Side branch predilatation during percutaneous coronary bifurcation intervention: Long-term mortality analysis

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ABSTRACT

Background: Side branch predilatation (SBPD) during coronary bifurcation interventions is a technique that is not recommended by the latest guidelines. However, the data about the clinical outcomes after SBPD are surprisingly few.

Aims: The current study aimed to explore the association between SBPD and mortality in long-term follow-up.

Methods: All patients with coronary bifurcation stenoses revascularized with percutaneous coronary intervention were included in a prospective registry. Patients with stable angina and a bifurcation lesion with ≥50% diameter stenosis were included in the current analysis. Patients were assigned to two groups — those with SBPD(+) and those without SBPD(–). Propensity score matching was performed to equalize the risk factors and severity of coronary artery disease between the groups. A Kaplan–Meier analysis with a log-rank test for between-group differences was also performed.

Results: From January 2013 to June 2021, 813 patients were included in the final study population. The mean age was 67 (10) years. After propensity score matching, 648 patients remained for analysis — 324 in each group. At a median follow-up of 57 months patients in the SBPD(+) group had a higher all-cause mortality (n = 107 (33%) vs. n = 98 [30.2%]; $P = 0.045$) and cardiovascular mortality (n = 82 [25.3%] vs. n = 70 [21.6%]; $P = 0.03$) when compared with SBPD(–) patients. SBPD was independently associated with all-cause and cardiovascular mortality.

Conclusion: SBPD treatment of coronary bifurcation stenoses is associated with worse patient survival in the follow-up of up to 8 years. SBPD treatment gives better angiographic results, but this did not translate into better clinical outcomes.

Key words: clinical outcomes, coronary bifurcation, percutaneous coronary revascularization, side branch

INTRODUCTION

During the last 20 years, considerable progress in the treatment of coronary bifurcation lesions has been achieved [1–4]. Nowadays there is much broader knowledge considering the treatment technique of coronary bifurcation stenoses which results in better clinical outcomes [5–8]. The one-stent technique with proximal optimization is fundamental in our current philosophy of coronary bifurcation intervention [4]. According to the latest European Bifurcation Club statements, side branch predilatation (SBPD) is generally not recommended [2–4]. The main reason is the

WHAT'S NEW?

This is the first study exploring the effects of side branch predilatation treatment during bifurcation percutaneous coronary intervention in long-term follow-up. The study showed that side branch predilatation during percutaneous coronary intervention results in better angiographic results. Surprisingly, this did not translate into better clinical outcomes. Earlier analyses failed to show an association between side branch predilatation and clinical outcomes, and a probable explanation is the shorter follow-up compared to our study. We hypothesize, that side branch predilatation could be a marker of a higher lesion complexity, as could be seen from its two independent predictors — long lesion length and high-grade side branch ostial stenosis. It could incorporate a much more global view of lesion complexity than could any score considering isolated characteristics of coronary bifurcation stenosis.

possibility of vessel dissection, which could be difficult to recross after main vessel stenting [9]. However, the data about the clinical outcomes after SBPD from dedicated studies are surprisingly few [10, 11].

The current study aimed to explore the effects of SBPD on immediate procedural results (angiographic and procedural success) and its association with all-cause and cardiovascular mortality at long-term follow-up.

METHODS

Patient selection

Data for the study were obtained from a multi-centre single-country prospective registry of all patients with coronary bifurcation stenoses from January 2013. The inclusion criteria were patients with stable angina and an angiographic bifurcation lesion in a native coronary artery with a diameter ≥2.5 mm and ≤4.5 mm and SB diameter ≥2.0 mm and >50% diameter stenosis in the main vessel. We excluded patients with non-cardiac co-morbid conditions and with a life expectancy of less than one year. We also excluded patients with left main coronary artery stenosis, total occlusion before SB occurrence, lesion of interest located in infarct-related artery, subjects with left ventricular ejection fraction <30%, subjects with moderate or severe degree valvular heart disease, or primary cardiomyopathy. All patients were managed in accordance with the Declaration of Helsinki. The study protocol was approved by the Local Ethics Committee of the two institutions — the Local Ethics Committee of University Hospital Alexandrovska, Sofia, Bulgaria, and the Local Ethics Committee of Medica Cor Hospital, Ruse, Bulgaria. All patients signed written informed consent to be included in the registry.

Procedures

Provisional stenting was the default percutaneous coronary intervention (PCI) procedure in all patients [2–4]. Predilatation of the main vessel (MV) was mandatory. The side branch was routinely wired before to MV predilatation.

