Hospital Discharge Risk Score System for the Assessment of Clinical Outcomes in Patients With Acute Myocardial Infarction (Korea Acute Myocardial Infarction Registry [KAMIR] Score)

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Assessment of risk at time of discharge could be a useful tool for guiding postdischarge management. The aim of this study was to develop a novel and simple assessment tool for better hospital discharge risk stratification. The study included 3,997 hospital-discharged patients with acute myocardial infarction who were enrolled in the nationwide prospective Korea Acute Myocardial Infarction Registry-1 (KAMIR-1) from November 2005 through December 2006. The new risk score system was tested in 1,461 hospital-discharged patients who were admitted from January 2007 through January 2008 (KAMIR-2). The new risk score system was compared to the Global Registry of Acute Coronary Events (GRACE) postdischarge risk model during a 12-month clinical follow-up. During 1-year follow-up, all-cause death occurred in 228 patients (5.7%) and 81 patients (5.5%) in the development and validation cohorts, respectively. The new risk score (KAMIR score) was constructed using 6 independent variables related to the primary end point using a multivariable Cox regression analysis: age, Killip class, serum creatinine, no in-hospital percutaneous coronary intervention, left ventricular ejection fraction, and admission glucose based on multivariate-adjusted risk relation. The KAMIR score demonstrated significant differences in its predictive accuracy for 1-year mortality compared to the GRACE score for the developmental and validation cohorts. In conclusion, the KAMIR score for patients with acute myocardial infarction is a simpler and better risk scoring system than the GRACE hospital discharge risk model in prediction of 1-year mortality. **(Am J Cardiol 2011;107:965–971)**

The Global Registry of Acute Coronary Events (GRACE) 6-month postdischarge model is a robust tool for the prediction of long-term clinical outcomes in patients with acute coronary syndrome (ACS)[1] and has been demonstrated to predict mortality for up to 4 years with good accuracy.[2] Risk stratification at hospital discharge could be very useful in guiding postdischarge care such as optimal medical therapy.[3] However, the GRACE model has several limitations. Patients and procedural characteristics in acute myocardial infarction (AMI) have been changing.[4,5] The GRACE risk model was developed and validated based on data from 1999 through 2003. Current clinical treatments may no longer fit the GRACE model with the following examples. Rate of percutaneous coronary intervention (PCI) and use of clopidogrel were merely 30% when the GRACE model was introduced. However, these treatments are currently used in approximately 90% of patients. Furthermore, the GRACE model does not consider the following risk factors: admission hyperglycemia, presence of stroke or peripheral artery disease, and left ventricular systolic function. These have been shown to be helpful in assessing risk in AMI.[6–9] None of the risk models have focused on new parameters in addition to the GRACE model in current clinical situations. Moreover, the GRACE score is difficult to calculate without the aid of a Web-based calculator or software. Accordingly, the aim of this study was to develop a novel score system based on the strongest factors independently associated with 1-year survival that would

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Table 1
Baseline clinical characteristics

Characteristics	Development Cohort (n = 3,997)	Validation Cohort (n = 1,461)
Age (years)	62.5 ± 12.5	62.6 ± 12.7
65–74	1,185 (29.6%)	432 (29.6%)
≥75	791 (18.0%)	270 (18.5%)
Women	1,147 (28.7%)	420 (28.7%)
Hypertension	1,902 (47.6%)	672 (46.0%)
Diabetes mellitus	1,068 (26.7%)	378 (26.1%)
Hypercholesterolemia	367 (9.2%)	159 (10.9%)
Current smoker	2,379 (59.9%)	905 (61.9%)
Previous myocardial infarction	291 (7.3%)	87 (6.0%)
Previous stroke or peripheral artery disease	279 (7.0%)	87 (6.0%)
Findings on admission		
Killip class II	422 (10.6%)	152 (10.4%)
Killip classes III to IV	463 (11.6%)	164 (11.3%)
Systolic blood pressure <100 mm Hg	348 (8.7%)	121 (8.3%)
Heart rate >100 beats/min	398 (10.0%)	148 (10.2%)
ST-segment depression	714 (18.6%)	269 (18.4%)
Left ventricular ejection fraction <40%	540 (13.5%)	184 (13.5%)
Serum creatinine ≥1.5 mg/dl	388 (9.8%)	154 (10.6%)
Admission glucose >180 mg/dl	1,147 (28.7%)	405 (28.3%)
Angiographic findings		
American College of Cardiology/American Heart Association lesion score C	1,764 (44.1%)	562 (38.5%)
3-vessel coronary disease	835 (20.9%)	400 (27.4%)
Left main coronary artery disease	115 (2.9%)	56 (3.8%)
In-hospital coronary revascularization		
No percutaneous coronary intervention	602 (15.1%)	212 (14.5%)
Multivessel percutaneous coronary intervention	872 (21.8%)	322 (22.1%)
Coronary artery bypass grafting	133 (3.3%)	54 (3.7%)

be used as a simple assessment tool for improving hospital discharge risk stratification in contemporary clinical practice.

