Incidence and Impact of Thrombocytopenia in Patients Undergoing Percutaneous Coronary Intervention With Drug-Eluting Stents

Sangwoo Park, MD[a], Jung-Min Ahn, MD[b], Tae Oh Kim, MD[b], Hanbit Park, MD[b], Sang-Cheol Cho, MD[b], Do-Yoon Kang, MD[b], Pil Hyung Lee, MD[b], Duk-Woo Park, MD[b,*], and Seung-Jung Park, MD[b], for the IRIS-DES Registry Investigators

Platelets are crucial in the pathophysiology of coronary artery disease and are a major target of antithrombotic agents in patients receiving percutaneous coronary intervention (PCI). We sought to evaluate the incidence and prognostic impact of thrombocytopenia on clinical outcomes in patients undergoing PCI with drug-eluting stents (DES). We evaluated consecutive patients who received PCI with DES in the IRIS-DES registry between April 2008 and December 2017. Patients were divided into 2 groups based on the presence of thrombocytopenia (platelet count $<150 \times 10^9$/L) at baseline. The primary outcome was all-cause mortality, and secondary outcomes included the composite outcome of death, myocardial infarction (MI), and stroke, and major bleeding. Complete follow-up data were available for 1 to 5 years (median, 3.1). Among 26,553 eligible patients, 1,823 (6.9%) had thrombocytopenia at baseline. At 5 years, the incidences of all-cause mortality (15.6% vs 8.1%, p <0.001), composite outcome (23.2% vs 15.6%, p <0.001), and major bleeding (3.7% vs 2.2%, p <0.001) were significantly higher in patients with thrombocytopenia than in those without thrombocytopenia. In multivariable Cox proportional-hazards models, thrombocytopenia was significantly associated with increased risks of all-cause mortality (hazard ratio 1.26, 95% confidence interval 1.07 to 1.48, p = 0.01) and major bleeding (hazard ratio 1.41, 95% confidence interval 1.04 to 1.91, P=0.03). In conclusion, among who patients underwent PCI with DES, the incidence of thrombocytopenia was 6.9%. Baseline thrombocytopenia was significantly associated with increased risks of mortality and major bleeding. **(Am J Cardiol 2020;134:55−61)**

Platelets play a crucial role in the pathophysiology of thrombosis and hemorrhage, which are major concerns in the management of patients undergoing percutaneous coronary intervention (PCI).[1] Thrombocytopenia is a not uncommon abnormality in a routine blood assay. Several studies reported the association between acquired thrombocytopenia and adverse clinical outcomes in patients with acute coronary syndrome (ACS).[2−4] However, data on the incidence and clinical impact of thrombocytopenia at baseline in patients receiving PCI are lacking. Furthermore, previous studies were limited in that they only included highly selected patients with high-risk ACS or myocardial infarction (MI), had relatively short follow-up periods, and were conducted in the past era of bare-metal stents or with substantial proportion of bare-metal stents.[4−9] We, therefore, sought to determine the incidence, risk factors, and prognostic impact of baseline thrombocytopenia on long-term clinical outcomes using the data from the real-world registry of patients undergoing PCI with drug-eluting stents (DES).

Methods

The study population was derived from the IRIS-DES (Interventional Cardiology Research In-Cooperation Society-Drug-Eluting Stents) registry. The design of the IRIS-DES registry and the associated ongoing analyses has been described.[10−14] In brief, the IRIS-DES is a prospective, multicenter registry of consecutive patients who underwent PCI with different types of DES in 46 academic and community hospitals in Korea between April 1, 2008 and December 31, 2017, and for whom follow-up data were available for at least 1 year and up to 5 years. The exclusion criteria were minimal. Patients with cardiogenic shock, malignant disease, or other co-morbid conditions with a life expectancy of <12 months, those treated with a mixture of different DES types, and those with a planned surgery necessitating the interruption of antiplatelet drugs within 6 months post-PCI were excluded. In the present study, we

[a]Department of Cardiology, Ulsan University Hospital, University of Ulsan College of Medicine, Ulsan, South Korea; and [b]Department of Cardiology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, South Korea. Manuscript received May 14, 2020; revised manuscript received and accepted July 28, 2020.