SB predilatation was left at the operator's discretion but generally recommended if ostial stenosis was severe (>75% diameter stenosis), the lesion length was visually more than 10 mm and wiring was difficult. SB predilatation was performed as single balloon inflation or with kissing balloon inflation in the MV. Afterward, stenting, and proximal optimization balloon inflation (left at the operator's discretion) were performed. The SB was stented in the case of flow less than TIMI 3, when diameter stenosis at the ostium assessed visually was more than 70%, despite balloon inflation or kissing balloon inflation (KBI), and flow was compromised (less than TIMI 3), when the patient was symptomatic (i.e., with chest pain). Angiographic success was defined as the end procedural post-procedural MV diameter stenosis (%DS) of <20% and SB diameter stenosis of <70% without significant dissection and flow impairment. The periprocedural myocardial infarction was defined according to the 4th universal definition of myocardial infarction [12]. Procedural success included angiographic success in the absence of in-hospital MACE (death, stroke, and myocardial infarction). All patients received double antiplatelet therapy with aspirin 75–100 mg and P2Y$_{12}$ inhibitor.

Angiographic analysis

All the analyses were performed with dedicated General Electric QCA software and additionally with MicroDicom QCA software, following the principles for coronary bifurcation stenosis analysis proposed by the Academic Research Consortium [13, 14]. The minimal luminal diameter (MLD), reference vessel diameter (RVD), and %DS ([<RVD–MLD>/ /RVD]*100) were measured for every segment of the bifurcation (i.e., proximal and distal MV — pMV, dMV, and SB) pre- and post-intervention. True bifurcation lesions were defined as percent diameter stenosis (%DS) >50% at the SB. Lesion length was measured from the proximal main vessel to the distal main branch (i.e., we considered beginning and ending points where hypothetically the stent will be implanted). SB lesion length was not measured systematically and thus was not reported. SB closure was defined as SB ostial stenosis ≥99% with TIMI 0–1 flow. All analyses were performed by two blinded and independent investigators (PN and GD) and in the case of disagreement, a consensus was formed with additional analysis from the first author (DV).

Definition of endpoints

All patients were followed up by telephone contact and/or clinical visit at 30 days and then monthly for vital status through the insurance number in the National Insurance

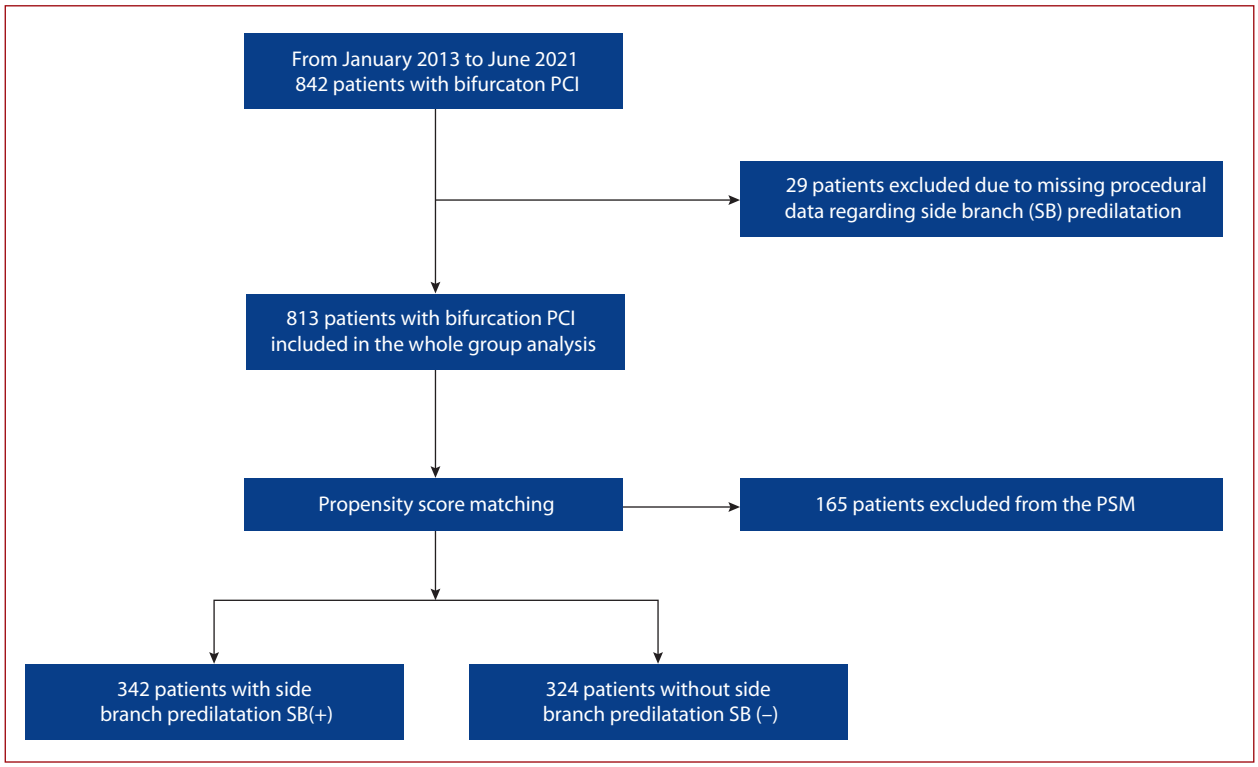

Figure 1. Flowchart. Patients included in the study

Abbreviations: PCI, percutaneous coronary intervention; PSM, propensity score matching

