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ORIGINAL ARTICLE

The impact of chronic kidney disease severity on clinical outcomes after current generation drug-eluting stent implantation for left main distal bifurcation lesions: the Milan and New-Tokyo registry

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ABSTRACT

Objectives. The impact of chronic kidney disease (CKD) on clinical outcomes after percutaneous coronary intervention (PCI) for unprotected left main distal bifurcation lesions (ULMD) is not fully understood in current generation drug eluting stent (cDES) era. We assessed clinical outcomes after PCI using cDES for ULMD according to CKD severity based on estimated glomerular filtration rate (eGFR). *Design*. We identified 720 consecutive patients who underwent PCI using cDES for ULMD at three high volume centers between January 2005 and December 2015. We divided those patients to the following five groups according to eGFR. Each group was defined as follows: no CKD (60 mL/min/1.73 $m^2 \leq$ eGFR), mild CKD ($45 \leq$ eGFR $<$ 60 mL/min/1.73 m^2), moderate CKD ($30 \leq$ eGFR $<$ 45 mL/min/1.73 m^2), severe CKD ($15 \leq$ eGFR $<$ 30 mL/min/1.73 m^2) and hemodialysis (HD). The primary endpoint was target lesion failure (TLF) at 3 years. TLF was defined as a composite of cardiac death, target lesion revascularization (TLR) and myocardial infarction (MI). *Results*. TLF occurred more frequently in severe CKD and HD group compared with other three groups. *Conclusions*. The patients who have severe CKD or are on HD, were extremely associated with worse clinical outcomes after PCI for ULMD even with cDES.

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KEYWORDS
Percutaneous coronary intervention; chronic kidney disease; current generation drug eluting stent; unprotected left main distal bifurcation lesions

Introduction

Recently, percutaneous coronary intervention (PCI) for unprotected left main distal bifurcation lesions (ULMD) has been widely performed [1,2]. Chronic kidney disease (CKD) is an independent risk factor for the development of coronary artery disease [3] and significantly increases the risk of death and cardiac adverse events after successful revascularization [4]. Previously, our team reported the severity of estimated glomerular filtration rate (eGFR)-based CKD was associated with an increased risk of clinical events after PCI for ULMD [5]. However, most of the patients included in that study were treated with early generation drug eluting stent (DES). Although the introduction of current generation DES (cDES) dramatically improved clinical outcomes after PCI compared to early generation DES (eDES) [6] in recent years, there are little available data about the impact of CKD severity on clinical outcomes after PCI using cDES for ULMD.

Therefore, we assessed clinical outcomes after PCI using cDES for ULMD according to CKD severity based on eGFR.

Materials and methods

Study population

Study flow chart is shown in Figure 1. We analyzed consecutive patients who underwent PCI for ULMD at New Tokyo Hospital, Matsudo, Japan, San Raffaele Scientific Institute and EMO-GVM Centro Cuore Columbus, Milan, Italy between January 2005 and December 2015. However, we excluded patients with acute myocardial infarction (MI) and in stent restenosis, those after coronary artery bypass graft surgery, those treated with bare metal stent, bioresorbable coronary scaffold and COMBO stent from the present study. The decision for treatment strategy was made to treat the left main distal bifurcation lesion through PCI after decision making meeting. In patients with high Syntax score, the reason on the decision was the frailty and comorbidity of patients.

Definition of variables

We divided those patients to the following 5 groups according to eGFR. CKD was defined as an eGFR of <60 mL/min/

