In-Hospital Outcomes of Chronic Total Occlusion Percutaneous Coronary Intervention in Patients With Chronic Kidney Disease

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ABSTRACT: Objectives. The effect of chronic kidney disease (CKD) on in-hospital outcomes of chronic total occlusion (CTO) percutaneous coronary intervention (PCI) has received limited study. **Methods.** We evaluated the prevalence of CKD and its impact on CTO-PCI outcomes in 1979 patients who underwent 2040 procedures between 2012 and 2017 at 18 centers. CKD was defined as preprocedural estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m^2. **Results.** Compared with patients without CKD (n = 1444; 73%), patients with CKD (n = 535; 27%) had more comorbidities (hypertension, diabetes mellitus, heart failure, peripheral arterial disease, prior myocardial infarction, PCI, coronary artery bypass graft surgery, and stroke), and more severe calcification and proximal vessel tortuosity. Patients with and without CKD had similar technical success rates (84% vs 86%; *P*=.49) and procedural success rates (83% vs 84%; *P*=.44). Patients with CKD had higher in-hospital mortality rate (1.9% vs 0.3%; *P*<.001) and in-hospital major adverse cardiovascular event (MACE) rate (4.3% vs 2.2%; *P*<.01). In-hospital mortality and MACE rates increased with decreasing eGFR levels (*P*=.03). In multivariate analysis, an independent association was observed between CKD and in-hospital mortality (adjusted odd ratio, 4.4; 95% confidence interval, 1.2-16.0; *P*=.02), but not overall MACE (adjusted odds ratio, 1.4; 95% confidence interval, 0.8-2.7; *P*=.28). **Conclusions.** CKD is common among patients undergoing CTO-PCI. High success rates can be achieved in patients with decreased glomerular filtration rate, but CKD may be associated with higher in-hospital mortality.

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Chronic kidney disease (CKD) has been associated with worse in-hospital and long-term outcomes after percutaneous coronary intervention (PCI).[1-7] However, little information exists regarding the effect of CKD on the outcomes of chronic total occlusion (CTO)-PCI.[7] Patients with CKD often have multiple comorbidities and increased coronary lesion complexity that could adversely affect PCI outcomes. Accordingly, we examined a contemporary multicenter CTO-PCI registry to examine the impact of CKD on the safety and efficacy of CTO-PCI.

Methods

We examined the clinical, angiographic, and procedural characteristics of 1979 patients who underwent 2040 CTO-PCIs and were enrolled in the PROGRESS CTO (Prospective Global Registry for the Study of Chronic Total Occlusion Intervention; NCT02061436) registry between May 2012 and November 2017 at 18 centers in the United States, Europe, and Russia (Appendix 1). The study was approved by the institutional review board of each site. Some centers only enrolled patients during part of the study period due to participation in other studies.

Renal function assessment. The estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula and the serum creatinine measurement obtained prior to and temporally closest to the index procedure.[8] Patient classification was based upon the Kidney Disease: Improving Global Outcomes (KDOKI)[9] and National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF-KDOQI)[10] guidelines: normal or high (G1), ≥90 mL/min/1.73 m^2; mildly decreased (G2), 60-89 mL/min/1.73 m^2; mildly to moderately decreased (G3a), 45-59 mL/min/1.73 m^2; moderately to severely decreased (G3b), 30-44 mL/min/1.73 m^2; severely decreased (G4), <29 mL/min/1.73 m^2; or kidney failure (G5), <29 mL/min/1.73 m^2. *CKD* was defined as eGFR <60 mL/min/1.73 m^2 (composite of the G3a, G3b, G4, and G5 groups); eGFR ≥60 mL/min/1.73 m^2 (composite of the G1

Table 1. Clinical characteristics of the study patients, classified according to preprocedural estimated glomerular filtration rate.

