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ORIGINAL ARTICLE / ÖZGÜN ARAŞTIRMA MAKALESİ

The Prognostic Value of MELD-XI Score in Emergency Department Patients with ST-Segment Elevation Myocardial Infarction

Acil Serviste ST-Segment Yükselmeli Miyokard Enfarktüsü Geçiren Hastalarda MELD-XI Skorunun Prognostik Değeri

Şirin Ertuğrul Esen[1], Yalcin Golcuk[2]

ABSTRACT

Aim: Effective risk stratification is essential for patients with acute ST-elevation myocardial infarction (STEMI) to enable timely and appropriate interventions. Liver dysfunction has emerged as a significant predictor of poor cardiovascular outcomes. The Model for End-stage Liver Disease (MELD)-XI score, initially designed to assess liver disease severity, has demonstrated prognostic value in cardiovascular contexts; however, its application to STEMI patients in emergency department (ED) settings remains underexplored.

Material and Methods: This retrospective study was conducted at Mugla Training and Research Hospital from July 2019 to January 2021. It included adult STEMI patients diagnosed per the European Society of Cardiology criteria. The primary outcome was 28-day all-cause mortality. Statistical analyses included univariate and Cox regression for mortality predictors and Receiver Operating Characteristic and Kaplan-Meier analyses to assess the MELD-XI score's predictive accuracy.

Results: Among 237 patients, 8.4% (n=20) died within 28 days. Non-survivors had significantly higher MELD-XI scores (10.72 vs. 9.44, p<0.001) and lower left ventricular ejection fractions (LVEF) (35% vs. 50%, p<0.001). A MELD-XI score threshold of 9.76 predicted mortality with 80% sensitivity and 70.5% specificity (AUC=0.813) and was negatively correlated with LVEF (r=-0.223, p<0.001). Kaplan-Meier analysis showed that patients with MELD-XI scores above 9.76 had significantly higher 28-day mortality (Log-rank test = 5.43, p<0.001). Independent predictors of mortality included MELD-XI score ≥9.76, age, cardiac arrest on admission, glucose, and hemoglobin levels.

Conclusion: The MELD-XI score is a valuable prognostic tool for assessing 28-day mortality risk in STEMI patients in EDs. By incorporating liver and renal function indicators, the MELD-XI score enhances conventional risk stratification and facilitates more targeted clinical interventions.

Keywords: Emergency department, MELD-XI score, mortality, risk stratification, STEMI.

ÖZ

Amaç: Akut ST-elevasyonu miyokard infarktüsü (STEMI) hastalarında etkili risk stratifikasyonu, zamanında ve uygun müdahalelerin yapılabilmesi için kritik öneme sahiptir. Karaciğer fonksiyon bozukluğu, kötü kardiyovasküler sonuçların güçlü bir prediktörü olarak öne çıkmaktadır. Başlangıçta karaciğer hastalığının şiddetini değerlendirmek amacıyla geliştirilen Model for End-stage Liver Disease (MELD)-XI skoru, kardiyovasküler hastalıklar açısından prognostik değer taşıdığı gösterilmiş olmasına rağmen, acil servis ortamında STEMI hastaları üzerindeki rolü henüz yeterince araştırılmamıştır.

Gereç ve Yöntemler: Bu retrospektif çalışma, Temmuz 2019 ile Ocak 2021 tarihleri arasında Muğla Eğitim ve Araştırma Hastanesi'nde gerçekleştirilmiştir. Çalışmaya, Avrupa Kardiyoloji Derneği kriterlerine göre tanı almış yetişkin STEMI hastaları dâhil edilmiştir. Birincil sonuç, 28 günlük tüm nedenlere bağlı mortalitedir. İstatistiksel analizler, mortalite prediktörlerini değerlendirmek için univariate ve Cox regresyonu, MELD-XI skorunun prediktif doğruluğunu belirlemek için ise Receiver Operating Characteristic ve Kaplan-Meier analizlerini içermektedir.

Bulgular: Çalışmaya dâhil edilen 237 hastadan %8,4'ü (n=20) 28 gün içinde hayatını kaybetmiştir. Ölen hastaların MELD-XI skorları (10,72 vs. 9,44, p<0,001) ve sol ventrikül ejeksiyon fraksiyonları (LVEF) (35% vs. 50%, p<0,001) anlamlı şekilde daha yüksek bulunmuştur. MELD-XI skoru 9,76'nın üzerinde olan hastalar, %80 duyarlılık ve %70,5 özgüllükle mortalite açısından yüksek risk taşıyan grup olarak belirlenmiştir (AUC=0,813). Ayrıca, MELD-XI skoru ile LVEF arasında negatif korelasyon saptanmıştır (r=-0,223, p<0,001). Kaplan-Meier analizi, MELD-XI skoru 9,76'nın üzerinde olan hastaların 28 günlük mortalitesinin anlamlı şekilde daha yüksek olduğunu göstermiştir (Log-rank testi = 5,43, p<0,001). Mortalitenin bağımsız prediktörleri arasında MELD-XI skoru ≥9,76, yaş, acil servise kabulde kardiyak arrest, glukoz ve hemoglobin seviyeleri yer almaktadır.

