ORIGINAL ARTICLE

A new simplified immediate prognostic risk score for patients with acute myocardial infarction

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Background: Immediate risk stratification of patients with myocardial infarction in the emergency department (ED) at the time of initial presentation is important for their optimal emergency treatment. Current risk scores for predicting mortality following acute myocardial infarction (AMI) are potentially flawed, having been derived from clinical trials with highly selective patient enrolment and requiring data not readily available in the ED. These scores may not accurately represent the spectrum of patients in clinical practice and may lead to inappropriate decision making.

Methods: This study cohort included 1212 consecutive patients with AMI who were admitted to the Mayo Clinic coronary care unit between 1988 and 2000. A risk score model was developed for predicting 30 day mortality using parameters available at initial hospital presentation in the ED. The model was developed on patients from the first era (training set—before 1997) and validated on patients in the second era (validation set—during or after 1997).

Results: The risk score included age, sex, systolic blood pressure, admission serum creatinine, extent of ST segment depression, QRS duration, Killip class, and infarct location. The predictive ability of the model in the validation set was strong ($c=0.78$).

Conclusion: The Mayo risk score for 30 day mortality showed excellent predictive capacity in a population based cohort of patients with a wide range of risk profiles. The present results suggest that even amidst changing patient profiles, treatment, and disease definitions, the Mayo model is useful for 30 day risk assessment following AMI.

Optimal management of acute myocardial infarction (AMI) requires rapid reperfusion strategies with a door-to-balloon time of 90 minutes or less, which is now expected for acute ST segment elevation myocardial infarction. The use of appropriate reperfusion strategies requires triage of all acute myocardial infarcts at the point of initial hospital contact—which most commonly is the emergency department. We sought to design a risk score algorithm for myocardial infarction that would be easy to implement in the emergency room with limited clinical and laboratory data.

Many risk factors and/or markers help to define prognosis in patients with AMI.^{1–4} Several simplified methods have been developed for risk stratification in this population,^{5–21} and these have been perceived as valuable.^{22–25} These methods generally use algorithms of varying degrees of complexity to summarise a patient's level of risk for mortality into a single numerical value based on multiple characteristics ostensibly obtainable at hospital presentation. However, there are limitations to the current models. These include:

- the requirement for detailed information from the patient's past medical record which may not be readily available at the time of emergency admission to physicians who initially evaluate and stabilise patients
- derivation from clinical trial databases which include highly selected patients enrolled over a short period of time
- exclusion of new or emerging prognostic variables
- a design applicable to either patients with non-ST elevation (NSTEMI) MI or STE MI, but not both.

For these reasons, attempts have been made to validate the TIMI and PURSUIT scoring systems in community based populations.^{26–30}

The impetus for this study was to develop a new model for prognosis following AMI, spanning an extended period of time in a broad unselected community based AMI population using easily obtainable variables present at the time of initial patient contact in the emergency room. We also considered recently described clinical predictors to determine if risk quantification could be improved. A longer time period was used for development to avoid the bias of a narrow timeframe, as has occurred with prior prognostic indices.

METHODS

Study sample

The Coronary Care Unit (CCU) database is a registry of all patients treated for AMI at the Mayo Clinic affiliated Saint Mary's Hospital in Rochester, MN, USA, beginning 1 January 1988. Data were collected from the medical record during initial hospitalisation. From this database, 1212 consecutive local Olmsted County, MN, patients diagnosed as having AMI between January 1988 and July 2000 were identified. Patients were not considered in our analysis if they refused to give research authorisation as required by Minnesota law, if they had less than 30 days' follow up and were not known to have died, or if their records were incomplete. The Mayo Foundation Institutional Review Board approved the study. For this study, we defined AMI according to World Health Organization criteria.³¹

