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One-year clinical outcomes between biodegradable-polymer-coated biolimus-eluting stent and durable-polymer-coated drug-eluting stents in STEMI patients with multivessel coronary artery disease undergoing culprit-only or multivessel PCI

Yong Hoon Kim[a,*,1], Ae-Young Her[a,1], Myung Ho Jeong[b], Byeong-Keuk Kim[c], Seung-Yul Lee[d], Sung-Jin Hong[c], Chul-Min Ahn[c], Jung-Sun Kim[c], Young-Guk Ko[c], Donghoon Choi[c], Myeong-Ki Hong[c], Yangsoo Jang[c]

[a] Division of Cardiology, Department of Internal Medicine, Kangwon National University School of Medicine, Chuncheon, South Korea
[b] Chonnam National University Hospital, Gwangju, South Korea
[c] Division of Cardiology, Severance Cardiovascular Hospital, Yonsei University College of Medicine, South Korea
[d] Sanbon Hospital, Wonkwang University College of Medicine, Gunpo, South Korea

HIGHLIGHTS

- Data comparing clinical outcomes among new-DESs in STEMI patients MVD who underwent primary PCI with culprit-only or multivessel PCI are limited.
- BP-BES and DP-DES showed comparable safety and efficacy in STEMI patients with MVD who underwent two different reperfusion strategies.
- In the total study population, the culprit-only PCI showed higher incidence of the total repeat revascularization than the multivessel PCI.

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ABSTRACT

Background and aims: There are limited data comparing clinical outcomes among new-generation drug-eluting stents (DES) in ST-segment elevation myocardial infarction (STEMI) patients with multivessel coronary artery disease (MVD) who underwent primary percutaneous coronary intervention (PCI) with culprit-only or multivessel PCI. We investigated 1-year clinical outcomes between biodegradable-polymer (BP)-coated biolimus-eluting stent (BES) and durable-polymer (DP)-coated DES in STEMI patients with MVD who underwent two different reperfusion strategies.

Methods: A total of 4255 patients were enrolled and divided into two groups, a culprit-only (n = 2571, BP- [n = 264] or DP-DES [n = 2307]) or a multivessel PCI group (n = 1684, BP- [n = 145] or DP-DES [n = 1539]). The primary endpoint was major adverse cardiac events (MACE) defined as all-cause death, recurrent myocardial infarction (re-MI), and total repeat revascularization. The secondary endpoint was the incidence of definite or probable stent thrombosis (ST).

Results: BP-BES and DP-DES showed a similar 1-year adjusted hazard ratio (HR) for MACE (culprit-only, adjusted hazard ratio [HR], 1.114; $p = 0.740$; multivessel, HR, 0.564; $p = 0.167$) and ST (culprit-only, HR, 1.110, $p = 0.891$; multivessel, HR, 0.375; $p = 0.402$). The adjusted HR for all-cause death, re-MI, and repeat revascularization were similar between the two groups. In the total population, the culprit-only PCI group showed a higher incidence of total repeat revascularization than the multivessel PCI group.

Conclusions: BP-BES and DP-DES showed comparable safety and efficacy in STEMI patients with MVD who underwent primary PCI with two different reperfusion strategies during a 1-year follow-up period.

* Corresponding author. Division of Cardiology, Department of Internal Medicine, Kangwon National University School of Medicine, 156 Baengnyeong Road, 24289, Chuncheon City, Gangwon Province, South Korea.
E-mail address: yhkim02@kangwon.ac.kr (Y.H. Kim).
[1] These authors contributed equally to this work.

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1. Introduction

Recent trials demonstrated that zotarolimus-eluting stents (ZES) and everolimus-eluting stents (EES) have improved clinical outcomes when compared with first-generation drug-eluting stents (DES) after percutaneous coronary intervention (PCI) [1–3]. In addition, stent platforms and polymers have rapidly evolved and newer anti-proliferative drugs, more biocompatible or biodegradable polymers, and polymer-free DES have been developed to reduce the rate of late stent thrombosis (ST) [4]. The cumulative incidences of major clinical outcomes were comparable between the biodegradable-polymer-coated biolimus-eluting stent (BP-BES) and the EES (5.2% vs. 4.8%, $p = 0.69$) during a 1-year follow-up period in the COMPARE II study [5]. Approximately, 50% of ST-segment elevation myocardial infarction (STEMI) is combined with multivessel coronary artery disease (MVD) and this is related to poor clinical outcomes [6,7]. Studies concerning the effect of two different reperfusion strategies such as culprit-only PCI or multivessel PCI in patients with STEMI with MVD on the major clinical outcomes of the new generations of DES such as BP-BES or durable-polymer (DP)-coated-DES are limited. Therefore, the aim of this study was to compare clinical outcomes between BES and DP-DES in STEMI with MVD who underwent primary PCI with these two different reperfusion strategies.

2. Materials and methods

2.1. Study design and population

Briefly, this was a non-randomized, multicenter, observational, retrospective cohort study comparing the 1-year clinical outcomes between BP-BES (either BioMatrix Flex stent, Biosensors International, Morges, Switzerland or Nobori stent, Terumo Corporation, Tokyo, Japan) and DP-DES (e.g., ZES [Resolute Integrity stent; Medtronic, Inc., Minneapolis, MN] or EES [Xience Prime stent, Abbott Vascular, Santa Clara, CA; or Promus Element stent, Boston Scientific, Natick, MA]) in STEMI patients with MVD, who underwent primary PCI with culprit-only or multivessel PCI from the Korea Acute Myocardial Infarction Registry (KAMIR). KAMIR is a nationwide, prospective, observational on-line registry in South Korea which started in November 2005; details of this registry can be found at the KAMIR website (http://www.kamir.or.kr). The study protocol was approved by the ethics committee at each participating center and informed consents were obtained from all individual participants included in the study prior to enrollment. These processes were conducted according to the ethical guidelines of the 1975 Declaration of Helsinki. A total of 26,011 STEMI patients who underwent PCI were enrolled between January 2006 and June 2015 in the KAMIR. Patients with the following conditions were excluded: (1) single vessel disease, left main disease, and chronic total occlusion (CTO) lesions (n = 14,733, 56.6%), (2) cardiogenic shock (n = 925, 3.6%), (3) fibrinolysis (n = 420, 1.6%), (4) coronary artery bypass graft (CABG, n = 72, 0.3%), (5) failed PCI (n = 176, 0.7%), (6) PCI was not done (n = 91, 0.3), (7) bare-metal stents (BMS) were deployed (n = 475, 1.8%), (8) first-generation and other kinds of DES except for BES, ZES, and EES were deployed (n = 2402, 9.2%), (9) different kinds of stents were deployed in the same patient (n = 498, 1.9%), (10) incomplete laboratory results (n = 899, 3.5%), (11) follow-up loss or non-participant (n = 1301, 5.0%). Finally, a total of 4255 STEMI patients with MVD who underwent successful primary PCI with new-generation DES (culprit-only PCI, n = 2571, 60.4% *vs.* multivessel PCI, n = 1684, 39.6%) were enrolled (Fig. 1). In this study, all 4255 patients completed a 1-year clinical follow-up by face-to-face interviews, phone calls, or chart review.

