Myocardial bridging (MB) is the most common congenital coronary anomaly, with an incidence between 1.5% and 16% as assessed by coronary angiography,1,2 and up to 80% as assessed at autopsy.3 The intramural course of certain portions of coronary artery, mainly the left anterior descending (LAD), was long considered an incidental and benign clinical finding; however, there is ample evidence in the literature documenting its association with compromised systolic and diastolic coronary blood flow causing clinical symptoms including angina, coronary thrombosis, myocardial infarction, life-threatening arrhythmias, Takotsubo like syndrome, sudden death, QT-interval changes, transient left ventricular dysfunction, coronary artery spasm, myocardial stunning, and even early death after cardiac transplantation.4-22 Standard treatment of angina caused by symptomatic myocardial bridging (SMB) involves beta-blockers and non-dihydropyridine calcium-channel blockers because they reduce heart rate and myocardial contractility.23,24 On the contrary, nitrates, by reducing the intrinsic coronary wall tension and increasing reflex sympathetic stimulation of contractility, may worsen symptoms, and their use is contraindicated.2,25 In patients with persistent symptoms resistant to medical therapy, the surgical procedure of choice is dissection of the overlying myocardial fibers (unroofing) with complete exposure of the coronary artery or bypass grafting, or the combination of the two.26-28 Intracoronary stent implantation as an alternative treatment in patients with SMB has been reported,29-33 confirming that intracoronary stent implantation results in relief of mural vessel compression and thus alleviation of myocardial ischemia.34-38 However, in only 1 existing long-term follow-up prospective trial consisting of 11 consecutive patients with SMB, relatively high in-stent restenosis (ISR) and target lesion revascularization (TLR) rate (36%) was reported in bare-metal stent (BMS) implantation of myocardial bridging.39 In another study involving 24 patients with obstructive coronary artery disease (CAD), stent placement in a LAD lesion such that the stent extended into previously angiographically not appreciated MB segment beyond the obstructive lesion, resulted in restenosis within the stented MB segment in 3 of 5 patients (60%) who required TLR.39 In another retrospective study, 12 patients received three different coronary stent types (BMS, sirolimus and paclitaxel drug-eluting stents [DESs]) following a failed attempt at maximal medical therapy and were compared with 17 medical therapy responders for recurrent severe angina, target vessel revascularization.
Myocardial Bridging and Drug-Eluting Stents

(TVR), myocardial infarction (MI), and death at follow-up. The authors concluded that coronary stent placement for medically refractory symptomatic myocardial bridge failed to relieve severe angina and was associated with high clinical restenosis.40

In addition, coronary artery perforation during stenting of MB has been reported in several instances.31-33 Nevertheless, no prospective long-term follow-up of a larger group of patients with DES implantation within bridged coronary segments has yet been reported. The objective of this study was to determine the long-term efficacy and dynamics of systolic and diastolic luminal changes within bridged segments of coronary arteries after intracoronary stenting with DES in patients with SMB and absence of obstructive coronary atherosclerosis.

Methods

Patient selection. The study population consisted of 15 consecutive non-CAD patients (13 men and 2 women) with SMB of the mid-portions of the LAD and minimal luminal diameter systolic narrowing of the tunneled segment of ≥50% and diastolic luminal reduction ≥20%, determined on two orthogonal projections. In 1 patient, MB was simultaneously present in the mid-portions of the LAD and LCX coronary artery. Patient demographic and clinical data and results of non-invasive tests for myocardial ischemia are given in Table 1.

All patients had multiple previous hospital admissions because of symptoms of angina, 1 had a history of previous non-transmural MI, and 1 was admitted to the hospital for non-ST elevation MI (NSTEMI). Treadmill exercise stress testing showed significant ST-segment depression of >0.2 mV or terminal T-wave inversion in the anterior leads during or after exercise in 6 patients (40%), while 9 patients (60%) had a positive Tc-99m sestamibi single-photon emission computed tomography (SPECT) with filling defects during stress which were reversible at rest. MB of the mid-portions of the LAD was detected in all patients on coronary angiography, with no evidence of coronary atherosclerosis. The study protocol was approved by the ethics committee of the Clinical Hospital Center Zagreb and School of Medicine University of Zagreb. Written informed consent was obtained from all patients.

