Clinical Outcome After Primary Percutaneous Coronary Intervention With Drug-Eluting and Bare Metal Stents in Patients With ST-Segment Elevation Myocardial Infarction

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Methods and Results—A total of 3756 consecutive patients with ST-segment elevation myocardial infarction treated with primary PCI and stent implantation, recorded in the Western Denmark Heart Registry from January 2002 through June 2005, were followed up for 2 years. We used Cox regression analysis to control for confounding. The 2-year incidence of definite stent thrombosis was 1.9% in the DES group and 1.1% in the BMS group (adjusted relative risk [RR]=1.53; 95% CI=0.84 to 2.78; P=0.17). Very late definite stent thrombosis (≥12 months) was seen in 0.4% in the DES group and 0.06% in the BMS group (adjusted RR=6.74; 95% CI=1.23 to 37.00; P=0.03). The 2-year incidence of myocardial infarction was similar in the 2 groups, 5.2% in the DES group versus 6.3% in the BMS group (P=0.28; adjusted RR=1.13; 95% CI=0.81 to 1.59; P=0.47). All-cause 2-year mortality was 7.8% in the DES group and 11.4% in BMS group (P<0.004; adjusted RR=0.79; 95% CI=0.60 to 1.04; P=0.09). The 2-year incidence of target lesion revascularization was 7.2% in the DES group and 8.7% in the BMS group (P=0.09; adjusted RR=0.70; 95% CI=0.52 to 0.92; P=0.012).

Conclusions—In ST-segment elevation myocardial infarction patients treated with primary PCI, target lesion revascularization was reduced by 30% in patients treated with a DES. The risk of very late definite stent thrombosis was low but increased in patients treated with DES. DES was not associated with increased risk of myocardial infarction or death, when compared with BMS. (Circ Cardiovasc Intervent. 2008;1:176-184.)

Key Words: mortality ■ myocardial infarction ■ primary PCI ■ restenosis ■ thrombosis

Primary percutaneous coronary intervention (PCI) is a well-established treatment for ST-segment elevation myocardial infarction (STEMI).1,2 Drug-eluting stents (DESs) were developed to address the problem of in-stent restenosis in patients who underwent bare metal stent (BMS) implantation. Randomized trials have shown that sirolimus-eluting stents are superior to BMS in STEMI patients in reducing the incidence of restenosis and target lesion revascularization (TLR).3,5 The beneficial effects of paclitaxel-eluting stents in STEMI patients are less well documented, with only 1 randomized trial showing a trend toward better outcome with paclitaxel-eluting stents.6

The long-term beneficial effects of DES have been questioned by reports of increased risks of stent thrombosis,7 myocardial infarction (MI), and death.8–10 However, these possible safety problems were not confirmed in other studies.10,11 For STEMI patients treated with primary PCI, data on long-term outcomes after DES implantation are limited. A clinical database study of a relatively small STEMI patient cohort detected an initial beneficial effect that disappeared...
over time. After 3 years of follow-up, the initial significantly lower target vessel revascularization rate in this group disappeared, and an increased rate of stent thrombosis was observed in the DES group (BMS, 1.6%; sirolimus-eluting stent, 2.7%; and paclitaxel-eluting stent, 2.9%). The current study assessed the use of DES versus BMS in a large cohort of primary PCI patients who received dual antiplatelet therapy for 12 months in accordance with recent recommendations. We used data from the Western Denmark Heart Registry (WDHR) to examine the rates of stent thrombosis, MI, death, and TLR after the implantation of DES or BMS in STEMI patients treated with primary PCI.

**Methods**

### Setting and Design

The study was conducted using Western Denmark’s health care databases, which cover the entire population of the region of ~3.0 million inhabitants (55% of the Danish population). All patients were followed-up for 24 months. A detailed description of the databases has been reported previously.

