Introduction

It is well known that type 2 diabetes mellitus (DM) is a major risk factor for chronic kidney disease (CKD). Previous studies have identified DM and CKD as independent and powerful predictors of ischemic complications in patients undergoing percutaneous coronary intervention (PCI) with stents. Although previous studies have addressed the adverse effects of CKD on clinical outcomes in DM patients undergoing PCI, these studies were conducted in the era of bare metal stents (BMS) or first-generation drug-eluting stents (DES). It is expected that improvement in interventional techniques over time, together with the use of newer-generation DES and adherence to optimal medical therapies have improved interventional outcomes. Therefore, this study
evaluated the impact of CKD on clinical outcomes in patients with DM who underwent PCI using newer-generation DES in a real-world setting.

**Subjects and Methods**

**Study population**

This study, conducted over a three-year period, was based on patients from an ongoing, tertiary care, single-center registry that prospectively enrolled unselected patients undergoing PCI. Patients with a variety of clinical indications including stable angina, unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI) were registered. Patients in whom stent implantation was not attempted during PCI were excluded. Data on patient medical history, presentation, angiographic findings, treatment method, and complications were collected prospectively using standardized case report forms. Clinical follow-up was performed either by telephone contact or by office visit at 6 and 12 months following PCI. The occurrence of major clinical events including death, myocardial infarction (MI), revascularization, and stent thrombosis was recorded. This registry was approved by the institutional review board. Informed consent was obtained from all patients.

Among 2303 patients who underwent PCI from January 2011 to December 2013, 887 (38.5%) with a history of DM or with HbA1c value greater than 6.5% at the time of admission to the hospital were selected. Serum creatinine level was measured at the time of admission. Renal function was assessed according to the estimated creatinine clearance (eCr), which was calculated using the Cockcroft-Gault equation. CKD was defined as an eCr value less than 60 mL/min/1.73 m². Patients were divided into groups without CKD and with CKD.

**Percutaneous coronary intervention**

All patients were given 300 mg of aspirin and 300 mg of clopidogrel (interchangeable with prasugrel 60 mg or ticagrelor 180 mg) as a loading dose at least six hours prior to the procedure, except in cases of emergency. All patients underwent PCI according to standardized methods. The decision for pre-dilatation or direct stenting, the selection of stent type, the use of glycoprotein (GP) IIb/IIIa inhibitors, the use of additional imaging modalities such as intravascular ultrasound or optical coherence tomography and the use of a pressure wire were made at the discretion of the operating physicians. Most patients were treated with either a biolimus-eluting stent (BioMatrix or BioMatrix Flex, Biosensors, Singapore, or Nobori, Terumo, Japan), an everolimus-eluting stent (Xience V or Xience Prime, Abbott, IL, USA), or a zotarolimus-eluting stent with Biolinx polymers (Endeavor Resolute or Endeavor Resolute Integrity, Medtronic, Minneapolis, MN, USA). All physicians tried to follow guideline-directed medical treatment, which includes the use of beta-blockers, angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers, and statins. After the index procedure, an aspirin dosage of 100 mg was prescribed permanently, and a 75-mg dosage of clopidogrel (interchangeable with prasugrel in a dosage of 10 mg daily or ticagrelor in a dosage of 90 mg twice daily) was prescribed for at least one year.

**Study endpoints and definition**

The primary endpoint was a patient-oriented composite outcome (POCO) at one year following the index procedure among survivors at discharge. The POCO was a composite consisting of all-cause mortality, MI, and revascularization. The secondary endpoints were POCO, device-oriented composite outcome (DOCO), bleeding complication, and contrast-induced nephropathy (CIN) during the index hospital stay, together with the DOCO at the one-year follow-up period. The DOCO was defined as a composite of cardiac death, MI (not clearly attributable to a non-target vessel), and target lesion revascularization (TLR). MI includes only spontaneous MI, which was diagnosed when at least two of the following three criteria were met: 1) clinical history of central chest pressure, pain, or tightness lasting ≥30 minutes, 2) ST-segment elevation greater than 0.1 mV in at least one standard or two precordial leads, and 3) troponin I value greater than the upper normal limit (UNL) or creatine kinase MB value greater than the UNL. Target lesion revascularization (TLR) was defined as any repeat PCI of the target lesion or bypass surgery of the target vessel performed for restenosis or other complication of the target lesion. The target lesion was defined as the treated segment from 5 mm proximal to the stent to 5 mm distal to the stent. Target vessel revascularization (TVR) was defined as any repeat PCI or surgical bypass of any segment of the target vessel. The target vessel was defined as the entire major coronary vessel proximal and distal to the target lesion, which includes upstream and downstream branches as well as the target lesion itself. Cardiac death was defined as any death due to a proximate cardiac cause (e.g., MI, low-output failure, fatal arrhythmia), unwitnessed death and death of unknown cause, and all procedure-related deaths including those related to concomitant treatment. Noncardiac death was defined as any death not covered by the above categories, such as death caused by infection, malignancy, sepsis, pulmonary causes, accident, suicide, or trauma. CIN was defined as a serum creatinine increase greater than or equal to 25% and/or 0.5 mg/dL within 48 hours after exposure to the contrast medium. Stent thrombosis was defined according to the criteria of the academic
Table 1. Baseline characteristics, initial laboratory findings, and medications at discharge