Clinical and non-invasive assessment of myocardial ischemia was done every 6 months over a 5-year period and repeat quantitative coronary angiography (QCA) was performed 12 and 24 months post procedure if not urged differently by recurrence of clinical symptoms and/or positive ischemia tests. The primary endpoints of the study were ISR (≥250% diameter stenosis in the stented segment), TLR (repeat percutaneous or surgical revascularization for a stenosis ≥50% by quantitative analysis anywhere within the stent or 5 mm beyond proximal or distal borders of the stent), LLL (the difference in millimeters between the minimal luminal diameter (MLD) post procedure and at angiographic follow-up exams), and permanent disappearance or significant improvement of clinical symptoms.

Quantitative coronary angiography and luminal diameter measurements. The minimal systolic luminal diameter (MSLD) and minimal diastolic luminal diameter (MDLD) of the bridged/stented segments were measured on cardiac work-station before, immediately after, and 12 and 24 months after stenting by two independent observers blinded to each other’s readings, using identical digital cinemages of at least 2 orthogonal projections of monoplane digital images and QCA commercial software (Centricity Radiology RA 600 Cardiac Review, version 7.0; GE Medical Systems, Inc).

In addition to MSLD and MDLD, the following variables were measured or automatically calculated: percent systolic and diastolic luminal diameter reduction and percent luminal cross-sectional area (CSA) reduction at the site of severest bridge narrowing during systole and mid-diastole, acute systolic and diastolic luminal gain (mm), in-stent LLL (mm), reference diameter of the adjacent proximal and distal epicardial segments (mm), and the total length of the bridged segment (mm).

Stent implantation. All patients received dual-antiplatelet treatment with 300 mg of aspirin and 600 mg of clopidogrel initiated 3 hours before the procedure, followed by 100 mg of aspirin and 75 mg of clopidogrel over a period of at least 9 months. In addition, to avoid possible pharmacological effect on the precise assessment of the length of the bridged segment, all vasoactive medication was stopped at least 2 days before stent implantation. In all patients in whom optimal visualization of the true length of the MB was inadequate at baseline angiography, provocative maneuvers were performed with intracoronary injection of glyceryl-trinitrate (0.2 mg) or dobutamine infusion (starting with 10 μg/kg body weight/min and increasing by 10 μg every 3 minutes), which resulted in increased systolic lumen reduction and thus allowed better delineation of the MB. At each dose, angiograms were performed until there was no further increase in the length of the MB or in the severity of maximum systolic compression.37,44 After careful determination of the bridged segment length, primary stenting with sirolimus-eluting stent (Cypher or Cypher Select; Cordis Corporation) was performed so that the stent/stents extended at least 3 mm on both sides beyond visible bridging into the epicardial portion of the coronary artery. Inflation pressures of 12–14 atm were used during DES deployment. Calculated balloon-to-artery ratio was 1.0. No intentional oversizing was attempted to achieve maximum luminal gain.

Follow-up. To assess clinical symptoms, subjective quality of life, any hospital admittances or physician visits for chest pain, and current drug treatment, all patients were clinically and non-invasively reevaluated for signs of ischemia every 6 months for a period of 5 years and invasively (angiographically) evaluated after 12 and 24 months or at any other time if required by deterioration of clinical symptoms and/or by objectively documented myocardial ischemia.

Statistical analysis. All values are given as mean ± standard deviation. MSLD, MDLD, percent luminal diameter reduction, percent luminal CSA reduction before, immediately after stenting, and 12 and 24 months post stenting, were compared using Wilcoxon signed-rank test, dependent t-test for paired samples, and one-way ANOVA. A probability value of P<.05 was considered significant. Interobserver and intraobserver variability was calculated by determination of correlation coefficient (r) and standard error of the estimate (SEE). Correlation coefficients and SEEs for absolute systolic and diastolic luminal diameter readings obtained by observers 1 and 2, using...
identical digital cineframes, before, immediately after, and 12 months after angioplasty were $r = 0.97$, SEE 3%; $r = 0.98$, SEE, 2%; and $r = 0.96$; SEE 4% (respectively). All statistical calculations were performed using statistical package WinSTAT for Microsoft Excel, version 2009.1