Abbreviations: ACE, angiotensin converting enzyme; AMI, acute myocardial infarction; CCU, coronary care unit; GRACE, Global Registry of Acute Coronary Events; GUSTO, Global Utilization of Streptokinase and Tissue plasminogen activator to treat Occluded arteries-1; PARAGON, Platelet IIb/IIIa Antagonist for the Reduction of Acute coronary syndrome events in a Global Organization Network; PURSUIT, Platelet IIb/IIIa in Unstable angina: Receptor Suppression Using Integrilin Therapy; STE, ST elevation; TIMI, Thrombolysis In Myocardial Infarction

Statistical analysis

In developing the risk score model, we considered all the variables in our database that should be readily available at the time of initial clinical presentation. These included demographic information, physical examination findings on admission, initial electrocardiogram (ECG) findings, and medical history including medical comorbidities. To facilitate temporal validation of the risk score model, we used a data-splitting technique^{32–34} to create a training set and a validation set using 1 January 1997 as the cut-off date. The training set (before 1997) consisted of 809 patients (67%) and the validation set (during or after 1997) consisted of 403 patients (33%). This is close to the two thirds split suggested by Harrell *et al.*³⁴ The risk score model was initially developed on the training set without knowledge of information regarding the validation set. To facilitate scoring and applying the model in a clinical setting, all the variables under consideration were put into categorical form. Binary variables were unchanged. Continuous variables were initially split into three categories in an effort to find any increased gradient of risk or possible non-linear or non-monotonic associations between a continuous variable and 30 day mortality. We chose the categories after examining graphical and numerical results as described by Mazumdar and Glassman,³⁵ who describe methods for finding the optimal split of a continuous variable with respect to some outcome.

Univariate associations between each of the candidate independent variables and 30 day mortality were assessed by means of a logistic regression model and described by odds ratios and 95% confidence intervals. We used a modified version of a variable selection technique described by Collett³⁶ to select variables from the training set to include in the final risk score model. The primary criterion for model inclusion was an odds ratio of 1.5 or greater in a multivariate logistic regression model. We chose to use an odds ratio approach to select variables since variable selection based on the more common p value approach can be dictated by sample size and prevalence of certain variables and hence may miss some clinically important effects. Clinical judgment was incorporated when necessary to exclude any variables demonstrating a clinically implausible relation with 30 day mortality.²⁴ Estimated odds ratios from the final multivariate logistic regression model were transformed into integer values, and followed closely to a process described by Morrow *et al.*¹⁷: 1 point for odds ratio ≥ 1.5 and < 2.0 ; 2 points for odds ratio ≥ 2.0 and ≤ 2.5 ; and 3 points for odds ratio > 2.5 . Categories from the three group variables were pooled if adjacent categories were found to have similar associations with 30 day outcome according to the odds ratio criteria.

The ability of the risk score model developed on the training set to predict 30 day mortality in the validation set was assessed with the c-index.³⁴ A model with a c-index near 1 demonstrates excellent predictive ability, while a c-index near 0.5 demonstrates poor predictive ability. The following criteria have been suggested for evaluating the c-index in prognostic models for acute coronary syndromes³⁷: $c < 0.6$, no clinical value; $0.6 \leq c \leq 0.7$, limited value; $0.7 < c \leq 0.8$, modest value; $c > 0.8$, genuine clinical utility. The c-index was calculated from a logistic regression model with the Mayo risk score as the sole independent variable. A similar logistic regression model adjusted for in-hospital therapy was developed to account for differential treatment across risk levels as well as potential changes in treatment over time.

RESULTS

Table 1 displays the demographics, admission examination characteristics, ECG characteristics, comorbidities, and medical history of the entire cohort. The 30 day mortality was 11.1%. Table 2 shows the univariate associations between

Table 1 Baseline characteristics of the entire study cohort (n = 1212)

