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Metabolic Syndrome and Coronary Artery Calcification in Renal Transplant Recipients

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Abstract

Background—Coronary artery calcification (CAC) and Metabolic Syndrome (MS) have been associated with increased cardiovascular risk. The study objective was to examine the association of MS with CAC presence and progression in renal transplant recipients.

Methods—We measured CAC progression in asymptomatic recipients who had no prior history of coronary artery disease.

Results—MS was common (55.4%). Median CAC scores were 0, 33.1, 98, and 261.9 for patients with 1, 2, 3, and 4 or more positive components of the MS, respectively. Severe CAC scores were more common in recipients with MS ($p=0.04$). Although recipients with MS had higher mean CAC scores at baseline and significant CAC progression [483 (590.6) vs. 619(813.8), $p=0.01$], MS was not an independent predictor of annualized rate of CAC change in a multivariate model

Conclusion—Future studies to evaluate if MS treatment improves cardiovascular outcomes are imperative.

Keywords

renal transplant; coronary calcification; metabolic syndrome

Introduction

Renal transplant recipients are at disproportionately high risk of mortality from cardiovascular disease (CVD) (1-3). A portion of this excess mortality can be attributed to higher prevalence of well-established CVD risk factors such as hypertension, diabetes, hyperlipidemia, and smoking (1),(4). Germane to the renal transplant population, immune-suppression and graft dysfunction confer additional CVD risk but do not completely explain the disparity (1),(2), (5,6).

The metabolic syndrome (MS), a harbinger of cardiovascular disease in the general population is associated with graft dysfunction and decreased survival among renal transplant recipients (7-13). Individually, several of the component risk factors of the MS have been associated with acute coronary syndromes post transplant (14,15). The prevalence of MS and the associated

cardiovascular risk appears to increase posttransplant secondary to the high incidence of obesity and insulin resistance(16).

The MS has been linked to coronary artery calcification (CAC), a correlate of atherosclerotic burden in the general population (17-23). CAC is prevalent in renal transplant patients (24, 25). To date, CAC and the MS have not been evaluated simultaneously in renal transplant recipients. The study objective was to examine the association of MS with the CAC presence and progression in renal transplant recipients. This is a secondary analysis of a prospective cohort recruited to evaluate the natural history of CAC (24,26).

Materials and Methods

One hundred and twelve incident renal transplant recipients were recruited from an adult program. Exclusion criteria included a history of coronary artery revascularization or myocardial infarction. Recipients of multiple organs, except pancreas were excluded. Cardiovascular disease was defined as history of cerebrovascular disease or peripheral vascular disease.

Recipients had a complete lipid profile (e.g. total cholesterol, triglycerides, HDL, LDL, apoA-1, and apoB), and C-Reactive Peptide measured prior to transplantation surgery. Laboratory testing was conducted using commercially available reagents, as previously described (24). We calibrated the measurement of serum creatinine by the PENN clinical laboratory against that of the MDRD core laboratory and used the MDRD equation to derive the estimated glomerular filtration rate (eGFR)(27). Patients underwent cardiac risk stratification as clinically indicated by their transplant nephrologists.

CAC was quantified using electron beam computed tomography (EBCT) in all study participants within 6 months of transplantation. Eighty-seven subjects had repeat EBCT performed. EBCT was not obtained in the remaining subjects due to: patient refusal or lost to follow-up (n=15), weight > 300 lbs (n=2), death (n=2), coronary artery disease requiring coronary artery bypass grafting post-transplant (n=1), and graft failure prior to second EBCT (n=5). Scans were performed on a C-150 Imatron scanner (GE, San Francisco, CA) using a standard protocol, as previously described (24).

Adapting the National Cholesterol Education Program Adult Treatment Panel III guidelines, MS was defined as the presence of 3 or more of the following five criteria: (1) obesity with BMI ≥ 30 kg/m²; (2) triglycerides ≥ 150 mg/dl or on treatment; (3) HDL <40 mg/dL in men and <50 mg/dL in women; (4) systolic blood pressure ≥ 130 mmHg, and/or diastolic blood pressure ≥ 85 mmHg, and/or on anti-hypertensive therapy; and (5) fasting glucose ≥ 100 mg/dL or on treatment. As waist circumference was not available for all patients, BMI was used as a surrogate, which has been shown to correlate in prior studies (10). We defined metabolic syndrome at 2 timepoints at the time of transplant and 3 months pos-transplant.