Institute. In the case of a registered death, the patient's general physician or relatives were contacted for more information regarding the cause of death. Cardiovascular death was defined as death with clearly determined cardiac origin or death from an unknown reason as recommended by the Academic Research Consortium-2 Consensus Document [15]. The censoring date for patients lost to follow-up was defined as the date of the last assessment.

Statistical analysis

Patients were allocated to two groups — those with performed SBPD(+) and those without SBPD(–). Continuous variables with normal distribution were expressed as the mean (standard deviation) and non-normally distributed variables as the median and interquartile range. Normal ranges were presented as the 5th and 95th percentiles. Categorical variables were expressed as counts and percentages. Differences between groups were examined with paired or unpaired t-tests as appropriate, with normal distributions. Otherwise, the Wilcoxon sign-ranked test and Mann–Whitney U-tests were used. Chi-square tests were applied for qualitative data. Correlation analysis was performed with Pearson or Spearman tests depending on the type of data. Propensity score matching (PSM) using the method of the nearest neighbor search was performed to equalize the effects of the following characteristics on the choice of SB predilatation strategy: age, sex, diabetes, smoking, hypertension, dyslipidemia, renal failure, cancer, chronic obstructive pulmonary disease, atrial fibrillation, left ventricular ejection fraction, and SYNTAX score. A Kaplan–Meier analysis with the log-rank test for differences between the groups was also performed. Additionally, univariate regression analysis was performed to identify independent predictors of all-cause and cardiovascular mortality. Variables that were found to be significant in the univariate analysis have been included in the multivariable Cox regression analysis, with backward elimination that was performed for the identification of independent predictors of all-cause death and cardiovascular death. Receiver operating characteristic (ROC) analysis was performed to evaluate factors influencing the performance of SBPD. The study was initiated by the investigators and approved by the ethical committees of the participating sites, where the team was collecting the data; all patients provided written informed consent for data collection. PSM was performed by using R Studio version 4.0.3. (R Foundation for Statistical Computing, Vienna, Austria) with the "MatchIt" package. Other statistical calculations were performed *via* SPSS version 23 (SPSS, US). The statistical differences were deemed significant if P <0.05.

RESULTS

Whole group

From January 2013 to June 2021 842 patients from two centers in Bulgaria (University Hospital Alexandrovska, Sofia, Bulgaria, and Specialized Heart Hospital Medica Cor, Ruse, Bulgaria) were included in a prospective registry according

