A Model for Predicting Mortality in Acute ST-Segment Elevation Myocardial Infarction Treated With Primary Percutaneous Coronary Intervention

Results From the Assessment of Pexelizumab in Acute Myocardial Infarction Trial

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Background—Accurate models to predict mortality are needed for risk stratification in patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI).

Methods and Results—We examined 5745 patients with STEMI undergoing primary PCI in the Assessment of Pexelizumab in Acute Myocardial Infarction Trial within 6 hours of symptom onset. A Cox proportional hazards model incorporating regression splines to accommodate nonlinearity in the log hazard ratio (HR) scale was used to determine baseline independent predictors of 90-day mortality. At 90 days, 271 (4.7%) of 5745 patients died. Independent correlates of 90-day mortality were (in descending order of statistical significance) age (HR, 2.03/10-y increments; 95% CI, 1.80 to 2.29), systolic blood pressure (HR, 0.86/10-mm Hg increments; 95% CI, 0.82 to 0.90), Killip class (class 3 or 4 versus 1 or 2) (HR, 4.24; 95% CI, 2.97 to 6.08), heart rate (>70 beats per minute) (HR, 1.45/10-beat increments; 95% CI, 1.31 to 1.59), creatinine (HR, 1.23/10-μmol/L increments >90 μmol/L; 95% CI, 1.13 to 1.34), sum of ST-segment deviations (HR, 1.25/10-mm increments; 95% CI, 1.11 to 1.40), and anterior STEMI location (HR, 1.47; 95% CI, 1.12 to 1.93) (c-index, 0.82). Internal validation with bootstrapping confirmed minimal overoptimism (c-index, 0.81).

Conclusions—Our study provides a practical method to assess intermediate-term prognosis of patients with STEMI undergoing primary PCI, using baseline clinical and ECG variables. This model identifies key factors affecting prognosis and enables quantitative risk stratification that may be helpful in guiding clinical care and for risk adjustment for observational analyses.

Key Words: myocardial infarction ■ mortality ■ risk factors
Methods
APEX AMI Trial and Study Population
Details of the APEX AMI trial have been published previously. Briefly, 5745 patients from 17 countries and 296 sites were enrolled in the trial between July 13, 2004, and May 11, 2006. Patients were eligible if they were aged ≥18 years and presented within 6 hours of symptom onset with high-risk STEMI defined as ECG evidence of at least 2-mm ST elevation in 2 anterior leads or 2-mm elevation in 2 inferior leads coupled with ST depression in 2 contiguous leads for a total of 8 mm or more or new left bundle branch block with at least 1-mm concordant ST elevation. Patients were excluded if they had inferior ST elevation alone without anterior ST depression, were pregnant, had active infection, or had received fibrinolytic therapy for the treatment of their qualifying events. Patients were random selected to receive an intravenous bolus of pexelizumab or matching placebo given in double-blinded fashion before PCI followed by an infusion of pexelizumab or placebo over the subsequent 24 hours. Concomitant medications and subsequent cardiac procedures were left to the discretion of the attending physician but were recommended to be in compliance with the acute STEMI treatment guidelines established by the American College of Cardiology, the American Heart Association, and the European Society of Cardiology.12,13
The primary end point of the APEX AMI trial (all-cause mortality through day 30) and the clinical event-adjudicated secondary end points (death and the composite incidence of death, cardiogenic shock, or congestive heart failure [HF] through days 30 and 90) were not significantly different between the pexelizumab and placebo groups. For the current analysis, we included all patients enrolled in the APEX AMI trial.

Data Collection, Definitions, and Clinical Outcomes
Electronic case report forms were used to prospectively collect all patient information, including demographics, medical history, presenting symptoms and signs, time to presentation from symptom onset, timing of randomization, presenting hemodynamic features, treatments, and in-hospital clinical events. This analysis focused on all-cause mortality measured between randomization (day 0) and 90 days postrandomization. Ninety-day follow-up was obtained on 99.96% of patients.