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*Corresponding author: Tel: +82-2-3010-3995; fax: +82-2-475-6898.

E-mail address: dwpark@amc.seoul.kr (D.-W. Park).

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also excluded patients in whom baseline laboratory data were unavailable.

Patients were categorized according to the presence of thrombocytopenia (baseline platelet count $<150 \times 10^9$/L). Patients with thrombocytopenia were further stratified into mild ($\geq$100 and $<150 \times 10^9$/L), moderate ($\geq$50 and $<100 \times 10^9$/L), and severe ($<50 \times 10^9$/L) based on previous studies and clinical relevance.[7,8,15–17] The ethics committee at each participating center approved the study protocol, which is registered in ClinicalTrials.gov (NCT01186133). All patients provided written informed consent for participation.

PCI was performed with standard techniques according to local practice. Before or during the procedure, all patients were given loading dose of aspirin and $P2Y_{12}$ receptor inhibitor. Heparin was administered throughout the procedure to maintain an activated clotting time of 250 seconds. The operator was responsible for deciding the treatment strategy including the use of intravascular ultrasound and the administration of glycoprotein IIb/IIIa inhibitors. After the procedure, aspirin was recommended indefinitely and $P2Y_{12}$ receptor inhibitor was prescribed for at least 12 months unless contraindicated. Other cardiac drugs were prescribed at the discretion of the attending physicians.

Clinical follow-up was conducted during hospitalization, at 1, 6, and 12 months, and every 6 months thereafter. Detailed information on baseline clinical or procedural characteristics and outcome data was collected by specialized personnel at each participating center using a dedicated electronic case report form as previously described.[10,14] Monitoring and verification of registry data were periodically performed in the participating hospitals by members of the academic coordinating center (Clinical Research Center, Asan Medical Center, Seoul, South Korea).

The primary outcome was all-cause mortality. The secondary outcomes included death from cardiac or noncardiac causes, MI (any, periprocedural, or spontaneous), stroke, stent thrombosis, major bleeding, and the composite of death, MI, and stroke.

The adjudication of the cause of death was performed according to the prespecified criteria. Death was considered to have cardiac causes unless a clear noncardiac cause could be established. The prespecified definition of MI was based on the universal definition of MI.[10,18] Procedure-related MI was defined as the development of new Q waves and either an elevation in the creatinine kinase-MB (CK-MB) fraction or troponin I concentration >3 times the upper limit, and spontaneous MI was defined as any CK-MB or troponin increase above the upper range limit with ischemic symptoms or signs. Stroke was defined as a neurologic deficit lasting more than 24 hours and was confirmed by a neurologist based on imaging. Definite or probable stent thrombosis was assessed according to the Academic Research Consortium criteria.[19] Major bleeding was assessed by the definition of the Thrombolysis in Myocardial Infarction classification,[20] in which intracranial bleeding, hemoglobin level drop $\geq$5 g/dl or fatal bleeding were considered as major bleeding events. For a more accurate assessment of clinical outcomes, additional information was obtained from telephone interviews and medical records obtained from other hospitals as necessary. All end points were confirmed by source documentation collected at each hospital and were adjudicated by independent clinicians who were unaware of the study purpose.

Baseline characteristics of the study population, including patient demographics, risk factors or co-morbidities, clinical presentation, and anatomic/procedural features were compared between patients with or without thrombocytopenia at baseline. Continuous variables were reported as mean $\pm$ standard deviation and compared using the Student's *t* test or the Wilcoxon rank sum test as appropriate. Categorical variables were presented as number (proportion) and compared using either the Pearson's chi-square test or the Fisher's exact test, as appropriate.

