Predictors and Outcomes of Post-Contrast Acute Kidney Injury After Endovascular Renal Artery Intervention

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Abstract

Purpose—To determine the incidence, predictors and clinical outcomes of post-contrast acute kidney injury (PC-AKI) following renal artery stent placement for atherosclerotic renal artery stenosis.

Materials and Methods—This retrospective study reviewed 1,052 patients who underwent renal artery stent placement for atherosclerotic renal artery stenosis. In total, 437 patients with follow-up were included in this study. Mean age was 73.6 ± 8.3 years. Post-contrast acute kidney injury was defined as an absolute serum creatinine increase $\geq$ 0.3 mg/dL or percentage increase in serum creatinine $\geq$ 50% within 48 hours of intervention. Logistic regression analysis was performed to identify risk factors for PC-AKI. The cumulative proportion of patients who died or went on to hemodialysis was determined using Kaplan-Meier survival analysis.

Results—Mean follow-up was 71.1 ± 68.4 months. Twenty-six patients (5.9%) developed PC-AKI. Patients who developed PC-AKI had significantly higher levels of baseline proteinuria compared to those who did not (Odds ratio 1.38; 95% CI 1.11–1.72; P=0.004). Prehydration, chronic kidney disease stage, baseline GFR, statin medications, contrast volume and iodine load were not associated with higher rates of PC-AKI. Dialysis-free survival and mortality rates were not significantly different between patients with and without PC-AKI (P=0.50 and P=0.17, respectively).

Conclusion—Elevated baseline proteinuria was the only predictor for PC-AKI in patients undergoing renal artery stent placement. Patients who developed PC-AKI were not at greater risk for hemodialysis or death.

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Introduction

Post-contrast acute kidney injury (PC-AKI) describes the acute decline of renal function after exposure to iodinated contrast material in the absence of another etiology (1). Studies have implicated iodinated contrast as one of the most common culprits of iatrogenic renal injury (2, 3). Furthermore, PC-AKI may be associated with increased risk for hemodialysis, mortality, longer hospital admissions and greater healthcare costs (4–6). However, other studies have questioned these implications by asserting a lack of causal evidence between contrast and renal injury as well as an overestimation of PC-AKI in the presence of physiologic creatinine fluctuations (7–10).

The majority of data on PC-AKI following intra-arterial contrast administration is based on cardiac angiography (8). The incidence of PC-AKI from intra-arterial contrast administration may range from 2% to 45% (11–14). In part, this wide range is a result of the variable definitions used for PC-AKI, many of which do not follow the definition adopted by the American College of Radiology (15). Limited studies have investigated PC-AKI in the setting of renal intervention despite the fact that these patients often have some degree of compromised renal function and may be at higher risk for acute kidney injury (16). Given this lack of data, the current study aimed to elucidate the incidence, predictors and outcomes of PC-AKI after renal artery stent implantation for atherosclerotic renal artery stenosis.

Materials and Methods

Patient population

Institutional review board approval was obtained for this Health Insurance Portability and Accountability Act compliant study. This was a retrospective, longitudinal follow-up study. Clinical data was reviewed for all patients who underwent renal artery stent implantation for atherosclerotic renal artery stenosis from January 1996 to June 2009 with follow-up to December 2015. In total, 1,052 patients underwent renal artery stent placement. Of these, 299 patients were excluded for incomplete baseline clinical data, 219 patients were excluded for inadequate information regarding iodinated contrast type or dose, and 97 patients excluded because they were lost to follow-up. The remaining 437 patients were included in the study; all of whom underwent renal artery stent implantation with iodinated contrast. Gadolinium and/or CO_2 were not used as contrast agents in these patients. Mean age of the study cohort was 73.6 ± 8.3 years. Baseline patient characteristics are summarized in Table 1.

