OVERVIEW OF PROGNOSTIC MODELS FOR MAJOR ADVERSE CARDIOVASCULAR EVENTS IN PATIENTS WITH ACUTE CORONARY SYNDROME

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Major adverse cardiovascular events (MACE) represent a key outcome in the management of acute coronary syndrome (ACS), requiring prognostic models with strong performance and standardized reporting. This review summarizes studies published from 2000 to 2025 investigating models predicting MACE in ACS, including classical clinical scores, biomarker-based models, nomograms, and machine learning approaches. Traditional models reported moderate discrimination for the TIMI score (C-statistic = 0.65) and higher accuracy for GRACE 2.0 (C = 0.75-0.81). In post-intervention cohorts, the CADILLAC, ACTION, and CAMI scores achieved C = 0.80-0.90. Biomarker-integrated models demonstrated improved performance, with BIPass showing C = 0.79 (95% CI 0.73-0.85). Nomogram-based tools predicting 6-24-month or post-PCI MACE reported AUC = 0.79-0.89. Machine learning models, including deep neural networks and long short-term memory algorithms using dynamic hs-troponin and ECG data, achieved AUC > 0.90 for short-term MACE prediction in emergency department chest-pain cohorts. The findings provide a quantitative overview of the prognostic performance of MACE prediction models in patients with ACS. Overall, the GRACE score remains the cornerstone of risk stratification in NSTEMI-ACS, while nomogram-based and machine learning models show substantial potential for individualized prognostication, and still require local recalibration and external validation to establish their clinical applicability.

Keywords: Major adverse cardiovascular events, prognostic models, acute coronary syndrome.

I. INTRODUCTION

Acute coronary syndrome (ACS) comprises ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), and unstable angina. Patients with ACS are at high risk for major adverse cardiovascular events (MACE), including death, recurrent myocardial infarction, stroke, or urgent coronary revascularization. Globally, ischemic heart disease remains the leading cause of mortality, and recent data indicate that more than 7 million individuals are diagnosed with ACS each year.[1] Despite advances in reperfusion strategies and secondary prevention, substantial residual risk persists, approximately one in four patients who survive an index ACS will experience recurrent myocardial infarction, stroke, or cardiovascular death during follow-up, and contemporary cohorts report annual MACE rates of around 3-4% beyond the first year of the acute event.[2,3] Predicting MACE in ACS is of major clinical importance, as it supports risk stratification, guides treatment strategies, and optimizes post-intervention care.[4] Current clinical guidelines recommend comprehensive risk assessment after ACS admission to determine the most appropriate management strategy for each patient.[5,6]

Over the past decades, numerous risk scores and prognostic models have been

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developed to estimate MACE risk in patients with ACS. Among them, the TIMI and GRACE scores are the most widely used and have been incorporated into international ACS management guidelines. However, both models were derived from patient cohorts in the early 2000s, when population characteristics and treatment strategies differed from those of current practice.[7] For example, the proportion of elderly patients, multimorbidity, and the use of newer therapies-including next-generation drug-eluting stents, modern P2Y12 inhibitors, and high-intensity statins-have substantially changed the clinical landscape of ACS management in recent years.[5,6] Consequently, earlier prognostic models may no longer fully capture contemporary patient risk, especially in underrepresented populations. Based on this rationale, this study aims to provide an overview of existing prognostic models for predicting major adverse cardiovascular events in patients with ACS. Unlike prior reviews focusing mainly on classical scores or ML-only tools, this review synthetizes guideline-era models (GRACE 2.0, CAMI, ABC-ACS…), biomarker-based models, and modern AI systems published in the last decade.

II. OVERVIEWS

1. Materials and methods

This work was designed as a narrative review with a structured search strategy. A narrative approach was chosen because prognostic models for MACE in ACS are highly heterogeneous in terms of outcome definitions, prediction horizons, target populations, and modeling techniques, which limits the feasibility and interpretability of a fully systematic or quantitative synthesis. Rather than attempting formal meta-analysis, our primary aim was to provide an integrated, clinically oriented overview of key models and their performance across different settings. This narrative review was conducted using studies published worldwide between 2000 and 2025.

Databases

The literature search was performed using major online biomedical databases, including PubMed/MEDLINE, Web of Science, and Google Scholar.

