Clinical Impact of Diabetes Mellitus on One-Year Clinical Outcomes Following Percutaneous Cardiac Intervention with Second-Generation Drug-Eluting Stents: A Real-World Observational Retrospective Study

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Abstract

Background: Diabetes mellitus (DM) is a risk factor for adverse clinical events following percutaneous coronary intervention (PCI). However, studies on the clinical impact of DM on second-generation drug-eluting stents (DES) are limited.

Methods: From April 1, 2009 to March 31, 2019, 888 patients with DM and 1,296 without DM, who underwent PCI with second-generation DES, were enrolled in this study. The primary outcome considered was one-year major adverse cardiac events (MACE), and the secondary outcomes were target lesion failure (TLF) and stent thrombosis (ST).

Results: In total, 2,184 patients with 3,344 lesions were enrolled in the pooled dataset. Observed one-year rates of MACE were significantly higher in patients with DM than in patients without DM (adjusted hazard ratio (HR) = 2.342 (1.311 - 4.184), p = 0.004). However, there was no significant difference in one-year rates of TLF (HR = 2.893 (0.980 - 8.543), p = 0.054). The cumulative incidence rates of MACE-free (log-rank test, p = 0.002) and TLF-free (log-rank test, p = 0.006) survival were significantly lower in patients with DM than in patients without DM. No cases of ST occurred in either of the two groups.

Conclusions: Our results indicated that the impact of DM significantly increased the rate of MACE in the early period (0-1 year).

Key words: diabetes mellitus, second-generation drug-eluting stents, major adverse cardiac events, target lesion failure, and stent thrombosis

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INTRODUCTION

The global population of diabetes mellitus (DM) patients is expected to increase from 463 million people (9.3%) in 2019 to 578 million people (10.2%) by 2030.[1] The total number of patients with DM in Taiwan increased from 1.3 million in 2009 to 2.2 million in 2014, and this number continues to increase every year.[2] DM is associated with the development of atherosclerosis and is a well-known risk factor for coronary artery disease (CAD). Approximately 25 to 30% of patients with DM undergo percutaneous coronary intervention (PCI) at some point in time.[3] Diabetic patients treated with drug-eluting stents (DES) have a lower rate of target vessel revascularization (TVR), subsequent myocardial infarction (MI) and mortality when compared with those receiving bare-metal stents (BMS).[4] DES have evolved rapidly in recent years and real-world studies have revealed better clinical outcomes with second-generation DES, compared to first-generation DES.[5] However, DM is still the main determinant of clinical outcomes in contemporary PCI practice. Therefore, we conducted this retrospective study of real-world cases to compare the overall clinical outcomes after second-generation DES implantation in patients with and without DM.

METHODS

Study Design and Population

In this retrospective cohort study, we enrolled patients aged ≥20 years with CAD attributable to native coronary artery stenosis with de novo lesions, who underwent PCI using second-generation DES at Tainan Municipal Hospital, Taiwan, ROC. Duration of the study period was from April 1, 2009 to March 31, 2019 (Figure 1). The exclusion criteria included a