Results

The measured bridge segment length was $26.6 \pm 9.2$ mm, mean number of stents was $1.6 \pm 0.88$, mean implanted stent length was $36.1 \pm 14.9$ mm, and average (nominal) stent diameter was $3.0 \pm 0.2$ mm (Table 2). The acute clinical success rate was 100% with respect to the absence of non-fatal MI, stroke, acute or subacute stent thrombosis, or death. However, due to the recurrence of angina, positive exercise stress test, and positive SPECT, early additional angiographic studies were necessary within the first 3-6 months in 3 patient who required repeat revascularization with plain old balloon angioplasty ($n = 2$) and DES implantation ($n = 1$) for ISR $>50%$. In all 3 patients, restenosis was focal and ranged from 53%-60%. In 1 patient with NSTEMI, stent implantation was complicated with type-3 coronary perforation (Figure 1) apparently caused by partial strut fracture (Figure 2). Quick stent-graft deployment at 14 atm (3.0/19 mm Jostent Graftmaster; Abbott Vascular) instantly stopped intrapericardial bleeding and permanently sealed off the perforation site (Figure 3). Further clinical course of the patient was entirely uneventful. Through the 5-year follow-up period, composite major adverse cardiac

Table 1. Demographic and clinical characteristics and results of non-invasive tests for myocardial ischemia in 15 patients with symptomatic myocardial bridging.

<table>
<thead>
<tr>
<th>Patients (n = 15)</th>
<th>Vessels (n = 16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>13 (86.66%)</td>
</tr>
<tr>
<td>Females</td>
<td>2 (13.33%)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>52.4 ± 11.1</td>
</tr>
<tr>
<td>Typical angina</td>
<td>7 (46.66%)</td>
</tr>
<tr>
<td>Atypical angina</td>
<td>6 (40%)</td>
</tr>
<tr>
<td>Non-ST elevation MI</td>
<td>1 (6.66%)</td>
</tr>
<tr>
<td>History of previous MI</td>
<td>1 (6.66%)</td>
</tr>
<tr>
<td>Non-specific symptoms</td>
<td>1 (6.66%)</td>
</tr>
<tr>
<td>ECG, old Q-wave</td>
<td>1 (6.66%)</td>
</tr>
<tr>
<td>ECG, non-specific ST alterations</td>
<td>2 (13.3%)</td>
</tr>
<tr>
<td>Positive stress exercise test</td>
<td>6 (40%)</td>
</tr>
<tr>
<td>Positive SPECT</td>
<td>9 (60%)</td>
</tr>
</tbody>
</table>

Data given as number (percentage) or mean ± standard deviation. MI = myocardial infarction; ECG = electrocardiogram.

Table 2. Descriptive statistical data of bridge segment length, number of stents, nominal stent diameter, and total stent length in 15 patients with 16 vessels stented for systolic myocardial bridging.

<table>
<thead>
<tr>
<th>Total Vessels (n = 16)</th>
<th>Bridge Length (mm)</th>
<th>Stents (n)</th>
<th>Stent Diameter (mm)</th>
<th>Stent Length (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>26.6</td>
<td>1.6</td>
<td>3.0</td>
<td>37.2</td>
</tr>
<tr>
<td>± SD</td>
<td>9.2</td>
<td>0.88</td>
<td>0.2</td>
<td>15.7</td>
</tr>
<tr>
<td>Median</td>
<td>24.1</td>
<td>1.0</td>
<td>3.0</td>
<td>30.5</td>
</tr>
</tbody>
</table>

Figure 1. Left lateral projection of left anterior descending immediately after stent deployment in a patient presenting with non-ST elevation myocardial infarction caused by myocardial bridging. The picture shows a type-3 coronary artery perforation with three tiny jets of contrast extravasation into free pericardial cavity at the mid-section of the fully expanded stent. The quick implantation of a stent-graft prevented the imminent cardiac tamponade and instantly stopped further intrapericardial bleeding.

Figure 2. After contrast washout, U- or V-shaped deformation of mid-portion of expanded stent, typical of partial strut fracture, is visible (black arrow).
and cardiovascular event rate, including death, non-fatal MI, TVR, stroke, late stent thrombosis, was 18.7% and was solely related to TLR (18.7%).

All patients, including patients with repeat revascularization and the patient with periprocedural coronary perforation, normalized their pre-stent positive exercise stress test (6 patients) and/or SPECT (9 patients) and remained asymptomatic up to the 5-year follow-up exam. All underwent repeat angiography after 12 and 24 months, with good long-term results and no late ISR. All patients reported disappearance or significant improvement in symptoms as well as improvement in physical exercise capacity. With the exception of beta-blockers, none of the patients were on chronic treatment with anti-ischemic drugs such as nitrates, calcium-channel blockers, or trimetazidine.