Clinical Characteristics	Overall (n = 1979)	Preprocedural eGFR (mL/min/1.73 m²)		*P*-Value
		<60 (n = 535)	≥60 (n = 1444)	
Age (years)	65.0 ± 10.0	70.2 ± 8.6	63.1 ± 8.6	<.001
Male gender	86%	82%	87%	<.01
Black race	7%	8%	6%	.17
BMI (kg/m²)	30.5 ± 6.1	30.7 ± 6.4	30.5 ± 6.0	.45
Baseline creatinine (mg/dL)	1.2 ± 0.9	1.8 ± 1.5	1.0 ± 0.2	<.001
eGFR CKD-EPI formula (mL/min/1.73 m²)	72.8 ± 21.9	45.1 ± 13.6	83.0 ± 14.2	<.001
Dialysis	2%	8%	0%	<.001
Current smoker	23%	16%	26%	<.001
Diabetes	44%	56%	39%	<.001
Dyslipidemia	92%	92%	93%	.68
Hypertension	90%	94%	88%	<.001
Prior MI	49%	47%	56%	<.01
Heart failure	31%	42%	27%	<.001
Prior PCI	60%	63%	58%	.05
Prior CABG	32%	40%	29%	<.001
Prior CVD	13%	15%	11%	.02
PAD	15%	22%	13%	<.001
Ad hoc CTO-PCI	14%	13%	14%	.61
Chronic lung disease	14%	14%	17%	.14
Left ventricular EF (%)	50.3 ± 13.4	47.8 ± 13.8	51.2 ± 13.1	<.001
CAD presentation				<.001
ACS	26%	32%	24%	
Stable angina	65%	58%	67%	
Other	10%	11%	9%	
CCS angina classification				.52
Class ≤1	10%	11%	10%	
Class ≥2	90%	89%	90%	
Antianginal medication used				
Long-acting nitrates	39%	42%	38%	.06
Beta-blockers	84%	85%	83%	.41
Calcium-channel blockers	24%	28%	23%	.03
Ranolazine	14%	17%	14%	.09

Data provided as percentage or mean ± standard deviation.
ACS = acute coronary syndrome; BMI = body mass index; CABG = coronary artery bypass graft; CKD-EPI = Chronic Kidney Disease Epidemiology Collaboration; CAD = coronary artery disease; CTO = chronic total occlusion; CVD = cerebrovascular disease; EF = ejection fraction; eGFR = estimated glomerular filtration rate; PCI = percutaneous coronary intervention; PAD = peripheral artery disease; MI = myocardial infarction.

and G2 groups) was considered normal. Patients undergoing dialysis were classified in the lowest eGFR group for all analyses. All patients had at least one creatinine measurement performed within 6 months prior to the index procedure.

Definitions. *Coronary CTOs* were defined as coronary lesions with Thrombolysis in Myocardial Infarction (TIMI) grade flow 0 of at least 3-month duration. *Estimation of the occlusion duration* was based on first onset of anginal symptoms, prior history of myocardial infarction in the target-vessel territory, or comparison with a prior angiogram. *Calcification* was assessed by angiography as mild (spots), moderate (involving ≤50% of the reference lesion diameter), or severe (involving >50% of the reference lesion diameter). *Moderate proximal vessel tortuosity* was defined as the presence of at least 2 bends >70° or 1 bend >90° and *severe tortuosity* as 2 bends >90° or 1 bend >120° in the CTO vessel. *Interventional collaterals* were defined as collaterals deemed amenable to crossing by a guidewire and a microcatheter by the operator. The *J-CTO score* was calculated as described by Morino et al,[11] the *PROGRESS CTO score* as described by Christopoulos et al,[12] and the *PROGRESS CTO Complication score* as described by Danek et al.[13] *Technical success of CTO-PCI* was defined as successful CTO revascularization with achievement of <30% residual diameter stenosis within the treated segment and restoration of TIMI grade 3 antegrade flow. *Procedural success* was defined as achievement of technical success with no in-hospital major adverse cardiac event (MACE). *In-hospital MACE* included any of the following adverse events prior to hospital discharge: death, myocardial infarction (MI), recurrent symptoms requiring urgent repeat target-vessel revascularization with PCI or coronary artery bypass graft (CABG) surgery, tamponade requiring either pericardiocentesis or surgery, and stroke. *Periprocedural and late in-hospital MI* were defined according to the Third Universal Definition

Table 2. Angiographic characteristics of the study lesions, classified according to preprocedural estimated glomerular filtration rate.