Sonuç: MELD-XI skoru, acil servislerde STEMI hastalarında 28 günlük mortalite riskini tahmin etmek için değerli bir prognostik araçtır. Karaciğer ve böbrek fonksiyonlarını göz önünde bulundurarak yapılan değerlendirmeler, geleneksel risk stratifikasyonunu geliştirir ve daha hedeflenmiş klinik müdahaleleri mümkün kılar.

Anahtar Kelimeler: Acil servis, MELD-XI skoru, mortalite, risk sınıflandırması, STEMI

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[1]Alasehir State Hospital, Emergency Medicine Service, Manisa, Türkiye

[2]Mugla Sitki Kocman University, Department of Emergency Medicine, Mugla, Türkiye.

Corresponding Author: Yalcin Golcuk, Assoc Prof. **Address:** Mugla Sitki Kocman University, Department of Emergency Medicine, Mugla, Türkiye. **Telephone:** +90 252 214 1323 **e-mail**: yalcingolcuk@mu.edu.tr.

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Introduction

Accurate risk stratification is crucial for patients presenting with acute ST-elevation myocardial infarction (STEMI), as timely and appropriate interventions are essential to improving clinical outcomes (1). Liver dysfunction, often associated with heart failure, is a significant predictor of adverse outcomes in cardiovascular diseases. Emergency Physicians frequently encounter the dual challenge of managing both cardiac and hepatic dysfunction, particularly in conditions such as congestive hepatopathy, which arises from reduced cardiac output and right ventricular failure. Elevated right atrial pressures and compromised hepatic perfusion lead to liver congestion and hypoxia, further worsening the patient's condition (2). Therefore, incorporating hepatic impairment into risk assessment models for acute coronary syndromes (ACS) is vital for optimal patient management.

The Model for End-stage Liver Disease (MELD) score was originally developed for patients with cirrhosis awaiting liver transplantation. It was later modified to the MELD-XI score by excluding the international normalized ratio, making it more suitable for patients on anticoagulation therapy (3,4). The MELD-XI score has demonstrated prognostic relevance in various cardiac conditions (5-10); however, its role in the emergency department (ED) for STEMI patients remains underexplored. This study aims to evaluate the prognostic significance of the MELD-XI score upon ED admission in STEMI patients, specifically assessing its predictive accuracy for 28-day all-cause mortality.

Material and Methods

Study Design and Setting

This retrospective, single-center, cross-sectional cohort study was conducted in the ED of Mugla Training and Research Hospital, a university-affiliated institution, between July 1, 2019, and January 1, 2021. The hospital has a capacity of 500 beds and the ED receives approximately 100,000 patient visits annually. Ethical approval was obtained from the Local Institutional Review Board (Protocol No: 220113-1), and the study was conducted in accordance with the Declaration of Helsinki. Due to the retrospective nature of the study, the requirement for obtaining written informed consent from participants was waived.

Selection of Participants

The study included adult patients aged 18 years and older who were diagnosed with STEMI upon presentation to the ED. Exclusion criteria included: individuals under 18 years of age; patients with inaccessible or incomplete medical records; those transferred to other healthcare facilities; individuals with conditions that mimic STEMI; patients who declined percutaneous coronary intervention (PCI); and those with end-stage liver cirrhosis, severe renal impairment requiring dialysis, malignant tumors, or a history of liver or kidney transplantation. All patients were diagnosed and managed according to the European Society of Cardiology (ESC) guidelines for STEMI (1), with follow-up conducted after 28 days.

Definitions and MELD-XI Score

STEMI was defined using the ESC diagnostic criteria, which included acute chest pain lasting more than 20 minutes and ST-segment elevation in at least two contiguous leads: ≥2.5 mm in men under 40 years, ≥2 mm in men aged 40 and above, or ≥1.5 mm in women in leads V2–V3, and/or ≥1 mm in other leads. A newly developed left bundle branch block was also considered indicative of STEMI. Significant coronary artery lesions identified during PCI were defined as stenosis of ≥50% in the infarct-related artery. Multivessel coronary artery disease was defined as significant stenosis in two or more major coronary arteries, including the left anterior descending artery (LAD), right coronary artery (RCA), and circumflex artery (CX) (1). The MELD-XI score was calculated using the formula: MELD-XI = [5.11 × ln(bilirubin)] + [11.76 × ln(creatinine)] + 9.44. Admission serum levels of creatinine and bilirubin were used, and in cases where multiple values were recorded on the day of admission, the highest values were selected. The minimum value for both bilirubin and creatinine was set at 1.0 mg/dL to ensure consistency in scoring (4).