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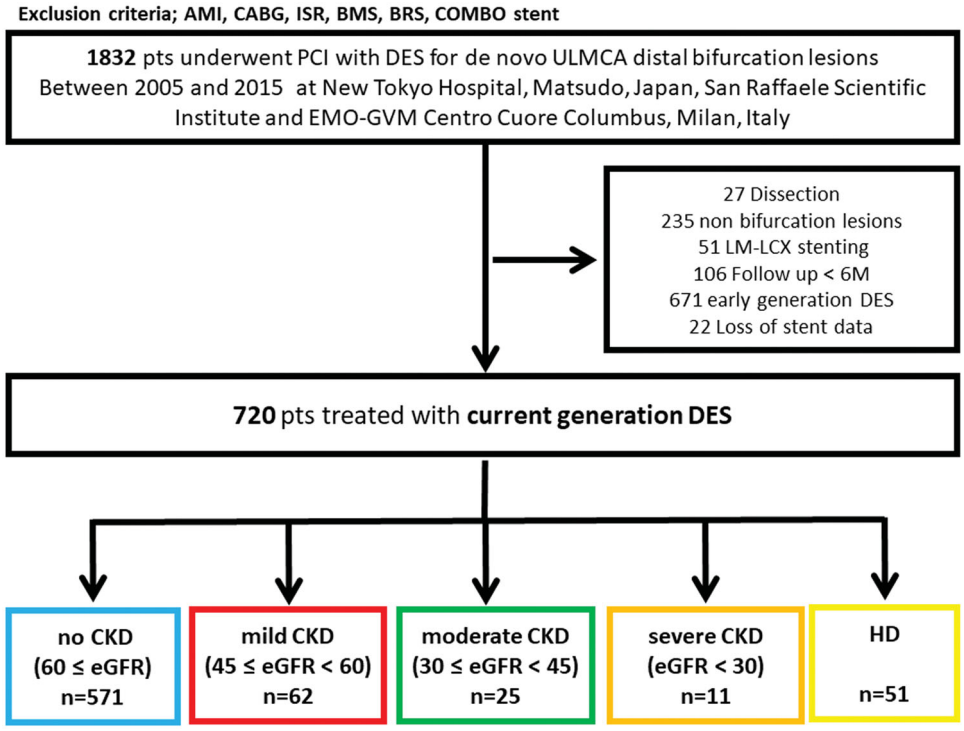

Figure 1. Study flow chart. AMI: acute myocardial infarction; BMS: bare metal stent; BRS: bioresorbable vascular scaffold; CABG: coronary artery bypass graft surgery; CKD: chronic kidney disease; DES: drug-eluting stent; DM: diabetes mellitus; ISR: in-stent restenosis; LCX: left circumflex artery; LM: left main; PCI: percutaneous coronary intervention; Pts: patients; ULMCA: unprotected left main coronary artery.

1.73 m^2 [7]. Each group was defined as follows; no CKD (60 mL/min/1.73 m^2 ≤ eGFR), mild CKD (45 ≤ eGFR < 60 mL/min/1.73 m^2), moderate CKD (30 ≤ eGFR < 45 mL/min/1.73 m^2), severe CKD (eGFR < 30 mL/min/1.73 m^2 and non-hemodialysis) and hemodialysis (HD). The eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula.

ULM disease was defined as a stenosis of at least 50% by visual evaluation that involved the ostium, body, or distal segment of the left main coronary artery or within the proximal 5 mm of the left anterior descending coronary artery (LAD) or left circumflex coronary artery (LCX) ostium. ULM lesions were divided into the following two groups: (1) ostium and body (non- bifurcation lesions) and (2) distal-bifurcation lesions. This study included only distal-bifurcation lesions because the previous paper reported the outcomes after PCI for non- bifurcation lesions [8]. Bifurcation lesions were classified according to the Medina classification [9] by two independent physicians. A true bifurcation lesion was defined as Medina class 1-1-1, 1-0-1, and 0-1-1. Coronary calcification was defined as "readily apparent densities seen within the artery wall and site of lesion as an X-ray absorbing mass". DM was defined according to the definition of the American Diabetes Association as follows [10]: (a) fasting plasma glucose of at least 126 mg/dl (7.0 mmol/l), or (b) 2-h plasma glucose of at least 200 mg/dl (11.1 mmol/l) during an oral glucose tolerance test, or (c) glycated hemoglobin of at least 6.5% (48 mmol/mol), or (d) presence of classic symptoms of hyperglycemia or hyperglycemic crisis with random plasma glucose of at least 200 mg/dl (11.1 mmol/l). Clinical data were collected during a hospital visit or by telephone contact at 6-month intervals. Angiographic follow-up was scheduled between 6 and 12 months or earlier if clinically indicated (evidence of ischemia on noninvasive evaluation or if there was suspicion of ischemia on clinical presentation). The relevant review boards in each institute approved the study protocol. We obtained written informed consent from each patient for data collection and analysis in accordance with the Declaration of Helsinki.

