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Risk of Acute Kidney Injury, Dialysis, and Mortality in Chronic Kidney Disease Patients following Intravenous Contrast Material Exposure

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Abstract

Objective—To examine the effect of intravenous iodinated contrast material administration on the subsequent development of acute kidney injury (AKI), emergent dialysis, and short-term mortality using a propensity score-adjusted analysis of Computed Tomography (CT) scan recipients with chronic kidney disease (CKD).

Patients and Methods—In this IRB approved retrospective study, all CKD patients who received a contrast-enhanced (contrast group) or unenhanced (noncontrast group) CT scan from January 2000 to August 2013 were identified. Patients were subdivided into CKD Stage III (baseline eGFR 30–59 ml/min/1.73m^2), and CKD Stage IV–V (baseline eGFR<30 ml/min/1.73m^2), subgroups and separately underwent propensity score generation, stratification, and 1:1 matching. Rates of AKI and 30-day emergent dialysis and mortality were compared between contrast and noncontrast groups. Sensitivity analyses examining only patients with stable prescan serum creatinine (SCr) and incorporating IV fluid administration at the time of scan into the model were also performed.

Results—A total of 6902 patients (4496 CKD Stage III, matched: 1220 contrast/1220 noncontrast; 2086 CKD Stage IV–V, matched: 491 contrast/491 noncontrast) were included in the

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study. Following propensity score adjustment, the rates of AKI, emergent dialysis, and mortality were not significantly higher in the contrast group compared to the noncontrast group in either CKD subset (CKD Stage III OR 0.65–1.00, *P*<.001–.99, CKD Stage IV–V OR 0.93–2.33, *P*=.22–.99). Both sensitivity analyses had similar results.

Conclusion—Intravenous contrast material administration was not associated with an increased risk of AKI, emergent dialysis, and short-term mortality in a cohort of patients with diminished renal function.

INTRODUCTION

Concern for the development of acute kidney injury (AKI) following administration of iodinated contrast material, also known as contrast-induced nephropathy (CIN), often limits the use of contrast material in patients at risk of developing this complication [1,2]. However, recent research suggests that the incidence and severity of CIN have been overestimated by prior uncontrolled studies [3–5]. In these prior studies, all instances of AKI following contrast administration were routinely ascribed to CIN, even though there are myriad causes of AKI among hospitalized patients. Controlled studies with clinically similar patients who did not receive contrast material are essential to help differentiate true CIN from contrast-independent AKI.

Two recent large retrospective studies by McDonald et al. and Davenport et al. used propensity score matching to compare contrast-enhanced Computed Tomography (CT) scan recipients and clinically similar patients who underwent an unenhanced CT scan [6,7]. Both studies found that the rate of AKI was similar between contrast recipients and control groups among patients with baseline eGFR > 30 ml/min/1.73m^2, providing evidence that CIN may not be a clinical concern in these patients. However, disparate results were reported for patients with baseline eGFR < 30 ml/min/1.73m^2, with the McDonald et al. study reporting similar rates of AKI between the two groups and the Davenport et al. study reporting significantly higher rates of AKI in contrast recipients suggestive of CIN. Several potential explanations for these dissimilar results have been postulated, including differences in clinical covariates included in the studies' propensity score models, differences in the clinical and demographic composition of the patient populations, and whether the study included or excluded patients with unstable serum creatinine prior to their CT scan [8,9].

The purpose of the current study was to perform a more rigorous propensity score analysis of CT scan recipients with renal insufficiency (eGFR < 60 ml/min/1.73m^2) and better determine the risk of AKI, emergent dialysis, and mortality following exposure to intravenous contrast material.

MATERIALS and METHODS

Study Design and Clinical Data Retrieval

Design and execution of this single-center retrospective study was subject to Institutional Review Board oversight and HIPAA privacy guidelines. The need for informed consent was waived. All clinical data were extracted from our electronic medical record (EMR) using a

combination of relational database software (DDQB, IBM, Armonk, New York) and manual chart review. Additional details of data retrieval and analysis are provided in the eAppendix.

