Impact of Chronic Kidney Disease on Contrast-Induced Nephropathy, Bleeding, and Long-Term Outcomes After Rotational Atherectomy: A Multicenter Retrospective Study

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Posted Date: 27 February 2026

doi: 10.20944/preprints202602.1883.v1

Keywords: rotational atherectomy (RA); chronic kidney disease (CKD); contrast-induced nephropathy (CIN)

 doi:10.20944/preprints202602.1883.v1

Article

Impact of Chronic Kidney Disease on Contrast-Induced Nephropathy, Bleeding, and Long-Term Outcomes After Rotational Atherectomy: A Multicenter Retrospective Study

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Abstract

Background and Objectives: Chronic kidney disease (CKD) is associated with severe coronary calcification and increased procedural risks. We aimed to evaluate the impact of CKD on contrast-induced nephropathy (CIN), bleeding, and long-term clinical outcomes in patients undergoing rotational atherectomy (RA). *Materials and Methods:* This study retrospectively analyzed 652 patients who underwent RA for calcified coronary lesions from the multicenter ROCK registry and a single-center extension between 2010 and 2025. Patients were classified into CKD (eGFR < 60 mL/min/1.73 m^2, n = 66) and non-CKD (n = 586) groups, excluding those on dialysis. The primary endpoint was a composite of CIN and in-hospital bleeding. Secondary endpoints included 3-year target vessel failure (TVF), myocardial infarction (MI), and total bleeding. *Results:* The primary composite outcome occurred more frequently in the CKD group (16.7% vs. 5.1%, p = 0.001). Specifically, CIN was significantly higher in CKD patients (15.2% vs. 1.7%, p < 0.001), while in-hospital bleeding did not differ significantly. In multivariate analysis, CKD was an independent predictor of the primary outcome (adjusted OR 3.02; 95% CI 1.36–6.69; p = 0.006). At 3-year follow-up, total bleeding (10.6% vs. 3.9%, p = 0.008) and MI (6.1% vs. 2.1%, p = 0.024) were higher in the CKD group, whereas TVF and cardiac death showed no significant difference. *Conclusions:* CKD is a robust independent risk factor for CIN and long-term bleeding in patients undergoing RA. However, comparable long-term

 doi:10.20944/preprints202602.1883.v1

efficacy outcomes suggest that RA remains a feasible strategy in CKD patients when early complications are carefully managed with contrast-minimizing strategies.

Keywords: rotational atherectomy (RA); chronic kidney disease (CKD); contrast-induced nephropathy (CIN)

1. Introduction

Chronic kidney disease (CKD) is associated not only with a higher burden of coronary artery disease but also with a disproportionately high prevalence of severe coronary artery calcification; a meta-analysis reported coronary artery calcification in approximately 60% of CKD patients, and observational data suggest that calcification can be present in up to 90% of patients with more advanced CKD group [1–3].

Severe coronary calcification is a well-studied risk factor or procedural complexity and is associated with suboptimal percutaneous coronary intervention (PCI) performance and worse clinical outcomes, including higher rates of repeat revascularization and adverse events after intervention [4,5].

In patients with CKD, contrast using invasive procedures such as coronary angiography and PCI carry a heightened risk of contras-induced nephropathy (CIN) and bleeding complications compared with patients with preserved renal function, reflecting both reduced renal reserve and a CKD-related bleeding diathesis [6]. Consistently, CKD has been linked to increased rates of major adverse cardiovascular events and mortality after PCI, with higher ischemic and hemorrhagic event rates observed in contemporary cohorts [7] and large trial datasets [8].

Rotational atherectomy (RA) is an established strategy for severely calcified, high complexity coronary lesions; however, it is used in a relatively small proportion of PCI procedures worldwide, typically on the order of 1-2% of all PCI cases in national datasets [9,10].

Despite the growing body of RA literature, data specifically evaluating CIN and bleeding, along with long-term clinical outcomes in RA-treated patients, particularly stratified by CKD status remain limited. Therefore, we conducted a retrospective analysis based on a RA registry to compare CIN, bleeding, and long-term outcomes between CKD and non-CKD patients undergoing RA.

2. Materials and Methods

2.1. Study Design and Population

The clinical data of 540 patients with severe calcific coronary artery disease who underwent PCI using RA between January 2010 and October 2019 at 9 tertiary centers from the 'ROtational atherectomy in Calcified lesions in Korea (ROCK)' registry and additional 178 patients from Saint Vincent hospital from November 2019 to August 2025 were retrospectively analyzed. Among them, 66 patients under dialysis were excluded since our main outcome, CIN was not able to be evaluated in dialysis patients. The clinical characteristics of total 652 patients were collected from each center using a standardized case report form of procedural details and follow-up data. Follow-up data were obtained from patient medical records or physician/patient interviews were retrospectively analyzed. The local ethics committee of each hospital approved this study. Informed consent to participants was waived because this study was designed as retrospective study. All methods were performed in accordance with the relevant guidelines and regulations. Between 2010 and 2019, consecutive patients with heavily calcific coronary lesions and significant stenosis (stenosis ≥ 70% of vessel diameter) who underwent PCI using RA were retrospectively enrolled from each institutional database. CKD was defined as an estimated glomerular filtration rate <60 mL/min/1.73 m^2, as calculated using the Modification of Renal Diet equation from baseline serum creatinine [11]. Study population and flow chart are described in Figure 1.