Stent information

The stents used in the present study were: sirolimus-eluting stents (SES) (Ultimaster; Terumo Corporation, Tokyo, Japan), zotarolimus-eluting stents (Resolute family, Medtronic, Santa Rosa, California), everolimus-eluting stents (EES) (Xience family [Abbott Vascular, Santa Clara, CA], Promus family and Synergy [Boston Scientific, Natick, MA, USA]), and biolimus-eluting stent (Nobori, Terumo Corporation, Tokyo, Japan). Single stent strategy was defined as the use of a single stent, regardless of the lesion type. Double stent strategy was defined as the use of two stents, regardless of the technique used. The main strategy of stenting was decided at the operator discretion. Kissing balloon inflation (KBI) and proximal optimization technique (POT) after stent implantation were dependent on the operator's discretion.

Medication

In patients who were not receiving aspirin, 200 mg of aspirin was administered before the procedure. The patients received a loading dose of either 300 mg of clopidogrel or 20 mg of prasugrel if they were not on a long-term

treatment. In the catheterization laboratory, heparin was administered to maintain an activated clotting time of ≥250 s or 200–250 s. After PCI, a lifelong administration of aspirin (100 mg/day) was prescribed, and clopidogrel (75 mg/day) or prasugrel (3.75 mg/day in Japan or 5.0 mg/day in Italy) was prescribed for at least 12 months, regardless of DES type. The patients treated with ticagrelor are not included in this study.

Investigated outcomes

The primary endpoint was target lesion failure (TLF). TLF was defined as a composite of cardiac death, target lesion revascularization (TLR) for LM-LAD and/or LCXos and MI. The individual components of TLF were also evaluated. Death was considered as cardiac in origin unless obvious non-cardiac causes were identified. The TLR was defined as a repeat revascularization by PCI or CABG of the target lesion. Periprocedural MI and ST were defined according to the Academic Research Consortium definitions [11]. Furthermore, the composite of sudden cardiac death (SCD) and definite/probable ST was also analyzed. Procedural success was defined as residual stenosis of <30% with a Thrombolysis in MI (TIMI) flow of 3 at the final angiography. During follow-up MI was defined by referring to the fourth universal definition of MI [12].

Statistical analysis

Values are reported as the mean ± standard deviation. Differences in categorical variables between the two groups were analyzed using the χ^2 test. Continuous variables were compared using the unpaired t-test. Time-to-event data were analyzed using the Kaplan–Meier method and log-rank test. Predictors of TLF were identified using a univariable Cox regression analysis. A multivariable model was built to identify the parameters independently associated with the occurrence of TLF at follow-up; all covariates with a statistically significant association with TLF at the univariable Cox regression analysis (with a 2-tailed p value <.10) or clinically relevant covariates were included in the final model. The models were also used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for clinical outcomes. All p values were two-sided, and $p < 0.05$ was considered statistically significant. The analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA).

Results

As Figure 1 shows, we identified 1832 consecutive patients who underwent PCI for ULMD at New Tokyo Hospital, Matsudo, Japan, San Raffaele Scientific Institute and EMO-GVM Centro Cuore Columbus, Milan, Italy between January 2005 and December 2015. Of 1832 patients, 720 patients were treated with cDES. Regarding to the distribution of patient's

Table 1. Baseline clinical, lesion and procedural characteristics.