2.2. PCI procedures and medical treatment

Diagnostic coronary angiography and PCI were done through either the femoral or the radial artery. Patient's activated clotting time (ACT) was maintained > 250 s during the procedure. All patients were given loading doses of 200–300 mg aspirin and 300–600 mg clopidogrel (when available or, alternatively, 180 mg ticagrelor or 60 mg prasugrel) before PCI. Revascularization was considered clinically indicated when the patient had typical angina and/or signs of ischemia and ≥50% diameter stenosis or ≥70% diameter stenosis in a coronary artery by visual estimation. During the in-hospital stay and after discharge, all patients' medical treatment included aspirin, clopidogrel, ticagrelor, prasugrel, cilostazol [Pletaal®, Otsuka Pharmaceutical Co., Tokyo, Japan], beta-blockers (BB), calcium channel blockers (CCB), angiotensin-converting enzyme inhibitors (ACEI) or angiotensin II type 1 receptor blockers (ARB) and lipid lowering agents (Table 1). The patients were maintained on 100–200 mg aspirin indefinitely and the combination of aspirin (100 mg/day) with clopidogrel (75 mg/day) or ticagrelor (90 mg/day) or prasugrel (5–10 mg/day) was recommended for at least 12 months to patients who had undergone PCI. Triple antiplatelet therapy (TAPT) (100 mg cilostazol twice a day added on to DAPT) was left to the discretion of the individual operators.

2.3. Study definitions and clinical endpoints

STEMI was defined as the patient who has experienced chest pain with ST-segment elevation in at least 2 contiguous leads of ≥2 mm (0.2 mV) in men or ≥1.5 mm (0.15 mV) in women in leads V2–V3 and/or of ≥1 mm (0.1 mV) in other contiguous chest leads or the limb leads, or new onset left bundle branch block on the admission electrocardiogram [8]. A successful PCI was defined as the achievement of an angiographic residual stenosis, which was less than 30%, and the final thrombolysis in myocardial infarction (TIMI) blood flow grade was three. MVD was defined as at least two major vessels (≥2 mm diameter) with > 70% stenosis of the diameter [9]. Complete revascularization (CR) was defined as the infarct-related artery (IRA) opened, followed by dilatation of all other significantly narrowed arteries during the primary procedure or index hospitalization. Incomplete revascularization (IR) was defined as IRA successfully opened, followed by dilatation of only the significantly narrowed artery in three-vessel disease during the primary procedure or index hospitalization [10]. The primary endpoint was the occurrence of major adverse cardiac events (MACE) defined as all-cause death, recurrent myocardial infarction (re-MI), total repeat revascularization including target lesion revascularization (TLR), target vessel revascularization (TVR), and non-TVR during the 1-year follow-up period. The secondary endpoint was the incidences of definite or probable stent thrombosis (ST). All-cause death was classified as cardiac (CD) or non-cardiac death. Re-MI was defined as the presence of clinical symptoms, electrocardiographic changes, or abnormal imaging findings of MI, combined with an increase in the creatine kinase myocardial band fraction above the upper normal limits or an increase in troponin-T/troponin-I to greater than the 99th percentile of the upper normal limit [8]. The definition of TLR, TVR, and non-TVR was previously published [11]. ST defined as acute (0–24 h), subacute (24 h - 30 days) and late (30 days - 1 year) according to the onset time of stent thrombosis [12].

2.4. Statistical analysis

All statistical analyses were performed using SPSS software, version 20 (SPSS Inc., Chicago, IL, USA). The data are expressed as means ± standard deviations or expressed as counts and percentages. For discrete variables, the differences between the two groups were analyzed with the χ^2 test or Fisher's exact test, as appropriate. Multivariable Cox proportional hazards regression, which includes baseline confounding factors, was used. We tested all available variables of potential relevance: age, gender (men), left ventricular ejection fraction (LVEF), body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), cardiovascular risk factors (e.g., hypertension, diabetes mellitus [DM], dyslipidemia, previous MI, previous PCI, previous

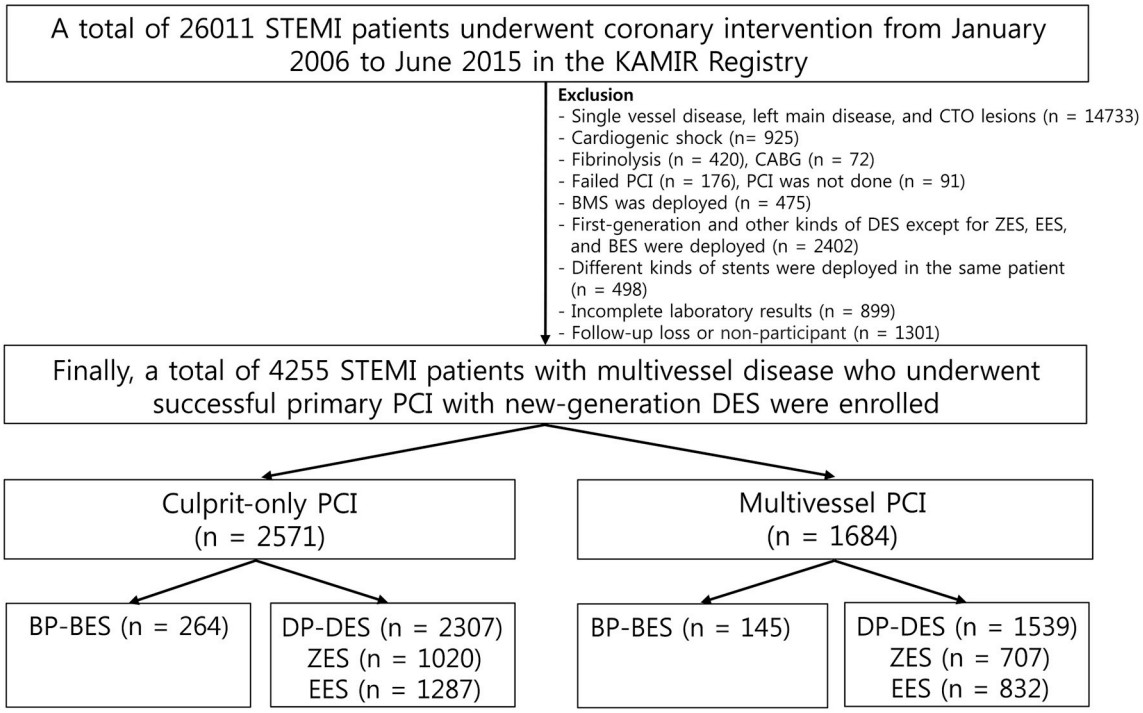

Fig. 1. Flow chart.