Clinical Characteristics	Overall (n = 2040)	Preprocedural eGFR (mL/min/1.73 m²)		*P*-Value
		<60 (n = 556)	≥60 (n = 1484)	
Target vessel				.62
RCA	54%	53%	55%	
LAD	24%	24%	24%	
LCX	21%	23%	20%	
Other	1%	1%	1%	
CTO length (mm)	32.7 ± 23.3	33.4 ± 23.3	32.4 ± 23.4	.41
Vessel diameter (mm)	2.8 ± 0.5	2.8 ± 0.5	2.8 ± 0.5	.88
Proximal cap ambiguity	33%	34%	33%	.58
Blunt/no stump	53%	51%	53%	.42
Bifurcation at distal cap	33%	35%	32%	.37
Diseased distal landing zone	32%	36%	30%	.02
Interventional collaterals	57%	56%	57%	.48
Moderate/severe calcification	49%	59%	46%	<.001
Moderate/severe tortuosity	33%	37%	32%	.02
In-stent restenosis	16%	16%	16%	.89
Previously failed CTO-PCI	20%	20%	20%	.65
J-CTO score	2.5 ± 1.3	2.6 ± 1.3	2.4 ± 1.3	.01
PROGRESS CTO score	1.3 ± 1.0	1.4 ± 1.1	1.3 ± 1.0	.03
PROGRESS CTO complication score	3.0 ± 2.0	3.7 ± 1.8	2.8 ± 2.0	<.001

Data provided as percentage or mean ± standard deviation.
CTO = chronic total occlusion; J = Japan; eGFR = estimated glomerular filtration rate; LAD = left anterior descending artery; LCX = left circumflex artery; PCI = percutaneous coronary intervention; PROGRESS = Prospective Global Registry of Chronic Total Occlusion Interventions; RCA = right coronary artery.

of Myocardial Infarction.[14] *Bleeding* was defined according to the National Cardiovascular Data Registry CathPCI database, and included suspected/confirmed bleeding occurring within 72 hours of the procedure and associated with any of the following: (1) hemoglobin drop of ≥3 g/dL; (2) transfusion with whole blood or packed red blood cells; or (3) procedural intervention/surgery at bleeding site to reverse or correct the bleed. *Procedure time* was calculated from administration of local anesthetic for vascular access to removal of the last catheter.

Statistical analysis. Categorical variables were described using percentages and compared between groups using Pearson's Chi-squared test or the Cochran-Armitage test for trend. Continuous variables were described as mean ± standard deviation or median (interquartile range [IQR]) and compared using the Student's t-test or Wilcoxon rank-sum test. Multivariable logistic regression was used to examine the association between eGFR/dialysis and MACE after adjusting for confounding variables selected on the grounds of (1) univariable association in the present study ($P<.10$); or (2) previously established links with MACE. Such variables included age, gender, body mass index, diabetes mellitus, hypertension, peripheral arterial disease, chronic lung disease, history of heart failure, MI, stroke, PCI or CABG, occlusion length, degree of lesion calcification, proximal cap morphology, and utilization of a retrograde approach. The group with the highest eGFR (≥90 mL/min/1.73 m²) was used as reference category for renal function. Stepwise backward elimination was used to form the final model. A two-sided *P*-value of <.05 was considered statistically significant. All statistical analyses were performed with JMP 13.0 (SAS Institute).

Results

The prevalence of CKD was 27% (535 of 1979 patients). Patients with CKD were more likely to be older and female, and to have hypertension, diabetes mellitus, heart failure, peripheral arterial disease, prior MI, PCI, CABG and stroke, and had lower left ventricular ejection fraction, but were less likely to be active smokers (Table 1). The most common CTO target vessel was the right coronary artery (54%), followed by the left anterior descending (24%) and the circumflex (21%) arteries. CKD patients were more likely to have lesions with moderate or severe calcification, proximal vessel tortuosity, and diseased distal target vessel. They also had more complex lesions with higher J-CTO scores (2.6 ± 1.3 vs 2.4 ± 1.3; $P=.01$) and PROGRESS CTO scores (1.4 ± 1.1 vs 1.3 ± 1.0; $P=.03$) (Table 2).