Age (years)*	70.7 (57.9–80.4)
Women (%)	40
Examination characteristics	
BMI (kg/m ²)*	26.9 (24.2–30.5)
Height (cm)*	170 (160–177)
Weight (kg)*	77 (66–90)
Heart rate (bpm)*	80 (68–96)
SBP (mm Hg)*	142 (120–166)
DBP (mm Hg)*	84 (70–99)
Killip class† > I (%)	21
CHF (%)	23
Anterior MI (%)	34
Creatinine (mg/dl)*	1.2 (1.0–1.4)
ECG characteristics	
ST elevation or LBBB (%)	50
ST depression (%)	
< 1 μ V	59
1–2 μ V	25
> 2 μ V	16
QRS duration (%)	
< 100 ms	53
100–120 ms	31
> 120 ms	16
T wave inversion (%)	
< 1 μ V	47
1–3 μ V	35
> 3 μ V	18
RBBB (%)	6
Comorbidities	
Renal disease (%)	3
Family history of CAD (%)	21
Diabetes (%)	18
Hyperlipidaemia (%)	50
Hypertension (%)	46
Smoking history (%)	59
PVD (%)	11
Medical history	
Stroke (%)	16
Angina (%)	25
MI (%)	21

*Values are mean (range).

†Killip classes: class I, no evidence of CHF; class II, rales $< 50\%$ of lung fields and/or S-3 gallop; class III, rales $> 50\%$ of lung fields; class IV, cardiogenic shock.

BMI, body mass index; bpm, beats per minute; CAD, coronary artery disease; CHF, congestive heart failure; DBP, diastolic blood pressure; ECG, electrocardiogram; LBBB, left bundle branch block; MI, myocardial infarction; PVD, peripheral vascular disease; RBBB, right bundle branch block; SBP, systolic blood pressure.

each of the variables and 30 day mortality in the training set of patients. The variables included in the final risk score model with corresponding odds ratios, 95% confidence intervals, and risk score point assignments are shown in table 3.

The final model thus has a maximum score of 18 although values greater than 10 were not common. Although inclusion into the final model was not based on p value criteria, all variables in the model were significant at the 0.05 level with the exception of ST depression 1–2 μ V ($p = 0.109$) and anterior location ($p = 0.052$). A post hoc analysis of the risk score factors revealed little change ($\leq 20\%$) in the odds ratio estimates from the training sample to the entire sample, with the exception of anterior location. The odds ratio estimate for anterior location changed from 1.64 in the training set to 1.03 in the overall set, a 37% decrease.

Table 4 shows the distribution of risk score factors between the training and validation sets, essentially examining the differences in risk score characteristics in the era before (training set) and after (validation set) 1 January 1997. Despite differences in many risk score factors across the different sets, the distribution of risk scores did not significantly differ (validation: mean (SD) 7 (3.4) v training:

Table 2 Univariate association with 30 day mortality (training set)

Demographics (n = 1212)	OR (95% CI)
Age (v <70 years)	
70–80	2.47 (1.37 to 4.44)
>80	4.50 (2.60 to 7.80)
Sex (female v male)	2.96 (1.87 to 4.66)
Examination characteristics	
BMI (v <25 kg/m ²)	
25–30	0.65 (0.39 to 1.08)
>30	0.71 (0.40 to 1.26)
Height (v <160 cm)	
160–170	0.68 (0.39 to 1.18)
>170	0.30 (0.17 to 0.54)
Weight (v <70 kg)	
70–80	0.54 (0.30 to 0.96)
>80	0.41 (0.25 to 0.69)
Heart rate (v <70 bpm)	
70–90	1.54 (0.81 to 2.90)
>90	2.80 (1.50 to 5.22)
SBP (v <110 mm Hg)	
110–140	0.70 (0.40 to 1.25)
>140	0.30 (0.16 to 0.54)
DBP (v <90 mm Hg)	
90–110	0.61 (0.36 to 1.03)
>110	0.47 (0.20 to 1.12)
Killip class* (>I v I)	4.65 (2.93 to 7.36)
CHF	4.30 (2.73 to 6.80)
Anterior MI	1.46 (0.93 to 2.28)
Cr at admission (v <1.2 mg/dL)	
1.2–1.4	1.60 (0.91 to 2.83)
>1.4	3.91 (2.26 to 6.77)
ECG characteristics	
ST elevation or LBBB	1.32 (0.85 to 2.08)
ST depression (v <1 µV)	
1–2	1.16 (0.67 to 2.03)
>2	2.43 (1.43 to 4.12)
QRS duration (v <100 ms)	
100–120	1.48 (0.89 to 2.47)
>120	2.50 (1.40 to 4.46)
T wave inversion (v <1 µV)	
1–3	1.26 (0.76 to 2.10)
>3	1.50 (0.85 to 2.65)
RBBB	1.62 (0.73 to 3.56)
Comorbidities	
Renal disease	2.04 (0.81 to 5.11)
Family history of CAD	0.70 (0.37 to 1.32)
Diabetes	1.48 (0.84 to 2.58)
Hyperlipidaemia	0.83 (0.53 to 1.30)
Hypertension	1.93 (1.23 to 3.02)
Smoking history	0.56 (0.36 to 0.87)
PVD	1.92 (1.08 to 3.42)
Medical history	
Stroke	1.75 (1.01 to 3.04)
Angina	1.31 (0.82 to 2.10)
MI	1.61 (0.98 to 2.63)