Study Population

Many patients in the current study were included in previous publications that examined the incidence of AKI, emergent dialysis, and mortality in patients who received a contrast-enhanced or unenhanced CT scan [7,10,11]. We wanted to improve upon these prior studies by 1) including a more comprehensive list of clinical variables related to renal insufficiency in the propensity score model to reduce confounding and better match contrast recipients and control patients, 2) performing a full chart review of the patient's record to confirm comorbidities and medical conditions instead of relying on ICD-9 diagnostic codes, which have been shown to be inaccurate in some cases [12–14], and 3) including CT scans performed through July, 2013 to better reflect current clinical practices.

Adult patients (18 years or older) were included in the current study if they 1) received an unenhanced (noncontrast group) or IV contrast-enhanced (contrast group) abdominal, pelvic, and thoracic CT scan from January 2000 to August 2013 at our institution; 2) had at least two pre-scan (within 24 hrs prior) SCr results and at least one post-scan (24–72 hrs post) SCr result; and 3) had a baseline eGFR < 60 ml/min/1.73m^2 at the time of CT scan as calculated below. Patients were excluded if they 1) had pre-existing renal dialysis requirements; 2) did not have the pre- and post-scan SCr results as described above; 3) were missing any clinical variables included in the propensity score model (listed in Table 1); or 4) received IV or intra-arterial contrast material from another exam or procedure within a 14-day period of the CT scan. When a patient received multiple CT scans over the study timeframe, only the last CT scan was included in the analysis to eliminate sampling bias and maximize the probability of identification of disease. Detailed information regarding inclusion and exclusion criteria is in the eAppendix.

Baseline Renal Function

All SCr data associated with each CT scan record were extracted from the EMR and temporally sorted with respect to the date of the scan. Baseline eGFR was calculated for each patient from the SCr result(s) 24 hours prior to CT scan using the MDRD (Modification of Diet in Renal Disease) equation based upon the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (KDOQI) recommendations as previously described [7]. Patients were stratified by baseline eGFR into 30–59 ml/min/1.73m^2 ("CKD Stage III") and <30 ml/min/1.73m^2 ("CKD Stage IV–V") subgroups to mirror the KDOQI classification of chronic kidney disease [15].

Outcome Variables

The outcomes examined in this study were acute kidney injury (AKI), emergent dialysis, and death following CT scan. AKI was defined as a rise in maximal observed SCr of either 1) ≥ 0.5 mg/dL ("standard AKI criteria") or 2) ≥ 0.3 mg/dL or ≥ 50% over baseline ("Acute Kidney Injury Network (AKIN) criteria") in the 24–72 hours following the time of CT scan. The former cutoff was chosen to maintain consistency with prior studies that used this definition of AKI, while the latter was chosen to reflect the more recent recommendations of

the AKIN [16]. Cases of emergent dialysis, defined as dialysis performed in a patient who did not previously require dialysis, and death within 30 days of CT scan were identified as previously described [11].

Propensity Score Analysis

Propensity score generation, stratification, and matching for patients in the contrast and noncontrast groups were performed using the R package MatchIt as previously described [10]. Logistic regression models derived from the 32 clinical variables numbered in Table 1 were separately created for the CKD Stage III and IV–V subgroups. Relative influence of propensity score model covariates was determined using the R package Twang [17].

Sensitivity Analyses of Pre-Scan SCr Stability and IV Fluid Administration

Two sensitivity analyses were performed to strengthen the confidence of our findings. In the first analysis, only patients with stable baseline renal function, defined as changes in pre-scan SCr < 0.5 mg/dL (eAppendix), were subjected to stratification and matching by propensity score as described above. This subgroup was created in order to remove patients with wide variability in renal function and/or undetected acute kidney injury prior to contrast material exposure that could potentially confound the results. In the second analysis, the amount of IV fluids administered to patients in the 24 hrs prior to their CT scan was included as a covariate in the propensity score model. Only CT scans performed after 12/2003 at our institution had IV fluid data entered into the EMR and therefore were included in this analysis. The amount of IV fluids administered on the day of and 24 hrs following CT scan were not included as covariates since they took place after the decision to administer contrast material and could potentially confound the result. These two post-hoc covariates were instead added as adjustment covariables to a conditional logistic regression model following matching with pre-scan IV fluids and other Table 1 covariates.