Statistical Analyses
Continuous variables were descriptively summarized using medians with 25th and 75th percentiles, and categorical factors were reported using percentages. The multivariable Cox proportional hazards regression model was used to identify baseline clinical factors that were associated with 90-day mortality.14 Factors examined in the modeling process are listed in Table 1 and included patient demographics (age, sex, race, body mass index), medical history and comorbid conditions (diabetes, hypertension, hyperlipidemia, atrial fibrillation, chronic inflammatory conditions, smoking status, prior coronary artery disease [CAD], prior MI, prior angina, prior PCI or coronary artery bypass graft [CABG] surgery, prior stroke or transient ischemic attack, prior HF, chronic obstructive pulmonary disease [COPD], peripheral vascular disease, chronic liver disease), family history of CAD, time from hospital arrival to randomization, time from symptom onset to randomization, time from symptom onset to hospital arrival, treatment, country of enrollment, ECG findings at baseline (MI location, total ST deviation), Killip class at enrollment, vital signs at presentation (heart rate, blood pressure), serum creatinine, estimated creatinine clearance (by Cockcroft-Gault equation), and treatment assignment.

For continuous clinical variables, we examined the shape and strength of the relation between individual variables and 90-day mortality using a flexible model-fitting approach involving restricted cubic spline functions and 5 knots. These functions were graphically and statistically examined to assess the assumption of this regression model that patient characteristics were linearly related to the logarithm of the hazard ratio (HR). When the test of nonlinearity from the spline transformation was statistically significant, the plot of the probability of outcome versus the variable of interest from the spline model provided a visual impression of a potential appropriate transformation. For the variables with statistically significant nonlinear relationships to outcome, it appeared that 2 slopes existed. A grid search of knot points around the visually determined inflection point identified the best fit, assuming a linear spline. The −2-log likelihood ratio test was compared for the model, assuming restricted cubic splines to that with linear splines. When the model test result with linear splines was not similar to that with restricted cubic splines, a restricted cubic spline transformation was applied during the modeling process. We also examined whether the prognostic model would change if the variable was included for particular levels of other important descriptors (ie, we tested for interactions among the prognostic clinical variables).

We performed variable selection with the Cox model using stepwise, forward, and backward elimination procedures to assess which baseline factors were consistently found to be strongly predictive of 90-day mortality. A P=0.05 was used for both inclusion and retention in the model for these analyses. Bootstrapping (200 samples using the complete study sample size) was performed on the baseline factors to assess how often and in what order these independent characteristics would be selected. For each bootstrap sample, the stepwise selection was repeated. The result of this process was reviewed to identify factors that were consistently found to be strongly predictive of outcome. Factors selected >75% of the time and that also were found in the original variable selection process were included in the final model.

To estimate the degree of optimism in the model’s performance, a second process involving bootstrapping was completed. For each bootstrap sample, the variables from the final model were fitted. Next, the original data set was fitted using the bootstrap sample model coefficients, and a c-index for this fit on the original data set was calculated. This provided an estimate of how well the model for particular levels of other important descriptors (ie, we tested for interactions among the prognostic clinical variables).

Calibration of the model predictions was assessed by comparison of the average model prediction to the observed mortality rate across deciles of predicted risk. Imputation was used to address missing data. Five imputations were performed using the SAS PROC MI software function. The SAS PROC MI assumes a multivariate normal distribution; all variables were distributed normally enough for PROC MI to produce a reasonable distribution. Minimum and maximum allowable values for each variable were set according to the actual data distribution. Modifications were made to the list of variables and to the minima and maxima until PROC MI converted and could find imputed values within all given ranges. Because there were very few missing data elements, the results from single imputation, no imputation, and multiple imputations were similar. All results presented in this article are based on the first imputed data set. All analyses were performed using SAS version 8.2 software (SAS Institute; Cary, NC).

To facilitate the application of this model for estimating risk in clinical practice, a simple scoring scheme for each prognostic variable was devised based on the regression coefficients calculated for the model. The products of these coefficients and the values for each factor in the model were associated with an estimate of the likelihood of experiencing the outcome. For the nomogram, each coefficient was multiplied by a range of values for its associated variable. For example, the values of age applied...
were 30, 40, …, 90. The products of the coefficients with the values of the characteristic were transformed to be integers. The sum of the integers of each characteristic for a patient were directly associated with the sum of the products of the coefficients with the values of the characteristics. Because these products were in turn associated with the probability of the outcome, so were the sum of the scores. The Splus statistical software program was used to derive the scores and nomogram. While using this nomogram to estimate 90-day mortality for a given patient, the scores for each prognostic factor that he or she has are summed to