	no CKD (60 ≤ eGFR) $n = 571$	mild CKD (45 ≤ eGFR < 60) $n = 62$	moderate CKD (30 ≤ eGFR < 45) $n = 25$	severe CKD (eGFR < 30) $n = 11$	HD $n = 51$	p-value
Age	69.5 ± 9.2	77.6 ± 7.7	78.9 ± 7.4	77.3 ± 6.5	69.1 ± 9.1	< .001
Male gender	459 (80.4)	43 (69.4)	22 (88.0)	8 (72.7)	42 (82.4)	.21
Previous myocardial infarction	174 (30.5)	22 (35.5)	13 (52.0)	5 (45.5)	14 (27.5)	.14
Previous stroke	32 (5.6)	8 (12.9)	7 (28.0)	1 (9.1)	6 (11.8)	< .001
Diabetes mellitus	231 (40.5)	30 (48.4)	12 (48.0)	5 (45.5)	34 (66.7)	.007
Insulin user	38 (6.7)	13 (21.0)	4 (16.0)	3 (27.3)	13 (25.5)	< .001
Hypertension	442 (77.4)	54 (87.1)	23 (92.0)	11 (100.0)	43 (84.3)	.046
Dyslipidemia	408 (71.5)	43 (69.4)	16 (64.0)	6 (54.5)	24 (47.1)	.006
Peripheral artery disease	53 (9.3)	15 (24.2)	12 (48.0)	1 (9.1)	30 (58.8)	< .001
Left ventricle ejection fraction	56.3 ± 10.3	53.1 ± 12.5	42.3 ± 11.6	54.3 ± 15.1	52.7 ± 11.3	< .001
Logistic EuroSCORE	3.9 ± 3.3	6.8 ± 8.5	12.4 ± 6.8	10.0 ± 11.3	10.2 ± 13.3	< .001
True bifurcation	303 (53.1)	35 (56.5)	15 (60.0)	6 (54.5)	33 (64.7)	.56
Three vessel disease	285 (50.3)	43 (69.4)	16 (64.0)	6 (54.5)	34 (66.7)	.01
Syntax score	26.4 ± 9.6	30.5 ± 11.3	28.4 ± 8.3	30.0 ± 9.4	27.7 ± 8.0	.08
Lesion calcification	273 (47.8)	34 (54.8)	12 (48.0)	6 (54.5)	39 (76.5)	.003
Rotational atherectomy	51 (8.9)	6 (9.7)	2 (8.0)	0 (0.0)	14 (27.5)	.001
Intravascular ultrasound	409 (71.6)	39 (62.9)	15 (60.0)	9 (81.8)	44 (86.3)	.04
Main branch stent diameter	3.50 ± 0.21	3.48 ± 0.20	3.50 ± 0.20	3.73 ± 0.26	3.54 ± 0.20	.005
Side branch stent diameter	3.07 ± 0.39	3.11 ± 0.36	3.40 ± 0.65	3.0 ± 0.0	3.13 ± 0.50	.45
Kissing balloon inflation	407 (71.3)	41 (66.1)	17 (68.0)	6 (54.5)	34 (66.7)	.65
Proximal optimization technique	457 (80.0)	45 (72.6)	18 (72.0)	8 (72.7)	46 (90.2)	.15
Proximal optimization technique balloon size	4.35 ± 0.43	4.32 ± 0.34	4.22 ± 0.42	4.63 ± 0.64	4.71 ± 0.43	< .001
Double stent strategy	177 (31.0)	15 (24.2)	6 (24.0)	2 (18.2)	17 (33.3)	.61
Stent name						
Xience	353 (61.8%)	36 (58.1)	13 (52.0)	5 (45.5)	36 (70.6)	
Resolute	46 (8.1)	8 (12.9)	4 (16.0)	0 (0.0)	6 (11.8)	
PROMUS	93 (16.3)	15 (24.2)	3 (12.0)	6 (54.5)	7 (13.7)	
Ultimaster	9 (1.6)	0 (0.0)	2 (8.0)	0 (0.0)	0 (0.0)	
Synergy	8 (1.4)	2 (3.2)	0 (0.0)	0 (0.0)	0 (0.0)	
NOBORI	62 (10.9)	1 (1.6)	3 (12.0)	0 (0.0)	2 (3.9)	

Data are presented as percentages and absolute numbers or means ± standard deviation.
CKD = chronic kidney disease, eGFR = estimated glomerular filtration rate.

number in each hospital, 542, 158, 20 patients were included from New Tokyo Hospital, San Raffaele Scientific Institute and Columbus Hospital, respectively.