Search Strategy

The search strategy combined Medical Subject Headings (MeSH) and free-text keywords, including: "acute coronary syndrome", "ACS", "major adverse cardiovascular events", "MACE", "risk score", "prognostic model", "NT-proBNP", "GDF-15", "nomogram", "machine learning", "deep learning". Boolean operators were applied as follows "acute coronary syndrome" OR ACS AND MACE OR "major adverse cardiovascular events" AND "risk model" OR "risk score" OR biomarker OR nomogram OR "machine learning".

Inclusion Criteria

Studies were included if they met the following criteria:

- Population: adults diagnosed with ACS (including STEMI, NSTEMI, or unstable angina).

- Study focus: development, updating, or validation of prognostic models or risk scores designed to predict short-term or long-term MACE.

- Study design: cohort studies, registry analyses, or secondary analyses of clinical trials reporting model performance.

- Outcomes: at least one composite or individual MACE outcome (e.g., all-cause or cardiovascular mortality, recurrent myocardial infarction, stroke, or urgent coronary revascularization). Variations in MACE definitions across studies were permitted but documented and considered in the qualitative synthesis.

- Reporting: provision of at least one measure of model performance, such as discrimination (C-statistic/area under the curve [AUC]) and, where available, calibration (e.g., calibration plots, Hosmer-Lemeshow test, or observed vs. predicted event rates).

- Language and timeframe: full-text articles published in English between 2000 and 2025.

Exclusion Criteria

- Studies that did not propose or evaluate a prognostic model (e.g., analyses of individual risk factors only).

- Non-ACS populations (e.g., stable coronary artery disease or heart failure without ACS).

- Animal studies, case reports, editorials, narrative commentaries without original data, and conference abstracts lacking sufficient methodological or performance details.

- Models derived solely from imaging or invasive physiology without reporting MACE outcomes.

Study selection and data extraction

Titles and abstracts identified through the search were screened to remove clearly irrelevant articles. Full texts of potentially eligible studies were then reviewed to confirm eligibility according to the predefined criteria. For each included study, we extracted:

- Study setting and population (e.g., STEMI, NSTEMI, overall ACS, post-PCI cohorts, national registries).

- Model type (clinical risk score, biomarker-integrated model, nomogram, or ML/AI-based approach).

- Predictor variables included in the final model.

- Outcome definitions and prediction horizons (e.g., in-hospital, 30-day, 6-month, 1- or 2-year MACE).

- Performance metrics (discrimination, calibration, and, where reported, net reclassification improvement [NRI] or integrated discrimination improvement [IDI]).

- Information on internal and external validation and, when available, clinical utility analyses (e.g., decision-curve analysis).

Given the narrative design, we synthesized findings qualitatively, grouping models into four broad categories (classical clinical scores, biomarker-based models, nomogram tools, and ML/AI-based models) and highlighting key sources of heterogeneity that influence comparison and potential implementation.