Methods

The Korea Acute Myocardial Infarction Registry (KAMIR), launched in November 2005, is a Korean prospective multicenter data collection registry that reflects real-world treatment practice and outcomes in Asian patients presenting with AMI. The registry consists of 50 community and teaching hospitals with facilities for primary PCI and on-site cardiac surgery. Data were collected by a trained study co-ordinator using a standardized case-report form and protocol. The study protocol was approved by the ethics committee at each participating institution.

Eligible patients were ≥18 years of age at time of hospital admission, had a suggestive history with electrocardiographic change (new ST-T segment change, new onset of left bundle branch block, or development of pathologic Q waves) with a concomitant increase of ≥1 cardiac biomarker value of necrosis above the decision limit for MI such as creatine kinase-MB and troponins I and T.

The study population was enrolled in a nationwide prospective KAMIR from November 2005 through January 2008 from 5,458 patients with AMI who survived to hospital discharge. The entire study population had complete 1-year follow-up data. The developmental cohort consisted

Table 2
Univariate analysis for predictors of one-year mortality

Characteristics	Beta Coefficient	P Value	HR (95% CI)
Age (years)			
65–74	1.309	<0.001	3.70 (2.58–5.31)
≥75	2.080	<0.001	8.00 (5.65–11.33)
Women	0.435	0.001	1.55 (1.19–2.01)
Hypertension	0.451	0.001	1.57 (1.22–2.03)
Diabetes mellitus	0.618	<0.001	1.86 (1.44–2.40)
Hypercholesterolemia	0.023	0.92	1.02 (0.65–1.61)
Current smoker	−0.433	0.001	0.65 (0.51–0.83)
Previous myocardial infarction	0.650	<0.001	1.92 (1.44–2.55)
Previous stroke or peripheral artery disease	1.074	<0.001	2.93 (2.11–4.07)
On admission			
Killip class			
II	1.166	<0.001	3.21 (2.26–4.56)
III to IV	2.519	<0.001	7.65 (5.80–10.07)
Systolic blood pressure <100 mm Hg	0.436	0.023	1.55 (1.06–2.25)
Heart rate >100 beats/min	1.163	<0.001	3.20 (2.39–4.27)
ST-segment depression	0.644	<0.001	1.90 (1.44–2.51)
Left ventricular ejection fraction <40%	1.342	<0.001	3.83 (2.91–5.03)
Serum creatinine ≥1.5 mg/dl	1.599	<0.001	4.75 (3.64–6.20)
Glucose >180 mg/dl	0.692	<0.001	2.00 (1.55–2.57)
Angiographic finding			
American College of Cardiology/American Heart Association lesion score C	0.217	0.232	1.24 (0.87–1.77)
3-vessel coronary disease	0.587	0.002	1.80 (1.25–2.59)
Left main coronary artery disease	0.900	0.014	2.46 (1.20–5.03)
In-hospital revascularization			
No percutaneous coronary intervention	1.372	<0.001	3.94 (3.04–5.11)
Multivessel percutaneous coronary intervention	0.204	0.30	1.23 (0.83–1.80)
No coronary artery bypass grafting	−0.377	0.22	0.69 (0.37–1.26)

Table 3
Multivariate analysis for predictors of one-year mortality

Characteristics	Beta Coefficient	p Value	HR (95% CI)
Age (years)			
65–74	0.871	0.001	2.39 (1.44–3.97)
>75	1.468	<0.001	4.34 (2.59–7.28)
Killip class			
II	0.850	0.001	2.34 (1.39–3.94)
III to IV	1.401	<0.001	4.06 (2.54–6.50)
No percutaneous coronary intervention	0.797	<0.001	2.22 (1.65–2.98)
Serum creatinine ≥1.5 mg/dl	0.580	0.012	1.79 (1.13–2.81)
Left ventricular ejection fraction <40%	0.805	<0.001	2.24 (1.47–3.41)
Admission glucose >180 mg/dl	0.417	0.040	1.52 (1.02–2.26)