*Killip classes: class I, no evidence of CHF; class II, rales <50% of lung fields and/or S-3 gallop; class III, rales >50% of lung fields; class IV, cardiogenic shock.
 BMI, body mass index; bpm, beats per minute; CAD, coronary artery disease; Cr, creatinine; DBP, diastolic blood pressure; ECG, electrocardiogram; MI, myocardial infarction; LBBB, left bundle branch block; PVD, peripheral vascular disease; OR, odds ratio; RBBB, right bundle branch block; SBP, systolic blood pressure.

Table 3 Final risk score model

Factor	OR (95% CI)	Points
Age		
>80 years	2.02 (1.19 to 3.44)	2
Sex		
Female	2.62 (1.56 to 4.39)	3
SBP		
≤140 mm Hg	2.75 (1.64 to 4.61)	3
Creatinine		
>1.4 mg/dl	1.89 (1.10 to 3.25)	1
ST depression		
1–2 µV	1.66 (0.90 to 3.09)	1
>2 µV	3.19 (1.75 to 5.83)	3
QRS duration		
≥100 ms	1.70 (1.02 to 2.83)	1
Killip class		
>I	3.25 (1.93 to 5.47)	3
MI location		
Anterior	1.64 (1.00 to 2.72)	1

MI, myocardial infarction; OR, odds ratio; SBP, systolic blood pressure.

Table 4 Distribution of risk score characteristics

Factor (%)	Training set	Validation set	p value
Age			
>80 years	23	34	<0.01
Sex			
Female	38	45	0.02
SBP			
≤140 mm Hg	48	47	0.61
Creatinine			
>1.4 mg/dL	20	24	0.13
ST depression			
1–2 µV	26	22	0.02
>2 µV	18	13	
QRS duration			
≥100 ms	48	47	0.92
Killip class			
>I	21	23	0.36
MI location			
Anterior	37	27	<0.01

MI, myocardial infarction; SBP, systolic blood pressure.

Figure 1 Distribution of risk scores between training and validation sets.

5.5 (3.2); $p = 0.20$, fig 1). There was also no difference in 30 day mortality by period (fig 2).

There were differences in treatment across the two eras. Patients in the latter era were more likely to receive angiotensin converting enzyme (ACE) inhibitors (28% validation set v 11% training set), aspirin (95% v 86%, respectively), β blockers (80% v 65%, respectively), and lipid lowering drugs (19% v 4%, respectively) during the initial day of hospitalisation (all $p < 0.001$). Reperfusion as primary treatment was less common among latter-era patients (32% v 43%, respectively, $p < 0.001$) reflecting an increased prevalence of NSTEMI admitted to our CCU during the second

time period, whereas use of stents was much commoner in these patients (39% v 5%, $p < 0.001$, respectively) reflecting the technological advances that have driven clinical practice.

