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Research Article

Integrating Inflammatory (CRP), Nutritional (Albumin), and Stress Hormonal Biomarkers (Cortisol and Catecholamines) for Risk Stratification in Acute Myocardial Infarction

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Abstract

Risk stratification in acute myocardial infarction (AMI) remains a cornerstone for guiding management and predicting outcomes. Conventional risk scores rely heavily on clinical and angiographic features, but recent evidence suggests that biochemical markers reflecting systemic inflammation, nutritional status, and stress physiology may provide additional predictive value. This prospective cohort study evaluated the integrated utility of inflammatory [C-reactive protein (CRP)], nutritional [serum albumin], and stress hormonal [cortisol and plasma catecholamines] biomarkers for risk stratification in AMI patients. A total of 240 patients presenting with confirmed AMI were enrolled. Baseline biomarker levels were measured within 24 hours of admission and correlated with 30-day major adverse cardiac events (MACE). Elevated CRP and cortisol, combined with hypoalbuminemia, significantly predicted higher MACE risk, while catecholamine surges correlated with early complications including arrhythmias and hemodynamic instability. A composite biomarker risk index demonstrated superior prognostic accuracy (AUC = 0.84) compared to conventional scores alone (AUC = 0.71). These findings support the clinical integration of multimodal biomarker profiling to enhance early risk stratification and optimize therapeutic decision-making in AMI.

Keywords: Acute myocardial infarction, CRP, albumin, cortisol, catecholamines, risk stratification

Introduction

Acute myocardial infarction (AMI) continues to be a leading cause of morbidity and mortality worldwide despite advances in reperfusion strategies, pharmacotherapy, and secondary prevention. Timely and accurate risk stratification is essential to identify high-risk patients who may benefit from aggressive interventions and closer monitoring. Conventional tools such as the TIMI and GRACE scores incorporate clinical parameters, hemodynamic variables, and laboratory findings like troponin, yet they fail to fully capture the systemic pathophysiological complexity of AMI.1-4

A growing body of research has highlighted the importance of biomarkers that extend beyond myocardial necrosis to reflect inflammatory activation, nutritional reserve, and neuroendocrine stress responses. These pathways are intricately linked to prognosis after AMI, influencing myocardial recovery, arrhythmia susceptibility, and long-term survival.5-7

Inflammation plays a pivotal role in atherosclerotic plaque rupture and post-infarction remodeling. CRP, as a sensitive marker of systemic inflammation, has been consistently associated with adverse cardiovascular outcomes. Persistently elevated CRP after AMI predicts recurrent ischemia, heart failure, and death.8-10

Nutritional status, often overlooked in acute care, influences resilience to ischemic stress. Hypoalbuminemia is not only a marker of poor nutrition but also reflects systemic inflammation and capillary leak. Several studies have shown that low albumin levels are linked to increased mortality and complications in AMI.

Stress hormonal biomarkers, particularly cortisol and catecholamines, capture the neuroendocrine activation that accompanies acute ischemia. Elevated cortisol indicates hypothalamic–pituitary–adrenal axis activation, which can worsen insulin resistance, endothelial dysfunction, and myocardial remodeling. Catecholamine surges, while adaptive in maintaining perfusion, predispose patients to malignant arrhythmias and hemodynamic compromise.

Individually, these biomarkers have prognostic significance. However, their integration into a composite index may provide a more holistic picture of patient vulnerability. By simultaneously

evaluating inflammation, nutrition, and stress responses, clinicians may achieve a refined stratification of AMI patients beyond conventional scoring systems.

The present study sought to systematically investigate the predictive role of CRP, albumin, cortisol, and catecholamines in AMI, both individually and in combination, for short-term outcomes. The central hypothesis was that a multimarker approach would yield superior prognostic accuracy compared to single biomarkers or conventional risk scores.

Methodology

This prospective cohort study was conducted in Shaikh Zayed Medical College, Lahore. Ethical approval was obtained, and informed consent was collected from all participants.

Sample size calculation: Using Epi Info v7.2, a minimum of 210 participants was required to detect a 15% difference in MACE incidence between high- and low-risk biomarker groups with 95% confidence and 80% power. To compensate for potential attrition, 240 patients were recruited.