Overall technical and procedural rates were 85% and 84%, respectively, and were similar in patients with and without CKD (Figure 1). Crossing strategies (Table 3) were similar for patients with and without CKD; however, retrograde techniques were used more frequently as the initial crossing approach in the CKD group (17% vs 13%; $P=.04$). Left ventricular assist devices were used more commonly in CKD patients (8% vs 4%; $P<.001$), for either prophylactic cardiac support (5% vs 3%; $P<.01$) or emergency cardiac support (2% vs 1%; $P<.01$). Procedures performed in CKD patients had longer procedural times (134 min [IQR, 85-200 min] vs 119 min [IQR, 77-185 min]; $P<.01$) and fluoroscopy times (48.9 min [IQR, 27.3-79.2 min] vs 41.4

Table 3. Procedural characteristics classified according to preprocedural estimated glomerular filtration rate.

Clinical Characteristics	Overall (n = 2040)	Preprocedural eGFR (mL/min/1.73 m²)		*P*-Value
		<60 (n = 556)	≥60 (n = 1484)	
Dual injection	70%	71%	70%	.70
Crossing strategies utilized				
AWE	82%	79%	83%	.07
Retrograde	35%	35%	34%	.59
ADR	32%	32%	32%	.98
First crossing strategy				.04
AWE	77%	74%	79%	
Retrograde	14%	17%	13%	
ADR	8%	9%	8%	
Successful crossing strategy				.71
AWE	46%	45%	46%	
Retrograde	21%	20%	21%	
ADR	19%	21%	18%	
None	15%	14%	15%	
Access site				
Right femoral	78%	80%	77%	.26
Left femoral	50%	53%	48%	.05
Right radial	33%	30%	34%	.08
Left radial	18%	19%	17%	.22
Number of stents[a]	2.4 ± 1.2	2.5 ± 1.3	2.4 ± 1.1	.06
Non-CTO PCI	28%	29%	28%	.58
Procedure time (min)	123 (80-190)	134 (85-200)	119 (77-185)	<.01
Contrast volume (mL)	255 (190-350)	250 (180-340)	260 (200-350)	.02
Fluoroscopy time (min)	43.4 (25.8-72.5)	48.9 (27.3-79.2)	41.4 (25.3-70.3)	<.01
Patient AK dose (Gray)	3.1 (1.9-4.8)	3.2 (2.0-5.2)	3.0 (1.8-4.7)	.14
LVAD used	5%	8%	4%	<.001
Urgent	1%	2%	1%	<.01
Prophylactic	3%	5%	3%	<.01
LVAD type				
Intraaortic balloon pump	1%	1%	1%	.82
Impella 2.5[b]	1%	2%	1%	.046
Impella 5.0[b]	0%	0%	0%	–
Impella CP[b]	2%	3%	1%	<.001
Tandem Heart[c]	1%	2%	1%	<.01
Length of in-hospital stay (days)	1 (1-2)	1 (1-2)	1 (1-2)	.35

Data provided as percentage, mean ± standard deviation, or median (interquartile range).
[a]For procedures with technical success only; [b]Manufactured by Abiomed; [c]Manufactured by TandemLife.
AWE = antegrade wire escalation; ADR = antegrade dissection and re-entry; AK = air kerma; CTO = chronic total occlusion; eGFR = estimated glomerular filtration rate; LVAD = left ventricular assist device; PCI = percutaneous coronary intervention; PHP = percutaneous heart pump; VA ECMO = veno-arterial extracorporeal membrane oxygenation.

min [IQR, 25.3-70.3 min]; *P*<.01), similar air kerma patient radiation dose (3.2 Gray [IQR, 2.0-5.2 Gray] vs 3.0 Gray [1.8-4.7 Gray]; *P*=.14), and lower contrast volume (250 mL [IQR, 180-340 mL] vs 260 mL [IQR, 200-350 mL]; *P*<.02).