Multivariable logistic regression was used to identify risk factors associated with the presence of thrombocytopenia at baseline. Cumulative events of clinical outcomes were evaluated based on Kaplan−Meier estimates and compared using the log-rank test. All analyses were truncated at 5 years of follow-up owing to different follow-up duration according to the types of DES. To explore the relation between thrombocytopenia and long-term clinical outcomes, a multivariable Cox proportional hazard model was used to adjust for the differences in baseline characteristics and clinically relevant covariates. Multivariable analysis included covariates that were statistically significant on a univariable Cox proportional hazard model and baseline characteristics that were significantly different between groups or those associated with baseline thrombocytopenia. Finally, independent predictors of primary outcome of all-cause mortality were also identified by using stepwise regressions with backward elimination.

All reported p Values were 2-sided and $p < 0.05$ was considered statistically significant. No adjustments were made for multiple comparisons. All analyses were performed with the use of SPSS software, version 22 (SPSS Inc, Chicago, Illinois).

Results

A total of 27,133 patients underwent PCI with DES during the study period. After the exclusion of 580 patients without results on complete blood assay, 26,553 patients were included in the current analysis. Among them, 1,823 (6.9%) patients had thrombocytopenia at baseline. Baseline clinical, angiographic, and procedural characteristics are shown in Table 1. The mean platelet counts ($\times 10^9$/L) in patients with or without thrombocytopenia were 125.0 $\pm$ 26.4 and 238.4 $\pm$ 73.3, respectively. Compared with patients without thrombocytopenia, those with thrombocytopenia were older, more male sex and more likely to have higher risk-profiles of clinical and angiographic characteristics. Although stent procedures were similar in both groups, complete revascularization was less frequently achieved in patients with thrombocytopenia. Discharge and follow-up medications are summarized in Table 2. At discharge, aspirin use was slightly less frequent in patients with thrombocytopenia. Although the proportion of $P2Y_{12}$ use was similar in both groups, the use of more potent $P2Y_{12}$ inhibitors (i.e., ticagrelor and prasugrel) was less common in patients with thrombocytopenia. The duration of dual

Table 1
Baseline clinical and procedural patient characteristics according to the presence of thrombocytopenia at baseline

