Impact of Single Long Stents Versus Overlapping Stents on Clinical Outcomes in Primary PCI

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Background: Patients with long coronary lesions undergoing primary percutaneous coronary intervention (pPCI) have higher rates of adverse clinical events. Both stent length and stent overlap are associated with worse outcomes; however, data comparing very long stent (VLS) to overlapping stents (OSs) are limited, particularly during pPCI. This study aimed to compare the impact of a single VLS versus ≥2 OSs on clinical outcomes in a multicenter registry of patients undergoing pPCI.

Methods: This study included patients with ST-segment elevation myocardial infarction (STEMI) who underwent pPCI using a single VLS (≥38 mm) or ≥2 OS (total stent length, ≥38 mm) in the culprit lesion. After propensity score matching based on tortuosity, calcification, Killip class, culprit lesion length ≥40 mm, and culprit vessel, the final cohort for analysis was selected. The primary endpoint was a combination of mortality and target lesion failure (reinfarction, stent thrombosis, or new revascularization) at 2 years.

Results: Among 647 consecutive STEMI patients who underwent pPCI between March 2016 and September 2022, 353 received VLS and 294 received OSs. After propensity score matching, 264 patients remained (132 in each group). The occurrence of the primary outcome (VLS: 12.9 vs. OS: 15.9%; $P = 0.86$), all-cause mortality (VLS: 7.6 vs. OS: 9.8%; $P = 0.51$), and target lesion failure (VLS: 8.3 vs. OS: 6.8, $P = 0.64$) were similar between the 2 groups.

Conclusions: In this cohort of real-world patients with STEMI undergoing pPCI, we found no significant difference in outcomes between VLS and OSs. Both strategies are reasonable treatment options for STEMI patients.

Key Words: mortality, percutaneous coronary intervention, ST-elevation myocardial infarction, stent thrombosis, target-vessel revascularization

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BACKGROUND

During percutaneous coronary intervention (PCI), stent overlapping can occur in up to 30% of cases due to various reasons such as incomplete lesion coverage, edge dissections, residual thrombus, or excessive tortuosity.[1–4]

The decision to implant a single very long stent (VLS) or overlapping stents (OSs) typically depends on the operator's discretion. However, despite improvements in stent polymers, platforms, and PCI techniques, treating very long coronary lesions remains challenging. This is particularly true in acute coronary syndromes, where inflammation plays a central role in the pathogenesis of coronary events. This proinflammatory state increases the risk of stent thrombosis and adverse events compared with stable coronary artery disease.[5–8] Newer-generation stents have shown lower procedural and late target-vessel myocardial infarction compared to older platforms, which may be explained by their thinner struts among other characteristics.[9,10] OSs increase the strut contact area with blood and its inflammatory milieu, which may be associated with adverse outcomes.

A subanalysis of the examination trial explored the use of overlapped stents versus single stents, but not necessarily in long lesions, and included the use of bare metal stents with higher rates of adverse outcomes in these patients.[11] Thus, there is currently a lack of real-world data that directly compare the use of contemporary single long drug-eluting stents (DES) versus overlapping DES in the context of acute coronary syndromes. Therefore, the objective of the present study was to compare the clinical outcomes associated with the implantation of VLS or OSs for the treatment of patients with ST-elevation myocardial infarction (STEMI) who underwent primary PCI (pPCI) in a real-world scenario.

METHODS

Study Design and Patients

This multicentric registry enrolled consecutive patients with STEMI who underwent pPCI at 4 institutions in Brazil and Spain from March 2016 to September 2022. All participating institutions were high-volume centers. Eligible patients were adults aged 18 years or older with suspected STEMI, identified by typical chest pain at rest and accompanied by ST-segment elevation or abnormalities meeting STEMI diagnostic criteria. STEMI diagnosis and treatment followed the latest guidelines applicable during the study period.[12,13] All patients were given dual antiplatelet therapy, comprising an initial dose of aspirin along with P2Y12 receptor antagonists (clopidogrel, prasugrel, or ticagrelor). This regimen was continued for at least 12 months post DES implantation. Patients admitted with STEMI (<12 hours) who underwent primary angioplasty with single VLSs (≥38 mm) or ≥2 OS (total stent length ≥38 mm) in culprit lesion were included. The exclusion criteria were treatment of bifurcations with the 2-stent technique, the use of bare metal stents, and long nonculprit lesions. The lesion length was measured by visual estimation. Written informed consent was obtained from all participants. This retrospective cohort study received approval from the Research Ethics Committee and adhered to current guidelines for medication and PCI strategies, chosen by the operators. Manuscript preparation followed the Strengthening the reporting of observational studies in epidemiology checklist guidelines.[14]