CABG, previous CVA, previous heart failure [HF], current smokers), blood chemistry results (blood glucose, hemoglobin $A1_C$, N-terminal pro-brain natriuretic peptide [NT-ProBNP], high-sensitivity CRP, serum creatinine, serum creatine kinase myocardial band [CK-MB], serum troponin-I, total cholesterol, triglyceride, low-density lipoprotein [LDL]-cholesterol, high-density lipoprotein [HDL]-cholesterol), discharge medications, IRA, treated vessels, pre-PCI TIMI grades, American College of Cardiology/American Heart Association (ACC/AHA) lesion types, the use of intravascular ultrasound (IVUS) or optical coherence tomography (OCT), door-to-balloon time, and the number of stents, mean stent diameter, and mean stent length. In addition, in patients with MVD, the timing of PCI for non-IRA (during index PCI or staged PCI before discharge) and completeness of MVD (CR or IR) were evaluated. Various clinical outcomes were estimated with Kaplan-Meier curve analysis, and differences between groups were compared with the log-rank test. A two-tailed *p*-value of < 0.05 was considered statistically significant.

3. Results

3.1. Baseline clinical, laboratory, angiographic and procedural characteristics

Baseline, laboratory and clinical characteristics of this study population are summarized in Table 1. In the culprit-only PCI group, the mean age between the two groups (BP-BES *vs.* DP-DES) was similar (64.1 ± 12.6 years *vs.* 64.1 ± 12.2 years, $p = 0.957$). However, the prescription rates of prasugrel, ARB, lipid lowering agents at discharge, and ACC/AHA type B2 lesion were higher in the BP-BES group and the levels of BMI, serum creatinine, prescription rates of clopidogrel, cilostazol, ACEI at discharge, and the mean length of deployed stent were higher in the DP-DES group. In the multivessel PCI group, the mean age between BP-BES and DP-DES was also similar (62.8 ± 12.5 years *vs.* 63.7 ± 12.3 years, $p = 0.399$). The level of blood glucose was higher in the BP-BES group and the level of hemoglobin A1c, HDL-cholesterol, prescription rates of cilostazol, ARB, CCB at discharge, and the mean length of deployed stent were higher in the DP-DES group.

3.2. Clinical outcomes

The cumulative incidences of major clinical outcomes at 1 year are listed in Tables 2 and 3, Fig. 2 and Supplementary Data 1. Supplementary Data 2 and 3 show independent predictors for MACE and stent thrombosis. Supplementary Data 4 shows the subgroup analysis for MACE in the culprit-only PCI and multivessel PCI groups.

3.2.1. BP-BES vs. DP-DES

In the culprit-only PCI group, the incidence of MACE was not significantly different between these two groups before adjustment (BP-BES *vs.* DP-DES = 8.8% *vs.* 8.1%, Log-rank $p = 0.723$; hazard ratio (HR), 0.923; 95% confidence interval (CI), 0.593–1.437; $p = 0.723$) and after adjustment (HR, 1.114; 95% CI, 0.588–2.113; $p = 0.740$). The incidence of definite or probable ST was similar between the two groups before (HR, 1.090; 95% CI, 0.254–4.681; $p = 0.907$) and after adjustment (adjusted HR, 1.110; 95% CI, 0.251–4.911; $p = 0.891$). The incidences of all-cause death (adjusted HR, 1.563; 95% CI, 0.467–5.232; $p = 0.469$), cardiac death (adjusted HR, 1.172; 95% CI, 0.342–4.021; $p = 0.800$), re-MI (adjusted HR, 0.737; 95% CI, 0.153–3.552; $p = 0.703$), and total repeat revascularization (adjusted HR, 0.919; 95% CI, 0.409–2.062; $p = 0.837$) were not significantly different between the two groups. In the multivessel PCI group, the incidence of MACE was also similar these two groups before adjustment (BP-BES *vs.* DP-DES = 7.8% *vs.* 6.4%, Log-rank $p = 0.504$; hazard ratio (HR), 0.809; 95% CI, 0.434–1.510; $p = 0.505$) and after adjustment (HR, 0.564; 95% CI, 0.250–1.270; $p = 0.167$). The incidence of definite or probable ST was similar between the two groups before (HR, 0.422; 95% CI, 0.091–1.955; $p = 0.270$) and after adjustment (adjusted HR, 0.375; 95% CI, 0.038–3.705; $p = 0.402$). The incidences of all-cause death (adjusted HR, 0.635; 95% CI, 0.186–2.167; $p = 0.468$), cardiac death (adjusted HR, 0.658; 95% CI, 0.193–2.246; $p = 0.504$), re-MI (adjusted HR, 0.584; 95% CI, 0.162–2.106; $p = 0.584$), and total repeat revascularization (adjusted HR, 0.519; 95% CI, 0.107–2.527; $p = 0.417$) were not significantly different between the two groups. The incidence of MACE was similar between the two groups regardless of the culprit-only PCI group or multivessel PCI group in this study.

Table 1
Baseline clinical, laboratory, and procedural characteristics.