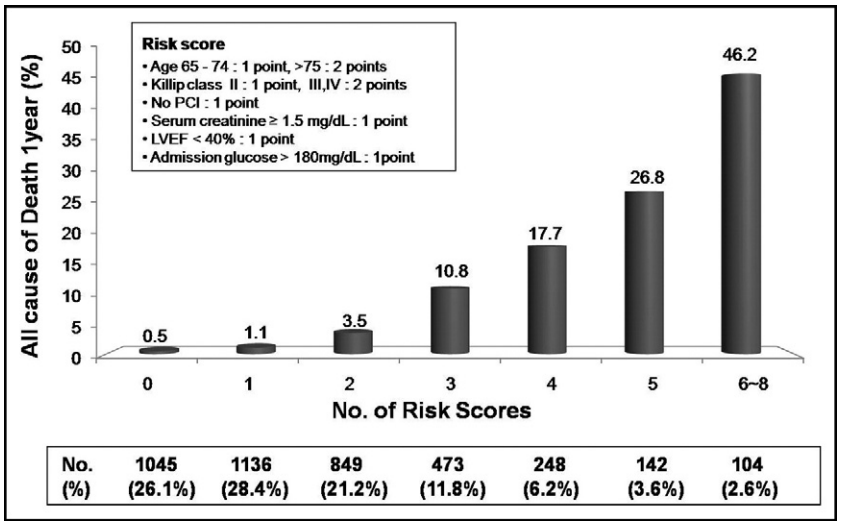

Figure 1. A new risk score predicting 1-year death from acute myocardial infarction. LVEF = left ventricular ejection fraction.

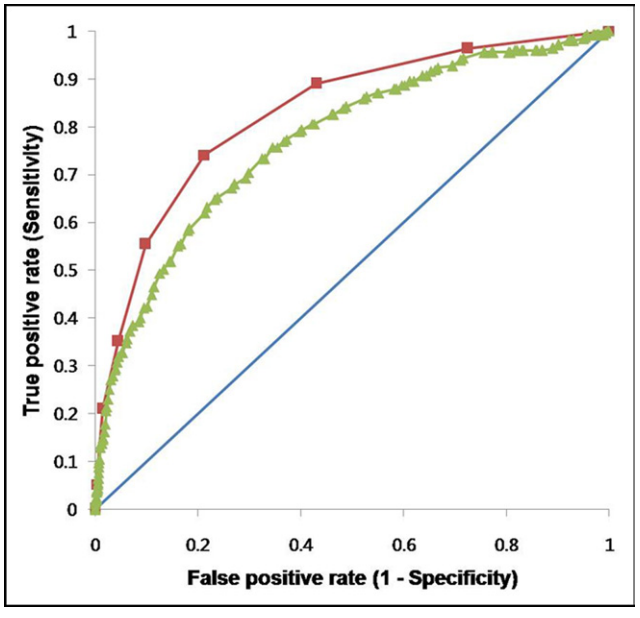

Figure 2. Receiver operator characteristic curves of no discrimination *(solid line)*, new risk score *(squares)*, and Global Registry of Acute Coronary Events score *(triangles)* for 1-year mortality in patients with acute myocardial infarction.

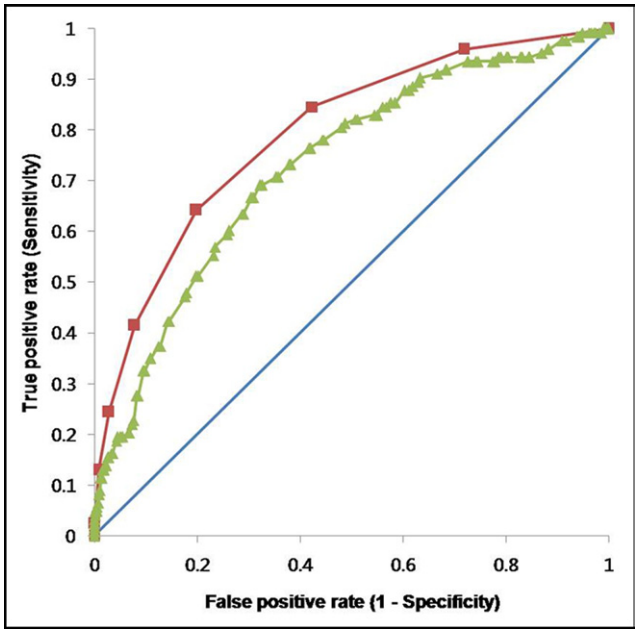

Figure 3. Receiver operator characteristic curves for no discrimination *(solid line)*, new risk score *(squares)*, and Global Registry of Acute Coronary Events score *(triangles)* for 1-year mortality in patients with ST-segment elevation myocardial infarction.

of 3,997 consecutive hospital-discharged patients who were enrolled in the nationwide prospective KAMIR-1 from November 2005 through December 2006. The new risk score system was tested in 1,461 hospital-discharged patients who were admitted from January 2007 through January 2008 (KAMIR-2).

