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ORIGINAL RESEARCH

ECG-Guided Antiarrhythmic Therapy in Acute Coronary Syndrome: A Multidimensional Assessment of Biomarkers, Cardiac Function, and Quality of Life

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Objective: To evaluate the impact of electrocardiogram (ECG)-guided antiarrhythmic strategy on serum biomarkers, cardiac function, and quality of life in patients with acute coronary syndrome (ACS) and arrhythmias.

Methods: This prospective observational cohort enrolled 80 ACS patients with arrhythmias managed via a monitor-alert-response-titration pathway (observation group) during January-December 2024. Propensity score-matched controls (n = 80) received conventional experience-based antiarrhythmic therapy. Comparisons included in-hospital antiarrhythmic interventions, arrhythmia control, adverse events, and myocardial injury/stress biomarkers (NT-proBNP, hs-cTnI, hs-cTnT, hs-CRP) at T0 (baseline), T1 (2-day), and T2 (7-day). Cardiac function (LVEF, LVEDD, LVEDVI, E/e′) and patient-reported outcomes [Seattle Angina Questionnaire (SAQ), 6-minute walk test (6MWT)] were assessed at T0, T3 (1-month), and T4 (6-month). Major adverse cardiovascular events (MACE) were recorded over 6 months.

Results: The observation group demonstrated higher β-blocker utilization, guideline adherence, and drug adjustment rates ($P < 0.05$). Arrhythmia control was superior in the observation group at T2 (93.75% vs 82.50%, $P < 0.05$). More pronounced reductions in all biomarkers were observed in the observation group at T1 and T2 ($P < 0.05$). Greater improvements in LVEF, LVEDD, LVEDVI, E/e′, SAQ scores, and 6MWT distance were sustained in the observation group at T3 and T4 ($P < 0.05$). The 6-month MACE rate was lower in the observation group (13.75% vs 32.50%, $P < 0.05$).

Conclusion: ECG-guided antiarrhythmic therapy improves pharmacotherapy quality and arrhythmia control, accelerating myocardial injury and stress attenuation. This strategy approach enhances cardiac recovery, reverse remodeling, functional capacity, and quality of life, ultimately reducing long-term cardiovascular risk.

Keywords: acute coronary syndrome, ECG-guided antiarrhythmic therapy, cardiac function, biomarkers, quality of life

Introduction

Acute coronary syndrome (ACS) constitutes a clinical spectrum of acute myocardial ischemia resulting from coronary atherosclerotic plaque rupture or erosion, with subsequent thrombus formation or severe vasospasm. This entity encompasses ST-segment elevation myocardial infarction (STEMI), non-STEMI (NSTEMI), and unstable angina (UA). The quality of early risk stratification and comprehensive management directly influences short-term mortality,

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heart failure incidence, and the long-term risk of major adverse cardiovascular events (MACE).[1–3] The 2023 ESC guidelines for ACS management underscore that structured care pathways from the pre-hospital to in-hospital phase, coupled with standardized pharmacological and revascularization strategies and evidence-based secondary prevention, are pivotal for further reducing mortality and complications.[1] Pathophysiologically, the *Fourth Universal Definition of MI* establishes the central diagnostic role of a rise and/or fall in cardiac troponin (cTn) values, especially high-sensitivity cTn (hs-cTn), in conjunction with clinical evidence of MI. It also emphasizes the critical need to differentiate MI from non-ischemic myocardial injury to prevent undertreatment and overtreatment.[4] This definition provides a unified framework for patient enrollment, endpoint definitions, and biomarker interpretation in clinical research.

Cardiac arrhythmias are a frequent complication in the acute phase of ACS. Sympathetic activation, electrophysiological heterogeneity induced by ischemia-reperfusion injury, and electrolyte imbalances can precipitate both atrial and ventricular arrhythmias, which in severe cases may lead to hemodynamic instability or even sudden cardiac death.[5–7] The 2023 AHA/ACC/HRS guideline for the management of ventricular arrhythmias and subsequent evidence highlight the central role of β-blockers in attenuating sympathetic drive, reducing myocardial oxygen demand, and decreasing the incidence of life-threatening ventricular arrhythmias.[8] However, the benefit of long-term routine β-blocker therapy in revascularized patients with preserved left ventricular ejection fraction (LVEF) remains a subject of debate,[9–12] necessitating management strategies optimized through individualized risk assessment and monitoring. Beyond reperfusion and antithrombotic therapy, a trend towards more meticulous monitor-guided care is gaining prominence. Continuous electrocardiographic (ECG) monitoring and mobile cardiac telemetry enhance arrhythmia detection rates, shorten intervention delays, and provide real-time guidance for drug titration and non-pharmacological interventions (eg, cardioversion/defibrillation, pacing, ablation), potentially translating into improved hard outcomes.[13–15] Although previous studies indicate a decline in the incidence of malignant arrhythmias in the contemporary era of intensive monitoring,[16,17] a standardized paradigm for converting monitoring signals into actionable therapeutic decisions, through structured alarm-response pathways, remains elusive, lacking robust prospective evidence. In routine care, arrhythmia management in ACS often remains experience-based, which may introduce inter-clinician variability, delayed responses to monitor alerts, and therapeutic uncertainty due to both overtreatment and undertreatment. Accordingly, this study focused on clinically significant in-hospital arrhythmias detected by continuous ECG monitoring, primarily premature ventricular complexes (PVCs), non-sustained ventricular tachycardia (NSVT), and new-onset atrial fibrillation/atrial flutter (AF/AFL).

Multidimensional outcome assessment further reflects the shift towards patient-centered care in ACS.[18] In addition to hard endpoints such as mortality and reinfarction, biomarkers including N-terminal pro-B-type natriuretic peptide (NT-proBNP), hs-cTn, and high-sensitivity C-reactive protein (hs-CRP) provide insights into the extent of myocardial injury, hemodynamic stress, and residual inflammatory risk.[19] Echocardiographic parameters, such as LVEF, left ventricular end-diastolic diameter/volume index (LVEDD/VI), and the ratio of early mitral inflow velocity to early diastolic mitral annular velocity (E/e′), characterize cardiac remodeling and diastolic function.[20] Meanwhile, disease-specific quality of life instruments like the Seattle Angina Questionnaire (SAQ) and functional capacity assessments such as the 6-minute walk test (6MWT) quantify functional status and patient-reported outcomes, having demonstrated good reliability and prognostic value in populations with coronary artery disease and cardiac rehabilitation.[21]

Against this backdrop, the present study aims to develop and evaluate a clinical pathway integrating ECG monitor alert, stratified response, and individualized antiarrhythmic drug titration within the framework of standard ACS care (Figure 1). We will systematically compare this protocol-driven approach against conventional, experience-based management, assessing its impact on arrhythmia control, myocardial injury/inflammatory burden, cardiac functional remodeling, quality of life, and medium-term MACE. The findings are expected to provide prospective evidence to inform structured and precision management strategies for ACS patients with concomitant arrhythmias.