Variables	Culprit-only PCI (n = 2571)			Multivessel PCI (n = 1684)		
	BP-BES (n = 264)	DP-DES (n = 2307)	*p* value	BP-BES (n = 145)	DP-DES (n = 1539)	*p* value
Men, n (%)	204 (77.3)	1730 (75.0)	0.416	113 (77.9)	1158 (75.2)	0.472
Age (years)	64.1 ± 12.6	64.1 ± 12.2	0.957	62.8 ± 12.5	63.7 ± 12.3	0.399
LVEF (%)	50.8 ± 11.0	50.5 ± 11.5	0.689	51.7 ± 10.1	50.3 ± 11.2	0.154
BMI (kg/m^2)	23.6 ± 3.2	24.0 ± 3.2	0.039	24.1 ± 3.1	24.2 ± 3.2	0.683
Systolic blood pressure (mmHg)	131.4 ± 25.4	130.1 ± 26.7	0.470	130.5 ± 28.1	129.9 ± 27.3	0.795
Diastolic blood pressure (mmHg)	78.8 ± 14.7	79.2 ± 16.4	0.660	79.2 ± 16.6	79.4 ± 16.3	0.906
Hypertension, n (%)	123 (46.9)	1198 (51.9)	0.100	64 (44.1)	776 (50.4)	0.165
Diabetes mellitus, n (%)	76 (28.8)	636 (27.6)	0.675	34 (23.4)	472 (30.7)	0.072
Dyslipidemia, n (%)	26 (9.8)	243 (10.5)	0.832	12 (8.3)	192 (12.5)	0.182
Previous MI, n (%)	6 (2.3)	75 (3.3)	0.462	5 (3.4)	42 (2.7)	0.615
Previous PCI, n (%)	9 (3.4)	125 (5.5)	0.189	8 (5.5)	53 (3.4)	0.201
Previous CABG, n (%)	1 (0.4)	6 (0.3)	0.532	0 (0.0)	3 (0.2)	0.595
Previous CVA, n (%)	22 (8.3)	138 (6.0)	0.134	7 (4.8)	96 (6.2)	0.590
Previous heart failure, n (%)	1 (0.4)	20 (0.9)	0.716	1 (0.7)	16 (1.0)	0.687
Current smokers, n (%)	127 (48.1)	1024 (44.4)	0.250	72 (49.7)	697 (45.3)	0.313
CK-MB (mg/dL)	162.2 ± 152.9	171.7 ± 196.1	0.360	147.6 ± 148.0	169.8 ± 203.4	0.106
Troponin-I (ng/mL)	61.3 ± 75.3	61.1 ± 98.7	0.982	75.4 ± 201.8	80.1 ± 468.4	0.863
Serum glucose (mg/dL)	162.2 ± 152.9	177.7 ± 196.1	0.160	185.8 ± 90.0	178.3 ± 78.3	0.003
Hemoglobin A1c (%)	6.8 ± 4.2	6.6 ± 1.5	0.540	6.4 ± 1.6	6.8 ± 2.3	0.012
NT-ProBNP (pg/mL)	1303.7 ± 4062.2	1653.7 ± 4580.1	0.322	1676.4 ± 5407.1	1616.5 ± 3759.5	0.895
High-sensitivity CRP (mg/dL)	7.2 ± 26.2	8.9 ± 42.3	0.423	7.3 ± 36.2	11.7 ± 55.8	0.249
Serum creatinine (mg/L)	1.0 ± 0.3	1.1 ± 0.7	0.026	1.0 ± 0.3	1.1 ± 2.0	0.415
Total cholesterol (mg/dL)	182.4 ± 46.0	184.6 ± 43.6	0.469	185.1 ± 43.8	187.6 ± 45.2	0.511
Triglyceride (mg/L)	133.9 ± 134.3	134.6 ± 103.6	0.936	133.2 ± 115.0	140.8 ± 113.0	0.458
HDL cholesterol (mg/L)	41.8 ± 11.6	42.9 ± 14.8	0.156	40.7 ± 11.0	43.6 ± 15.5	0.030
LDL cholesterol (mg/L)	114.3 ± 37.1	116.9 ± 37.4	0.294	120.0 ± 36.9	118.2 ± 40.0	0.596
Discharge medications, n (%)						
Aspirin, n (%)	254 (96.2)	2189 (94.9)	0.348	139 (95.9)	1460 (94.9)	0.601
Clopidogrel, n (%)	215 (81.4)	2018 (87.5)	0.006	125 (86.2)	1325 (86.1)	0.970
Ticagrelor, n (%)	28 (10.6)	180 (7.8)	0.114	14 (9.7)	100 (6.5)	0.148
Prasugrel, n (%)	20 (7.6)	78 (3.4)	0.001	5 (3.4)	61 (4.0)	0.760
Cilostazole, n (%)	25 (9.5)	410 (17.8)	< 0.001	20 (13.8)	394 (25.6)	0.001
Beta-blocker, n (%)	233 (84.5)	1936 (83.9)	0.817	122 (84.1)	1250 (81.2)	0.388
ACEI, n (%)	140 (53.0)	1414 (61.3)	0.009	72 (49.7)	866 (56.3)	0.125
ARB, n (%)	77 (29.2)	460 (19.9)	< 0.001	46 (31.7)	353 (22.9)	0.017
CCB, n (%)	9 (3.4)	79 (3.4)	1.000	1 (0.7)	82 (5.3)	0.008
Lipid lowering agents	228 (86.4)	1876 (81.3)	0.044	124 (85.5)	1300 (84.5)	0.739
Infarct-related artery (IRA)						
Left anterior descending, n (%)	133 (50.4)	1062 (46.0)	0.180	66 (45.5)	741 (48.1)	0.602
Left circumflex, n (%)	26 (9.8)	199 (8.6)	0.505	17 (11.7)	189 (12.3)	0.845
Right coronary artery, n (%)	104 (39.4)	1042 (45.2)	0.078	61 (42.1)	608 (39.5)	0.547
Treated vessel						
Left anterior descending, n (%)	133 (50.4)	1062 (46.0)	0.180	109 (75.2)	1132 (73.6)	0.672
Left circumflex, n (%)	26 (9.8)	199 (8.6)	0.505	62 (42.8)	656 (42.6)	0.975
Right coronary artery, n (%)	104 (39.4)	1042 (45.2)	0.078	91 (62.8)	935 (60.8)	0.636
TIMI flow in culprit lesion before PCI						
0	176 (66.7)	1499 (65.0)	0.894	91 (62.8)	930 (60.4)	0.704
1	23 (8.7)	267 (11.6)	0.219	13 (9.0)	190 (12.4)	0.412
2	22 (8.3)	255 (11.0)	0.275	21 (14.4)	188 (12.2)	0.429
3	43 (16.3)	286 (12.4)	0.073	20 (13.8)	231 (15.0)	0.807
ACC/AHA lesion type						
Type B1, n (%)	27 (10.2)	305 (13.2)	0.207	18 (12.4)	179 (11.6)	0.779
Type B2, n (%)	89 (33.7)	576 (25.0)	0.002	52 (35.9)	462 (30.0)	0.144
Type C, n (%)	130 (49.2)	1232 (53.4)	0.200	58 (40.0)	720 (46.8)	0.138
IVUS	53 (20.1)	400 (17.3)	0.269	36 (24.8)	316 (20.5)	0.224
OCT	1 (0.4)	11 (0.5)	0.825	1 (0.7)	4 (0.3)	0.363
FFR	3 (1.1)	15 (0.7)	0.369	2 (1.4)	10 (0.6)	0.318
Door-to-balloon time, min	60.7 ± 28.8	58.7 ± 38.2	0.892	52.1 ± 52.5	68.8 ± 41.1	0.196
PCI for non-IRA						
During index PCI				96 (66.2)	1001 (65.0)	0.778
Staged PCI before discharge				49 (33.8)	538 (35.0)	0.778
Completeness of multivessel PCI						
Complete revascularization				102 (70.3)	1104 (71.7)	0.723
Incomplete revascularization				43 (29.7)	435 (28.3)	0.723
Stent diameter (mm)	3.17 ± 0.38	3.16 ± 0.43	0.861	3.14 ± 0.38	3.11 ± 0.41	0.490