Our standard immunosuppressive regimen comprised induction with rabbit antithymocyte globulin, followed by maintenance therapy with calcineurin inhibitor (tacrolimus or cyclosporin), mycophenolate mofetil and low dose prednisone (5 mg PO at 3 months pos-transplant).

For the statistical analysis, continuous variables were summarized using means (standard deviations) and medians. Percentages were used to summarize categorical variables. Log transformation was used for variables with a skewed distribution. We used ordinal logistic regression and/or test for trends to evaluate variables with ordinal value such as positive components of the MS or categories of CAC score. We estimated the annualized rate of change using the following formula: [(second CAC score-baseline score)/interval between scans]. We

performed a multiple regression adjusting for known predictors of CAC progression in renal transplant recipients to determine independent association of MS and any of its components with progression of CAC. Statistical analyses were performed with STATA, version 10 (College Station, TX). Informed consent was obtained from all participants and the Institutional Review Board approved the study protocol.

Results

The average age of study participants was 49(standard deviation, 12.5) years. The majority of subjects were male (62%) Caucasian (62.5%), and deceased donor transplant recipients (60.7%). Thirty-four percent of subjects were African-American. A third had a history of diabetes (34%) and few had a history of cardiovascular disease (9%). Preemptive transplant was seen in 25% of recipients. The average dialysis vintage for the remaining subjects was 3 (2.7) years.

It was common for recipients to be on calcium containing phosphorus binders (71%) or noncalcium containing binders (23%) at the time of transplant. Almost a quarter (24%) listed Vitamin D therapy and a bisphosphonate was used in 19% of subjects.

MS was common (55.4%) at time of transplant. Sixty-six percent of patients had CAC detected by EBCT. Patients with MS had higher median CAC scores compared to subjects without MS [111.7 vs. 20.4, $p=0.04$]. The median CAC score increased with the number of positive components of the MS. Median CAC scores were 0, 33.1, 98, and 261.9 for patients with 1, 2, 3, and 4 or more positive components of the MS, respectively. Severe CAC scores were more frequent in subjects with MS compared to subjects without MS ($p=0.04$, Table 1). While 37.7 % of subjects with MS had severe CAC, only 19.6% of subjects without MS had severe CAC.

The prevalence of metabolic syndrome 3 months post-transplant decreased to 38.3% but by 6 months it had increased to 49.4%. These changes were largely secondary to improvements in the immediate post-transplant period of HDL and triglycerides. HDL improved significantly post-transplant from 45.1(17.2) at baseline to 59.8(20.6) at 3 months post-transplant. Triglycerides also had a favorable shift from 183.4(163.2) to 166.8(99.4). There were improvements in systolic blood pressure (145.5(21.7) vs. 135.3(22.7) and diastolic blood pressure (82.8(12.9) vs. 74(11.5)). There were no significant changes in average BMI and glucose. Glucose levels were higher post-transplant in non-diabetics compared to glucose levels at time of transplant [at baseline 85.1 (19.6), at 3 months. 91.3(20.5) and at 6 months 98.1(68.5)].

In univariate analysis, the presence of MS at time of transplant was not associated with gender, race, calcium, phosphorus or dialysis vintage (Table 2). Patients with MS were more likely to have increased CRP ($p=0.01$) but had lower apo A1 levels as expected since it is the main protein content of HDL ($p < .001$). Hematocrit was lower in the MS group, which achieved borderline statistical significance [35.7% vs 37.5%, $p=0.06$].