GRACE risk scores were calculated in all patients. Age, history of heart failure, history of MI, increased heart rate at rest, low systolic blood pressure on arrival, ST-segment depression, increased initial serum creatinine, increased cardiac biomarkers, and not having in-hospital PCI were used for scoring.

ST-segment depression was defined as new horizontal or downsloping depression 0.05 mV in 2 contiguous leads.[10]

Proposed diagnostic criteria for acute kidney injury were an abrupt (within 48 hours) absolute increase in serum creatinine concentration of ≥0.3 mg/dl from baseline, percent increase in serum creatinine concentration of ≥50%, or oliguria of <0.5 ml/kg per hour for >6 hours.[11] Urine volume and prehospital serum creatinine level were not checked in the KAMIR database; level of creatinine for renal injury was defined as an increase >0.3 mg/dl above the upper limit of normal range (1.5 mg/dl). Significant hyperglycemia in AMI was defined as >180 mg/dl according to the American Heart Association scientific statement for hyperglycemia management in patients with ACS.[12] The American Heart Association has recommended that plasma

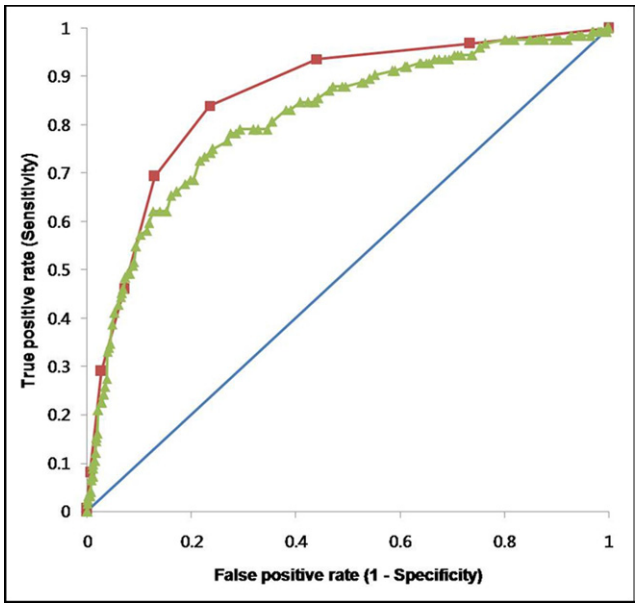

Figure 4. Receiver operator characteristic curves for no discrimination *(solid line)*, new risk score *(squares)*, and Global Registry of Acute Coronary Events score *(triangles)* for 1-year mortality in patients with non–ST-segment elevation myocardial infarction.

Table 4
Model performance in validation cohort

Variable	c-Statistic (95% CI)		P Value
	New Risk Score	GRACE Score	
Acute myocardial infarction	0.83 (0.79–0.88)	0.76 (0.72–0.83)	0.0089
ST-segment elevation myocardial infarction	0.81 (0.74–0.87)	0.73 (0.65–0.80)	0.0223
Non-ST-segment elevation myocardial infarction	0.86 (0.80–0.91)	0.78 (0.72–0.85)	0.0279

glucose concentrations in patients with ACS be measured and considered for intensive glucose control with intravenous or subcutaneous insulin. Assessment of left ventricular ejection fraction was performed using echocardiography, which is a class I recommendation in the American College of Cardiology/American Heart Association guidelines.[13]

The primary clinical end point was the composite of all-cause death at 1 year. Data were collected by a trained study co-ordinator using a standardized case-report form and protocol. To analyze prediction for 1-year mortality, data from the risk score and added parameters were employed as independent variables. Breslow-Day test was performed to assess homogeneity of relative risk across participating centers. Univariate relation between baseline characteristics and 1-year mortality was assessed by univariate Cox regression analysis. Multivariate analysis by stepwise Cox regression models (backward elimination) tested variables that were significant at a p value <0.2 in univariate analysis. All variables in the final model met the assumptions for proportional hazards.