(*continued on next page*)

Table 1 (*continued*)

Variables	Culprit-only PCI (n = 2571)			Multivessel PCI (n = 1684)		
	BP-BES (n = 264)	DP-DES (n = 2307)	*p* value	BP-BES (n = 145)	DP-DES (n = 1539)	*p* value
Stent length (mm)	24.5 ± 7.8	28.1 ± 11.3	< 0.001	25.2 ± 9.0	27.3 ± 10.2	0.020
Number of stent	1.32 ± 0.63	1.35 ± 0.62	0.527	2.03 ± 0.88	2.14 ± 0.94	0.208

Values are means ± SD or numbers and percentages. The *p* value for continuous data from analysis of variance. The *p* value for categorical data from chi-square or Fisher's exact test.

LVEF, left ventricular ejection fraction; BMI, body mass index; MI, myocardial infarction; PCI, percutaneous coronary intervention; CABG, coronary artery bypass graft; CK-MB, creatine kinase myocardial band; NT-ProBNP, N-terminal pro-brain natriuretic peptide; CRP, C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; ACEI, angiotensin converting enzyme inhibitors; ARB, angiotensin II type 1 receptor blockers; CCB, calcium channel blockers; TIMI, Thrombolysis In Myocardial Infarction; ACC/AHA, American College of Cardiology/American Heart Association; IVUS, intravascular ultrasound; OCT, optical coherence tomography; FFR, fractional flow reserve; PCI, percutaneous coronary intervention; BP, biodegradable-polymer; DP, durable-polymer; BES, biolimus-eluting stents; DES, drug-eluting stents.

3.2.2. Culprit-only vs. multivessel PCI

Table 2 also shows major clinical outcomes between the culprit-only and the multivessel PCI group according to the types of polymer (BP-BES or DP-DES). In the BP-BES group, the cumulative incidences of MACE (adjusted HR, 1.297; 95% CI, 0.550–3.057; $p = 0.553$) and definite or probable ST (adjusted HR, 1.447; 95% CI, 0.156–13.46; $p = 0.745$) between the culprit-only and the multivessel PCI group were similar. The adjusted HR for all-cause death, CD, Re-MI, and total revacularization rates between the two groups were also similar. However, in the DP-DES group, the incidence of total revacularization was significantly higher in the culprit-only PCI group compared with the multivessel-PCI group before (unadjusted HR, 0.460; 95% CI, 0.294–0.721; $p = 0.001$) and after adjustment (adjusted HR, 0.439; 95% CI, 0.240–0.803; $p = 0.007$). However, in the total population, regardless of the types of polymer, the cumulative incidence of the total revascularization rate was significantly higher in the culprit only group compared with the multivessel PCI group (3.8% *vs.* 1.7%, Log-rank $p < 0.001$; adjusted HR, 0.488; 95% CI, 0.287–0.832; $p = 0.008$)

Table 2
Comparison of clinical outcomes at 1-year.

Outcomes	BP-BES	DP-DES	Log-Rank	Unadjusted		Adjusted[a]	
				HR (95% CI)	*p* value	HR (95% CI)	*p* value
Culprit-only PCI							
MACE	22 (8.8)	182 (8.1)	0.723	0.923 (0.593–1.437)	0.723	1.114 (0.588–2.113)	0.740
All-cause death	9 (3.5)	85 (3.7)	0.840	1.073 (0.540–2.133)	0.840	1.563 (0.467–5.232)	0.469
Cardiac death	6 (2.3)	70 (3.1)	0.503	1.328 (0.577–3.057)	0.505	1.172 (0.342–4.021)	0.800
Re-MI	4 (1.6)	23 (1.0)	0.416	0.646 (0.223–1.868)	0.420	0.737 (0.153–3.552)	0.703
Total repeat revascularization	10 (4.3)	81 (3.7)	0.748	0.898 (0.466–1.732)	0.748	0.919 (0.409–2.062)	0.837
Definite or probable ST	2 (0.7)	19 (0.8)	0.907	1.090 (0.254–4.681)	0.907	1.110 (0.251–4.911)	0.891
Multivessel PCI							
MACE	11 (7.8)	96 (6.4)	0.504	0.809 (0.434–1.510)	0.505	0.564 (0.250–1.270)	0.167
All-cause death	5 (3.5)	51 (3.4)	0.922	0.955 (0.381–2.394)	0.922	0.635 (0.186–2.167)	0.468
Cardiac death	4 (2.8)	44 (2.9)	0.953	1.031 (0.371–2.870)	0.953	0.658 (0.193–2.246)	0.504
Re-MI	5 (3.7)	25 (1.7)	0.109	0.465 (0.178–1.215)	0.118	0.584 (0.162–2.106)	0.584
Total repeat revascularization	2 (1.6)	25 (1.8)	0.848	1.151 (0.273–4.858)	0.849	0.519 (0.107–2.527)	0.417
Definite or probable ST	2 (1.4)	9 (0.6)	0.256	0.422 (0.091–1.955)	0.270	0.375 (0.038–3.705)	0.402

