Predicting Restenosis of Drug-Eluting Stents Placed in Real-World Clinical Practice
Derivation and Validation of a Risk Model From the EVENT Registry

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Background—Prediction of restenosis after percutaneous coronary intervention (PCI) remains challenging, and existing risk assessment algorithms were developed before the widespread adoption of drug-eluting stents (DES).

Methods and Results—We used data from the EVENT registry to develop a risk model for predicting target lesion revascularization (TLR) in 8829 unselected patients undergoing DES implantation between 2004 and 2007. Using a split-sample validation technique, predictors of TLR at 1 year were identified from two thirds of the subjects (derivation cohort) using multiple logistic regression. Integer point values were created for each predictor, and the summed risk score (range, 0 to 10) was applied to the remaining sample (validation cohort). At 1 year, TLR occurred in 4.2% of patients, and after excluding stent thrombosis and early mechanical complications, the incidence of late TLR (more likely representing restenosis-related TLR) was 3.6%. Predictors of TLR were age ≥60, prior PCI, unprotected left main PCI, saphenous vein graft PCI, minimum stent diameter ≤2.5 mm, and total stent length ≥40 mm. Comparison of observed versus predicted rates of TLR according to risk score demonstrated good model fit in the validation set. There was more than a 3-fold difference in TLR rates between the lowest risk category (score 0; TLR rate, 2.2%) and the highest risk category (score ≥5; TLR rate, 7.5%).

Conclusions—The overall incidence of TLR remains low among unselected patients receiving DES in routine clinical practice. A simple risk model incorporating 6 readily available clinical and angiographic variables helps identify individuals at extremely low (<2%) and modestly increased (7%) risk of TLR after DES implantation. (Circ Cardiovasc Interv. 2010;3:327-334.)

Key Words: restenosis • drug-eluting stents • percutaneous coronary intervention • risk assessment

Recent clinical trials have demonstrated marked reductions in restenosis by drug-eluting stents (DES) when compared with bare metal stents.1 As a result, rapid adoption of DES by the cardiology community has resulted in frequent placement of DES in clinical settings for which efficacy and safety data are limited. These “off-label” patients and lesions have higher rates of adverse events including repeat revascularization,2-5 which likely contributes to the continued 5% to 7% incidence of clinical restenosis reported in contemporary registries of percutaneous coronary intervention (PCI).6,7

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Identifying patients at high risk of DES restenosis would provide clinicians with an opportunity to select therapeutic options best tailored for the individual patient, based on that patient’s unique set of clinical characteristics. Ideally, risk stratification would be based on factors known before PCI so that clinical decision-making (eg, choosing medical therapy or bypass surgery versus PCI, type of stent, number of lesions to revascularize, etc) would maximize benefit and minimize the risk of adverse outcomes such as restenosis. To better identify patients at higher risk of restenosis, several investigators have developed risk-prediction models based on clinical and angiographic variables associated with target lesion revascularization (TLR). However, these models were developed before widespread availability of DES,8,9 in single-center experiences,10,11 or in restrictive patient subsets.4 In addition, none of the predictors identified in DES populations have undergone independent validation. With DES use increasing well beyond the original indications granted by the US Food and Drug Administration, clinicians need additional guidance to predict outcomes of PCI outside of randomized controlled trials. The present study used data from an observational registry to develop a risk model for predicting TLR in DES-treated patients, which demonstrated good model fit in an independent sample. The risk stratification model identified 6 readily available variables for TLR that could be used to guide individualized clinical decision-making in DES-treated patients.
controlled trials and, in particular, in settings in which angiographic follow-up is not routine.

We therefore used the Evaluation of Drug Eluting Stents and Ischemic Events (EVENT) registry to identify predictors of clinically-driven TLR among unselected DES patients treated in “real-world” clinical practice. We then constructed a risk score for TLR and tested the model for predictive accuracy in a separate population.