2. Results

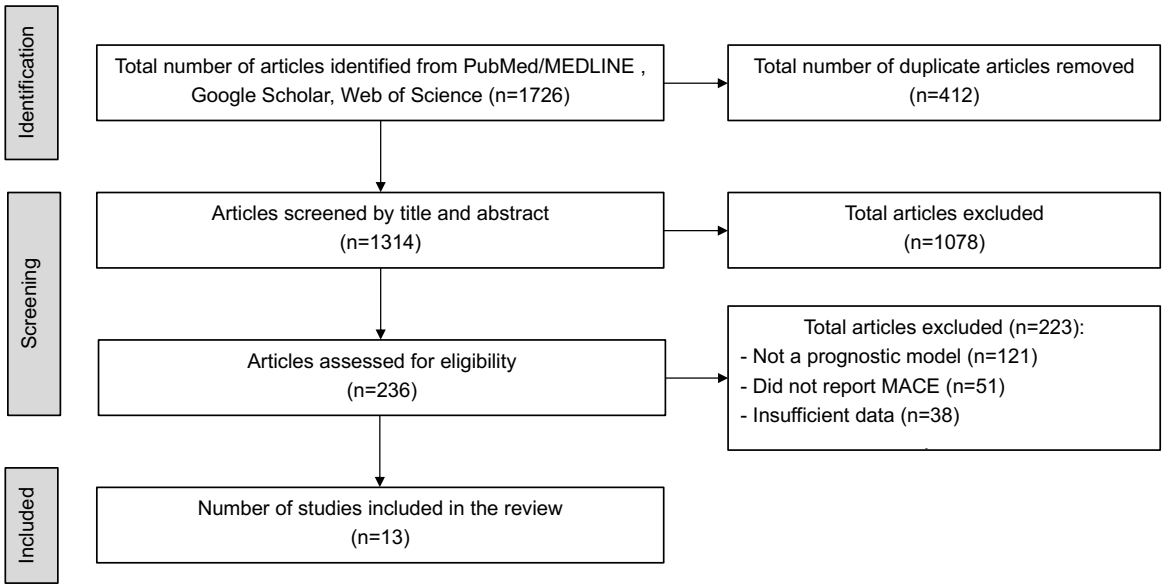

Figure 1. Flow diagram of study identification and selection

Clinical Risk Scores

Classical clinical risk scores remain the foundation of prognostic assessment in ACS. As summarized in Table 1, the TIMI score provides simple bedside risk stratification but only moderate discrimination (C-statistic =0.65) for short-term composite outcomes. In contrast, GRACE and GRACE 2.0, derived from large multinational registries, consistently show higher accuracy for in-hospital and post-discharge mortality (C ≈0.75-0.81) and are recommended as reference tools in current guidelines.[5,6] Post-PCI scores such as CADILLAC, PAMI, Zwolle, ACTION and CAMI generally achieve C-statistics in the 0.80-0.90 range for 30-day and 1-year mortality in selected STEMI or registry cohorts, but their applicability is often restricted to specific interventional or regional settings.

Table 1. Classical clinical risk scores and biomarker-based prognostic models for major adverse cardiovascular events in acute coronary syndrome

Model/study		Description
TIMI (Thrombolysis In Myocardial Infarction) risk score[8]	Population	Patients with NSTEMI/unstable angina from the TIMI 11B and ESSENCE trials.
	Outcome	Composite of death, myocardial infarction, or severe ischemia requiring urgent revascularization; 14-day follow-up.
	Key predictors	Age ≥65 years old; ≥3 coronary risk factors; prior stenosis ≥50%; aspirin use within the previous 7 days; ≥2 anginal episodes within 24 hours; ST-segment deviation; elevated cardiac biomarkers.
	Performance	C-statistic =0.65; event rates rising from 5% (score 0-1) to >40% (score 6-7).

Model/study		Description
GRACE / GRACE 2.0 (Global Registry of Acute Coronary Events)[5,6,9]	Population	All patients with ACS (STEMI, NSTEMI, unstable angina) from the GRACE registry; GRACE 2.0 updated using contemporary cohorts.
	Outcome	In-hospital and 6-month mortality; some studies additionally report MI or composite MACE.
	Key predictors	Age; blood pressure; heart rate; Killip class; cardiac arrest; troponin; creatinine; ST-segment deviation (GRACE 2.0 incorporates hs-troponin and updated coefficients).
	Performance	C-statistic =0.81 in derivation and =0.75 in external validation; consistently higher accuracy than TIMI in comparative analyses.
CADILLAC / PAMI / Zwolle Scores[10]	Population	Patients with STEMI undergoing primary or rescue PCI.
	Outcome	30-day and 1-year mortality following PCI.
	Key predictors	Age; LVEF; Killip class; creatinine; peripheral arterial disease; multivessel disease; stent failure (CADILLAC); additional clinical and angiographic variables in PAMI and Zwolle.
	Performance	C-statistic =0.80-0.90 for 30-day and 1-year mortality; comparable to or slightly lower than GRACE in certain comparisons.
NCDR ACTION Risk Score[11]	Population	ACS patients in the ACTION Registry-GWTG with an external validation cohort in China.
	Outcome	In-hospital mortality.
	Key predictors	Age; hemodynamic variables; heart failure; renal dysfunction; heart rate; blood pressure; ST-segment deviation; additional clinical factors.
	Performance	C-statistic 0.945 for in-hospital mortality in the Chinese cohort; higher than GRACE, GRACE 2.0, TIMI, and CPACS (GRACE =0.92; TIMI =0.81; CPACS =0.84).
CPACS Score[11]	Population	ACS patients from the CPACS program (China).
	Outcome	In-hospital mortality or short-term MACE.
	Key predictors	Clinical variables at presentation (age, vital signs, clinical status).
	Performance	C-statistic =0.84; does not outperform GRACE in comparative analyses.