Statistical Analysis

Statistical analyses were performed using R (version 3.0.3, R Foundation for Statistical Computing, Vienna, Austria) [18]. Dichotomous variables were displayed as counts with percentages, categorical data were displayed as relative frequencies (%), and continuous data were presented as medians with interquartile ranges (IQR). Differences in clinical characteristics and rates of AKI, emergent dialysis, and mortality between the contrast and noncontrast groups prior to matching were assessed using the Wilcoxon rank-sum test for continuous clinical characteristics and Fisher's Exact test or Pearson's chi-squared test for categorical clinical characteristics and outcomes. The collective risk of AKI, emergent dialysis, and mortality following stratification by propensity score was assessed using Cochran-Mantel-Haenszel estimates. Differences in clinical characteristics and rates of AKI, emergent dialysis, and mortality following 1:1 matching were measured using conditional logistic regression, conditioned on the unique ID assigned to each match.

RESULTS

Study Population and Propensity Score Adjustment

A total of 6902 CT scan records (4496 Chronic kidney disease (CKD) Stage III, 2086 CKD Stage IV–V) met all study inclusion criteria (eTables 3 and 4). Before propensity score adjustment, patients in the contrast and noncontrast groups had significant differences in numerous clinical variables, including baseline renal function, acute and chronic comorbidities, and medication use.

Propensity score distributions for both CKD subgroups are shown in eFigure 1. The relative influence of all covariates on the propensity score model of each CKD subgroup is shown in eFigure 2. The five most influential covariates for the CKD Stage III subgroup were baseline eGFR, pre-existing hypertension, age, admit type, and gender. The five most influential covariates for the CKD Stage IV–V were baseline eGFR, pre-existing chronic kidney disease, pre-scan SCr stability, year of scan, and age.

Among all study patients, stratification of propensity score into deciles eliminated all significant differences for all covariates between the contrast and noncontrast groups in both CKD subgroups (eTables 3 and 4). One-to-one matching based on the propensity score yielded a smaller, more rigorously matched cohort of 2440 CT scan recipients for the CKD Stage III subgroup (1220 contrast/1220 noncontrast) and a cohort of 982 matched CT scan recipients for the CKD Stage IV–V subgroup (491 contrast/491 noncontrast) (Tables 1 and 2). This matching also eliminated all significant differences of all covariates between the contrast and noncontrast groups in both CKD subgroups.

Propensity Score Adjusted Outcome Rates

Patient outcomes following stratification and matching by propensity score are shown in Tables 3 and 4. Following stratification, the rate of AKI was not significantly higher in the contrast group compared to the noncontrast group in either CKD subgroup (CKD Stage III: AKIN criteria P=<.001, standard AKI criteria P=.29; RI Stage IV–V: AKIN criteria P=.99, standard AKI criteria P=.90). A similar pattern was observed following propensity score matching (CKD Stage III: AKIN criteria P<.001, standard AKI criteria P=.38; CKD Stage IV–V: AKIN criteria P=.47, standard AKI criteria P=.92). Use of emergent dialysis was rare and not significantly different between the contrast and noncontrast groups in either CKD subgroup following stratification (CKD Stage III: P=.62; CKD Stage IV–V: P=.31) or matching (CKD Stage III: P=.99; CKD Stage IV–V: P=.22). The rate of mortality was also not significantly different between the contrast and noncontrast groups in either CKD subgroup following stratification (CKD Stage III: P=.25; CKD Stage IV–V: P=.89) or matching (CKD Stage III: P=.06; CKD Stage IV–V: P=.71).