Table 1. Baseline Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Died Within 90 Days</th>
<th>Alive at 90 Days</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>n (%)</td>
<td>271 (4.7)</td>
<td>5461 (95.3)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Age, y</td>
<td>73 (61, 80)</td>
<td>61 (55, 70)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Female sex, %</td>
<td>35.1</td>
<td>22.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>80 (70, 91)</td>
<td>75 (65, 86)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>White race, %</td>
<td>93.7</td>
<td>94.3</td>
<td>0.6703</td>
</tr>
</tbody>
</table>

Region of enrollment

- Canada, New Zealand, Australia: 11.1, 15.8, 0.0360
- All other enrolling countries: 88.9, 84.2

Clinical history, %

- Hypertension: 62.7, 48.8, <0.0001
- Diabetes mellitus: 25.5, 15.4, <0.0001
- Current-smoker: 24.8, 44.2, <0.0001
- Hypercholesterolemia: 53.8, 49.5, 0.2264
- Family history of CAD: 13.0, 19.2, 0.0111
- Prior angina: 31.0, 23.7, 0.0060
- Prior CAD: 27.7, 15.9, <0.0001
- Prior MI: 21.6, 11.6, <0.0001
- Prior PCI: 14.4, 9.6, <0.0001
- Prior CABG surgery: 5.5, 2.0, <0.0001
- Prior congestive HF: 10.3, 3.3, <0.0001
- Prior atrial fibrillation: 10.7, 3.8, <0.0001
- Prior stroke: 8.1, 3.6, <0.0001
- COPD: 12.6, 4.5, <0.0001
- PVD: 9.2, 4.1, <0.0001
- Dialysis: 2.2, 0.2, <0.0001

Presenting characteristics

- Heart rate, bpm: 83 (70, 100), 75 (65, 86), <0.0001
- Systolic BP, mm Hg: 120 (100, 142), 133 (118, 150), <0.0001
- Diastolic BP, mm Hg: 72 (60, 85), 80 (70, 90), <0.0001
- Infarct location
  - Inferior, %: 26.0, 41.5
  - Anterior, %: 72.0, 58.5
- Renal insufficiency: 3.0, 3.9, 0.2109
- Sum of ST-segment deviation at baseline, mm*: 14.5 (10, 22), 13.0 (9, 14), <0.0001
- Killip class
  - 1: 63.5, 90.7
  - 2: 20.3, 7.9
  - 3: 7.0, 0.8
  - 4: 9.2, 0.6
- Creatinine, μmol/L: 106.1 (88.4, 131.0), 88.4 (79.6, 106.1), <0.0001
- Time to hospital arrival from symptom onset, h: 2.5 (1.4, 3.9), 2.2 (1.4, 3.3), 0.0043
- Time to randomization from symptom onset, h: 3.3 (2.1, 4.5), 2.8 (2.0, 3.9), 0.0003
- Time to randomization from hospital arrival, h: 0.5 (0.3, 1.0), 0.5 (0.2, 0.9), 0.1293

Data are presented as median (25th, 75th) or percentages. BP indicates blood pressure; bpm, beats per minute; PVD, peripheral vascular disease.