|                               | Patients without CKD (n=549) | Patients with CKD (n=338) | p       |
|-------------------------------|------------------------------|---------------------------|---------|
| Male                          | 388 (70.7)                  | 169 (50.0)                | <0.001  |
| Age (years)                   | 63.3±10.1                   | 71.9±7.6                  | <0.001  |
| BMI (kg/m²)                   | 25.3±2.9                    | 23.8±2.9                  | <0.001  |
| HTN                           | 349 (63.6)                  | 262 (77.7)                | <0.001  |
| Insulin-requiring diabetes    | 25 (4.6)                    | 42 (12.4)                 | <0.001  |
| Dyslipidemia                  | 129 (23.5)                  | 76 (22.5)                 | 0.728   |
| Dialysis-dependent CKD        | 0 (0)                       | 21 (6.4)                  | <0.001  |
| Smoking                       | 292 (53.9)                  | 130 (38.8)                | <0.001  |
| Prior MI                      | 59 (10.7)                   | 35 (10.4)                 | 0.854   |
| Prior PCI                     | 109 (19.9)                  | 67 (19.8)                 | 0.991   |
| Prior CABG                    | 2 (0.4)                     | 4 (1.2)                   | 0.209*  |
| Prior CVA                     | 38 (7.0)                    | 29 (8.7)                  | 0.358   |
| Stable angina                 | 94 (17.1)                   | 39 (11.5)                 | 0.024   |
| Unstable angina               | 162 (29.5)                  | 99 (29.3)                 | 0.945   |
| NSTEMI                         | 95 (17.3)                   | 85 (25.1)                 | 0.005   |
| STEMI                          | 102 (18.6)                  | 57 (16.9)                 | 0.518   |
| SBP (mmHg)                    | 130.7±24.5                  | 131.0±30.7                | 0.863   |
| DBP (mmHg)                    | 76.6±14.5                   | 73.6±16.4                 | 0.007   |
| HR (bpm)                      | 78.1±17.5                   | 79.1±20.8                 | 0.467   |
| TC (mg/dL)                    | 174.8±48.1                  | 166.2±44.9                | 0.008   |
| TG (mg/dL)                    | 119.5 (86.3, 177.8)         | 116.0 (81.0, 166.8)       | 0.097   |
| HDL-cholesterol (mg/dL)       | 44.7±11.6                   | 42.9±12.7                 | 0.031   |
| LDL-cholesterol (mg/dL)       | 107.2±39.2                  | 100.5±37.9                | 0.015   |
| BUN (mg/dL)                   | 16 (13, 18)                 | 22 (17, 30)               | <0.001  |
| Cr (mg/dL)                    | 0.80 (0.64, 0.90)           | 1.20 (0.94, 1.67)         | <0.001  |
| eCCr (mL/min/1.73 m²)         | 92.6±28.6                   | 40.6±15.0                 | <0.001  |
| Glucose (mg/dL)               | 146 (114, 199)              | 154 (114, 228)            | 0.129   |
| Hb (g/dL)                     | 14 (13, 15)                 | 12 (11, 14)               | <0.001  |

