Evaluation the role of HEART Score in Risk Stratification and Management of Patients with Chest pain presented to Emergency Department of Benha University Hospital

Abstract

Background: Undifferentiated chest pain stands out as both frequent and potentially dangerous. Conventional reliance on cardiovascular risk factors alone is frequently inadequate when clinicians attempt to separate true acute coronary syndrome (ACS) from benign causes. The HEART score, has seen little investigation within high-risk Egyptian populations. Accordingly, the present work assessed how well this tool performs at forecasting 30-day Major Adverse Cardiovascular Events (MACE) and at flagging low-risk individuals who may be discharged early and safely.

Methods: A prospective observational study at Benha University Hospital, the investigation enrolled 260 ED attendees whose acute chest pain raised suspicion of a cardiac origin. Each participant had a baseline HEART score recorded and was then tracked across a 30-day window for any MACE. The discriminative performance of the score was examined through Receiver Operating Characteristic (ROC) analysis, with particular attention paid to the Negative Predictive Value (NPV).

Results: Within the 30-day window, MACE occurred in 21.5% (n = 56) of the cohort. Median HEART scores ran markedly higher among those who reached this endpoint than among those who did not (7 vs. 4, $p < 0.001$). For predicting 30-day MACE the score reached strong diagnostic accuracy (AUC = 0.945), together with an exceptionally high NPV (96.7% for MACE; 98.9% for mortality). An adverse event was recorded in just one patient classified as low-risk.

Conclusion: Within the Egyptian ED setting, Beyond forecasting 30-day MACE reliably, the HEART score supplied strong diagnostic value when the aim is to pinpoint low-risk candidates appropriate for early discharge.

Keywords: Acute Coronary Syndrome, Emergency Department, Risk Stratification, Major Adverse Cardiovascular Events, Chest Pain.

Introduction

Acute undifferentiated chest pain is one of the most commonly reported clinical complaints that are encountered heavily in day-to-day clinical practice in the emergency department (ED) [1]. A chief and serious etiology of chest pain is acute coronary syndrome (ACS), which needs to be rapidly and accurately identified and treated to prevent its significant morbidity and mortality [2]. Current diagnostic challenges that are associated with the detection of ACS include the frequent overlap of symptoms and the fact that initial electrocardiograms and cardiac biomarkers can be non-diagnostic in the early stages of ischemia, which hinders the diagnostic process and makes it conduct proper risk-stratification for patients with chest pain of suspected cardiac origin [3]. This is clinically significant, because differentiating high-risk patients who require immediate invasive intervention from low-risk patients who can be safely discharged is crucial for patient safety, as well as enhancing the process of resources-utilization, which includes the prevention of unnecessary hospital admissions, reducing the costs of healthcare and downsizing the overcrowding of ED [4].

Previous reports demonstrated that traditional risk factors relying on isolated clinical parameters are not accurate, because compensatory mechanisms can maintain stable

vital signs even in the presence of impending severe cardiac events [5,6]. Therefore, to overcome these limitations, the Heart Score was developed, with the rational that by comprehensively combining the subjective clinical history taking with the objective ischemic markers, this score can create a practical framework that can be effectively utilized in initial triage and patient risk stratification, as well as in standardizing and streamlining management pathways[7] .

While the HEART score has demonstrated strong predictive capabilities internationally, there is a notable paucity of data validating its performance within Egyptian cohorts. The significance of this gap relies on the fact that the Egyptian Population often exhibit high risk cardiovascular profiles, meaning that studies used to validate HEART score in other western and eastern population may not perfectly translate to local practice [8,9,10]. Also, confirming the score's ability to distinguish low-risk patients and discharge them early is logistically critical to minimize overcrowding and improve resource-utilization is such limited resource region. Therefore, we conducted this prospective study to evaluate and validate the prognostic ability of HEART score in adult Egyptian patients presenting to the ED with acute chest pain with a clinical suspicion of ACS.

Methodology

Study Design and Setting

Before any enrolment took place, the Research Ethics Committee of the Faculty of Medicine, Benha University, granted ethical approval (approval code: MS 40-4-2025), and every patient, or a guardian acting on their behalf, provided written informed consent. This study was done as a prospective observational study in the emergency department, Benha University Hospitals. Recruitment spanned a 12-month interval running from May 2025 to May 2026 and was confined to adults who arrived at the ED with undifferentiated chest pain accompanied by clinical suspicion of ACS. Our objective was to gauge how valid and accurate the HEART score is for guiding risk stratification and management, which we did by testing its capacity to anticipate 30-day Major Adverse Cardiovascular Events (MACE) and to single out, with safety, low-risk candidates eligible for early discharge.

Study Population and Eligibility Criteria

Only individuals who finished the full 30-day follow-up were retained for analysis. Whether or not the primary composite endpoint had occurred determined their assignment into one of two principal cohorts, namely the MACE and No-MACE groups. Eligibility was restricted to consenting adults aged 18 years or older who came to the ED on account of acute undifferentiated chest pain thought to stem from a cardiac source. Conversely, a patient was ruled out when presentation already showed confirmed MI, when hemodynamic instability was present, when an unambiguous non-cardiac explanation for the pain existed, or when the person could not or would not give informed consent.

Study Procedures

Upon a patient's arrival in the ED, a uniform clinical work-up was carried out, during which baseline demographics, namely body mass index, age and sex, were documented together with cardiovascular risk factors such as hypertension, diabetes mellitus,

hypercholesterolemia, a history of smoking, and any family background of coronary artery disease. The narrative of the chest pain itself was then graded as slightly, moderately, or highly suspicious. Every participant underwent a thorough physical examination spanning both general and local components, with emphasis placed on recording vital signs {heart rate (HR), respiratory rate (RR), temperature, together with systolic and diastolic blood pressure (SBP and DBP)}. In addition, we noted the exact interval from pain onset, taken as the precise time that had elapsed between when the pain began and the moment of hospital assessment.