†Comparing Killip class 1 and 2 versus 3 and 4.
Table 2. Independent Correlates of 90-Day Death in Descending Order of Their \( \chi^2 \) Value With Imputation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Adjusted ( \chi^2 )</th>
<th>HR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, per increments of 10 y</td>
<td>78.98</td>
<td>1.94</td>
<td>1.69–2.22</td>
</tr>
<tr>
<td>Systolic BP, per increments of 10 mm Hg</td>
<td>62.9</td>
<td>0.85</td>
<td>0.81–0.89</td>
</tr>
<tr>
<td>Killip class 3 or 4 (vs 1 or 2)</td>
<td>47.0</td>
<td>3.85</td>
<td>2.67–5.57</td>
</tr>
<tr>
<td>Heart rate &gt;70 to &lt;110 bpm per increments of 10 bpm</td>
<td>40.0</td>
<td>1.41</td>
<td>1.27–1.55</td>
</tr>
<tr>
<td>Heart rate ≤70 bpm per increments of 10 bpm</td>
<td>0.5</td>
<td>1.03</td>
<td>0.82–1.30</td>
</tr>
<tr>
<td>Total ST deviation at baseline per increments of 10 mm</td>
<td>19.5</td>
<td>1.31</td>
<td>1.17–1.48</td>
</tr>
<tr>
<td>Creatinine &gt;90 ( \mu \text{mol/L} ) per increments of 10 ( \mu \text{mol/L} )</td>
<td>18.9</td>
<td>1.22</td>
<td>1.12–1.34</td>
</tr>
<tr>
<td>Creatinine &lt;90 ( \mu \text{mol/L} ) per increments of 10 ( \mu \text{mol/L} )</td>
<td>0.2</td>
<td>1.03</td>
<td>0.90–1.17</td>
</tr>
<tr>
<td>Anterior MI location (vs other)</td>
<td>8.1</td>
<td>1.45</td>
<td>1.10–1.93</td>
</tr>
<tr>
<td>Time from symptom onset to randomization per increments of 1 h</td>
<td>7.6</td>
<td>1.19</td>
<td>1.05–1.35</td>
</tr>
<tr>
<td>History of COPD (vs none)</td>
<td>5.3</td>
<td>1.48</td>
<td>1.02–2.17</td>
</tr>
<tr>
<td>Prior MI (vs none)</td>
<td>3.9</td>
<td>1.40</td>
<td>1.01–1.92</td>
</tr>
<tr>
<td>Current-smoker (vs current-nonsmoker)</td>
<td>2.8</td>
<td>0.78</td>
<td>0.57–1.06</td>
</tr>
<tr>
<td>History of diabetes mellitus (vs none)</td>
<td>2.5</td>
<td>1.32</td>
<td>0.98–1.77</td>
</tr>
</tbody>
</table>

C-index, 0.82. Heart rate was a linear spline with increasing risk from 0 to 70 bpm, greater increase in risk for increasing values from 70 to 110 bpm, and the same risk for all values >110. Serum creatinine was a linear variable, with increasing risk from 90 \( \mu \text{mol/L} \) and the same risk for all values <90 \( \mu \text{mol/L} \). BP indicates blood pressure; bpm, beats per minute.

Table 3. Independent Correlates of 90-Day Death in Descending Order of Their \( \chi^2 \) Value, Taking Bootstrapping Results Into Account

<table>
<thead>
<tr>
<th>Variable</th>
<th>Adjusted ( \chi^2 )</th>
<th>HR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, per increments of 10 y</td>
<td>117.1</td>
<td>2.03</td>
<td>1.80–2.29</td>
</tr>
<tr>
<td>Systolic BP, per increments of 10 mm Hg</td>
<td>50.5</td>
<td>0.86</td>
<td>0.82–0.90</td>
</tr>
<tr>
<td>Killip class 3 or 4 (vs 1 or 2)</td>
<td>49.9</td>
<td>4.24</td>
<td>2.97–6.08</td>
</tr>
<tr>
<td>Heart rate &gt;70 to &lt;110 bpm per increments of 10 bpm</td>
<td>49.1</td>
<td>1.45</td>
<td>1.31–1.59</td>
</tr>
<tr>
<td>Heart rate ≤70 bpm per increments of 10 bpm</td>
<td>0.5</td>
<td>1.04</td>
<td>0.83–1.31</td>
</tr>
<tr>
<td>Creatinine, &gt;90 ( \mu \text{mol/L} ) per increments of 10 ( \mu \text{mol/L} )</td>
<td>21.6</td>
<td>1.23</td>
<td>1.13–1.34</td>
</tr>
<tr>
<td>Creatinine, ≤90 ( \mu \text{mol/L} ) per increments of 10 ( \mu \text{mol/L} )</td>
<td>0.5</td>
<td>1.05</td>
<td>0.92–1.19</td>
</tr>
<tr>
<td>Total ST deviation at baseline per increments of 10 mm</td>
<td>13.6</td>
<td>1.25</td>
<td>1.11–1.40</td>
</tr>
<tr>
<td>Anterior MI location (vs other)</td>
<td>9.2</td>
<td>1.47</td>
<td>1.12–1.93</td>
</tr>
</tbody>
</table>

C-index, 0.81. Heart rate was a linear spline with increasing risk from 0 to 70 bpm, greater increase in risk for increasing values from 70 to 110 bpm, and the same risk for all values >110 bpm. Serum creatinine was a linear variable with increasing risk from >90 \( \mu \text{mol/L} \) and the same risk for all values <90 \( \mu \text{mol/L} \). BP indicates blood pressure; bpm, beats per minute.