Table 1 summarizes baseline clinical characteristics and lesion and procedural characteristics among the five groups, respectively. As the table shows, the patients with more severe CKD had significantly more severe morbidity. Patients in severe CKD and HD group had higher performance rate of intravascular ultrasound (IVUS), the implantation of a larger stent in main branch and POT using a larger balloon. In our population, intra-aortic balloon pumping was used in 50, 9, 4, 0 and 8 patients in no CKD, mild CKD, moderate CKD, severe CKD and HD group, respectively. The difference of the usage rate is not significant among the five groups ($p = .16$). Additionally, only one patient in no CKD group was underwent emergent CABG.

The median follow-up period was 1488 days (IQR: 1038-1975) and 3 years clinical follow-up was available in 88.2% of the patients. Among the five groups, the performance rate of angiographic follow up during 3 years after PCI is significantly lower in moderate and severe CKD group (81.1% in no CKD group vs. 67.7% in mild CKD group vs. 48.0% in moderate CKD group vs. 45.5% in severe CKD group vs. 76.5% in HD group, $p < .001$). As it is shown in Figure 2, TLF and TLR occurred more frequently in severe CKD and HD group compared with other 3 groups. Table 2 demonstrated significantly stronger association with adverse events after PCI in patients with severe CKD or on HD, who have eGFR less than 30. Furthermore, eGFR value of pre and post PCI are shown in Table 3. There were no patients required hemodialysis for exacerbation of renal function during perioperative period. Three patients in severe CKD group required hemodialysis during follow up periods (within 3 years after PCI).

The results of the multivariable analysis with Cox regression analysis are shown in Table 4. The independent predictors of TLF were severe CKD, HD, left ventricular ejection fraction, lesion calcification, female and age.

Discussion

The present study demonstrated that the patients with eGFR less than 30 mL/min/1.73 m^2, who has severe CKD or are on HD, were extremely associated with worse clinical outcomes after PCI even with cDES, mainly driven by increasing cardiac death, target lesion revascularization and myocardial infarction.

CKD is an independent risk factor for developing cardiovascular disease (CVD)[13]. Cardiovascular morbidity and mortality in CKD patients are high and the presence of