Medication at discharge among survivors

|                               | Patients without CKD (n=549) | Patients with CKD (n=338) | p       |
|-------------------------------|------------------------------|---------------------------|---------|
| Aspirin                       | 526 (97.0)                  | 303 (96.2)                | 0.551   |
| ADP receptor inhibitor        | 529 (97.6)                  | 306 (97.1)                | 0.682   |
| Clopidogrel                   | 506 (93.4)                  | 300 (95.2)                | 0.262   |
| Ticagrelor or prasugrel       | 23 (4.2)                    | 6 (1.9)                   | 0.068   |
| Cilostazol                    | 60 (11.1)                   | 33 (10.5)                 | 0.788   |
| VKA                           | 4 (0.7)                     | 4 (1.3)                   | 0.475*  |
| Statin                        | 481 (88.7)                  | 257 (81.6)                | 0.003   |
| RAS inhibitor                 | 364 (67.2)                  | 208 (66.0)                | 0.736   |
| CCB                           | 58 (10.7)                   | 44 (14.0)                 | 0.154   |
| Beta-blocker                  | 366 (67.5)                  | 207 (65.7)                | 0.587   |
| Nitrate                       | 266 (49.1)                  | 144 (45.7)                | 0.342   |
| Nicorandil                    | 359 (66.1)                  | 224 (71.1)                | 0.126   |

Values are presented as n (%) or mean±standard deviation or median (interquartile range). *Fisher's exact test. CKD: chronic kidney disease, BMI: body mass index, HTN: hypertension, MI: myocardial infarction, PCI: percutaneous coronary intervention, CABG: coronary artery bypass graft, CVA: cerebrovascular accident, NSTEMI: non-ST segment elevation myocardial infarction, STEMI: ST segment elevation myocardial infarction, SBP: systolic blood pressure, DBP: diastolic blood pressure, HR: heart rate, TC: total cholesterol, TG: triglyceride, HDL: high-density lipoprotein, LDL: low-density lipoprotein, BUN: blood urea nitrogen, Cr: creatinine, eCCr: estimated creatinine clearance, Hb: hemoglobin, ADP: adenosine diphosphate, VKA: vitamin K antagonist, RAS: renin-angiotensin system, CCB: calcium channel blocker

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research consortium (ARC). The incidence of ST included definite and probable ST. Bleeding was defined according to the standards of the bleeding ARC (BARC).9)

Statistical analysis
Continuous variables were described as mean ± standard deviation or median (interquartile range) and were analyzed by Student’s unpaired t-test or the Mann-Whitney U test, as appropriate. Categorical variables were described as counts (percentages) and were compared by χ² or Fisher’s exact test, as appropriate. Follow-up began on the date of the index PCI and continued until the date of death or for one year, whichever occurred sooner. Kaplan-Meier curves for the POCO and DOCO of both patient groups were obtained. Clinical outcomes were compared between the groups with the log rank p test. Cox proportional hazards regression analysis was used to estimate the hazard ratio (HR) of POCO and

Table 2. Angiographic and procedural characteristics

|                                | Patients without CKD (n=549) | Patients with CKD (n=338) | p     |
|--------------------------------|-----------------------------|---------------------------|-------|
| Heparin use before procedure   | 180 (32.8)                  | 110 (32.5)                | 0.940 |
| Use of GP IIb/IIIa inhibitor   | 71 (12.9)                   | 38 (11.2)                 | 0.457 |
| Disease extent                 |                             |                           | <0.001|
| 1-VD                           | 208 (37.9)                  | 63 (18.6)                 |       |
| 2-VD                           | 182 (33.2)                  | 126 (37.3)                |       |
| 3-VD                           | 159 (29.0)                  | 149 (44.1)                |       |
| Transradial intervention       | 527 (96.0)                  | 265 (78.4)                | <0.001|
| Elective PCI                   | 434 (79.1)                  | 264 (78.1)                | 0.736 |
| Primary PCI                    | 99 (18.0)                   | 53 (15.7)                 | 0.409 |
| Use of IABP                    | 3 (0.5)                     | 11 (3.3)                  | 0.002 |
| Use of ECMO                     | 5 (0.9)                     | 1 (0.3)                   | 0.416*|
| Use of IVUS                     | 527 (96.0)                  | 310 (91.7)                | 0.007 |
| Use of thrombus aspiration     | 110 (20.0)                  | 62 (18.3)                 | 0.536 |
| Bifurcation PCI                | 193 (35.2)                  | 109 (32.2)                | 0.375 |
| CTO PCI                        | 34 (6.2)                    | 15 (4.4)                  | 0.266 |
| Treated lesion territory       |                             |                           |       |
| LAD                            | 341 (62.1)                  | 199 (58.9)                | 0.337 |
| LCX                            | 135 (24.6)                  | 82 (24.3)                 | 0.912 |
| RCA                            | 187 (34.1)                  | 145 (42.9)                | 0.008 |
| LM                             | 29 (5.3)                    | 18 (5.3)                  | 0.978 |
| Multi-vessel PCI               | 146 (26.6)                  | 104 (30.8)                | 0.179 |
| Implanted stent type           |                             |                           | 0.266 |
| BES                            | 161 (29.3)                  | 90 (26.6)                 |       |
| EES                            | 191 (34.6)                  | 109 (32.2)                |       |
| ZES-R                          | 161 (29.3)                  | 115 (34.0)                |       |
| Implanted stent diameter (mm)  | 3.2±0.5                     | 3.0±0.5                   | 0.002 |
| Implanted stent length (mm)    | 42.1±24.7                   | 44.7±26.7                 | 0.152 |
| Implanted stent number (n)     | 1.7±0.9                     | 1.8±0.9                   | 0.113 |
| Total procedure time (min)     | 56.5±24.7                   | 57.9±29.1                 | 0.470 |
| Contrast volume (mL)           | 193.2±67.4                  | 199.5±100.1               | 0.311 |