Methods

Study Population
EVENT is a prospective observational registry designed to evaluate contemporary interventional practice patterns at approximately 50 US centers.12 Using broad enrollment criteria to minimize selection bias, EVENT enrolled patients age 18 years and older with a wide variety of clinical indications for PCI; the only exclusion criteria were PCI or bypass surgery within the previous 4 weeks or prior participation in the EVENT registry. Study coordinators collected demographic, clinical, and treatment variables using standardized case report forms, which were submitted to a data coordinating center (Harvard Clinical Research Institute, Boston, Mass) for cleaning and analysis. Detailed descriptions of patient angiographic characteristics and medication use before, during, and after the PCI procedure were collected as well. Periprocedural and in-hospital clinical events were recorded, and patients or their primary physicians were contacted by telephone at 6 and 12 months after the index PCI to identify specific events including death, myocardial infarction, and repeat revascularization. In the case of suspected clinical events, additional source data were obtained to allow detailed review of each event. Each institution’s human studies committee approved the study protocol, and written consent was obtained from each subject.

The present analysis included all patients who underwent placement of 1 or more DES for any clinical indication. To reduce potential bias introduced at the time of repeat revascularization for other lesions (ie, “innocent bystander PCI”), patients receiving bare metal stents or balloon angioplasty alone to any lesion at the index PCI were excluded.

Analytic Approach
Continuous variables are described as mean±standard deviation, and categorical variables are described as proportions. Statistical analyses were performed with SAS software (version 8.2, SAS Institute, Cary, NC), and probability values ≤0.05 were considered statistically significant unless otherwise specified.

Primary End Point
The primary end point was the occurrence of TLR in a given patient during the 12 months after index PCI. TLR was defined as repeat PCI or bypass graft placement for a stenosis in the DES placed at index PCI, or occurring within 5 mm of the stent (“edge effect”) as determined by the investigator at each site. Patients with missing follow-up data or those who died during follow-up (without first having TLR) were included in the analysis but were counted as having no TLR events. All follow-up events were reviewed by experienced clinical cardiologists (J.M.S., J.B.L., and D.J.C.), and repeat revascularizations were adjudicated by reviewing the associated narrative descriptions or by contacting the enrolling center when necessary.

Secondary Analysis
Because our main objective was to identify risk factors for developing clinical restenosis in DES, we performed a prespecified sensitivity analysis using late-TLR as an alternative end point. Late TLR was defined as TLR occurring between 1 and 12 months after index PCI (ie, excluding early TLR events more likely to be related to mechanical complications) and in the absence of angiographically confirmed stent thrombosis.

Derivation Cohort
We used a split-sample technique to derive and independently validate our risk score. First, we randomly designated two thirds of the study population as the derivation cohort. Within the derivation group, patients with and without TLR were compared using χ² and t tests for categorical and continuous variables, respectively, or with nonparametric alternatives when appropriate. We then constructed univariable logistic regression models to select candidate variables for predicting TLR; candidate variables were those with nominal statistical significance (at P<0.2 level) or established predictors of in-stent restenosis from prior studies. The variables considered in the univariable analyses were sociodemographic factors (age, sex, body mass index, tobacco use), clinical factors (hypertension, diabetes, hyperlipidemia, prior myocardial infarction, prior PCI, prior coronary bypass surgery, heart failure, peripheral arterial disease, indication for PCI, glomerular filtration rate), and patient-level angiographic characteristics (number of diseased vessels, PCI vessel location, number of lesions undergoing PCI, bifurcation location, in-stent restenosis, TIMI flow grade before PCI, lesion severity, presence of thrombus before PCI, maximal lesion stenosis, total stent number, minimum stent diameter, total stent length, DES stent type).

Risk Model Construction
In the derivation cohort, we used logistic regression with backward stepwise elimination (stay criterion P≤0.05) to evaluate the multivariable predictors of TLR along with their associated odds ratios (OR), 95% confidence intervals (CI), and β-coefficients. To verify that our results were not biased by patients who died or had missing follow-up data, the multivariable predictors of TLR were derived separately using Cox proportional hazards regression. This process was repeated using late-TLR as the end point of interest (secondary analysis) to determine whether predictors of the more restenosis-specific TLR definition were similar to the predictors of any TLR event.

After the TLR predictors were confirmed, we identified cut-points for the continuous variables from logistic regression by constructing a univariate plot of TLR versus deciles of the continuous variable. On the basis of these graphs, we then dichotomized each variable at a clinically relevant cut-point, using clinical judgment to help round up or down to simplify the calculation of a given patient’s TLR risk score. To estimate TLR risk using a simple additive score, we assigned each variable a “weight” based on the rounded β-coefficient for that patient characteristic.13 The overall TLR risk score for each patient was then calculated by adding the number of points for each TLR predictor identified in that individual. Only those subjects with complete cases (ie, no missing data for the predictor variables) were included in the final models. Model performance was evaluated by calculating the c-statistic for comparison with prior published TLR risk models.