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Clinical Data and Outcomes

Clinical data were collected during the hospital stay and included: baseline clinical and angiographic characteristics, medical history, and procedural aspects. Echocardiography assessment was performed during the hospital stay according to the hospital routine. The primary endpoint was the combined outcome of mortality and target lesion failure (TLF) on the culprit vessel (reinfarction, stent thrombosis, or new revascularization) up to a maximum follow-up of 2 years. The secondary endpoint was each individual outcome analyzed. Long-term follow-up was ascertained through clinical visits or telephone contact with patients or their families. Time-to-event was expressed in months.

Definitions

Sudden cardiac arrest was identified as an episode of cardiac arrest necessitating resuscitation interventions such as ventilation, chest compressions, and defibrillation. Chronic kidney disease was classified as either kidney damage or a glomerular filtration rate persistently below 60 mL/min/1.73 m² for a duration of 3 months or longer, regardless of the underlying cause.[15] A positive family history of cardiovascular disease was described as a self-reported instance of cardiovascular disease diagnosed in first-degree relatives (parents, siblings, or children) at an age of onset younger than 55 years for men and younger than 65 years for women.[16]

Procedural outcomes were evaluated as well. A successful procedure was determined by achieving the final thrombolysis in myocardial infarction flow grading system 2 or 3 with residual stenosis below 30%. No reflow indicated inadequate myocardial reperfusion in a segment of the coronary circulation without any visible mechanical vessel blockage on angiography. Distal embolization was characterized by a sudden "cutoff" in one of the peripheral coronary artery branches of the infarct-related vessel, beyond the angioplasty site, along with a distal filling defect. The type and model of DES used were at the operator's discretion and center availability (Table S1, Supplemental Digital Content, http://links.lww.com/HPC/A267).

Statistical Analysis

Continuous variables were expressed as mean (standard deviation) or median (interquartile range) based on the presence of symmetrical and asymmetrical distribution, respectively. The normality of the distribution of each variable was assessed by the Shapiro–Wilk test. Categorical variables were expressed as relative and absolute frequencies. Differences between groups were compared using Student *t* test or Mann–Whitney *U* test as appropriate. Categorical variables were presented as absolute and percentage numbers and compared using the χ^2 test or Fisher exact test, when appropriate.

Patients treated with VLS were compared to those treated with OSs. To limit bias, propensity score matching (PSM) analysis was performed. Because of different baseline characteristics between the 2 groups and similar sample sizes, we randomly selected 40% of the OS patients to reduce the propensity score distance and large score discrepancies between groups. Random selection was performed using the SPSS platform (select cases > random sample of cases > 40% of all cases). Logistic regression was performed with stent length type as a dependent variable and the following independent variables: tortuosity, calcification, Killip class 3 or 4, culprit lesion length ≥40 mm, and culprit vessel. The calibration of the logistic regression model was assessed using the Hosmer–Lemeshow test. Then, PSM was performed using nearest neighbor methods; 2 groups of 132 patients each were created. PSM analysis was performed using RStudio (Version 2022.07.1; RStudio, R version 4.1.3). Absolute standardized differences for the covariates are listed in Table S2, Supplemental Digital Content, http://links.lww.com/HPC/A267. Univariate Cox regressions for long-term follow-up event rates of primary and secondary endpoints were calculated for the unmatched and matched groups. No multiple variable entries were used, as a multivariable analysis had already been conducted through PSM. Kaplan–Meier survival curves were constructed to present the unadjusted time-to-event data for the investigated endpoints and were compared using the log-rank test. All hypothesis tests had a 2-sided significance level of 0.05. Kaplan–Meier survival curves were created using MedCalc Statistical Software version 14.8.1 (MedCalc Software bvba, Ostend, Belgium). All remaining data were analyzed using the Statistical Package for the Social Science (SPSS), version 26.0.