Variable	Thrombocytopenia ($<150 \times 10^9$/L)		p Value
	Yes (n = 1823)	No (n = 24730)	
Age (years)	67.9 ± 10.2	63.8 ± 10.9	<0.001
Men	1459 (80.0%)	17340 (70.1%)	<0.001
Body mass index (kg/m^2)	24.4 ± 3.2	24.8 ± 3.2	<0.001
Platelet count ($\times 10^9$/L)	125.0 ± 26.4	238.4 ± 73.3	<0.001
Hypertension	1197 (65.7%)	15292 (61.8%)	0.001
Diabetes mellitus	732 (40.2%)	8102 (32.8%)	<0.001
Diabetes mellitus on insulin	114 (6.3%)	989 (4.0%)	<0.001
Hyperlipidemia	822 (45.1%)	12405 (50.2%)	<0.001
Smoker	369 (20.2%)	7113 (28.8%)	<0.001
Previous myocardial infarction	137 (7.5%)	1346 (5.4%)	<0.001
Previous percutaneous coronary intervention	369 (20.2%)	3325 (13.4%)	<0.001
Previous coronary artery bypass grafting	59 (3.2%)	408 (1.6%)	<0.001
Previous cerebrovascular accident	144 (7.9%)	1719 (7.0%)	0.14
Peripheral artery disease	50 (2.7%)	424 (1.7%)	0.002
Chronic lung disease	38 (2.1%)	564 (2.3%)	0.64
Chronic renal failure	189 (10.4%)	833 (3.4%)	<0.001
Dialysis	118 (6.5%)	348 (1.4%)	<0.001
Congestive heart failure	63 (3.5%)	536 (2.2%)	<0.001
Ejection fraction (%)	57.3 ± 10.7	58.5 ± 10.0	<0.001
Ejection fraction <40%	139 (7.6%)	1343 (5.4%)	<0.001
Atrial fibrillation	139 (7.6%)	739 (3.0%)	<0.001
Clinical indication			<0.001
Stable angina pectoris	828 (45.4%)	10138 (41.0%)	
Unstable angina pectoris	566 (31.0%)	7639 (30.9%)	
Non-ST elevation myocardial infarction	257 (14.1%)	3682 (14.9%)	
ST elevation myocardial infarction	172 (9.4%)	3271 (13.2%)	
Angiographic and procedural characteristics			
No. of narrowed coronary arteries			0.02
1	879 (48.2%)	12711 (51.4%)	
2	583 (32.0%)	7618 (30.8%)	
3	361 (19.8%)	4401 (17.8%)	
Left main disease	157 (8.6%)	1742 (7.0%)	0.01
Bifurcation disease	474 (26.0%)	6528 (26.4%)	0.73
American College of Cardiology/American Heart Association Type B2, C lesion	1411 (77.4%)	18737 (75.8%)	0.12
Moderate to severe calcium	220 (12.1%)	2398 (9.7%)	0.001
In stent restenosis	96 (5.3%)	898 (3.6%)	<0.001
Treated vessel, Left main	128 (7.0%)	1515 (6.1%)	0.14
Treated vessel, Left anterior descending	1130 (62.0%)	16202 (65.5%)	0.002
Treated vessel, Left circumflex	523 (28.7%)	6820 (27.6%)	0.32
Treated vessel, Right	679 (37.2%)	8671 (35.1%)	0.06
Stents per patient	1.6 ± 0.9	1.6 ± 0.9	0.70
Average stent diameter (mm)	3.2 ± 0.4	3.2 ± 0.4	0.87
Total stent length (mm)	30.8 ± 16.0	30.3 ± 15.8	0.17
Use of glycoprotein IIb/IIIa inhibitors	44 (2.4%)	887 (3.6%)	0.01
Femoral approach	1026 (56.3%)	13991 (56.6%)	0.83
Intravascular ultrasound guidance	783 (43.0%)	11020 (44.6%)	0.19
Complete revascularization	118 (64.7%)	16948 (68.5%)	0.003

Data are mean ± SD or number (%).

antiplatelet therapy (DAPT) was significantly shorter in patients with thrombocytopenia. Several key clinical factors were identified as independent predictors of thrombocytopenia at baseline (Table 3).

During the 1 to 5 years of follow-up (median, 3.1 years), there were 1,400 deaths (891 cardiac and 509 noncardiac), 1,685 MIs (1,459 periprocedural and 227 spontaneous), 488 strokes, 110 definite or probable stent thromboses, and 448 major bleeding events. The observed incidence and the Kaplan-Meier event curves of the clinical outcomes stratified by the presence of thrombocytopenia are shown in Table 4 and Figure 1, respectively. The 5-year incidence of primary outcome of all-cause mortality was significantly higher in patients with thrombocytopenia than in those

Table 2
Medications at discharge and follow-up

Variable	Thrombocytopenia (<150 × 10^9/L)		p Value
	Yes (n = 1823)	No (n = 24730)	
Medications at discharge			
Aspirin	1778 (97.5%)	24334 (98.4%)	0.007
$P2Y_{12}$ inhibitor	1761 (96.6%)	23997 (97.0%)	0.32
Clopidogrel	1656 (90.8%)	21785 (88.1%)	<0.001
Ticagrelor	93 (5.1%)	1984 (8.0%)	<0.001
Prasugrel	23 (1.3%)	473 (1.9%)	0.06
Dual antiplatelet therapy on discharge	1743 (95.6%)	23823 (96.3%)	0.13
Cilostazol	197 (10.8%)	2910 (11.8%)	0.23
Beta blocker	1082 (59.4%)	15080 (61.0%)	0.18
Calcium channel blocker	605 (33.2%)	8813 (35.6%)	0.04
Angiotensin-converting enzyme inhibitor or angiotensin II receptor blocker	934 (51.2%)	12957 (52.4%)	0.35
Vitamin K antagonist	51 (2.8%)	319 (1.3%)	<0.001
Statin	1463 (80.3%)	21214 (85.8%)	<0.001
Medications on follow-up*			
Dual antiplatelet therapy duration, years	1.8 ± 1.4	1.9 ± 1.5	<0.001
Ongoing dual antiplatelet therapy at 1-month	1704 (95.6%)	23483 (96.3%)	0.11
Ongoing dual antiplatelet therapy at 6-months	1405 (82.9%)	20462 (87.3%)	<0.001
Ongoing dual antiplatelet therapy at 1-year	957 (66.1%)	14109 (69.8%)	0.003