Model Validation
In the remaining one third of the study population, risk scores were calculated for each patient by adding the points assigned to each variable in the TLR risk model. Rates of TLR were then evaluated and stratified according to risk score in these subjects, and a validation plot was constructed to test model accuracy. Goodness-of-fit was evaluated using Hosmer-Lemeshow testing.

Results

Overall Population and Derivation Cohort
Between July 2004 and June 2007, EVENT enrolled 10 144 subjects, of whom 8829 (87%) were treated exclusively with DES and comprised the full analytic cohort for this study (Figure 1). Of these, 235 patients died (2.7%) and an additional 593 (6.7%) had incomplete 12-month follow-up data. The overall incidence of TLR and target vessel revascularization (TVR) at 12 months were 4.2% and 5.9%, respectively. After excluding events related to stent thrombo-
sis and TLR within the first 30 days, the secondary end point (late TLR) occurred in 321 patients (3.6%). After excluding 54 individuals with missing data, the derivation cohort consisted of 5863 patients who received an average of 1.6 stents (in 1.4 lesions) per patient. Any TLR occurred in 236 patients (4.0%) and late TLR occurred in 198 patients (3.4%) in the derivation cohort.

Baseline clinical characteristics of patients with and without TLR in the derivation cohort are summarized in Table 1. Patients who had TLR were younger and had more symptomatic angina at baseline according to the Canadian Cardiovascular Society angina classification. Diabetes mellitus, hypertension, hyperlipidemia, prior PCI, and prior bypass surgery were associated with increased TLR in bivariate analysis as well. Angiographic characteristics associated with TLR are listed in Table 2 and included treatment of a saphenous vein graft (SVG) lesion, unprotected left main PCI, greater lesion complexity, in-stent restenosis lesion, greater number of lesions treated, greater total stent length, greater number of stents implanted, and smaller minimum stent diameter.

Predictors of TLR After the multivariable analysis, predictors of TLR in the derivation cohort were younger age, previous PCI, SVG lesion location, unprotected left main PCI, smaller stent diameter, and greater overall stent length. The same set of 6 predictors was identified after the multivariable analysis was repeated using proportional hazards regression and in the secondary analysis to predict late TLR. After assigning dichotomous cut-points to age (<60 years), minimum stent diameter (≤2.5 mm), and total stent length (≥40 mm), the variables in the final risk model remained unchanged (Table 3). The performance of the fully categorical model was not significantly different than the original model containing both categorical and continuous variables (c-statistic changed from 0.677 to 0.672). Based on the β-coefficients for this categorical model, integer point scores were assigned to each covariate ranging from 1 to 3 points (Table 3); the resulting summed risk score ranged from 0 to 10 points.

Risk Score Validation The validation cohort consisted of the remaining one third of DES patients in EVENT (n=2884 with complete data), with rates of TLR and late TLR at 12 months of 4.7% and 4.1%, respectively. There were no major differences in clinical or angiographic characteristics between the validation and derivation cohorts, with the exception of slightly lower diastolic blood pressure at enrollment (75 versus 76 mm Hg) and slightly more right coronary artery PCI (38% versus 36%) in the validation group. The distribution of patients by risk score in the validation set and the observed TLR frequencies for each score are displayed in Figure 2. Patients with the lowest risk scores (0 to 1 points) represented one third of the validation cohort and had TLR at a rate of 2% to 3%, whereas high-risk patients (4 to 10 points) represented 20% of the validation set and had a 1-year TLR rate of 6% to 8%—approximately 3 times higher than the lowest risk group. Comparison of observed versus expected rates of TLR according to risk score (Table 4), and within subgroups of predicted risk (Figure 3), demonstrated good model fit in the validation cohort (Hosmer-Lemeshow χ²=0.68, P=0.95).

Discussion Despite recent reports demonstrating high rates of DES utilization in patient populations and lesion subsets that have been excluded from most clinical trials, as well as higher complication rates in these “off-label” applications,2-4 we found that the incidence of TLR at 1 year remains <5% among unselected patients receiving DES in contemporary US practice. In our study, 6 clinical and angiographic characteristics were independently associated with TLR in DES recipients. Moreover, we found that a simple integer risk score based on these 6 factors was a valid predictor of TLR in the overall PCI population. This risk score was most useful for identifying individuals at modestly increased risk (6% to
7%) and very low risk (≈2%) of requiring clinically driven TLR after DES placement. Thus, despite the low rate of TLR in the general PCI population, our risk score may be a useful tool to help clinicians and patients select the optimal treatment strategy when considering the need for PCI.

