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Transient and Persistent Renal Dysfunction are Predictors of Survival after Percutaneous Coronary Intervention:

Insights from the Dartmouth Dynamic Registry

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Abstract

Objectives: We sought to determine if transient and persistent elevations in creatinine following percutaneous coronary intervention (PCI) resulted in poor survival.

Background: Limited survival data exist that define the natural survival history of transient and persistent post PCI renal dysfunction.

Methods: Data were collected prospectively on 7,856 consecutive patients undergoing PCI from January 1, 2000 to July 31, 2006. Ninety-three patients were excluded due to pre-PCI dialysis. Patients were stratified into three categories of renal dysfunction: no renal dysfunction from baseline (<0.5 mg/dL increase in creatinine within 48 hours of the procedure), transient renal dysfunction (0.5 mg/dL increase in creatinine within 48 hours with return to normal within 2 weeks), and persistent renal dysfunction (0.5 mg/dL increase in creatinine without returning to normal within 2 weeks of the procedure). Mortality was determined by a match to the Social Security Death Master File.

Results: Median survival was 3.2 years (mean 3.4). Renal dysfunction occurred in 250 patients (0.5 mg/dL increase in creatinine). Survival was significantly different between patients at 1, 3.2 and 7.5 years (P-value < 0.001): no renal dysfunction (95%, 88%, 75%), with transient (61%, 42%, 0%) and with persistent (58%, 44%, 36%) renal dysfunction. Patients with transient or persistent renal dysfunction had a 2–3 fold increased adjusted risk of 7.5-year mortality compared with patients with no renal dysfunction.

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Disclosures

There are no conflicts of interest to disclose.

Conclusions: Both transient and persistent post procedural renal dysfunction are prognostically significant for mortality during extended follow-up. Renal dysfunction should be closely monitored before and after PCI.

Condensed Abstract:

Creatinine elevations following PCI have been disregarded as transient events with little-to-no impact on morbidity or mortality. We evaluated the short- and long-term natural history of transient and persistent renal dysfunction following 7,759 PCI patients over 7.5 years. We discovered both *persistent* and *transient* creatinine elevations occur after PCI and that *persistent* and *transient* elevations in creatinine were associated with a significant 2–3 fold increased risk of poor survival. We provide new evidence for the increased mortality of those with renal dysfunction over the 7.5-years of follow-up. Patients developing renal dysfunction following PCI should be closely monitored even if creatinine normalizes prior to discharge.

Keywords

Renal dysfunction; creatinine; PCI; angioplasty; survival

Introduction

Renal dysfunction is a serious consequence of percutaneous coronary intervention (PCI) and is commonly defined as a 0.5 (mg/dL) increase in creatinine from baseline within 48 hours of exposure.(1,2) Renal dysfunction has been reported to occur in 3–14% of patients and results in increased in-hospital and long-term mortality.(3,4) Renal dysfunction is the third most common contributor of renal failure during hospitalization.(5)

Damage to the renal tubules and function elevates creatinine within 48 hours of exposure and may return to baseline within two weeks.(6) Creatinine elevations that return to baseline within two weeks are *transient* creatinine elevations, whereas elevations in creatinine not returning to baseline are *persistent*. The effect of transient versus persistent increases in creatinine after PCI procedures on survival is not known.

The purpose of this investigation was to determine the relationship of transient post-PCI renal dysfunction on survival and define the natural survival history of patients with transient and persistent post-procedural renal dysfunction.

Methods

Study population

The Dartmouth Dynamic Registry (DDR) is a large prospective, clinical, consecutive patient registry of patients having diagnostic or interventional cardiovascular catheterization procedures at Dartmouth-Hitchcock Medical Center. In the present study, data was prospectively collected on 7,856 consecutive PCI patients from January 1, 2000 to July 31, 2006. Ninety-three patients were excluded if they were on dialysis prior to the procedure, leaving 7,759. The registry was approved by the Institutional Review Board (Dartmouth Center for the Protection of Human Subjects), patient consent was waived.