Figure 3 shows 30 day mortality rates across risk scores, demonstrating the increased gradient of risk for 30 day

Figure 2 30 day mortality between training and validation sets.

mortality as the risk score increases. The strong association is also evident from the odds ratio estimates per unit increase in risk score for both the training (OR 1.46, 95% CI 1.34 to 1.58) and validation (1.37, 1.24 to 1.53) sets. The prognostic ability of the risk score was weakened little in the validation set ($c = 0.78$) from the training set ($c = 0.81$). This degree of attenuation found in the validation set was similar to that found by Morrow *et al* (0.78 to 0.75) with the TIMI risk score.¹⁷ When we tested the altered risk score algorithm as suggested by the entire cohort (removing anterior location from the risk score calculation) on the validation set, the results were similar to the initially developed model ($c = 0.79$).

The risk score model demonstrated improved prognostic capability over time in the latter era. The c -index by year for the years 1997 through 2000 was 0.74, 0.73, 0.82, and 0.89, respectively. The odds ratios for the effect of a 1 unit increase in risk score ranged from 1.28 (1.09, 1.50) in 1998 to 1.77 (1.15 to 2.72) in 2000. The c -index by year in the training set ranged from 0.73 to 0.92, with odds ratios ranging from 1.28 (1.03 to 1.60) to 2.04 (1.36 to 3.06), not showing an obvious temporal trend in either direction.

Table 5 displays the change in therapy across the spectrum of risk scores. Those with higher risk scores were significantly less likely ($p < 0.001$) to receive all reported medications within 24 hours of admission with the exception of ACE inhibitors. They were also less likely to receive reperfusion as their primary treatment, any angioplasty during their admission, or a stent, than those in the lower risk score categories (all $p < 0.05$). The risk score remained strongly associated with 30 day mortality in both sets of patients even after adjusting for some of the more clinically significant treatment variables (angioplasty, stent, and aspirin and β blocker use) (training set OR 1.27, 1.14 to 1.41, $p < 0.001$; validation set OR 1.37 1.19 to 1.57).

DISCUSSION

Our data have validated a new and unique prognostic tool for predicting 30 day mortality in a population of consecutive unselected patients with AMI. This study is based on a community sample without the obligatory exclusions associated with clinical trials. We used clinical data readily available upon arrival in the emergency department, and patients were included over a 12 year period. This approach obviated many of the weaknesses associated with prior studies on AMI risk scores. In keeping with the use of unselected patients, our patient cohort was sicker (as evinced by increased heart rates and more advanced Killip class), older, and more apt to be female than highly selected patient samples gleaned from clinical trials that have heretofore been used to develop risk scores (data not shown). Thirty day mortality was also higher in our cohort (11.1% *v* 3.6–7.0%). We also had the advantage of utilising a patient population presenting over a 12 year period of time rather than over the

Figure 3 Thirty day mortality by risk score group in the training and validation sets.

typical 6–24 month period of most randomised clinical trials. We temporally validated the scoring system over several years, allowing assessment of the system's performance amidst changing patient profiles, treatment strategies, and disease definitions. Our approach incorporated the important prognostic variables identified by previous risk assessment studies^{17–20} as well as new or emerging risk markers such as ST segment deviation, elevated biomarkers and renal insufficiency. Finally, our model can be applied easily to all AMI populations.

We applied rigorous statistical validation methodology by temporally dividing our AMI cohort into two groups. We then identified the prognostic variables in the initial cohort or test set, and validated the prognostic associations in a subsequent cohort set, or validation set. We included in our cohort only local patients from Olmsted County, MN, who were admitted to our CCU since 1988. We excluded the large number of patients admitted to our CCU after transfer from regional and/or national referral networks to avoid “referral bias”. In addition, since the patients in our CCU database cover a time period of over 12 years, the prognostic ability of the risk score could be assessed over multiple treatment strategies and disease definitions. Changes in treatment and disease will most certainly continue, so prognostic tools must be robust enough to withstand such changes. To our knowledge, our risk assessment tool is the first to be examined and tested over such an extended period of time.