Procedure

Patients were referred to the Intervention Radiology Division for renal artery revascularization if Doppler ultrasound demonstrated a peak systolic velocity >180 cm/s or a renal-to-aortic ratio >3.5 or if the stenosis exceeded 50% of the luminal diameter on computed tomography angiography or conventional angiography. Renal artery stent placement was performed by three board-certified interventional radiologists. Antihypertensive and statin medications were continued until the day of the procedure in all patients. Intravascular access was achieved via the common femoral artery. Upon obtaining

access, the renal arteries were selected and angiograms were performed. Identified stenoses were treated with balloon-mounted bare-metal stents. None of the patients received embolic protection.

A variety of contrast media were used including low-osmolar agents, Iohexol (Omnipaque; GE Healthcare, Chicago, IL, USA) and iopamidol (Isovue; Bracco Diagnostics, Milan, Italy), as well as iso-osmolar, iodixanol (Visipaque; GE Healthcare). Seventy-eight percent of patients with CKD stage 3B or greater (Glomerular filtration rate (GFR) 44 mL/min/ 1.73 m^2) were admitted within 24 hours of the procedure and pretreated with intravenous isotonic fluids at a rate of 1 mL/kg/hr. Other potential renoprotective treatments such as bicarbonate and N-acetylcysteine (NAC) were not recorded for this study.

Measured outcomes

The primary outcome was the incidence of PC-AKI within 48 hours of renal stent placement. Secondary endpoints included time to hemodialysis and death. Estimated glomerular filtration rate (eGFR), 24-hour proteinuria, medications and comorbidities including chronic kidney disease (CKD), cardiac disease, metabolic disease, and smoking history were analyzed as possible contributing factors for PC-AKI. Urine protein levels were obtained within one month of the procedure. Information on the need for dialysis was obtained by querying the USRDS database. Mortality information was obtained by querying the death data in the US Social Security Death Index and health system medical record.

Glomerular filtration rate (GFR) was calculated based on preintervention serum creatinine levels and patient demographic information using the Modification of Diet in Renal Disease equation (17). Chronic kidney disease stages were determined based on the Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group classification (18).

Post-contrast acute kidney injury was determined using the Acute Kidney Injury Network (AKIN) criteria, which defines stage 1 PC-AKI as an absolute serum creatinine increase 0.3 mg/dL or a relative increase in serum creatinine 50% within 48 hours of intervention (19). Although a reduction in urine output 0.5 mL/kg/hr for at least 6 hours within 48 hours of a nephrotoxic event is also considered criteria for acute kidney injury, this metric was not routinely recorded during or after intervention and was excluded from analysis.

Statistical analysis

Statistical analysis was performed with SAS version 9.4 (SAS Institute, Cary, NC). Comparisons were made between normally distributed continuous variables with the Student paired *t*-test. Categorical variables were compared using the Pearson chi-squared test. Logistic regression analysis was performed to identify risk factors associated with PC-AKI after renal artery stent placement. Statistical significance was defined as p<0.05. The cumulative proportion of patients surviving with and without hemodialysis as well as mortality w determined using Kaplan-Meier survival analysis.

Results

A total of 437 patients fulfilled the inclusion criteria and were analyzed. Mean follow-up was 71.1±68.4 months. Bilateral stents were placed in 98 (22.4%) individuals. Average stent diameter was 6.0±0.9 mm. Twenty-six (5.9%) patients developed PC-AKI consistent with at least AKIN stage 1 renal injury. None of the patients required hemodialysis for immediate management of PC-AKI. No major postprocedural complications requiring intervention or prolonged hospitalization occurred.

Predictors for post-contrast acute kidney injury

Risk factors associated with PC-AKI were determined with logistic regression analysis (Table 2). The only significant risk factor was the severity of baseline 24-hr proteinuria, where patients with PC-AKI had higher levels of urine protein (Odds ratio 1.38; 95% CI 1.11–1.72; P=0.004). Baseline creatinine, eGFR and CKD stage were not significantly associated with PC-AKI (P=0.72, P=0.29 and P=0.12–0.97, respectively).