Data collection

Patient, disease, and procedural data were recorded prospectively for all patients. Patient characteristics included: age, sex, weight, height, body surface area, body mass index, morbid obesity, diabetes, hypertension, peripheral vascular disease, chronic obstructive pulmonary disease. Coronary disease characteristics included: history of coronary artery disease, congestive heart failure, chest pain, stable angina, acute coronary syndrome, acute myocardial infarction (MI), recent MI, remote MI, cardiogenic shock, ejection fraction (estimated and/or calculated), prior thrombolysis therapy, prior PCI, prior coronary artery bypass graft surgery (CABG), priority of procedure (elective, urgent, emergent). Laboratory measures included: hypercholesterolemia, positive troponin (> 0.2 ng/mL), creatinine (baseline up until 2 weeks following procedure), estimated glomerular filtration rate (eGFR) based on National Kidney Foundation definitions using the Modification of Diet in Renal Disease (MDRD) equation (mL/min/1.73 m^2): $186 \times (\text{serum creatinine mg/dL})^{-1.154} \times (\text{age})^{-0.203} \times (0.742 \text{ for women})$.(7–9) Procedural variables recorded: type of procedure (diagnostic, interventional), type of contrast (ionic, non-ionic), contrast dye load (mL), contrast agent, fluorography time (min), pre-procedure resting mean aortic pressure, coronary disease classification (none, single, double, triple vessel disease, critical, significant left main disease, indeterminant), intra-aortic balloon pump (IABP). Clinical outcomes monitored: cardiac events (new MI, cardiac arrest, coronary stent thrombosis, not including recurrent angina or new congestive heart failure), cerebrovascular accident (stroke), index-admission mortality, new onset of dialysis-dependent renal failure, length of hospitalization (pre and post-procedure), time from procedure to last recorded creatinine within 2 weeks. Maximum acceptable contrast dose (MACD) was calculated as $(5 \text{ mL} \times \text{body weight kg}) / \text{baseline creatinine}$.(10) High and low contrast dose categories were defined as ≤ 140 (mL) and > 140 (mL).(11,12)

Renal function

Renal function was assessed prior to PCI at baseline, within 48 hours post-PCI and up to 2 weeks following the procedure. At baseline, serum creatinine was measured and eGFR was calculated using the MDRD equation. The highest creatinine within 48 hours after the procedure was used to diagnose renal dysfunction defined as ≥ 0.5 (mg/dL) increase in creatinine from baseline within 48 hours. Patients were stratified into three categories of exposures: no creatinine elevation from baseline (< 0.5 mg/dL increase in creatinine within 48 hours of the procedure), transient renal dysfunction (≥ 0.5 mg/dL increase in creatinine within 48 hours with return to normal within 2 weeks), and persistent renal dysfunction (≥ 0.5 mg/dL increase in creatinine without returning to normal within 2 weeks of the procedure).

Patient follow-up

The primary outcome of this analysis was all-cause mortality up to 7.5 years of follow-up. Mortality was determined by a match of the DDR registry to the Social Security Administration Death Master File (SSDMF, US Department of Commerce National Technical Information Service) using social security number and date of birth. The SSDMF includes all deaths reported to the Social Security Administration occurring in or outside of

the United States, with 92% sensitivity for all deaths.(13) In comparison, the National Death Index sensitivity has been documented between 87 and 98%.(14–16) Time to death for index-admission deaths were recorded from case to discharge. Time to death for post-discharge deaths were calculated from case to date of death recorded by the SSDMF.