### Patients and Procedures

Primary PCI for STEMI has been the recommended treatment in Denmark after the publication of the DANish trial in Acute Myocardial Infarction-2 (DANAMI-2) study in 2003. To be eligible for primary PCI, patients must meet the following criteria: (1) symptoms present <12 hours from onset of pain to time of catheterization, and (2) ST-segment elevation (at least 1 mm in 2 or more standard leads or at least 2 mm in 2 or more contiguous precordial leads) or a new left bundle-branch block. We used the WDHR to identify all primary PCIs performed from January 1, 2002 through June 30, 2005. For inclusion in the current study, patients had to receive either DES or BMS. Those treated with balloon angioplasty without stent implantation or with a combination of BMS and DES were excluded (n=91; 2.4%). During the first year of the study, only BMS were available. From 2003, DES or BMS was selected according to the preference of the PCI operator. PCI was performed according to the standard practices of the participating centers. A glycoprotein IIb/IIIa receptor blocker was administered at the operator’s discretion. The post-intervention antiplatelet regimen included lifelong acetylsalicylic acid (75 to 150 mg once daily) and clopidogrel with a loading dose of 300 mg followed by maintenance with 75 mg daily. The recommended duration of clopidogrel treatment was 3 to 12 months until November 2002 and 12 months thereafter. In patients with definite stent thrombosis, medical records were reviewed to document the use of antiplatelet therapy.

### End Points

The end points were the time to stent thrombosis (definite, probable, and possible), MI, all-cause mortality, cardiac death, and TLR. All end points were assessed within 24 months of the PCI date and ascertained from the WDHR, the Danish National Patient Registry (NPR) covering all Danish hospitals and the Danish Registry of Causes of Death. All patients were followed-up for 24 months.

### Stent Thrombosis

We defined stent thrombosis using the Academic Research Consortium’s definition, with a modification for probable stent thrombosis. Probable stent thrombosis Any unexplained death within the first 30 days after intracoronary stenting.

### Definite Stent Thrombosis

Angiographic confirmation of stent thrombosis and at least 1 of the following signs present within 48 hours: new onset of ischemic symptoms at rest, new electrocardiographic changes suggestive of acute ischemia, or typical increase and decrease of cardiac biomarkers.

### Probable Stent Thrombosis

Any unexplained death occurring from 30 days after intracoronary stenting until the end of the follow-up period. Stent thrombosis was further characterized as acute (0 to <24 hours), subacute (≥1 day to <30 days), late (≥30 days to <1 year), and very late (≥1 year to 24 months).

We defined new MIs as hospitalization for MI occurring ≥28 days after the index PCI. We ascertained admissions and readmissions for MI (ICD-10 codes I21-I21.9) from the Danish National Patient Registry and deaths from the Civil Registration System. We validated the recorded cause of death using original death certificates obtained from the National Registry of Causes of Death and classified deaths according to their underlying cause.

From the WDHR, we ascertained TLR, defined as a repeat PCI of the index lesion or coronary artery bypass grafting, occurring within 2 years after the index stent implantation. We assessed all clinical end points occurring within 2 years of the index PCI. An expert committee reviewed relevant records and adjudicated the end points regarding stent thrombosis and cause of death. For all cases of stent thrombosis, we reviewed relevant medical records and catheterization films.

### Covariates

From the WDHR, we retrieved data on potential predictors for subsequent cardiovascular events, including the characteristics of the patients, procedures, and lesions. For each patient, we obtained data on all discharge diagnoses from the Danish National Patient Registry. We then computed the comorbidity scores using the Charlson comorbidity index, which covers 19 major disease categories, including diabetes mellitus, heart failure, cerebrovascular diseases, and cancer. The index value is a weighted summary of the diagnoses, with each weight calculated based on 1-year mortality associated with each disease in the original Charlson dataset.

Data on all key patient and procedure characteristics were >95% complete, and ascertainment of end points (stent thrombosis, death, MI, and TLR) was 100% complete.

### Statistical Methods

Distributions of continuous variables in the 2 groups were compared using either the 2-sample t test or the Mann–Whitney test, depending on whether the data followed the normal distribution. Continuous variables are presented either as median and interquartile range (25th to 75th) or mean ± SD depending on whether the data followed the normal distribution or not. Distributions of categorical variables were compared using the χ² test.

We counted end point events that occurred during the follow-up period and compared their rates for the cohort of patients with DES and the cohort with BMS. Follow-up began on the date of the index PCI procedure. In analyses with stent thrombosis as the outcome, follow-up continued until date of definite stent thrombosis, death, emigration, or until 24 months after implantation, whichever came first. In analyses with death as the outcome, follow-up continued until date of death, emigration or until 24 months after implantation. In analyses with MI as the outcome, follow-up continued until date of MI, death, emigration, or until 24 months after implantation.