Immediately on arrival, each evaluated patient was placed on the standard emergency pathway, which combined a 12-lead electrocardiogram (ECG), subsequently labelled normal, non-specific, or as displaying significant ST-segment depression, with a laboratory panel comprising routine blood tests and serial cardiac biomarkers. The first troponin specimen was obtained right after presentation; when the pain had started under 6 hours earlier, a follow-up sample was taken three hours afterward, and persistence of symptoms prompted further draws extending to 6 hours. Scoring subsequently relied on whichever troponin reading proved highest.

A HEART score spanning 0 to 10 was derived for every patient to inform management, with 0, 1, or 2 points allotted to each of five clinical domains, namely History, ECG, Age, Risk Factors and Troponin [8]. The summed value then placed patients within one of three strata: those at Low Risk (score 0-3) were regarded as suitable for safe, early discharge; an Intermediate Risk total (score 4-6) pointed toward hospital admission, observation, and non-invasive testing; and a High Risk result (score 7-10) flagged candidates for prompt invasive management, for instance coronary angiography on either a primary or an elective basis.

Outcomes and Follow-up

A 30-day follow-up, accomplished either through direct telephone contact or by examining hospital and clinic records, served to test how well the baseline HEART score anticipated later clinical events. The principal outcome of interest was any MACE, a composite spanning acute myocardial infarction (AMI), the need for revascularisation by percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG), and death from any cause.

Statistical Analysis

All data handling and statistical computation were carried out in Jamovi. The normality of continuous variables was judged with the Shapiro-Wilk test; according to its outcome, normally distributed measures appeared as mean ± standard deviation (SD), whereas those that were non-parametric were expressed as median (range). Counts of categorical observations were given as frequencies (percentages). When groups were compared directly, continuous data were handled by either Student's t-test or the Mann-Whitney U test depending on their distribution, while categorical comparisons relied on the Chi-square (χ^2) test or Fisher's exact test. The capacity of the HEART score to discriminate clinical outcomes was assessed by Receiver Operating Characteristic (ROC) analysis, and the area under this ROC curve (AUC) was then obtained, together with the sensitivity and specificity and both predictive values, the positive one (PPV) and the negative one (NPV), to characterise the diagnostic precision of the score.

Results

Ultimately, 260 patients who saw the 30-day follow-up through to completion made up the analytic cohort. When the primary composite endpoint was examined, a MACE was detected in 56 patients (21.5%), whereas the remaining 204 patients (78.5%) stayed free of any such event. Figure 1

Male sex was considerably more frequent within the MACE group (76.8% vs 57.8%, $p = 0.012$), as was a family history of CAD (35.7% vs 15.7%, $p > 0.001$). By contrast, neither age ($p = 0.310$) nor BMI ($p = 0.480$) separated the two groups to any significant degree. Patients who developed MACE had a median age of 60 years old and a mean BMI of 30.5 kg/m2, against a median of 58 years old and a BMI of 30.0 kg/m2 among their No-MACE counterparts. The remaining risk factors, including hypertension, diabetes, hypercholesterolemia and smoking history, were comparable across the groups (all $p < 0.05$). Clinical assessment likewise uncovered no meaningful divergence in vital signs between the MACE and No-MACE patients (all $p < 0.05$). Symptom timing followed an almost identical course in both, the mean delay from pain onset to hospital arrival sitting at roughly 5 hours either way (4.9 vs 5.3 hours, $p =0.450$), and over half of each group arrived inside the crucial 6-hour window (57.1% vs 56.8%, $p = 0.970$). Table 1

High-risk attributes dominated the MACE group across every domain: a highly suspicious clinical history was present in 67.8%, significant ST-segment depression in 39.3%, an age of 65 years or older in 55.4%, and troponin elevations exceeding three times the upper limit in 60.7%. The NO-MACE group, on the other hand, leaned toward low-risk profiles, with only 12.4% showing a highly suspicious history, 4.4% having significant ST-segment depression on ECG, 57.8% recording normal ECGs, and 74.0% returning negative troponin results. Such contrasts produced a markedly greater median Total HEART Score in the MACE than the No-MACE group (7 vs 4, $p < 0.001$); most patients who sustained a MACE fell into the high-risk (67.9%) or intermediate-risk (30.4%) bands, whereas the No-MACE group was dominated by low-risk (39.2%) and intermediate-risk (50.5%) individuals. Strikingly, an adverse event befell merely one patient (1.8%) within the low-risk tier. Table 2

Thirty days of observation brought to light pronounced differences between the cohorts. MI proved common in the MACE group, affecting 85.7%, and 6 patients (10.7%) died over the follow-up, whereas the No-MACE group registered neither MI nor any death. The MACE group additionally tended toward a longer hospital stay ($p > 0.001$). Medical therapy on its own sufficed for the overwhelming majority of No-MACE patients (95.1%), but the MACE group called for invasive treatment far more often, with 66.1% undergoing PCI and 17.8% receiving CABG. Table 2

Diagnostic performance of the HEART score was outstanding, reflected in an AUC of 0.945 for 30-day MACE, 0.897 for MI, and 0.872 for revascularization. Although prediction of mortality was somewhat weaker (AUC 0.717), the NPV stayed remarkably elevated throughout, reaching 96.7% for MACE and 98.9% for mortality. Taken together, these figures affirm that the HEART score is a dependable clinical instrument, especially adept at excluding major adverse events and recognising low-risk patients fit for safe discharge. Table 3 and Figures 2-4

Case Presentations

Case 1: Low-Risk Profile

A 58-year-old diabetic and hypertensive female presented with acute chest pain 5 hours post-onset. Her symptoms were slightly suspicious for ACS, and she lacked a family history of CAD. Vital signs were stable. A 12-lead ECG was entirely normal, showing a normal sinus rhythm with no ischemic changes (Figure 5). Cardiac troponin levels were negative. She was assigned a Total HEART Score of 2 (Low Risk) and was subsequently safely discharged on conservative medical therapy. During the 30-day follow-up period, she did not experience any MACE.