Statistical Analysis

Baseline patient and disease characteristics were compared across categories of renal dysfunction (none, transient and persistent) and across renal dysfunction categories alone (transient and persistent) using percentages and means ($\pm$ standard deviation). We used χ^2 tests and non-parametric tests of trend to compare similarities between categories of renal dysfunction. For in-hospital clinical outcomes, χ^2 tests, tests of trend. Kaplan-Meier and log-rank techniques were used to conduct the survival analysis; patients were stratified by renal dysfunction (none, transient and persistent). Incidence of death (deaths per 100 person-years of follow-up) was calculated for each category. Cox's proportional hazard models were used to calculate crude and adjusted hazard ratios (HR): no renal dysfunction as the referent category, adjusting for age, sex, diabetes, prior myocardial infarction, ejection fraction<35%, non-elective priority, length of post-procedural hospitalization, morbid obesity, prior cardiac intervention, baseline eGFR<60 (mL/min/1.73m²). Crude and adjusted Kaplan-Meier curves were created. Adjusted curves were created using the Ghali method, (17) adjusting for age, sex, diabetes, prior myocardial infarction, ejection fraction<35%, non-elective priority, morbid obesity, prior cardiac intervention, length of postoperative stay (1 day, >1 day), and baseline eGFR<60 (mL/min/1.73m²). Analyses were conducted using Stata 9.0 (Stata, College Station, TX). Statistical methods have been described previously. (18,19)

Results

Patient and disease characteristics differed significantly across categories of renal dysfunction (none, transient and persistent, Table 1). Patients developing dysfunction were on average 9 years older, more likely to be female, have more comorbidities, more severe coronary disease and renal function and more urgent or emergent priority for PCI. However, there were few significant differences between the transient and persistent renal dysfunction groups. Patients with transient or persistent renal dysfunction were statistically similar for all characteristics.

Procedural characteristics showed less variability among renal dysfunction categories (Table 2). The proportion of patients receiving iso- or low-osmolar non-ionic dye did not differ across groups. Fluoroscopy time was significantly longer in the renal dysfunction groups. Patients exceeding the maximum acceptable contrast dose were more likely to develop renal dysfunction (P<0.001). There were no significant differences between transient or persistent renal dysfunction procedure characteristics. Both transient and persistent renal dysfunction groups had more extensive coronary disease, including more left main disease, and 5-fold the rate of IABP use during the procedure.

Clinical Outcomes

The rate of in-hospital clinical outcomes was significantly higher among patients with transient or persistent renal dysfunction (Table 3). Patients with renal dysfunction were more likely to develop cardiac events, new onset of dialysis-dependent renal failure and death. Patients with renal dysfunction had significantly longer length of post-procedure stay: none (2.0 ± 4.0), transient (7.9 ± 10.8) and persistent renal dysfunction (7.5 ± 10.5 ; P -value < 0.001).

Long-term Survival

Patients were linked to the Social Security Death Master File with 42,238 person-years of follow-up and 1,225 deaths. Median survival was 3.2 years (mean 3.4). Renal dysfunction (0.5 mg/dL increase in creatinine) occurred in 250 patients. Survival was significantly different between patients at 1, 3.2 and 7.5 years (P -value < 0.001): no renal dysfunction (95%, 88%, 75%), with transient (61%, 42%, 0%) and with persistent (58%, 44%, 36%) renal dysfunction (Figure 1). Patients with transient or persistent renal dysfunction had a 2–3 fold increased adjusted risk of survival compared with patients with no renal dysfunction: transient (HR: 3.13; 95% CI: 2.44–4.02) and persistent (HR: 2.11; 95% CI: 1.60–2.78). Transient and persistent renal dysfunction had similar survival (HR: 0.73; 95% CI: 0.51–1.06).

Discussion

Renal dysfunction is a common and serious consequence following PCI. In this present study, renal dysfunction occurred in 3.2% of patients; 1.6% of the patients developing renal dysfunction (0.5 mg/dL increase in creatinine) within 48 hours of their procedure experienced a transient elevation in creatinine where creatinine returned to normal within two weeks; 1.6% of the patients developing renal dysfunction had persistent elevation in creatinine in which their creatinine did not return to normal within two weeks of the procedure. Renal dysfunction was associated with an increased risk for major cardiac events, in-hospital mortality and new onset of dialysis dependent renal failure during hospitalization. Seven-year follow-up demonstrated renal dysfunction (0.5 mg/dL increase in creatinine) as significant independent predictors of reduced survival. Both transient and persistent renal dysfunction were associated with significantly decreased survival even after adjustment for relevant covariates.