Outcomes	Culprit-only PCI	Multivessel PCI	Log-Rank	Unadjusted		Adjusted[a]	
				HR (95% CI)	*p* value	HR (95% CI)	*p* value
BP-BES							
MACE	22 (8.8)	11 (7.8)	0.799	0.910 (0.441–1.877)	0.799	1.297 (0.550–3.057)	0.553
All-cause death	9 (3.5)	5 (3.4)	0.978	0.985 (0.340–3.029)	0.978	1.816 (0.505–6.534)	0.361
Cardiac death	6 (2.3)	4 (2.8)	0.760	1.218 (0.344–4.316)	0.760	2.731 (0.655–11.39)	0.168
Re-MI	4 (1.6)	5 (3.7)	0.199	2.310 (0.620–8.603)	0.212	2.905 (0.589–14.32)	0.190
Total repeat revascularization	10 (4.3)	2 (1.6)	0.166	0.358 (0.078–1.635)	0.185	0.354 (0.064–1.957)	0.234
Definite or probable ST	2 (0.7)	2 (1.4)	0.540	1.830 (0.258–12.99)	0.546	1.447 (0.156–13.46)	0.745
DP-DES							
MACE	182 (8.1)	96 (6.4)	0.063	0.792 (0.618–1.014)	0.064	0.720 (0.512–1.013)	0.059
All-cause death	85 (3.7)	51 (3.4)	0.561	0.902 (0.638–1.277)	0.561	0.857 (0.528–1.390)	0.532
Cardiac death	70 (3.1)	44 (2.9)	0.766	0.944 (0.648–1.377)	0.766	0.873 (0.522–1.461)	0.605
Re-MI	23 (1.0)	25 (1.7)	0.082	1.644 (0.933–2.897)	0.085	1.157 (0.537–2.492)	0.710
Total repeat revascularization	81 (3.7)	25 (1.8)	0.001	0.460 (0.294–0.721)	0.001	0.439 (0.240–0.803)	0.007
Definite or probable ST	19 (0.8)	9 (0.6)	0.395	0.710 (0.321–1.569)	0.397	0.182 (0.030–1.087)	0.062

BP, biodegradable-polymer; DP, durable-polymer; BES, biolimus-eluting stents; DES, drug-eluting stents; PCI, percutaneous coronary intervention; MACE, major adverse cardiac events; Re-MI, recurrent myocardial infarction; ST, stent thrombosis; HR, hazard ratio; CI, confidence interval; LVEF, left ventricular ejection fraction; MI, myocardial infarction; CABG, coronary artery bypass graft; HF, heart failure; CVA, cerebrovascular accidents; Hb, hemoglobin; HDL, high-density lipoprotein; ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin II type 1 receptor blockers; ACC/AHA, American College of Cardiology/American Heart Association.

[a] Adjusted by age, gender (men), LVEF, hypertension, diabetes, dyslipidemia, previous history of MI, PCI, CABG, HF, CVA, HbA1c, HDL-cholesterol, clopidogrel, cilostazole, ACEI, ARB, lipid lowering agent, ACC/AHA type B1, B2 lesion, stent diameter, stent length.

Table 3
Comparison of clinical outcomes between culprit-only and multivessel PCI in entire patients at 1-year.

Outcomes	Culprit-only PCI	Multivessel PCI	Log-Rank	Unadjusted		Adjusted	
				HR (95% CI)	*p* value	HR (95% CI)	*p* value
MACE	204 (8.1)	107 (6.5)	0.063	0.801 (0.634–1012)	0.063	0.755 (0.556–1.024)	0.755
All-cause death	94 (3.7)	56 (3.4)	0.585	0.912 (0.655–1.270)	0.586	0.902 (0.582–1.400)	0.647
Cardiac death	76 (3.0)	48 (2.9)	0.853	0.966 (0.673–1.387)	0.853	0.964 (0.600–1.548)	0.879
Re-MI	27 (1.1)	30 (1.8)	0.040	1.712 (1.018–2.880)	0.043	1.113 (0.567–2.185)	0.755
Total repeat revascularization	91 (3.8)	27 (1.7)	< 0.001	0.450 (0.293–0.691)	< 0.001	0.488 (0.287–0.832)	0.008
TLR	20 (0.8)	8 (0.5)	0.240	0.614 (0.271–1.395)	0.244	0.626 (0.220–1.780)	0.379
TVR	32 (1.3)	16 (1.0)	0.387	0.768 (0.421–1.399)	0.388	0.656 (0.284–1.512)	0.323
Non-TVR	56 (2.3)	11 (0.7)	< 0.001	0.298 (0.156–0.570)	< 0.001	0.303 (0.134–0.685)	0.004
Definite or probable ST	21 (0.8)	11 (0.7)	0.548	0.800 (0.386–1.659)	0.548	0.883 (0.365–2.135)	0.782

MACE, major adverse cardiac events; Re-MI, recurrent myocardial infarction; TLR, target lesion revascularization; TVR, target vessel revascularization; ST, stent thrombosis; PCI, percutaneous coronary intervention; HR, hazard ratio; CI, confidence interval.

(Table 3). Before adjustment, old age (age ≥65 years), decreased LVEF (< 50%), DM, low serum HDL-cholesterol (< 40 mg/dL) level, the use of cilostazole, ACEI, and lipid lowering agent as discharge medications, and size of deployed stent being less than 3 mm were the independent predictors for MACE in the culprit-only PCI. However, after adjustment, low serum HDL-cholesterol (< 40 mg/dL) level and the use of ACEI and ARB as discharge medications were statistically significant independent predictors of MACE in this group. In case of multivessel PCI, before adjustment, old age (age ≥65 years), men, decreased LVEF (< 50%), hypertension, DM, use of cilostazole, ACEI, and ARB as discharge medications and size of deployed stent being less than 3 mm were significant independent predictors for MACE. After adjustment, hypertension and use of ACEI and lipid lowering agents as discharge medications were significant independent predictors for MACE (Supplementary Data 2). In addition, in the culprit-only PCI group, the independent predictors for stent thrombosis were DM, low serum HDL-cholesterol (< 40 mg/dL) level, use of ACEI as discharge medication, and size of deployed stent being less than 3 mm were significant independent predictors for ST before adjustment. After adjustment, low serum HDL-cholesterol (< 40 mg/dL) level and use of ACEI as discharge medication were significant independent predictors for ST. In the multivessel PCI group, use of cilostazole as discharge medication and size of deployed stent being less than 3 mm were significant independent predictors for ST. However, after adjustment, significant predictors for ST were not apparent in this study (Supplementary Data 3).