The QCA measurements are given in Table 3. The mean MB length was 26.6 ± 9.2 mm (range, 17.2-42.6 mm). Mean maximal systolic luminal diameter reduction before stent implantation was 61 ± 14.6% (range, 51%-98%). Immediately after stent implantation, it was 14.5 ± 10.5% (range, 0%-28%); \( \mathit{P}<.001 \), giving a mean acute systolic luminal gain of 1.36 ± 0.6 mm (range, 0.7-2.4 mm). After 12 months, it was 23 ± 18% (range, 3%-60%); \( \mathit{P}=1.6 \) (non-significant; versus systolic luminal diameter reduction immediately after stenting), and after

### Table 3. Results of quantitative coronary angiography in 15 patients with 16 treated vessels.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>After Stent Deployment</th>
<th>( \mathit{P}^a )</th>
<th>After 12 Months</th>
<th>( \mathit{P}^b )</th>
<th>After 24 months</th>
<th>( \mathit{P}^c )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reference diameter (mm)</td>
<td>2.8 ± 0.5 median, 2.8</td>
<td>2.9 ± 0.4 median, 2.9</td>
<td>.80</td>
<td>2.8 ± 0.6 median, 3</td>
<td>.80</td>
<td>2.9 ± 0.4 median, 3</td>
<td>.18</td>
</tr>
<tr>
<td>Minimal systolic luminal diameter (mm)</td>
<td>1.17 ± 0.5 median, 1.3</td>
<td>2.53 ± 0.35 median, 2.5</td>
<td>.001</td>
<td>2.28 ± 0.55 median, 2.4</td>
<td>.06</td>
<td>2.46 ± 0.35 median, 2.5</td>
<td>.22</td>
</tr>
<tr>
<td>% Systolic luminal diameter reduction (% stenosis)</td>
<td>61 ± 14.6 median, 57</td>
<td>14.5 ± 10.5 median, 17.5</td>
<td>&lt;.001</td>
<td>23 ± 18 median, 18.8</td>
<td>.16</td>
<td>16.5 ± 10 median, 12.8</td>
<td>.80</td>
</tr>
<tr>
<td>% Cross-sectional area reduction in systole</td>
<td>82.4 ± 8.3 median, 82</td>
<td>28.5 ± 17.3 median, 34.6</td>
<td>&lt;.001</td>
<td>37.9 ± 24.7 median, 34</td>
<td>.33</td>
<td>27.4 ± 13.8 median, 23.5</td>
<td>.08</td>
</tr>
<tr>
<td>Minimal diastolic luminal diameter (mm)</td>
<td>1.79 ± 0.27 median, 1.9</td>
<td>2.45 ± 0.29 median, 2.5</td>
<td>&lt;.001</td>
<td>2.27 ± 0.55 median, 2.5</td>
<td>.39</td>
<td>2.42 ± 0.3 median, 2.5</td>
<td>.73</td>
</tr>
<tr>
<td>% Diastolic luminal diameter reduction (% stenosis)</td>
<td>34.36 ± 8.68 median, 34.85</td>
<td>12.07 ± 6.07 median, 11.06</td>
<td>&lt;.001</td>
<td>20.76 ± 12.34 median, 20.38</td>
<td>.01</td>
<td>15.38 ± 7.31 median, 16.88</td>
<td>.07</td>
</tr>
<tr>
<td>% Cross-sectional area reduction in diastole</td>
<td>46.56 ± 12.77 median, 49</td>
<td>23 ± 9.59 median, 21.05</td>
<td>&lt;.001</td>
<td>35.87 ± 19 median, 37.5</td>
<td>.009</td>
<td>26.24 ± 12.47 median, 26.33</td>
<td>.33</td>
</tr>
<tr>
<td>Acute diastolic luminal gain (mm)</td>
<td>0.74 ± 0.4 range, 0.2-1.4 median, 0.718</td>
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</tr>
<tr>
<td>Acute systolic luminal gain (mm)</td>
<td>1.359 ± 0.6 range, 0.7-2.4 median, 1.265</td>
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<tr>
<td>In-stent late luminal loss (systole) (mm)</td>
<td>0.24 ± 0.56 range, -0.5-1.6 median, 0.05</td>
<td>0.24 ± 0.56 range, -0.5-1.6 median, 0.05</td>
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</tr>
<tr>
<td>In-stent late luminal loss (diastole) (mm)</td>
<td>0.3 ± 0.45 range, -0.3-0.8 median, 0.03</td>
<td>0.3 ± 0.45 range, -0.3-0.8 median, 0.03</td>
<td>0.3 ± 0.45 range, -0.3-0.8 median, 0.03</td>
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<td>0.3 ± 0.45 range, -0.3-0.8 median, 0.03</td>
</tr>
</tbody>
</table>