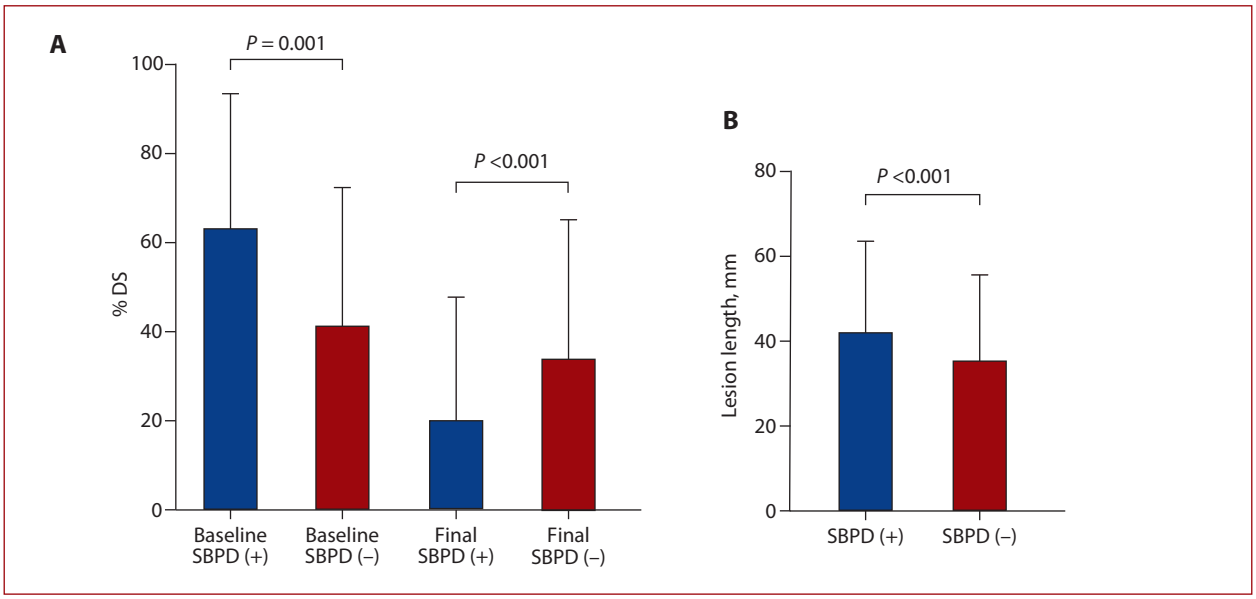

Figure 2. Comparison of the percentage of SB diameter stenosis at baseline and post-PCI in patients with and without SBPD (**A**). Lesion length in patients with and without SBPD (**B**)

Abbreviations: %DS, percentage of diameter stenosis; SB, side branch; SBPD, side branch predilatation

to the above inclusion criteria (Figure 1). Most patients (573, 68%) were enrolled in the Alexandrovska Hospital, and 269 patients (32%) were enrolled in the Medical Cor Hospital. For 29 patients, data on SB dilatation were missing, and the final study population included 813 patients who formed the study group. The mean age was 67 (10) years, 70% were males, and 40% diabetics. True bifurcations (Medina xx1) were 65% of cases. Predilatation of the SB only was performed in 5% (n = 42), whereas combined SB and MB predilatation, consecutively or simultaneously with KBI was performed in 35% (n = 249). In total, the SB was predilated in 40% of the patients. There was no significant difference between the rates of SB predilatation in the two centers (39% vs. 41%). The differences in demographic and angiographic characteristics between the groups with and without SB predilatation are presented in Supplementary material, *Table S1*.

There were significant angiographic differences between groups SBPD(+) and SBPD(–) (Supplementary material, *Table S2*). The patients with SBPD had more severe stenoses in all segments of bifurcation stenosis, longer MV lesions, and more advanced atherosclerotic disease reflected by higher SYNTAX scores and higher frequency of multivessel disease (Figure 2). The more severe disease in the SBPD group resulted in more SB stenting (36% vs. 6%; P <0.001), longer total stent length (55 [31] mm vs. 42 [23] mm; P <0.001), and a higher mean number of implanted stents per procedure (2.01 [0.6] vs. 1.71 [0.5]; P <0.001). SBPD resulted in a higher number of final KBI and a higher number of final SB post dilatations — 72% vs. 52%; P <0.001. However, the rate of final angiographic success was better in the SBPD(+) group — 269 (83%) vs. 362 (74%) in SBPD(–); P = 0.01. The rate of SB closure after main vessel stenting was non-significantly different in the SBPD(+) and SBPD(–) groups (3.6% vs. 2.2%; P = 0.27). Furthermore, the rate of periprocedural myocardial infarction (MI) was not different between the groups with and without SB closure — 52% vs. 34%; P = 0.07. A ROC analysis was performed to evaluate factors influencing the performance of SBPD (Supplementary material, *Table S3*). A logistic regression analysis demonstrated that SB %DS ≥65% (OR = 6.681; CI, 4.631–9.638); P <0.001) and lesion length ≥25 mm (OR = 1.551; CI, 1.004–2.397; P = 0.048) were independent predictors of SBPD.

At up to 96 months of follow-up (median 61 months, interquartile range 39–83 months) 252 patients died (31%), of whom 187 (22%) died from cardiac reasons. Figure 3 illustrates the Kaplan–Meier survival curves for all-cause (A) and cardiac mortality (B). As seen from the figures, SBPD patients had higher all-cause (107/324, 33% vs. 145/489, 29.7%, log-rank P = 0.01) and cardiac mortality (82/324, 25.3% vs. 102/489, 20.8%, log-rank P = 0.01). After adjustments for covariates with Cox proportional hazards logistic regression, SBPD was independently related to all-cause and cardiac mortality (Table 1). The type of bifurcation (true vs. non-true) was not associated with all-cause and cardiovascular survival (log-rank P = 0.17 and log-rank P = 0.37).