Results

Patient Characteristics and In-Hospital Management

Of the 5745 patients enrolled in APEX AMI, 271 (4.7%) died within 90 days after randomization. The majority of deaths occurred in the first 30 days (30-day mortality, 3.9% [83.3% of overall deaths]). Table 1 shows the differences in baseline characteristics between the patients who died within 90 days and those who did not. On average, patients who died were >1 decade older, and a higher proportion were women. Most comorbid conditions were significantly more common among patients who died than among those who survived. Patients who died were less likely to have been active-smokers. Presenting heart rate, Killip class, and serum creatinine were significantly higher and systolic blood pressure significantly lower among patients who died than among those who survived. The location of MI was more likely to be anterior than inferior in the patients who died. The median time from symptom onset to hospital arrival and randomization was half an hour longer in the patients who died.

Multivariable Modeling Results

Twelve factors were found to be independently predictive of 90-day mortality using the variable selection procedures and imputed data (Table 2). In descending order of significance (based on their Wald \( \chi^2 \) value), these factors were older age, lower systolic blood pressure, higher Killip class, higher heart rate, more baseline total ST deviations, higher serum creatinine >90 \( \mu \text{mol/L} \), anterior location of MI, longer time from symptom onset to randomization, history of COPD, and prior MI. Current-nonsmoker and history of diabetes were associated with borderline increased risk of 90-day death. The same set of 12 variables was consistently selected regardless of the variable selection algorithm used. The major gradient of risk in the baseline heart rate was in the range of 70 to 110 beats per minute and in the range of >90 \( \mu \text{mol/L} \) for creatinine.

To assess the stability of these variables in the model selection process, 200 bootstrap samples were generated, and the significance of the 12 variables was examined in each sample. We determined a priori that to be included as a predictor in our final model, a variable must be consistently and independently significant in at least 75% of the bootstrap replications. Time from symptom onset to randomization was selected in <50% of the samples and, thus, was not included in the final model. Other variables shown in Table 2 with weaker relationships that did not meet this criterion were current-smoker, prior MI, history of COPD, and prior MI. Current-nonsmoker and history of diabetes were associated with borderline increased risk of 90-day death. The same set of 12 variables was consistently selected regardless of the variable selection algorithm used. The major gradient of risk in the baseline heart rate was in the range of 70 to 110 beats per minute and in the range of >90 \( \mu \text{mol/L} \) for creatinine.

The c-index for the final model was robust at 0.821, and internal validation with bootstrapping revealed very minimal overoptimism (c-index, 0.817). To graphically display the substantial gradient of risk in 90-day mortality reflected by 3 important predictive baseline characteristics...
(ie, age, Killip class, and heart rate), Kaplan–Meier curves for selected illustrative groupings of these variables are shown in Figures 1 to 3. Figure 4 shows the calibration (observed versus expected) of the final model, reflecting excellent agreement between the predicted and observed 90-day mortality rates across deciles of predicted risk. Figure 5 provides an algorithm and demonstrates how to estimate risk of 90-day death in a hypothetical patient.

Discussion

We found 7 variables at the time of presentation that may help to accurately discriminate levels of risk and reliably predict the likelihood of early mortality in a population of patients with STEMI and planned primary PCI. It is remarkable that even though the reperfusion method is different and more effective, many of the predictors are the same as the most important predictors (older age, lower blood pressure, higher heart rate, Killip class, anterior MI location) identified in the early 1990s in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries mortality model for patients undergoing fibrinolysis.2

In the United States, ≈400,000 patients experience from acute STEMI each year, and the number is much larger globally.20 The majority of these patients initially present to the emergency department, where timely initial triage and treatments have significantly improved survival. With respect to reperfusion therapy, timely primary PCI compared with fibrinolytic therapy has been shown to achieve greater infarct artery patency rates and higher thrombolysis in MI (TIMI) flow rates. Because this translates into better outcomes,21 including lower mortality, recurrent ischemia and infarction, stroke, congestive HF, cardiogenic shock, and the need for subsequent revascularization, it carries an American College of Cardiology/American Heart Association class IA recommendation.12 Nevertheless, a gradient of risk exists across the
spectrum of patients with STEMI that may have an impact on patient management, including the length of time in a cardiac care unit.