Model/study		Description
CAMI / SCAMI-NSTEMI Scores[7]	Population	Patients with MI from the CAMI registry; SCAMI-NSTEMI for the NSTEMI subgroup.
	Outcome	In-hospital mortality.
	Key predictors	Age; sex; blood pressure; heart rate; creatinine; hematologic/electrolyte markers; Killip class; ST-segment deviation; medical history.
	Performance	AUC =0.83-0.84; well-suited to Asian populations.
ABC-ACS Model[13]	Population	Patients with ACS from the PLATO and TRACER trials after PCI.
	Outcome	Cardiovascular death or MI at 1 year; composite ischemic outcomes.
	Key predictors	Age; NT-proBNP; GDF-15; extent of CAD; vascular disease history; Killip class; ACS type; P2Y12 inhibitor use.
	Performance	C-statistic =0.72 in derivation and validation; outperforms GRACE 2.0 for certain endpoints.
BIPass Model[14]	Population	ACS patients from the BIPass registry (derivation n=4407; validation n=1409).
	Outcome	12-month MACE (death, MI, stroke, or revascularization).
	Key predictors	Age; hypertension; prior MI; prior stroke; Killip class; heart rate; NT-proBNP.
	Performance	C-statistic 0.79 (95% CI: 0.73-0.85); superior discrimination to GRACE and TIMI in the same cohort; good calibration and clear risk stratification.

Prognostic models based on risk factors and laboratory parameters

Beyond classical scores, several models have incorporated more detailed clinical variables, procedural factors and laboratory parameters (Table 1). The model by Kassaian et al. combined diabetes, renal dysfunction, reduced left ventricular ejection fraction, ACS presentation and multivessel disease with PCI-related variables to predict 1-year MACE after PCI, but achieved only moderate discrimination (C-statistic 0.63).[9]

In contrast, biomarker-integrated tools such as ABC-ACS and BIPass explicitly include NT-proBNP, GDF-15 and other prognostic markers on top of clinical predictors.[10,11] In trial-based cohorts and the BIPass registry, these models reached C-statistics in the upper 0.7 range and showed better discrimination than GRACE and TIMI in some comparisons, with acceptable calibration and clearer separation across risk strata. However, most evaluations have been conducted in specific trial or registry populations, and wider external validation remains limited.

Nomogram and machine learning models

Several recent studies have proposed nomogram-based tools to facilitate individualized risk estimation after ACS (Table 2). A non-invasive nomogram by Wang et al. combined autonomic indices (LF/HF ratio) with age, diabetes, prior myocardial infarction and smoking to predict 1- and 2-year MACE-free survival, achieving AUCs of 0.79-0.89 with good calibration and clear risk stratification (Figure 2).[15] Other nomograms incorporating the HALP index, soluble ST2 or blood lactate in elderly NSTEMI patients or ACS-PCI cohorts reported AUCs around 0.75-0.80 and modest improvements over traditional scores, but remain based on relatively small, single-center samples.[16]

In parallel, ML-based approaches have been developed in large emergency department chest-pain cohorts, using high-dimensional clinical, ECG and serial high-sensitivity troponin data (Table 2).[4] Deep neural networks achieved AUCs up to 0.93 for 30-day mortality and 0.97 for cardiac arrest, while recurrent models such as Long Short-Term Memory networks showed high discrimination for 30-day MACE using time-series biomarker and ECG trajectories. Although these findings highlight the potential of ML for dynamic prediction, external validation, calibration and clinical utility analyses are still scarce.

Table 2. Nomogram and machine learning models for predicting major adverse cardiovascular events in patients with acute coronary syndrome

Model/study		Description
Non-invasive Autonomic Nervous System Nomogram[15]	Population	ACS patients assessed for autonomic nervous system function; Chinese cohort.
	Outcome	MACE-free survival at 1 year and 2 years.
	Key predictors	LF/HF ratio; age; diabetes; prior myocardial infarction; smoking.
	Performance	AUC 0.79 (derivation) and 0.89 (validation) for 1-year MACE; approximately 0.80 for 2-year prediction; good calibration and clear separation of low-, intermediate-, and high-risk groups.
Nomogram for Elderly NSTEMI Patients After PCI[16]	Population	Elderly patients with NSTEMI following PCI.
	Outcome	1-year MACE.
	Key predictors	HALP index; soluble ST2 (sST2); conventional clinical variables.
	Performance	AUC in the moderate range (~0.75-0.80); modest improvement over traditional scores; limited sample size and requires further external validation.