The propensity score matched group

After propensity score matching, 648 patients remained for analysis — 324 in each group. The groups were well-balanced regarding all parameters, excluding clopidogrel treatment (not used in matching), which was used more frequently in the SBPD(–) group (Table 2). The groups were well balanced regarding the severity of coronary artery disease, with no significant differences in SYNTAX scores

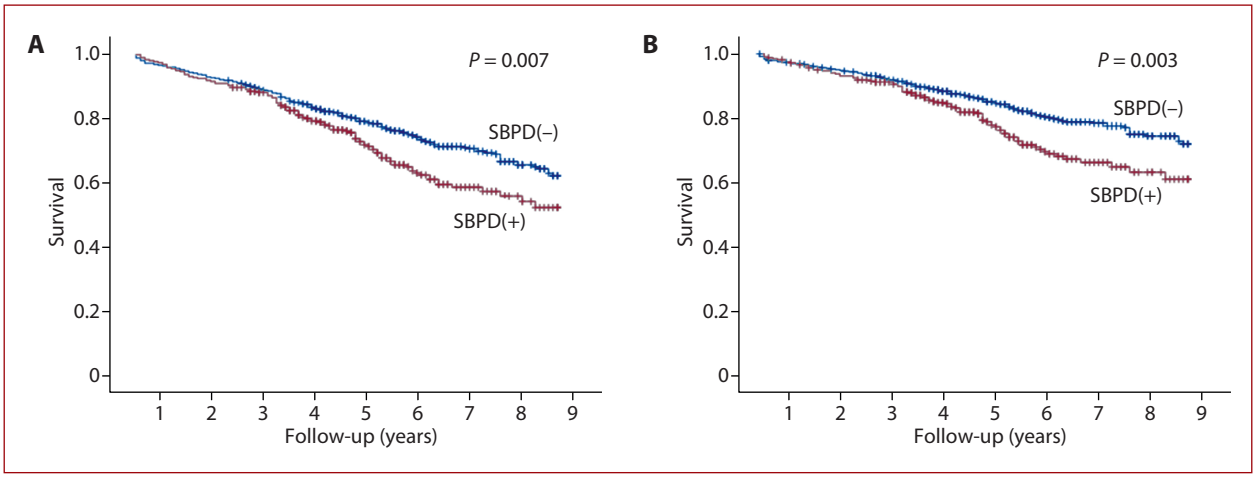

Figure 3. Whole group Kaplan–Meier survival curves. **A.** All-cause mortality. **B.** Cardiovascular mortality

Abbreviation: see Figure 2

Table 1. Independent predictors of all-cause and cardiovascular mortality

Predictor of all-cause mortality	HR	95% CI	*P*-value
Age, years	1.029	1.014–1.045	<0.001
SBPD(+)	1.329	1.006–1.756	0.045
Troponin >012 ng/ml	1.540	1.123–2.111	0.01
COPD	1.978	1.429–2.738	<0.001
Hemoglobin, g/l	0.984	0.976–0.992	0.03
Mitral regurgitation	1.307	1.074–1.592	0.01
LV PWT, mm	1.139	1.050–1.236	0.01
LVEF, %	0.985	0.972–0.999	0.04
Total model includes also: NYHA, DLP, cancer, stroke, renal failure, aldosterone inhibitor, atrial fibrillation, VKA			
Predictors of cardiac mortality			
SBPD	1.430	1.040–1.967	0.03
Age, years	1.032	1.014–1.051	0.01
Diabetes	1.645	1.185–2.283	0.01
COPD	2.246	1.563–3.228	<0.001
Troponin >0.012 ng/ml	1.756	1.200–2.570	0.01
LV PWT	1.148	1.038–1.271	0.01

Abbreviations: CI, confidence interval; COPD, chronic obstructive pulmonary disease; DLP, dyslipidemia; HR, hazard ratio; LVEF, left ventricular ejection fraction; LV PWT, left ventricular posterior wall thickness; NYHA, New York Heart Association; VKA, vitamin K-antagonist; other — see Figure 2

and frequency of multivessel disease. However, as in the whole group, SBPD(+) patients had higher-grade stenoses at all bifurcation segments and longer lesions (Table 3). The angiographic success rate was better in the SBPD(+) group (83% vs. 72%; *P* <0.001) but at the expense of higher rates of periprocedural myocardial infarction (46% vs. 30%; *P* <0.001) and troponin rise (86% vs. 78%; *P* = 0.02). Acute SB closure after stenting occurred at similar rates in the SBPD(+) and SBPD(–) groups (3.6% vs. 2.7%; *P* = 0.51), without difference in rates of MI regardless of whether SB was closed or not (n = 10/17, 59% vs. n = 227/631, 36%; *P* = 0.06). At the end of the procedure, there were 2 closed SB in the SBPD group (0.7%) and 4 closed SB in SBPD(–) (*P* = 0.46). Interestingly — in 2 of 6 finally closed SBs, the troponin rise fulfilled the criteria for myocardial infarction; however, in all patients, troponin increased by >20% from the baseline level. After logistic regression analysis, the only independent predictors of SBPD were SB %DS ≥65% (OR = 5.320; CI, 3.603–7.855; *P* <0.001) and lesion length ≥25 mm (OR = 1.685; CI, 1.060–2.679; *P* = 0.03). Again, as in the whole group, the rate of SB stent implantation was higher in the SBPD(+) group than in the SBPD(–) group (36% vs. 7%; *P* <0.001).