In our analysis, we chose a less sensitive and more specific definition of renal dysfunction (0.5 mg/dL absolute increase in creatinine), which was clinically relevant and demonstrating a substantially reduced survival. However, the literature has accepted a 25% increase in creatinine as a definition of renal dysfunction.(3,20) We re-analyzed our data using the 25% increase in creatinine and present the survival curves in Figure 2. Using the 25% definition, patients with transient or persistent renal dysfunction had a 2-fold increased risk of 7.5-year adjusted survival: transient (HR: 2.18; 95% CI: 1.76–2.69) and persistent (HR: 1.61; 95% CI: 1.30–1.98). The result for both definitions is similar: 2–3-fold increased risk of reduced survival. However the magnitude of difference was larger in the 0.5 (mg/dL) analysis compared with the 25% analysis.

Some discrepancy exists surrounding the definition of renal dysfunction. The more commonly accepted window has been defined at 48 hours, which is the definition we have chosen to use for renal dysfunction.(1–3,20) Our measurement for renal dysfunction recovery to diagnose transient or persistent renal dysfunction was two weeks; although it may be argued additional time would be needed to allow for recovery from renal injury. Rocha-Singh *et al.*, suggest 95% of patients recover from renal injury within a year following exposure(21), while others suggest creatinine returns to baseline within 2 weeks following the procedure.(6) We believe the present study strongly supports the prognostic significance of all post procedural creatinine elevation 0.5 (mg/dL) and 25% within 48 hours irrespective of the “recovery” time window.

In past studies, renal dysfunction has been associated with both short- and long-term mortality. Marenzi *et al.*, reported renal dysfunction rates of 33% without prophylactic treatment with in-hospital mortality at 11%. With the use of N-acetylcysteine, renal dysfunction rates were lower at 8% (double dose) 15% with lower mortality at 3% and 4%. (22) Rich *et al.*, identified patients with creatinine elevations 44μmol/L within 48 hours following cardiac catheterization had an increase risk of in-hospital mortality (14.3%) compared to patients without elevations in post-procedure creatinine (6.2%).(23) Gruberg *et al.*, confirmed Rich’s findings among patients with pre-existing renal dysfunction (baseline serum creatinine 1.8 mg/dL) experiencing 25% increase in creatinine within 48 hours of PCI.(20) Patients with renal dysfunction had an in-hospital mortality of 14.9% compared to patients without renal dysfunction 4.9%. There was a three-fold difference in cardiac related death for patients with renal dysfunction compared with no renal dysfunction: 9.6% versus 3.3%.(20) Rihal and Chertow defined renal dysfunction as 0.5 mg/dL increase in creatinine was associated with an increased risk of in-hospital mortality.(4,24) Patients with renal dysfunction had a 22% mortality rate compared to 1.4%.(4) Patients admitted to the hospital for all causes developing renal injury were 6.5-times more likely to die in the hospital compared to patient not developing renal injury; on average these patients had 3.5 more days in the hospital and \$7500 excess in hospital costs.(24) Our findings support these reports on index-admission mortality and expand these findings by examining the effects of transient and persistent elevations in creatinine with seven-year survival as well as additional resulting morbidities including cardiac events and dialysis.