\( \mathit{P}<.05 \) considered significant. \( \mathit{P}^a \) = baseline versus post stent deployment measurements; \( \mathit{P}^b \) = post stent deployment versus 12-month follow-up measurements; \( \mathit{P}^c \) = post stent deployment versus 24-month follow-up measurements; \( \mathit{P}^d \) = 12-month follow-up versus 24-month follow-up measurements.
Despite neointimal proliferation within the first year after stent implantation, both diastolic luminal reductions after 12 months. Nevertheless, the difference was not statistically significant. In addition, and probably equally important, there was a persistent mean diastolic luminal diameter reduction before stent implantation of 34.4 ± 8.7% (range, 24.7%-46.8%). Immediately after stent implantation, it was 12.1 ± 6.1% (range, 1.5%-22.4%); \( P < .001 \), giving a mean acute diastolic luminal gain of 0.74 ± 0.4 mm (range, 0.2-1.39 mm). After 12 months, it was 20.8 ± 12.3% (range, 5%-45.9%); \( P = .01 \) (non-significant; versus diastolic luminal diameter reduction immediately after stenting), and after 24 months it was 15.4 ± 7.3% (range, 5%-25.6%); \( P = .07 \) (non-significant; versus diastolic luminal diameter reduction after 12 months). Despite neointimal proliferation within the first year after stent implantation, both diastolic luminal reductions after 12 and 24 months showed persistent improvement compared to baseline values: \( P = .005 \) and \( P = .001 \), respectively. As a measure of neointimal proliferation, in-stent LLL determined after 12 months in systole and diastole for the whole group of patients (including patients with TLR) was similar at 0.24 ± 0.56 mm (range, -0.5–1.6 mm) and 0.3 ± 0.45 mm (range, -0.3–0.8), respectively, and there was no significant additional luminal diameter loss after the first year of follow-up. Actually, in 3 patients with TLR, a further increase in luminal diameter was noted on 12-month and 24-month angiography, which accounted for negative values of LLL after 24 months. This was explained by the effect of repeated balloon angioplasty and additional stent implantation at higher pressures and possibly by positive vascular remodeling of the stented segment after stent implantation. When analyzed separately, patients with ISR and TLR had LLL at 12-month follow-up of 1.12 ± 0.5 mm, as opposed to 0.08 ± 0.37 mm in patients without significant ISR. Two of 3 patients with ISR had only positive pre-stent exercise stress test, while 1 patient had both positive pre-stent SPECT and exercise stress test, thus making functional testing results non-predictive of early ISR in patients with SMB.

**Discussion**

Intracoronary stent implantation has been performed before in patients with MB. However, the information available from these isolated clinical cases and one small clinical trial using BMS are scarce, limited, and over 10 years old. In addition, the rare case reports on DES implantation in individual patients with MB are sporadic and anecdotal. DES implantation has dramatically reduced the rates of ISR and TLR compared to BMS use in patients with CAD. As yet, no prospective long-term follow-up of a group of patients with DES implantation for SMB has been reported. Although the mechanism of ischemia in fixed obstructive CAD and in dynamic obstruction in MB is distinctly different, it is reasonable and conceivable to expect that DESs should prove equally efficient in reducing ISR in patients with SMB, since they proved to do so in CAD patients.

Since common recommendations on indications for percutaneous revascularization in patients with SMB are non-existent, the indications in our 15 patients were made in agreement with the traditionally accepted cut-off value of >50% angiographic narrowing as significant, and according to ESC/EACTS Guidelines on Myocardial Revascularization, “any stenosis >50% with limiting angina or angina equivalent, unresponsive to optimal medical therapy.”