The overall in-hospital MACE rate was 2.7% (54 patients) (Table 4) and was higher among CKD patients (4.3% vs 2.2%; *P*<.01) (Figure 1), driven by higher in-hospital mortality (1.9% vs 0.3%; *P*<.001). We observed an inverse graded dose-response relationship between MACE and eGFR (*P* for trend =.03) (Figure 2); this was largely due to an increase in in-hospital mortality with worsening eGFR. Four patients died in the non-CKD group due to coronary perforation (1 hemothorax, 1 intramural cardiac hematoma, 2 coronary tamponade with subsequent cardiogenic shock), and 10 patients died in the CKD group (4 patients suffered cardiogenic shock after coronary perforation; 1 patient suffered cardiac arrest after new MI; 1 patient died from multiple organ dysfunction; 2 patients died from progressive cardiogenic shock despite use of a left ventricular assist device; 1 patient died from hemorrhagic stroke; and 1 patient died from hemorrhagic shock secondary to a vascular access complication). After adjustment for potential confounders, no independent association was found between renal function/dialysis status and overall MACE rate (Table 5); however, the association

Table 4. Procedural and in-hospital complications, classified according to preprocedural estimated glomerular filtration rate.

Clinical Characteristics	Overall (n = 1979)	Preprocedural eGFR (mL/min/1.73 m²)		P-Value
		<60 (n = 535)	≥60 (n = 1444)	
Major adverse cardiovascular events	2.7%	4.3%	2.2%	.01
Death	0.7%	1.9%	0.3%	<.001
Acute myocardial infarction	1.0%	1.1%	0.9%	.65
Stroke	0.3%	0.6%	0.2%	.20
Emergency re-PCI	0.2%	0.0%	0.3%	.22
Emergency surgery	0.2%	0.2%	0.1%	.81
Pericardiocentesis	1.0%	1.5%	0.8%	.14
Contrast induced nephropathy[a]	0.4%	1.6%	0.0%	<.01
Perforation	3.3%	3.7%	3.2%	.54
Bleeding	1.1%	2.2%	0.7%	<.01

Data presented as percentages. [a]Prospective data collection started in 2016.
PCI = percutaneous coronary intervention; GFR = estimated glomerular filtration rate.

Table 5. Crude and adjusted odds ratios for in-hospital major adverse cardiovascular events by different stratifications of renal function.

	Unadjusted		Adjusted	
	Odds ratio (95% CI)	P-Value	Odds ratio (95% CI)	P-Value
CKD vs no CKD				
≥60 mL/min/1.73 m²	1.0 (Ref)	–	1.0 (Ref)	–
<60 mL/min/1.73 m²	2.1 (1.2-3.5)	.01	1.4 (0.8, 2.7)	.28
According to eGFR stage				
≥90 mL/min/1.73 m²	1.0 (Ref)	–	1.0 (Ref)	–
60-90 mL/min/1.73 m²	2.1 (0.8-5.1)	.11	1.1 (0.4-2.8)	.92
30-60 mL/min/1.73 m²	3.2 (1.3-8.2)	.01	1.4 (0.5-4.2)	.54
<30 mL/min/1.73 m²	5.4 (1.6-18.3)	<.01	2.1 (0.5-8.6)	.30
According to dialysis status				
Non-dialyzed	1.0 (Ref)	–	1.0 (Ref)	-
Dialyzed	2.6 (0.8-8.7)	.12	3.1 (0.8-11.3)	.10
According to CV/eGFR ratio[a]				
≥60 ml/min/1.73m²	1.15 (0.97-1.37)	.13	0.97 (0.76-1.24)	.79
<60 ml/min/1.73m²	1.02 (1.00-1.04)	.10	1.02 (1.00-1.04)	.07

[a]Per 1 unit change.
CI = confidence interval; CKD = chronic kidney disease; CV = contrast volume; eGFR = estimated glomerular filtration rate.

between CKD and in-hospital mortality persisted after adjustment (odds ratio [OR], 4.4; 95% confidence interval [CI], 1.2-16.0; *P*=.02). Bleeding was observed in 22 patients (1.1%), as follows: access site in 14 patients, gastrointestinal in 2 patients, retroperitoneal in 4 patients, genito-urinary in 1 patient, and hemothorax in 1 patient. Bleeding was more frequent among patients with CKD (2.2% vs 0.7%; *P*<.01). Contrast-induced nephropathy (CIN) was diagnosed in 4 patients (0.4%) during the hospital stay, requiring new dialysis in 1 patient. Acute kidney injury only occurred in patients with decreased kidney function (1.6% vs 0.0%; *P*<.01).