Several groups have also expanded the work on renal dysfunction and short-term outcomes to long-term survival following interventional procedures. Three studies defined renal dysfunction as 25%(20,25,26) and/or 0.5 mg/dL(25,26) increase in creatinine from baseline. Three additional studies defined renal dysfunction as 0.5 mg/dL increase in creatinine,(4,27,28) while one study defined renal dysfunction as 1.0 mg/dL(29) increase in creatinine and another as 50%(30) increase from baseline. Nikolsky *et al.*, associated renal dysfunction following PCI with a 2.75 times increased risk of 1-year mortality (95%CI: 1.84–4.12). Dangas *et al.*, showed a 2.37-fold increased risk for 1-year mortality (95%CI: 1.63–3.44) among patients with pre-existing renal dysfunction (eGFR<60), whereas patients without pre-existing renal dysfunction (eGFR ≥60) had a relative risk of only 1.78 (95%CI: 1.22–2.60). Gruberg *et al.*, also among patients with pre-existing renal dysfunction (baseline creatinine 1.8 mg/dL) had a 3.86 fold increased risk of death at 1-year (95%CI: 1.96–2.58). Sadeghi *et al.*, demonstrated patients with renal dysfunction at any point during the index-

admission had an increased risk of mortality at 30-days (RR: 13.8; 95%CI: 7.3–26.2) and 1-year (RR: 7.4; 95%CI: 4.7–11.7). Rihal *et al.*, from the Mayo Clinic PCI Registry showed renal dysfunction patients had higher mortality over non-renal dysfunction patients at 6-months (9.8% vs. 2.3%), 1-year (12.1% vs. 3.7%) and 5-years (44.6% vs. 14.6%), respectively. Lindsay *et al.*, evaluated creatinine and CK-MB abnormalities following PCI; they demonstrated patients with renal dysfunction and normal CK-MB had a 3.7-fold increased odds of dying in 1-year (95%CI: 2.2–6.4) and patients with renal dysfunction and CK-MB abnormalities had a 4.6-fold (95%CI: 2.1–9.8) likelihood of dying within one year compared with patients not developing renal dysfunction or CK-MB abnormalities.(27) In another study, Lindsay *et al.*, defined renal dysfunction as 50% elevation in creatinine from baseline and reported patients with renal dysfunction were more likely to die within 1-year of PCI, odds ratio of 2.7 (95%CI: 1.5–4.9).(30) Gupta *et al.*, defined severe renal dysfunction as an absolute increase in creatinine of 1 mg/dL or greater and identified patients with severe renal dysfunction as having an adjusted hazard ratio of 1.77 (95%CI: 1.24–2.54), again demonstrating an increased risk of long-term mortality associated with renal dysfunction.(29) Our findings are in agreement with these reports and extend these findings by associating both transient and persistent renal dysfunction with 7-year survival among patients undergoing PCI.

There are physiological differences that may account for transient and persistent creatinine elevations that should be considered. Here we have demonstrated that despite pathophysiological differences, both transient and persistent elevations are associated with a poor prognosis for short- and long-term morbidity and mortality. We also found that patients developing transient renal dysfunction had poorer survival than patients with persistent renal dysfunction. This effect may be due to the likelihood of more patients with persistent renal dysfunction undergoing dialysis treatment prior to discharge where as patients with transient elevations were less likely to undergo dialysis. This effect may be due to clinical oversight of transient elevations as not severe, where as persistent elevations may have resulted in closer monitoring and treatment prior to discharge.

Quality improvement strategies are required to advance and monitor preventive measures in PCI. Efforts to reduce renal dysfunction by hydration, prophylactic treatment of N-acetylcysteine and sodium bicarbonate should be followed.(11,22,31) Patients should be risk-stratified prior to undergoing PCI to estimate their risk of developing renal dysfunction and to calculate the maximum allowable contrast dose to be administered.(32,33) Patients at high risk of renal dysfunction should be targeted for adequate volume expansion 3–12 hours prior to the procedure and 6–12 hours following contrast exposure. Identification of risk, monitoring of quality indicators to reduce renal dysfunction should be monitored and reported by the cardiology section at least on a monthly basis for continuous monitoring and improvement to reduce renal injury resulting from contrast.(1,34–36)