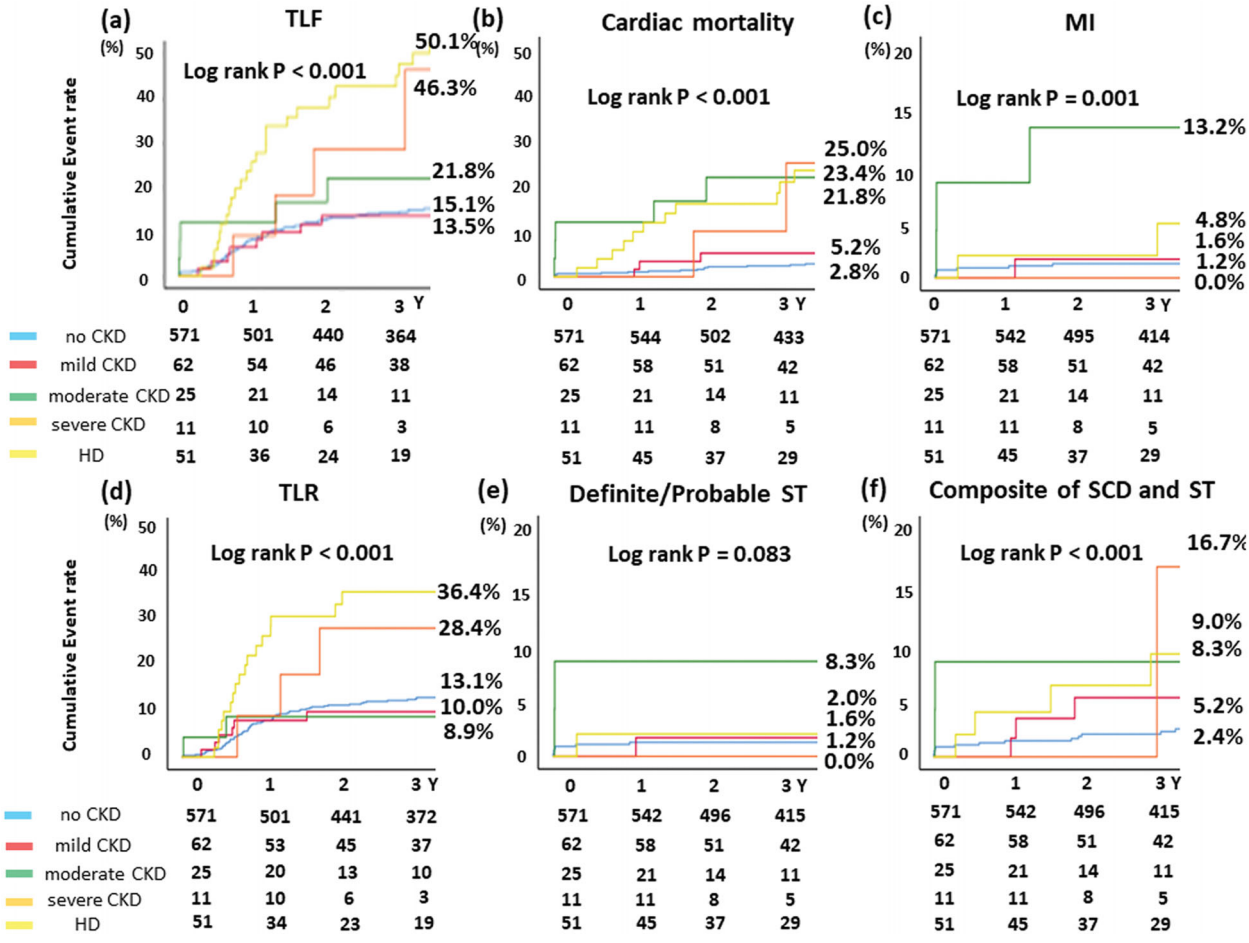

Figure 2. Kaplan–Meier curve of TLF, cardiac mortality, TLR, MI and ST.
(a) TLF; (b) Cardiac mortality; (c) MI; (d)TLR; (e)Definite/probable ST; (f)Composite of SCD and ST. TLF: target lesion failure; MI: myocardial infarction; TLR: target lesion revascularization; ST: stent thrombosis; SCD: sudden cardiac death; CKD: chronic kidney disease; HD: hemodialysis.

Table 2. Clinical outcomes.

	Unadjusted HR			Adjusted HR		
	95% CI			95% CI		
	Severe CKD	HD	eGFR < 30	Severe CKD	HD	eGFR < 30
Target lesion failure	2.80 (1.14-6.84) $p = .024$	4.38 (2.91-6.60) $p < .001$	4.32 (2.93-6.38) $p < .001$	3.25 (1.26-8.37) $p = .015$	3.18 (1.97-5.14) $p < .001$	3.34 (2.15-5.20) $p < .001$
Cardiac death	5.49 (1.70-17.8) $p = .004$	6.49 (3.61-11.7) $p < .001$	7.16 (4.07-12.6) $p < .001$	7.48 (2.10-27.0) $p = .002$	3.54 (1.75-7.18) $p < .001$	4.50 (2.30-8.80) $p <.001$
Target lesion revascularization	2.01 (0.64-6.36) $p = .23$	3.63 (2.21-5.98) $p < .001$	3.44 (2.15-5.51) $p < .001$	2.60 (0.78-8.62) $p = .12$	2.87 (1.62-5.10) $p <.001$	2.91 (1. 71-4.94) $p <.001$
Myocardial infarction	–	2.45 (0.54-11.1) $p = .24$	1.97 (0.44-8.89) $p = .38$	–	1.02 (0.19-5.40) $p = .98$	0.89 (0.17-4.57) $p = .89$
Definite/probable stent thrombosis	–	1.32 (0.17-10.3) $p = .79$	1.06 (0.14-8.30) $p = .95$	–	0.71 (0.07-6.12) $p = .71$	0.53 (0.06-4.77) $p = .57$
Sudden cardiac death and/or Stent thrombosis	2.81 (0.38-20.7) $p = .31$	4.47 (2.02-9.88) $p < .001$	4.44 (2.07-9.53) $p < .001$	2.80 (0.35-22.7) $p = .34$	2.74 (1.10-6.86) $p = .031$	2.87 (1.21-6.85) $p = .017$