RESULTS

Between 2016 and 2022, a total of 803 patients were admitted to pPCI with a total stent length ≥38 mm and were screened for this study. Of these, 156 were excluded because they met the exclusion criteria. Thus, 647 patients were included in the final analysis, with 353 and 294 patients in the VSL and OS groups, respectively. Figure 1 shows a flow diagram of the study.

The rates of sudden cardiac arrest (VLS, 3.2% vs. OS, 8.9%), Killip class 3 or 4 at admission (VLS, 8.2% vs. OS, 16.7%), and

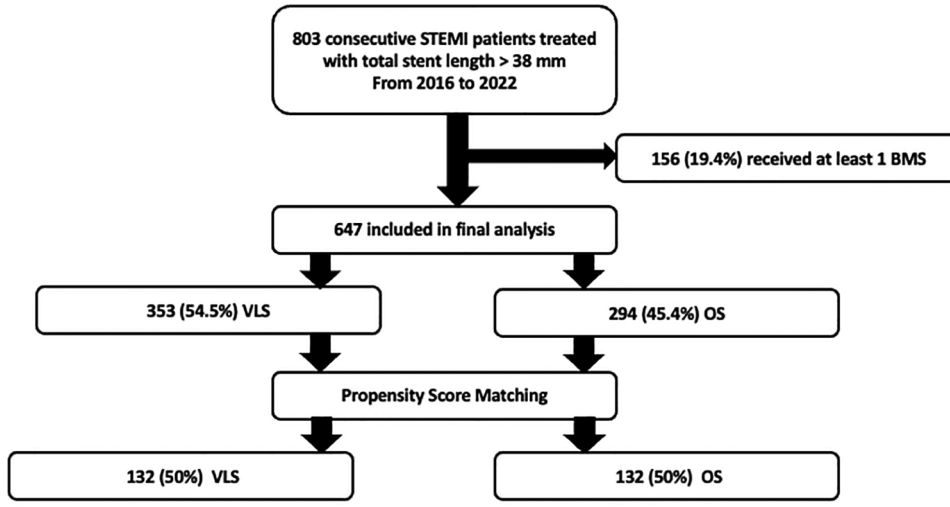

FIGURE 1. Flowchart of the study.

TABLE 1. Baseline Characteristics

	Overall			Propensity score matching		
	VLS (length ≥38 mm) n = 353	OS ≥2 stents (total length ≥38 mm) n = 294	*P*	VLS (length ≥38 mm) n = 132	OS ≥2 stents (total length ≥38 mm) n =132	*P*
Age, y	61.8 (±11.5)	62.5 (±11.6)	0.43	60.5 (±11.5)	62.8 (±11.1)	0.26
Male sex	268 (75.9)	194 (66.0)	0.05	111 (84.1)	89 (67.4)	0.002
BMI, m/kg²	27.7 (±4.7)	27.5 (±4.8)	0.50	27.8 (±4.5)	27.2 (±4.3)	0.30
Hypertension	222 (62.9)	198 (67.3)	0.23	80 (60.6)	86 (65.2)	0.44
Diabetes	99 (28)	84 (28.6)	0.88	35 (26.5)	33 (25.0)	0.77
CKD	22 (6.3)	12 (4.1)	0.22	13 (9.8)	6 (4.5)	0.09
Smoking, current or previous	215 (60.9)	165 (56.1)	0.21	91 (68.9)	82 (62.1)	0.24
Family history of CAD	54 (15.3)	38 (13.0)	0.39	22 (16.8)	14 (10.7)	0.15
Atrial fibrillation	8 (2.3)	12 (4.1)	0.18	5 (3.8)	2 (1.5)	0.25
Chronic obstructive pulmonary disease	32 (9.1)	20 (6.8)	0.29	16 (12.1)	9 (6.8)	0.14
Previous MI	50 (14.2)	53 (18.0)	0.18	13 (9.8)	19 (14.4)	0.25
Previous HF	18 (5.1)	15 (5.1)	0.99	6 (4.5)	5 (3.8)	0.75
Precious PCI	51 (14.4)	47 (16.0)	0.58	15 (11.4)	15 (11.4)	1.00
Previous CABG	9 (2.5)	7 (2.4)	0.89	2 (1.5)	2 (1.5)	1.00
Previous stroke	18 (5.1)	19 (6.5)	0.45	10 (7.6)	3 (2.3)	0.04
Sudden cardiac arrest	11 (3.2)	26 (8.9)	0.002	3 (2.3)	7 (5.3)	0.19
Anterior MI	152 (43.9)	113 (38.4)	0.21	57 (43.2)	48 (36.4)	0.25
Killip 3 or 4	29 (8.2)	49 (16.7)	0.001	13 (9.8)	21 (15.9)	0.14
Heart rate, bpm	80 (±18)	80 (±20)	0.99	81 (±18)	79 (±19)	0.42
Mean blood pressure, mm Hg	102 (±24)	105 (±28.2)	0.33	99 (±24)	103 (±23)	0.40
Total ischemic time, min	364 (237–514)	361 (238–538)	0.92	318 (245–494)	264 (240–600)	0.34
Creatinine, md/dL	1.06 (0.8–1.1)	0.98 (0.77–1.2)	0.66	0.98 (0.83–1.2)	0.95 (0.7–1.1)	0.53
LVEF, %	48 (±11)	49 (±12)	0.38	49 (±11)	49 (±12)	0.88