Values are presented as n (%) or mean±standard deviation. *Fisher’s exact test. CKD: chronic kidney disease, VD: vessel disease, PCI: percutaneous coronary intervention, IABP: intra-aortic balloon pump, ECMO: extracorporeal membrane oxygenation, IVUS: intravascular ultrasound GP: glycoprotein, CTO: chronic total occlusion, LAD: left anterior descending, LCX: left circumflex artery, RCA: right coronary artery, LM: left main, BES: biolimus-eluting stent, EES: everolimus-eluting stent, ZES: resolute zotarolimus-eluting stent.
DOC0 during a one-year period of follow-up among survivors at discharge. Adjusted HRs with 95% confidence intervals (CIs) were computed. Multivariable Cox regression models were adjusted for baseline differences in demographic, clinical, and angiographic factors. The variables used for adjustment in the analysis of one-year outcomes were CKD; age; sex; body mass index; history of hypertension, smoking, MI, or PCI; acute MI; diastolic blood pressure; multi-vessel disease; multi-vessel PCI; implanted stent type, number, diameter, and length; low-density lipoprotein (LDL)-cholesterol; hemoglobin; and discharge medications including aspirin, adenosine diphosphate (ADP) receptor inhibitor, statin, beta-blocker, calcium channel blocker (CCB), and renin–angiotensin system (RAS) inhibitor. Logistic multivariate regression analysis using the same variables as in the multivariable Cox regression model (with the exception of
discharge medications) was performed to estimate the odds ratio for in-hospital outcomes such as POCO, DOCO, bleeding complications, and CIN among all patients. All statistical analyses were performed with IBM SPSS Statistics version 20.0 (IBM Inc., Chicago, IL, USA). A two-tailed p value of 0.05 was considered statistically significant.

Results

Baseline characteristics

Among a total of 887 consecutive patients with DM, approximately 62% (549) had normal renal function (eCcr of ≥60 mL/min/1.73 m²), whereas 38% (338) had CKD (eCcr of <60 mL/min/1.73 m²). Baseline characteristics, initial laboratory findings, and medications at discharge are presented in Table 1. Compared with patients without CKD, those with CKD were older, less often obese, and more likely to be female. Patients with CKD were more likely to have a history of hypertension as well as insulin-dependent DM. However, these patients were less likely to be smokers than patients without CKD. The incidence of NSTEMI was higher in patients with CKD. Initial systolic blood pressure and heart rate were similar between the two groups, but diastolic blood pressure was significantly lower in patients with CKD. On initial laboratory findings, total cholesterol and high- and low-density lipoprotein cholesterol were lower in patients with CKD. Hemoglobin was also lower in patients with CKD. Although the rate of prescription of antplatelet, ADP receptor inhibitor, beta-blocker, CCB, or RAS inhibitor was similar between the two groups, the rate of statin prescriptions was significantly lower in patients with CKD.

Angiographic and procedural characteristics

Angiographic and procedural characteristics are presented in Table 2. Although the rate of multi-vessel disease was higher in patients with CKD, the rate of multi-vessel PCI was not different between the two groups. The rate of transradial intervention was significantly higher in patients without CKD. The indication for PCI was similar between the two groups. The type of implanted stent, number of stents, and length of stents were similar between the groups, but implanted stent diameter was smaller in patients with CKD.