Inclusion criteria: Adults aged 18–75 years with confirmed ST-elevation or non-ST-elevation MI diagnosed by ECG and troponin elevation.
Exclusion criteria: Chronic inflammatory diseases, malignancy, chronic steroid therapy, end-stage renal or hepatic disease.

Data collection:

- CRP measured via high-sensitivity immunoassay.
- Serum albumin assessed by bromocresol green method.
- Serum cortisol measured by electrochemiluminescence.
- Plasma catecholamines (epinephrine, norepinephrine) quantified by HPLC.

Outcomes: Primary outcome was 30-day MACE (composite of recurrent MI, heart failure, arrhythmia requiring intervention, or death). Secondary outcomes included individual complication rates.

Statistical analysis: Continuous variables were expressed as mean ± SD. Paired and independent t-tests were used where appropriate. Logistic regression models tested associations between biomarkers and outcomes. ROC curves compared predictive accuracy of individual biomarkers, conventional scores, and composite biomarker index. $p < 0.05$ was considered significant.

Results

Table 1: Demographic and Baseline Characteristics

Variable	Total (n=240)
Age (years, mean ± SD)	59.3 ± 11.4
Male sex, n (%)	172 (71.7%)
STEMI / NSTEMI, n (%)	148 (61.7%) / 92 (38.3%)
Hypertension, n (%)	126 (52.5%)
Diabetes mellitus, n (%)	94 (39.2%)
Killip class ≥ II, n (%)	58 (24.2%)

Interpretation: The cohort reflected typical AMI demographics with a predominance of older males and common comorbidities.

Table 2: Biomarker Profiles in Patients With and Without MACE at 30 Days

Biomarker	No MACE (n=178)	MACE (n=62)	p-value
CRP (mg/L)	8.2 ± 3.6	15.9 ± 5.8	<0.001
Albumin (g/dL)	3.9 ± 0.5	3.2 ± 0.6	<0.001
Cortisol (µg/dL)	17.4 ± 6.3	28.1 ± 7.4	<0.001
Catecholamines (pg/mL)	220 ± 86	388 ± 104	<0.001

Interpretation: Patients with MACE had significantly higher inflammatory and stress biomarkers and lower albumin.

Table 3: Predictive Accuracy of Biomarker Models

Model	**AUC**	**Sensitivity (%)**	**Specificity (%)**
CRP alone	0.72	68	70
Albumin alone	0.69	64	67
Cortisol alone	0.74	71	73
Catecholamines alone	0.70	65	72
Conventional GRACE score	0.71	70	69
Composite biomarker index	0.84	79	81

Interpretation: The composite biomarker index outperformed individual biomarkers and the conventional GRACE score.

Discussion

This study demonstrated that CRP, albumin, cortisol, and catecholamines are strongly associated with short-term adverse outcomes following AMI. Their integration into a composite index significantly improved prognostic accuracy. 11-14 Elevated CRP underscores the central role of inflammation in AMI pathophysiology. Persistent inflammatory activation likely contributes to impaired myocardial healing and arrhythmogenic substrate. Hypoalbuminemia reflects both poor baseline nutritional status and acute-phase response. Its predictive value highlights the interplay between systemic health and cardiac recovery.15-16 Stress hormonal markers captured acute neuroendocrine activation. Elevated cortisol levels paralleled increased adverse outcomes, consistent with the maladaptive effects of prolonged HPA axis stimulation. Similarly, catecholamine surges correlated with arrhythmias, reflecting heightened adrenergic drive.17-18 Importantly, the composite biomarker approach provided incremental predictive value beyond the GRACE score. This suggests that biochemical profiling can refine existing risk tools.19-20 From a clinical standpoint, integrating these assays into routine early evaluation could identify patients needing intensive monitoring, early invasive strategies, or adjunctive anti-inflammatory and nutritional interventions. Limitations include single-center design, relatively short follow-up, and lack of long-term mortality data. Larger multicenter trials are warranted to validate the biomarker index and assess cost-effectiveness.

Conclusion

Integrated biomarker assessment combining CRP, albumin, cortisol, and catecholamines significantly enhances risk stratification in AMI. The composite index outperformed conventional scoring systems, offering a holistic reflection of inflammatory, nutritional, and stress responses. Incorporation of such multimarker strategies could optimize clinical decision-making and improve patient outcomes.

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