BMI indicates body mass index; CAD, coronary artery disease; CAGB, coronary artery bypass graft; CKD, chronic kidney disease; HF, heart failure; MI, myocardial infarction; LVEF, left ventricular ejection fraction.

occurrence of vessel dissection (VLS, 0.8% vs. OS, 5.4%) were significantly lower in the VLS group. The patients in the VLS group were also older (VLS, 75 vs. OS, 66 years). Lesion and stent lengths differed between the groups, with the OS group presenting longer lesions and stents (Tables 1 and 2 for further details).

After PSM, the study sample comprised 264 patients (132 in the VLS group and 132 in the OS group). Differences in the baseline characteristics described above were no longer statistically significant, except for age and angiographic aspects, which were already included in the model. The baseline characteristics of patients in the VLS and OS groups before and after PSM are summarized in Table 1, and the procedural characteristics are shown in Table 2.

Unadjusted Outcomes

After a median follow-up of 16 months, the overall long-term mortality rate was 10.4% and the TLF rate was 7.9%. In the overall population, there was no significant difference in the primary endpoint between the VLS and OS groups. Moreover, the occurrence of all-cause mortality, TLF, stent thrombosis, and new revascularization was also similar between the groups, but the occurrence of new myocardial infarction was higher in the OS group (Table 3 and Figures S1–S3, Supplemental Digital Content, http://links.lww.com/HPC/A267).

Propensity Score Matching

After PSM, the overall mortality and TLF rates were 8.7% and 7.6%, respectively. After adjustments by PSM, the occurrence of the primary outcome, all-cause mortality, TLF, reinfarction, stent thrombosis, and new revascularization were similar between the groups. These findings are summarized in Table 3. During the follow-up period, cumulative event-free rates of all-cause mortality and TLF rates were comparable between the VLS and OS groups (Fig. 2).

DISCUSSION

In this prospective cohort study evaluating STEMI patients undergoing primary PCI, we observed no significant differences in overall mortality and TLF between patients treated with a single VLS and those treated with OSs. To the best of our knowledge, this is the first study to compare the outcomes of VLS and OSs in STEMI patients undergoing pPCI with only DESs.

Despite the lower deliverability and increased risk of stent fracture attributed to VLS,[17–19] its use might have some potential advantages in primary PCI. Covering the culprit lesion with a single VLS means that an OS segment will be absent, which has been associated with a lower risk of stent thrombosis and restenosis.[20] Single balloon inflation may also be desired since increased manipulation is associated with slow/no reflow in the STEMI context. Another advantage is the potentially lower procedural time and contrast volume, as there is only one stent deployed. On the other hand, OS has some advantages, such as flexibility in treating multiple or diffuse lesions, better apposition in the vessel wall, better deliverability in very tortuous vessels, and lower risk of stent fracture.[21]

The potential reasons for the worse OS outcomes can be attributed to several factors.