A new risk score was developed using independent variables associated with 1-year mortality. The lowest hazard ratio (HR) was 1.52, with 1 point for an HR <3.04 and 2

Table 5
Risk scores

GRACE Model		KAMIR Score	
Age (years)		age (years)	
40–49	18	65–74	1
50–59	36	≥75	2
60–69	55		
70–79	73		
80–89	91		
≥90	100		
History of heart failure	24		
History of myocardial infarction	12		
Findings at initial hospital presentation		findings at initial hospital presentation	
Heart rate at rest (beat/min)		Killip class	
50–69	3	II	1
70–89	9	III to IV	2
90–109	14		
110–149	23		
150–199	35		
≥200	43		
Systolic blood pressure (mm Hg)			
≤79	24		
80–99	22		
100–119	18		
120–139	14		
140–159	10		
160–199	4		
ST-segment depression	11		
Findings during hospitalization		findings during hospitalization	
Initial serum creatinine (mg/dl)		serum creatinine ≥1.5 mg/dl	1
0–0.39	1	admission glucose >180 mg/dl	1
0.4–0.79	3	left ventricular ejection fraction <40%	1
0.8–1.19	5	no percutaneous coronary intervention	1
1.2–1.59	7		
1.6–1.99	9		
2.0–3.99	15		
≥4	20		
Elevated cardiac enzymes	15		
No percutaneous coronary intervention	14		

points for 3.05 to 4.56. In summary, the risk score was calculated by a simple sum of variables based on the multivariate-adjusted risk relation.

Predicted accuracy of the risk score was assessed using area under the receiver operator characteristic curve (or c-statistic).[14] A new risk score (KAMIR score) system was compared to the GRACE risk score by differences between 2 areas under the curve in the 95% confidential interval (CI). For evaluation of the risk score in all patients, missing variables contributed 0 point to the total score. Data were missing in 48 patients for serum creatinine, 86 patients for serum glucose, 124 patients for Killip class, 29 patients for systolic blood pressure, 27 patients for heart rate, 167 patients for ST-segment depression, and 13 patients for performed PCI; 311 patients (5.7%) did not have an echocar-

diogram. Except for these variables, no data were missing. A p value <0.05 was considered statistically significant. Analyses were performed using SPSS 15.0 (SPSS, Inc., Chicago, Illinois). Comparison between receiver operator characteristic curves was performed using Analyze-it standard edition (Analyse-it Software, Ltd., Leeds, United Kingdom).

Results

In total 5,458 discharged patients with AMI were included in this study. Baseline characteristics are presented in Table 1. During 1-year follow up, all-cause death occurred in 228 patients (5.7%) and 81 patients (5.5%) and cardiac death occurred in 166 patients (4.2%) and 56 patients (3.8%) in the development and validation cohorts, respectively. In addition, 5,152 patients (94.4%) underwent coronary angiography, and 197 patients (3.6%) had no significant stenosis (normal coronary artery 1.5%, myocardial bridge 0.2%, spasm 1.1%, mild stenosis 0.8%). PCI was performed in 4,617 patients (84.6%). The test for assessing homogeneity of relative risk across the center was not significant for 1-year mortality (Breslow-Day test, p = 0.641).

Univariate predictors of 1-year mortality were age, female gender, hypertension, diabetes mellitus, smoking, previous MI, history of stroke or peripheral artery disease, Killip class, initial heart rate, systolic blood pressure, ST-segment depression on electrocardiogram, left ventricular ejection fraction on echocardiogram, left main coronary artery lesion, 3-vessel disease, and baseline serum creatinine and glucose levels (Table 2). By multivariate Cox regression analysis, the 6 independent factors that increased the risk of 1-year mortality were age 65 to 74 years (HR 2.35, 95% CI 1.58 to 3.51), age >75 years (HR 4.78, 95% CI 3.25 to 7.04), Killip class II (HR 2.14, 95% CI 1.44 to 3.18), Killip classes III to IV (HR 3.76, 95% CI 2.68 to 5.27), no PCI (HR 2.22, 95% CI 1.65 to 2.98), serum creatinine ≥1.5 mg/dl (HR 2.04, 95% CI 1.49 to 2.79), left ventricular ejection fraction <40% (HR 1.86, 95% CI 1.37 to 2.52), and admission glucose >180 mg/dl (HR 1.62, 95% CI 1.06 to 2.19; Table 3).

A new risk score (KAMIR score) for AMI showed a strong graded relation to 1-year mortality (0.5% to 46.2%; Figure 1). Accuracy for 1-year mortality by the GRACE and KAMIR score systems were 0.77 (area under the curve, CI 0.74 to 0.80) and 0.83 (CI 0.80 to 0.86), respectively. A significant difference existed (0.77 vs 0.83, p <0.0001). The KAMIR score (area under the receiver operator characteristic curve 0.79, CI 0.75 to 0.83) demonstrated a significant difference in predictive accuracy compared to the GRACE score (0.79 vs 0.73, p = 0.0007) for ST-segment elevation MI and non–ST-segment elevation MI (0.86 vs 0.81, p = 0.0079; Figures 2 through 4). The KAMIR score also demonstrated significant differences in predictive accuracy compared to the GRACE model for the validation cohort in the entire range of AMI (Table 4). The KAMIR score included more in-hospital variables rather than medical history (Table 5).