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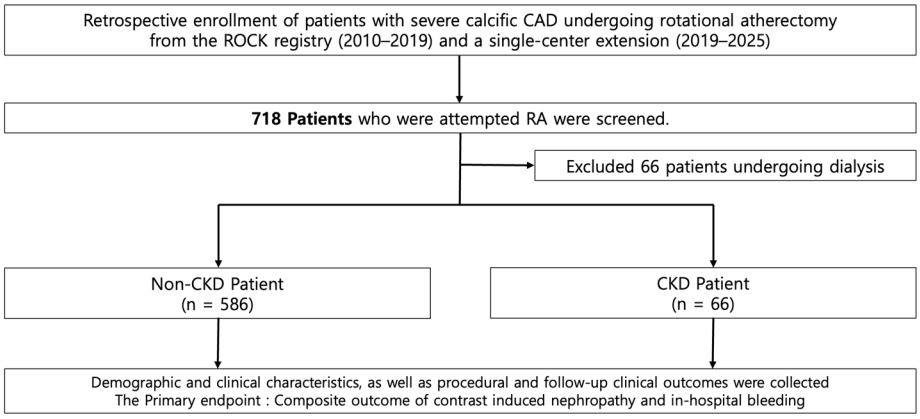

Figure 1. Study population and flow chart.

2.2. RA Procedure

Following the protocol of ROCK Registry, the treatment strategy, including decisions of performing/timing of RA, burr size, and choice of vascular access, was at the discretion of the attending cardiologists with careful consideration of clinical risk factors, anatomical complexity, patients' conditions. Standard techniques guided all procedures. All RA procedures were performed using the Rotablator ™ RA system (Boston Scientific, Marlborough, MA, USA). During RA, pauses in ablation runs and intracoronary nitroglycerin and/or verapamil were used to avoid coronary spasm and slow flow phenomenon. Antiplatelet therapy and peri-procedural anticoagulation were performed following the accepted guidelines [12,13]. Also, direct RA is included in our study defined as the early application of RA (1) before pre-balloon; (2) pre-balloon dilation with balloon size of less than 2.0 mm; (3) judged by tactile sense of balloon passage by operators; or (4) presence severe calcification on imaging modalities. On the other hand, indirect RA was defined as RA after the balloon was expanded to a size greater than 2.0 mm.

2.3. Study Outcomes

The primary endpoint of the study was a composite of CIN and in-hospital bleeding. And the individual outcome was also evaluated. CIN was defined as the impairment of kidney function measured as either a 25% increase in serum creatinine from baseline or a 0.5 mg/dL increase in absolute serum creatinine value within 48–72 h after the procedure. The secondary endpoint were 3-year outcome of target-vessel failure (TVF), defined as cardiac death, target-vessel spontaneous myocardial infarction (TVMI), or target vessel revascularization (TVR), all-cause death, cardiac death (CD), any MI, TVMI, any repeat revascularization (RR), TVR, target-lesion revascularization (TLR), stent thrombosis (ST), cerebrovascular accident (CVA), and bleeding. Technical success was defined as the achievement of residual stenosis < 30% in the presence of grade III Thrombolysis in myocardial infarction flow. Procedural success was defined as achieving technical success without in-hospital major adverse cerebral and cardiac events (MACCEs), including in-hospital death, in-hospital CVA, urgent revascularization (CABG or PCI), peri-procedural MI or ST during the hospitalization period. Procedural complications included cardiac tamponade, coronary perforation, severe coronary dissection, defined from The National Heart, Lung, and Blood Institute classification system as type D, E, and F, temporary pacemaker insertion, CIN, or in-hospital bleeding. We investigated procedure time, radiation dose, and contrast amount to assess procedural efficiency and safety. Death was defined as death from any cause. TVMI was spontaneous MI clearly attributable to the target vessel. Spontaneous MI was defined as any creatine kinase-myocardial band or troponin increase above the upper limit of the normal range with ischemic symptoms or signs during follow-up after discharge. Peri-procedural MI was defined as peak elevations of the creatine kinase-myocardial band of over 10-fold above the upper reference limit within 48 h after the procedure [14]. RR was defined as any

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percutaneous or surgical revascularization in any vessel. TVR was defined as any percutaneous or surgical revascularization of the treated vessel. TLR was defined as any percutaneous or surgical revascularization of the treated lesion. CVA was defined as a focal neurological deficit of central origin lasting >24 h, confirmed by a neurologist and imaging. All clinical events were confirmed by source documentation collected at each hospital and centrally adjudicated by an independent group of clinicians unaware of the revascularization type.