4. Discussion

The main findings of this study are as follows: (1) BP-BES and DP-DES showed similar 1-year adjusted HR for MACE and ST, (2) the adjusted HR for all-cause death, re-MI, and total repeat revascularization were also similar between the two groups, (3) in the total study population, the culprit-only PCI group showed a higher incidence of total

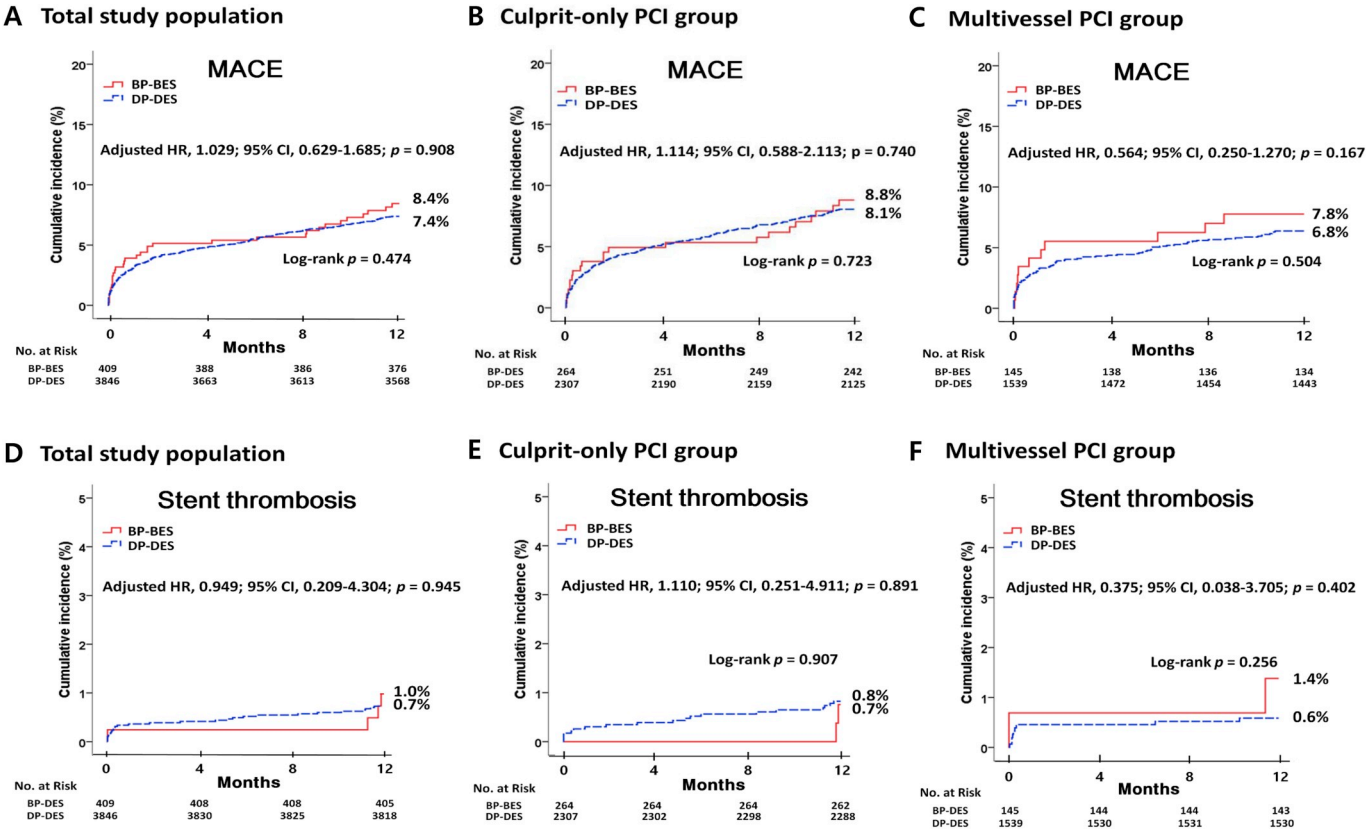

Fig. 2. Kaplan-Meier analysis for MACE (A, B, C) and stent thrombosis (D, E, F) in the total study population, culprit-only PCI, and multivessel PCI group.

repeat revascularization than the multivessel PCI group.

The polymer of BP-BES contains polylactic acid which is fully degraded into carbon dioxide and water within 6 months [13]. Therefore, 6 months after the index PCI, the stent surface remained as a BMS without any chronic inflammatory stimulus. However, previous randomized controlled trials demonstrated that BP-DES was comparable to DP-DES in terms of safety and efficacy during 9 months, 1-year [14,15] or 4-year follow-up periods [16]. In case of AMI, recent studies have reported BES has similar safety and efficacy compared with EES during long-term follow-up periods [5,17]. Furthermore, the comparative clinical outcomes between ZES and EES were not significantly different. In the DUTCH PEERS trial [18], the cobalt-chromium-based ZES (Resolute Integrity®, Medtronic, Santa Rosa, CA, USA) and platinum-chromium-based EES (Promus Element®, Boston Scientific, Natick, MA, USA) showed similar incidences of target vessel failure (HR, 1.17; 95% CI, 0.80–1.71; $p = 0.42$) and definite, probable, or possible, ST (HR, 0.93; 95% CI, 0.44–1.96; $p = 0.85$) in 1811 AMI patients during a 1 year follow-up period. In this study, the cumulative incidence of MACE for BP-BES *vs.* DP-DES (culprit-only PCI group; 8.8% *vs.* 8.1%, Log-rank $p = 0.723$, adjusted HR, 1.114; 95% CI, 0.588–2.113; $p = 0.740$ *vs.* multivessel PCI group; 7.8% *vs.* 6.4%, Log-rank $p = 0.504$, adjusted HR, 0.564; 95% CI, 0.250–1.270; $p = 0.167$) and ST for BP-BES *vs.* DP-DES (culprit-only PCI group; 0.7% *vs.* 0.8%, Log-rank $p = 0.907$, adjusted HR, 1.110; 95% CI, 0.251–4.911; $p = 0.891$ *vs.* multivessel PCI group; 1.4% *vs.* 0.6%, Log-rank $p = 0.256$, adjusted HR, 0.375; 95% CI, 0.038–3.705; $p = 0.402$) were similar (Fig. 2) and comparable with previous studies. Therefore, in the two different reperfusion strategies, culprit-only PCI or multivessel PCI, BP-BES showed similar major clinical outcomes compared with DP-DES in STEMI patients who undergo primary PCI. Furthermore, the comparisons of the cumulative incidences of MACE and ST between the culprit-only and the multivessel PCI group according to the types of polymer (BP-BES or DP-DES) were similar in this study (Table 2). However, in the DP-DES group, the adjusted HR for total repeat revascularization rate was significantly higher in the culprit-only PCI group compared with the multivessel-PCI group (adjusted HR, 0.439; 95% CI, 0.240–0.803: $p = 0.007$). Even though the adjusted HR for the total repeat revascularization rate was not significantly different between the culprit-only PCI group and the multivessel PCI group in the BP-BES, in the total population, regardless of the types of polymer, the cumulative incidences of total revacularization rate were significantly higher in the culprit only group compared with the multivessel PCI group (Table 3). Therefore, the reason for similar cumulative incidences of any revascularziation (4.3% *vs.* 1.6%, Log-rank $p = 0.166$) in the BP-BES group between the culprit only PCI group and the multivessel PCI group was attributed to the relatively small total number of culprit-only PCI patients in the BP-BES group (409/4255, 9.6%) compared to the entire group (2571/4255, 60.4%). In this study, even though there were some limitations, the multivessel PCI group showed better outcomes than the culprit-only PCI group regarding the total repeat revacularization rate. The treatment strategy whether culprit-only or multivessel PCI was determined by the operator's discretion without any restriction. Therefore, in the culprit-only PCI group, lesions estimated as significant during the initial procedure but not treated, might have been planned PCI later. These PCIs were included as revascularization events in this study and could explain the observed difference. The new-generation DES in our study (BES *vs.* ZES and EES) have different stent platforms, polymer and antiproliferative drugs, but the primary and secondary endpoints did not different between the two groups. Until now, the relationship between stent strut thickness and platform design and long-term safety and efficacy of DES was not well defined [19,20].