We obtained 87 repeat EBCTs in this cohort at least one year later. Due to missing data, we were able to determine the metabolic syndrome status of 84 participants. There were 31 subjects that did not have metabolic syndrome at time of transplant or at 3 months. These patients did not have progression of the mean CAC score [384.2(877.1) vs. 439.4(963.6), $p=0.23$] or the median CAC score [20.4 vs. 30.4, $p=0.34$]. However, subjects that had metabolic syndrome at both time points ($n=22$) had higher baseline CAC scores and significant progression of their mean CAC score [483 (590.6) vs. 619(813.8), $p=0.01$ and their median CAC score [231.3 vs. 322, $p=0.05$]. Subjects with metabolic syndrome at 1 of these 2 time points had a higher median CAC score at baseline and there was CAC progression [72.9 vs. 93.1, $p=0.04$].

We have previously described Caucasian race, eGFR at 3 months, baseline CAC score, BMI and blood pressure as independent risk factors for annualized rate of CAC progression in renal recipients(28). Controlling for race, eGFR at 3 months, baseline CAC score,, presence of MS at baseline or at 3 months post-transplant does not emerge as an independent risk factor from progression (Table 3).If one examines the individual components of the MS, only BMI and triglycerides components had a significant association with annualized rate of CAC progression at baseline, but not at 3 months post-transplant.

Discussion

Our study is the first to examine the relationship between the MS and CAC in renal recipients in a diverse population. CAC was common (66%) as confirmed by others (24,25). The median CAC score was significantly higher among recipients with the MS. The median CAC score increased with increasing number of positive components of the MS. Although, recipients with MS had higher mean and median CAC scores at baseline and significant progression, MS was not an independent predictor of annualized rate of CAC change after adjustment for baseline CAC score, race and eGFR at 3 months.

Progressive increase in prevalence of CAC with more positive components of the MS has been found in non-CKD population based cohorts(21) (23). As the MS is thought to promote atherosclerosis, it is not surprising that more risk factors of the MS correlate with greater burden of atherosclerosis as measured by CAC at the time of renal transplant. Further, the presence of metabolic syndrome is a predictor of cardiovascular events among renal transplant recipients. Compared to renal transplant recipients without the metabolic syndrome, patients with the metabolic syndrome at 1 year posttransplant were 3.4 fold more likely to have an atherosclerotic event (29). Faenza and colleagues studied the relationship between the metabolic syndrome 1 year posttransplant and cardiovascular morbidity and mortality. The authors found that a positive association between the number of positive components of the metabolic syndrome and the incidence of cardiovascular events over a 4 year follow up period(30).

The role of CAC is still being evaluated in the general population and in patients with end stage renal disease (19,31). It is generally accepted that no calcification yields a low risk of CAD and higher scores are associated with increased risk (31,32). Wong et al found in the general population that presence of MS and an intermediate or higher CAC scores was associated with increased risk of inducible myocardial ischemia (33). Studies by Matzouka (34) and Blacher (35) demonstrate that vascular calcification increase risk of cardiovascular events in dialysis patients. Treatment to lower CAC may reduce cardiovascular events in dialysis patients(36).

The prevalence of MS was higher than the 23% observed by Porrini et al in Spanish recipients 1 year after transplantation(10). However, it is similar to a study by deVries and colleagues where 63% of renal recipients had the MS at 6 years of follow up (11). MS has been associated with chronic graft dysfunction, a risk factors for atherosclerotic events among renal transplant recipients (10-12,15). We believe that levels of HDL increased post-transplant secondary to the use of prednisone post-transplant. HDL cholesterol levels decreases by 14.0% in women and 22.0% in men after prednisone is discontinued(37).

Ducoux and colleagues found that the Framingham Heart Study model underestimated cardiovascular risk in renal recipients. In the same study, non-traditional risk factors such as hyperhomocysteinemia and elevated CRP were associated with ischemic heart disease (5). In our study, we found an association between CRP and the presence of MS.

There are important limitations to this study. This is a relatively small cohort and the analysis should be viewed as exploratory and in need of larger confirmatory studies. Only one laboratory measurement was performed at each timepoint. Repeat CT scans were obtained in 78% of the

study population which may have introduced a survivorship bias. The study population is representative of the renal recipients from our geographic area, but may not be representative of other areas with different ethnic composition. Future studies to evaluate if treatment of MS improves cardiovascular outcomes in renal transplant recipients are imperative.