We constructed Kaplan–Meier curves for patients and lesions treated with DES or BMS and used the life-table method to compute the 2-year incidence for each end point (proportion of the population at risk with the outcome of interest). Cox proportional-hazards regression was used to compute hazard ratios as estimates of the relative risk (RR) for each end point. Because the hazards were not proportional throughout the follow-up period, we computed the estimates of RR within separate time windows, where the proportionality assumption held. The RR in these analyses reflected the risk among patients alive and at risk of the specific end point at the start of each time period (eg, after 30 days or 1 year of follow-up). We controlled for age, gender, diabetes mellitus, and procedure time in
Table 1. Characteristics of Patients With STEMI Treated With Primary PCI and DES or BMS in Western Denmark from January 2002 Through June 2005

<table>
<thead>
<tr>
<th></th>
<th>DES</th>
<th>BMS</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>783</td>
<td>2973</td>
<td></td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>573 (73.2)</td>
<td>2179 (73.3)</td>
<td>0.95</td>
</tr>
<tr>
<td>Age, years (interquartile range)</td>
<td>60.0 (50–69)</td>
<td>64 (55–73)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Family history, n (%)</td>
<td>236 (30.1)</td>
<td>812 (27.3)</td>
<td>0.19</td>
</tr>
<tr>
<td>Smoking, n (%)</td>
<td>359 (45.9)</td>
<td>1268 (42.7)</td>
<td>0.10</td>
</tr>
<tr>
<td>Diabetes mellitus, n (%)</td>
<td>88 (11.2)</td>
<td>240 (8.1)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>169 (21.6)</td>
<td>657 (22.1)</td>
<td>0.23</td>
</tr>
<tr>
<td>Previous coronary artery bypass grafting, n (%)</td>
<td>18 (2.3)</td>
<td>34 (1.1)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Previous percutaneous coronary intervention, n (%)</td>
<td>35 (4.5)</td>
<td>118 (4.0)</td>
<td>0.03</td>
</tr>
<tr>
<td>Previous MI, n (%)</td>
<td>67 (8.6)</td>
<td>341 (11.5)</td>
<td>0.007</td>
</tr>
<tr>
<td>Lipid lowering therapy, n (%)</td>
<td>116 (14.8)</td>
<td>317 (10.7)</td>
<td>0.0006</td>
</tr>
<tr>
<td>Glycoprotein IIb/IIIa receptor blocker, n (%)</td>
<td>514 (65.6)</td>
<td>1717 (57.8)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Comorbidity index score, n (%)</td>
<td>0 (0)</td>
<td>547 (70.0) 2004 (67.4)</td>
<td>0.40</td>
</tr>
<tr>
<td>1–2</td>
<td>190 (24.3)</td>
<td>790 (26.6)</td>
<td></td>
</tr>
<tr>
<td>3+</td>
<td>46 (5.9)</td>
<td>179 (6.0)</td>
<td></td>
</tr>
</tbody>
</table>

Table 1 shows the characteristics of patients with STEMI treated with primary PCI and DES or BMS in Western Denmark from January 2002 through June 2005. The table highlights differences in variables such as age, family history, smoking, diabetes mellitus, and hypertension among patients treated with DES and BMS.

Results

Descriptive Data
A total of 3756 STEMI patients (4469 lesions) treated with primary PCI and stenting were followed up for 24 months. Of these, 2973 patients with 3486 lesions were treated with BMS, whereas 783 patients with 983 lesions received DES. The number of patients treated with DES was 783 (Cypher stent: n=465 [59.4%], Taxus stent: n=318 [40.6%]), and those treated with BMS was 2973. The patients’ median age was 63 years (interquartile range, 54 to 73 years), and 30.9% of patients were older than 75 years. Baseline patient and procedure characteristics (Table 1) and lesion characteristics (Table 2) differed substantially between the DES and BMS groups.

Stent Thrombosis

Definite Stent Thrombosis
The 2-year incidence of definite stent thrombosis did not differ significantly between patients treated with DES (n=19; 1.9%) and BMS (n=38; 1.1%; adjusted RR=1.53; 95% CI=0.84 to 2.78; P=0.17). Definite stent thrombosis occurred within a week in 34 lesions (60%; DES, 0.9% [n=9]; and BMS, 0.7% [n=25]) and within 30 days in 42 lesions (74%; DES, 1.2% [n=12]; and BMS, 0.9% [n=30]). Median time to occurrence of definite stent thrombosis was 5 days (interquartile range, 1 to 37 days). Rates of acute, subacute, and late definite stent thrombosis were also similar in the 2 groups (Figure 1A). The cumulative incidence of stent thrombosis over time showed an initial steep rise, followed by an almost linear slight increase up to 24 months. Very late definite stent thrombosis occurred in 4 lesions (0.4%) in the DES group and in 2 lesions (0.06%) in the BMS group (adjusted RR=6.74; 95% CI=1.23 to 37.00; P=0.03; Table 3 and Figure 2A).