Case 2: High-Risk Profile

A 66-year-old male with established atherosclerosis presented 4.5 hours post-onset with highly suspicious acute chest pain. The ECG demonstrated sinus rhythm (HR ~100 bpm), normal axis, normal PR, and a narrow QRS, alongside significant ST-segment depression in leads V4-V6, I, II, III, and aVF. Echocardiography revealed hypokinesia in the basal inferior septum, basal inferior, mid inferior septum, and inferior walls (Figure 6 details the abnormal ECG and Echo findings). Cardiac troponins were elevated >3x the normal limit, yielding a HEART Score of 10 (High Risk). Admitted for invasive management, he underwent successful PCI for critical stenoses in the proximal LAD and mid RCA, and survived his 30-day follow-up.

Discussion

At baseline, our analysis demonstrated that both groups (Mace and No-Mace) had comparable median age (60 vs 58 years) and BMI (30.5 vs 30.0 kg/m2). In agreement with our findings, previous reports indicated that while age is built into the score, it is not the sole independent driver of acute MACE [11]. Nomura et al. reported a significantly older population compared to our study (median 76 vs 69), but similar to our results, age did not significantly differ between the two groups. Notably, the older cohort could be related to the vastly older demographic baseline of Japan compared to Egypt [12].

In our study, gender was a significant factor (p=0.012) in the development of MACE in our analysis, with males comprising 76.8% of the MACE group compared to 57.8% of the No-MACE group. This male predominance is in alignment with epidemiological data consistently showing that obstructive CAD and the subsequent sudden plaque rupture incident tend to develop more in earlier age in males compared to females, whose microvascular disease often presents more atypically [13]. In agreement with our findings, previous studies noted the significance of gender differences in coronary artery disease and incorporated the variable of gender in the newly modified HEARTS3 score [11].

Regarding the clinical presentation of the included patients, our findings demonstrated that physiological parameters were uniform across both cohorts, with no statistically significant differences in SBP, DBP, HR, RR, or temperature. Generally, our findings encapsulate the true danger of the early ACT, which could be clinically silent and difficult to identify. Current literature emphasizes heavily the fact that compensatory hemodynamic and neural mechanisms can maintain normal vital signs until extensive myocardial infarction or exacerbating heart failure occurs [14].

The 30-day outcomes heavily validated the severity of the MACE cohort: 85.7% suffered MI, 10.7% died, and the majority of the patients required PCI (66.1%) or CABG (17.8%). On the other hand, the vast majority of the No-MACE group were treated with conservative medical therapy (95.1%). Similar to our study, Backus et al. also reported that among the MACE cohort, the overwhelming majority required PCI or CABG. (15). In disagreement with our results, **Kim et al**. [9] reported much lower MACE severity; AMI was ~21%, and mortality was only 1.9% in their validation group. This could be related to their inclusion of a broader, lower-risk patient population with more atypical chest pain, unlike our study, which focused on patients presenting with classic highly suspicious criteria. **Kumar et al.** [10] also reported much lower MACE severity: PCI in 17.1%, CABG in 4.6%, and mortality at just 1.1%; however, this is directly attributed to their study design, which excluded high-risk patients (HEART score ≥7).

The distribution of HEART score components showed profound, highly significant differences between the groups (all $p < 0.001$, except Risk Factors at $p = 0.002$). In alignment with our results, a systematic review and meta-analysis of 30 different studies by **Fernando et al.** [16], comprising a pooled population of 44,202 adult ED patients presenting with chest pain, found that high-risk scores (≥7) heavily concentrate events, yielding a 95.0% specificity for MACE. In discordance with our results, **Kim et al.** [9] found that all components of the HEART score were associated with significant differences between the groups except age, which failed to reach statistical significance. Subsequently, this led authors to create a modified version of the HEART score called mHEART, without the score of age. Notably, while some reports in the literature reported that the History component of the HEART score could be the weakest link due to its subjectivity, our findings show that it was highly discriminative, likely due to rigorous clinical evaluation [17] More notably, our findings strongly demonstrated the synergistic combination of age, subjective history, and objective ischemia (ECG/Troponin), which if done correctly has the ability to overcome the blind spots of using any single metric alone.

In agreement, **Kumar et al.,** [10] reported an AUC of 0.874 and an NPV of 100% for the 0-3 threshold, highlighting an exceptionally strong clinical safety net to rule out low-risk patients. In consistent with our findings, Liu et al., conducted a systematic review of 29 studies for risk stratification of ED chest pain patients and reported that compared to traditional risk scores like TIMI and GRACE, Heart score was consistently among the top performers, with AUCs generally between 0.72 and 0.89 for identifying low-risk vs high-risk patients [18]. Therefore, our results indicate that the true clinical utility of the HEART score is in its exceptional NPV, which enables it to accurately exclude the low-risk patients, while minimize the risk of missing ischemic events.