Sensitivity Analysis: Adjusted Outcome Rates in Patients with Stable Pre-scan SCr

Propensity score matching after excluding any patients who had widely fluctuating SCr results (delta ≥ 0.5 mg/dL) prior to their CT scan yielded a cohort of 2146 matched CT scan recipients for the CKD Stage III subgroup (1073 contrast/1073 noncontrast) and a cohort of 496 matched CT scan recipients for the CKD Stage IV–V subgroup (248 contrast/248

noncontrast) (eTables 5 and 6). Following matching, there were no significant differences between the contrast and noncontrast groups in any covariates in both CKD subgroups. The rates of AKI, emergent dialysis, and mortality were again not significantly higher in the contrast group compared to the noncontrast group in either CKD subgroup, regardless of AKI criteria or whether patients were stratified or matched by propensity score (eTables 7 and 8).

Sensitivity Analysis: Adjusted Outcome Rates Including IV Fluid Administration in the Propensity Score Model

Incorporation of IV fluids administered in the 24 hrs prior to CT scan in the propensity score model yielded a cohort of 1734 matched CT scan recipients for the CKD Stage III subgroup (867 contrast recipients/867 noncontrast recipients) and a cohort of 572 matched CT scan recipients for the CKD Stage IV–V subgroup (286 contrast recipients /286 noncontrast recipients) (eTables 9 and 10). Following matching, there were no significant differences in clinical covariates between the contrast and noncontrast groups in either CKD subgroup. The rates of AKI, emergent dialysis, and mortality were again not significantly higher in the contrast group compared to the noncontrast group in either CKD subgroup, after incorporating pre-scan IV fluid administration, regardless of AKI criteria or whether patients were stratified or matched by propensity score (eTables 11 and 12).

The administration of IV fluids on the day of CT scan or the day after scan was not included in the propensity score model, as only covariates that are present at the time of treatment can be included. In the matched CKD Stage III subgroup, contrast recipients received significantly more fluids on the day of scan compared to patients in the noncontrast group (P=.04, eTable 9). In the matched CKD Stage IV–V subgroup, contrast recipients and patients in the noncontrast group had similar likelihoods of receiving fluids on the day of scan and day after scan and received similar amounts of fluids (eTable 10). Among patients with similar IV fluid administration, AKI, dialysis, and mortality rates were again not significantly higher in the contrast group compared to the noncontrast group (eTables 13 and 14).

DISCUSSION

This large, propensity score adjusted, retrospective study suggests that intravenous contrast material administration for CT scanning is not associated with an increased risk of acute kidney injury (AKI) in a cohort of patients with renal insufficiency. These results were observed regardless of propensity score adjustment method, AKI cutoff, or subgroup analysis. These findings provide more robust further evidence that the risk of contrast-induced nephropathy (CIN) is extremely low in the vast majority of patients undergoing CT scanning.

Our findings corroborate prior propensity score studies that also found similar rates of AKI, emergent dialysis, and short-term mortality between contrast-enhanced and unenhanced CT scan recipients, even in patients with renal insufficiency [7,10,11,19]. Our current study builds upon these findings in multiple ways. First, our study included almost all of the reported risk factors for AKI into the propensity score model, including use of potentially nephrotoxic

medications, the presence of associated chronic or acute conditions, and the presence of stable or unstable renal function at the time of CT scan. Second, we performed a manual chart review to validate model covariates instead of relying on automated retrievals of ICD-9 diagnostic codes, which are known to be less accurate [12–14]. Third, we accounted for IV fluid administration data in our analysis to better characterize patients in terms of hydration status. Other AKI prophylactic measures, including N-acetylcysteine and sodium bicarbonate, were not included in the model because there is insufficient evidence of their efficacy [1,20–22].

We found a significantly lower risk of AKI in Stage III contrast recipients compared to propensity score stratified or matched control patients if a cutoff of ≥ 0.3 mg/dL or ≥ 50% over baseline SCr was used to define AKI. There are several potential reasons for this observation. One possibility is that the control patients in this cohort more frequently had minor variability in SCr following CT scan compared to contrast recipients, and this variability could have been interpreted as AKI. Another possibility is that an unmeasured confounder remains in this cohort after propensity score adjustment that results in a higher rate of AKI in the control group. Slightly lower risks of AKI when defined by a cutoff of ≥ 0.5 mg/dL over baseline SCr, emergent dialysis, and mortality were also observed in the Stage III contrast recipients compared to control patients, supporting this hypothesis.