Materials and Methods

Patient Characteristics

Based on previous data, the incidence of 6-month MACE in conventionally managed patients was approximately 30%.[22] It was projected that the implementation of the monitor-response-titration pathway could reduce this incidence to 15%-

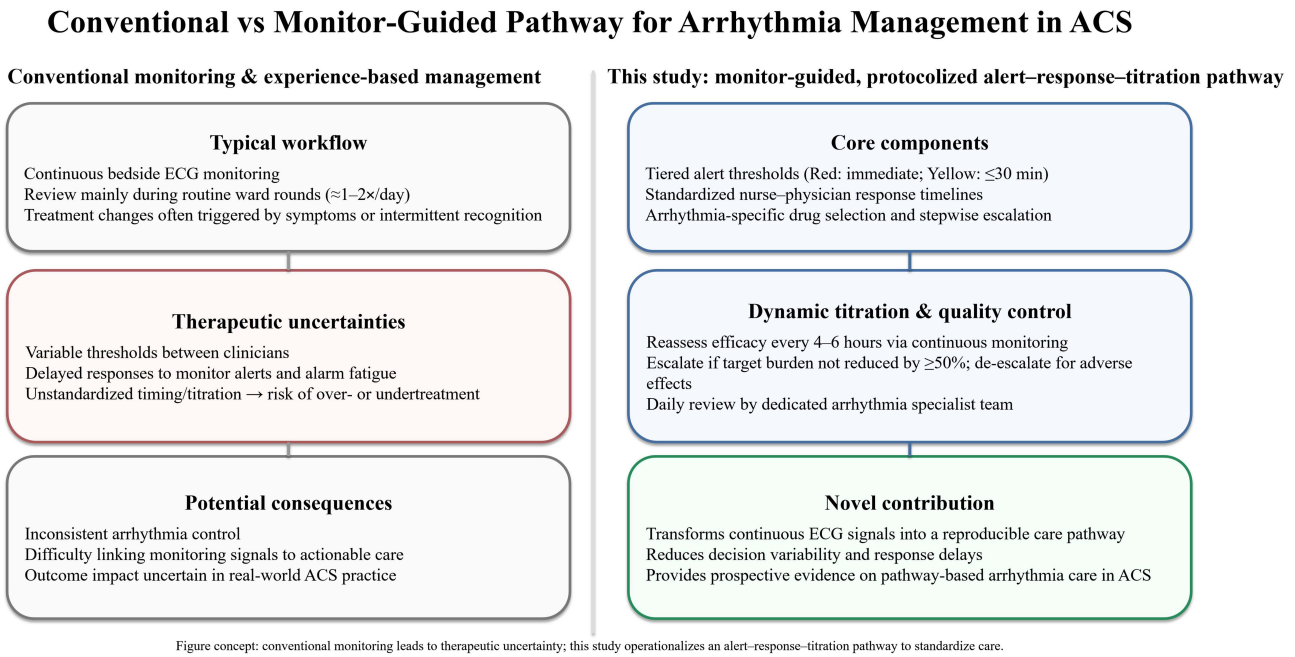

Figure 1 Conventional vs Monitor-Guided Pathway for Arrhythmia Management in ACS.

20%. To adopt a conservative and realistic estimate, sample size calculation and scenario verification were performed assuming proportions of $p_1 = 0.325$ (control group) and $p_2 = 0.1375$ (observation group). Using the formula for comparing two independent proportions (two-sided $\alpha = 0.05$, $1\text{-}\beta = 0.80$), the required sample size was approximately $n \approx 78$ per group (total ≈ 156). Accounting for an anticipated 5%–10% loss to follow-up or incomplete data, a total sample size of 160 patients was targeted. The final study included 80 patients per group, meeting the pre-specified power requirement. This was a prospective observational cohort study without researcher intervention. From January to December 2024, an individualized antiarrhythmic management pathway based on ECG monitor alerts and structured response was implemented as an in-hospital quality improvement initiative. Patients with ACS and clinically significant arrhythmias who were managed according to this pathway during this period constituted the exposed cohort, or the observation group (n = 80). A control cohort (n = 80) was then established by performing 1: 1 propensity score matching (caliper width = 0.2 × standard deviation of the logit of the propensity score) from a pool of contemporaneous ACS patients with arrhythmias who received conventional management but were not enrolled in the pathway (Figure 2). Matching was based on age, sex, ACS type, Global Registry of Acute Coronary Events (GRACE) risk score at admission, LVEF, peak hs-cTn level, and comorbidities to minimize selection bias. No statistically significant differences were observed in baseline characteristics between the two groups ($P > 0.05$ for all), confirming their comparability (Table 1).

Inclusion Criteria

Patients were required to meet the following criteria: 1) diagnosis of ACS according to established criteria,[23] confirmed by ECG findings, coronary angiography, or dynamic evolution of myocardial injury biomarkers; 2) presence of clinically significant arrhythmia, verified by continuous cardiac monitoring, 12-lead ECG, or 24-hour ambulatory ECG monitoring after admission; 3) first-onset ACS or hospitalization for an acute exacerbation of pre-existing coronary artery disease; 4) presentation to the hospital within 24 hours of ACS symptom onset (≤ 12 hours for STEMI patients); 5) an expected survival of ≥ 6 months; 6) age between 18 and 80 years; and 7) provision of written informed consent by patients or their legally authorized representative, following approval of the study protocol by the hospital's Ethics Committee.

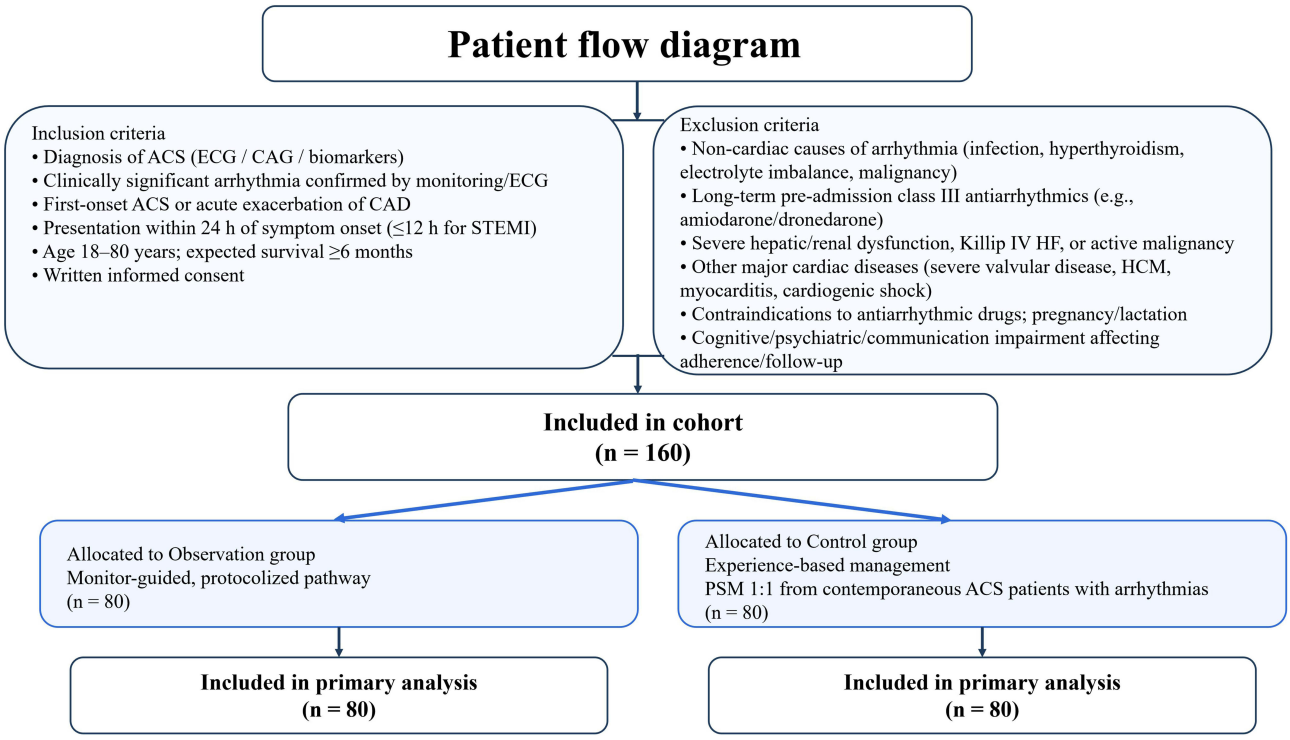

Figure 2 Patient flow diagram.