2.4. Statistical Analyses

Continuous variables were presented as mean ± standard deviation (SD) and compared using the Student t-test or Mann Whitney U test. Categorical variables were presented as counts (percentages) and compared using the chi-square or Fisher exact test, as appropriate. The primary outcomes were analyzed using multivariable logistic regression models. Odds ratios with 95% confidence intervals were calculated to estimate the independent association between CKD status and the occurrence of each outcome after adjustment for predefined covariates. Event rates were estimated on Kaplan–Meier estimates in time-to-first-event analyses, and they were compared using the log-rank test. A univariate Cox regression analysis was performed to obtain the hazard ratio (HR) for clinical outcomes. Then, to find out the independent predictors with the clinical outcomes, a multivariate Cox proportional hazard regression model was performed using important clinical covariates including clinically relevant variables and statistically significant variables with a p-value < 0.05 by univariate analysis. For subgroup analysis, Cox regression analysis was performed and visualized using forest plots. All statistical analyses were conducted using SAS 9.4 (SAS Institute Inc., Cary, NC, USA), and a p-value < 0.05 was considered statistically significant.

3. Results

3.1. Baseline Characteristics

Baseline characteristics are summarized in Tables 1-3. A total of 652 patients who underwent ROTA were included in the analysis, of whom 66 (10.1%) were classified as having CKD (eGFR < 60 mL/min/1.73 m²) and 586 (89.9%) as non-CKD. Diabetes mellitus (DM) was higher in the CKD group (78.8% vs. 54.2%, $p < 0.001$). Regarding cardiovascular history, prior peripheral vascular disease were more frequent in the CKD group (21.2% vs. 6.5%, $p < 0.001$), whereas prior myocardial infarction and prior coronary artery bypass grafting showed no statistically significant differences between groups. Clinical diagnosis of STEMI/NSTEMI was more included in CKD group (39.4% vs. 26.0%, $p = 0.021$). Patients with CKD had a lower pre-procedural left ventricular ejection fraction compared with non-CKD patients (48.4 ± 14.0 vs. 54.0 ± 12.9, $p = 0.001$). Other vessel revascularization was performed more frequently in CKD group (54.6% vs. 41.9, $p = 0.049$). Lastly, contrast volume was significantly less used in CKD group (189.4 ± 95.6 vs. 214.1 ± 91.9, $p = 0.044$).

Table 1. Baseline characteristics.

	non CKD	CKD	
	n = 586	n = 66	*p*-value
Age	72.0±9.6	74.3±8.3	0.069
Sex	351(60.0)	38(57.6)	0.703
BMI	24.2±3.9	24.1±3.6	0.874
Systolic BP	131.9±24.1	133.5±18.2	0.581
Diastolic BP	74.8±12.5	74.3±10.9	0.737
Smoking	111(19.0)	7(10.6)	0.094
HTN	446(76.2)	57(86.4)	0.063
DM	317(54.2)	52(78.8)	<0.001

 doi:10.20944/preprints202602.1883.v1

Dyslipidemia	301(51.5)	33(50.0)	0.823
CKD (no dialysis)	0(0.0)	66(100.0)	<0.001
LV Ejection Fraction	54.0±12.9	48.4±14.0	0.001
Medical History			
PCI	136(23.3)	19(28.8)	0.317
CABG	24(4.1)	4(6.1)	0.515
MI	64(10.9)	9(13.6)	0.511
CVA	93(15.9)	10(15.2)	0.875
PVD	38(6.5)	14(21.2)	<0.001
Chronic lung disease	34(5.8)	8(12.1)	0.061
Heart failure	73(12.5)	12(18.2)	0.192
Atrial fibrillation	42(7.2)	10(15.2)	0.024
Clinical_diagnosis (STEMI/NSTEMI)	152(26.0)	26(39.4)	0.021

CKD, chronic kidney disease; BMI, body mass index; BP, blood pressure; HTN, hypertension; DM, diabetes mellitus; CKD, chronic kidney disease; LV, left ventricle; PCI, percutaneous coronary intervention; CABG, coronary artery bypass graft; MI, myocardial infarction; CVA, cerebrovascular accident; PVD, peripheral vascular disease; NSTEMI, non-ST segmental elevation myocardial infarction; STEMI, ST segmental elevation myocardial infarction.

Table 2. Laboratory analyses and medications.

	non CKD	CKD	
	n = 586	n = 66	*p*-value
Labaratory analyses			
Hb	12.6±2.3	11.1±1.8	<0.001
Platelet	220.8±65.5	219.8±93.0	0.910
Triglyceride	119.0±74.2	123.1±66.0	0.686
Total cholesterol	146.5±40.1	134.6±35.3	0.030
LDL cholesterol	85.2±39.3	75.6±31.9	0.079
HDL cholesterol	47.1±14.7	40.2±14.3	<0.001
Hs-CRP	2.6±10.6	2.5±6.8	0.971
HbA1c	6.7±1.6	7.4±1.5	0.002
Medication			
NOAC	21(3.6)	5(7.6)	0.171
DAPT	559(95.6)	61(92.4)	0.230
Aspirin	571(97.6)	62(93.9)	0.099
P2Y12_inhibitor	574(98.1)	66(100.0)	0.614
Cilostazol	73(12.5)	6(9.1)	0.424
Beta-blocker	392(67.0)	45(68.2)	0.848
ACEi or ARB	377(64.4)	39(59.1)	0.391
Statin	552(94.4)	62(93.9)	0.782

Hb, hemoglobin; LDL, low density lipoprotein; HDL, high density lipoprotein; Hs-CRP, high sensitivity C-reactive protein; NOAC, new oral anticoagulant; DAPT, dual antiplatelet therapy; ACEI/ARB, angiotensin converting enzyme inhibitor / angiotensin II receptor blocker.