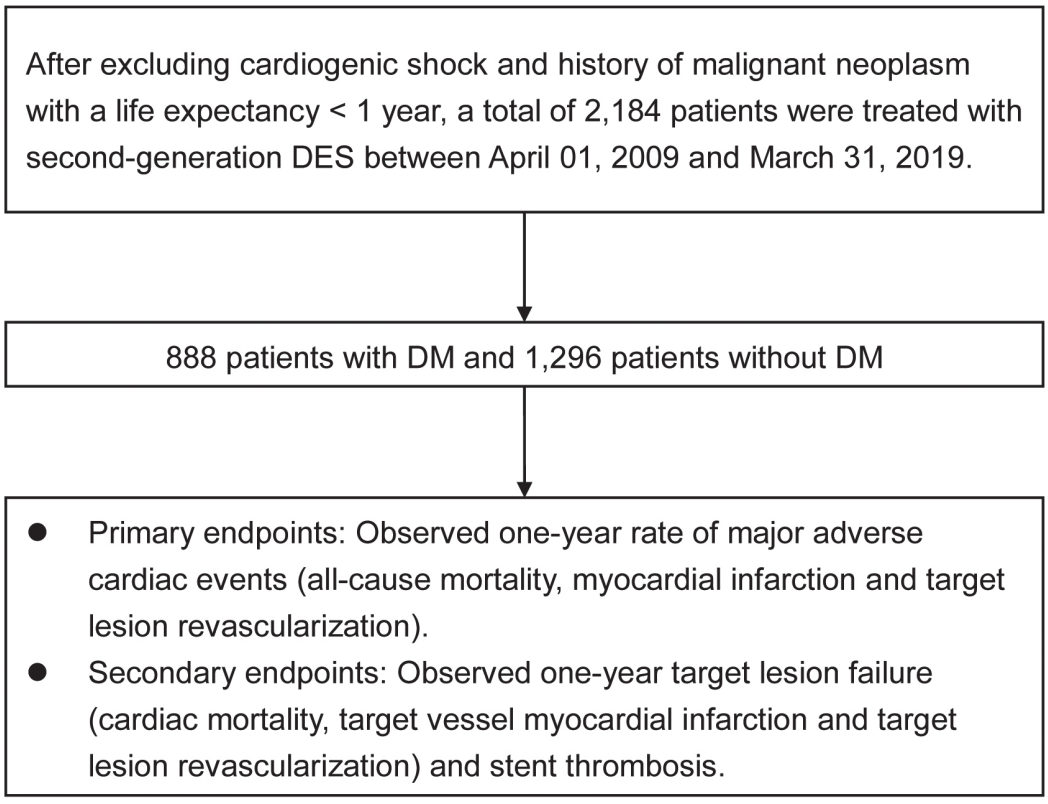

Figure 1. Study flow chart. DES: drug-eluting stents; DM: diabetes mellitus.

history of cardiogenic shock and prior diagnosis of malignancy with a life expectancy < 1 year. The definition of DM was either a previous diagnosis of DM or new DM diagnosed according to the criteria of the American Diabetes Association.[6]

PCI Procedure

Diagnostic coronary angiographies and PCI were performed using standard techniques at each lesion in order to achieve optimal stent apposition. Unfractionated heparin was administered according to the standard regimen during PCI. The use of intravascular ultrasound (IVUS) and post-dilatation with a high-pressure noncompliant balloon for each lesion was at the discretion of each operator. Dual antiplatelet therapy comprising aspirin (100 mg/day) combined with clopidogrel (300 mg loading dose followed by 75 mg once daily) or ticagrelor (180 mg loading dose followed by 90 mg twice daily) was administered before or at the time of PCI and continued for the recommended period of one year.

Definition of Clinical Endpoints

Clinical intake was done during hospitalization with follow-up at one-year intervals after PCI. The primary endpoint of the study was major adverse cardiac events (MACE), defined as a composite of all-cause mortality, MI and clinically driven target lesion revascularization (TLR). The secondary endpoint was target lesion failure (TLF), defined as a composite of cardiac mortality, target vessel-related MI and clinically driven TLR.

The definition of cardiac mortality was death due to cardiac causes, such as MI, heart failure or fatal arrhythmia. The cause of death was considered cardiac related unless a definite noncardiac cause was established.