A prior study by Davenport et al. reported a significantly higher rate of AKI in patients with eGFR < 30 ml/min/1.73m^2 that received IV contrast material compared to a propensity score matched control group [6]. Our current findings suggest patients with eGFR < 30 ml/min/1.73m^2 were not at increased risk of CIN. There are several possible reasons for this discrepancy. First, a more comprehensive list of clinical covariates was used in our propensity score models and different methods were used to retrieve covariates from the medical record (i.e. automated ICD-9 diagnostic code retrieval vs. manual chart review). Second, Davenport et al created one propensity score model for all patients while our study created separate models for the CKD Stage III and IV–V subgroups. We believe these two groups represent very different patient populations that require separate propensity score models, a hypothesis strengthened by our finding that different clinical covariates have different relative influences on the propensity scores of the two subgroups. Finally, the discrepancy between study findings may reflect differences in patient populations or clinical practices. Additional prospective and large sample-size retrospective studies are needed, particularly those that examine AKI sequelae including dialysis and death, to determine the safety of intravenous contrast material in patients with severe renal insufficiency.

Our study has several limitations. First, retrospective statistical methods including propensity score adjustment can only account for measured confounders. To our knowledge, our current propensity score model with 32 clinical covariates is the most robust model to date. This expanded model had similar results to our prior propensity score study with fewer covariates. However, unmeasured confounders may still remain in our current study that could affect patient outcomes. Second, since we could only examine patients with sufficient pre- and post-scan SCr results, we were limited to a predominantly inpatient cohort. However, this bias favorable enriches the number of inpatients in our study population, increasing the probability of observing AKI in a more acutely ill population when compared

to outpatients. Third, we were unable to determine whether contrast osmolality affected differences in outcomes since only a small percentage of patients in our cohort (6% of CKD Stage III patients and 17% of CKD Stage IV–V patients) received iso-osmolar contrast material. Fourth, while we used KDOQI chronic kidney disease stage cutoffs to stratify patients by eGFR, a percentage of patients were likely assigned to these subgroups because of acute or sub-acute changes in renal function instead of the presence of true chronic kidney disease. Finally, while prospective randomized controlled trials of CIN are the best way to determine causality, such trials are ethically challenging and require large sample sizes to be sufficiently powered to examine rare outcomes such as emergent dialysis. Additional observational studies incorporating different clinical covariates, patient populations, and clinical practices are needed to confirm the true risk of CIN.

CONCLUSION

Our findings provide additional evidence that the administration of intravenous contrast material does not increase the risk of AKI, emergent dialysis, and mortality, even in patients with substantially compromised renal function.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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ABBREVIATIONS

AKI	acute kidney injury
CT	Computed Tomography
CKD	chronic kidney disease
SCr	serum creatinine
CIN	contrast-induced nephropathy
eGFR	estimated glomerular filtration rate
EMR	electronic medical record
KDOQI	Kidney Disease Outcomes Quality Initiative
AKIN	Acute Kidney Injury Network

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Table 1

Demographics of matched CKD stage III cohort[a]