Model/study		Description
Lactate-Based Nomogram[4]	Population	ACS patients undergoing PCI with measured blood lactate levels.
	Outcome	MACE at 6 months, 1 year, and long-term follow-up.
	Key predictors	Blood lactate concentration combined with clinical variables and coronary angiographic characteristics.
	Performance	AUC typically 0.75-0.80; improved sensitivity in certain risk subgroups; external validation still needed.
ML Models for Emergency Department Chest Pain Patients[4]	Population	Emergency department patients presenting with chest pain and suspected cardiac disease; findings summarized from a scoping review of ML models.
	Outcome	30-day MACE (death, myocardial infarction, cardiac arrest).
	Key predictors	Demographics; vital signs; ECG; cardiac biomarkers (including time-series hs-troponin); comorbidities; additional EHR-derived variables.
	Performance	Deep neural network achieved AUC =0.93 for 30-day mortality and =0.97 for cardiac arrest; logistic regression performed lower (=0.89) within the same dataset.
Deep Learning LSTM Models Using Time-Series Data[4]	Population	Patients with available time-series data (serial troponin; repeated or continuous ECG).
	Outcome	30-day MACE predicted from biomarker kinetics and ECG dynamics.
	Key predictors	Serial hs-troponin measurements; ECG data; additional clinical covariates.
	Performance	High discriminative ability for short-term MACE (AUC often >0.85) in internal validation; no long-term validation yet and not implemented in routine clinical practice.

In Figure 2, the non-invasive autonomic nomogram proposed by Wang et al. assigns points to LF/HF ratio, age, diabetes, prior myocardial infarction and smoking, with the total score mapped to 1- and 2-year MACE-free survival.[15]

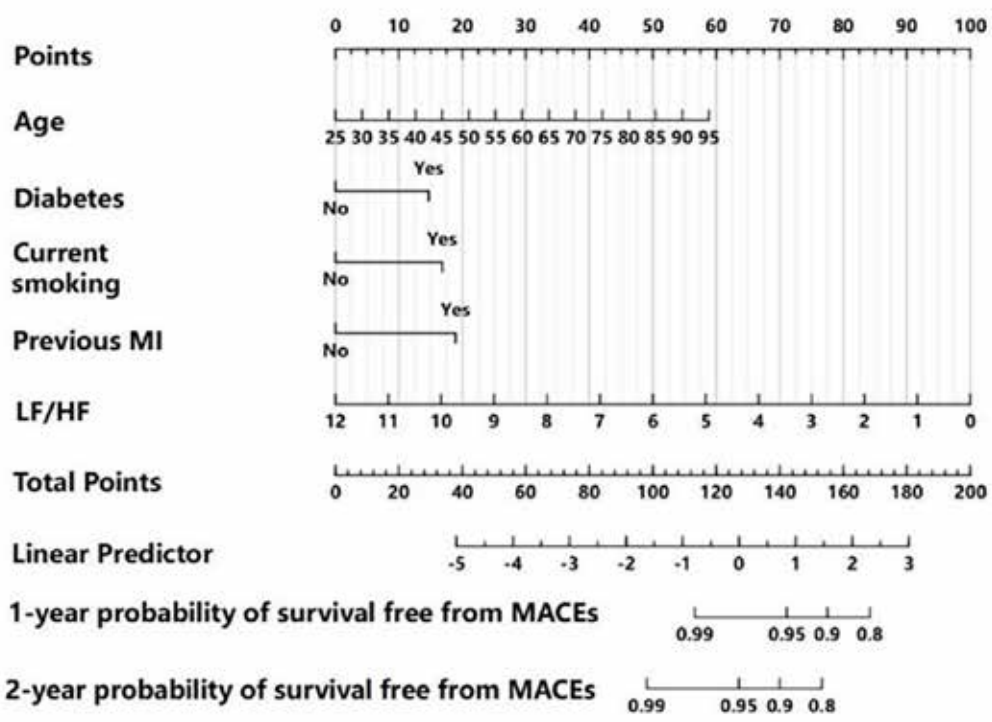

Figure 2. Nomogram model predicting 1- and 2-year MACE-free survival in patients with ACS[15]