Exclusion Criteria

Exclusion criteria comprised the following: 1) arrhythmias secondary to non-cardiac conditions, such as severe infection, hyperthyroidism, electrolyte imbalances, or malignant tumors; 2) pre-admission long-term use of class III antiarrhythmic agents (eg, amiodarone, dronedarone); 3) concomitant severe hepatic or renal dysfunction, Killip class IV heart failure, or active malignancy; 4) presence of other significant cardiac diseases, including severe valvular heart disease, obstructive hypertrophic cardiomyopathy, acute myocarditis, or cardiogenic shock; 5) contraindications to antiarrhythmic drug therapy, such as sick sinus syndrome without a pacemaker, second-degree or higher atrioventricular block, long QT

Table 1 Comparison of Baseline Characteristics

Characteristic	Observation Group (n = 80)	Control Group (n = 80)	χ^2/t/Z	p
Sex			0.413	0.521
Male	45 (56.25)	49 (61.25)	–	–
Female	35 (43.75)	31 (38.75)	–	–
Age (years)	61.90 ± 8.93	62.49 ± 8.99	0.415	0.679
BMI (kg/m^2)	25.09 ± 3.29	24.86 ± 3.34	0.449	0.654
Cardiovascular risk factors				
Hypertension	51 (63.75)	52 (65.00)	0.027	0.869
Diabetes mellitus	34 (42.50)	29 (36.25)	0.655	0.418
Dyslipidemia	41 (51.25)	46 (57.50)	0.630	0.427
Previous MI	12 (15.00)	10 (12.50)	0.211	0.646
ACS type			0.689	0.708
STEMI	39 (48.75)	34 (42.50)	–	–
NSTEMI	26 (32.50)	28 (35.00)	–	–
UA	15 (18.75)	18 (22.50)	–	–

(*Continued*)

Table 1 (Continued).

Characteristic	Observation Group (n = 80)	Control Group (n = 80)	$\chi^2/t/Z$	*p*
Arrhythmia type			0.990	0.827
PVC	44 (55.00)	46 (57.50)	–	–
NSVT	23 (28.75)	20 (25.00)	–	–
AF/AFL	11 (13.75)	10 (12.50)	–	–
Others	2 (2.50)	4 (5.00)	–	–
Heart rate (beats/min)	78.61 ± 13.14	77.23 ± 13.96	0.647	0.518
Systolic blood pressure (mmHg)	136.24 ± 19.45	134.64 ± 20.11	0.512	0.610
Diastolic blood pressure (mmHg)	83.30 ± 11.43	82.75 ± 12.36	0.292	0.771
Serum creatinine (mg/dL)	0.94 (0.68, 1.22)	0.92 (0.64, 1.21)	0.459	0.646
Killip class			0.428	0.807
I	48 (60.00)	52 (65.00)	–	–
II	25 (31.25)	22 (27.50)	–	–
III	7 (8.75)	6 (7.50)	–	–
ST-segment deviation	65 (81.25)	62 (77.50)	0.344	0.558
Elevated cardiac enzymes	73 (91.25)	75 (93.75)	0.360	0.548
GRACE risk score	144.45±25.96	143.31±25.01	0.282	0.778
ACS management strategy			0.573	0.449
Pharmacological therapy	16 (20.00)	20 (25.00)	-	-
PCI	64 (80.00)	60 (75.00)	-	-

Abbreviations: BMI, Body mass index; ACS, Acute coronary syndrome; MI, Myocardial ischemia; STEMI, ST-segment elevation myocardial ischemia; NSTEMI, Non-ST-segment elevation myocardial ischemia; UA, Unstable angina; PVC, Premature ventricular complexes; NSVT, Non-sustained ventricular tachycardia; AF/AFL, Atrial fibrillation/Atrial flutter; GRACE, Global Registry of Acute Coronary Events; PCI, Percutaneous coronary intervention.

syndrome, or asthma; 6) pregnancy or lactation; and 7) cognitive, psychiatric, or communication impairments that would preclude adherence to treatment, completion of questionnaires, or follow-up assessments.

Study Protocols

Shared Management Measures

Both groups received identical foundational ACS management. This included continuous ECG monitoring for all patients upon admission (using the Philips IntelliVue MX800 system), revascularization decisions strictly guided by current guidelines,[1] prompt correction of electrolyte imbalances (particularly serum potassium and magnesium), and regular assessment of antiarrhythmic drug safety, incorporating daily QT interval monitoring.

Control Group: Conventional, Experience-Based Management

Patients in this group received standard ACS pharmacotherapy, including dual antiplatelet therapy, anticoagulants, statins, β-blockers, and angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers (ACEI/ARB). Antiarrhythmic management was administered at the discretion of the attending physician, based on clinical experience. The specific approach involved: ① Review of arrhythmia data from continuous ECG monitoring during routine ward rounds (typically once or twice daily); ② Initiation, dose adjustment, or switching of antiarrhythmic drugs primarily triggered by patient-reported symptoms (eg, palpitations, dizziness) or significant arrhythmias identified during these routine checks; ③ Drug selection referenced the Chinese Expert Consensus on Clinical Use of Antiarrhythmic Drugs,[24] without a standardized protocol for timing or titration; ④ Emergency protocols were followed for hemodynamically unstable malignant arrhythmias.

Observation Group: Monitor-Guided, Protocolized Management

In addition to standard ACS care, this group received a structured, individualized antiarrhythmic strategy driven by a predefined clinical pathway for ECG monitor alerts: ① Structured alert pathway: A tiered alarm system was

established: Red alerts (immediate response) included sustained ventricular tachycardia (VT), ventricular fibrillation, polymorphic VT, and pauses > 3 seconds. Yellow alerts (response within 30 minutes) included non-sustained VT (NSVT, > 5 consecutive beats), frequent premature ventricular complexes (PVC; > 30/hour), and new-onset atrial fibrillation (AF) with rapid ventricular response (> 120 bpm). ② Standardized response protocol: For red alerts, nurses immediately summoned a physician for bedside evaluation, initiating an emergency management plan within 5 minutes. For yellow alerts, the responsible nurse documented and reported the event to a physician, who performed a clinical assessment and adjusted therapy within 30 minutes. ③ Individualized drug titration strategy: Antiarrhythmic agents were selected based on the predominant arrhythmia: β-blockers were first-line for ventricular arrhythmias, with a stepwise addition of amiodarone if response was inadequate. For AF/atrial flutter (AFL), rate control was prioritized using β-blockers or amiodarone, selected according to cardiac function. Therapy was dynamically adjusted based on continuous monitoring feedback. Efficacy was assessed every 4–6 hours, and treatment was escalated if the target arrhythmia burden failed to reduce by ≥ 50%. Therapy was de-escalated for adverse effects (eg, sinus bradycardia < 50 bpm, QTc interval > 500 ms). ④ Quality control: A dedicated cardiac arrhythmia specialist team reviewed all management daily to ensure strict adherence to the clinical pathway. Arrhythmia burden was quantified from continuous ECG monitor data as PVC frequency (events per hour), NSVT burden (number of NSVT episodes per 24 hours and longest run), and AF/AFL burden (cumulative duration and percentage of monitored time in AF/AFL); the ≥50% reduction criterion was calculated relative to the 24-hour baseline preceding treatment initiation. All arrhythmia alerts and classifications were centrally reviewed using stored rhythm strips by the dedicated arrhythmia specialist team, and ambiguous events were adjudicated by senior cardiologists until consensus was reached.

Outcome Measures

Antiarrhythmic Interventions During Hospitalization

The utilization of pharmacological and non-pharmacological interventions was compared between the two groups. For pharmacological interventions, parameters included the usage rates of β-blockers and amiodarone, the rate of combination antiarrhythmic therapy, guideline adherence rate (assessed for both drug selection and dosage), and the overall drug adjustment rate (encompassing changes in drug type or dosage due to insufficient efficacy, safety concerns, or empirical decisions). In non-pharmacological interventions, the application rates of the following procedures were recorded: electrical cardioversion/defibrillation, temporary cardiac pacing, radiofrequency catheter ablation (RFCA), implantable cardioverter-defibrillator (ICD) implantation, and cardiac resynchronization therapy (CRT).