TABLE 2. Procedural Characteristics

	Overall			Propensity score matching		
	VLS (length ≥38 mm) N = 353	**OS ≥2 stents (total length ≥38 mm) n = 294**	***P***	**VLS (length ≥38 mm) n = 132**	**OS ≥2 stents (total length ≥38 mm) n = 132**	***P***
Culprit vessel			0.60			0.65
LAD	158 (40)	116 (39.5)		58(43.9)	51(38.6)	
Cx	20 (5.8)	15 (5.8)		9 (6.8)	6(5.3)	
RCA	164 (46.7)	154 (52.4)		63(47.7)	56(55.3)	
LM	2 (0.9)	3 (1.0)		1 (0.8)	0 (0.0)	
Other	7 (2.0)	6 (2.0)		1(0.8)	1(0;8)	
Multivessel disease	174 (49.7)	156 (53.1)	0.39	58 (44.3)	61 (46.2)	0.75
Tortuosity	14 (4.0)	27 (9.3)	0.07	10(7.6)	11 (8.3)	0.82
Calcification	21 (6.0)	22 (7.6)	0.43	9 (6.8)	7 (5.3)	0.60
Femoral access	85 (24.4)	78 (26.7)	0.50	35 (26.7)	32 (24.2)	0.64
Preprocedure TIMI flow			0.29			0.79
0	296 (83.9)	231 (81.5)		109 (82.6)	103 (78.0)	
1	37 (10.5)	40 (13.6)		16 (12.1)	21(15)	
2	13 (3.7)	12 (4.1)		5 (3.8)	5 (3.8)	
3	7(2.0)	11 (3.7)		2 (1.5)	3 (2.3)	
Thrombus aspiration	74 (21.1)	51 (17.5)	0.25	40 (30.3)	28 (21.4)	0.09
TIMI thrombus scale			0.01			0.31
1	8 (2.3)	3 (1.1)		3 (2.3)	2 (1.5)	
2	14 (4.1)	9 (2.8)		6 (4.5)	4 (3.0)	
3	22 (6.4)	19 (6.7)		8 (6.1)	6 (4.5)	
4	26 (7.6)	45 (15.8)		11 (8.3)	22 (16.7)	
5	274 (79.7)	209 (73.6)		104 (78.8)	98 (74.2)	
Bifurcation (medina)			0.0001			0.22
001	2 (0.6)	2 (0.7)		0 (0.0)	1 (0.8)	
010	5 (1.4)	6 (2.0)		1 (0.8)	4 (3.0)	
011	2 (0.6)	8 (2.7)		1 (0.8)	3 (2.3)	
100	1 (0.3)	1 (0.3)		0 (0)	0 (0)	
101	6 (1.7)	4 (1.4)		3 (2.3)	3 (2.3)	
110	13 (3.7)	27 (9.2)		6 (4.5)	14 (10.6)	
111	12 (3.4)	17 (6.5)		7 (5.4)	8 (6.1)	
Bifurcation	48 (13.8)	76 (26.3)	<0.0001	20 (15.3)	38 (28)	0.008
Postprocedure TIMI flow			0.58			0.80
0	1 (0.3)	3 (1.0)		1 (0.8)	1 (0.9)	
1	6 (1.7)	3 (1.0)		1 (0.8)	2 (0.9)	
2	21 (6.0)	18 (6.1)		6 (4.6)	9 (6.8)	
3	323 (92.0)	269 (91.8)		123 (93.9)	120 (90.9)	
Lesion length, mm	40 (±8)	54 (±15)	0.0001	46 (±11)	55 (±14)	0.0001
Lesion length >40 mm	107 (30.7)	224 (77.2)	0.0001	106 (80.3)	106 (80.3)	1.00
Total stents in procedure	1 (1–1)	2 (2–3)	0.0001	1 (1–2)	2 (2–3)	0.0001
Total stent length, mm	42 (±8.9)	69 (18.7)	0.0001	47 (±9.4)	67 (±17.0)	0.0001
Mean stent diameter, mm	3.1 (±0.4)	3.1 (±0.4)	0.19	3.0 (±0.4)	3.1 (±0.4)	0.38
Polymer			<0.0001			<0.0001
Durable	211 (61.9)	142 (50.9)		76 (59.8)	64 (51.2)	
Biodegradable	127 (37.2)	76 (27.2)		48 (37.8)	37 (29.6)	
Both	0 (0)	61 (21.9)		(0.0)	24 (19.2)	
Drug			<0.0001			<0.0001
Everolimus	130 (36.9)	84 (28.9)		67 (50.8)	37 (28.2)	
Zotarolimus	49 (13.9)	49 (16.8)		10 (7.60	22 (16.8)	
Sirolimus	170 (48.3)	76 (27.1)		54 (40.9)	39 (29.8)	
Others	3 (0.9)	2 (0.7)		1 (0.1)	0 (0.0)	
More than 1	0 (0)	77 (26.5)		0 (0)	33 (25.2)	
Predilatation	257 (72.8)	261 (88.8)	<0.0001	94 (71.2)	112 (84.8)	0.007