In-hospital and one-year clinical outcomes

In-hospital outcomes in overall patients and one-year clinical outcomes among survivors at discharge are presented in Table 3. Kaplan-Meier curves for the survivors at discharge are displayed in Fig. 1. During index hospitalization, patients with CKD were associated with both ischemic and bleeding complications. The incidence of POCO was significantly higher in patients with CKD, driven primarily by a greater incidence of death by any cause. The incidence of DOCO, driven primarily by a greater incidence of

| Table 4. Multivariate analysis |
|-------------------------------|
|                               | POCO  | DOCO  | Bleeding Cx | CIN    |
| In-hospital outcome*          |       |       |             |       |
| Crude                         | 4.586 (2.105-9.990) | 4.870 (2.037-11.644) | 6.327 (2.082-19.228) | 3.435 (2.155-5.478) |
| Model 1                       | 4.636 (1.959-10.975) | 4.636 (1.959-10.975) | 8.935 (2.589-30.835) | 3.643 (2.159-6.146) |
| Model 2                       | 2.966 (1.081-8.138) | 2.882 (0.962-8.630) | 12.531 (3.080-50.977) | 2.458 (1.399-4.318) |
| Model 3                       | 2.769 (0.963-7.962) | 2.794 (0.889-8.781) | 11.512 (2.726-48.618) | 2.468 (1.389-4.385) |

| One-year outcome†             |       |       |             |       |
| Crude                         | 2.842 (1.778-4.543) | 4.000 (1.520-10.529) | N/A  | N/A  |
| Model 1                       | 2.366 (1.414-3.957) | 2.817 (0.990-8.016) | N/A  | N/A  |
| Model 4                       | 1.964 (1.114-3.371) | 2.091 (0.703-6.220) | N/A  | N/A  |
| Model 5                       | 1.824 (1.065-3.124) | 2.082 (0.690-6.278) | N/A  | N/A  |

Values are presented as odds ratio (or hazard ratio) and 95% confidence interval. *Logistic multivariate regression analysis. †Cox proportional hazards regression analysis. Model 1: adjusted for age and sex. Model 2: adjusted for model 1+BMI, HTN, smoking, prior MI, prior PCI, AMI, DBP, LDL-cholesterol, and Hb. Model 3: adjusted for model 2+MV disease, MV PCI, implanted stent type, number, diameter, and length. Model 4: adjusted for model 2+discharge medications including aspirin, ADP receptor inhibitor, statin, beta-blocker, CCB, and RAS inhibitor. Model 5: adjusted for model 3+discharge medications including aspirin, ADP receptor inhibitor, statin, beta-blocker, CCB, and RAS inhibitor. POCO: patient-oriented composite outcome, DOCO: device-oriented composite outcome, Cx: complication, CIN: contrast-induced nephropathy, BMI: body mass index, HTN: hypertension, MI: myocardial infarction, PCI: percutaneous coronary intervention, AMI: acute myocardial infarction, DBP: diastolic blood pressure, LDL: low-density lipoprotein, Hb: hemoglobin, MV: multi-vessel, ADP: adenosine diphosphate, CCB: calcium channel blocker, RAS: renin–angiotensin system.
cardiac death, was also significantly higher in patients with CKD. The rates of bleeding complications and CIN were also significantly higher in patients with CKD. During the one-year follow-up period, the incidence of POCO, driven primarily by a higher incidence of any death and any MI, and the incidence of DOCO, driven primarily by a higher incidence of cardiac death, were significantly higher in patients with CKD. Although the rate of TVR was significantly higher in patients with CKD, the rates of TLR and stent thrombosis were similar between the groups.

**Multivariate analysis**

On multivariate analysis (Table 4), presence of CKD was an independent predictor of bleeding complication (HR: 11.512, 95% CI: 2.726–48.618) and CIN (HR: 2.468, 95% CI: 1.389–4.385), but not of POCO (HR: 2.769, 95% CI: 0.963–7.962) or DOCO (HR: 2.794, 95% CI: 0.889–8.781) during index hospitalization. During the one-year follow-up period, presence of CKD was an independent predictor of POCO (HR: 1.824, 95% CI: 1.065–3.124), but not of DOCO (HR: 2.082, 95% CI: 0.690–6.278) among survivors at discharge.