One hundred and sixty-six (38%) patients were admitted within 24 hours prior to intervention for intravenous hydration. These patients did not have lower rates of PC-AKI (P=0.189). No increased risk for PC-AKI was observed based on stent diameter (P=0.65), bilateral intervention (P=0.27), or medication including statins (P=0.95), calcium channel blockers (P=0.28), and ACEI/ARB (P=0.22).

Mean iodinated contrast volume was 135.2±72.2 mL. The iodine load was calculated to account for differences in iodine concentration among contrast agents. This parameter was calculated for each procedure by multiplying the contrast concentration by the volume of contrast delivered. Mean iodine load was 42.5±22.5 g. Neither contrast volume nor iodine load were associated with PC-AKI (P=0.17 and P=0.15, respectively).

Outcomes after post-contrast acute kidney injury

The Kaplan-Meier survival analyses for the cumulative incidence of hemodialysis and death are demonstrated in Figure 1. Median time to hemodialysis among patients with PC-AKI was 43.7±31.8 months compared to 46.4±43.6 months among those without PC-AKI. Patients who developed PC-AKI did not have significantly greater risk for long-term hemodialysis (Hazard ratio [HR] 1.16; 95% CI 0.75–1.81; P=0.50). Two hundred and nineteen patients died within the follow-up period. Mortality rates were not significantly different between the two groups (HR 1.44; 95% CI 0.85–2.44; P=0.17).

Discussion

The incidence and clinical significance of PC-AKI is controversial. In this large, single-institution, retrospective study, 5.9% of patients who underwent renal artery stent placement developed PC-AKI. A systematic review of patients who underwent peripheral angiography and endovascular therapy by Prasad et al demonstrated a median PC-AKI incidence rate of 10% (range: 0–45%) among 10,316 peripheral procedures with a median volume of contrast of 138.2 mL (range: 49.0–240.5 mL) (14). The incidence of PC-AKI in the present study was at the lower end of this range despite higher mean contrast volume. Peng et al reported a

PC-AKI rate of 17.3% in patients (n=150) who underwent renal artery intervention (16). In their study, PC-AKI was defined as a relative increase in serum creatinine by 25% or an absolute increase in serum creatinine by 0.5 mg/dL within 72 hours. The use of less stringent parameters for defining PC-AKI with regard to time and relative creatinine change compared to the current study may have contributed to the higher incidence rate.

Patients with PC-AKI had higher baseline 24-hour proteinuria than patients who did not develop PC-AKI. In 2010, a large cohort study with 920,985 adults reported that the risk of acute kidney injury increased substantially with the presence and severity of proteinuria (20). Subsequent studies implicated proteinuria as a risk factor of AKI after intra-arterial contrast administration in the settings of percutaneous coronary intervention and stroke intervention (21–23). Piskinpasa et al reported that PC-AKI was significantly higher when proteinuria exceeded 1 g/day (24). The authors of the Cardiovascular Outcomes in Renal Atherosclerotic Lesions (CORAL) trial reported that patients had significantly better event-free survival, cardiovascular disease-related death, progressive renal insufficiency, and overall survival when the albumin/creatinine ratio was 22.5 mg/g (25). The cutoff of 22.5 mg/g was established by taking median ratio of the study population, which consisted of 826 patients. Further investigation with larger populations is needed to establish a more discrete threshold level of proteinuria that predicts the risk for developing PC-AKI. While proteinuria may be important for assessing renal function and PC-AKI risk, accurate urine protein evaluation, performed over 24 hours, is time intensive. Therefore, using the albumin/creatinine ratio, which relies on spot albumin, may be more clinically feasible.

The mechanism linking proteinuria to PC-AKI is unclear. Proteinuria is an etiology for tubulointerstitial damage though upregulation of inflammatory mediators such as endothelin-1, NF-κB and monocyte chemoattractant protein-1 (26). Greater levels of proteinuria may represent more advanced renal compromise with less functional reserve to withstand additional insults from nephrotoxic chemicals including iodinated contrast.