At a median follow-up of 57 months (interquartile range 37–78 months), 205 patients died (31.6%); 152 patients died from cardiovascular reasons (23.5%). There was no difference in all-cause and cardiovascular mortality between patients with or without periprocedural myocardial infarction (all-cause death log-rank *P* = 0.27, cardiovascular death log-rank *P* = 0.24) or those with or without SB stenting (all-cause death log-rank *P* = 0.798, cardiovascular death log-rank *P* = 0.37). SBPD(+) patients had a higher all-cause (107/324, 33% vs. 98/324, 30.2%, log-rank *P* = 0.045) and cardiovascular mortality (82/324, 25.3% vs. 70/324, 21.6%, log-rank *P* = 0.03) when compared with the SBPD(–) group (Figure 4). On multivariable Cox survival analysis, SBPD was

Table 2. Propensity score matching group — demographic characteristics

Patient characteristics	SBPD(+) n = 324	SBPD(–) n = 324	*P*-value
Age, years, mean (SD)	67(10)	68(10)	0.55
Sex, males, n (%)	230 (71)	230 (71)	0.93
Hypertension, n (%)	320 (99)	324 (100)	0.18
Hyperlipidemia, n (%)	311 (96)	314 (97)	0.54
Diabetes, n (%)	152 (47)	155 (48)	0.75
Renal failure, n (%)	104 (32)	94 (29)	0.50
Smoking, n (%)	129 (40)	120 (37)	0.49
Previous stroke, n (%)	48 (15)	55 (17)	0.57
Peripheral artery disease, n (%)	39 (12)	39 (12)	0.75
Previous myocardial infarction, n (%)	84 (26)	104 (32)	0.13
Previous PCI, n (%)	172 (53)	165 (51)	0.61
COPD, n (%)	42 (13)	42 (13)	1.00
Atrial fibrillation, n (%)	74 (23)	71 (22)	0.71
DOAC, n (%)	29 (9)	16 (5)	0.10
Clopidogrel treatment, n (%)	249 (77)	275 (85)	0.01
LVEF, %	55 (10)	55 (10)	0.74
Clopidogrel, n (%)	253 (78)	256 (79)	0.40
Prasugrel, n (%)	23 (7)	25 (8)	0.21
Ticagrelor, n (%)	48 (15)	43 (13)	0.03

Abbreviations: DOAC, direct oral anticoagulant; PCI, percutaneous coronary intervention; SD, standard deviation; other — see Table 1

Table 3. PSM group — angiographic and procedural characteristics

Angiographic parameter	SBPD(+)	SBPD(–)	*P*-value
SYNTAX score, mean (SD)	13 (7)	13 (6)	0.23
pMV RVD, mm, mean (SD)	3.26 (0.41)	3.37 (0.43)	<0.001
pMV %DS, %, mean (SD)	62 (29)	57 (31)	0.05
dMV RVD, mm, mean (SD)	2.89 (0.37)	3.11 (1.88)	0.06
dMV %DS, %, mean (SD)	71 (24)	68 (25)	0.06
SB RVD, mm, mean (SD)	2.36 (0.31)	2.37 (1.08)	0.93
SB %DS, %, mean (SD)	68 (25)	41 (32)	<0.001
SB %DS, %, after stenting, mean (SD)	42 (36)	48 (33)	0.03
SB %DS, final, mean (SD)	20 (29)	34 (31)	<0.001
SB stent, mean (SD)	36 (9)	6 (4)	<0.001
Lesion length, mm, mean (SD)	42 (21)	36(20)	<0.001
MB postdilatation, POT, (%), mean (SD)	249 (77)	204 (63)	<0.001
KBI, n (%)	165 (51)	71 (22)	<0.001
Multivessel disease, n (%)	249 (77)	227 (70)	<0.001

Abbreviations: %DS, percentage of diameter stenosis; dMV, distal main vessel; KBI, kissing balloon inflation; pMV, proximal main vessel; POT, proximal optimization technique; RVD, reference vessel diameter; SB, side branch; other — see Figure 2