The APEX AMI trial, which examined the largest number of patients with STEMI undergoing primary PCI in a clinical trial to date, provides novel information that makes such risk assessment feasible at presentation before patients with STEMI undergo primary PCI. Seven variables at baseline were found to be strongly associated with increased risk of 90-day mortality. Importantly, the risk stratification model in our study was created without invoking factors other than those readily available at baseline before the primary PCI procedure was performed.

Various published studies have evaluated predictors of mortality in the primary PCI population. Addala et al. examined the pooled Primary Angioplasty in Myocardial Infarction trial data on 3252 patients who presented within 12 hours and had coronary anatomy suitable for primary PCI. They identified the following 6 factors to be independently associated with increased risk of mortality at 1 year: age, Killip class, heart rate, diabetes mellitus, and anterior STEMI or left bundle branch block (c-index, 0.78). Similarly, Halkins et al. examined 2082 patients randomly selected <12 hours from symptom onset in the Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications trial, in which patients were randomly selected after coronary angiography defined a lesion suitable for PCI, and found 7 factors that independently correlated with 1-year mortality: baseline left ventricular ejection fraction, renal insufficiency, Killip class, final TIMI flow grades, age, anemia, and 3-vessel CAD (c-index, 0.79 for 1-year mortality). Finally, De Luca et al. examined 1791 patients with STEMI after treatment with primary PCI in a single center and identified Killip class, postprocedural TIMI flow, age, ischemia time, anterior STEMI, and number of diseased vessels as important predictors of 30-day mortality (c-index, 0.90).
Significant predictor variables and similarities and differences between these studies and our study are shown in Table 4. All studies, including ours, involved selected patients with STEMI and various times of assessment, yet many of the variables found to be predictive of mortality were common among these studies. Our study, one of the few to include patients with the entire spectrum of renal dysfunction, found serum creatinine to be an important factor, similar to the Global Registry of Acute Coronary Events model.

Three studies discussed herein integrated the risk model into a risk scoring system. Although the study by Addala et al., like ours, focused on baseline features, it did not include validation. In contrast, angiographic variables were included in the other 2 studies, precluding their use before angiographic variables are available. These investigators did perform external validation of their models in separate validation sets. Additionally, our study included baseline ECG variables that included not only infarct location, but also the total of ST-segment deviation, which was not available and not used in other studies.

The c-indices to predict mortality of other published models that included angiographic variables were similar to the c-index of our model (that only included variables evident before angiography). This suggests that much of the predictive information for mortality is explained by the readily available baseline variables, with angiographic variables adding only modestly.

Limitations
A possible limitation of our modeling analysis is the fact that it is based on patients enrolled in a large clinical trial with excellent quality of care. Even though the trial had few exclusion criteria (and included patients with renal failure and shock), trial populations for whom informed consent must be obtained tend to be at lower risk than the general population. For example, our study would not have included patients unable to provide consent because of coma, confusion following cardiac arrest, or severe hemodynamic instability because of cardiogenic shock. Although we performed internal validation of our model, external validation through testing the accuracy of the model predictions in a separate independent population would be helpful. Finally, in keeping with our study objective, we included only those variables available before angiography. Thus, we are unable to provide insight into the incremental prognostic value of infarct size as