Arrhythmia Control and Treatment-Related Adverse Events

Arrhythmia control rates were assessed at two time points during hospitalization: T1 (2 days post-treatment initiation) and T2 (7 days post-treatment initiation). The incidence of treatment-related adverse events was also monitored, categorized as: ① Hemodynamic abnormalities: Symptomatic bradycardia (heart rate < 50 bpm), atrioventricular block, and hypotension (systolic blood pressure < 90 mmHg). ② Electrophysiological abnormalities: Proarrhythmic effects, notably marked QTc interval prolongation (> 500 ms) and new-onset malignant arrhythmias such as Torsades de Pointes. ③ Organ toxicity: Amiodarone-associated thyroid dysfunction [abnormal thyroid-stimulating hormone (TSH) levels], hepatotoxicity [alanine aminotransferase/aspartate aminotransferase (ALT/AST) levels >3 times the upper limit of normal], and pulmonary toxicity. ④ Other systemic adverse effects: Fatigue, dizziness, and bronchospasm related to β-blockers or calcium channel blockers, as well as gastrointestinal symptoms (nausea, vomiting) potentially associated with any antiarrhythmic agent.

Myocardial Injury and Stress Biomarkers

Fasting venous blood samples (5 mL) were collected from the cubital vein of patients in both groups at three time points: T0 (within 24 hours before treatment), T1, and T2. The blood samples were placed in vacuum tubes without anticoagulant, allowed to clot at room temperature for 30 minutes, and then centrifuged at 3,000 r/min for 15 minutes. The resulting supernatant serum was carefully separated, aliquoted, and stored at −80°C in an ultra-low temperature freezer for subsequent batch analysis. Serum levels of NT-proBNP and hs-cTnT were measured using electrochemiluminescence

immunoassay on a Roche Cobas e 801 fully automated analyzer with matched reagent kits (Roche Diagnostics, Switzerland). hs-cTnI levels were determined by chemiluminescent microparticle immunoassay on an ARCHITECT i2000SR automated immunoassay analyzer with corresponding reagents (Abbott Laboratories, USA). hs-CRP levels were quantified via immunonephelometry on a BN ProSpec® fully automated protein analyzer with matched reagents (Siemens Healthineers, Germany). All procedures were strictly performed according to the manufacturers' instructions and standardized operating protocols, with internal quality controls implemented throughout.

Cardiac Function Assessment

Transthoracic echocardiography was performed at T0, T3 (1 month after discharge), and T4 (6 months after discharge) using a Philips EPIQ 7C color Doppler ultrasound system (Philips, Netherlands; S5-1 probe, frequency 1–5 MHz) by two senior sonographers who were blinded to group allocation. With patients in the left lateral decubitus position and under synchronous ECG monitoring, the following parameters were measured during quiet respiration: LVEF, LVEDD, LVEDVI, and E/e′. All measurements were obtained over three consecutive cardiac cycles and averaged for the final analysis.

Quality of Life and Functional Status

Patient-reported outcomes were evaluated at T0, T3, and T4 using both the SAQ and the 6MWT distance. The SAQ[25] comprises 19 items across five domains: physical limitation (PL, 9 items in Question 1), angina stability (AS, Question 2), angina frequency (AF, Questions 3–4), treatment satisfaction (TS, Questions 5–8), and Disease Perception (DP, Questions 9–11). Items in Questions 1, 3, 4, and 5 are scored on a 1–6 scale, while remaining items use a 1–5 scale. Uniformly trained researchers administered the questionnaire in a quiet environment, reading each item aloud. Patients responded based on their experiences during the preceding four weeks. Standardized domain scores were calculated as: (actual score - minimum domain score)/(maximum domain score - minimum domain score) × 100. All domain scores range from 0 to 100, with higher scores indicating better quality of life and functional status. The 6MWT[26] was conducted along a 30-meter straight, flat corridor. Patients avoided vigorous activity for 1 hour and heavy meals for 2 hours preceding the test. Starting from a standing position at the corridor's end, patients received standardized instructions: "Please walk back and forth along this corridor as far as possible in 6 minutes. Your goal is to cover the maximum distance, but do not run or jog". Standardized encouragement (eg, "You are doing well, please continue") was provided every minute. The test was immediately terminated if patients developed chest pain, intolerable dyspnea, dizziness, or pallor. The total distance walked in 6 minutes was recorded in meters as the primary measure of functional exercise capacity.

MACE

The composite MACE endpoint was assessed over the 6-month period following hospital discharge. Components included: ① Cardiac death: Death attributable to direct cardiac causes, including acute MI, malignant arrhythmias, or heart failure. ② Non-fatal recurrent MI: Defined according to the *Fourth Universal Definition of MI*,[27] requiring a rise and/or fall of myocardial injury markers (preferably hs-cTn) with at least one value exceeding the 99th percentile upper reference limit, accompanied by clinical evidence of acute myocardial ischemia (eg, ischemic symptoms, new ischemic ECG changes, imaging evidence of new loss of viable myocardium, or identification of a coronary thrombus by angiography). ③ Rehospitalization for UA or heart failure: Hospital readmission required due to new-onset or worsening symptoms of UA or heart failure (eg, dyspnea, edema). ④ Unplanned revascularization: Including either urgent/emergent or elective PCI or coronary artery bypass grafting (CABG) performed for recurrent ischemia related to the target or a non-target vessel. ⑤ Malignant arrhythmic events: Episodes of hemodynamically unstable sustained ventricular tachycardia or ventricular fibrillation, or appropriate ICD therapy delivered for an arrhythmic event.

Statistical Analysis

All statistical analyses were performed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed using the Shapiro–Wilk test. Data were presented as mean ± standard deviation (± *s*) for normally distributed variables. Between-group comparisons for these parameters were conducted using the

independent samples *t*-test when the assumption of homogeneity of variance (verified by the Levene's test) was met; otherwise, the the Welch *t*-test was applied. Within-group comparisons for paired measurements were performed using the paired samples *t*-test. Non-normally distributed data were expressed as M (P25, P75). The Mann–Whitney *U*-test was used for between-group comparisons of such data, while the Wilcoxon signed-rank test was employed for within-group comparisons. For analyzing repeated-measures data, repeated-measures analysis of variance was applied if the data met assumptions of normality and sphericity. If these assumptions were violated, the non-parametric Friedman test was used instead. Post-hoc analyses for multiple comparisons were adjusted using the Bonferroni correction. Categorical data were summarized as (n, %) and compared between groups using the Chi-square (χ^2) test. A two-sided significance level of $\alpha = 0.05$ was predefined for all tests, with a *P*-value of less than 0.05 considered statistically significant.

Results

Comparison of Antiarrhythmic Interventions During Hospitalization

Significant differences were observed in pharmacological management strategies between the two groups. Patients in the observation group demonstrated a significantly higher β-blocker utilization rate, a greater rate of guideline-adherent prescribing, and a more frequent rate of drug therapy adjustments compared to the control group ($P < 0.05$ for all). In contrast, the usage rates of amiodarone and the proportion of patients receiving combination antiarrhythmic therapy were comparable between the two groups ($P > 0.05$). Regarding non-pharmacological interventions, no statistically significant differences were found in the application rates of any procedures ($P > 0.05$ for all) (Table 2).