There are limitations associated with these analyses. These findings were derived from a single medical center and they may not be generalizable to other geographic areas. However, Dartmouth participates in the Northern New England Cardiovascular Disease Study Group, which in turn has been found to be generalizable to other geographic areas.(37,38) We were unable to report on prophylactic treatments. Currently at our institution prophylactic

treatments are conducted before and after the procedure, however these are not recorded in the registry. Efforts are underway to capture this information. Lastly, creatinine at baseline may not reflect a true baseline of renal function due to the presence of acute myocardial infarction, unstable angina, left ventricular dysfunction, heart failure or cardiogenic shock. While this may not reflect a true baseline, the last creatinine measure prior the procedure is a measure of renal function nearest to the procedure and point of contrast exposure.

Renal dysfunction is not an uncommon by-product of PCI and is one of many potential etiologies of post-procedural renal dysfunction. Both transient and persistent renal dysfunction were associated with a poor prognosis for short- and long-term survival and in-hospital morbidities. Efforts to identify patients at risk of impaired renal function after PCI should be identified prior to the procedure. Hydration protocols and minimal contrast volume should be adopted as common practice. Whether this “unmasked” renal dysfunction serves only as a prognostic marker, or if suppression of this phenomenon can improve outcomes, remains a subject of intense investigation.

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Abbreviations List:

CABG	Coronary artery bypass graft surgery
DDR	Dartmouth Dynamic Registry
IABP	Intra-Aortic Balloon Pump
MACD	Maximum acceptable contrast dose
MDRD	Modification of Diet in Renal Disease
MI	acute Myocardial Infarction
PCI	Percutaneous Coronary Intervention
SSADMf	Social Security Association Death Master File

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Figure 1:

Adjusted Kaplan-Meier Survival Plot Stratified By Transient and Persistent Renal Dysfunction. Adjusted Kaplan-Meier survival curves, adjusted for age, sex, diabetes, prior myocardial infarction, ejection fraction <35%, non-elective priority, length of post-procedural hospitalization, morbid obesity, prior cardiac intervention, baseline eGFR <60 (mL/min/1.73m²). Blue: no renal dysfunction (<0.5 mg/dL increase in creatinine from baseline). Red: transient renal dysfunction (≥0.5 mg/dL increase in creatinine from baseline and creatinine returned to normal within 2 weeks). Green: persistent renal dysfunction (≥0.5 mg/dL increase in creatinine from baseline and creatinine remained elevated within 2 weeks).

Table 1:

Patient and disease characteristics

Variables	Renal Dysfunction Category			All P-value	Transient vs. Persistent P-value
	None	Transient	Persistent		
Number (N=7,759)	7,509	125	125		
Patient and disease characteristics					
Age (years)	64.3±12.4	73.3±9.9	73.0±12.4	<0.001	0.960
Sex (female, %)	29.9	43.2	44.8	<0.001	0.799
BMI	29.1±5.9	29.7±6.4	28.5±6.9	0.226	0.107
Morbid obesity (%)	8.4	14.4	12.6	0.053	0.549
Diabetes (%)	26.8	46.3	45.9	<0.001	0.998
Hypertension (%)	63.6	75.2	66.4	0.004	0.296
Peripheral vascular disease (%)	8.5	20.7	17.8	<0.001	0.519
COPD (%)	9.2	20.0	12.1	0.001	0.097
Cardiac disease					
Congestive heart failure (%)	10.2	41.5	32.0	<0.001	0.123
Chest pain (%)	65.3	55.0	60.7	0.001	0.597
Stable angina (%)	17.2	6.5	10.4	<0.001	0.495
Acute Coronary Syndrome (%)	32.3	48.2	36.3	0.001	0.196
Prior myocardial infarction (%)	53.0	72.7	65.6	<0.001	0.228
Cardiogenic shock (%)	1.2	9.6	11.2	<0.001	0.679
Ejection fraction <35% (%)	4.9	12.2	15.6	<0.001	0.444
Ejection fraction (%)					
70	14.8	4.8	12.0	0.116	0.939
60–69	37.7	28.6	32.0		
50–59	25.9	19.1	20.0		
40–49	12.6	23.8	16.0		
<40	9.0	23.8	20.0		
Prior therapy and priority					
Prior thrombolysis (%)	8.1	6.0	8.0	0.945	0.567
Prior coronary intervention (%)	26.3	20.3	24.4	<0.001	0.298
Prior CABG (%)	14.5	23.0	26.1	0.001	0.576
Priority (%)					
Elective	58.1	43.2	38.4	<0.001	0.481
Urgent	26.4	36.0	34.4		
Emergent	15.5	20.8	27.2		
Laboratory measures					
Hypercholesterolemia (%)	62.6	60.8	54.1	0.118	0.109
Positive troponin (%)	9.0	12.8	12.0	0.189	0.848
Baseline creatinine (mg/dL)	1.0±0.4	1.6±1.0	1.7±1.0	<0.001	0.100
Baseline eGFR (mL/min/1.73 ² m)	73.2±27.2	53.7±24.6	49.7±26.9	<0.001	0.095
Baseline eGFR (mL/min/1.73 ² m)					