Predictors of Definite Stent Thrombosis
Treatment with glycoprotein IIb/IIIa receptor blockers reduced the risk of definite stent thrombosis (adjusted RR=0.47; 95% CI=0.27 to 0.83; P=0.008). Stent length (adjusted RR=1.03; 95% CI=1.00 to 1.06; P=0.07) and procedure time (adjusted RR=1.01; 95% CI=1.00 to 1.02; P=0.12) did not influence the risk of definite stent thrombosis significantly. Neither infarct-related artery nor TIMI flow before intervention (adjusted RR=0.72; 95% CI=0.36 to 1.56; P=0.37) and TIMI flow after intervention (adjusted RR=1.52; 95% CI=0.54 to 4.25; P=0.42) were related to risk of stent thrombosis.

Definite, Probable, or Possible Stent Thrombosis
Definite, probable, or possible stent thrombosis was found in 106 patients treated with DES (2-year incidence, 3.6%) and in 28 patients treated with BMS (2-year incidence, 3.6%; RR=0.99; 95% CI=0.65 to 1.50; P=0.96; Figure 1B). After controlling for covariates, the risk of stent thrombosis (definite, probable, or possible) did not differ between the 2 groups (adjusted RR=1.09; 95% CI=0.71 to 1.67; P=0.68; Table 4 and Figure 2B).

Definite Stent Thrombosis and Antiplatelet Therapy
Among the 57 patients who developed definite stent thrombosis, 82.5% (n=47) were receiving dual antiplatelet therapy (aspirin and clopidogrel) at the time of the thrombotic event. In the 6 patients with very late stent thrombosis, 1 patient was on dual antiplatelet therapy (DES=1), 1 patient was treated with aspirin only (DES=1), and 4 patients (DES=2 and BMS=2) had discontinued both aspirin and clopidogrel after 12 months.

Mortality
All-cause 2-year mortality was lower in DES than in BMS patients (7.8% versus 11.4%; P<0.004; RR=0.67; 95% CI=0.51 to 0.88; P=0.0039; Figure 1C). This difference was not statistically significant after adjustment for covariates (adjusted RR, 0.79; 95% CI=0.60 to 1.04; P=0.09; Figure 2C). All-cause mortality 12 to 24 months after initial stenting was similar in the 2 groups (2.2% versus 2.4%; P=0.75; adjusted RR=1.04; 95% CI=0.59 to 1.83; P=0.90).

Cardiac mortality during the first 30 days was 4.6%, lower in the DES group (3.2%) than in the BMS group (4.9%) (P=0.039; RR=0.65; 95% CI=0.43 to 0.98; P=0.04). After adjustment, risk of cardiac death during the first 30 days no longer differed significantly between the 2 groups (adjusted RR=0.77; 95% CI=0.50 to 1.18; P=0.23). Cardiac mortality...
after 2 years was 6.6%, with lower cardiac mortality among DES patients than among BMS patients (5.0% versus 7.0%; \(P=0.037\); RR=0.70; 95% CI=0.50 to 0.99; \(P=0.04\)). After adjustment, risk of cardiac death did not differ significantly between the 2 groups (adjusted RR=0.83; 95% CI=0.59 to 1.18; \(P=0.31\)).

Noncardiac 2-year mortality was 3.3%, which was similar in DES-treated (2.6%) and BMS-treated (3.5%) patients (RR=0.70; 95% CI=0.44 to 1.13; \(P=0.15\)). Controlling for covariates did not change this result (adjusted RR=0.82; 95% CI=0.51 to 1.33; \(P=0.42\)).

During the second half of the follow-up period (12 to 24 months), after discontinuation of the recommended dual antiplatelet therapy, neither all-cause mortality, nor cardiac mortality, nor noncardiac mortality differed between patients treated with DES or with BMS (Table 4).

**Myocardial Infarction**

The 2-year incidence of MI was similar in the 2 stent groups (6.0% in the DES group versus 4.9% in the BMS group; \(P=0.28\) [Figure 1D]; adjusted RR=1.13; 95% CI=0.81 to 1.59; \(P=0.47\) [Figure 2D]).