Figure 3 shows a similar point-based nomogram integrating the HALP index, soluble ST2 and conventional clinical factors to estimate 1-year MACE risk in elderly NSTEMI patients after PCI.[16]

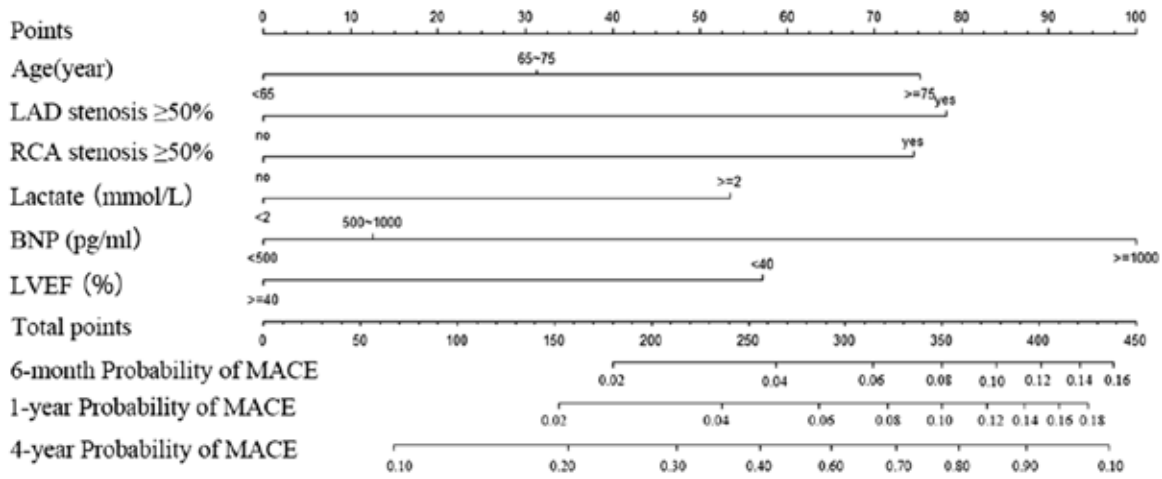

Figure 3. Nomogram for predicting MACEs in patients with ACS after PCI[16]

3. Discussion

This narrative review summarizes the evolution of prognostic models for major adverse cardiovascular events in patients with acute coronary syndrome, spanning classical scores, biomarker-integrated tools, nomograms, and ML-based approaches. Across these categories, we observed substantial variability in model design, target populations, outcome definitions, and prediction horizons, which complicates direct comparison and has important implications for clinical implementation.

Clinical risk scores such as TIMI and GRACE remain the foundation of ACS risk stratification.[8,9] TIMI offers simplicity and ease of bedside use but provides only moderate discrimination for short-term composite outcomes. In contrast, GRACE and its updated version, GRACE 2.0, consistently demonstrate higher accuracy for both in-hospital and post-discharge mortality and MACE, and they are endorsed by major guidelines as reference tools for non-ST-elevation ACS.[5,6] In comparative analyses, GRACE often outperforms TIMI and other STEMI-focused scores, although specialized scores such as CADILLAC, PAMI, Zwolle, ACTION, and CAMI may provide superior discrimination in narrowly defined settings such as primary PCI cohorts or registry-based STEMI populations.[7,10,11] These specialized models, however, are less generalizable outside their derivation context and may exhibit suboptimal calibration in very high-risk groups, as illustrated by the underestimation of mortality in the highest ACTION risk strata.[11] Overall, the evidence suggests that GRACE 2.0 currently offers the best balance between discrimination, calibration, and applicability across a broad ACS population, while other scores may be considered in selected post-PCI or STEMI scenarios.

Biomarker-integrated models represent an important step toward more biologically informed risk prediction. NT-proBNP, GDF-15, and other biomarkers reflecting myocardial stretch, inflammation, and cellular stress have shown incremental prognostic value over clinical variables alone.[12,13,14] The ABC-ACS and BIPass models exemplify this approach by combining biomarkers with established clinical predictors and achieving C-statistics in the upper 0.7 range.[13,14] However, the magnitude of improvement over GRACE 2.0 is modest rather than transformative, and external validation remains limited. Biomarker-based models also depend on assay standardization, timing of blood sampling, and the influence of comorbidities such as renal dysfunction, which may attenuate their performance in routine practice. Moreover, relatively few biomarker studies have reported calibration metrics, reclassification indices (NRI, IDI), or decision-curve analyses, making it difficult to determine whether statistically significant gains translate into clinically meaningful net benefit.