The objective of this controlled clinical trial was to determine the long-term efficacy and dynamics of systolic and diastolic luminal diameter changes within 16 bridged segments after intracoronary stenting with DES in 15 patients with SMB, resistant to optimal medical therapy and in the absence of coronary atherosclerosis. In the only available prospective study using BMS implantation to treat patients with SMB, the percentage of mid-diastolic diameter reduction immediately after stenting was entirely abolished by neointimal proliferation at 7 weeks repeat angiography and even greater than baseline.
diastolic reduction at 6-month angiography. Furthermore, an overall ISR rate of 36% was reported. The present study not only confirmed the previously reported acute and long-term relief of systolic luminal compression\textsuperscript{37} immediately after stent implantation, but also confirmed that in spite of distinctly smaller but inevitable neointimal proliferation, DES implantation resulted in persistent and durable lessening of diastolic luminal reduction over a period of 24 months. With respect to unusually long stented segments (26.6 ± 9.2 mm), total stent length (37.2 ± 15.7 mm), ISR of 18.7%, and in-stent LLL of 0.2–0.3 mm are comparable to results obtained in unselected patients with long and complex lesions and CAD.\textsuperscript{48–50} During stent deployment, a coronary perforation with imminent cardiac tamponade occurred in 1 of our patients (6.3%). There are several reports of unexplained coronary perforation caused by stent implantation in patients with MB\textsuperscript{52,50–55} including stent fracture following stenting of MB.\textsuperscript{55} In our patient, partial strut fracture (Figure 2), which was probably induced by non-uniform stent expansion in a highly mobile and hard bridged segment area together with a forceful bridge contraction in opposite direction to balloon expansion, resulted in coronary perforation (Figure 1). Usually, stent fracture is not recognized at the time of stent placement,\textsuperscript{56} but is rather diagnosed on repeat angiography for adverse outcomes such as ISR.\textsuperscript{57} Stent thrombosis, coronary dissection with coronary aneurysm formation, and typically delayed perforation.\textsuperscript{58,59} In our patient, stent fracture caused instantaneous perforation, which is a very rare complication. This rare but serious complication may limit the operator’s tendency to utilize DES implantation on a larger scale in patients with SMB.

**Study limitations.** Given the scarcity of eligible candidates with SMB unresponsive to medical therapy, the small number of study patients, although understandable, is a major limitation of the study. Another limitation is the reliance on anatomical QCA measurements without additional use of intravascular ultrasound, optical coherence tomography, and/or fractional flow reserve (FFR), which would potentially allow for more accurate functional assessment of myocardial ischemia, better delineation of dynamics of neointimal proliferation, and more precise control of stent expansion. It is even more true since FFR became a supplemental standard to guide the decision on whether to proceed with PCI in atherosclerotic lesions by evaluating the hemodynamic significance of a fixed epicardial stenosis and is generally considered to be highly specific and to have a high positive predictive value.\textsuperscript{60} However, despite these data, it is unclear whether FFR would help guide revascularization decisions in patients with SMB, because unlike the fixed stenosis of coronary atherosclerosis, MB is a dynamic stenosis, and the usefulness of FFR in this setting, except anecdotal reports,\textsuperscript{36,61} has not been validated. Moreover, uncertainties about the right methodology (adenosine versus dobutamine challenge) together with reported limitations\textsuperscript{62} leave the issue of FFR usefulness in patients with MB unresolved and controversial. However, in spite of its objective limitations, this is the only and largest prospective long-term follow-up study of patients treated with DES for SMB.

**Conclusion**

DES implantation in selected patients with SMB unresponsive to optimal medical therapy is an effective and fairly safe therapeutic option. It provides significant and persistent long-term relief of systolic and diastolic luminal reduction and concomitant myocardial ischemia. However, there is a remaining issue of ISR and TLR of 18.7% (versus 36% with BMS) which, we believe, might further improve in the future with the use of newer-generations DESs. Nonetheless, there is a small but definite risk of coronary perforation at the time of stent deployment within MB, which in our experience appeared in 1 of 16 stented vessels (6.3%). However the exact mechanism of coronary perforation may be unclear and complex,\textsuperscript{65} apparently, partial stent fracture may play a major role in certain cases. Due to this rare, but real and potentially serious complication, patients will need to be chosen carefully and provided with individualized treatment.

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