Notably, despite achieving a significant NPV of 98.9%, the HEART score was only a modest predictor of 30-day mortality in our cohort (AUC 0.717). In alignment with our results, **Nomura et al.,** [12]reported that HEART score achieved only a modest prediction ability for the detection of 30-day mortality, despite achieving higher accuracy in the prediction of MACE and MI. In contrast to our results, Fernando et al. found that a HEART score ≥4 had an excellent sensitivity of 95.0% for mortality. In our study, the relatively small sample size yielded a low amount of overall mortality events, which might have resulted in the modest prediction of 30-day mortality. Unlike our

study, **Fernando et al.** [16] included much larger sample, where deaths were frequent enough to power the accuracy metrics.

Several constraints temper this work. Its single-centre conduct and the comparatively modest number of participants may narrow how far the results can be generalised. Furthermore, while the chosen follow-up window suited the appraisal of acute, short-term outcomes and complications, it falls short of capturing the longer-range prognostic worth of the HEART score for cardiovascular events that emerge later.

Conclusion

For acute chest pain encountered in the emergency department, the HEART score serves as an accurate and safe means of triage; it couples excellent negative predictive value for spotting low-risk patients who may leave early with reliable forecasting of 30-day major adverse cardiovascular events.

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Author contribution

All authors made an equal contribution to this study.

Conflicts of Interest

No conflicts of interest

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List of Abbreviations

- **ACS:** Acute Coronary Syndrome
- **AMI:** Acute Myocardial Infarction
- **AUC:** Area Under the Curve
- **BMI:** Body Mass Index
- **CABG:** Coronary Artery Bypass Grafting
- **CAD:** Coronary Artery Disease
- **CI:** Confidence Interval
- **DBP:** Diastolic Blood Pressure
- **ED:** Emergency Department
- **eGFR:** Estimated Glomerular Filtration Rate
- **ECG:** Electrocardiogram
- **HK:** Hypokinesia
- **HR:** Heart Rate
- **LAD:** Left Anterior Descending
- **LVEF:** Left Ventricular Ejection Fraction
- **MACE:** Major Adverse Cardiovascular Events
- **MI:** Myocardial Infarction
- **NPV:** Negative Predictive Value
- **PCI:** Percutaneous Coronary Intervention
- **PPV:** Positive Predictive Value
- **RCA:** Right Coronary Artery
- **ROC:** Receiver Operating Characteristic
- **RR:** Respiratory Rate
- **SBP:** Systolic Blood Pressure
- **SD:** Standard Deviation

Table 1. Baseline Demographics, Clinical Presentation, and Admitted Patient Characteristics

Characteristic	MACE (N = 56)	No-MACE (N = 204)	p-value
Demographics			
Age (years)	60 (38 - 84)	58 (35 - 82)	0.310
Gender (Male)	43 (76.8%)	118 (57.8%)	0.012*
BMI (kg/m²)	30.5 ± 4.5	30.0 ± 4.9	0.480
Comorbidities & Risk Factors, n (%)			
Hypertension	36 (64.3%)	112 (54.9%)	0.240
Diabetes Mellitus	34 (60.7%)	101 (49.5%)	0.120
Hypercholesterolemia	29 (51.8%)	83 (40.7%)	0.180
History of Smoking	33 (58.9%)	89 (43.6%)	0.080
Family History of CAD	20 (35.7%)	32 (15.7%)	< 0.010*
Clinical Presentation & Vitals			
SBP (mmHg)	144.2 ± 23.5	142.1 ± 22.1	0.540
DBP (mmHg)	87.1 ± 14.0	85.9 ± 13.4	0.570
HR (beats/min)	85.5 ± 15.8	84.3 ± 15.0	0.600
RR (breaths/min)	19.1 ± 2.8	18.3 ± 2.3	0.280
Temperature (°C)	36.9 ± 0.6	36.9 ± 0.4	0.900
Pain onset duration (hours)	4.9 ± 3.2	5.3 ± 3.8	0.450
Presented ≤ 6 hours	32 (57.1%)	116 (56.8%)	0.970

*Data are reported as mean ± SD for continuous parametric data, median (range) for non-parametric continuous data, and frequency (percentage) for categorical data. *; a statistically significant p-value ≤ 0.05. Abbreviations: BMI, body mass index; CAD, coronary artery disease; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; HR, heart rate; LVEF, left ventricular ejection fraction; MACE, major adverse cardiovascular events; RR, respiratory rate; SBP, systolic blood pressure.*

Table 2. HEART Score Components, Risk Stratification, and 30-Day Clinical Outcomes

Characteristic	MACE (N = 56)	No-MACE (N = 204)	p-value
HEART Score Components			
History Score			< 0.001*
Slightly suspicious (0)	3 (5.4%)	87 (42.6%)	
Moderately suspicious (1)	15 (26.8%)	92 (45.1%)	
Highly suspicious (2)	38 (67.8%)	25 (12.3%)	
ECG Score			< 0.001*
Normal (0)	12 (21.4%)	118 (57.8%)	
Non-specific changes (1)	22 (39.3%)	77 (37.8%)	
ST depression (2)	22 (39.3%)	9 (4.4%)	
Age Score			< 0.001*
< 45 years (0)	1 (1.8%)	37 (18.1%)	
45-64 years (1)	24 (42.8%)	108 (53.0%)	
≥ 65 years (2)	31 (55.4%)	59 (28.9%)	
Risk Factor Score			0.002*
No risk factors (0)	2 (3.6%)	34 (16.7%)	
1-2 risk factors (1)	20 (35.7%)	106 (51.9%)	
≥ 3 risk factors (2)	34 (60.7%)	64 (31.4%)	
Troponin Score			< 0.001*
Negative (0)	9 (16.1%)	151 (74.0%)	
1-3x normal (1)	13 (23.2%)	34 (16.7%)	
> 3x normal (2)	34 (60.7%)	19 (9.3%)	
Total HEART Score	**7 (4 - 10)**	**4 (0 - 9)**	**< 0.001***
Risk Stratification			
Low Risk (Score 0-3)	1 (1.8%)	80 (39.2%)	< 0.001*
Intermediate Risk (Score 4-6)	17 (30.4%)	103 (50.5%)	< 0.001*
High Risk (Score 7-10)	38 (67.9%)	21 (10.3%)	< 0.001*
30-Day Clinical Outcomes			
Myocardial Infarction	48 (85.7%)	0 (0.0%)	< 0.001*
30-Day Mortality	6 (10.7%)	0 (0.0%)	< 0.001*
Length of Hospital Stay (days)	6.5 ± 3.1	1.5 ± 1.2	< 0.001*