Target vessel-related MI was MI that could be attributed to the target vessel or could not be clearly attributed to a non-target vessel. We also observed the risk of stent thrombosis (ST), including acute (within 24 hours), subacute (within 30 days) and late (30 days to 12 months). The definition of ST was based on the guidelines of the Academic Research Consortium.[7]

Statistical Analysis

Standard statistical methods were used in this study to compare clinical characteristics and outcomes between two groups of patients. For categorical variables (e.g., presence or absence of diabetes), the Chi-squared test was used to compare the proportions between the groups. For continuous variables (e.g., age, body mass index (BMI)), Student's t-test was used to compare the means between the groups. The results were presented as numbers (proportions) for categorical variables and mean ± standard deviation for continuous variables. To investigate the relationship between diabetes and outcomes, a multivariate Cox proportional hazard regression model was used. This allowed the researchers to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for the primary and secondary composite outcomes (MACE and TLF) after adjusting for potential confounders (age, sex, BMI, hypertension, hyperlipidemia and tobacco smoking). Kaplan-Meier analysis was used to calculate the observed event rate at one year and the cumulative incidence of event-free survival. The log-rank test was used to compare the differences between the two groups. Finally, data analysis was performed using SPSS version 22.0 (IBM Corp., IBM SPSS Statistic for Windows, Armonk, NY, USA). A p-value of less than 0.05 was considered statistically significant.

RESULTS

Baseline clinical characteristics of the study patients

Between April 1, 2009 and March 31, 2019, a total of 2,184 patients with CAD, who had been treated using second-generation DES, were enrolled in this study. The baseline clinical characteristics of the patients, grouped by DM status, are shown in Table 1, whereby 888 patients had DM and 1,296 did not. Overall, there were significant differences between patients with and

Table 1. Baseline clinical characteristics of patients

Characteristic	DM (n=888)	Non-DM (n=1,296)	*p*-value
Age (years)	67.7 ± 10.4	66.2 ± 12.5	<0.001
Male	578 (65.1%)	975 (75.2%)	<0.001
BMI (kg/m2)	26.4 ± 3.8	25.9 ± 3.7	0.004
Hypertension	694 (78.2%)	844 (65.1%)	<0.001
Hyperlipidemia	511 (57.5%)	690 (53.2%)	0.026
Tobacco smoker	206 (23.2%)	394 (30.4%)	<0.001
eGFR < 60 ml/min/1.73m^2	373 (42.0%)	359 (27.7%)	<0.001
ESRD	84 (9.5%)	58 (4.5%)	<0.001
LVEF (%)	62.0 ± 11.7	61.9 ± 10.9	0.837
Clinical presentation			
Stable angina	318 (35.8%)	438 (33.8%)	0.177
Unstable angina	419 (47.2%)	593 (45.8%)	0.270
NSTEMI	101 (11.4%)	133 (10.3%)	0.225
STEMI	60 (6.8%)	162 (12.5%)	<0.001
ACEI or ARB	313 (35.2%)	426 (32.9%)	0.134
β- blockers	124 (14.0%)	263 (20.3%)	<0.001
Statin	689 (77.6%)	1051 (81.1%)	0.026

* Age, BMI and baseline LVEF (%) data are expressed as mean ± standard deviation and other data are expressed as n (%).
* Abbreviations: DM: diabetes mellitus; BMI: body mass index; eGFR: estimated glomerular filtration rate; ESRD: end-stage renal disease; LVEF: left ventricular ejection fraction; NSTEMI: non-ST elevation myocardial infarction; STEMI: ST-elevation myocardial infarction; ACEI: angiotensin-converting enzymes inhibitors; ARB: angiotensin receptor blockers.

without DM, in age, sex and key clinical factors (BMI, hypertension, hyperlipidemia, tobacco smoking, estimated glomerular filtration rate (eGFR) < 60 ml/min/1.73m^2 and end-stage renal disease (ESRD)) and ST-elevation myocardial infarction (STEMI). As baseline medications, Angiotensin Converting Enzyme Inhibitors (ACEI) or Angiotensin Receptor Blockers (ARB) were prescribed similarly for patients with and without DM (p = 0.134). However, the prescription of β-blockers and statins was more common in patients without DM than in patients with DM (p < 0.001 and p = 0.026 respectively).

