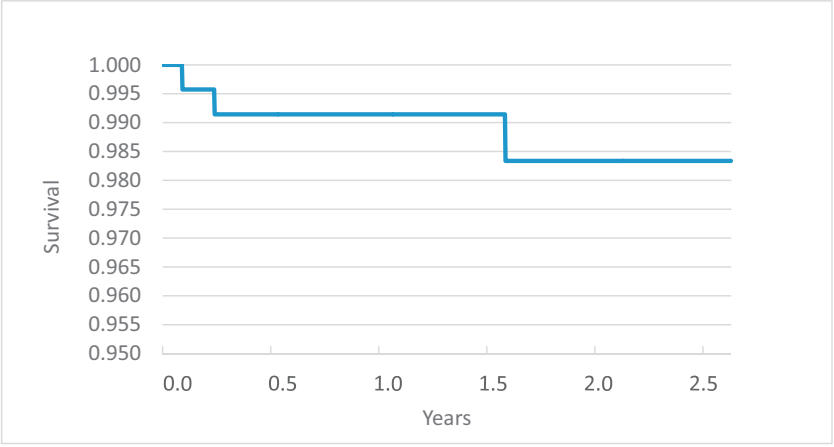

Fig. 2. Kaplan-Meier survival curve. Post-transplant overall survival from death of any cause during our follow-up period. A total of 3 patients died during our follow-up period with two falling within 1 year of transplant.