Arrhythmia Control and Treatment-Related Adverse Events During Hospitalization

The arrhythmia control rate in the observation group was significantly higher than that in the control group at T2 (93.75% vs 82.50%, $P < 0.05$). In contrast, no statistically significant difference in control rates was observed between the two groups at the T1 assessment ($P > 0.05$). Regarding treatment-related adverse events, the incidence rates of hemodynamic abnormalities, electrophysiological abnormalities, organ toxicity, and other systemic adverse effects were all comparable between the two groups, with no statistically significant differences identified ($P > 0.05$ for all) (Table 3).

Comparison of Myocardial Injury and Stress Biomarkers

No statistically significant differences in the levels of NT-proBNP, hs-cTnI, hs-cTnT, or hs-CRP were observed between the two groups at baseline (T0; $P > 0.05$ for all). At both subsequent time points, T1 and T2, levels of all four biomarkers demonstrated significant decreases from baseline values within each group ($P < 0.05$). Furthermore, the observation group consistently exhibited significantly lower levels of NT-proBNP, hs-cTnI, hs-cTnT, and hs-CRP compared to the control group at both T1 and T2 ($P < 0.05$ for all comparisons) (Table 4).

Table 2 Comparison of Antiarrhythmic Interventions During Hospitalization [n (%)]

Intervention	Observation Group (n = 80)	Control Group (n = 80)	χ^2	*P*
Pharmacological therapy				
β-blocker utilization rate	76 (95.00)	68 (85.00)	4.444	0.035
Amiodarone utilization rate	27 (33.75)	32 (40.00)	0.671	0.413
Combination therapy rate	18 (22.50)	15 (18.75)	0.344	0.558
Guideline adherence rate	73 (91.25)	58 (72.50)	9.476	0.002
Drug adjustment rate	65 (81.25)	42 (52.50)	14.925	< 0.001
Non-pharmacological therapy				
Electrical cardioversion/Defibrillation	5 (6.25)	9 (11.25)	1.252	0.263
Temporary cardiac pacing	1 (1.25)	3 (3.75)	1.026	0.311
RFCA	2 (2.50)	3 (3.75)	0.206	0.650
ICD	1 (1.25)	2 (2.50)	0.340	0.560
CRT	0 (0.00)	0 (0.00)	-	-

Abbreviations: RFCA, Radiofrequency catheter ablation; ICD, Implantable cardioverter-defibrillator; CRT, Cardiac resynchronization therapy.

Table 3 Comparison of Arrhythmia Control and Treatment-Related Adverse Event Rates During Hospitalization [n (%)]

Outcome Measure	Observation Group (n = 80)	Control Group (n = 80)	χ^2	*P*
Arrhythmia control rate				
T1	67 (83.75)	60 (75.00)	1.871	0.171
T2	75 (93.75)	66 (82.50)	4.838	0.028
Adverse event rate				
Hemodynamic abnormalities	7 (8.75)	14 (17.50)	2.686	0.101
Electrophysiological abnormalities	3 (3.75)	8 (10.00)	2.441	0.118
Organ toxicity	2 (2.50)	3 (3.75)	0.206	0.650
Other systemic adverse effects	10 (12.50)	9 (11.25)	0.060	0.807

Notes: T1, 2 days post-treatment initiation; T2, 7 days post-treatment initiation.

Table 4 Comparison of Myocardial Injury and Stress Biomarkers

Time Point	Observation Group (n = 80)	Control Group (n = 80)	*Z*	*P*
T0				
NT-proBNP (pg/mL)	1499.00 (1247.00, 1866.50)	1511.50 (1231.00, 1851.50)	0.346	0.729
hs-cTnI (pg/mL)	22.25 (14.45, 31.40)	20.80 (12.90, 30.75)	0.770	0.442
hs-cTnT (ng/mL)	1.60 (1.20, 2.00)	1.55 (1.20, 2.00)	0.048	0.962
hs-CRP (mg/L)	25.20 (21.80, 28.50)	25.50 (22.35, 28.75)	0.456	0.649
T1				
NT-proBNP (pg/mL)	796.50 (646.50, 973.00)[a]	1010.00 (830.00, 1211.00)[a]	5.291	< 0.001
hs-cTnI (pg/mL)	7.70 (5.00, 11.65)[a]	11.50 (7.60, 16.40)[a]	3.978	< 0.001
hs-cTnT (ng/mL)	0.70 (0.60, 1.00)[a]	0.90 (0.70, 1.20)[a]	3.702	< 0.001
hs-CRP (mg/L)	15.15 (13.05, 17.35)[a]	18.45 (15.80, 21.10)[a]	5.613	< 0.001
T2				
NT-proBNP (pg/mL)	299.50 (221.50, 379.00)[a]	357.50 (269.00, 443.50)[a]	3.164	0.002
hs-cTnI (pg/mL)	1.90 (1.15, 2.85)[a]	2.70 (1.60, 4.20)[a]	2.955	0.003
hs-cTnT (ng/mL)	0.20 (0.10, 0.20)[a]	0.20 (0.10, 0.30)[a]	2.336	0.019
hs-CRP (mg/L)	5.20 (3.85, 6.60)[a]	6.40 (4.55, 8.40)[a]	3.256	0.001

Notes: [a]$P < 0.05$ compared with T0 within the same group. T0, baseline (within 24 hours pre-treatment); T1, 2 days post-treatment; T2, 7 days post-treatment.
Abbreviations: NT-proBNP, N-terminal pro-B-type natriuretic peptide; hs-cTnI, High-sensitivity troponin I; hs-cTnT, High-sensitivity troponin T; hs-CRP, High-sensitivity C-reactive protein.

Comparison of Cardiac Function Parameters

No significant differences in LVEF, LVEDD, LVEDVI, or the E/e′ ratio were observed between the two groups at baseline (T0; $P > 0.05$ for all). At both follow-up time points (T3 and T4), both groups showed significant improvement from their respective T0 values, characterized by higher LVEF and lower LVEDD, LVEDVI, and E/e′ ratio ($P < 0.05$ for all intra-group comparisons). Notably, the extent of these improvements was significantly more pronounced in the observation group compared to the control group at both T3 and T4, with all inter-group differences reaching statistical significance ($P < 0.05$) (Table 5).

Comparison of SAQ Scores

No significant differences in any of the five SAQ domain scores were observed between the two groups at baseline (T0; $P > 0.05$ for all). At the T3 and T4 follow-up assessments, both groups exhibited statistically significant improvements from their T0 scores across all SAQ domains, including PL, AS, AF, TS, and DP ($P < 0.05$ for all within-group comparisons). Furthermore, the improvements in the observation group were significantly greater than those in the control group at both time points, with all inter-group differences being statistically significant ($P < 0.05$) (Table 6).

Table 5 Comparison of Cardiac Function Parameters

Time Point	Observation Group (n = 80)	Control Group (n = 80)	*t*	*P*
T0				
LVEF (%)	48.36 ± 5.16	47.89 ± 5.36	0.571	0.569
LVEDD (mm)	53.72 ± 3.73	54.26 ± 3.74	0.923	0.357
LVEDVI (mL/m^2)	76.05 ± 7.39	76.65 ± 7.66	0.505	0.614
E/e′	12.96 ± 2.15	13.16 ± 2.24	0.569	0.570
T3				
LVEF (%)	54.26 ± 5.60[a]	51.49 ± 5.98[a]	3.029	0.003
LVEDD (mm)	50.57 ± 3.70[a]	52.51 ± 4.13[a]	3.117	0.002
LVEDVI (mL/m^2)	69.94 ± 6.86[a]	73.36 ± 7.43[a]	3.022	0.003
E/e′	10.25 ± 1.77[a]	11.09 ± 2.00[a]	2.809	0.006
T4				
LVEF (%)	58.61 ± 5.26[a]	56.43 ± 5.70[a]	2.522	0.013
LVEDD (mm)	49.82 ± 3.39[a]	51.08 ± 4.04[a]	2.130	0.035
LVEDVI (mL/m^2)	67.55 ± 6.49[a]	69.93 ± 7.39[a]	2.167	0.032
E/e′	8.81 ± 1.51[a]	9.36 ± 1.81[a]	2.072	0.040

Notes: E/e′, ratio of early mitral inflow velocity to early diastolic mitral annular velocity. T0, baseline; T3, 1 month post-discharge; T4, 6 months post-discharge. [a]$P < 0.05$ compared with T0 within the same group.
Abbreviations: LVEF, Left ventricular ejection fraction; LVEDD, Left ventricular end-diastolic diameter; LVEDVI, Left ventricular end-diastolic volume index.