### Comparison With Previous Studies

Several of the characteristics associated with TLR in our study have been reported previously, including longer stent length,9 smaller reference vessel diameter, 14 and prior PCI.8–10 However, several key predictors—Younger patient age, performance of unprotected left main PCI, and SVG location—have not been reported consistently in prior models. One single-center analysis of bare metal stents by Spertus et al.8 identified age ≈55 years as a predictor of restenosis, but...
several more recent DES studies have not reported this finding.\textsuperscript{10,11} Left main PCI was much less likely to be undertaken before widespread acceptance of DES and therefore was unlikely to have been adequately represented in prior observational registries to be identified as a TLR risk factor.

The strong association between SVG lesion location and clinical restenosis is an important finding because previous studies have resulted in conflicting conclusions about the incremental benefit of DES over bare metal stents in vein grafts.\textsuperscript{15,16} Although several small randomized trials have demonstrated reductions in angiographic restenosis with DES in SVGs,\textsuperscript{17,18} at least 1 trial has demonstrated that this benefit is attenuated substantially with extended follow-up.\textsuperscript{19} Whether this reflects a difference in the underlying pathological mechanism of restenosis in SVGs or is driven by progressive disease in untreated segments of the graft during early follow-up is uncertain. Regardless of the underlying mechanism, the present study has important ramifications for the clinical management of SVG stenoses in which the risk:benefit ratio may not favor DES as consistently as for stenoses in other vessels.

Of note, the treatment of bifurcation stenosis was not associated with increased TLR in our study—a finding that is in contrast to several previous trials that have incorporated routine angiographic follow-up.\textsuperscript{20,21} Another notable finding in our study is the absence of diabetes as a predictor of TLR—a finding that is in striking contrast to the consistent association between diabetes and both angiographic and clinical restenosis after bare metal stent implantation\textsuperscript{9,22–25} and with angiographic restenosis in early randomized clinical trials of DES.\textsuperscript{26,27} There are several possible explanations for this finding. By suppressing neointimal proliferation to a great extent, DES have substantially reduced differences in angiographic restenosis between diabetic and nondiabetic patients.\textsuperscript{28,29} Another explanation could be the lack of classic anginal symptoms exhibited by many patients with diabetes, leading to lower rates of clinically driven TLR despite underlying differences in angiographic restenosis. Finally, given the relatively low rates of TLR observed in the present analysis, we may have lacked statistical power to detect small differences in rates of TLR between diabetic and nondiabetic patients despite the large sample size. Of note, several other single center and multicenter registries have failed to identify diabetes as a predictor of clinically driven TLR among DES recipients.\textsuperscript{30,31}

### Clinical Implications and Applications

By using real-world registry data from multiple hospitals across the United States, the rates and predictors of TLR identified in EVENT probably reflect clinical restenosis much more closely than data derived from either highly selected patients enrolled in randomized controlled trials or in

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**Table 3. Final TLR Predictor Model and Risk Score**

<table>
<thead>
<tr>
<th>Variable</th>
<th>OR</th>
<th>95% CI</th>
<th>( \beta )-Coefficient</th>
<th>( P ) Value</th>
<th>Point Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient age &lt;60 y</td>
<td>1.49</td>
<td>1.14–1.95</td>
<td>0.401</td>
<td>0.0035</td>
<td>1</td>
</tr>
<tr>
<td>Prior PCI</td>
<td>1.83</td>
<td>1.40–2.39</td>
<td>0.604</td>
<td>(&lt;0.0001)</td>
<td>2</td>
</tr>
<tr>
<td>Left main PCI</td>
<td>3.14</td>
<td>1.30–7.57</td>
<td>1.144</td>
<td>0.0109</td>
<td>3</td>
</tr>
<tr>
<td>SVG location</td>
<td>2.40</td>
<td>1.62–3.57</td>
<td>0.876</td>
<td>(&lt;0.0001)</td>
<td>2</td>
</tr>
<tr>
<td>Minimum stent diameter ≤2.5 mm</td>
<td>1.54</td>
<td>1.17–2.02</td>
<td>0.430</td>
<td>0.0018</td>
<td>1</td>
</tr>
<tr>
<td>Total stent length ≥40 mm</td>
<td>1.78</td>
<td>1.35–2.35</td>
<td>0.577</td>
<td>(&lt;0.0001)</td>
<td>1</td>
</tr>
</tbody>
</table>

Model c-statistic=0.672; discrimination slope=0.01656.