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Table 3. Lesion and procedural characteristics.

	non CKD	CKD	
	n = 586	n = 66	*p*-value
Lesion Classification(B2/C)	549(93.9)	62(93.9)	>0.999
Vessel disease			
1VD	124(21.2)	9(13.6)	0.312
2VD	187(32.0)	25(37.9)	
3VD	274(46.8)	32(48.5)	
Multivessel disease (MVD)	461(78.8)	57(86.4)	0.149
LM disease	86(14.7)	11(16.7)	0.671
IVUS	303(51.8)	37(56.1)	0.511
Direct	211(36.1)	24(36.4)	0.962
Other vessel revascularization	245(41.9)	36(54.6)	0.049
Procedural approach			
Radial	291(49.7)	31(47.0)	0.669
Femoral	294(50.3)	35(53.0)	
Procedure success	555(94.9)	64(97.0)	0.762
Technical success	562(96.1)	66(100.0)	0.155
Contrast (mL)	214.1±91.9	189.4±95.6	0.044
Procedural time	78.0±51.4	86.5±56.4	0.211
Radiation dose	3,811.6±3,643.3	4,354.1±2,684.3	0.574
Rotational atherectomy			
Size of burr (start)	1.5±0.2	1.5±0.2	0.951
Size of burr (max)	1.5±0.6	1.5±0.2	0.795
Number of burr	1.2±0.4	1.2±0.5	0.991
Stent			
Stent diameter	3.0±0.4	3.0±0.3	0.384
Total number of stent	2.5±1.3	2.6±1.3	0.377
Total length of stent	69.6±37.7	74.3±38.4	0.343
Peri-procedural complication			
Coronary dissection	192(32.8)	19(28.8)	0.507
Coronary perforation	21(3.6)	2(3.0)	>0.999
Urgent intervention for tamponade	3(0.5)	0(0.0)	>0.999
Temporary pacemaker	39(6.7)	6(9.1)	0.442
Periprocedural MI	54(9.2)	10(15.2)	0.126
Inhospital outcome			
Inhospital death	18(3.1)	2(3.0)	>0.999
Inhospital CVA	2(0.3)	0(0.0)	>0.999
Urgent CABG	2(0.3)	0(0.0)	>0.999
Urgent PCI	5(0.9)	1(1.5)	0.475

LM, left main; IVUS, intravascular ultrasound sonography; MI, myocardial infarction; CVA, cerebrovascular accident; CABG, coronary artery bypass graft; PCI, percutaneous coronary intervention.

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3.2. Primary Outcomes

Primary in-hospital outcomes are presented in Table 4. The composite outcome of CIN or in-hospital bleeding occurred more frequently in the CKD group compared with the non-CKD group (16.7% vs. 5.1%, $p = 0.001$). After adjustment for age, sex, smoking, hypertension, diabetes, clinical diagnosis of STEMI/NSTEMI, MVD, other-vessel revascularization, procedural approach, and pre-procedural LV ejection fraction, CKD remained independently associated with the composite outcome (adjusted OR, 3.02; 95% CI, 1.36-6.69; $p = 0.006$). CIN occurred significantly more often in patients with CKD (15.2% vs. 1.7%, $p < 0.001$). In multivariable analysis using the same adjustment model, CKD was an independent predictor of CIN (adjusted OR, 7.18; 95% CI, 2.32-22.21, $p < 0.001$). In-hospital bleeding alone did not differ significantly between the non-CKD and CKD groups (3.0% vs. 3.6%, $p > 0.999$).

Table 4. Clinical outcomes (Primary and Secondary outcomes).