The risk score algorithm described here was developed in a manner similar to instruments for predicting 30 day mortality previously reported in the literature.^{17–20} Thus, many of the same variables were found to be significant in our model. Age, systolic blood pressure, and indicators of heart failure (Killip class >1 or presence of rales) are common to all the risk scores (table 6). The PURSUIT and GUSTO-1 models displayed increasing risk per decade of age, whereas the TIMI model grouped age into <65 , 65–74, and >75 . Our initial grouping of age into <70 , 70–80, and >80 showed little difference between the <70 and 70–80 year old groups. Not surprisingly, our unselected CCU population presented much older patients (median age = 71) than those in any of those trials (medians = 62–64), perhaps partially explaining this finding. Our final grouping of systolic blood pressure into ≤ 140 versus >140 is also a much different threshold than suggested by previous literature. The TIMI model used 100 mm Hg as the cut-point, while the PURSUIT model used two thresholds, one at 100 and one at 120. Our suggested threshold is close to the median systolic blood pressure seen in our patients (CCU median = 142), where the 100 and 120 thresholds are much lower than the medians seen in three of the reported trials (median = 140, 130, 130). Our grouping of Killip class (>1 *v* 1) was identical to two of the other risk score algorithms.

Table 5 Medications and procedures by risk score categories

	0-1 (n = 148)	2-3 (n = 181)	4-5 (n = 302)	6-7 (n = 255)	8-9 (n = 169)	10+ (n = 157)	p value
Medications within 24 hours							
ACE inhibitors	11	11	18	14	25	21	<0.001
Aspirin	94	93	91	88	85	80	<0.001
β Blockers	82	78	74	73	54	56	<0.001
Heparin	98	94	93	86	89	81	<0.001
Lipid lowering	17	10	10	7	8	3	<0.001
Nitrates	54	52	46	45	44	41	0.006
Primary treatment							
Lytic	15	20	21	16	9	11	<0.001
PTCA	24	22	27	20	27	11	
CABG	1	1	0	1	0	1	
Medications	60	57	52	63	64	77	
Reperfusion	40	43	48	37	36	23	<0.001
Other	60	57	52	63	64	77	
Other treatment							
CABG	13	9	8	9	7	6	0.056
PTCA	56	51	46	41	37	22	<0.001
Stent	28	15	19	16	13	8	<0.001

ACE, angiotensin-converting enzyme; CABG, coronary artery bypass graft; PTCA, percutaneous transluminal coronary angioplasty.

Our model showed a significantly detrimental association between female sex and 30 day mortality, a finding not displayed by any of the other models, yet previously reported by us and others.³⁸⁻⁴⁰ A recent paper by MacIntyre *et al* suggests that the increased risk for women was most prominent among younger AMI patients, but weakened among older patients.³⁸ Our data support this finding.

The ECG parameters assessed here were only included in two of the four trials reported previously. The PURSUIT investigators found ST depression prognostic; our model suggests an increased gradient of risk as ST depression increases. This is in keeping with the recent PARAGON results which showed a similar pattern.⁴¹ The second GUSTO-1 model also reported our finding of a prolonged QRS duration as prognostic. Two recent reports corroborate this finding.⁴²⁻⁴³

Heart rate was highly associated with 30 day mortality in all 4 clinical trial models but was not found independently associated with 30 day mortality in our population. Conversely, creatinine level was found prognostic in our patients, but there was no evidence of its consideration in the trial populations. This may be due to the exclusion of patients with renal failure in most clinical trials. Anterior location was also found significant in both GUSTO models; our post hoc

analysis of the entire cohort suggests this variable may not be associated with 30 day mortality.