SVG indicates saphenous vein graft.

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**Figure 2.** A, Distribution of patients, and B, TLR frequencies by TLR risk score in the validation cohort.

**Table 4. Predicted and Observed Rates of TLR According to Risk Score in the Validation Cohort**

<table>
<thead>
<tr>
<th>Points</th>
<th>Predicted TLR Rate, %</th>
<th>95% CI</th>
<th>Observed TLR Rate, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>2.3</td>
<td>2.2–2.4</td>
<td>2.2</td>
</tr>
<tr>
<td>1</td>
<td>3.5</td>
<td>3.4–3.6</td>
<td>3.3</td>
</tr>
<tr>
<td>2</td>
<td>4.4</td>
<td>4.3–4.5</td>
<td>4.3</td>
</tr>
<tr>
<td>3</td>
<td>4.9</td>
<td>4.8–5.1</td>
<td>5.5</td>
</tr>
<tr>
<td>4</td>
<td>6.3</td>
<td>6.1–6.6</td>
<td>6.7</td>
</tr>
<tr>
<td>5–10</td>
<td>9.7</td>
<td>9.0–10.4</td>
<td>7.5</td>
</tr>
</tbody>
</table>

Predicted TLR rate (and 95% CI) was calculated using \( \beta \)-coefficients from the final TLR risk model; observed TLR rate was the directly-measured value (for each patient subgroup according to point level) in the validation cohort.
As a result, patients with relatively high baseline clinical and angiographic characteristics were predictors of TLR in patients receiving drug-eluting stents (DES). We found that 6 easily obtainable clinical and angiographic variables were predictors of TLR after DES implantation with the potential to improve risk assessment using contemporary stents or systemic therapies designed to further reduce restenosis may be particularly appealing for patients with multivessel coronary disease. First, the finding that overall TLR rates were <5% is reassuring, particularly with regard to the effectiveness of DES in clinical practice involving a high prevalence of “off-label” applications. In addition, future generations of stents or systemic therapies designed to further reduce restenosis may be compared against estimates of clinically driven TLR in the first-generation DES evaluated in our analysis, and, based on our validated risk score, individuals at high risk of TLR could be targeted for enrollment in clinical trials of these therapies. Finally, at the patient level, our risk model could be used to provide real-time, patient-specific estimates of the probability of TLR after DES implantation with the potential to improve patient-centered decision-making at the time of PCI. This approach recently has been pilot-tested at several hospitals including our own, whereby specialized software is used to develop a customized informed consent document for each patient that describes individualized risks of restenosis, bleeding, and mortality based on that specific patient’s clinical characteristics.

As a result, patients with relatively high TLR risk could be targeted for closer outpatient follow-up after DES placement or potentially for medical management in appropriate subjects. Such an application could be particularly appealing for patients with multivessel coronary disease such as those studied in the Synergy between PCI with Taxus and Cardiac Surgery (SYNTAX) trial, as patients with lower TLR risk could be considered for multivessel PCI whereas higher-risk individuals might be better served with bypass surgery.

Our study has several potential applications relevant to the treatment of patients with obstructive coronary disease. First, the finding that overall TLR rates were <5% is reassuring, particularly with regard to the effectiveness of DES in clinical practice involving a high prevalence of “off-label” applications. In addition, future generations of stents or systemic therapies designed to further reduce restenosis may be compared against estimates of clinically driven TLR in the first-generation DES evaluated in our analysis, and, based on our validated risk score, individuals at high risk of TLR could be targeted for enrollment in clinical trials of these therapies. Finally, at the patient level, our risk model could be used to provide real-time, patient-specific estimates of the probability of TLR after DES implantation with the potential to improve patient-centered decision-making at the time of PCI. This approach recently has been pilot-tested at several hospitals including our own, whereby specialized software is used to develop a customized informed consent document for each patient that describes individualized risks of restenosis, bleeding, and mortality based on that specific patient’s clinical characteristics.