	non CKD	CKD		univariate				multivariate			
Primary outcome	n = 586	n = 66	*p*-value	OR	95%CI (lower – upper)		p-value	OR	95%CI (lower – upper)		p-value
Composite	30(5.1)	11(16.7)	0.001	3.700	1.758	7.789	<0.001	3.019	1.363	6.685	0.006
CIN	10(1.7)	10(15.2)	<0.001	10.269	4.099	25.727	<0.001	7.184	2.324	22.210	<0.001
In-hospital bleeding	21(3.6)	2(3.0)	>0.999	0.839	0.192	3.662	0.816	0.779	0.170	3.565	0.747
Secondary outcome (3-year outcome)	n = 586	n = 66	*p*-value	Log-rank p-value	HR	95%CI (lower – upper)		p-value	HR	95%CI (lower – upper)		p-value
TVF	63(10.8)	9(13.6)	0.481	0.349	1.395	0.693	2.807	0.351	1.317	0.639	2.715	0.456
AD	55(9.4)	9(13.6)	0.273	0.210	1.565	0.772	3.171	0.214	1.326	0.641	2.743	0.447
CD	41(7.0)	6(9.1)	0.461	0.468	1.373	0.582	3.236	0.469	1.260	0.522	3.043	0.607
MI	12(2.1)	4(6.1)	0.069	0.024	3.417	1.097	10.645	0.034	3.975	1.155	13.683	0.029
TVMI	7(1.2)	2(3.0)	0.229	0.191	2.735	0.568	13.175	0.210	3.904	0.616	24.735	0.148
RR	50(8.6)	6(9.1)	0.881	0.723	1.165	0.499	2.719	0.724	0.933	0.390	2.233	0.876
TVR	35(6.0)	6(9.1)	0.291	0.209	1.733	0.727	4.128	0.215	1.459	0.590	3.611	0.414
TLR	31(5.3)	4(6.1)	0.772	0.632	1.289	0.454	3.660	0.633	1.091	0.370	3.216	0.875
NLR	25(4.3)	3(4.6)	0.756	0.756	1.209	0.365	4.010	0.756	1.149	0.333	3.971	0.826
CVA	8(1.4)	3(4.6)	0.091	0.054	3.419	0.906	12.913	0.070	3.296	0.820	13.250	0.093
ST	5(0.9)	1(1.5)	0.475	0.603	1.756	0.205	15.028	0.607	1.731	0.179	16.745	0.635

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Total bleeding	23(3.9)	7(10.6)	0.025	0.008	2.983	1.275	6.978	0.012	3.392	1.374	8.370	0.008
Minor bleeding	9(1.5)	5(7.6)	0.009	<0.001	5.776	1.918	17.396	0.002	7.798	2.282	26.646	0.001
Major bleeding	9(1.5)	1(1.5)	>0.999	0.940	1.082	0.137	8.577	0.940	1.336	0.151	11.854	0.795

CIN, contrast induced nephropathy; TVF, target-vessel failure; AD, all-cause death; CD, cardiac death; MI, myocardial infarction; TVMI, target-vessel spontaneous MI; RR, repeat revascularization; TVR, target-vessel revascularization; TLR, target-lesion revascularization; NLR, neutrophil to lymphocyte ratio; CVA, cerebrovascular accident; ST, stent thrombosis. TVF is defined as composite of CD, TVMI, TVR.

3.3. Secondary Outcomes

3-year outcomes are summarized in Table 4. Median follow-up duration was 1.5 (IQR, 0.6-2.9) years. Myocardial infarction occurred more frequently in CKD patients (6.1% vs. 2.1%, log-rank p = 0.024) and CKD was independently associated with an increased risk of myocardial infarction in multivariable analysis (adjusted HR, 4.00; 95% CI, 1.16-13.68, log-rank p = 0.029). However, TVF, CD, TVMI, TVR, CVA, and ST were more common in the CKD group, though this did not reach statistical significance. Total bleeding events during follow-up occurred markedly more often in CKD patients (10.6% vs. 3.9%, log-rank p = 0.008), with CKD showing a strong independent association after multivariable adjustment (adjusted HR, 3.39; 95% CI, 1.37-8.37, log-rank p = 0.008). This association was primarily driven by minor bleeding events (adjusted HR, 7.80; 95% CI, 2.28-26.65, log-rank p = 0.001), whereas major bleeding did not differ significantly between two groups. Kaplan-Meier curves representing 3-year outcomes of TVF, CD, MI, TVMI, TVR, and total bleeding are shown in Figure 2.