Management Strategy			< 0.001*
Medical Therapy	9 (16.1%)	194 (95.1%)	
PCI	37 (66.1%)	10 (4.9%)	
CABG	10 (17.8%)	0 (0.0%)	

*Data are reported as median (range) for non-parametric continuous data, and frequency (percentage) for categorical data. *; a statistically significant p-value ≤ 0.05. Abbreviations: CABG, coronary artery bypass grafting; ECG, electrocardiogram; MACE, major adverse cardiovascular events; PCI, percutaneous coronary intervention.*

Table 3. Diagnostic Performance (AUC Analysis) of the HEART Score

Diagnostic Endpoint	AUC (95% CI)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
30-Day MACE	0.945 (0.915 - 0.975)	89.3	85.8	63.3	96.7
Myocardial Infarction	0.897 (0.853 - 0.940)	85.7	80.3	45.6	96.7
Coronary Revascularization	0.872 (0.815 - 0.929)	82.5	84.2	59.5	94.5
30-Day Mortality	0.717 (0.552 - 0.882)	66.7	70.5	5.1	98.9

AUC, area under the curve; CI, confidence interval; MACE, major adverse cardiovascular events; NPV, negative predictive value; PPV, positive predictive value.

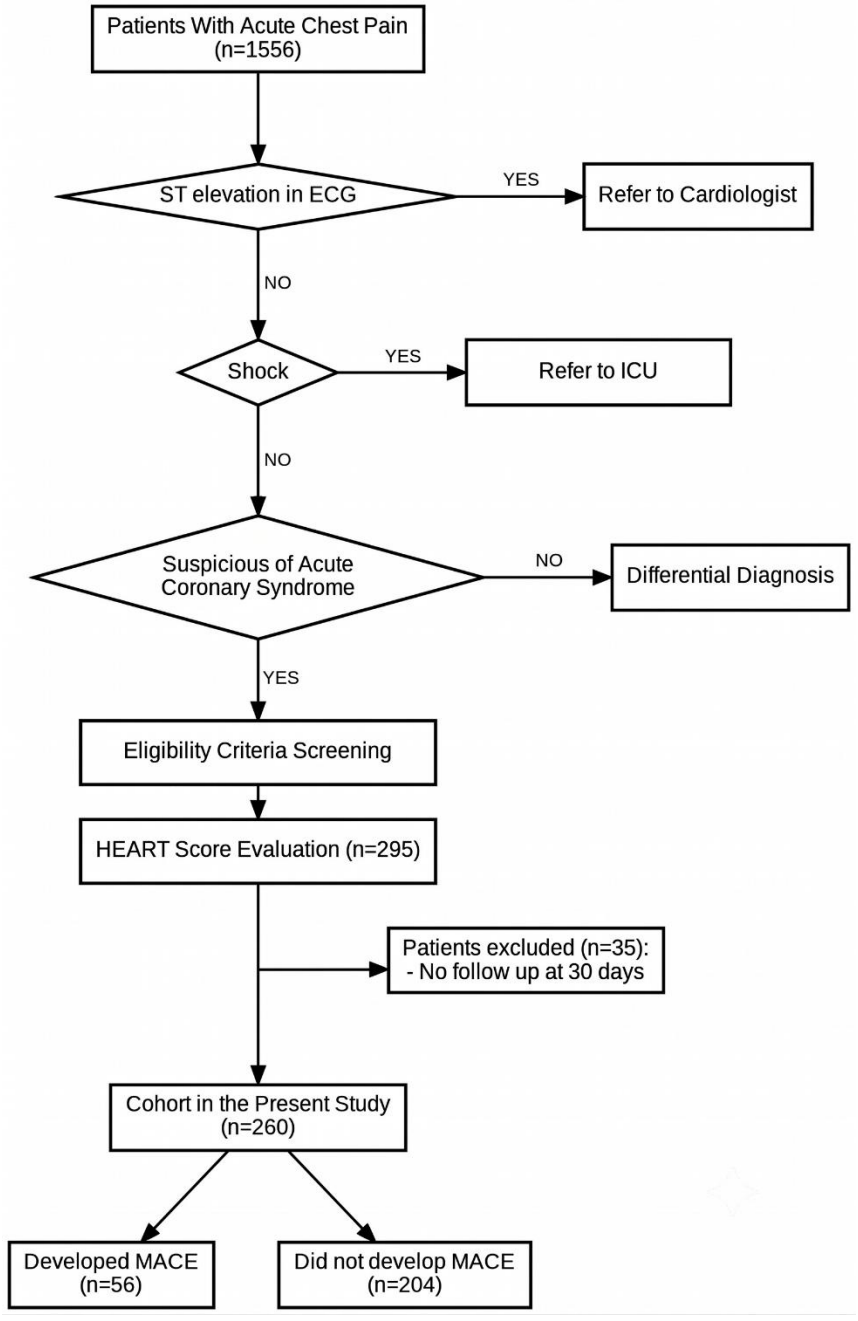

Figure 1. Flow chart for included participants

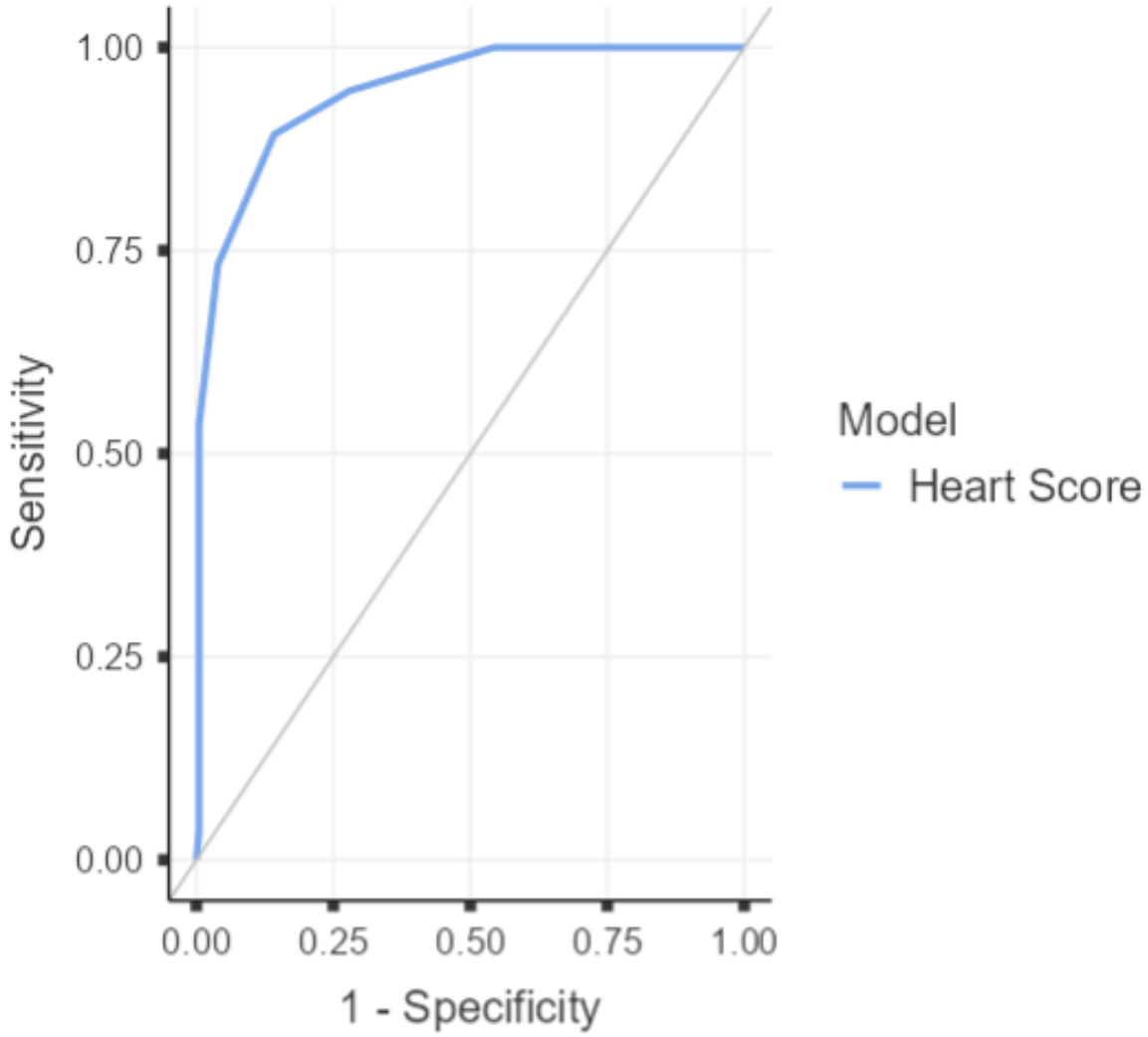

Figure 2. ROC curve for ability of Heart Score to detect 30-Day MACE

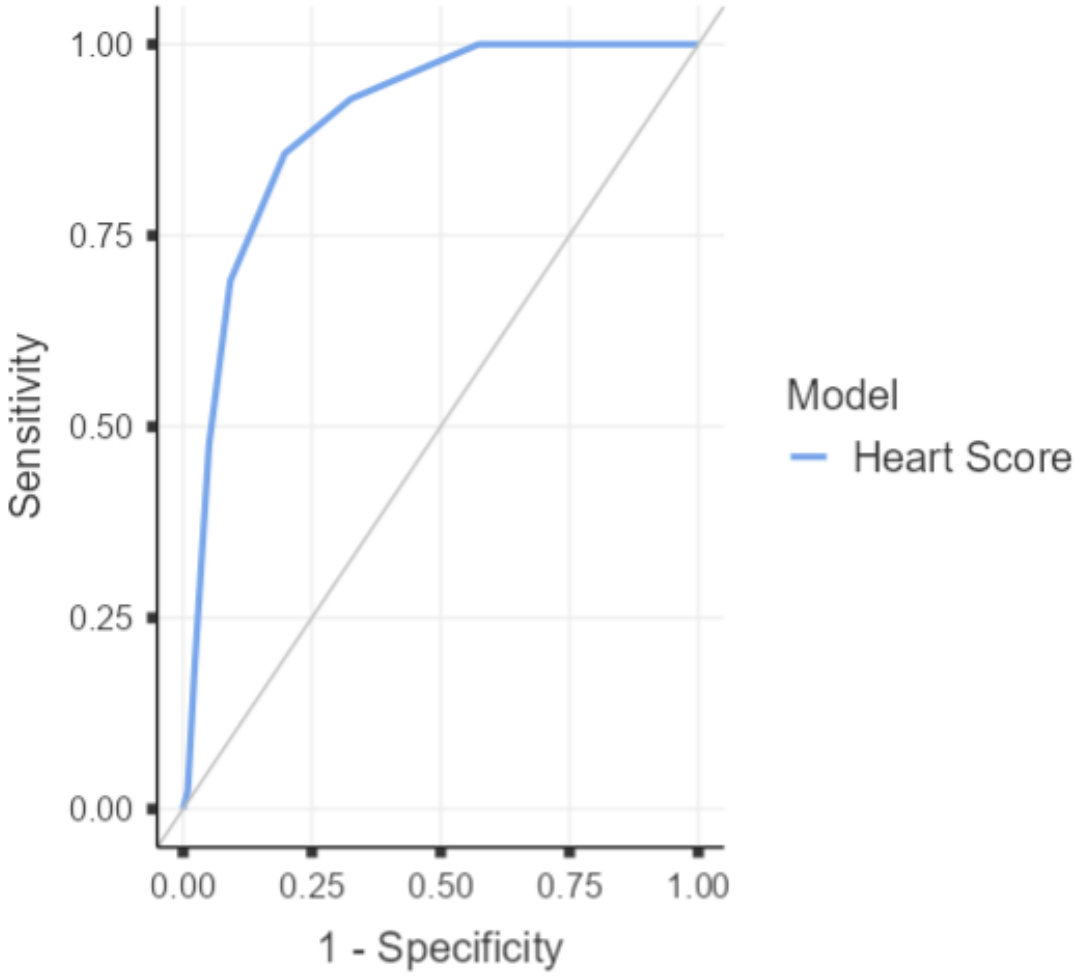

Figure 3. ROC curve for ability of Heart Score to detect 30-Day MI