CKD = chronic kidney disease, HD = haemodialysis, eGFR = estimated glomerular filtration rate.

Table 3. The value of eGFR of pre and post PCI.

	The value of eGFR		
CKD group	Pre PCI	Post PCI	*p*-value
Mild CKD	53.6 ± 4.0	56.6 ± 12.7	.10
Moderate CKD	38.5 ± 4.2	40.9 ± 8.4	.08
Severe CKD	22.1 ± 4.4	22.1 ± 6.2	.99

Data are presented as absolute numbers or means ± standard deviation.
eGFR = estimated glomerular filtration rate, PCI = percutaneous coronary intervention, CKD = chronic kidney disease.

CKD worsens clinical outcomes of CVD [14]. Previous studies have also demonstrated that CKD patients have poorer clinical outcomes even after successful revascularization [15]. In this study, we analyzed only patients with ULMD. Stent failure in ULMD could lead to life threatening events such as sudden cardiac death. It's most different point from non-ULM lesion. However, there are little available data about clinical outcomes after PCI using for ULMD in CKD patients. Therefore, we analyzed the patients with ULMD because we consider that it is very important to fully understand the impact of CKD on clinical outcomes after PCI for ULMD also in cDES era. Our study firstly demonstrated the worse clinical outcomes after PCI even with cDES for ULMD especially in CKD patients with eGFR less than 30 mL/min/1.73 m^2.

CKD is also associated with serious complications including vascular calcification due to CKD-mineral and bone disorder (CKD-MBD) [16]. Previous studies have reported that severe coronary calcification was associated with unfavorable clinical outcomes after PCI [17]. The most likely explanations were stent recoil [18] and excessive neointimal tissue [19]. CKD-MBD causes hyperphosphatemia and elevated serum alkaline phosphatase, low serum vitamin D and hypocalcemia and is associated with increased risk for severe cardiovascular calcification, morbidity, and mortality [20,21]. Additionally, reactive oxygen species (ROS) is also strongly associated with adverse events after PCI [22]. An increased level of ROS is associated with endothelial dysfunction, neointimal hyperplasia, vascular smooth muscle cells hypertrophy and migration involved in the post-PCI remodeling process [23,24]. Because CKD-MBD and oxidative stress are present even in the early stage of CKD [25] and the stage of renal failure progresses in a time-dependent manner, it means that patients with severe CKD and on HD have a longer disease duration than those with mild and moderate CKD. These previous findings could reasonably explain our results that patients with eGFR less than 30 mL/min/1.73 m^2 was extremely associated with worse clinical outcomes. Considering that advanced renal dysfunction increases the risk of adverse events, we believe that these patients should be carefully evaluated before performing PCI even with cDES.

Angiographic follow-up could affect clinical outcomes, especially in TLR. In this study, the performance rate of angiographic follow-up during 3 years after PCI is significantly lower in severe CKD group. This result could reasonably explain to reflect the physician's desire to avoid the use of contrast media. In general, the higher rate of angiographic follow up could lead to any events, mainly TLR. Therefore, our result indicates that angiographic follow up did not affect the clinical outcomes. Additionally, patients in severe CKD and HD group had higher performance rate of IVUS, the implantation of a larger stent in main branch and POT using a larger balloon. Considering IVUS and POT are associated with more favorable clinical outcomes [26–28], it is considered that the finding reflects the inferiority in patients with eGFR less than 30 mL/min/1.73 m^2.