	Contrast group	Noncontrast group	*P* value
Number of scans (N)	1220	1220	
[1]Year of scan	2006 (2003–2010)	2006 (2003–2010)	.24
[2]Age (median, IQR)	75 (65–83)	75 (64–83)	.74
[3]Female (%)	631 (52%)	605 (50%)	.29
[4]Caucasian race	1108 (91%)	1117 (92%)	.52
[5]Admission			.48
Inpatient	658 (54%)	686 (56%)	
ER/Inpatient	377 (31%)	361 (30%)	
Outpatient	185 (15%)	173 (14%)	
[6]ICU at time of scan	183 (15%)	179 (15%)	.82
Pre-existing comorbidities			
[7]Diabetes mellitus	257 (21%)	279 (23%)	.26
[8]Diabetic nephropathy	40 (3.3%)	42 (3.4%)	.82
[9]Hypertension	538 (44%)	567 (46%)	.19
[10]Chronic kidney disease	371 (30%)	408 (33%)	.08
[11]Multiple myeloma	10 (0.8%)	10 (0.8%)	.99
[12]Congestive heart failure	247 (20%)	261 (21%)	.46
[13]Charlson Comorbidity score	3 (1–6)	3 (2–6)	.23
Conditions within 7d of scan			
[14]AKI	115 (9.4%)	120 (9.8%)	.72
[15]Renal stone	37 (3.0%)	49 (4.0%)	.18
[16]Sepsis	56 (4.6%)	59 (4.8%)	.77
[17]Major surgery	273 (22%)	246 (20%)	.17
Prescribed nephrotoxic medication at time of scan			
[18]Antibiotics other than vancomycin	104 (8.5%)	104 (8.5%)	.99
[19]Vancomycin	138 (11%)	144 (12%)	.69
[20]ACE inhibitors	289 (24%)	295 (24%)	.77
[21]ARBs	112 (9.2%)	116 (9.5%)	.78
[22]Chemotherapeutics	18 (1.5%)	18 (1.5%)	.99
[23]COX2 inhibitors	31 (2.5%)	23 (1.9%)	.27
[24]Loop diuretics	382 (31%)	393 (32%)	.63
[25]HCTZ	154 (13%)	126 (10%)	.07
[26]Immunosuppressants other than sirolimus	35 (2.9%)	36 (3.0%)	.90
[27]Sirolimus	1 (0.1%)	1 (0.1%)	.99
[28]NSAIDs	53 (4.3%)	55 (4.5%)	.85

	Contrast group	Noncontrast group	*P* value
[29]Statins	381 (31%)	386 (32%)	.83
[30]Baseline eGFR	47 (40–52)	46 (39–52)	.07
[31]SCr stability prior to scan			.94
Stable	1083 (89%)	1088 (89%)	
Unstable - Increasing	83 (6.8%)	81 (6.6%)	
Unstable - Decreasing	54 (4.4%)	51 (4.2%)	
[32]SCr Delta (SCr max-min)	0.2 (0.1–0.3)	0.2 (0.1–0.3)	.71

[a]Numbered variables were used to generate the propensity score model.

Table 2

Demographics of matched CKD stage IV–V cohort

	Contrast group	Noncontrast group	*P* value
Number of scans (N)	419	419	
Year of scan	2006 (2003–2008)	2006 (2002–2009)	.82
Age (median, IQR)	70 (61–79)	71 (60–80)	.93
Female (%)	270 (64%)	278 (66%)	.54
Caucasian race	405 (97%)	409 (98%)	.40
Year scan performed			
Admission			.38
Inpatient	347 (83%)	347 (83%)	
ER/Inpatient	52 (12%)	59 (14%)	
Outpatient	20 (4.8%)	13 (3.1%)	
ICU at time of scan	77 (18%)	77 (18%)	.99
Pre-existing comorbidities			
Diabetes mellitus	155 (37%)	158 (38%)	.83
Diabetic nephropathy	21 (5.0%)	25 (6.0%)	.56
Hypertension	341 (81%)	342 (82%)	.93
Chronic kidney disease	201 (48%)	203 (48%)	.87
Multiple myeloma	7 (1.7%)	9 (2.2%)	.62
Congestive heart failure	138 (33%)	152 (36%)	.30
Charlson Comorbidity score	4 (2–8)	4 (2–8)	.67
Conditions within 7d of scan			
AKI	252 (60%)	260 (62%)	.54
Renal stone	7 (1.7%)	7 (1.7%)	.99
Sepsis	57 (14%)	61 (15%)	.69
Major surgery	58 (14%)	63 (15%)	.62
Prescribed nephrotoxic/nephromodulatory medication at time of scan			
Antibiotics other than vancomycin	49 (12%)	60 (14%)	.24
Vancomycin	64 (15%)	64 (15%)	.99
ACE inhibitors	132 (32%)	136 (32%)	.76
ARBs	62 (15%)	54 (13%)	.43
Chemotherapeutics	5 (1.2%)	3 (0.7%)	.48
COX2 inhibitors	15 (3.6%)	18 (4.3%)	.59
Loop diuretics	209 (50%)	227 (54%)	.20
HCTZ	66 (16%)	62 (15%)	.71
Immunosuppressants other than sirolimus	32 (7.6%)	38 (9.1%)	.47
Sirolimus	5 (1.2%)	7 (1.7%)	.57
NSAIDs	28 (6.7%)	30 (7.2%)	.78
Statins	157 (37%)	156 (37%)	.94