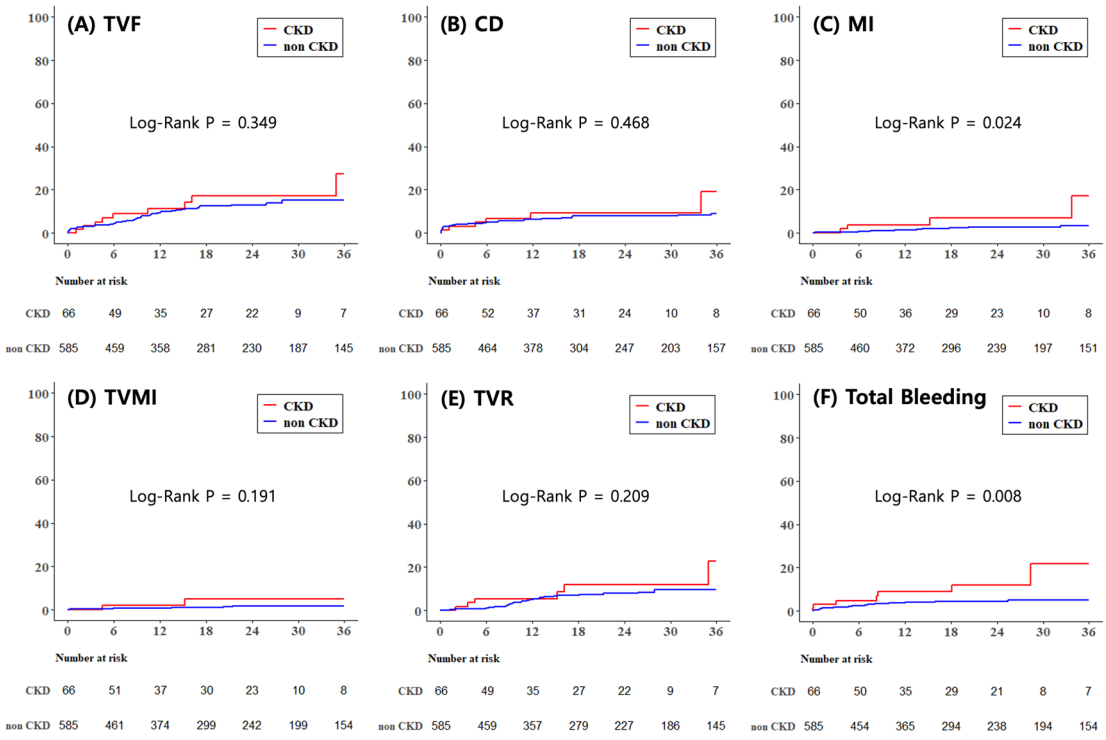

Figure 2. Kaplan–Meier curves for secondary outcomes during the follow-up period. (A) TVF, target vessel failure; (B) CD, cardiac death; (C) MI, myocardial infarction; (D) TVMI, target vessel myocardial infarction; (E) TVR, target vessel revascularization; (F) Total bleeding.

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3.4. Subgroup Analyses

Subgroup analyses were performed to evaluate whether the association between CKD and the composite primary outcome (CIN or in-hospital bleeding) differed across clinically relevant subgroups, including age (< 70 vs. ≥ 70 years), sex, presence of MVD, other-vessel revascularization, use of a direct RA strategy, and pre-procedural LVEF (Figure 3). CKD was consistently associated with an increased risk of the composite primary outcome across most subgroups. The magnitude of risk associated with CKD was particularly pronounced in patients aged ≥ 70 years (OR, 3.31; 95% CI, 1.44-7.62, $p = 0.005$) , male patients (OR, 4.51; 95% CI, 1.62-12.55, $p = 0.004$), those with MVD (OR, 4.46; 95% CI, 1.98-10.03, $p < 0.001$), patients undergoing other-vessel revascularization (OR, 5.67; 95% CI, 2.01-16.05, $p = 0.001$),those with direct RA approach (OR, 5.10; 95% CI, 2.14-12.16, $p < 0.001$), and those with pre-procedural LVEF under 50% (OR, 5.20; 95% CI, 1.92-14.13, $p = 0.001$) indicating that the adverse effect of CKD on the primary outcome was consistent across these subgroups.