Baseline characteristics of lesions and procedures

The baseline characteristics of lesions and procedures are shown in Table 2. There were 1,448 lesions among patients with DM and 1,896 lesions among patients without DM. The treated vessels did not differ significantly between the two groups. Regarding the lesion type, there were only two and one type A lesions in patients with DM and without DM, respectively, with no significant difference (p = 0.400), but there were more type B lesions in patients without DM (p < 0.001) and more type C lesions in patients with DM (p < 0.001). Patients without DM had more chronic total occlusion (CTO) lesions (p = 0.013). There was no significant difference in bifurcation lesions between the two groups (p = 0.318). However, patients with DM had a much greater lesion length (p = 0.001) and a much narrower reference vessel diameter (p = 0.003). The use of high-pressure noncompliant balloon post-dilatation was similar for the two groups (p = 0.262), but IVUS was

Table 2. Baseline characteristics of lesions and procedures

Characteristic	DM (n=1,448)	Non-DM (n=1,896)	*p*-value
Target lesion			
LM	60 (4.1%)	85 (4.5%)	0.349
LAD	670 (46.3%)	886 (46.7%)	0.410
LCX	258 (17.8%)	330 (17.4%)	0.395
RCA	460 (31.8%)	587 (31.0%)	0.322
ACC-AHA lesion type			
A	2 (0.1%)	1 (0.1%)	0.400
B	657 (45.4%)	1,000 (52.7%)	<0.001
C	789 (54.5%)	895 (47.2%)	<0.001
CTO	95 (6.6%)	165 (8.7%)	0.013
Bifurcation	293 (20.2%)	370 (19.5%)	0.318
Lesion length (mm)	22.2 ± 9.1	21.2 ± 9.2	0.001
RVD (mm)	2.9 ± 0.5	3.0 ± 0.4	0.003
High-pressure post-dilatation	888 (61.3%)	1,141 (60.2%)	0.262
IVUS	555 (38.3%)	663 (35.0%)	0.025
Length of DES (mm)	25.7 ± 8.6	24.9 ± 8.6	0.009
MLD (mm)			
Pre-PCI	0.50 ± 0.3	0.48 ± 0.4	<0.001
Post-PCI	2.82 ± 0.7	2.84 ± 0.4	0.193
Diameter stenosis (%)			
Pre-PCI	84.2 ± 11.1	85.2 ± 11.7	0.154
Post-PCI	2.75 ± 24.1	3.64 ± 6.62	0.138

* Data are expressed as mean ± standard deviation or as n (%).
* Abbreviations: DM: diabetes mellitus; LM: left main; LAD: left anterior descending; LCX: left circumflex; RCA: right coronary artery; ACC: American College of Cardiology; AHA: American Heart Association; CTO: chronic total occlusion; RVD: reference vessel diameter; IVUS: intravascular ultrasound; DES: drug-eluting stents; MLD: minimal lumen diameter; PCI: percutaneous coronary intervention.

used more frequently in DM patients, compared to non-DM patients (p = 0.025). Notably, the length of DES was greater in patients with DM than in those without DM (p = 0.009).

Clinical outcomes

Table 3 shows the primary (MACE) and secondary (TLF and ST) clinical outcomes at the one-year follow-up. The incidence rates of MACE and TLF after one year were significantly higher in patients with DM than in those without DM. MACE occurred in 6.0% of patients with DM and in 2.7% of patients without DM (p < 0.001). TLF occurred in 2.4% of patients with DM and in 0.8% of patients without DM (p = 0.006). Furthermore, all-cause mortality, including cardiogenic and non-cardiogenic death, was also significantly higher in patients with DM. However, there was no significant difference in the incidence of MI and TLR in patients with DM compared to those without. There were no cases of ST in the two groups within one year.