Data are mean ± SD or number (%).
* Note that proportion of ongoing dual antiplatelet therapy is the proportion of patients who are taking dual antiplatelet therapy among patients survived at that point.

Table 3
Independent predictors of baseline thrombocytopenia

Variables	Odds ratio (95% confidence interval)	p Value
Age	1.04 (1.04−1.05)	<0.001
Male sex	2.45 (2.16−2.78)	<0.001
Acute coronary syndrome	0.88 (0.80−0.97)	0.009
Diabetes mellitus	1.22 (1.10−1.35)	<0.001
Chronic renal failure	2.64 (2.21−3.14)	<0.001
Chronic lung disease	0.64 (0.46−0.90)	0.01
Smoker	0.71 (0.63−0.81)	<0.001
Hyperlipidemia	0.81 (0.73−0.89)	<0.001
Prior coronary artery bypass grafting	1.46 (1.10−1.95)	0.01
Prior percutaneous coronary intervention	1.42 (1.25−1.60)	<0.001
Atrial fibrillation	2.07 (1.70−2.51)	<0.001

without thrombocytopenia (15.6 % vs 8.1%, p < 0.001). The incidences of cardiac and noncardiac death were significantly higher in patients with thrombocytopenia. In addition, the incidences of stroke (3.5% vs 2.6%, p = 0.02), composite of death, MI and stroke (23.2 % vs 15.6 %, p <0.001), and major bleeding (3.7% vs 2.2%, p <0.001) were also significantly higher in patients with thrombocytopenia. The incidences of MI and stent thrombosis were similar between the 2 groups. After multivariable adjustment of baseline covariates, the presence of thrombocytopenia was significantly associated with increased risks of all-cause mortality (hazard ratio [HR] 1.26, 95% confidence interval [CI] 1.07 to 1.48, p = 0.01) and major bleeding (HR 1.41, 95% CI 1.04 to 1.91, p = 0.03). The association of thrombocytopenia with all-cause mortality and major bleeding was maintained after further adjustment for the duration of DAPT (Supplemental Table 1). Baseline thrombocytopenia tended to be associated with an increased risk of the composite of death, MI, and stroke (HR 1.12, 95% CI 1.00 to 1.27, p = 0.06). However, baseline thrombocytopenia was not associated with the risks for cardiac death or thrombotic events.

We further assessed the prognostic impact of thrombocytopenia by stratifying into mild or moderate to severe thrombocytopenia (Supplemental Table 2 and Supplemental Figure S1). Compared with patients without thrombocytopenia, those with mild thrombocytopenia had a higher adjusted risk of all-cause mortality (HR 1.20, 95% CI 1.01 to 1.44, p = 0.04) but not of major bleeding (HR 1.22, 95% CI 0.86 to 1.72, p = 0.26). However, patients with moderate-to-severe thrombocytopenia had significantly higher risks of all-cause mortality (HR 1.59, 95% CI 1.10 to 2.29, p = 0.01) and major bleeding (HR 2.72, 95% CI 1.52 to 4.87, p = 0.001).

