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Predictors of Major Adverse Cardiovascular Events in Patients Presenting with Acute Myocardial Infarction

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ABSTRACT

Background: Despite current reperfusion and secondary prevention, acute myocardial infarction (AMI) continues to be linked to high early and late cardiovascular risk. **Background:** Identify clinical, laboratory, angiographic and echocardiographic predictors of 1-year major adverse cardiovascular events (MACE) after AMI.

Methods: This was a prospective cohort study of 428 consecutive adults who had suffered a ST-elevation or non-ST-elevation myocardial infarction (MI) and followed for 12 months. MACE included cardiovascular death, nonfatal recurrent myocardial infarction, nonfatal ischemic stroke, or unplanned coronary revascularization. Independent predictors were identified by Cox regression and baseline variables were compared between patients with and without MACE.

Results: Mean age was 59.6 ± 11.8 years and 326 patients (76.2%) were men. 62.6% of presentations were STEMI. MACE was observed in 94 patients (22.0%): cardiovascular death (31 patients, 7.2%), recurrent myocardial infarction (29 patients, 6.8%), ischemic stroke (11 patients, 2.6%) and unplanned revascularization (38 patients, 8.9%), some patients having more than one event. MACE was more common in Killip class $\geq$ II, anterior STEMI, admission glucose $\geq$ 180 mg/dL, eGFR $<$ 60 mL/min/1.73 m², LVEF $<$ 40%, multivessel coronary disease, and incomplete revascularization in patients $\geq$ 65 years of age. Independent predictors were Killip class $\geq$ II (adjusted HR 2.18, 95% CI 1.42–3.34), LVEF $<$ 40% (HR 1.89, 95% CI 1.24–2.88), eGFR $<$ 60 (HR 1.72, 95% CI 1.12–2.64), admission glucose $\geq$ 180 mg/dL (HR 1.61, 95% CI 1.05–2.47), and multivessel disease (HR 1.58, 95% CI 1.02–2.44).

Conclusions: AMI patients were identified as having a higher risk of 1-year MACE due to a combination of hemodynamic compromise, ventricular dysfunction, renal impairment, metabolic stress and coronary disease burden.

Keywords: acute myocardial infarction; major adverse cardiovascular events; risk prediction; Killip class; ejection fraction; renal function.

INTRODUCTION

Acute myocardial infarction (AMI) is still a major cause of morbidity and mortality in cardiovascular disease. In fact, current ACS guidelines focus on the early diagnosis, timely reperfusion, powerful antiplatelet and antithrombotic therapy, intensive lipid lowering and thorough secondary prevention, as residual risk remains after technically successful revascularization [1]. The European guidelines also emphasize risk stratification from the first medical contact to long-term follow-up and suggest the combination of clinical status, electrocardiographic parameters, cardiac biomarkers, ventricular function, coronary anatomy and the presence of comorbidities [2].

Post-AMI risk is variable. The classic Killip classification showed that the degree and presence of heart failure at the time of AMI is a strong predictor of mortality [3]. Further large-scale data from the reperfusion period validated age, systolic blood pressure, heart rate, anterior infarction, and clinical heart failure as early prognostic factors. Simple bedside scores were thus created to convert these frequently available features into reproducible risk estimates.

The TIMI risk scores for STEMI and non-ST-elevation acute coronary syndromes set the groundwork for practical prognosticating early in the care of these patients [4,5]. The GRACE investigators then created and tested models that included age, heart rate, systolic blood pressure, creatinine, Killip class, cardiac arrest, ST-segment deviation and biomarker evidence of myocardial necrosis [6]. GRACE-derived estimates also have a prognostic value following discharge and can predict risk beyond the index hospitalization [7].

But, the results of the current era are influenced by factors which are not fully captured by the older models. The clinical course of AMI has been transformed by high-sensitivity troponin testing, primary PCI, radial access, newer antiplatelet therapy, routine echocardiography, staged complete revascularization, contemporary lipid-lowering therapy, and improved heart-failure therapy. Meanwhile, renal dysfunction, stress hyperglycemia, impaired left ventricular ejection fraction and multivessel coronary disease remain to be markers of biologically vulnerable patients. Recent evidence also supports the negative prognostic relationship between admission hyperglycemia in both diabetic and non-diabetic AMI patients [8].

The current study, therefore, examined predictors of 1-year MACE in a contemporary cohort of AMI patients. We hypothesized that hemodynamic severity, ventricular dysfunction, renal impairment, metabolic stress, and coronary anatomic burden would be independent predictors of increased risk even after receiving guideline-directed acute treatment.