Nomogram-based tools have gained popularity in recent years, particularly in Asian cohorts, as they offer an intuitive graphical representation of multivariable models and facilitate individualized risk estimation.[15,16] Many nomogram models integrate conventional risk factors with emerging indices, such as heart rate variability or composite inflammatory scores, and report AUCs close to or exceeding 0.80 for intermediate- to long-term MACE prediction. Nevertheless, most nomograms arise from single-center or relatively small datasets with limited external validation, raising concerns about overfitting and transportability. When applied in different hospitals, health systems, or ethnic groups, these models may

require recalibration and re-estimation of baseline risk to maintain accuracy and avoid misclassification.

ML- and AI-based models constitute the most recent wave of innovation in ACS prognostication.[4] Deep neural networks, random forests, and recurrent architectures such as long short-term memory networks can exploit high-dimensional clinical and time-series data (e.g., serial troponin measurements, continuous ECG monitoring) and often achieve impressive discrimination, with AUCs frequently above 0.90 in derivation and internal validation datasets.[4] However, these results should be interpreted with caution. Many ML studies rely on large single-system cohorts, use complex feature engineering, and do not consistently report calibration, external validation, or robustness analyses. Overfitting to local practice patterns, temporal shifts in treatment, and unmeasured confounding may all undermine the apparent superiority of ML models when they are transported to new settings. In addition, limited interpretability and the absence of user-friendly interfaces can impede clinical acceptance and regulatory approval. To move beyond proof-of-concept, ML models will need transparent reporting, external validation across diverse healthcare systems, and evaluation of clinical utility using frameworks such as decision-curve analysis.

The heterogeneity in MACE definitions, prediction horizons, and patient populations across the included studies deserves particular emphasis. Some models target in-hospital events, whereas others focus on 30-day, 6-month, or 1-2-year outcomes; MACE composites also vary in whether they include heart failure, repeat revascularization, or hospitalization. This variability partly explains the wide range of reported AUCs and complicates head-to-head comparisons. From a clinical perspective, a model optimized for short-term intensive care triage may not be directly comparable to one designed for long-term secondary prevention, even if both are labeled as MACE prediction tools. Future model development and validation efforts should more clearly specify the intended use case, time horizon, and outcome definition, and adhere to established reporting and risk-of-bias frameworks for prognostic models.

This narrative review has several limitations. First, although we employed a structured search strategy, we did not conduct a formal systematic review or meta-analysis, nor did we apply standardized tools such as PROBAST to assess risk of bias across prognostic models. As a result, our synthesis is qualitative and may not fully capture all relevant studies, particularly those published outside major databases. Second, the included studies differed substantially in patient populations, outcome definitions, and follow-up durations; these sources of heterogeneity limit direct comparison of performance metrics and may partly account for the wide variation in reported AUCs. Finally, we relied on performance measures as reported by the original authors; incomplete reporting of calibration metrics, reclassification indices, and clinical utility made detailed comparison between models challenging.

III. CONCLUSION

Prognostic models for major adverse cardiovascular events in patients with acute coronary syndrome have evolved from simple clinical risk scores to biomarker-integrated and artificial intelligence-based models. Among these, GRACE 2.0 still appears to be the most commonly recommended tool for risk stratification, largely because of its relatively stable predictive accuracy and acceptable calibration in multiple studies. Post-intervention models such as CADILLAC, ACTION, and

CAMI, as well as models incorporating NT-proBNP, have shown comparable or, in some settings, better performance in specific populations. Nomogram-based models may facilitate visual representation of individualized risk, while machine learning approaches appear promising for dynamic prediction and more personalized management, but further validation and implementation studies are still needed.

REFERENCES

1. Timmis A, Kazakiewicz D, Townsend N, Huculeci R, Aboyans V, Vardas P. Global epidemiology of acute coronary syndromes. *Nat Rev Cardiol*. 2023; 20(11): 778-788. doi:10.1038/s41569-023-00884-0.