Variables	Renal Dysfunction Category			All P-value	Transient vs. Persistent P-value
	None	Transient	Persistent		
90	24.5	7.2	8.0	<0.001	0.056
60–89	48.7	33.6	17.6		
30–59	21.8	41.6	53.6		
15–29	1.4	15.2	16.0		
<15	3.6	2.4	4.8		

Renal dysfunction: 0.5mg/dL increase in creatinine from baseline within 48 hours of procedure; transient: elevation with return to normal; persistent: elevation without return to normal; P-value: chi-square test or test of trend; BMI: body mass index; COPD: chronic obstructive pulmonary disease; CAD: coronary artery disease; CABG: coronary artery bypass graft surgery; eGFR: estimated glomerular filtration rate using MDRD equation.

Table 2:

Procedural characteristics

Variables	Renal Dysfunction Category			All P-value	Transient vs. Persistent P-value
	None	Transient	Persistent		
Contrast					
Omnipaque (%)	26.7	33.6	25.0	0.795	0.147
Visipaque (%)	73.3	66.4	75.0		
Fluoro time (min)	22.1±15.8	27.1±17.6	27.5±17.7	<0.001	0.911
Exceeded MACD (%)	17.5	44.0	48.8	<0.001	0.447
Pre-resting aortic pressure	91.7±25.6	83.3±25.5	82.8±24.7	<0.001	0.880
Coronary disease (%)					
No coronary artery disease	1.2	0.0	0.8	<0.001	0.779
Single vessel	28.5	13.6	13.6		
Double vessel	27.9	20.0	16.0		
Triple vessel	23.2	30.4	29.6		
Critical left main	1.9	9.6	8.0		
Significant left main	4.7	10.4	16.0		
Indeterminant	12.6	16.0	16.0		
Intra-aortic balloon pump (%)	2.5	14.4	20.0	<0.001	0.241

P-value for chi-square test or test of trend; MACD: maximum acceptable contrast dose: ((5mL*body weight kg) creatinine).

Table 3:**In-hospital Outcomes**

In-hospital outcomes	Renal Dysfunction Category				
	None	Transient	P-value	Persistent	P-value
Cardiac events [*] (%)	1.0	4.0 (2.10–7.46)	<0.001	2.6 (1.28–5.49)	0.009
Stroke (%)	1.0	1.6 (0.21–11.98)	0.659	3.0 (0.68–13.40)	0.147
Mortality (%)	1.0	8.6 (5.10–14.40)	<0.001	8.9 (5.29–14.81)	<0.001
Dialysis (%)	1.0	80.2 (25.65–251.10)	<0.001	190.5(67.95–533.86)	<0.001

^{*} Cardiac events (new MI, cardiac arrest, stent thrombosis, not including recurrent angina or new congestive heart failure; Odds ratios and 95%CI from multivariate logistic regression.