Discussion

An ideal risk score system is required for an accurate prediction of prognosis, and it needs to be simple and easily accessible for wide use. However, accuracy is often accompanied by a complex calculation.[15] The KAMIR score in the present study provided simplicity and accuracy for long-term prognosis by adding independent parameters that were not included in the GRACE risk model.

Determining heart failure parameters is pivotal to improve accuracy of prediction. In the GRACE model, history of heart failure is a variable for heart failure, but this variable reflects a patient's previous status and may increase the prediction for death compared to risk after discharge or after AMI. As a mirror of heart failure, Killip classification is used to categorize patients according to their risk based on the presence of simple physical examination findings.[16] The higher the Killip class on presentation, the greater the subsequent mortality in AMI.[17] Left ventricular systolic function is an important predictor of long-term mortality after AMI.[18,19] These clinical noninvasive assessments provide powerful prognostic information and their effect is synergistic.[20] When applying receiver operating characteristic curve for 1-year mortality, area under the receiver operator characteristic curve for Killip classification was 0.72 (95% CI 0.68 to 0.76) and that for left ventricular ejection fraction <40% was 0.62 (95% CI 0.58 to 0.66). Discriminatory accuracy in the 2 parameters was superior to previous heart failure (0.52, 95% CI 0.48 to 0.58) in the KAMIR data (p <0.001). Therefore, these parameters were added as a baseline variable instead of previous heart failure.

Renal dysfunction is independently associated with increased risk of death.[21,22] Combining estimated glomerular filtration rate with a heart function indicator (left ventricular ejection fraction, N-terminal pro–brain natriuretic peptide) after MI is a predictor of 10-year mortality.[23] The GRACE and KAMIR models used serum creatinine as a renal failure indicator. However, creatinine level is not an ideal indicator of renal function particularly in older smaller patients. Therefore, we considered estimated glomerular filtration rate a variable. Estimated glomerular filtration rate was calculated by the Cockcroft–Gault formula, and then patients were categorized as having ≥60 or <60 ml/min. Estimated glomerular filtration rate was a univariate predictor of 1-year mortality (HR 6.65, 95% CI 4.56 to 9.68, beta coefficient 1.894, p <0.001), and by multivariate Cox regression analysis estimated glomerular filtration rate was an independent predictive factor. However, age 65 to 74 years was not a predictor of mortality (HR 1.28, 95% CI 0.75 to 2.19, β coefficient 1.276, p = 0.376). This may be due to repeated use of "age" as a parameter. Independent parameters were age >75 years, estimated glomerular filtration rate, admission hyperglycemia, ejection fraction, and Killip class. We calculated this score by the same method as in our study. Accuracy was not superior to our risk model despite complex calculation for 1-year mortality (area under receiver operator characteristic curve 0.821, 95% CI 0.793 to 0.849).

Age is an important parameter for short- and long-term survival after MI.[24] In the Thrombolysis In Myocardial Infarction III registry, patients >75 years of age with non–ST-segment elevation ACS had more diffuse and severe coronary disease and increased adverse outcomes than those <75 years of age.[25] The KAMIR model for the age param-

eter is much easier to calculate than the GRACE model, but area under the receiver operator characteristic curve of the KAMIR model (0.731, 95% CI 0.698 to 0.764) was almost same as the GRACE model (0.739, 95% CI 0.708 to 0.771) when applying receiver operator characteristic curve for 1-year mortality.

Hyperglycemia at presentation was associated with greater myocardial necrosis and increased 10-month mortality rate regardless of previous diabetes.[6,7] A recent study has demonstrated that tight glycemic control decreases apoptosis in peri-infarct lesions and remodeling in patients with AMI by decreasing oxidative stress and inflammation.[26] That study noted that severe hyperglycemia on admission (>180 mg/dl) is also an independent predictor and reported greater myocardial necrosis (peak creatinine kinase 1,520.9 ± 2,133.6 vs 1,335.0 ± 1,790.7, $p = 0.029$), higher creatinine levels (1.25 ± 1.06 vs 1.08 ± 1.95, $p < 0.001$), and lower ejection fraction (49.9 ± 12.6 vs 53.1 ± 19.6, $p < 0.001$). However, hyperglycemia remains underappreciated as a risk factor and untreated in patients with AMI. Only 21.3% of patients were administered subcutaneous or intravenous insulin for severe hyperglycemia in this study.