(Continued)

TABLE 2. Continued

	Overall			Propensity score matching		
	VLS (length ≥38 mm) N = 353	OS ≥2 stents (total length ≥38 mm) n = 294	*P*	VLS (length ≥38 mm) n = 132	OS ≥2 stents (total length ≥38 mm) n = 132	*P*
Postdilatation	206 (58.7)	228 (77.6)	<0.0001	69 (52.7)	97 (73.5)	0.001
No. overlapped stents	—	2 (2–2)	—	—	2 (2–2)	—
Strut thickness			0.07			0.34
Ultrathin	187 (54.8)	117 (42.4)		65 (51.2)	56 (45.2)	
Thin	152 (44.6)	158 (57.1)		62 (48.8)	68 (54.8)	
Other	2 (0.06)	1 (0.4)		0 (0.0)	0 (0.0)	
IVUS	0 (0.0)	8 (2.7)	0.02	0	3 (2.3)	0.08
OCT	1 (0.3)	0 (0.0)	0.36	0	0	1.00
Contrast volume, mL	204 (±82)	207 (±72)	0.68	206 (±85)	212 (±72)	0.65
Angiographic success	345 (98.0)	287 (97.6)	0.73	129 (97.7)	129 (97.7)	1.00
Procedural complications						
No reflow	18 (5.1)	26(8.8)	0.06	7 (5.3)	15 (11.4)	0.07
Distal embolization	11 (3.1)	17 (5.8)	0.09	3 (2.3)	11 (8.3)	0.02
Vessel dissection	3 (0.8)	16 (5.4)	0.001	1 (0.8)	9 (6.8)	0.01

CX indicates circumflex; IVUS, intravascular ultrasound; LAD, left anterior descending artery; LM, left main; OCT, optical coherence tomography; RCA, right coronary artery; TIMI, thrombolysis in myocardial infarction.

TABLE 3. Outcomes (Univariate and Multivariate Analyses)

	Overall				Propensity score matching			
	VLS (length ≥38 mm) n= 353	OS ≥2 stents (total length ≥38 mm) n = 292	HR (95% CI)	*P*	VLS (length ≥38 mm) n = 132	OS ≥2 stents (total length ≥38 mm) n = 132	HR (95% CI)	*P*
Primary outcome								
Mortality and TLF	51 (14.4)	52(17.7)	1.3 (0.8–1.9)	0.14	17 (12.9)	21 (15.9)	1.2 (0.6–2.4)	0.41
Secondary outcome								
All-cause death	35 (9.9)	32 (10.0)	1.8 (0.7–1.9)	0.49	10 (7.6)	13 (9.8)	1.4 (0.6–3.1)	0.41
TLF	25(7.1)	26 (8.8)	1.3 (0.8–2.4)	0.22	11 (8.3)	9 (6.8)	0.9 (0.3–2.2)	0.86
New MI culprit vessel	9 (2.5)	16 (5.4)	2.4 (1.1–5.5)	0.02	4 (3.0)	6 (4.5)	1.8 (0.5–6.6)	0.30
Stent thrombosis culprit vessel	7 (2.0)	9 (3.1)	1.7 (0.6–4.6)	0.27	3 (2.3)	3 (2.3)	1.1 (0.2–5.7)	0.85
Lesion revascularization	20 (5.7)	18 (6.1)	1.1 (0.6–2.2)	0.62	7 (5.3)	7 (5.3)	1.0 (0.3–3.1)	0.87

CI indicates confidence interval; HR, hazard ratio; MI, myocardial infarction.