Forty-five patients (2%) were undergoing dialysis at the time the procedure was performed. Compared with non-dialysis patients, those undergoing dialysis had numerically lower technical success (80% vs 85%; *P*=.31) and procedural success (78% vs 84%; *P*=.28) and numerically higher incidence of in-hospital MACE (6.7% vs 2.7%; *P*=.10).

Discussion

Our study provides novel insights into the acute outcomes of CKD patients undergoing CTO-PCI, as follows: (1) CKD was common in the CTO-PCI population, with approximately one-third of patients having an eGFR of <60 mL/min/1.73 m²; (2) CKD patients, even those with severely reduced renal function (eGFR<30 mL/min/1.73 m²) had similarly high technical success rates vs non-CKD patients; and (3) CKD patients had higher in-hospital mortality rates.

Several pathophysiological links have been established between renal dysfunction and progression of coronary artery disease, including a pro-inflammatory and hypercoagulable state,[15] homocysteinemia,[16] arterial calcification,[17] and endothelial dysfunction.[18,19] CKD is consistently associated with worse in-hospital[3,6,20] and long-term outcomes[1,5,21,22] after PCI, but to date, limited information has been published regarding procedural and in-hospital outcomes of patients with CKD undergoing CTO-PCI.

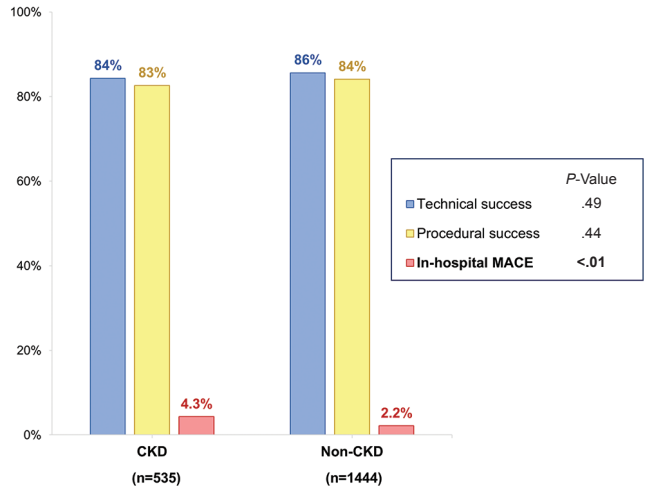

FIGURE 1. Technical success, procedural success, and in-hospital major adverse cardiovascular event (MACE) rates in patients with and without chronic kidney disease (CKD).

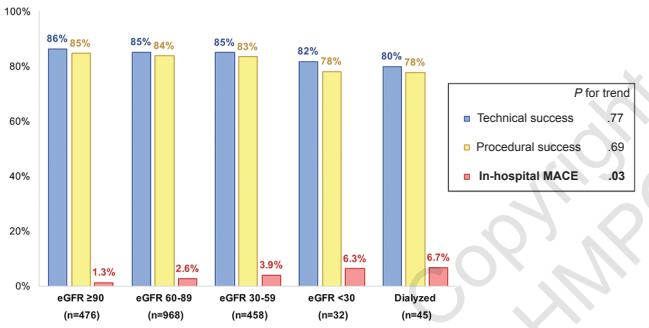

FIGURE 2. Technical success, procedural success, and in-hospital major adverse cardiovascular event (MACE) rates in the study patients, classified according to preprocedural estimated glomerular filtration rate (eGFR).

In our study, 27% of patients undergoing CTO-PCI had an eGFR of <60 mL/min/1.73 m^2, a finding consistent with previous reports.[7,23,24] Declining renal function did not significantly affect the technical success of CTO-PCI despite being associated with several comorbidities, such as prior MI and prior CABG, which have been associated with technical failure in other studies.[25] Moreover, patients with CKD were more likely to have moderate or severe calcification and proximal tortuosity that could also hinder coronary revascularization.[12,25] Although overall in-hospital MACE increased significantly and incrementally with decreasing renal function, this association was no longer present on multivariable analysis. However, in-hospital mortality was significantly higher in patients with CKD, an association that persisted after multivariable adjustment. This finding, which is in line with previous reports of outcomes after non-CTO PCI and CTO-PCI in patients with CKD, should be incorporated in the decision-making process for patients with a CTO and CKD. Current prediction models for technical success and adverse events of CTO-PCI, such as the PROGRESS CTO score[12] and PROGRESS CTO Complications score,[13] do not include CKD.

