A bedside prediction tool predicted all cause mortality 6 months after discharge for acute coronary syndrome


Clinical impact ratings GP/FP/Primary care ****** Internal medicine ******* Cardiology *******

Q In patients hospitalised for acute coronary syndrome (ACS), does a bedside prediction tool predict all cause mortality?

CONCLUSION

In patients admitted to hospital for acute coronary syndrome, a bedside prediction tool predicted all cause mortality within 6 months of discharge.

Abstract and commentary also appear in ACP Journal Club.

Commentary

Accurate prognostic information in ACS is a key factor in adapting interventions to individual patient risk. The current classification of ACS is based mainly on quantifying the extent of myocardial injury. It identifies patient groups with different prognoses and intervention needs. However, it has been proposed that more comprehensive evaluation of long term risk is needed, beyond the information provided by biochemical markers of myocardial necrosis. In the study by Eagle et al, the GRACE risk score integrated simple and readily obtainable patient characteristics, including whether the patient had received inhospital percutaneous coronary intervention, and serum markers to complement the prognostic information pertaining to the type of ACS. Other risk prediction tools are available for unstable angina, NSTEMI, and STEMI, based on data from randomized controlled trials. These scores showed predictive ability for a composite score, including new or recurrent MI, need for revascularization, and death. Besides the fact that the scores were designed for short term prognosis (up to 30 d) and that each one covered only a part of the ACS spectrum, their main problem was that patient outcome reflected the highly controlled environment of clinical trials and not the usual circumstances of care. In contrast, the GRACE risk score was derived and validated in 2 large, multinational prospective cohorts that better reflected everyday practice.

To clarify the role of the GRACE risk score as a therapeutic decision aid, it is desirable that the score’s risk estimates identify groups with different responses to interventions. For example, in patients with unstable angina or NSTEMI and higher TIMI risk scores, enoxaparin was more effective than unfractionated heparin. We still need data on the efficacy of guiding therapeutic decisions based on GRACE risk score estimates. However, the strong validity, simplicity, and greater generalisability of this tool compared with clinical trial based tools make the GRACE risk score a solid candidate to help guide ACS management.

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METHODS

Design: 2 cohort studies, 1 for derivation and 1 for validation (Global Registry of Acute Coronary Events [GRACE]).

Setting: 94 hospitals in 14 countries.

Patients: patients who were ≥18 years of age, alive at discharge, and hospitalised for presumed ACS and had ≥1 of electrocardiographic changes consistent with ACS, serial increases in serum cardiac biomarkers, or confirmed coronary artery disease. The qualifying ACS must not have been precipitated by non-cardiovascular comorbid conditions. At discharge, patients were assigned to ST segment elevation myocardial infarction (STEMI), non-ST segment elevation myocardial infarction (NSTEMI), or unstable angina categories. 15 007 patients formed the derivation cohort and 7638 patients formed the validation cohort.

Description of prediction guide: baseline characteristics, symptoms and signs at presentation, inhospital treatments and procedures, and inhospital complications were included in a stepwise Cox proportional hazards regression model to develop the prediction tool. The tool assigned point totals for each variable, and a total point score for each patient was calculated.

Outcomes: all cause mortality within 6 months of hospital discharge.

MAIN RESULTS

6 month all cause mortality rates were similar between the derivation and validation cohorts (4.8% and 4.7%, respectively). 9 multivariate predictors of death were identified in the derivation cohort that performed similarly in the validation cohort (table). The prediction tool performed well in all forms of ACS (STEMI, NSTEMI, and unstable angina); the tool performed similarly in the validation cohort (table). The prediction tool performed well in all forms of ACS (STEMI, NSTEMI, and unstable angina); the c statistic was 0.81 in the derivation cohort and 0.75 in the validation cohort.

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Multivariate predictors for all cause mortality 6 months after hospital discharge in acute coronary syndrome (ACS)*

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Derivation cohort</th>
<th>Hazard ratio (95% CI)</th>
<th>Validation cohort</th>
<th>All patients with ACS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age per 10 y increase</td>
<td>1.8 (1.6 to 1.9)</td>
<td>1.7 (1.5 to 1.9)</td>
<td>1.7 (1.6 to 1.8)</td>
<td></td>
</tr>
<tr>
<td>History of myocardial infarction</td>
<td>1.5 (1.3 to 1.8)</td>
<td>1.2 (0.9 to 1.5)</td>
<td>1.4 (1.2 to 1.6)</td>
<td></td>
</tr>
<tr>
<td>History of congestive heart failure</td>
<td>2.2 (1.8 to 2.6)</td>
<td>2.0 (1.5 to 2.7)</td>
<td>2.1 (1.8 to 2.5)</td>
<td></td>
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<tr>
<td>Pulse per 30/min increase</td>
<td>1.3 (1.2 to 1.4)</td>
<td>1.4 (1.2 to 1.7)</td>
<td>1.3 (1.2 to 1.5)</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure per 20 mm Hg decrease</td>
<td>1.1 (1.08 to 1.2)</td>
<td>1.0 (0.9 to 1.2)</td>
<td>1.1 (1.06 to 1.2)</td>
<td></td>
</tr>
<tr>
<td>Initial serum creatinine concentration per 1 mg/dl increase</td>
<td>1.2 (1.1 to 1.24)</td>
<td>1.2 (1.1 to 1.3)</td>
<td>1.2 (1.1 to 1.23)</td>
<td></td>
</tr>
<tr>
<td>Initial cardiac enzyme elevation</td>
<td>1.6 (1.4 to 1.9)</td>
<td>1.5 (1.2 to 2.0)</td>
<td>1.5 (1.3 to 1.8)</td>
<td></td>
</tr>
<tr>
<td>ST segment depression</td>
<td>1.4 (1.2 to 1.7)</td>
<td>1.6 (1.3 to 2.1)</td>
<td>1.5 (1.3 to 1.7)</td>
<td></td>
</tr>
<tr>
<td>No inhospital PCI</td>
<td>1.6 (1.2 to 2.0)</td>
<td>1.5 (1.1 to 2.1)</td>
<td>1.9 (1.3 to 1.9)</td>
<td></td>
</tr>
</tbody>
</table>

*PCI = percutaneous coronary intervention. CI defined in glossary.