Patients taking statin medications did not have lower rates of PC-AKI. Some studies have demonstrated that statins lowered PC-AKI risk (27–29). For example, a placebo-controlled, double-blind clinical trial with 220 subjects found 80 mg of atorvastatin combined with NAC and standard intravenous hydration decreased the risk of renal injury within 24 hours of percutaneous coronary intervention compared to a combination of placebo, NAC and hydration. However, the rate of acute kidney injury was not statistically different at 48 hours (30). Other studies have not found an association between statins and kidney injury reduction (31–33). The value of NAC for PC-AKI prevention is controversial and studies have suggested NAC may reduce creatinine without actually preventing renal injury (19). Thus, NAC was not investigated in this study.

Contrast volume and iodine load were not observed risk factors for PC-AKI. Prior studies have reported a positive correlation between contrast volume and PC-AKI (34, 35). There are limited data on the effect of iodine load on PC-AKI incidence (36). Iodine load is a derivative of contrast volume and iodine concentration and may have a more direct effect on PC-AKI than volume alone. Iodine concentration influences contrast viscosity, which negatively correlates with renal oxygenation and potentially increases the risk for kidney

injury (37). Additional previously reported risk factors including advanced chronic kidney disease and diabetes were not associated with increased rates of PC-AKI in this study (38, 39).

Long-term mortality may be increased by PC-AKI, especially in patients with advanced CKD (15, 40, 41). Patients in this study who developed renal injury within 48 hours of intervention did not have an increased risk for hemodialysis or mortality. These findings parallel those reported by McDonald et al who reported intravenous contrast material was not associated with increased risk of hemodialysis and mortality among 6,902 subjects (42).

This study was limited by its nonrandomized retrospective design. As a result, follow-up did not occur at regular intervals and medical therapy was not standardized. In many patients, multiple contrast agents with different osmotic concentrations were administered, undermining the feasibility of a subgroup analysis on the effects of contrast osmolarity. However, only low-osmolar or iso-osmolar contrast media were used in this study and the efficacy of iso-osmolar agents compared to that of low-osmolar agents in reducing AKI has been debated (43, 44). Other clinical factors such as potentially nephrotoxic medications may impact renal function, but were not measured in this study. The contribution of atherosclerotic emboli to renal injury remains indeterminant, but embolic protection, which was not used in this study, was not a confounding variable.

The incidence of PC-AKI in this study was at the lower end of the reported range for intra-arterially administered contrast. Proteinuria was a significant predictor for PC-AKI and should be assessed prior to renal artery endovascular intervention when clinically feasible. PC-AKI did not have a significant impact on hemodialysis or mortality in this study.

Acknowledgments

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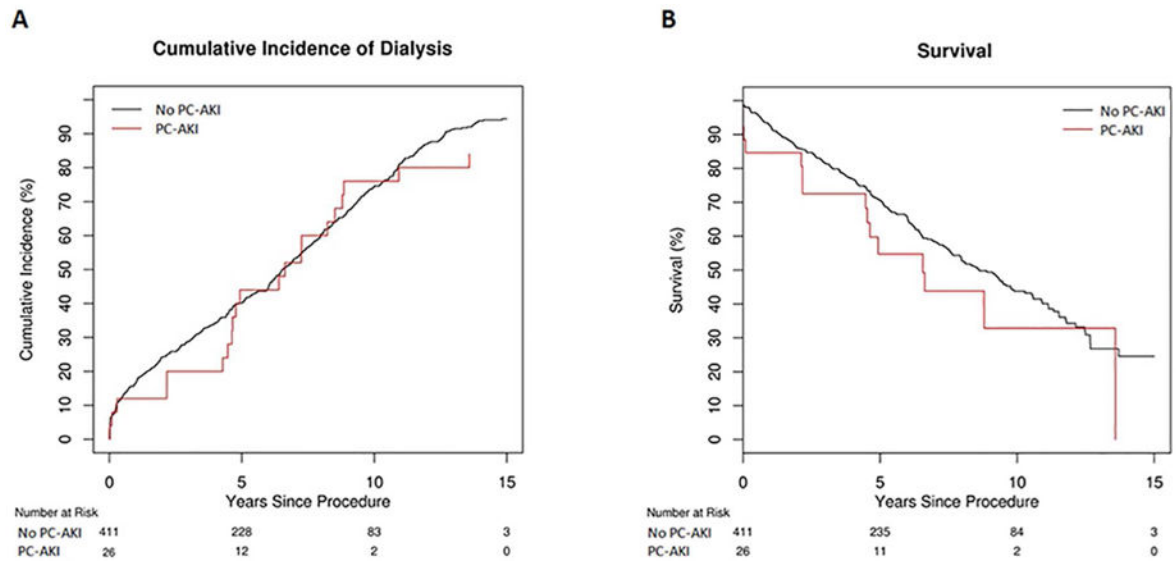
Figure 1.