The independent predictors of all-cause mortality are shown in Supplemental Table 3. The presence of thrombocytopenia at baseline was an independent predictor of all-cause mortality, and this association was more pronounced in patients with moderate-to-severe thrombocytopenia. Meanwhile, mild thrombocytopenia was not an independent predictor of major bleeding, but moderate-to-severe thrombocytopenia was the most important predictor of major bleeding (Supplemental Table 4).

Discussion

The present study evaluated the incidence and long-term clinical impact of baseline thrombocytopenia in patients

Table 4
Event rates and hazard ratios for clinical outcomes according to the presence of baseline thrombocytopenia

Outcomes	No. of events (%) at 5-Years		Unadjusted		Multivariable-adjusted*	
	Thrombocytopenia		Hazard ratio (95% confidence interval)	p Value	Hazard ratio (95% confidence interval)	p Value
	Yes (n = 1,823)	No (n = 24,730)				
All-cause mortality	176 (15.6%)	1224 (8.1%)	2.07 (1.77−2.43)	<0.001	1.26 (1.07−1.48)	0.01
Cardiac death	104 (9.7%)	787 (5.3%)	1.91 (1.55−2.34)	<0.001	1.18 (0.95−1.45)	0.13
Non-cardiac death	72 (6.6%)	437 (2.9%)	2.37 (1.85−3.04)	<0.001	1.38 (1.07−1.78)	0.02
Myocardial infarction	121 (8.0%)	1564 (6.9%)	1.06 (0.88−1.27)	0.55	1.00 (0.83−1.21)	0.99
Periprocedural	100 (5.8%)	1359 (5.6%)	1.00 (0.82−1.22)	0.99	0.97 (0.79−1.19)	0.78
Spontaneous	21 (2.2%)	206 (1.3%)	1.46 (0.93−2.28)	0.10	1.21 (0.77−1.92)	0.41
Stroke	45 (3.5%)	443 (2.6%)	1.45 (1.07−1.97)	0.02	1.13 (0.82−1.54)	0.45
Composite of death, myocardial infarction, and stroke	304 (23.2%)	2941 (15.6%)	1.45 (1.29−1.64)	<0.001	1.12 (1.00−1.27)	0.06
Stent thrombosis	4 (0.6%)	106 (0.6%)	0.54 (0.20−1.45)	0.22	0.52 (0.19−1.41)	0.20
Major bleeding	49 (3.7%)	399 (2.2%)	1.74 (1.29−2.33)	<0.001	1.41 (1.04−1.91)	0.03

Hazard ratios are for the patients with thrombocytopenia (<150 × 10^9/L) compared with those without thrombocytopenia.

* Hazard ratios were adjusted for age, sex, body mass index, hypertension, diabetes, hyperlipidemia, smoking, previous percutaneous coronary intervention, previous coronary artery bypass grafting, previous cerebrovascular accident, peripheral artery disease, chronic renal failure, chronic lung disease, congestive heart failure, ejection fraction <40%, atrial fibrillation, acute coronary syndrome, extent of diseased vessel, left main disease, total stent length >40 mm, transfemoral approach, use of statin, and complete revascularization.