Unadjusted and adjusted analyses of the primary composite clinical outcome of MACE and its components at one year are shown in Table 4 and the secondary composite clinical outcome of TLF and its components at one year are shown in Table 5. After adjustment, we found a significantly higher risk of MACE and all-cause mortality in patients with DM, compared to those without DM (MACE: adjusted HR = 2.342 (95% CI: 1.311 - 4.184), p = 0.004; all-cause mortality: adjusted HR = 2.390 (95% CI: 1.319 - 4.329), p = 0.004). There was no significant difference in the risk of MI and TLR (TLR adjusted HR = 2.668 (95% CI: 0.237 - 30.058), p = 0.427). However, there was also no significant difference in TLF and its components in patients with DM, compared to those without DM (TLF: adjusted HR = 2.893 (95% CI: 0.980 - 8.543), p = 0.054; cardiac mortality: adjusted HR = 2.954 (95% CI: 0.880 - 9.918), p = 0.080; TLR adjusted HR = 2.668 (95% CI: 0.237 - 30.058), p = 0.427).

The one-year cumulative MACE- and TLF-free survival rates are shown in Figure 2. There were significant differences in MACE-free survival (Figure 2A: log-rank test, p = 0.002) and TLF-free survival (Figure 2B: log-rank test, p = 0.006).

Table 3. Clinical outcomes at one-year follow-up in patients with DM and patients without DM

	DM (N=888)	Non-DM (N=1,296)	*p*-value
MACE	53 (6.0%)	35 (2.7%)	<0.001
TLF	21 (2.4%)	10 (0.8%)	0.006
All-cause mortality	51 (5.7%)	34 (2.6%)	0.001
Cardiac	18 (2.0%)	9 (0.7%)	0.013
Non-cardiac	33 (3.7%)	25 (1.9%)	0.027
MI	3 (0.3%)	1 (0.08%)	0.232
Culprit (target vessel MI)	1 (0.1%)	0 (0%)	0.444
Non-culprit	2 (0.2%)	1 (0.08%)	0.416
TLR	3 (0.3%)	1 (0.08%)	0.187
Stent thrombosis	0	0	-

* Data are expressed as the number of clinical outcomes, N (%).
* Abbreviations: MACE: major adverse cardiac events; TLF: target lesion failure; MI: myocardial infarction; TLR: target lesion revascularization.

Table 4. Hazard ratios of one-year event rates for MACE by presence of diabetes mellitus

	DM vs Non-DM			
	Unadjusted HR (95% CI)	*p*-value	Adjusted HR (95% CI)	*p*-value
MACE	2.135 (1.396 - 3.266)	0.000	2.342 (1.311 - 4.184)	0.004
All-cause mortality	2.112 (1.371 - 3.252)	0.001	2.390 (1.319 - 4.329)	0.004
MI	4.457 (0.464 - 42.849)	0.196	0.001 (0.000 - >10)	NA
TLR	4.458 (0.464 - 42.861)	0.195	2.668 (0.237 - 30.058)	0.427

* Values expressed as unadjusted HR (95% CI) and adjusted HR (95% CI). Adjusted HR was estimated by multivariate Cox proportional hazard regression model after adjustment for age, sex, body mass index, hypertension, hyperlipidemia and tobacco smoking.
* Abbreviations: MACE: major adverse cardiac events; DM: diabetes mellitus; HR: hazard ratio; CI: confidence interval; MI: myocardial infarction; NA: not available; TLR: target lesion revascularization.

Table 5. Hazard ratios of one-year event rates for TLF by presence of diabetes mellitus

	DM vs Non-DM			
	Unadjusted HR (95% CI)	p-value	HR (95% CI)	p-value
TLF	2.692 (1.290 - 5.618)	0.008	2.893 (0.980 - 8.543)	0.054
Cardiac mortality	2.515 (1.152 - 5.493)	0.021	2.954 (0.880 - 9.918)	0.080
Target vessel MI	104.143 (0.000 - >10)	0.585	NA	NA
TLR	4.458 (0.464 - 42.861)	0.195	2.668 (0.237 - 30.058)	0.427

* Values expressed as unadjusted HR (95% CI) and adjusted HR (95% CI). Adjusted HR was estimated by multivariate Cox proportional hazard regression model after adjustment for age, sex, body mass index, hypertension, hyperlipidemia and tobacco smoking.
* Abbreviations: TLF: target lesion failure; DM: diabetes mellitus; HR: hazard ratio; CI: confidence interval; MI: myocardial infarction; NA: not available; TLR: target lesion revascularization.