**Discussion**

Our study demonstrated that CKD was associated with increased in-hospital and one-year adverse clinical outcomes, as well as with bleeding complications, in diabetic patients in the era of newer-generation DES, even after adjustment for other clinical factors that could potentially confound these associations. It also demonstrated that CKD was an independent predictor of CIN in diabetic patients in this timeframe. Despite these important clinical adverse outcomes, CKD was not shown to be associated with an increased risk of device-related events, including stent thrombosis and TLR.

**Ischemic outcomes in CKD**

DM is the most common cause of CKD worldwide. The world’s diabetic population is expected to approximately double from 2.8% in 2000 to 4.4% in 2030. The incidence of CKD is also expected to escalate in parallel to this trend. Both DM and CKD are associated with an increased risk of coronary events due to the associated prothrombotic state and proinflammatory milieu. Both DM and CKD are strong and independent predictors of adverse clinical outcomes in patients with coronary artery disease (CAD) treated with PCI. In line with the findings of previous studies, our study also demonstrated that the incidence of POCO comprised of any death, any MI, and any revascularization in diabetic CKD patients was significantly higher than in patients without CKD. In contrast to previous studies, we demonstrated that the incidence of device-related events, especially ST and TLR in diabetic CKD patients, was similar to the incidence of device-related deaths in patients without CKD. These results may be related to overall improvement.
in interventional techniques and the use of newer-generation DES. Most previous studies were conducted in the era of BMS and first-generation DES. In contrast, our study included only patients treated with newer-generation DES. Recent pooled analysis has shown that newer-generation DES could reduce the DOCO by 32% in comparison to early-generation DES in patients with higher CAD complexity assessed according to synergy between PCI with taxus and cardiac surgery (SYNTAX) score.\(^{40}\) It also showed that newer-generation DES significantly reduced TLR by 65% and definite ST by 72%.

**Bleeding complications in CKD**

In our study, overt bleeding was only observed only in patients with CKD. The work of Latif et al.\(^ {15}\) demonstrated a graded increase in bleeding complications with worsening renal function. The work of Alexander et al.\(^ {7}\) demonstrated that renal impairment independently predicted the overdosing of heparin and GP IIb/IIIa inhibitors and major bleeding in acute coronary syndrome patients. Moreover, the correct dosing of heparin in patients with CKD has not been established. Patients with CKD exhibit platelet dysfunction manifested by reduced ATP release and decreased serotonin content in dense granules. Our results are consistent with the results of previous studies.

**CIN in CKD**

Diabetic CKD confers a high risk for CIN, a major noncardiac complication associated with high morbidity and mortality, including the need for temporary dialysis or even permanent impairment of kidney function.\(^ {50}\) Decline in renal function after PCI portends a worse prognosis in patients with already impaired renal function. Our study found a significantly high occurrence rate of CIN after PCI (17% vs. 6%) in patients with CKD. This finding is similar to the findings of a study conducted by Nikolsky et al.\(^ {41}\) (27% vs. 15%) in diabetics who have undergone PCI. In that study, volume of contrast and CKD were independent predictors of CIN, and CIN was an independent predictor of one-year mortality. The findings therein showed concordance with our study. However, the absolute incidence of CIN was lower in our study than in that previous research. The cause of this discrepancy could be the use of less contrast in our study. As mentioned in Nikolsky’s article, there was a stepwise increase in CIN with increasing amount of contrast medium.

**Limitations**

This study has several limitations. First, this was a single-center study with a modest number of patients and a relatively short follow-up period, with a relatively low incidence of endpoints. Thus, these results may not be consistent with the results of a large cohort study. In particular, we were limited in our ability to evaluate some rare endpoints such as stent thrombosis. Second, the Cockcroft-Gault equation used for estimation of creatinine clearance (eCCr) might overestimate creatinine clearance in obese patients and is not applicable in patients with end-stage renal disease (ESRD). However, even if all dialysis-dependent patients were included in the CKD group, this would not affect the results, as ESRD is a known independent predictor of poor outcomes. Finally, this study involved heterogeneities in clinical and angiographic characteristics between patients with or without CKD. Multivariate analysis was performed to reduce this limitation, but potential bias could still exist.

**Conclusion**

Among diabetic patients, CKD, as determined by eCCr, is a powerful and independent predictor of in-hospital bleeding complications, CIN, and one-year patient-related ischemic outcomes in the era of newer-generation DES. However, CKD is not related to in-hospital or one-year device-related ischemic outcomes, particularly stent thrombosis and TLR. It remains to be determined whether the absence of differences in device-related outcomes between CKD and non-CKD patient groups is sustained during long-term follow-up.

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