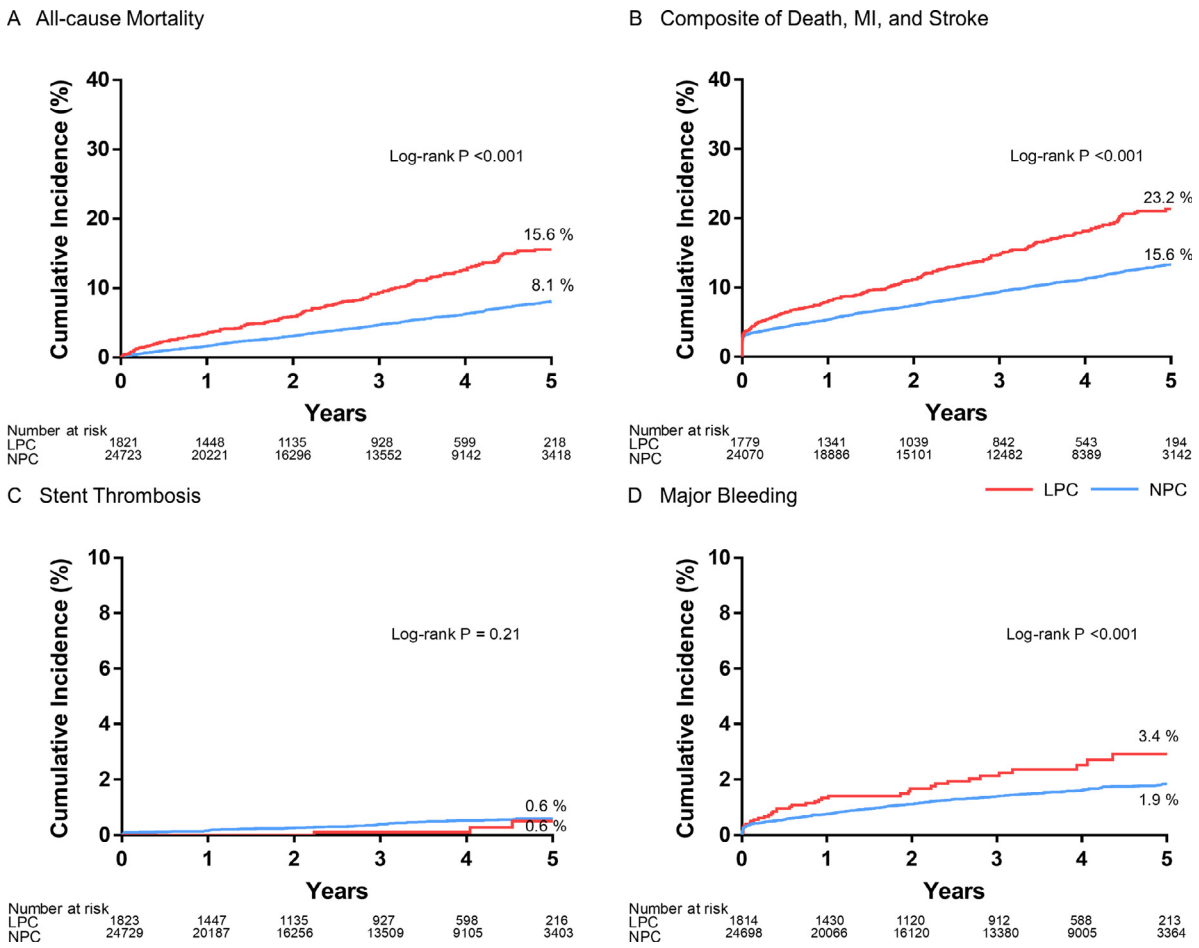

Figure 1. Cumulative event rates of all-cause mortality and secondary outcomes according to presence of baseline thrombocytopenia in patients who underwent PCI with DES. *(A)* all-cause mortality; *(B)* composite of death, MI, and stroke; *(C)* stent thrombosis; *(D)* major bleeding. DES = drug-eluting stents; LPC = low platelet count; MI = myocardial infarction; NPC = normal platelet count; PCI = percutaneous coronary intervention.

who received PCI with DES. In the contemporary clinical-practice IRIS-DES registry, the incidence of thrombocytopenia at baseline was approximately 7% and the presence of thrombocytopenia was significantly associated with an increased risk of all-cause mortality and major bleeding. This association was more prominent in patients with moderate-to-severe thrombocytopenia than in those with mild thrombocytopenia.

Thrombocytopenia is a commonly observed abnormality in routine blood tests.[3] Understanding the clinical impact of baseline thrombocytopenia on mortality, major cardiovascular events, and major bleeding events in patients undergoing PCI are crucial for balancing ischemic and bleeding risks and optimal decision-making for antithrombotic strategy and duration of DAPT. Nevertheless, clinical evidences regarding the prognostic significance of thrombocytopenia in patients undergoing PCI with contemporary DES are still lacking. Therefore, our study may provide valuable clinical insights on management and risk stratification of PCI patients treated with DES according to baseline thrombocytopenia.