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**Table 4**

<table>
<thead>
<tr>
<th>Systolic BP Score</th>
<th>Heart Rate Score</th>
<th>Age Score</th>
<th>Creatinine Score</th>
<th>Total ST Deviation Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;=80</td>
<td>29</td>
<td>&lt;=35</td>
<td>0</td>
<td>&lt;=30</td>
</tr>
<tr>
<td>80-90</td>
<td>19</td>
<td>35-45</td>
<td>13</td>
<td>30-40</td>
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<tr>
<td>100-120</td>
<td>8</td>
<td>45-55</td>
<td>27</td>
<td>60-90</td>
</tr>
<tr>
<td>&gt;=120</td>
<td>0</td>
<td>55-65</td>
<td>40</td>
<td>90-120</td>
</tr>
<tr>
<td>70-80</td>
<td>7</td>
<td>65-75</td>
<td>53</td>
<td>120-150</td>
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<td>80-90</td>
<td>15</td>
<td>75-85</td>
<td>66</td>
<td>150-180</td>
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<td>90-100</td>
<td>23</td>
<td>&gt;85</td>
<td>73</td>
<td>180-210</td>
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<td>100-110</td>
<td>30</td>
<td>210-240</td>
<td>73</td>
<td>&gt;270</td>
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<tr>
<td>&gt;=110</td>
<td>38</td>
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<td>87</td>
<td>&gt;270 100</td>
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</table>

Add the total number of points for Clinical Evaluation from the above table:_______

**Figure 5.** APEX AMI mortality risk model nomogram. Clinical evaluation scores (A). Predicted rate of 90-day mortality by nomogram score (B). Based on the nomogram, a patient aged 75 years (score 66) with anterior STEMI (score 9), systolic blood pressure of 110 mm Hg (score 8), heart rate of 65 beats per minute (score 6), Killip class 1 (score 0), total ST-segment deviation of 25 (score 9), and serum creatinine of 95 μmol/L (score 19) would have a total score of 117 with estimated probability of 90-day death of approximately 14%. BP indicates blood pressure.
estimated by cardiac biomarkers, ECG variables such as ST resolution or other angiographic variables as pre- and post-procedure TIMI flow, number of diseased vessels, left ventricular ejection fraction, and so forth. Despite these limitations, the comprehensive collection of baseline characteristics and the overall consistency of predictive factors seen with other studies support the reliability of our findings.

Conclusions
Our study provides insight into the important independent predictors of mortality in a primary PCI population. It also offers a tool for clinicians to assess patients with STEMI treated with primary PCI with regard to intermediate-term prognosis using clinical information available at the time of presentation. This information may be helpful in guiding clinical care as well as risk adjustment when performing other observational and quality improvement studies.

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Disclosures
None.

References
Models to predict mortality using variables available before procedure are needed for risk stratification in patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI). We used the Cox proportional hazards model to determine baseline independent predictors of 90-day mortality among 5745 patients with STEMI undergoing primary PCI in the Assessment of Pexelizumab in Acute Myocardial Infarction Trial. At 90 days, 271 (4.7%) of the 5745 patients died. Baseline variables independently associated with 90-day mortality included age (hazard ratio [HR], 2.07/10-y increment; 95% CI, 1.84 to 2.33), Killip class (class 3 or 4 HR, 4.08; 95% CI 2.84 to 5.84), heart rate >70 beats per minute (HR, 1.45/10 beats per minute, 95% CI, 1.32 to 1.60), systolic blood pressure (HR, 0.86/10 mm Hg; 95% CI, 0.82 to 0.90), creatinine >90 μmol/L (HR, 1.10/10-μmol/L increment; 95% CI, 1.06 to 1.13), sum of ST-segment deviations (HR, 1.26/mm; 95% CI, 1.12 to 1.41), and anterior STEMI location (HR, 1.44; 95% CI, 1.09 to 1.89) (c-index, 0.82). Our study provides a clinically practical method to assess intermediate-term prognosis of patients with STEMI undergoing primary PCI using baseline clinical and ECG variables that may be helpful in guiding clinical care and for risk adjustment for observational analyses.
A Model for Predicting Mortality in Acute ST-Segment Elevation Myocardial Infarction Treated With Primary Percutaneous Coronary Intervention: Results From the Assessment of Pexelizumab in Acute Myocardial Infarction Trial

Amanda Stebbins, Rajendra H. Mehta, Paul W. Armstrong, Kerry L. Lee, Christian Hamm, Frans Van de Werf, Stefan James, Torsten Toftegaard-Nielsen, Ricardo Seabra-Gomes, Harvey D. White, Christopher B. Granger and for the Assessment of Pexelizumab in Acute Myocardial Infarction (APEX AMI Investigators)

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