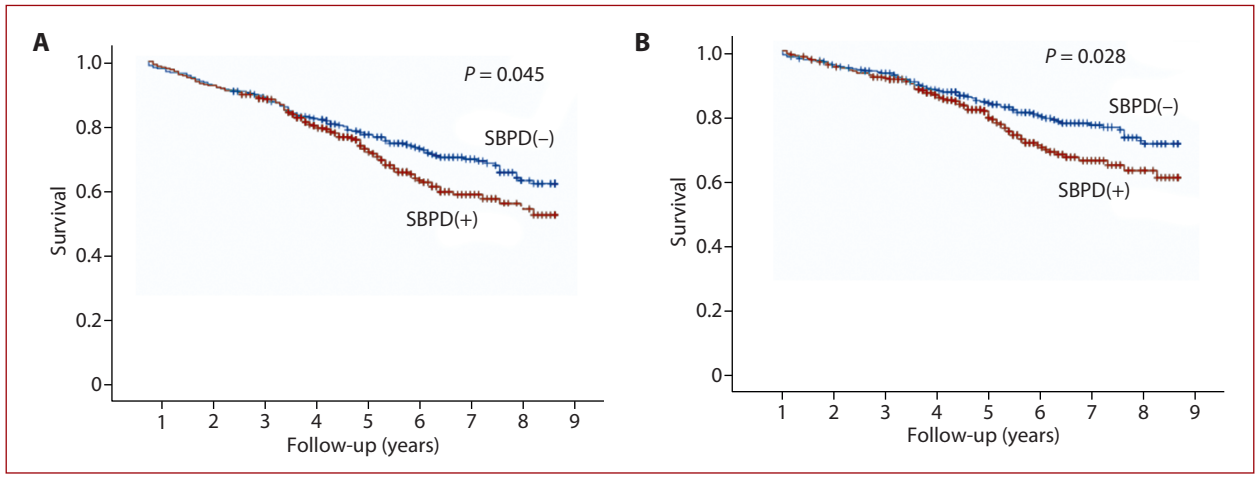

Figure 4. Kaplan–Meier survival curves for propensity score matching group. **A.** All-cause mortality. **B.** Cardiac mortality

Abbreviation: see Figure 2

Table 4. Predictors of mortality in the group matched after propensity score matching

Predictor of all-cause mortality	HR	95% CI	*P*-value
Age, years	1.033	1.015–1.051	<0.001
SBPD	1.363	1.011–1.836	0.04
Troponin >0.012 ng/ml	1.625	1.120–2.358	0.01
COPD	1.802	1.254–2.591	<0.001
Hemoglobin, g/l	0.989	0.979–0.998	0.02
Mitral regurgitation	1.269	1.017–1.584	0.04
LV PWT, mm	1.136	1.034–1.248	0.01
Statin treatment	0.500	0.251–0.999	0.05
Total model includes also: diabetes, smoking, NYHA, cancer, renal failure, SYNTAX score, atrial fibrillation, LVEF			
Predictors of cardiac mortality			
SBPD	1.483	1.047–2.099	0.03
Age, years	1.042	1.019–1.065	0.01
Diabetes	1.626	1.131–2.339	0.01
COPD	2.146	1.445–3.185	<0.001
Troponin >0.012 ng/ml	1.858	1.204–2.866	0.01
LV PWT	1.111	1.003–1.231	0.04
Statin treatment	0.345	0.161–0.739	0.01

Total model includes also: smoking, NYHA class, renal failure, hemoglobin, treatment with aldosterone inhibitor, atrial fibrillation, VKA, SYNTAX score, mitral regurgitation, LVEF, LBBB

Abbreviations: LBBB, left bundle branch block; other — see Figure 2 and Table 1

independently associated with all-cause and cardiovascular death (Table 4). Similar, to the whole group in the PSM population, the type of bifurcation (true vs. non-true) was not associated with all-cause and cardiovascular survival (log-rank $P = 0.512$ and log-rank $P > 0.05$).

DISCUSSION

To the best of our knowledge, this is the first study with long follow-up exploring the effects of SB predilatation. The main findings can be summarized as follows: 1) For the first time, it was demonstrated that SBPD is an independent predictor of higher all-cause and cardiac mortality; 2) The difference in mortality between the two groups/became evident at more than two years after PCI; 3) This effect was observed in the whole group analysis and after propensity score matching.

The largest randomized study compared the effect of SBPD on the immediate procedural outcome and very short-term clinical outcomes, at 6 months [10]. The study included patients with left-main (LM) and non-LM stenoses, which makes reaching definitive conclusions challenging. Interestingly, the study findings (better SB flow during all procedure steps, equal difficulty of SB rewiring after main vessel stenting) contradict all consequent recommendations, which are against SBPD [2–4]. Most of the data considering the clinical impact of SBPD come from Korean databases — they investigated in the COBIS (Coronary Bifurcation Stenting) registry [16–20]. Regarding the procedural effects in SBPD groups, there were better angiographic results despite higher rates of SB ostial dissection and higher rates of SB stent implantation. The earlier data, from the first round of recruitment, demonstrated worse results with predilatation, while the more recent data showed better results, with no differences regarding the frequency of target vessel failure, without affecting the rates of mortality and myocardial infarction [16–18]. Moreover, the type of SBPD (balloon only or kissing-balloons inflation) does not seem to affect the outcome [17]. The follow-up in both analyses was with a limited duration of approximately 2 years.