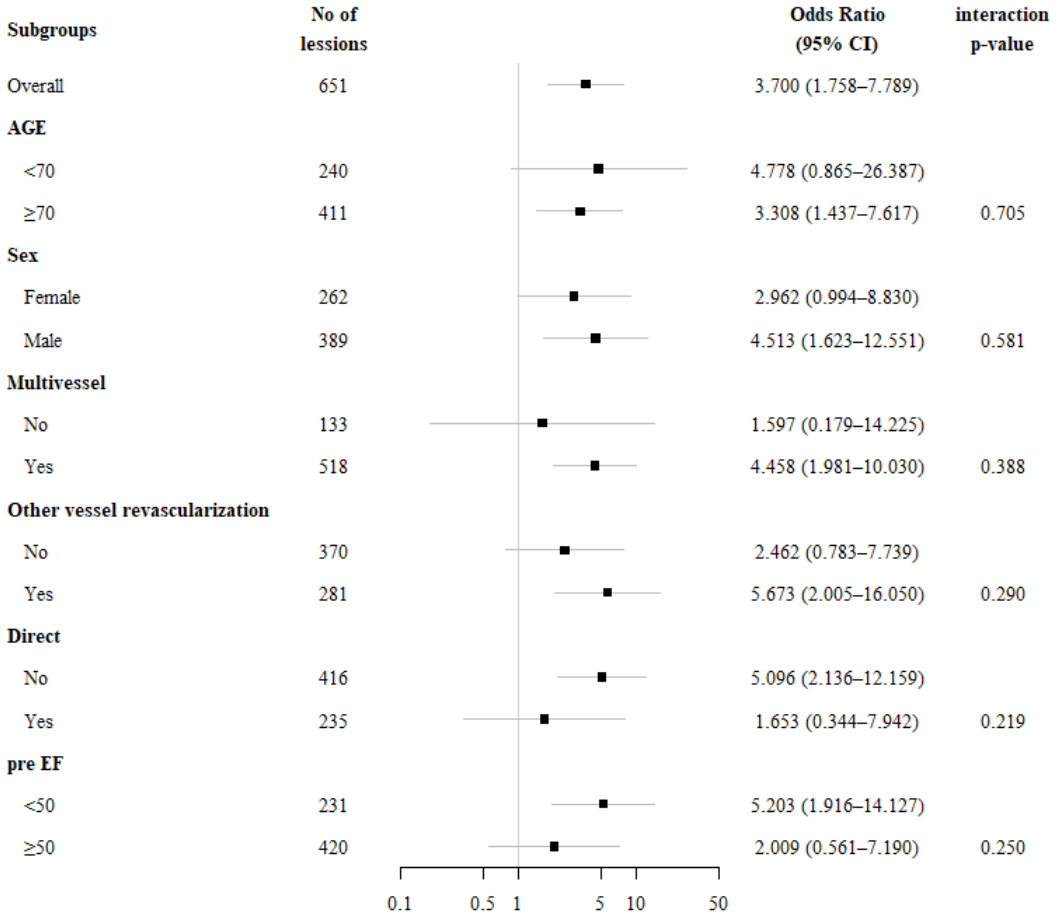

Figure 3. Forest plot for subgroup analysis comparing the incidence of composite primary outcome between CKD and non-CKD groups.

4. Discussion

In this retrospective cohort study, CKD was independently associated with a significantly higher risk of CIN in patients undergoing ROTA. Subgroup analyses demonstrated that this association remained consistent across most clinically relevant strata, including age ≥70 years, sex, MVD, other-vessel revascularization, direct ROTA strategy, and pre-procedural ejection fraction. With respect to 3-year outcomes, myocardial infarction and total bleeding events occurred significantly more frequently in the CKD group, whereas major adverse cardiovascular events (MACE), cerebrovascular

 doi:10.20944/preprints202602.1883.v1

accident (CVA), and stent thrombosis (ST) were numerically higher but did not reach statistical significance.

Coronary artery calcification in patients with CKD represents a critical determinant of both treatment strategy and long-term prognosis. Patients with CKD not only have a higher burden of coronary artery disease but also exhibit more advanced vascular calcification, often necessitating coronary intervention. Such interventions inherently require invasive procedures involving antithrombotic therapy with potential bleeding risk, as well as the unavoidable use of iodinated contrast agents, a well-established precipitant of acute kidney injury. In this context, CKD embodies a clinical phenotype intrinsically vulnerable to coronary procedures.

For patients with severe coronary calcification, preparation with RA is often required beyond conventional balloon angioplasty. Several studies have evaluated the clinical outcomes of RA-treated populations. Although not stratifying patients specifically by CKD status, prior investigations have identified low preoperative mean arterial pressure as an important predictor of CIN in RA-treated patients [15]. Moreover, studies examining plaque-modification strategies have shown that patients requiring additional intervention for severe calcification experience higher rates of CIN compared with those undergoing less complex PCI [16]. Conversely, one study comparing RA and non-RA groups reported reduced MACE rates in the RA group during in-hospital and 2-year follow-up, suggesting potential procedural benefits in selected patients [17].

It is well established that CKD patients experience higher rates of CIN and bleeding complications following contrast-based invasive procedures compared with individuals with preserved renal function [18,19]. Several pathophysiological mechanisms may explain these observations. CKD patients are more susceptible to contrast-induced renal injury even at comparable contrast volumes due to impaired renal reserve and altered microvascular autoregulation. Although RA-treated patients are generally expected to require higher contrast volumes than non-RA PCI cases, the contrast volume used in our study was much lower than that reported in prior ROTA versus non-ROTA comparisons [20]. Notably, even with relatively lower contrast exposure in the CKD group, the risk of CIN remained substantially higher than in the non-CKD group, reaffirming CKD itself as a major independent determinant of CIN.

Regarding bleeding risk, despite the interventional complexity associated with RA, in-hospital bleeding did not significantly differ between CKD and non-CKD groups. Although total bleeding events during 3-year follow-up were significantly higher in CKD patients, this difference was primarily driven by minor bleeding events. This observation may be partly explained by the absence of significant differences in vascular access site distribution (radial vs. femoral) and other peri-procedural factors, including procedural time, between the two groups. Nevertheless, given the significantly increased long-term bleeding risk observed in CKD patients, individualized therapeutic planning that incorporates bleeding propensity remains important. The biological basis for increased bleeding in CKD is well recognized. CKD is associated with platelet dysfunction, endothelial abnormalities, and uremia-related coagulopathy, all contributing to an elevated bleeding tendency [21]. Current guidelines also recognize CKD as a major determinant of bleeding risk and recommend consideration of early de-escalation or shortening of dual antiplatelet therapy when clinically appropriate [22–24]. Furthermore, the preferential use of polymer-free or bioabsorbable-polymer stents may facilitate shorter durations of dual antiplatelet therapy without compromising ischemic protection in this high-risk population [25].

In subgroup analyses, although post-procedural bleeding complications are generally more common in women [26], the magnitude of risk associated with CKD was higher in male patients in our cohort. In patients with MVD, longer procedural times, higher contrast volumes, and repeated intra-procedural anticoagulation may contribute to increased bleeding and renal stress. Similarly, in cases where direct RA was not performed, the need for additional lesion preparation may increase procedural burden and contrast exposure, potentially explaining the observed increase in risk.