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Study Limitations
The results of our study should be considered in the context of several important limitations. First, many of the covariates in our risk models were assessed by the investigators themselves, rather than at a centralized core laboratory. By introducing additional variability into these covariates (especially the angiographic factors), it is likely that we have somewhat underestimated the true discriminative ability of our model. On the other hand, site-reported data have the advantage of being directly generalizable to the types of patients, physicians, and practice environments represented in our study population. In addition, the use of TLR as an end point (and late TLR in particular) is an approximation of clinical restenosis, but TLR does not account for undetected restenosis or restenotic lesions treated medically after being diagnosed at repeat diagnostic catheterization. The duration of follow-up may also affect this study’s interpretability because very late TLR beyond 1 year was not assessed in EVENT. Finally, the relatively modest c-statistic of our multivariable model (0.672) indicates that TLR prediction remains suboptimal using standard clinical and angiographic variables. Of note, studies of TLR before the DES era reported model c-statistics even lower than our current model (0.63 to 0.65), and similar findings have been noted in other well-accepted risk models.

Conclusion
In this large-scale, multicenter study of unselected DES patients, we found that the incidence of clinically driven TLR remains low (<5%) despite their frequent use in “off-label” settings and multivessel/multivessel procedures. Moreover, we found that 6 easily obtainable clinical and angiographic characteristics were predictors of TLR in patients receiving DES. The integer risk score constructed and validated in this study provides important patient-centered estimation of risk for clinical restenosis in the 12 months after DES placement—particularly for those individuals at low (≤2%) and modestly elevated (>7%) risk of TLR. Further validation of this risk score in other populations may allow this tool to be incorporated into future research studies and for risk assessment algorithms designed to predict clinical restenosis in contemporary practice.

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Disclosures
Dr Stolker reports serving on the speakers’ bureau for AstraZeneca Pharmaceuticals and serving as a consultant to Novo Nordisk and Educational Testing Consultants, LLC. Dr Lindley reports serving as a consultant to Novo Nordisk. Dr Marso has served as a consultant to Novo Nordisk and Abbott Vascular, and he has received research grants from Volcano Corporation, Terumo, Amylin Pharmaceuticals, The Medicines Company, and Boston Scientific. Dr Kleiman has received research grants from Schering Plough, Boston Scientific, Cordis Corporation, and BMS-Sanofi. Dr Mauri has served as a consultant to Cordis Corporation and has received research grants to the Harvard Clinical Research Institute from Abbott Vascular, Boston Scientific, Cordis Corporation, Medtronic Vascular, Eli Lilly, Daiichi Sankyo, Bristol Myers Squibb, and Sanofi-Aventis. Dr
References


CLINICAL PERSPECTIVE

Prediction of restenosis after percutaneous coronary intervention (PCI) remains challenging because existing risk assessment algorithms were developed before widespread adoption of drug-eluting stents. Identifying patients at high risk of DES restenosis would provide clinicians with an opportunity to select therapeutic options best tailored for the individual patient (eg, choosing medical therapy or bypass surgery versus PCI, number of lesions to revascularize, and type of stent to implant). We therefore used a large multicenter registry of contemporary PCI to identify predictors of clinically driven target lesion revascularization (TLR) among 8829 drug-eluting stents patients treated in “real-world” clinical practice between 2004 and 2007. We then constructed a risk score for TLR and tested the model for predictive accuracy in a separate population. At 1-year follow-up, TLR occurred in 4.2% of patients. Using multiple logistic regression, we identified 6 predictors of TLR at 1 year: age <60, prior PCI, unprotected left main PCI, saphenous vein graft PCI, minimum stent diameter ≤2.5 mm, and total stent length ≤40 mm. There was more than a 3-fold difference in TLR rates between the lowest risk category (score=0; TLR rate, 2.2%) and the highest risk category (score ≥5; TLR rate, 7.5%). In conclusion, the overall incidence of clinically driven TLR remains low (<5%) among unselected patients receiving drug-eluting stents in routine clinical practice. A simple risk model incorporating 6 readily available clinical and angiographic variables helps provide important patient-centered estimation of risk for clinical restenosis in the 12 months after drug-eluting stents placement—particularly for those individuals at low (≈2%) and modestly elevated (≥7%) risk of TLR.
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