Table 6 Comparison of SAQ Scores

Time Point	Observation Group (n = 80)	Control Group (n = 80)	*Z*	*P*
T0				
PL	33.50 (28.50, 39.00)	34.00 (29.50, 40.00)	0.940	0.347
AS	29.00 (24.50, 35.00)	30.00 (25.50, 36.00)	0.830	0.406
AF	31.50 (26.50, 37.50)	32.00 (27.50, 38.00)	0.673	0.501
TS	43.00 (37.00, 50.00)	42.00 (36.00, 50.00)	0.388	0.698
DP	49.00 (44.00, 55.00)	50.00 (45.00, 56.00)	0.905	0.365
T3				
PL	71.50 (65.00, 78.00)[a]	65.00 (58.00, 72.50)[a]	3.977	< 0.001
AS	74.50 (66.50, 83.00)[a]	67.00 (58.00, 76.50)[a]	3.841	< 0.001
AF	76.00 (68.50, 84.00)[a]	68.00 (61.50, 77.50)[a]	3.905	< 0.001
TS	78.50 (73.00, 85.00)[a]	72.00 (65.50, 79.00)[a]	4.362	< 0.001
DP	77.00 (71.50, 83.00)[a]	70.00 (63.50, 77.00)[a]	4.804	< 0.001
T4				
PL	85.00 (80.00, 91.00)[a]	81.00 (74.00, 87.00)[a]	3.291	< 0.001
AS	92.00 (86.00, 97.00)[a]	86.00 (79.00, 93.50)[a]	3.567	< 0.001
AF	92.00 (87.50, 96.00)[a]	87.50 (82.00, 95.00)[a]	3.085	0.002
TS	90.00 (85.50, 94.50)[a]	86.00 (80.00, 92.00)[a]	3.238	0.001
DP	91.00 (86.00, 95.00)[a]	86.00 (80.00, 92.50)[a]	3.484	< 0.001

Notes: T0, baseline; T3, 1 month post-discharge; T4, 6 months post-discharge. [a]$P < 0.05$ compared with T0 within the same group.
Abbreviations: SAQ, Seattle Angina Questionnaire; PL, Physical limitation; AS, Angina stability; AF, Angina frequency; TS, Treatment satisfaction; DP, Disease perception.

Comparison of 6MWT Results

No significant difference in the baseline 6MWT distance was observed between the two groups at T0 ($P > 0.05$). At both follow-up assessments (T3 and T4), patients in both groups demonstrated a statistically significant improvement in the 6MWT distance compared to their own baseline values ($P < 0.05$). Furthermore, the observation group achieved

Table 7 Comparison of 6MWT Distances

Time Point	Observation Group (n = 80)	Control Group (n = 80)	*t*	*P*
T0	301.86 ± 38.96	300.45 ± 39.33	0.228	0.820
T3	398.25 ± 45.04[a]	375.15 ± 46.80[a]	3.181	0.002
T4	452.34 ± 43.57[a]	436.11 ± 45.86[a]	2.294	0.023

Notes: T0, baseline; T3, 1 month post-discharge; T4, 6 months post-discharge. [a]$P < 0.05$ compared with T0 within the same group.
Abbreviation: 6MWT, 6-minute walk test.

a significantly longer walking distance than the control group at both the T3 and T4 time points ($P < 0.05$ for both comparisons) (Table 7).

Comparison of MACE Within 6 months Post-Discharge

During the 6-month follow-up period after discharge, the occurrence rate of the composite MACE endpoint was significantly lower in the observation group compared to the control group (13.75% vs 32.50%, $P < 0.05$) (Table 8).

Discussion

This study established a management model centered on an ECG monitor alert-stratified response-stepwise drug titration-team-based quality control pathway, built upon a foundation of standardized ACS care. The results demonstrated that, compared to conventional experience-driven management, this protocol significantly enhanced the rational initiation and timely adjustment of key medications like β-blockers, reduced the time delay from detection to intervention, and was associated with superior short-term arrhythmia control; however, given the observational design, residual confounding and differences in care intensity cannot be fully excluded. These benefits were paralleled by a more rapid and pronounced reduction in biomarkers reflecting three distinct biological pathways: myocardial injury (hs-cTn), hemodynamic stress (NT-proBNP), and inflammatory burden (hs-CRP). At the 1- and 6-month follow-ups, the observation group exhibited sustained improvements in echocardiographic parameters of left ventricular systolic/filling function and remodeling (eg, increased LVEF, decreased LVEDD/VI and E/e′), significantly better patient-reported outcomes across all SAQ domains and the 6MWT, alongside a lower incidence of medium-term MACE, a finding that should be interpreted as hypothesis-generating, which may reflect the benefits of a structured pathway but may also be influenced by unmeasured confounders and differences in care intensity. Collectively, this evidence suggests that the mere presence of monitoring is insufficient to confer clinical benefit. The crucial factor lies in integrating monitoring signals into actionable and quantifiable management processes, thereby transforming passive recording into an active trigger for intervention. This approach achieved multi-dimensional benefits at the organ, molecular, and patient levels without increasing the need for invasive procedures or costly equipment.

Regarding the underlying mechanisms, sympathetic activation, dispersion of action potential duration, and repolarization heterogeneity during acute ischemia and reperfusion provide a fertile substrate for arrhythmogenesis. Rhythm disturbances and tachycardia further exacerbate transient wall stress and filling pressures, induce endothelial dysfunction and microcirculatory perfusion fluctuations, and amplify inflammation and oxidative stress, ultimately establishing

Table 8 Comparison of MACE Within 6 months Post-Discharge [n (%)]

MACE	Observation Group (n = 80)	Control Group (n = 80)	χ^2	*P*
Cardiac death	1 (1.25)	2 (2.50)	–	–
Non-fatal recurrent MI	2 (2.50)	5 (6.25)	–	–
Rehospitalization for UA/heart failure	4 (5.00)	9 (11.25)	–	–
Unplanned revascularization	3 (3.75)	6 (7.50)	–	–
Malignant arrhythmic events	1 (1.25)	4 (5.00)	–	–
Total MACE	11 (13.75)	26 (32.50)	7.910	0.005

Abbreviations: MACE, Major adverse cardiovascular events; MI, Myocardial infarction; UA, Unstable angina.

a multi-loop "electrophysiological-hemodynamic-inflammatory" positive feedback cycle.[28,29] Integrating ECG monitoring with a stratified response protocol essentially interrupts this positive feedback loop at its origin. Predefined trigger events, such as NSVT, polymorphic PVC, or new-onset rapid AF, prompt immediate bedside assessment and drug titration. This reduces the arrhythmic burden and heart rate-related oxygen consumption, stabilizes blood pressure and coronary perfusion, thereby mitigating internal environmental insults. Within this framework, β-blockers play a pivotal role in disrupting the sympathetic-electrophysiological cascade. By reducing heart rate and blood pressure, prolonging diastole, and improving the myocardial oxygen supply-demand balance and coronary perfusion, they not only directly suppress the risk of life-threatening ventricular events but also indirectly reduce cumulative troponin release by diminishing triggers and re-entry opportunities during reperfusion. The control of heart rate and load leads to decreased chamber wall stress, reflected in the subsequent decline of NT-proBNP. Furthermore, rhythm stabilization and containment of the catecholamine storm can suppress inflammatory amplification, manifesting as a more rapid decrease in hs-CRP.[8,30] In essence, the monitor-response-titration chain interlinks these three parallel biological pathways, resulting in observable, quantifiable, and synergistic short-term biomarker improvements, which translate into mid-term event-free survival benefits through the early reversal of structural remodeling.