One prior study evaluating ROTA in patients with advanced CKD, including those with end-stage renal disease on dialysis, reported no significant difference in acute procedural complications

 doi:10.20944/preprints202602.1883.v1

according to renal function and emphasized the feasibility of RA in advanced CKD [27]. Consistent with our findings, while CKD itself confers an unavoidable increase in acute procedural risk—particularly for CIN—long-term MACE did not significantly differ after 3 years. This suggests that with meticulous peri-procedural management and careful complication control, RA remains a viable therapeutic option in CKD patients.

Taken together, our findings support a more individualized interventional strategy in CKD patients undergoing RA. In those with multivessel disease, early consideration of staged PCI may help reduce contrast burden. In elderly CKD patients, thorough pre-procedural imaging assessment and strategic planning—including contrast minimization and the use of a direct ROTA approach to reduce procedural time—may prevent renal injury. Although acute complications may not be entirely avoidable, appropriate early management may allow CKD patients to achieve long-term outcomes comparable to those without CKD, supporting the feasibility of RA in this high-risk population.

This study has several limitations. First, the retrospective study design precludes causal inference and may allow residual confounding; however, we applied multivariable adjustment and subgroup analyses to strengthen the validity of our findings. Second, though the sample size was not large, the cohort consisted exclusively of patients undergoing RA, making the study adequately powered within this high-risk population. Third, patients with end-stage renal disease on dialysis were excluded because CIN was a primary endpoint, which limits generalizability but was necessary to accurately evaluate contrast-related renal injury. Finally, procedural strategies and contrast use were operator-dependent, and minor bleeding events may have been underreported; though, key clinical variables were incorporated into adjusted analyses.

In patients undergoing RA for severely calcified coronary lesions, CKD independently increases the risk of CIN and long-term bleeding, as well as ischemic events such as MI. However, comparable 3-year major adverse cardiovascular outcomes suggest that RA remains a feasible strategy in CKD when early complications are carefully managed. These findings emphasize the need for contrast-minimizing procedural strategies, thoughtful lesion preparation planning, and tailored antiplatelet regimens in CKD patients undergoing complex coronary intervention.

5. Conclusions

In patients undergoing rotational atherectomy for severely calcified coronary lesions, CKD is a robust independent risk factor for CIN and long-term bleeding complications, particularly minor bleeding. However, the long-term efficacy outcomes, including target vessel failure and cardiac death, were comparable between CKD and non-CKD groups. These findings support the feasibility of rotational atherectomy in CKD patients, emphasizing the importance of optimized peri-procedural strategies to minimize contrast burden and bleeding complications.

Author Contributions: Conceptualization, J.L.,S.-S.C., J.J., K.K., Y.K., K.L. and S.-H.H.; data curation, S.-R.L., W.-Y.J., J.-H.L. (Jang-Hoon Lee), I.-J.C., K.-H.Y., S.-W.L., J.-H.L. (Jae-Hwan Lee), H.-J.L., S. H.H., S.-N.L. and K.L.; formal analysis, S.-H.H.; investigation, J.L., Y.K. and J.J.; methodology, S.-H.H. and K.-D.Y.; project administration, S.-H.H., K.L. and J.J.; resources, K.-W.M., K.-D.Y. and D.M.; software, J.L.,S.-S.C.; supervision, K.-D.Y., S.-H.H. and K.L.; validation, S.-H.H., K.K. and J.J.; visualization, J.L., S. S.C., K.K. and J.J.; writing—original draft preparation, J.L., S.-S.C., J.J. and S.-H.H.; writing—review and editing, S.-H.H., K.-D.Y., S.-R.L. and K.L. Each author has reviewed and consented to the final manuscript as submitted for publication. All authors have read and agreed to the published version of the manuscript.

Funding: This study received no external funding.

Institutional Review Board Statement: The study was executed following the guidelines of the Declaration of Helsinki and authorized by the Institutional Review Board (IRB) of Daejeon St. Mary's Hospital (approval code: DC19REDI0066, approval date: 30 July 2019).

Informed Consent Statement: All participants in the study provided their informed consent.

 doi:10.20944/preprints202602.1883.v1

Data Availability Statement: The data included in this manuscript cannot be shared publicly, due to the need to protect the privacy of the included subjects. Data may be shared upon reasonable request to the corresponding author.

Conflicts of Interest: The authors declare no conflict of interest.

Abbreviations

The following abbreviations are used in this manuscript:

CABG	coronary artery bypass graft
CD	cardiac death
CIN	contrast induced nephropathy
CKD	chronic kidney disease
CVA	cerebrovascular accident
LVEF	left ventricle ejection fraction
MVD	multivessel disease
PCI	percutaneous coronary intervention
RA	rotational atherectomy
ST	stent thrombosis
RR	repeat revascularization
TVF	target vessel failure
TVMI	target-vessel spontaneous myocardial infarction
TVR	target-vessel revascularization

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