Current generation DES had been proved to be superior to eDES in many studies [29,30]. The cDES has many advantages compared to eDES [31,32]. Due to platinum or cobalt–chrome platforms, durable polymer coatings and thinner struts, better biocompatibility, cDES shows a significant improvement in clinical outcomes for treatment of native coronary arteries. However, these beneficial effects of cDES could be not sufficient to overcome the negative

Table 4. The Predictors of target lesion failure on Cox regression analysis.

	Univariable hazard ratio (95% Confidence interval)	*p*-value	Adjusted hazard ratio (95% Confidence interval)	*p*-value
HD	4.38 (2.91–6.60)	<.001	3.30 (2.0–5.44)	<.001
severe CKD (eGFR < 30)	2.80 (1.14–6.84)	.024	3.66 (1.40–9.58)	.008
moderate CKD (30 ≤ eGFR < 45)	1.50 (0.66–3.39)	.34	1.60 (0.66–3.88)	.30
mild CKD (45 ≤ eGFR < 60)	0.77 (0.40–1.46)	.42	0.82 (0.41–1.66)	.58
Left ventricular ejection fraction	0.98 (0.97–0.99)	.003	0.98 (0.97–0.99)	.036
Lesion calcification	1.68 (1.20–2.35)	.003	1.48 (1.02–2.14)	.037
Female	1.33 (0.92–1.95)	.14	1.51 (1.01–2.25)	.042
Age	0.98 (0.97–1.01)	10.065	0.98 (0.96–0.99)	.036
Main branch stent diameter	0.71 (0.32–1.57)	.39	0.50 (0.22–1.15)	.10
True bifurcation	1.62 (1.15–2.28)	.005	1.36 (0.95–1.94)	.09
Proximal optimization technique	0.53 (0.38–0.75)	<.001	1.03 (0.65–1.63)	.89
Insulin user	2.47 (1.62–3.75)	<.001	1.45 (0.88–2.40)	.14
Peripheral artery disease	1.85 (1.26–2.71)	.002	1.04 (0.66–1.65)	.86
Diabetes mellitus	1.75 (1.26–2.43)	.001	1.38 (0.95–2.01)	.09
Intravascular ultrasound	0.99 (0.68–1.43)	.95	0.81 (0.54–1.21)	.31

HD: hemodialysis; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate.

factors present in patients with eGFR less than 30 mL/min/1.73 m^2, who has severe CKD or are on HD.

This study has several limitations. First, this was a non-randomized, retrospective study. The data could have resulted in selection bias. Second, although IVUS is strongly recommended during LM stenting, IVUS was not used in all cases. Third, since a routine angiographic follow-up was not performed, angiographic follow-up and TLR were performed according to each institution's strategy and each operator's discretion. Forth, the difference of each institution could not be analyzed sufficiently. Fifth, all patients were received DAPT at discharge and DAPT was continued at least 12 months, but the duration of DAPT beyond 12 months could not be fully analyzed. Sixth, although the previous paper reported that serum creatinine usually begins to rise within 24 h after exposure to contrast media and peaks between 3 and 5 days [33], we have only the data about serum creatinine for next day of index PCI. Therefore, we do not discuss about contrast induced acute kidney injury in the present paper. Finally, severe CKD group had a higher risk of MI, sudden cardiac death, and stent thrombosis compared to the HD group in this study. This surprising result can be explained by non-performance of angiographic follow up to avoid the use of contrast media due to concerns about impaired renal function. Further study is required to improve clinical outcomes in those high-risk patients.

Acknowledgments

The patients who have severe CKD or are on HD, were extremely associated with worse clinical outcomes after PCI for ULMD even with cDES, mainly driven by increasing cardiac death, target lesion revascularization and myocardial infarction.

Disclosure statement

The authors have no conflicts of interest to declare.

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