Comparison with prior evidence highlights the distinct value of our study. While earlier research often focused on the impact of whether to implement continuous/telemetry monitoring on the detection rates of malignant arrhythmias and in-hospital outcomes,[31] our findings emphasize how to translate monitoring into action. This involves creating a closed-loop system through standardized alarm thresholds, mandatory response time limits, standardized operating procedures for drug titration, and daily team-based quality control, thereby elevating monitoring from an information-gathering tool to an integral component of treatment intensity. It is noteworthy that recent randomized and real-world studies have yielded conflicting evidence regarding the consistent survival benefit of long-termβ-blocker therapy in revascularized patients with preserved LVEF.[9–11] However, the benefits observed in our study primarily occurred during intensification and optimization at the acute-subacute phase, which does not contradict the concept of individualized de-escalation/reassessment in the long term. We advocate for a comprehensive time-stratified + risk-stratified strategy, emphasizing protocol-driven titration and achieving target doses during the early phase of high ischemic burden, strong sympathetic drive, and rhythm instability to obtain early reversal of structure and function. Subsequently, long-term maintenance or gradual dose reduction should be guided by LVEF, heart rate/rhythm status, ischemic burden, and adverse effects, avoiding a one-size-fits-all approach to prolonged overtreatment.

Our study also provides clues regarding the modifiability of residual inflammatory risk. Although guidelines are relatively cautious about routine monitoring of inflammatory markers such as hs-CRP, growing evidence indicates its independent association with short- to medium-term MACE and its complementary value to biomarkers like troponin and NT-proBNP in risk stratification.[32] The greater and faster reduction of hs-CRP in the protocol-managed group suggests that rhythm and load control, potentially by reducing endothelial shear stress fluctuations and improving microcirculatory function, may indirectly attenuate inflammatory drivers. This offers a rationale for future exploration of integrated inflammation-load-injury risk models to guide personalized follow-up and rehabilitation interventions.[33] The consistent improvements in functional capacity (6MWT) and patient-reported outcomes (SAQ) align with the imaging benefits, indicating a coherent transmission of benefits from the molecular and organ levels to the patient level. Rhythm and heart rate stabilization not only improve hemodynamics and energy matching but also translate into patient-perceptible symptom relief, enhanced exercise tolerance, and greater treatment satisfaction, aligning with the current direction of patient-centered quality improvement.[34,35]

Regarding clinical generalizability, the proposed pathway is characterized by low barriers to implementation and high reproducibility. It leverages existing ECG monitoring platforms and nurse-physician collaborative response mechanisms, achieving standardization from alarm to action through parameter thresholds, color-coded alert levels, and time-bound nodes, without requiring expensive new equipment or complex algorithms. It is thus applicable in most in-hospital settings for ACS patients with clinically significant arrhythmias. For resource-limited primary care or non-cardiology specialty hospitals, the key lies in localizing alarm thresholds and institutionalizing team training and daily quality control. In well-equipped centers, this pathway could subsequently be enhanced by integrating AI-assisted rhythm

identification, noise suppression, and false-alarm reduction to further shorten response times, reduce staff fatigue, and improve protocol adherence consistency.

Of course, this study has several limitations that warrant careful consideration. Although propensity score matching improves baseline comparability, differences in post-matching ACS care processes (eg, reperfusion timing, infarct size surrogates, and intensity of guideline-directed medical therapy such as high-intensity statins) may still exist and could influence biomarker trajectories. Accordingly, the observed biomarker changes may partially reflect greater care responsiveness and earlier risk-factor optimization enabled by the pathway, rather than rhythm control alone. First, its single-center, prospective, non-randomized controlled design, while mitigated by baseline equipoise, repeated measurements, and multidimensional outcomes, cannot eliminate residual confounding. Second, the pathway incorporates multiple elements (alert stratification, timed response, drug titration steps, team quality control), making it difficult to disentangle the independent contribution of each component to the final outcomes. Third, some heterogeneity existed in the specific β-blocker agents and titration schemes used, which, while guideline-concordant, might dilute the effect size estimation of any single strategy. Fourth, the follow-up was primarily up to 6 months, thus the sustainability of hard endpoint and quality-of-life benefits requires longer-term assessment. Fifth, the proportion of non-pharmacological rhythm interventions (eg, catheter ablation, ICD/CRT) was low in this cohort, limiting the generalizability of our conclusions to populations at high electrophysiological risk requiring invasive strategies. These limitations suggest that future research should employ multicenter randomized controlled trials to verify whether the differences between standard monitoring + experiential decision-making and protocolized monitor-guided therapy can be consistently replicated over 1 year or longer. Furthermore, factorial design elements could help deconstruct the marginal contributions and identify the minimal effective combination of key components like alarm thresholds, response times, titration steps, and quality control frequency.

Based on the current findings, future research could focus on three main directions: First, establishing a time-axis strategy (acute phase intensification-subacute phase optimization-long-term de-escalation) for β-blocker therapy, defining criteria for when to intensify, maintain, or de-escalate based on LVEF, ischemic burden, rhythm status, and adverse effects. Second, developing integrated risk scores combining inflammatory markers (eg, hs-CRP/interleukin-6), load indicators (NT-proBNP), injury markers (hs-cTn), and echocardiographic/ECG parameters to explore their value in guiding follow-up frequency, rehabilitation prescriptions, and rehospitalization warnings. Third, coupling AI algorithms with the existing pathway to evaluate their real-world benefits in reducing false alarm rates, improving intervention consistency, shortening response delays, and saving manpower, and observing whether this algorithm-process synergy can further reduce MACE and all-cause mortality. Overall, this study demonstrates the potential of the monitoring-guided optimization framework with low implementation costs and high reproducibility, providing actionable evidence for the bedside management of ACS with concomitant arrhythmias and laying a methodological foundation for subsequent randomized trials and quality improvement initiatives.

Data Sharing Statement

The data used during the current study are available from the corresponding author (Long Li) on reasonable request.

Ethical Approval

The study protocol was approved by The People's Hospital of Menghai County's Ethics Committee (No. HYLL-2025-LW002). All procedures involving human participants were performed in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

Xiaolong Li and Xianfeng Liu are co-first authors for this study. The authors declare no competing interests in this work.