**Target Lesion Revascularization**

TLR within 2 years occurred slightly less frequently in DES patients than in BMS patients (2-year incidence, 7.2% versus 8.7%; \(P=0.09\); Figure 1E). After controlling for age, gender, diabetes mellitus, stent length, reference vessel size, and glycoprotein IIb/IIIa, the risk reduction with DES was 30% (adjusted RR=0.70; 95% CI=0.52 to 0.92; \(P=0.012\); Figure 2E). Diabetes mellitus (RR=1.66; 95% CI=1.19 to 2.31; \(P=0.003\)) and stent length (RR [per 1 mm]=1.02; 95% CI=1.00 to 1.03; \(P=0.01\)) increased the risk of TLR within 24 months. Reference vessel size was inversely related to TLR (RR [per 1 mm]=0.82; 95% CI=0.67 to 1.00; \(P=0.05\)).

**Propensity Score Analysis**

The estimates of effect were unchanged when we used Cox regression with adjustment for propensity score (calculated by logistic regression as predicted probability of use of BMSs...
Figure 1. A, Risk of definite stent thrombosis among patients treated with DES or BMS. B, Risk of definite, probable, and possible stent thrombosis among patients treated with DES or BMS. C, All-cause mortality among patients treated with DES or BMS. D, Risk of MI among patients treated with DES or BMS. E, TLR among patients treated with DES or BMS (unadjusted data).
Table 3. Risk of Definite Stent Thrombosis in Patients With STEMI Treated With Primary PCI and DES (n=983) or BMS (n=3486) in Western Denmark

<table>
<thead>
<tr>
<th></th>
<th>DES</th>
<th>BMS</th>
<th>RR* (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Events</td>
<td>No./100 Person-Year</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>19</td>
<td>1.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute (24 h)</td>
<td>5</td>
<td>187.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 day to &lt;30 days</td>
<td>7</td>
<td>9.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 days to 12 months</td>
<td>3</td>
<td>0.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;12 months</td>
<td>4</td>
<td>0.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Adjusted for age, sex, diabetes mellitus, stent length, reference vessel size, procedure time, and glycoprotein IIb/IIIa.

Discussion

The current study shows that primary PCI patients treated with either DES or BMS in a real-world setting have similar 2-year risks of stent thrombosis, MI, or death. Within the same period, the use of DES was associated with 30% reduction in the risk of clinically driven TLR. To our knowledge, our study represents the largest published database of STEMI patients treated with primary PCI. The 3 interventional centers in our study are all high-volume PCI centers, and one third of the PCI procedures are primary PCI including optimized transportation logistics.1

An important clinical dilemma exists as to whether the benefit of reduced repeat revascularization with DES outweighs the harm of a possible increased risk of the rare but serious complication of late stent thrombosis. Another concern is whether the net TLR reduction after DES is maintained during longer-term follow-up, especially after cessation of dual antiplatelet therapy. Our data show that 25% of definite stent thromboses occurred acutely within 24 hours and that another 50% occurred within 30 days. During these first 30 days, the risk of definite stent thrombosis was similar in patients treated with DES or BMS. Previous studies on BMS implanted during primary PCI for MI have found high thrombosis rates. However, this is limited by the absence of a uniform definition for stent thrombosis in these studies. Current studies comparing DES and BMS in this setting have also found high rates of stent thrombosis in both groups. Stent thrombosis is therefore more frequent in primary PCI for acute MI compared with planned procedures and the rates of stent thrombosis are similar for DES and BMS. However, longer follow-up is necessary to follow the risk of very late stent thrombosis to ensure that this risk will not outweigh the benefit with reduction in TLR.

Several factors are associated with an increased risk of stent thrombosis in STEMI patients treated with primary PCI, including the risk of incomplete stent apposition19 or under-expansion of the stent itself.19 Further, the increased platelet activation in acute MI,20 coupled with delayed healing, risk of lack of endothelialization, and exposure to proinflammatory and thrombogenic environment, might explain the increased risk of stent thrombosis in STEMI patients.21 However, neither in our study nor in the 3 randomized studies (TYPHOON,24 PASSION,6 and SESAMI3) did these possible pathophysiological processes result in an increased risk of early stent thrombosis after DES implantation. In the current study, definite stent thrombosis during the first year after the cessation of dual antiplatelet therapy was more common after DES than after BMS treatment. This difference, although it was extremely small, is still important. At the same time, DES was associated with a marked and persistent reduction in TLR.