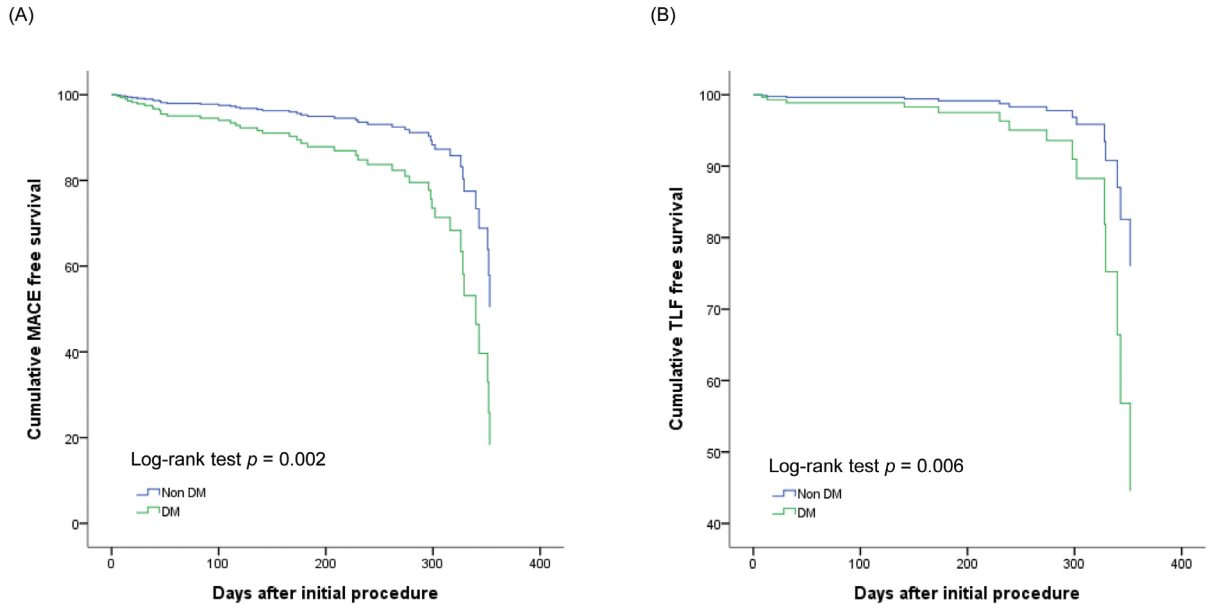

Figure 2. The Kaplan-Meier curves of cumulative incidence of (A) MACE-free and (B) TLF-free survival among patients with DM and without DM at one-year follow-up after implantation of second-generation DES. MACE: major adverse cardiovascular events; TLF: target lesion failure; DM: diabetes mellitus.

DISCUSSION

The major findings of our study were: (1) At one-year follow-up, after adjustment, the risks of MACE and all-cause mortality were significantly higher in patients with DM who had undergone second-generation DES; (2) At one-year follow-up, after adjustment, the risks of MI, TLR, TLF, cardiac mortality and target-vessel MI did not differ significantly between DM and non-DM patients who had undergone second-generation DES; (3) The cumulative MACE-free and TLF-free survival rates were significantly lower in patients with DM in the early (0-1 year) period after second-generation DES implantation.