The key finding of the present study is that the presence of thrombocytopenia at baseline was significantly associated with an increased risk of all-cause mortality after PCI with DES. Although there have been several studies suggesting the prognostic significance of acquired thrombocytopenia during ACS,[2–4] the direct relation between baseline thrombocytopenia and mortality in patients with stable coronary artery disease is not yet fully determined. In our study involving a diverse clinical spectrum of coronary artery disease in the real-world practice, thrombocytopenia was independently associated with an increased risk of all-cause mortality, which was incremental with the severity of thrombocytopenia. The exact mechanism underlying the relation between thrombocytopenia and all-cause mortality is still unclear; however, a previous study suggested that the presence of thrombocytopenia might be a marker of higher-risk profiles of baseline co-morbidity and also be associated with higher risks of ischemic and bleeding complications associated with PCI procedures and antithrombotic drugs.[21]

We also identified moderate-to-severe thrombocytopenia as a strong predictor for major bleeding after PCI with DES. It is well-known that major bleeding complications have led to subsequent major adverse cardiovascular events such as death, MI, stroke, or stent thrombosis in ACS or stable angina in patients who underwent PCI.[22–24] Recommended strategies for maximizing antithrombotic effects and minimizing bleeding risk in patients with thrombocytopenia undergoing PCI might involve individualized DAPT regimen, transradial approach, use of DES with safer profiles necessitating shorter DAPT, prescription of proton pump inhibitor, and avoidance of glycoprotein IIb-IIIa inhibitors.[17,25] As for the duration of DAPT, a recent study showed that patients who underwent complex PCI had a higher risk of ischemic events, but benefitted from long-term DAPT only if high-bleeding risk features were not present.[25] Such data suggests when concordant, bleeding risk, more than ischemic risk, should inform decision-making on the duration of DAPT. In this context, a tailored approach to DAPT should be considered in PCI patients with thrombocytopenia.

This study had several limitations. First, this study is observational, nonrandomized study. Although a wide range of baseline characteristics and covariates were adjusted in the multivariable adjusted models, unmeasured confounders (e.g., frailty, hematologic disease, liver disease, infection, patient's compliance to antiplatelet therapy, hidden malignancy, history of gastrointestinal bleeding, concomitant use of proton pump inhibitor) might influence observed findings. Second, causality between thrombocytopenia and clinical outcomes cannot be exactly established because unmeasured confounding might be present. Third, in our study, the exact causes of thrombocytopenia at baseline were not fully evaluated. Several factors might influence thrombocytopenia such as the use of noncardiac medications, antiplatelet antibodies, infection, or disseminated intravascular coagulation, which were not collected in our registry; thus, the impact of these preexisting conditions on subsequent treatment patterns and downstream outcomes could not be assessed. Lastly, because the duration of DAPT was decided at the discretion of the attending physicians, the relative effect of DAPT duration in patients with thrombocytopenia should be confirmed by further insights from other clinical trials.

In conclusion, in this large-sized, real-world cohort of patients who underwent PCI with DES, thrombocytopenia at baseline was significantly associated with increased risks of all-cause mortality and major bleeding.

Disclosures

The authors have no conflicts of interest to declare. The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Author's Contribution

Sangwoo Park: Writing - Original Draft, Methodology, Formal analysis. Jung-Min Ahn: Resources, Investigation. Tae Oh Kim: Data Curation, Visualization. Hanbit Park: Data Curation, Investigation. Sang-Cheol Cho: Data Curation, Investigation. Do-Yoon Kang: Project administration. Pil Hyung Lee: Validation. Duk-Woo Park: Conceptualization, Writing - Review & Editing. Seung-Jung Park: Supervision.

Finally all authors have read and approved the manuscript.

Supplementary materials

Supplementary material associated with this article can be found in the online version at https://doi.org/10.1016/j.amjcard.2020.07.059.

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