Another important finding in our study is the significantly lower risk of definite stent thrombosis in patients treated with a glycoprotein IIb/IIIa receptor blocker. In the CADILLAC trial,22 subacute thrombosis was reduced by 74% in patients treated with abciximab. However, abciximab treatment did not affect the composite end point at 1 year. Although the glycoprotein IIb/IIIa receptor blocker treatment in our study was administered at the discretion of the PCI operator, we observed a similar risk reduction of 53% in definite stent thrombosis after glycoprotein IIb/IIIa receptor blocker administration compared with abciximab administration. In the STRATEGY trial,23 patients treated with DES plus tirofiban had less angiographic stent thrombosis at 30 days compared with patients treated with the combination of BMS plus abciximab (0 versus 3 stent thromboses). In the recently published MULTISTRATEGY trial,24 tirofiban therapy, when compared with abciximab, was associated with noninferior resolution of ST-segment elevation at 90 minutes after coronary intervention, whereas sirolimus-eluting stent implantation was associated with a significantly lower than uncoated stents risk of major adverse cardiac events within 8 months after intervention. The ADMIRAL study25 did not present data on stent thrombosis, but it demonstrated that early inhibition of the platelet glycoprotein (GP) IIb/IIIa receptor with abciximab led to improved coronary patency, left ventricular function, and better clinical outcomes. Long-term follow-up in the ADMIRAL study25,26 showed that adjunctive abciximab to primary stenting for STEMI elicits
Figure 2. A, Risk of definite stent thrombosis among patients treated with DES or BMS. B, Risk of definite, probable, and possible stent thrombosis among patients treated with DES or BMS. C, All-cause mortality among patients treated with DES or BMS. D, Risk of MI among patients treated with DES or BMS. E, TLR among patients treated with DES or BMS (adjusted data).
favorable clinical outcomes with the same absolute reductions in risks of “hard” clinical outcomes from 30 days up to 3 years of follow-up.

Mortality in the current study was generally higher for patients treated with DES or BMS compared with mortality estimates reported in both a meta-analysis and in randomized clinical trials of DES versus BMS in primary PCI. This may be important for the interpretation of our data, because randomized trials (and the meta-analysis based on these trials) usually include highly selected patients. Thus, our study extends the results of the randomized trials to a real-world setting. Clinically driven TLR was lower in our study than in the randomized trials, probably because routine angiographic follow-up in such trials increases the TLR rate.

The current data did not demonstrate significant differences in the cumulative rates of death or MI after 2 years for patients treated with DES or BMS. Furthermore, all-cause mortality and cardiac mortality for patients treated with primary PCI were similar to the findings in the DANAMI-2 study.

**Limitations**

The validity of our findings depends on data quality and the ability to control for potential confounding. Our design is based on computerized registries with complete nationwide coverage, enabling study of a well-defined large population with complete follow-up. However, like all observational studies, our study is prone to biases related to unmeasured factors. A bias attributable to an unknown variable cannot be eliminated.

We collected data over a 3-year period, during which the prevalence of DES stents implanted increased from zero to 53%. To reduce bias during this transition period, we followed-up every patient for up to 24 months. It also is important to note that although we report 12 months as the recommended duration of dual antiplatelet treatment, we had only access to the actual duration of this treatment among patients with definite stent thrombosis. Finally, according to the ARC definition of probable and possible stent thrombosis, we categorized all unexplained deaths as stent thromboses. However, it is unlikely that stent thrombosis was the cause of death in all cases of unexplained death.

**Conclusions**

The results of the current study indicated that patients treated with stent implantation during primary PCI the overall rates of stent thrombosis, MI, and death were of similar magnitude in patients receiving DES or BMS after 2 years. Compared with BMS, DES improved the clinical outcome by reducing the risk of clinically driven TLR.

**Disclosures**

Dr Jensen has received lecture fees from Abbott, AstraZeneca, Cordis, and Merck; Dr Maeng has received lecture fees from Cordis and consultant fees from Novo Nordisk and Medtronic; Dr Kaltoft has received lecture fees from Cordis; Dr Lassen has received lecture fees from Bristol-Meyers Squibb, Nycomed, Boston Scientific, GlaxoSmithKline, and Cordis; and Dr Thuesen has received lecture fees from Medtronic, Abbott, Cordis, and Boston Scientific, consulting fees from Abbott, is in the advisory board of Boston Scientific, and has received research grant support from Medtronic, Abbott, Cordis, and Boston Scientific.

**References**

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