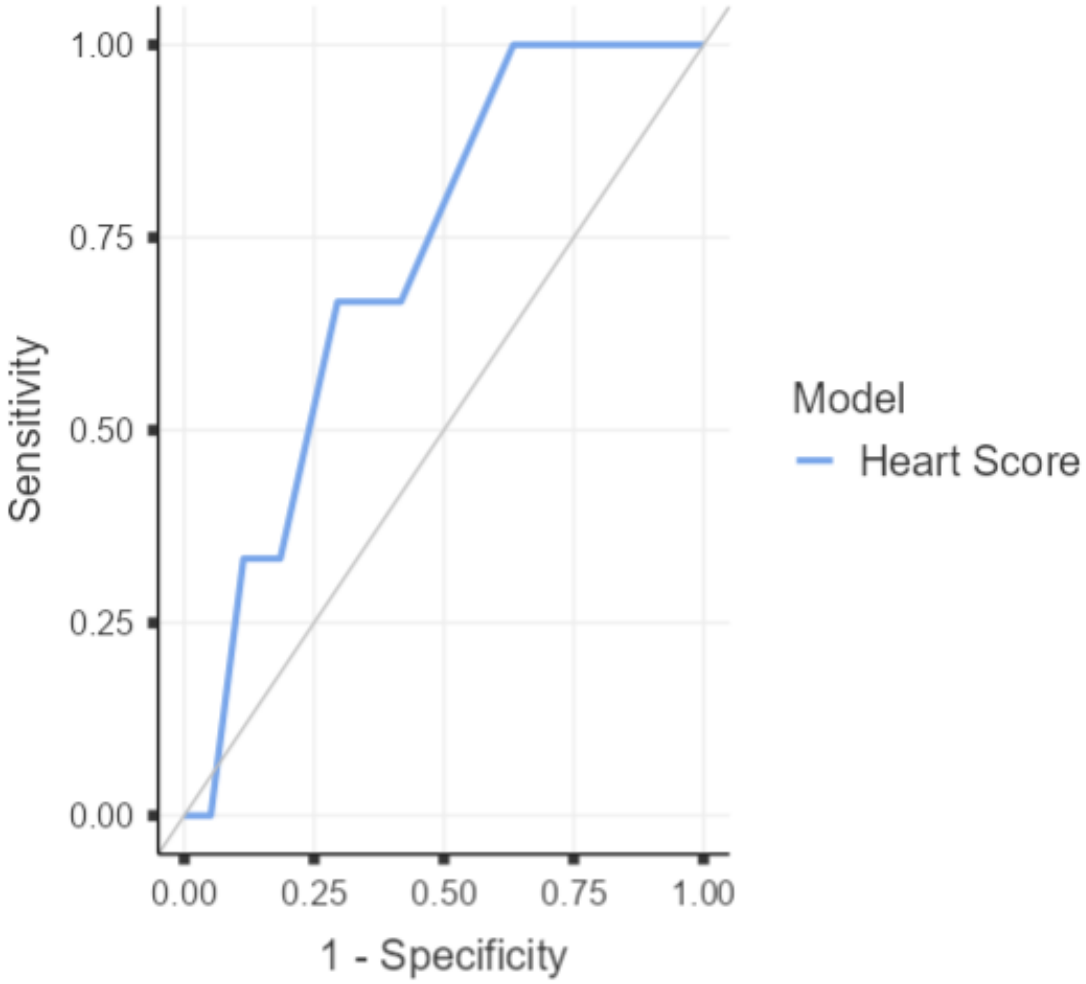

Figure 4. ROC curve for ability of Heart Score to detect 30-Day Mortality

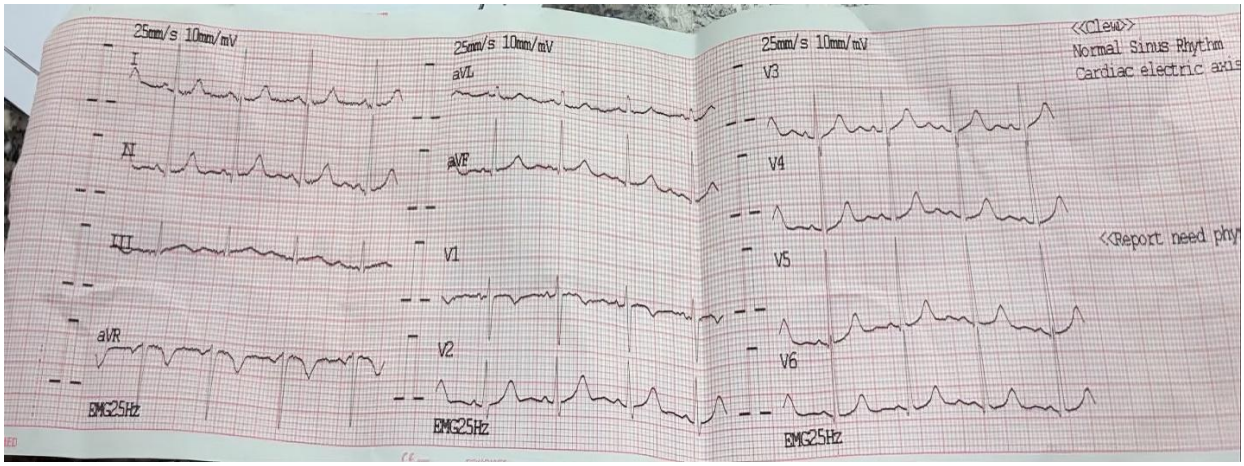

Figure 5. Normal ECG of the 58-year-old female patient

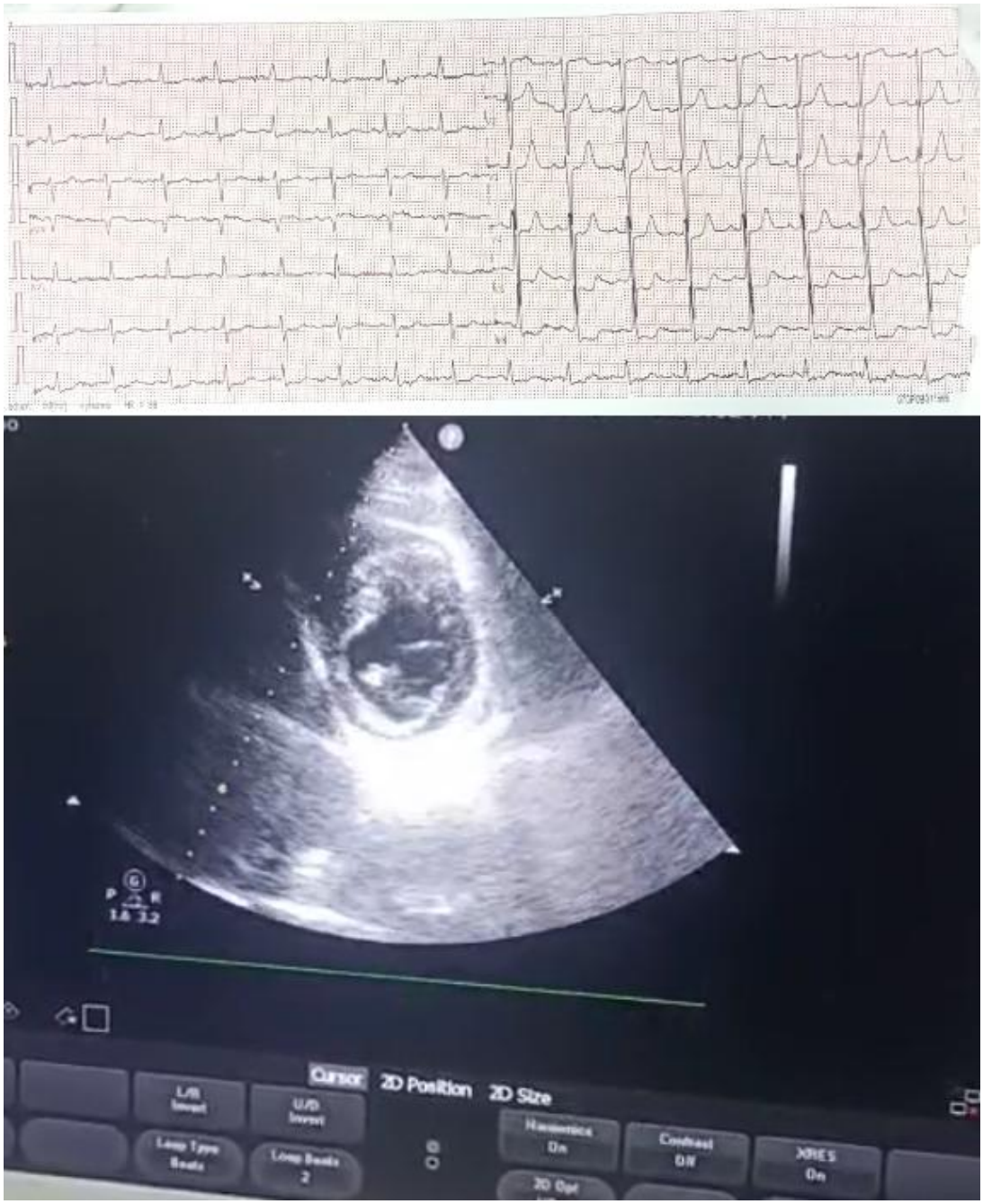

Figure 6. Abnormal ECG demonstrating significant ST depression and Echocardiography revealing regional hypokinesia in the 66-year-old male patient