MATERIALS AND METHODS

Study design and population

A total of 428 consecutive adults with AMI were included in this prospective observational study. AMI was diagnosed based on a compatible clinical presentation, dynamic cardiac troponin elevation with at least one value above the 99th percentile of the assay, and supporting electrocardiographic, imaging, or angiographic evidence of myocardial ischemia. Both STEMI and NSTEMI were eligible. Patients were excluded from the study if the diagnosis was type 2 myocardial infarction, myocarditis was diagnosed, or Takotsubo syndrome was diagnosed, if there was no follow-up, or if the life expectancy from non-cardiovascular disease was less than 12 months.

A baseline assessment and treatment should be provided.

Demographic data, traditional cardiovascular risk factors, history of coronary disease, symptom-to-door time, heart rate, systolic blood pressure, Killip class, admission glucose, hemoglobin, creatinine, eGFR, and peak high-sensitivity cardiac troponin were documented. Twelve-lead electrocardiograms were classified according to the infarct territory. Echocardiography was done at the time of admission, and LVEF was estimated by the biplane Simpson method, if possible. Coronary angiography revealed the culprit vessel, the number of epicardial coronary arteries involved, the involvement of the left-main artery, final TIMI flow and completeness of revascularization. The treating cardiology team provided reperfusion, antithrombotic therapy, and secondary prevention as per the standard practice in the institution at that time.

The definition of outcomes and follow up.

The main outcome was the first event of MACE within 12 months, which comprised cardiovascular death, nonfatal recurrent myocardial infarction, nonfatal ischemic stroke, or unplanned coronary revascularization. Recurrent myocardial infarction was defined as a new ischemic presentation with evidence of myocardial injury in the biomarkers and objective evidence of ischemia. Unplanned revascularization was defined as urgent or clinically indicated PCI or coronary bypass surgery following discharge. Follow-up was conducted by clinic review, telephone calls and review of hospital records. The events were judged according to a set of criteria.

Statistical analysis

Continuous variables were reported as mean $\pm$ standard deviation or median (interquartile range) and categorical variables as n (%). Student's t test or Mann-Whitney U test was used for continuous variables and chi-square or Fisher exact test was used for categorical variables to compare patients with and without MACE. Kaplan-Meier methods were used to create time-to-event curves. Multivariate Cox proportional-hazards regression was used to determine independent predictors of MACE. Clinical relevance and univariable association were used to select candidate variables. The collinearity of the variables was checked prior to modeling. The adjusted hazard ratios (HR) with 95% confidence intervals (CI) were reported. Two-sided $p < 0.05$ was considered to be statistically significant.

RESULTS

Among 428 patients, mean age was 59.6 ± 11.8 years, 326 (76.2%) were men, and 268 (62.6%) presented with STEMI. Diabetes mellitus was present in 168 (39.3%), hypertension in 249 (58.2%), and current smoking in 171 (40.0%). Killip class II–IV was observed in 92 patients (21.5%) at presentation. Mean LVEF was $45.2 \pm 9.8\%$; 114 patients (26.6%) had LVEF $< 40\%$. Primary PCI was performed in most eligible STEMI patients, while an invasive strategy was used for the majority of NSTEMI patients.

At 12 months, 94 patients (22.0%) experienced at least one MACE. Patients with MACE were older and more frequently had diabetes, Killip class $\geq$ II, reduced eGFR, high admission glucose, LVEF $< 40\%$, multivessel coronary artery disease, and incomplete revascularization. They also had longer median symptom-to-door time and a higher frequency of anterior STEMI.

Table 1. Baseline characteristics according to 1-year MACE status

Characteristic	MACE (n=94)	No MACE (n=334)	p value
Age, years	64.8 ± 11.2	58.1 ± 11.5	< 0.001
Male sex, n (%)	69 (73.4)	257 (76.9)	0.48
Diabetes mellitus, n (%)	48 (51.1)	120 (35.9)	0.008
STEMI, n (%)	64 (68.1)	204 (61.1)	0.22
Anterior STEMI, n (%)	39 (41.5)	91 (27.2)	0.008
Killip class $\geq$ II, n (%)	39 (41.5)	53 (15.9)	< 0.001
Admission glucose, mg/dL	184 ± 72	153 ± 58	< 0.001
eGFR, mL/min/1.73 m ²	63.1 ± 22.4	76.8 ± 20.6	< 0.001
LVEF, %	39.9 ± 9.6	46.7 ± 9.2	< 0.001

MACE: major adverse cardiovascular events; STEMI: ST-elevation myocardial infarction; eGFR: estimated glomerular filtration rate; LVEF: left ventricular ejection fraction.