According to the European guideline recommendation [21], the optimal treatment strategies for STMI with MVD is immediate revascularization of both culprit and nonculprit lesions. The U.S. guideline [22] recommended that appropriate-use score criteria consider immediate revascularization of both culprit and nonculprit arteries during the same procedure to be highly appropriate. Pasceri V et al. [23] demonstrated CR with PCI can reduce the risk of death and MI (relative risk: 0.76; 95% CI: 0.56–0.99; $p = 0.04$) compared with culprit-only PCI. Wang et al. [24] also showed that the CR treatment strategy was preferred over the culprit-only PCI in STEMI with MVD undergoing primary PCI. In this study, the cumulative incidences of MACE, all-cause death, CD and definite or probable ST were similar between the culprit-only PCI and the multivessel PCI, except for the total repeat revacularization rate (Tables 2 and 3). We think that the main cause of these findings were associated with selection bias because many patients were excluded by the exclusion criteria such as, first-generation and other kinds of DES except for ZES, EES, and BES were deployed (n = 2402, 9.2%), incomplete laboratory results (n = 899, 3.5%), follow-up loss or non-participant (n = 1301, 5.0%) etc. In addition, the patients who had left main coronary artery disease, cardiogenic shock, and CTO lesions were also excluded in this study. Therefore, one must be cautious in interpretation of these results.

The study populations of previous studies [16,23–25] were different compared with this study; in previous studies the numbers of enrolled STEMI patients were only 20–30%, and the studies did not focus on the comparison of major clinical outcomes between the BP-BES and DP-DES, whereas in this study, all enrolled patients were composed of STEMI. We think that the results of this study could provide an important message to the interventional cardiologist during primary PCI, especially in case of STEMI patients with MVD undergoing culprit-only PCI or multivessel PCI in the era of new-generation DES, especially in the case of BP-BES or DP-DES.

In our study, there were several limitations. First, the present study was a non-randomized study and there may be some under-reporting and/or missed data. Second, there may be sample selection bias, because the total number of BP-BES received patents in this study were relatively lower than DP-DES, this factor may act as an important bias in this study. Third, because the use of ZES, EES, or BES was at the discretion of the physician; this may play a bias role in this study. Fourth, even though multivariate analysis was done; variables not included in this registry may have affected the study outcomes. Fifth, the 1-year follow-up period of this study was relatively short to determine the long-term major clinical outcomes, especially because the patients who could show very late ST were not followed up in this study. Sixth, this study enrolled only Korean patients; the present results may not be generalizable to all other ethnicities in different parts of the world. Finally, even though the authors classified ZES and EES as a DP-DES, these DES have different kinds of eluting drugs, different release kinetics, and different stent platforms. Therefore, DP-DES is not a homogenous group and these conclusions may not be valid for all stents included in it.

In conclusion, BP-BES and DP-DES showed comparable safety and efficacy in STEMI patients with MVD who underwent primary PCI with two different treatment strategies during a 1-year follow-up period. Hence, DP-DES and BP-BES are equally acceptable in STEMI patients with MVD who are undergoing primary PCI with culprit-only or multivessel PCI.

Conflicts of interest

The authors declared they do not have anything to disclose regarding conflict of interest with respect to this manuscript.

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Korea acute myocardial infarction registry (KAMIR) investigators

Myung Ho Jeong, MD, Youngkeun Ahn, MD, Sung Chul Chae, MD,

Jong Hyun Kim, MD, Seung-Ho Hur, MD, Young Jo Kim, MD, In Whan Seong, MD, Donghoon Choi, MD, Jei Keon Chae, MD, Taek Jong Hong, MD, Jae Young Rhew, MD, Doo-Il Kim, MD, In-Ho Chae, MD, Junghan Yoon, MD, Bon-Kwon Koo, MD, Byung-Ok Kim, MD, Myoung Yong Lee, MD, Kee-Sik Kim, MD, Jin-Yong Hwang, MD, Myeong Chan Cho, MD, Seok Kyu Oh, MD, Nae-Hee Lee, MD, Kyoung Tae Jeong, MD, Seung-Jea Tahk, MD, Jang-Ho Bae, MD, Seung-Woon Rha, MD, Keum-Soo Park, MD, Chong Jin Kim, MD, Kyoo-Rok Han, MD, Tae Hoon Ahn, MD, Moo-Hyun Kim, MD, Ki Bae Seung, MD, Wook Sung Chung, MD, Ju-Young Yang, MD, Chong Yun Rhim, MD, Hyeon-Cheol Gwon, MD, Seong-Wook Park, MD, Young-Youp Koh, MD, Seung Jae Joo, MD, Soo-Joong Kim, MD, Dong Kyu Jin, MD, Jin Man Cho, MD, Sang-Wook Kim, MD, Jeong Kyung Kim, MD, Tae Ik Kim, MD, Deug Young Nah, MD, Si Hoon Park, MD, Sang Hyun Lee, MD, Seung Uk Lee, MD, Hang-Jae Chung, MD, Jang-Hyun Cho, MD, Seung Won Jin, MD, Myeong-Ki Hong, MD, Yangsoo Jang, MD, Jeong Gwan Cho, MD, Hyo-Soo Kim, MD, and Seung Jung Park, MD.

Appendix A. Supplementary data

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