In a single-center study, Stahli et al examined long-term outcomes after CTO-PCI among 2002 patients stratified by the patients' baseline eGFR.[7] During a median follow-up of 2.6 years, higher eGFR was associated with lower all-cause mortality (hazard ratio [HR], 0.98; 95% CI, 0.98-0.99; *P*<.001). However, patients with failed CTO-PCI had worse long-term survival, regardless of whether they had CKD (eGFR <60 mL/min/1.73 m^2; HR, 1.59; 95% CI, 1.08-2.32; *P*=.02) or preserved renal function (eGFR >60 mL/min/1.73 m^2; HR, 1.73; 95% CI, 1.15-2.60; *P*<.01). Stahli et al reported significantly different procedural success rates for CTO-PCI in patients with different stages of renal dysfunction (range, 69%-86%; *P* for trend <.001); however, in our study, the procedural outcomes were similar in patients with various degrees of renal dysfunction (range, 78%-85%; *P* for trend =.69).

Dialysis patients are known to have worse in-hospital and long-term outcomes after PCI,[3] as was also observed in our study. This is likely related to higher angiographic complexity (especially more severe calcification) and more comorbidities that may predispose to complications (such as bleeding) or decrease the tolerance of a complication. If feasible, CABG might provide good long-term outcomes in dialysis patients.[26-28]

Prior publications of CTO-PCI in CKD patients focused mostly on the incidence of CIN (6.2%-9.4%) (Table 6).[23,24,29-33] Liu et al compared patients with renal failure (defined as creatinine clearance of <90 mL/min/1.73 m^2) who did (n = 359) or did not undergo CTO-PCI (n = 142), and reported that high technical success (89%) and improved long-term outcomes could be achieved in the former group, without increasing the risk for CIN (adjusted OR, 0.88; 95% CI, 0.41-1.93).[24] Liu et al developed a risk-stratification model for predicting CIN from a cohort of 728 patients: age ≥75 years, left ventricular ejection fraction <40%, and baseline serum creatinine >1.5 mg/dL were identified as independent predictors for CIN.[33] Bataille et al investigated the interaction between CKD and CTO in a non-infarct related artery on short-term (30-day) and long-term (1-year) outcomes after PCI for ST-segment elevation acute MI,[31] and found that the prevalence of CTOs was twice as high in CKD patients as compared with patients who did not have CKD. Lee et al evaluated the effect of CTO-PCI with periprocedural MI on long-term outcomes of 1058 patients who underwent successful CTO revascularization.[34] During a median follow-up of 4.4 years, CKD was independently associated with higher all-cause mortality (HR, 3.39; 95% CI, 1.48-7.75; *P*<.01).

Study limitations. First, this was a retrospective, observational study, and is subject to all the limitations of such

Table 6. Studies examining the effect of chronic kidney disease (CKD) on chronic total occlusion (CTO) percutaneous coronary intervention (PCI).

Author	Year	Prevalence of CKD[a] in Patients With CTO-PCI	Technical Success[a] in CKD Group	In-hospital MACE[a] in CKD Group	Note
Aguiar-Souto et al[30]	2008	65 (28%)	n/a	n/a	CIN[a] incidence: 6.2%. Contrast volume, eGFR, and Mehran risk score were not predictive of CIN.
Bataille et al[31]	2013	45 (29%)	n/a	n/a	Compared to patients with normal renal function, patients with CKD undergoing primary PCI for STEMI were twice as likely to have a CTO in a non-infarct related artery (13.4% vs 7.1%; *P*<.001).
Lin et al[32]	2014	206 (40%)	n/a	n/a	CIN incidence: 5.4%. Age >75 years, Mehran risk score, and severe proximal tortuosity were independent predictors of CIN after CTO-PCI.
Liu et al[29]	2015	85 (100%[c])	85%	Death: 0.0% Stroke: 0.0%	CIN incidence was similar in patients without CTO, patients with CTO target lesion, and patients with CTO as non-target lesion (11.4% vs 9.4% vs 6.7%, respectively; *P*=.34). Age ≥75 years was the only independent predictor of CIN.
Liu et al[24]	2016	359/154[b] (100%[c])	89%	MI: 0.0% Stroke: 0.3% Death: 0.6%	In patients with CTO and chronic kidney disease undergoing angiography, attempted CTO recanalization was associated with reduced long-term mortality (adjusted hazard ratio: 0.38; 95% confidence interval, 0.18-0.83; *P*=.02), without increase in CIN incidence (8.6% vs 10%; *P*=.13).
Stahli et al[7]	2018	418 (20%)	76%	Stroke: 0.2% All-cause death: 2.0% Bleeding: 1.7%	Decreasing baseline renal function was associated with higher all-cause mortality. CTO-PCI failure was associated with higher long-term mortality regardless of baseline renal function.
Zhang et al[23]	2016	86 (18%)	100%[c]	n/a	Patients with CTO benefit from revascularization regardless of CKD status (longer overall and MACE-free survival), but CKD attenuated this benefit.
Current study	2018	535 (27%)	84%	Stroke: 0.6% All-cause death: 1.9% Bleeding: 2.2%	High success rate of CTO-PCI can be achieved in patients with and without CKD, but CKD may be associated with increased in-hospital mortality but not MACE.