Table 2. Angiographic characteristics and 1-year outcomes

Variable	MACE (n=94)	No MACE (n=334)	p value
Multivessel coronary disease, n (%)	61 (64.9)	151 (45.2)	0.001
Left-main/left-main equivalent disease, n (%)	11 (11.7)	17 (5.1)	0.024
Final culprit-vessel TIMI 3 flow, n (%)	82 (87.2)	315 (94.3)	0.020
Incomplete revascularization, n (%)	37 (39.4)	72 (21.6)	< 0.001
Cardiovascular death, n (%)	31 (33.0)	0	—
Recurrent MI, n (%)	29 (30.9)	0	—
Ischemic stroke, n (%)	11 (11.7)	0	—
Unplanned revascularization, n (%)	38 (40.4)	0	—

TIMI: Thrombolysis in Myocardial Infarction; MI: myocardial infarction. Individual MACE components are not mutually exclusive.

Table 3. Independent predictors of 1-year MACE

Predictor	Adjusted HR	95% CI	p value
Killip class $\geq$ II	2.18	1.42–3.34	< 0.001
LVEF $< 40\%$	1.89	1.24–2.88	0.003

Predictor	Adjusted HR	95% CI	p value
eGFR <60 mL/min/1.73 m ²	1.72	1.12–2.64	0.013
Admission glucose ≥180 mg/dL	1.61	1.05–2.47	0.029
Multivessel coronary disease	1.58	1.02–2.44	0.039
Age ≥65 years	1.47	0.98–2.22	0.064
Complete revascularization	0.66	0.43–1.00	0.051

HR: hazard ratio; CI: confidence interval. Model adjusted additionally for sex, diabetes, infarct type, anterior location, systolic blood pressure, and symptom-to-door time.

Cardiovascular death occurred in 31 patients (7.2%), recurrent myocardial infarction in 29 (6.8%), ischemic stroke in 11 (2.6%), and unplanned coronary revascularization in 38 (8.9%). Because some patients experienced multiple events, component counts exceeded the number with first MACE. Most first events occurred within the initial 6 months, but clinically important residual risk continued throughout the follow-up period.

In multivariable Cox regression, Killip class ≥II was the strongest independent predictor of MACE. LVEF <40%, eGFR <60 mL/min/1.73 m², admission glucose ≥180 mg/dL, and multivessel coronary disease also independently predicted higher risk. The association of age ≥65 years was attenuated after adjustment, while complete revascularization showed a protective direction that narrowly missed conventional statistical significance.

DISCUSSION

This study was able to define a clinically meaningful set of predictors for 1-year MACE after AMI. Each of the following factors at presentation provided independent prognostic information: hemodynamic compromise, impaired LV systolic function, renal dysfunction, and marked admission hyperglycemia, and multivessel coronary disease. The results of this study demonstrate that residual risk after AMI is more than just the culprit lesion; it also represents the patient's physiologic reserve, myocardial injury, systemic comorbidity, and overall atherosclerotic burden.

Killip class ≥II was the most important independent predictor. This is in line with the initial observation by Killip and Kimball that during AMI, clinical heart failure is correlated with a dramatic increase in mortality [3]. Despite the significant improvements in reperfusion, intensive care and pharmacotherapy, pulmonary congestion or shock at presentation remains a marker of severe myocardial dysfunction, high filling pressure, neurohormonal activation and low hemodynamic reserve. This is biologically and clinically plausible because the persistence of Killip class in current risk models.

Our results also are consistent with existing bedside risk tools. The TIMI score for STEMI includes age, blood pressure, heart rate, Killip class, location of the infarct, weight, history of diabetes and hypertension, and delay in treatment [4] whereas the TIMI score for unstable angina/NSTEMI includes higher risk based on different clinical and electrocardiographic variables [5]. The GRACE model expanded this and showed the benefit of adding age, heart rate, systolic pressure, creatinine, Killip class, arrest, ST deviation and biomarker elevation [6]. Importantly, GRACE-derived assessment continues to be useful after discharge, and predicts longer-term mortality [7].

The reduced LVEF was an independent predictor of MACE in our cohort. LVEF incorporates the functional implications of infarct size, previous myocardial damage, stunning, remodeling and residual ischemia. An LVEF < 40% also identifies patients who may require specific heart-failure therapies and who are eligible for further evaluation for device therapy. The association between low LVEF and adverse events seen here highlights the importance of regular echocardiographic follow-up during the initial admission and at the time of recovery, if clinically indicated.

Another strong predictor was renal dysfunction. Low eGFR can be a sign of chronic vascular disease, diabetes, hypertension, endothelial dysfunction or cardiorenal interaction. It also makes it difficult to use contrast and affects the dosage of antithrombotics, and it can lead to bleeding and heart failure. These are the reasons why renal function is included in the GRACE model [6]. Clinically, an AMI patient with eGFR <60 mL/min/1.73 m² should be considered to be at higher risk even if reperfused.

Hyperglycemia at admission was an independent risk factor for MACE. Acute hyperglycemia can be a sign of undiagnosed diabetes, poor glycemic control or a stress response due to catecholamines. It can cause oxidative stress, endothelial dysfunction, platelet activation, inflammation and impaired microvascular reperfusion. An adverse association between admission hyperglycemia and outcomes in AMI was again reported in a recent systematic review and meta-analysis,

irrespective of whether the patients were diabetic or non-diabetic [8]. Therefore, the glucose value at admission can be used as a prognostic parameter, in addition to a binary diabetes history.