The present study had some limitations. First, validation is as important as development for a new risk model. We, however, could not access other patient populations in a more worldwide sample. Another year of data in the same registry (KAMIR-2) was used as a validation cohort in this study. Although all parameters in the KAMIR model were known strong predictors of mortality in multination or Western studies, further validation may be needed for wide use of the KAMIR model. Second, all management decisions were made by the attending cardiologist. Treatment method was not controlled by the given controlling guidelines. Hence, it is difficult to assess the benefits of individual therapy. Third, laboratory testing routinely assessed measurements of the respective hospitals in this study. Fourth, the predictive value of this score stops at 1 year after the event. Further analysis of these patients with longer follow-up may be needed to strengthen the value of this score.

1. Eagle KA, Lim MJ, Dabbous OH, Pieper KS, Goldberg RJ, Van de Werf F, Goodman SG, Granger CB, Steg PG, Gore JM, Budaj A, Avezum A, Flather MD, Fox KA; GRACE Investigators. A validated prediction model for all forms of acute coronary syndrome: estimating the risk of 6-month postdischarge death in an international registry. *JAMA* 2004;291:2727–2733.
2. Allen LA, Tang EW, Wong CK, Herbison P. Global Registry of Acute Coronary Events (GRACE) hospital discharge risk score accurately predicts long-term mortality post acute coronary syndrome. *Am Heart J* 2007;153:29–35.
3. Yan AT, Yan RT, Tan M, Huynh T, Soghrati K, Brunner LJ, DeYoung P, Fitchett DH, Langer A, Goodman SG; Canadian ACS Registries Investigators. Optimal medical therapy at discharge in patients with acute coronary syndromes: temporal changes, characteristics, and 1-year outcome. *Am Heart J* 2007;154:1108–1115.
4. Roe MT, Messenger JC, Weintraub WS, Cannon CP, Fonarow GC, Dai D, Chen AY, Klein LW, Masoudi FA, McKay C, Hewitt K, Brindis RG, Peterson ED, Rumsfeld JS. Treatments, trends, and outcomes of acute myocardial infarction and percutaneous coronary intervention. *J Am Coll Cardiol* 2010;56:254–263.
5. Peterson ED, Shah BR, Parsons L, Pollack CV Jr, French WJ, Canto JG, Gibson CM, Rogers WJ. Trends in quality of care for patients with acute myocardial infarction in the National Registry of Myocardial Infarction from 1990 to 2006. *Am Heart J* 2008;156:1045–1055.
6. Bhadriraju S, Ray KK, Defranco AC, Barber K, Bhadriraju P, Murphy SA, Morrow DA, McCabe CH, Gibson CM, Cannon CP, Braunwald E. Association between blood glucose and long-term mortality in patients with acute coronary syndromes in the OPUS-TIMI 16 trial. *Am J Cardiol* 2006;97:1573–1577.
7. Meier JJ, Deifuss S, Klamann A, Launhardt V, Schmiegel WH, Nauck MA. Plasma glucose at hospital admission and previous metabolic control determine myocardial infarct size and survival in patients with and without type 2 diabetes: the Langendreer Myocardial Infarction and Blood Glucose in Diabetic Patients Assessment (LAMBDA). *Diabetes Care* 2005;28:2551–2553.
8. Mukherjee D, Eagle KA, Kline-Rogers E, Feldman LJ, Juliard JM, Agnelli G, Budaj A, Avezum A, Allegrone J, FitzGerald G, Steg PG; GRACE Investigators. Impact of prior peripheral arterial disease and stroke on outcomes of acute coronary syndromes and effect of evidence-based therapies (from the Global Registry of Acute Coronary Events). *Am J Cardiol* 2007;100:1–6.
9. Kontos M, Jamal S, Tatum J, Ornato J, Jesse RL. Predictive power of systolic function and congestive heart failure in patients with patients admitted for chest pain without ST elevation in the troponin era. *Am Heart J* 2008;156:329–335.
10. Thygesen K, Alpert JS, White HD; Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction. Universal definition of myocardial infarction. *Eur Heart J* 2007;28:2525–2538.
11. Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, Levin A; Acute Kidney Injury Network. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. *Crit Care* 2007;11(suppl):R31.
12. Deedwania P, Kosiborod M, Barrett E, Ceriello A, Isley W, Mazzone T, Raskin P. Hyperglycemia and acute coronary syndrome: a scientific statement from the American Heart Association diabetes committee of the council on nutrition, physical activity, and metabolism. *Circulation* 2008;117:1610–1619.
13. Anderson JL, Adams CD, Antman EM, Bridges CR, Califf RM, Casey DE Jr, Chavey WE II, Fesmire FM, Hochman JS, Levin TN, Lincoff AM, Peterson ED, Theroux P, Wenger NK, Wright RS, Smith SC Jr, Jacobs AK, Adams CD, Anderson JL, Antman EM, Halperin JL, Hunt SA, Krumholz HM, Kushner FG, Lytle BW, Nishimura R, Ornato JP, Page RL, Riegel B. ACC/AHA 2007 guidelines for the management of patients with unstable angina/non–ST-Elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction) developed in collaboration with the American College of Emergency Physicians, the Society for Cardiovascular Angiography and Interventions, and the Society of Thoracic Surgeons endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation and the Society for Academic Emergency Medicine. *J Am Coll Cardiol* 2007;50(suppl):e1–e157.
14. Hanley JA, McNeil BJ. The meaning and use of the area under a receiver operating characteristic (ROC) curve. *Radiology* 1982;143:29–36.
15. Yan AT, Yan RT, Tan M, Casanova A, Labinaz M, Sridhar K, Fitchett DH, Langer A, Goodman SG. Risk scores for risk stratification in acute coronary syndromes: useful but simpler is not necessarily better. *Eur Heart J* 2007;28:1072–1078.
16. Killip T III, Kimball JT. Treatment of myocardial infarction in a coronary care unit. A two year experience with 250 patients. *Am J Cardiol* 1967;20:457–464.
17. Khot UN, Jia G, Moliterno DJ, Lincoff AM, Khot MB, Harrington RA, Topol EJ. Prognostic importance of physical examination for heart failure in non–ST-elevation acute coronary syndromes: the enduring value of Killip classification. *JAMA* 2003;290:2174–2181.
18. Køber L, Torp-Pedersen C, Pedersen OD, Høiberg S, Camm AJ. Importance of congestive heart failure and interaction of congestive heart failure and left ventricular systolic function on prognosis in patients with acute myocardial infarction. *Am J Cardiol* 1996;78:1124–1128.
19. Køber L, Torp-Pedersen C, Jørgensen S, Eliasen P, Camm AJ. Changes in absolute and relative importance in the prognostic value of