(A) Kaplan-Meier hemodialysis-free survival curves for patients with and without PC-AKI.

(B) Kaplan-Meier overall survival curves for 437 patients with and without PC-AKI.

Table 1

Baseline characteristics of patients who underwent renal artery stent placement.

	CIN (N=26)	No CIN (N=411)	Total (N=437)	P
Gender, n (%)				
Female	16(61.5)	195(47.4)	211(55.8)	0.16
Male	10(38.5)	216(52.6)	226(51.7)	
Age, mean (SD), years	75.7(6.8)	72.9(8.9)		0.12
Bilateral renal artery stenosis, n (%)	2(7.7)	66(16.1)	68(55.6)	0.25
GFR, mean (SD)	49.6(27.5)	46.0(15.9)	46.3(16.8)	0.31
Proteinuria, mean (SD), (mg/24hr)	1103(2386.1)	376.9(854.3)	419.5(1018.9)	<0.001
CKD stage, n (%)				
1/2	3(11.5)	69(16.8)	72(16.5)	0.47
3A	11(42.3)	142(34.5)	153(35.0)	0.42
3B	9(34.6)	142(34.5)	151(34.6)	0.99
4	2(7.7)	55(13.4)	57(13.0)	0.40
5	1(3.8)	3(0.7)	4(0.9)	0.11
Current smoker, n (%)	5(19.2)	76(18.5)	81(18.5)	0.93
Diabetes, n (%)	12(46.2)	126(30.7)	138(31.6)	0.10
Coronary artery disease, n (%)	18(69.2)	246(59.8)	264(60.4)	0.34
Hyperlipidemia, n (%)	20(76.9)	333(81.0)	353(80.8)	0.61
Hypertension, n (%)	26(100.0)	403(98.1)	429(98.2)	0.47

Table 2

Logistic regression analysis for predictors of post-contrast acute kidney injury following renal artery stent placement.

	N	Odds Ratio	Lower 95% CI	Upper 95% CI	P
Female sex	211	1.77	0.79	4.00	0.17
Proteinuria[†]		1.38	1.11	1.72	0.004
Creatinine		1.12	0.06	2.10	0.72
eGFR		1.01	0.99	1.03	0.29
CKD stage					
1/2	72	0.65	0.19	2.21	0.49
3A	153	1.78	0.48	6.59	0.97
3B	151	1.46	0.38	5.56	0.65
4	57	0.84	0.14	5.18	0.25
5	4	7.67	0.61	97.4	0.12
Statin medication	351	1.03	0.38	2.82	0.95
Antihypertensive medication					
ACEI/ARB	385	3.54	0.47	26.7	0.22
Calcium channel blocker	350	1.97	0.58	6.72	0.28
Prehydration	165	0.59	0.27	1.30	0.19
Contrast volume		1.00	0.99	1.01	0.17
Total iodine mass		1.01	0.99	1.03	0.15
Stent diameter		0.90	0.59	1.40	0.65
Bilateral intervention	68	0.44	0.10	1.89	0.27
Current smoker	81	0.99	0.36	2.73	0.99
Diabetes	138	1.93	0.87	4.30	0.11
Coronary artery disease	264	1.47	0.63	3.47	0.38
Hypertension	429	0.99	0.05	21.57	0.99
Hyperlipidemia	353	0.75	0.29[*]	1.93	0.55