First, OS creates a zone with a higher strut density, which may cause narrowing of the vessel and delayed healing of the endothelium, leading to incomplete healing and increased inflammation. Second, OS segments can alter blood flow and increase drug release both locally and distally, leading to excessive inflammation and hindered healing.[20,22,23] Additionally, the proinflammatory state of STEMI patients can increase the risk of stent thrombosis, a severe complication of PCI associated with recurrent ischemic events and death. Chronic inflammation can impair endothelial function and increase platelet activation, promoting thrombus formation on the stent surface and increasing the risk of stent thrombosis.[5,8,17]

Few studies have directly compared overlapping DES with a single long DES, but none specifically in STEMI patients. Three studies showed no differences in major adverse cardiovascular events and target-vessel revascularization after short- and long-term follow-up in patients treated with single long stents versus OSs. It is worth noting that very few patients with acute coronary syndromes were included. Our study corroborates these findings, suggesting that concerns regarding worse outcomes with OS are not confirmed in clinical practice despite angiographically more complex lesions.[1,24,25]

On the other hand, Negreira-Caamaño et al compared 678 patients, with 262 receiving VLS of 40 mm or more and 416 receiving 2 OSs. The VLS group had a lower incidence of major adverse cardiovascular events (4.4% vs. 10.7%; $P < 0.01$), driven by lower target lesion revascularization rates (1.1% vs. 4.7%; $P < 0.01$).[2] However, only 3 STEMI patients were included in the study. Additionally, 23% of patients in the OS group received at least one bare metal stent compared to 12% in the single stent group, which may have influenced the results.