left ventricular systolic function and congestive heart failure after acute myocardial infarction. TRACE Study Group. Trandolapril Cardiac Evaluation. *Am J Cardiol* 1998;81:1292–1297.
20. Møller JE, Hillis GS, Oh JK, Reeder GS, Gersh BJ, Pellikka PA. Wall motion score index and ejection fraction for risk stratification after acute myocardial infarction. *Am Heart J* 2006;151:419–425.
21. Al Suwaidi J, Reddan DN, Williams K, Pieper KS, Harrington RA, Califf RM, Granger CB, Ohman EM, Holmes DR Jr; GUSTO-IIb, GUSTO-III, PURSUIT. Global Use of Strategies to Open Occluded Coronary Arteries. Platelet Glycoprotein IIb/IIIa in Unstable Angina: Receptor Suppression Using Integrilin Therapy; PARAGON-A Investigators. Platelet IIb/IIIa antagonism for the reduction of acute coronary syndrome events in a global organization network. Prognostic implications of abnormalities in renal function in patients with acute coronary syndromes. *Circulation* 2002;106:974–980.
22. Anavekar NS, McMurray JJ, Velazquez EJ, Solomon SD, Kober L, Rouleau JL, White HD, Nordlander R, Maggioni A, Dickstein K, Zelenkofske S, Leimberger JD, Califf RM, Pfeffer MA. Relation between renal dysfunction and cardiovascular outcomes after myocardial infarction. *N Engl J Med* 2004;351:1285–1295.
23. Palmer SC, Yandle TG, Frampton CM, Troughton RW, Nicholls MG, Richards AM. Renal and cardiac function for long-term (10 year) risk stratification after myocardial infarction. *Eur Heart J* 2009;30:1486–1494.
24. Goldberg RJ, McCormick D, Gurwitz JH, Yarzebski J, Lessard D, Gore JM. Age-related trends in short- and long-term survival after acute myocardial infarction: a 20-year population-based perspective (1975–1995). *Am J Cardiol* 1998;82:1311–1317.
25. Stone PH, Thompson B, Anderson HV, Kronenberg MW, Gibson RS, Rogers WJ, Diver DJ, Théroux P, Warnica JW, Nasmith JB, Kells C, Kleiman N, McCabe CH, Schactman M, Knatterud GL, Braunwald E. Influence of race, sex, and age on management of unstable angina and non–Q-wave myocardial infarction: the TIMI III registry. *JAMA* 1996; 275:1104–1112.
26. Marfella R, Di Filippo C, Portoghese M, Ferraraccio F, Rizzo MR, Siniscalchi M, Musacchio E, D'Amico M, Rossi F, Paolisso G. Tight glycemic control reduces heart inflammation and remodeling during acute myocardial infarction in hyperglycemic patients. *J Am Coll Cardiol* 2009;53:1425–1436.

Appendix

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