	Contrast group	Noncontrast group	*P* value
Baseline eGFR	24 (20–27)	24 (20–27)	.87
SCr stability prior to scan			.84
Stable	251 (60%)	245 (58%)	
Unstable - Increasing	49 (12%)	54 (13%)	
Unstable - Decreasing	119 (28%)	120 (29%)	
SCr Delta (SCr max-min)	0.3 (0.2–0.8)	0.4 (0.2–0.7)	.60

Table 3

CKD stage III cohort outcomes after propensity score analysis

	Contrast group	Noncontrast group	Odds/Hazard ratio (95% CI) [b]	*P* value
Stratified Analysis (# Patients)	2310	2186		
AKI[a]				
AKIN criteria	229 (9.9%)	360 (16%)	0.68 (0.55–0.84)	**<.001**
Standard criteria	102 (4.4%)	157 (7.2%)	0.84 (0.63–1.14)	.29
Dialysis within 30d post-scan	5 (0.2%)	26 (1.2%)	0.69 (0.25–1.93)	.62
Death within 30d post-scan	177 (7.7%)	275 (13%)	0.86 (0.68–1.09)	.25
1:1 Matched Analysis (# Patients)	1220	1220		
AKI[a]				
AKIN criteria	126 (10%)	185 (15%)	0.65 (0.41–0.89)	**<.001**
Standard criteria	61 (5.0%)	71 (5.8%)	0.86 (0.51–1.20)	.38
Dialysis within 30d post-scan	5 (0.4%)	5 (0.4%)	1.00 (0.24–2.24)	.99
Death within 30d post-scan	109 (8.9%)	137 (11%)	0.77 (0.50–1.04)	.06

[a] AKIN criteria defined as ≥ 0.3 mg/dL or 50% over baseline SCr, standard criteria defined as ≥ 0.5 mg/dL over baseline SCr.

[b] Odds of contrast group versus noncontrast group.

Table 4

CKD stage IV–V cohort outcomes after propensity score analysis

	Contrast group	Noncontrast group	Odds ratio (95% CI) [b]	*P* value
Stratified Analysis (# Patients)	474	1612		
AKI[a]				
AKIN criteria	94 (20%)	458 (28%)	1.01 (0.77–1.33)	.99
Standard criteria	65 (14%)	361 (22%)	0.90 (0.66–1.24)	.90
Dialysis within 30d post-scan	7 (1.5%)	28 (1.7%)	1.83 (0.73–4.55)	.31
Death within 30d post-scan	84 (18%)	266 (17%)	1.03 (0.77–1.38)	.89
1:1 Matched Analysis (# Patients)	419	419		
AKI[a]				
AKIN criteria	89 (21%)	81 (20%)	1.14 (0.78–1.50)	.47
Standard criteria	62 (15%)	61 (15%)	1.02 (0.63–1.41)	.92
Dialysis within 30d post-scan	7 (1.7%)	3 (0.7%)	2.33 (0.98–3.68)	.22
Death within 30d post-scan	77 (18%)	81 (19%)	0.93 (0.57–1.29)	.71

[a] AKIN criteria defined as ≥ 0.3 mg/dL or 50% over baseline SCr, standard criteria defined as ≥ 0.5 mg/dL over baseline SCr.

[b] Odds of contrast group versus noncontrast group.