References

1. 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:10.1093/eurheartj/ehad191
2. Kraler S, Mueller C, Libby P, Bhatt DL. Acute coronary syndromes: mechanisms, challenges, and new opportunities. *Eur Heart J*. 2025;46(29):2866–2889. doi:10.1093/eurheartj/ehaf289
3. Georgiopoulos G, Kraler S, Mueller-Hennessen M, et al. Modification of the GRACE Risk Score for Risk Prediction in Patients With Acute Coronary Syndromes. *JAMA Cardiol*. 2023;8(10):946–956. doi:10.1001/jamacardio.2023.2741
4. Sandoval Y, Apple FS, Mahler SA, et al. High-Sensitivity Cardiac Troponin and the 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guidelines for the Evaluation and Diagnosis of Acute Chest Pain. *Circulation*. 2022;146(7):569–581. doi:10.1161/CIRCULATIONAHA.122.059678
5. Frampton J, Ortengren AR, Zeitler EP. Arrhythmias After Acute Myocardial Infarction. *Yale J Biol Med*. 2023;96(1):83–94. doi:10.59249/LSWK8578
6. Demidova MM, Holmqvist F, Erlinge D, Platonov PG. Ventricular arrhythmias during ST-segment elevation myocardial infarction and arrhythmic complications during recurrent ischaemic events. *Eur Heart J*. 2024;45(5):393–395. doi:10.1093/eurheartj/ehad740
7. Salerno N, Ielapi J, Cersosimo A, et al. Incidence and outcomes of transient new-onset atrial fibrillation complicating acute coronary syndromes: results from a systematic review and meta-analysis. *Eur Heart J Cardiovasc Pharmacother*. 2025;10(8):652–661. doi:10.1093/ehjcvp/pvae066
8. Wolowiec L, Grzesk G, Osiak J, et al. Beta-blockers in cardiac arrhythmias-Clinical pharmacologist's point of view. *Front Pharmacol*. 2022;13:1043714. doi:10.3389/fphar.2022.1043714
9. Yndigegn T, Lindahl B, Mars K, et al. Beta-Blockers after Myocardial Infarction and Preserved Ejection Fraction. *N Engl J Med*. 2024;390(15):1372–1381. doi:10.1056/NEJMoa2401479
10. Silvain J, Cayla G, Ferrari E, et al. Beta-Blocker Interruption or Continuation after Myocardial Infarction. *N Engl J Med*. 2024;391(14):1277–1286. doi:10.1056/NEJMoa2404204
11. Ishak D, Aktaa S, Lindhagen L, et al. Association of beta-blockers beyond 1 year after myocardial infarction and cardiovascular outcomes. *Heart*. 2023;109(15):1159–1165. doi:10.1136/heartjnl-2022-322115
12. Gomes RAF, Furtado LCC, Montenegro MV, Filho DCS. Beta-blockers in post-myocardial infarction with preserved ejection fraction: systematic review and meta-analysis. *Cardiovasc Diagn Ther*. 2025;15(2):398–413. doi:10.21037/cdt-24-368
13. Gladstone DJ, Wachter R, Schmalstieg-Bahr K, et al. Screening for Atrial Fibrillation in the Older Population: a Randomized Clinical Trial. *JAMA Cardiol*. 2021;6(5):558–567. doi:10.1001/jamacardio.2021.0038
14. Kwun JS, Lee JH, Park BE, et al. Diagnostic Value of a Wearable Continuous Electrocardiogram Monitoring Device (AT-Patch) for New-Onset Atrial Fibrillation in High-Risk Patients: prospective Cohort Study. *J Med Internet Res*. 2023:25e45760. doi:10.2196/45760.
15. Ferrick AM, Raj SR, Deneke T, et al. 2023 HRS/EHRA/APHRS/LAHRS expert consensus statement on practical management of the remote device clinic. *Heart Rhythm*. 2023;20(9):e92–e144. doi:10.1016/j.hrthm.2023.03.1525
16. Garcia R, Marijon E, Karam N, et al. Ventricular fibrillation in acute myocardial infarction: 20-year trends in the FAST-MI study. *Eur Heart J*. 2022;43(47):4887–4896. doi:10.1093/eurheartj/ehac579
17. Pelter MM. Hospital-Based Electrocardiographic Monitoring: the Good, the Not So Good, and Untapped Potential. *Am J Crit Care*. 2024;33(4):247–259. doi:10.4037/ajcc2024781
18. Goldfarb MJ, Saylor MA, Bozkurt B, et al. Patient-Centered Adult Cardiovascular Care: a Scientific Statement From the American Heart Association. *Circulation*. 2024;149(20):e1176–e1188. doi:10.1161/CIR.0000000000001233
19. Katsioupa M, Kourampi I, Oikonomou E, et al. Novel Biomarkers and Their Role in the Diagnosis and Prognosis of Acute Coronary Syndrome. *Life*. 2023;13(10). doi:10.3390/life13101992
20. Nagueh SF, Sanborn DY, Oh JK, et al. Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography and for Heart Failure With Preserved Ejection Fraction Diagnosis: an Update From the American Society of Echocardiography. *J Am Soc Echocardiogr*. 2025;38(7):537–569. doi:10.1016/j.echo.2025.03.011
21. Virani SS, Newby LK, Arnold SV, et al. 2023 AHA/ACC/ACCP/ASPC/NLA/PCNA Guideline for the Management of Patients With Chronic Coronary Disease: a Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. *Circulation*. 2023;148(9):e9–e119. doi:10.1161/CIR.0000000000001168
22. Goldberg RJ, Currie K, White K, et al. Six-month outcomes in a multinational registry of patients hospitalized with an acute coronary syndrome (the Global Registry of Acute Coronary Events [GRACE]). *Am J Cardiol*. 2004;93(3):288–293. doi:10.1016/j.amjcard.2003.10.006
23. Acute coronary syndromes. London: National Institute for Health and Care Excellence (NICE); 2020. PMID: 33301270.
24. Chinese Society of Cardiology CMA. Chinese Heart Rhythm Society CSoBE. [Chinese expert consensus on clinical use of antiarrhythmic drugs]. *Zhonghua Xin Xue Guan Bing Za Zhi*. 2023;51(3):256–269. doi:10.3760/cma.j.cn112148-20230130-00047.
25. Thomas M, Jones PG, Arnold SV, Spertus JA. Interpretation of the Seattle Angina Questionnaire as an Outcome Measure in Clinical Trials and Clinical Care: a Review. *JAMA Cardiol*. 2021;6(5):593–599. doi:10.1001/jamacardio.2020.7478
26. Kammin EJ. The 6-Minute Walk Test: indications and Guidelines for Use in Outpatient Practices. *J Nurse Pract*. 2022;18(6):608–610. doi:10.1016/j.nurpra.2022.04.013

27. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth Universal Definition of Myocardial Infarction (2018). *Glob Heart*. 2018;13(4):305–338. doi:10.1016/j.gheart.2018.08.004
28. Bergfeldt L, Gransberg L, Lundahl G. Ischemia induced repolarization dispersion changes and ventricular arrhythmia: validation of Frank vectorcardiography parameters: a review. *Heart Rhythm O2*. 2025;6(5):709–719. doi:10.1016/j.hroo.2025.02.005
29. Drera A, Rodella L, Brangi E, Riccardi M, Vizzardi E. Endothelial Dysfunction in Heart Failure: what Is Its Role? *J Clin Med*. 2024;13(9). doi:10.3390/jcm13092534
30. 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:10.1161/CIR.0000000000001309
31. Holm MS, Falun N, Pettersen TR, et al. Appropriateness and outcomes of hospitalized patients telemetry monitored for cardiac arrhythmias in accordance with the American Heart Association Practice Standards-A multicenter study. *Heart Lung*. 2024;68:217–226. doi:10.1016/j.hrtlng.2024.07.005
32. Li L, Liu S, Zhang Z, et al. Prognostic value of high-sensitivity C-reactive protein in patients undergoing percutaneous coronary intervention with different glycemic metabolism status. *Cardiovasc Diabetol*. 2023;22(1):223. doi:10.1186/s12933-023-01932-2
33. Malandish A, Karimi A, Naderi M, Ghadamyari N, Gulati M. Impacts of Exercise Interventions on Inflammatory Markers and Vascular Adhesion Molecules in Patients With Heart Failure: a Meta-analysis of RCTs. *CJC Open*. 2023;5(6):429–453. doi:10.1016/j.cjco.2023.02.009
34. Mavromatis K, Jones PG, Ali ZA, et al. Complete Revascularization and Angina-Related Health Status in the ISCHEMIA Trial. *J Am Coll Cardiol*. 2023;82(4):295–313. doi:10.1016/j.jacc.2023.05.025
35. Coulshed A, Coulshed D, Pathan F. Systematic Review of the Use of the 6-Minute Walk Test in Measuring and Improving Prognosis in Patients With Ischemic Heart Disease. *CJC Open*. 2023;5(11):816–825. doi:10.1016/j.cjco.2023.08.003