Earlier analyses failed to show an association between SBPD and clinical outcomes, and the probable explanation is the shorter follow-up compared to our study [19]. It is possible that more intensive antiplatelet therapy used during the first and second year after PCI (much higher rates of use of ticagrelor/prasugrel) exhibited a protective effect. However, the discontinuation of those medications later contributed to exposure to more aggressive underlying atherosclerotic disease. We hypothesize, that SB predilatation could be a marker of a higher lesion complexity, as could be seen from its two independent predictors, i.e., long lesion length and high-grade SB ostial stenosis. That could be seen even after PSM (considering SYNTAX scores). The SBPD patients had much more complex stenoses, resembling higher grade stenoses in all segments of bifurcations and longer lesion lengths, as well as more frequent SB stenting. It could incorporate a much more global view of lesion complexity than could any score considering isolated characteristics of coronary bifurcation stenosis. There was no angiographic characteristic that was independently associated with survival other than SBPD in the whole group and after PSM. We could not observe a difference in survival in true vs. non-true coronary bifurcation stenoses, even in different strata, with and without SB predilatation. This is a difference from the previous study, and the reason is unclear [20]. We can speculate, that a simple division of bifurcation stenoses into true and non-true lesions based only on the SB ostial stenosis of 50% is not sensitive enough

when considering a population with a much higher risk profile. It is possible, that for patients with less severe risk factor profile, this division is appropriate, but with the disease advancement, this becomes irrelevant.

Our results are different from the COBIS registries [16, 18, 19]. The reasons are not clear enough but could be related to the different risk profiles of the patients and more advanced atherosclerotic disease in our population, as well as selection bias, as in any observational study. Our patients had much higher risk profiles — almost everyone had hypertension, dyslipidemia, diabetes (around 40%) nicotine addiction, and chronic renal insufficiency (30%) — all rates were several times higher than in other studies [11–15]. Practically, half of our study population had previous PCI, and, as such, the calculated SYNTAX score resembled mainly the contribution of coronary bifurcation stenosis to total coronary artery disease burden.

In our series of patients, the rates of acute SB closure (3%) and final closure (0.9%) are similar to the results of Pan et al. [10], much lower than in Korean studies — at around 10% post-main vessel stenting SB closure [16–19]. That could have resulted from different definitions our definition of SB ostial occlusion (TIMI 0–1 flow) and other definitions (less than TIMI 3 flow during PCI). As with previous studies, patients with SB closure had much higher rates of periprocedural myocardial infarction, but this did not translate into worse clinical outcomes, even in patients who remained with a closed SB at the end of the procedure. The rates of angiographic success of PCI were higher with SBPD, but that did not result in lower periprocedural MI or better survival. There was no influence on survival of periprocedural MI, probably because most of those infarcts were small and did not influence the prognosis of the patients. We performed an ROC analysis to identify eventual CK, CK-MB, and troponin values before and after PCI, which could be associated with higher mortality, and only the preprocedural troponin values were associated with all-cause and cardiovascular death. The identified value (>0.012 ng/ml) was below the normal range (0.014 ng/ml), which will be further investigated.

As for the technical concern that SBPD could restrict side branch rewiring and therefore increase the rate of SB compromise or closure, this was not confirmed in our analysis. The rates of final SB closure were numerically lower (but not statistically significant) when SBPD was performed. These data were observed also after propensity score matching. Our data are in agreement with previous data from other studies [16, 18]. Interestingly, in the NORDIC I study, routine SB predilatation was a recommended strategy and again no signs of worse immediate procedural outcomes were observed [23].

Limitations

As with any observational study, our study was prone to selection bias — the patients were treated in tertiary interventional cardiology centers and therefore had more severe risk factors and significant anatomical characteristics. Hence, our data could be especially relevant for this most severe group of patients. Due to the large size of the included cohort and the long follow-up period, detailed information about the specific cause of cardiovascular death was not available.

CONCLUSION

The side branch predilatation treatment of coronary bifurcation stenoses was associated with worse patient survival in the 8-years follow-up. We suggest that SBPD is an important marker of lesion severity. It gives better angiographic results, but this did not translate into better outcomes.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/polish_heart_journal.

Article information

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