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Table 1

Relationship between degree of coronary calcification and presence of metabolic syndrome at time of transplant

Coronary Calcification Score	No Metabolic Syndrome(%) N=51	Metabolic Syndrome(%) N=61
<10 (n=42)	45.1	31.1
10-<100 (n=19)	17.6	16.4
100-<400 (n=18)	17.6	18.8
400-<1000 (n=21)	11.8	24.6
>1000 (n=12)	7.8	13.1

Table 2

Distribution of demographic and laboratory parameters by presence of metabolic syndrome at time of transplant

	No Metabolic Syndrome N=51	Metabolic Syndrome N=61	P-value
Age (years) mean (SD) [§]	46.7(12.8)	50.9 (12.01)	0.07
Dialysis vintage (years)	2.7(2.7)	3.3 (2.7)	0.39
BMI (kg/m ²)	25.3 (3.6)	28.8 (6.7)	0.001
Female (%)	41.2	36.1	0.58
Caucasian (%)	62.7	62.3	0.96
Diabetes (%)	25.5	42.6	0.06
Never on Dialysis (%)	23.5	24.6	0.90
Living donor (%)	45.1	34.4	0.25
Never Smoked (%)	52	57.4	0.57
History of cardiovascular disease (%)	3.9	13.1	0.09
Total cholesterol (mg/dl)	174.2 (40.2)	174.9 (50.8)	0.93
Triglycerides (mg/dl)	145.9 (158.7)	213.5 (160.7)	0.03
HDL (mg/dl)	52.4 (17.5)	39.1 (14.2)	<0.001
LDL (mg/dl)	93 (33.9)	93.9 (36.6)	0.90
Apo A1 (mg/dl)	133.2 (25)	116.2 (25.8)	<0.001
Apo B (mg/dl)	79.4 (22.1)	81.7 (21.5)	0.59
Lipoprotein (a) median (mg/dl)	29.5	23.1	
Glucose (mg/dl)	89.6 (39.5)	127.3 (100.4)	0.01
CRP [*]	2.7 (2.4)	5.2 (7.4)	0.01 (log)
S-ICAM	281.1(98.9)	278.4 (70.5)	0.87
WBC	6.4 (2.1)	7.2 (2.2)	0.06
Calcium (mg/dl)	9.1 (1.2)	9.3 (1.0)	0.31
Phosphate (mg/dl)	5.1 (1.27)	5.0 (1.4)	0.84
Hematocrit (%)	37.5 (5.1)	35.7 (4.8)	0.06
SBP (mmHg)	143.75 (22.5)	147.6 (20.9)	0.35
DBP(mmHg)	84.3 (13.4)	81.9 (12.6)	0.34
eGFR at 3 months post-transplant (ml/min)	62.0 (16.6)	59.8 (17.7)	0.51
Presence of CAC score >400 (%)	19.6	37.7	0.04
CAC score (median)	20.4	111.7	0.04
CAC score (mean) [*]	301.6 (706.3)	423 (662.4)	0.10 (log)

* reflects log transformation

[§]Expressed as a mean with standard deviation in parentheses

Table 3

Association of Annualized Rate Of CAC Score Change with Metabolic Syndrome and its Components

	Coefficient*	p-value
Presence of Metabolic Syndrome	33.1 (29.7)	0.28
Presence of Metabolic Syndrome at 3 months posttransplant	44.1(31.1)	0.16
Component		
<i>Blood Pressure</i>	15.1 (63.1)	0.81
<i>HDL</i>	-4.48 (31.3)	0.89
<i>Triglycerides</i>	57.3 (30.1)	0.06
<i>Glucose</i>	11.7 (29.7)	0.70
<i>BMI</i>	80.6 (38.2)	0.04
Component at 3months posttransplant		
<i>Blood Pressure</i>	32.6(44.9)	0.47
<i>HDL</i>	-8.8(52.5)	0.87
<i>Triglycerides</i>	13.1(35.5)	0.71
<i>Glucose</i>	11.6(34.5)	0.74
<i>BMI</i>	61.8(40.3)	0.13

* adjusted for race, baseline CAC score, eGFR at 3 months