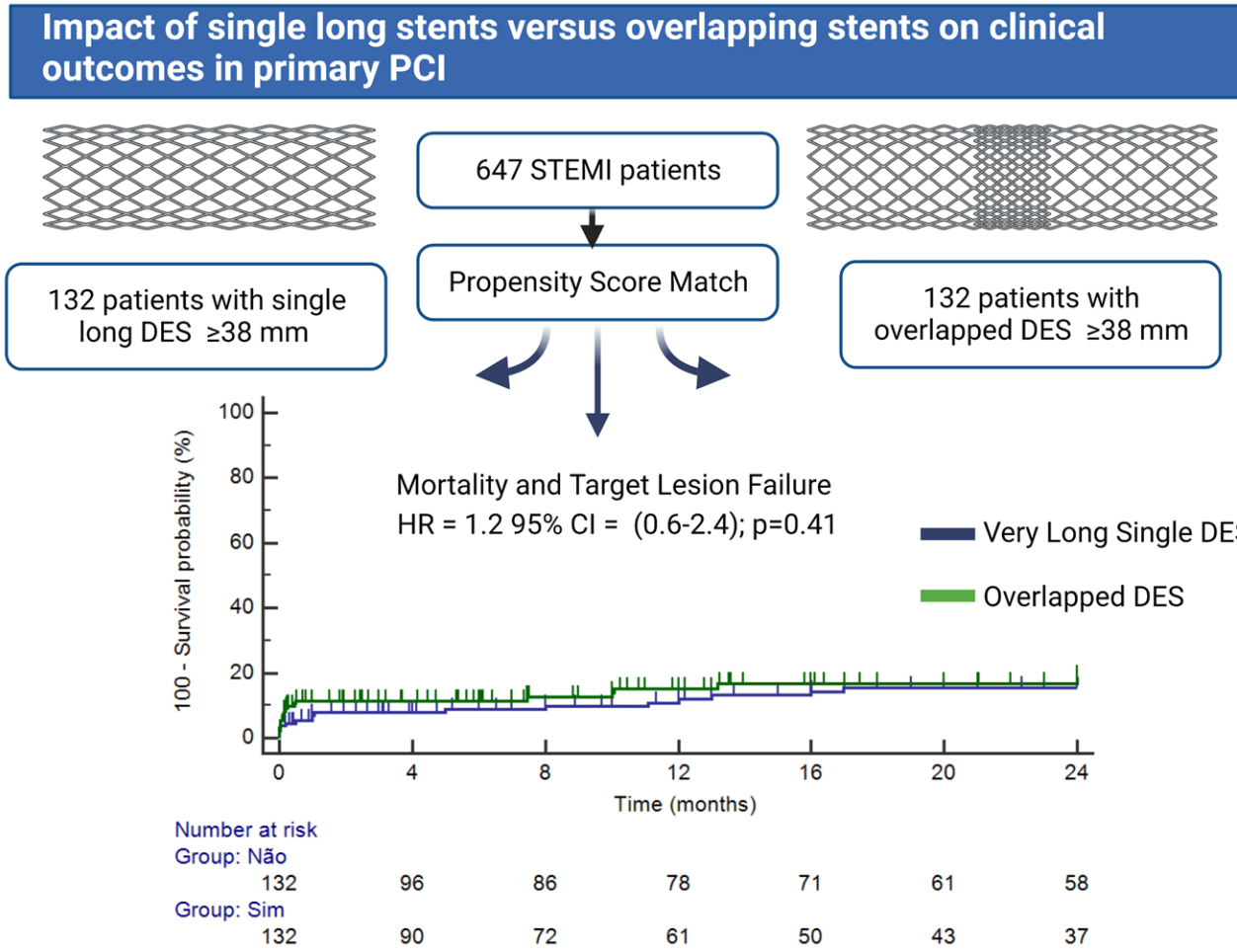

FIGURE 2. Time-to-event curves for primary endpoint. Event rates were calculated with the use of Kaplan–Meier methods. Figure created with Biorender.com.

A recent meta-analysis observed that individuals who received OS had a greater risk of cardiac mortality and target lesion revascularization than those who received VLS. Also, as expected, the fluoroscopy time and contrast volume in the OS group were higher than in the VLS group.[26] These findings are contrary to ours; however, this pooled analysis included mostly patients with stable coronary artery disease and both DES and bare metal stent, while our analysis included exclusively DES and STEMI patients.

In another prospective registry of DESs, O'Sullivan et al evaluated the clinical outcomes of 3133 patients over a 3-year period based on stent type [sirolimus-eluting stents (n =1532) vs. everolimus-eluting stents (n = 1601)] and the presence of OSs. The primary endpoint was higher in patients with OS than in those with multiple stents without overlapping or a single stent. However, a stratified analysis by stent type revealed a higher risk of the primary outcome in sirolimus-eluting stents with OS, but no significant difference between everolimus-eluting stents with OS or multiple eluting stents.[27] In our analysis, most patients received everolimus or sirolimus stenting, although a quarter of the patients in the OS group received more than one type of stent. Although some evidence suggests that OS with different stent platforms may be beneficial,[4] it is challenging to accurately determine the actual impact of multiple DESs on clinical outcomes as multiple combinations can occur. Furthermore, this finding is representative of daily clinical practice, where a mix of stent types is often used, depending on availability and operator preference.

The strengths and limitations of this study are noteworthy. First, this study was not a randomized comparison of VSL and OS; therefore, it has limitations that are inherent to observational studies such as significant residual confounding, particularly related to lesion and procedural characteristics, and unmeasured confounding may have influenced our results. Second, data regarding the total procedure time, fluoroscopy time, and radiation dose were not available. Some of these parameters could be improved with the use of VLS. Third, in some cases, OS is implanted because of residual dissection, which may have an impact on flow and ischemia, especially in the STEMI setting. Moreover, according to baseline characteristics, it appears that patients in OS group had a higher degree of coronary complexity. Also, the relatively short follow-up duration, with a median of 16 months, limits our ability to assess long-term effects and outcomes comprehensively. However, despite the few discrepancies between groups after PSM, this tool included relevant variables directly related to the primary outcome and the adjusted analysis was well-balanced. Finally, we present results from both unadjusted analyses and those adjusted for the main confounding factors in a multicenter STEMI registry with a significant number of consecutive patients, with a high applicability to daily clinical practice.

CONCLUSIONS

In our cohort of consecutive patients with STEMI treated with long stent length, we found similar rates of mortality and TLF for VSL and OS in patients submitted to pPCI. Both strategies are reasonable treatment options in the setting of STEMI.

DISCLOSURES

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