Multivessel coronary disease also was an independent predictor of events. This is probably due to increased total atherosclerotic burden and increased number of plaques with future potential for destabilization. The association of incomplete treatment and incomplete revascularization was in a protective direction, but the association was not statistically significant in this cohort. The current guidelines focus on individual decision making for complete revascularization based on presentation of the infarct, infarct anatomy, hemodynamic status, lesion complexity, and the presence of comorbidities [1,2]. The results of this study provide strong rationale for careful post-AMI planning for residual disease and aggressive secondary prevention, regardless of whether the remaining lesions are treated immediately, during the index admission or in a staged fashion.

The study supports a pragmatic method of post-AMI care. A multidisciplinary approach to early care and meticulous optimization of secondary prevention is warranted in patients with heart failure, low LVEF, renal impairment, marked hyperglycemia, and multivessel disease, as well as closer monitoring after discharge. Risk assessment should not be static and should not only be done at admission but can change significantly in the first months, as may ventricular function, renal function, symptoms, medication adherence and recurrent ischemia.

There are a number of caveats to be noted. The design of the observations is susceptible to residual confounding. Less common endpoints (stroke) were not analyzed and there was no adequate interaction testing for STEMI versus NSTEMI. Treatment decisions were not made by protocol, and therefore associations with revascularization completeness may be due to selection factors. Standard care did not include the standardization of troponin assays or infarct-size imaging. Lastly, the definition of MACE differs between studies and makes it difficult to compare the absolute event rates. However, clinically relevant endpoints were used and predictors were consistent with known pathophysiology and validated risk models.

Future studies should integrate these traditional risk factors with longitudinal biomarkers, quantitative infarct imaging, coronary plaque features, frailty scores, and machine-learning techniques, while maintaining interpretability. It is also important to assess any new tool for calibration in the local population and to determine whether it alters management, or merely enhances statistical discrimination.

The present results are complemented by large reperfusion-era datasets. Among the most important factors influencing 30-day mortality in an international group of over 41,000 patients with acute myocardial infarction were age, hemodynamic variables, infarct characteristics and severity of heart failure [9]. The prospective multinational GRACE study also showed that easily available clinical, electrocardiographic, renal, and biomarker data can stratify the risk of death and recurrent infarction throughout the spectrum of acute coronary syndromes [10]. These observations are supportive of the ongoing use of integrated bedside risk assessment despite the ongoing development of modern reperfusion and pharmacologic therapy.

There is a need for uniformity in the definition and adjudication of recurrent infarction. The Fourth Universal Definition of Myocardial Infarction provided a unified definition that incorporates biomarker change with clinical evidence of acute ischemia and continues to be relevant in the presence of composite MACE endpoints that include recurrent MI [11]. This is especially important in observational follow-up, where isolated troponin elevation should not be considered as a marker of reinfarction in the absence of ischemic markers.

Another important determinant of outcome is the reperfusion time, particularly in STEMI. De Luca et al. showed a linear relationship between ischemic delay and 1-year mortality following primary angioplasty [12]. Likewise, Terkelsen et al. demonstrated that independently, system delay was associated with mortality in patients receiving primary PCI [13]. Our observation that patients with more severe hemodynamic compromise and more extensive myocardial injury continue to be vulnerable despite current intervention is supported by these findings, and highlights the need to reduce patient- and system-related delays.

Laboratory markers gave further prognostic information. Anemia was identified as an independent clinically relevant risk marker in a large acute-coronary-syndrome analysis [14]. In-hospital and 1-year mortality have also been shown to be significantly higher in patients with renal deterioration following STEMI [15]. Last but not least, cardiogenic shock is the most extreme part of the hemodynamic-risk spectrum; large AMI cohorts have demonstrated that age, prior infarction, altered perfusion, oliguria and adverse hemodynamic findings are associated with particularly high mortality rates in patients with shock [16]. All these data support the concept of a multimarker approach in which clinical severity, ventricular

function, renal status, metabolic disturbance, anemia, and coronary anatomy should be read together when estimating the risk after AMI.

CONCLUSION

Multivessel coronary disease, Killip class $\geq$ II, LVEF $<40\%$, eGFR <60 mL/min/1.73 m², and admission glucose ≥ 180 mg/dL were independent risk factors for 1-year MACE in patients with AMI. These variables are easily obtained at the index hospitalization and can be used to aid pragmatic risk stratification. Patients with more than one high-risk feature might require more aggressive secondary prevention, heart failure and renal management, assessment of remaining coronary disease, and more frequent post-discharge follow-up.

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