A recently derived risk scoring system has been published from the GRACE registry.⁴⁴ The GRACE score is an elegant and comprehensive scoring system that assigns various point amounts to various clinical strata and was derived from patients admitted at 94 different hospitals across 18 countries. Our scoring system has similarities, including being derived from a non-selected population. The GRACE score omitted patients who died within the initial 24 hours of hospitalisation whereas our method did not. The GRACE score omitted low risk patients but our score did not. Our score examined several ECG variables known to be prognostic including QRS duration and degree of ST segment depression. Finally, the GRACE score predicts inhospital mortality whereas our data predict 30 day mortality. The GRACE score is an excellent addition to the risk score literature and extends what is known from the GUSTO and TIMI scoring systems. We believe our score builds upon the knowledge from the GRACE score and represents a simpler, easy to use prognostic score that can readily be determined in the emergency department setting when evaluating patients with AMI.

Our risk score model was additionally validated in a set of patients not used in model development, showing excellent predictive ability in those patients admitted to our CCU in 1997 and beyond. The risk score showed increasing predictive power over time during this period, with an overall c-index approaching 0.8. In a recent paper describing prognostic models in patients with acute coronary syndromes, Ohman *et al*³⁷ describe a c-index of “greater than 0.8 [as having] discrimination adequate for genuine clinical utility”. Our model demonstrates a predictive capacity near this threshold in a validation set, which makes it a promising tool for prognosis in AMI patients. Some may raise concerns about creating a scoring system in one time period and testing it in a second time period, even though this is the statistically recommended methodology.³²⁻³⁴ We did not observe any differences in overall mortality between the training and testing sets, nor did we see any statistically significant differences in mortality by risk score between the training and testing sets. Thus, we believe our testing and training sets are internally consistent and are not biased by treatment period differences.

Table 6 Comparison between TIMI, GRACE, and Mayo risk scores

	Mayo score	GRACE score	TIMI score*	GUSTO-1 predictors
Clinical presentation	No	Yes†	Yes	No
Sex	Yes	No	No	Yes
Age	Yes	Yes	Yes	Yes
Systolic blood pressure	Yes	Yes	No	Yes
Heart rate	No	Yes	No	Yes
Cardiovascular risk factors	No	No	Yes	No
Killip class	Yes	Yes	No	Yes
Renal function	Yes	Yes	No	No
ST deviation	Yes	Yes	Yes	No
QRS duration	Yes	No	No	No
Anterior MI location	Yes	No	No	Yes
Cardiac enzyme abnormality	No	Yes	Yes	No

*TIMI score for non-ST elevation myocardial infarction/UA.

†Cardiac arrest.

Our odds ratio estimates from logistic regression models are further evidence of the strong relation between the risk score and 30 day mortality. Although patients of higher risk are treated less aggressively than their lower risk counterparts with newer and more potent agents, the relation between the risk score and 30 day mortality in the validation set remained strong even after adjusting for these clinically important treatment variables.

The initial proposed risk score model was based on the first two thirds of the patients seen at the CCU since 1988. Our results suggest that one of the factors found significant in the training set (anterior location) may not be significant when considering the entire cohort. It is likely that the observed diminution of anterior MI location as a prognostic marker is a reflection of our using a cohort combining STE AMI and NSTEMI AMI. The declining prevalence of STE AMI in the testing set weakens the overall association of anterior MI location but not the clinical validity of this marker, and we elected to keep anterior MI location in our final model. Anterior location was also found to be significantly associated with 30 day mortality in the GUSTO-I trial, supporting this observation in our data.

Some factors recently shown to be associated with 30 day mortality could not be considered in our analysis because of the unavailability of appropriate data. These include the newer biomarkers,⁴⁵ ECG parameters up to three hours after hospital presentation,⁴⁶ socioeconomic status,⁴⁷ and parameters based on data after initial evaluation.^{48–50} Finally, the Olmsted County population is predominantly Caucasian, and our analysis may have weaknesses when applied to other ethnic groups.

SUMMARY

Our risk score instrument for 30 day mortality has excellent predictive capacity in a cohort of patients with a wide range of risk profiles over an extended period of time. Our results suggest that even amidst changing patient profiles, treatment strategies, and disease definitions, the Mayo risk score model is a promising tool for prognosis of 30 day mortality among AMI patients.

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