[a]Definition varies according to study.
[b]359 patients had a creatinine clearance (CrCl) <90 mL/min and 154 patients had CrCl <60 mL/min.
[c]Variable examined was an inclusion criterion in these studies.
CIN = contrast-induced nephropathy; eGFR = estimated glomerular filtration rate; MACE = major adverse cardiovascular event; MI = myocardial infarction; n/a = data not available.

studies. Second, eGFR calculations were made using only a single creatinine measurement performed closest to the procedure that was recorded as part of the study; data on urine albumin, or specific functional and structural indicators for CKD, were not collected. Given these limitations, CKD classification in our group could be subject to selection bias; however, the prevalence of CKD in our study is in line with prior studies. Third, there was no core laboratory adjudication of angiograms and no clinical event adjudication. Fourth, limited data were available on the incidence of

Appendix 1. Participating centers.	
1.	Appleton Cardiology, Appleton, Wisconsin
2.	Baylor Heart and Vascular Hospital, Dallas, Texas
3.	Beth Israel Deaconess Medical Center, Boston, Massachusetts
4.	Henry Ford Hospital, Detroit, Michigan
5.	Korgialeneio-Benakeio Hellenic Red Cross General Hospital of Athens, Athens, Greece
6.	Massachusetts General Hospital, Boston, Massachusetts
7.	Medical Center of the Rockies, Loveland, Colorado
8.	Minneapolis Heart Institute, Abbott Northwestern Hospital, Minneapolis, Minnesota
9.	Meshalkin Siberian Federal Biomedical Research Center, Ministry of Health of Russian Federation, Novosibirsk, Russian Federation
10.	North Texas VA Healthcare System, Dallas, Texas
11.	PeaceHealth St. Joseph Medical Center, Bellingham, Washington
12.	St. Luke's Mid America Heart Institute, Kansas City, Missouri
13.	Torrance Memorial Hospital, Torrance, California
14.	The Heart Hospital Baylor Plano, Plano, Texas
15.	University of California San Diego, La Jolla, California
16.	University of Pittsburgh Medical Center Presbyterian, Pittsburgh, Pennsylvania
17.	VA Central Arkansas Healthcare System, Little Rock, Arkansas
18.	VA Minneapolis Healthcare System and University of Minnesota, Minneapolis, Minnesota

CIN and new need for in-hospital dialysis due to the shorter data collection period. Fifth, no data were available on preprocedural hydration, other interventional or diagnostic procedures that were performed prior to PCI, or the type of contrast agent utilized.

Conclusion

CKD is common among patients undergoing CTO-PCI. High procedural success rates can be achieved in CKD patients, but they may have increased risk for in-hospital death.

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Study data were collected and managed using REDCap electronic data capture tools.[35] REDCap (Research Electronic Data Capture) is a secure, web-based application designed to support data capture for research studies, providing (1) an intuitive interface for validated data entry; (2) audit trails for tracking data manipulation and export procedures; (3) automated export procedures for seamless data downloads to common statistical packages; and (4) procedures for importing data from external sources.

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