2. Zhu XY, Jiang ZM, Li X, Su FF, Tian JW. Establishment and validation of post-PCI nomogram in elderly patients with acute coronary syndromes. *Front Cardiovasc Med*. 2025;12:1529476. Published 2025 Mar 7. doi:10.3389/fcvm.2025.1529476.

3. Abu-Assi E, López-López A, González-Salvado V, et al. The Risk of Cardiovascular Events After an Acute Coronary Event Remains High, Especially During the First Year, Despite Revascularization. *Rev Esp Cardiol (Engl Ed)*. 2016; 69(1): 11-18. doi:10.1016/j.rec.2015.06.015.

4. Chopannejad S, Sadoughi F, Bagherzadeh R, Shekarchi S. Predicting Major Adverse Cardiovascular Events in Acute Coronary Syndrome: A Scoping Review of Machine Learning Approaches. *Appl Clin Inform*. 2022; 13(3): 720-740. doi: https://doi.org/10.1055/a-1863-1589.

5. Byrne RA, Rossello X, Coughlan JJ, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. *Eur Heart J*. 2023; 44(38): 3720-3826. doi: https://doi.org/10.1093/eurheartj/ehad191.

6. Rao SV, O'Donoghue ML, Ruel M, et al. 2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*. 2025; 151(13): e771-e862. doi: https://doi.org/10.1161/CIR.0000000000001309.

7. Song, C., Fu, R., Dou, K. et al. The CAMI-score: A Novel Tool derived From CAMI Registry to Predict In-hospital Death among Acute Myocardial Infarction Patients. *Sci Rep* 8, 9082 (2018). https://doi.org/10.1038/s41598-018-26861-z.

8. Antman EM, Cohen M, Bernink PJ, et al. The TIMI risk score for unstable angina/non-ST elevation MI: A method for prognostication and therapeutic decision making. *JAMA*. 2000; 284(7): 835-842. doi: https://doi.org/10.1001/jama.284.7.835.

9. Eagle KA, Lim MJ, Dabbous OH, et al. 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(22): 2727-2733. doi: https://doi.org/10.1001/jama.291.22.2727.

10. Méndez-Eirín E, Flores-Ríos X, García-López F, et al. Comparison of the prognostic predictive value of the TIMI, PAMI, CADILLAC, and GRACE risk scores in STEACS undergoing primary or rescue PCI. *Rev Esp Cardiol (Engl Ed)*. 2012; 65(3): 227-233. doi: https://doi.org/10.1016/j.recesp.2011.10.019.

11. Bai L, Li YM, Yang BS, et al. Performance of the Risk Scores for Predicting In-Hospital Mortality in Patients with Acute Coronary Syndrome in a Chinese Cohort. *Rev Cardiovasc Med*. 2023; 24(12): 356. Published 2023 Dec 19. doi: https://doi.org/10.31083/j.rcm2412356.

12. Kassaian SE, Saroukhani S, Alaeddini

F, et al. A Risk-Scoring Model to Predict One-year Major Adverse Cardiac Events after Percutaneous Coronary Intervention. *J Tehran Heart Cent*. 2015; 10(4): 167-175.

13. Batra G, Lindbäck J, Becker RC, et al. Biomarker-Based Prediction of Recurrent Ischemic Events in Patients With Acute Coronary Syndromes. *J Am Coll Cardiol*. 2022; 80(18): 1735-1747. doi: https://doi.org/10.1016/j.jacc.2022.08.767.

14. Wang J, Gao W, Chen G, et al. Biomarker-based risk model to predict cardiovascular events in patients with acute coronary syndromes - Results from BIPass registry. *Lancet Reg Health West Pac*. 2022; 25:100479. Published 2022 May 30. doi: https://doi.org/10.1016/j.lanwpc.2022.100479.

15. Wang J, Wu X, Sun J, et al. Prediction of major adverse cardiovascular events in patients with acute coronary syndrome: Development and validation of a non-invasive nomogram model based on autonomic nervous system assessment. *Front Cardiovasc Med*. 2022;9:1053470. Published 2022 Nov 3. doi: https://doi.org/10.3389/fcvm.2022.1053470.

16. Kong, S., Chen, C., Zheng, G. *et al.* A prognostic nomogram for long-term major adverse cardiovascular events in patients with acute coronary syndrome after percutaneous coronary intervention. *BMC Cardiovasc Disord* 21, 253 (2021). https://doi.org/10.1186/s12872-021-02051-0.