In 1979, Kannel et al. utilized the data

from the Framingham heart study (FHS) to confirm that DM was a major risk factor for cardiovascular disease (CVD).[8] It is particularly worth mentioning that DM is a well-known strong risk factor for CAD and acute myocardial infarction (AMI). DM patients are at high risk for AMI, cardiac mortality, in-stent restenosis and ST after PCI. Approximately one-third of DM patients receive PCI procedures in the United States of America, whereby the clinical outcomes after PCI procedures are relatively ineffective compared to those without DM.[9] In our study, we found that patients with DM experienced higher incidence of MACE (DM vs. non-DM = 6.0% vs. 2.7%) and all-cause mortality (DM vs. non-DM = 5.7% vs. 2.6%). Our result was in accordance with other studies. Asmir I. Syed showed that DM patients have a relatively high incidence of MACE (DM vs. non-DM = 18.5% vs. 9.4%) and all-cause mortality (DM vs. non-DM = 12.9% vs. 4.6%) at the one-year follow-up after DES implantation.[10] Yong-Jin Jian et al. also showed that DM patients have a relatively high incidence of MACE (DM vs. non-DM 12.5% vs. 5.0%) and all-cause mortality (DM vs. non-DM = 5.0% vs. 2.2%) at the two-year follow-up after DES implantation.[11] In addition, compared to second-generation DES, first-generation DES have a similar MACE rate (HR = 0.89 (95% CI: 0.6 - 1.31), p = 0.57) and a higher risk of ST (HR = 5.75 (95% CI: 1.16 - 28.47), p = 0.03) at the one-year follow-up, in patients with DM.[12] However, Cheol Hyun Lee et al. found that even in the era of second-generation DES, DM patients had significantly higher risk of MACE (HR = 3.04 (95% CI: 1.97 – 4.68), p < 0.001) after adjustment, compared to patients without DM, in the early period (0-1 year) after DES implantation.[13] Similar results were also found in our study (DM vs. Non-DM: MACE (adjusted HR = 2.342, (95% CI: 1.311 - 4.184), p = 0.004). It is worth noting that the reduction in the risk of death and AMI was most obvious within the first six months before maximum impact of the restenosis prevention. This phenomenon may be due to neointimal hyperplasia over several months after DES implantation, and following angiography at around 9 months.

TLF is the key parameter as regards efficacy and safety, and reflects any lesion- and device- associated adverse clinical events that occur during follow-up. Second-generation DES have significantly decreased the rate of in-stent restenosis (ISR) and TLR, compared with first-generation DES. While second-generation DES do offer a high level of safety and efficacy, the BIOSCIENCE trial confirmed that DM patients remain at increased risk of TLF, mainly due to higher rates of TLR, compared to patients without DM.[14] The BIONICS Randomized Trial also showed that DM patients had significantly higher risk of TLF (HR = 1.86, (95% CI: 1.25 – 2.76), p = 0.002) at the one-year follow-up.[15] However, in our study there was no significant difference in TLF (adjusted HR = 2.893 (95% CI: 0.980 - 8.543), p = 0.054) and its components in patients with DM, compared to those without DM. In DM patients, the higher rates of repeat revascularization after PCI are mainly caused by both ISR and disease progression. The leading cause of ISR is an obviously accelerated neointimal hyperplasia in combination with endothelial dysfunction, vascular inflammation, and the growth factor-like effect of insulin on vascular smooth muscle and neointimal cells.[15] Thus, second-generation DES play an important role in ISR since more advanced technology allows for the manufacture of thinner struts with more biocompatible polymers, which can result in greater deliverability and flexibility, reduce vascular inflammation, decrease neointimal proliferation and accelerate endothelization. Thus, the technology can provide sufficient arterial repair after DES implantation.[16]

Patients with DM are mostly at risk of ST even though the incidence of ST is quite low (< 0.1%) for all types of second-generation DES. The increased ST risk in patients with DM may be in part related to a hypercoagulable state because DM patients have high blood glucose, endothelial

dysfunction, platelet hyperactivity, elevated vascular shear forces and oxidative stress which could create a perilous concurrence of risk factors for ST.[17] Yong Hoon Kim et al. found that while DM patients had a higher incidence of ST (1.0%), this was not the case for patients with prediabetes (0.6%) and patients with normoglycemia (0.5%).[18] A systematic review and meta-analysis that compared early and late ST in DM and non-DM patients following PCI with DES showed that DM patients had a higher rate of late ST than non-DM patients (odds ratio (OR) = 1.95, (95% CI: 1.35 - 2.81), p = 0.0004), but a similar rate of early ST (OR = 1.30, (95% CI: 0.89 - 1.91), p = 0.18).[19] Inadequate stent expansion has also been associated with ST, whereby optimal stent apposition and expansion can be evaluated via IVUS.[20] By contrast, IVUS-guided high-pressure noncompliant balloon post-dilatation after DES implantation can improve stent expansion and decrease the risk of ISR and ST.[21] However, in our study, there was no acute, sub-acute or late ST among DM and non-DM patients at the one-year follow-up after PCI with second-generation DES. The study by Abdurrahman Tasal et al. also demonstrated that high-pressure noncompliant post-dilatation seems to optimize stent expansion and decrease the risk of ST at the 6-month follow-up after PCI.[22] In our study, the high usage of high-pressure noncompliant balloon post-dilatation of second-generation DES played an integral role in the PCI procedure, and also achieved maximal luminal area and optimal stent expansion.

Finally, significantly lower rates of one-year cumulative MACE-free survival and TLF-free survival were observed in DM patients, compared to non-DM patients. However, multiple factors are associated with clinical outcomes in DM patients who have undergone second-generation DES implantation, including age, poor glycemic control, high BMI, chronic kidney disease (CKD), multiple lesions, longer lesion length, small vessel size, type of DES, tobacco smoking and genetic factors etc.[23] Further detailed research is warranted to understand the development of clinical outcomes and proper treatment in DM patients receiving second-generation DES.

STUDY LIMITATIONS

Several limitations of this study must be addressed. (1) The study was a non-randomized retrospective cohort trial, which had some missing data, such as type of DM (type 1 or 2), prior duration of DM and level of HbA1c. (2) A long-term follow-up is preferred because it is an important strength of the study. With longer follow-up duration the DM status may change in some patients and potentially dilute findings regarding the comparison between DM and non-DM patients.[24] (3) We did not collect data about oral antidiabetic agents and insulin for DM treatment during the study. The RESET and NEXT trials demonstrated that second-generation DES have a significantly lower adjusted HR for TLR compared with first-generation DES in insulin-treated DM patients (adjusted HR = 0.54 (95% CI 0.32–0.96), p = 0.04),[25] so dividing between insulin-dependent and non-insulin-dependent DM patients could provide additional evidence for the optimal treatment of DM patients who have undergone PCI with second-generation DES, and thus enrich our study. (4) We did not collect specific data on the type of second-generation DES, so we could not compare the safety and efficacy among different second-generation DES. Yujin Yang et al. found that there are no significant differences in MACE over three years among different types of second-generation DES.[26] (5) The use of IVUS and post-dilatation with a high- pressure noncompliant balloon for each lesion was at the discretion of each operator, and thus, was subject to selection bias. (6) The data reflected our own experience in Tainan Municipal Hospital (Managed by Show Chwan Medical Care Corporation). We need further study with pooled results from multiple centers to confirm our results. (7) Similar to the RESOLUTE Japan Small Vessel Study,[27] our

study did not report any definite ST. However, it is important to note that not every patient in real world treatment receives routine follow-up catheterization laboratory examinations within one year after the initial procedure. Therefore, it is necessary to observe the clinical outcomes of coronary intervention over a longer period of time.

CONCLUSIONS

This single-center, one-year follow-up study demonstrated that, despite being treated with state-of-the-art second-generation DES technology, DM patients still face a significantly higher risk of MACE. Therefore, for patients with DM, it is necessary to evaluate the risk factors within one year after PCI with second-generation DES. It is therefore recommended to consider a long-term follow-up clinical trial on the usage of the newly developed DES.

CONFLICT OF INTEREST

All authors declare no conflicts of interest

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