Data Collection

Demographic characteristics (e.g., gender, age), clinical features (e.g., arterial blood pressure, pulse rate, ECG findings, left ventricular ejection fraction [LVEF]), and medical history (e.g., prior PCI, comorbidities, smoking status, and medication use) were meticulously recorded using a standardized electronic spreadsheet. The presence of cardiac arrest upon ED presentation was also documented. Initial laboratory results (e.g., complete blood count, serum chemistry, blood gas analysis, Troponin T, CK-MB, and lipid profiles) were systematically extracted. Significant coronary artery lesions and multivessel coronary artery disease identified during PCI were noted for both the infarct-related artery and other major coronary arteries. Hospital admission status, length of stay (LOS), and patient survival status at 28 days post-admission were comprehensively recorded. MELD-XI scores were calculated for all patients. Survival status and dates of death were obtained through telephone interviews with patients or their relatives during the 28-day follow-up and validated by reviewing hospital medical records. For deaths outside the hospital, the local civil registration database was consulted to confirm daily reported fatalities within the study area.

Statistical Analysis

The normality of quantitative variable distributions was evaluated using the Kolmogorov-Smirnov test. Descriptive statistics were reported as mean ± standard deviation for normally distributed variables and as median (minimum–maximum) for non-normally distributed variables. Frequency counts and percentages were used for categorical variables. Differences in mean values of quantitative variables were compared using the Student's t-test for normally distributed data and the Mann-Whitney U test for non-normally distributed data. Categorical variables were compared using the χ^2 test or Fisher's exact test, as appropriate. Univariate analysis was applied to all demographic, clinical, laboratory, and outcome variables, with significant variables subsequently included in a Cox regression analysis to identify independent predictors of 28-day all-cause mortality. The Receiver Operating Characteristic (ROC) analysis was used to assess the predictive value of the MELD-XI score for 28-day mortality, and a Kaplan–Meier survival curve was constructed and compared using the log-rank test. Statistical significance was

defined as P <0.05. All statistical analyses were conducted using SPSS version 20 software (SPSS Inc., Chicago, IL, USA).

Results

During the study period, a total of 315 patients diagnosed with STEMI who presented to the ED were reviewed through hospital electronic health records for eligibility in the final analysis. Exclusions included 25 patients with unavailable 28-day mortality data and 48 patients with missing critical admission data for serum bilirubin or creatinine. Additionally, two patients died before undergoing PCI, one patient refused treatment, and two cases involved conditions mimicking STEMI, specifically Kounis syndrome and vasospastic angina. After these exclusions, a final cohort of 237 patients was analyzed.

Among the 237 patients, 199 (84%) were male, with a mean age of 60.41 ± 12.67 years. Notably, 20 patients (8%) died within the 28-day follow-up period. ECG findings indicated that 49.8% presented with inferior, 46.8% with anterior, 10.1% with lateral, 8.9% with posterior, and 7.2% with right-sided infarctions. Angiographic findings revealed that the majority of patients had significant lesions in the LAD (75.5%), followed by the RCA (59.5%) and the CX (43.0%). Additionally, 22.4% of the cohort had multivessel coronary artery disease. A significant association between critical stenosis of the LAD and mortality was identified (P = 0.032). Thirteen patients (5.5%) presented to the ED in cardiac arrest, and the overall 28-day all-cause mortality rate was 8.4% (n = 20). The median LOS was 3 days (ranging from 1 to 30 days). Detailed demographic, laboratory, and clinical characteristics of the cohort are provided in Tables 1 and 2.

Characteristics	Survivors (n = 217)	Nonsurvivors (n = 20)	*p* value
Age, years	59.3 ± 12.2	72.3 ± 11.2	<0.001
Men/Women, n	185/32	14/6	0.104
Previous medical history, n (%)			
Myocardial infarction	26 (12)	4 (20)	0.295
Chronic pulmonary disease	19 (8.8)	2 (10)	0.693
Diabetes mellitus	42 (19.4)	7 (35)	0.144
Hypertension	72 (33.2)	12 (60)	0.031
Heart failure	6 (2.8)	2 (10)	0.139
Hyperlipidaemia	19 (8.8)	3 (15)	0.410
Cerebrovascular disease	5 (2.3)	1 (5)	0.414
Atrial fibrillation	6 (2.8)	1 (5)	0.465
Smoking	155 (71.4)	15 (75)	0.936
Previous PCI	39 (18)	4 (20)	0.766
Medications used, n (%)			
Aspirin	38 (17.5)	4 (20)	0.762
P2Y12 inhibitors	12 (5.5)	3 (15)	0.121
Anticoagulant	3 (1.4)	1 (5)	0.299
ACE/ARB inhibitor	48 (22.1)	6 (30)	0.411
Diuretic	5 (2.3)	2 (10)	0.110
Statin	27 (12.4)	6 (30)	0.042
Laboratory results			
White blood cell count (x10^3/µL)	10.6 (2.2-29.6)	14.0 (5.5-33.6)	0.014
Red blood cell count (x10^3/µL)	494 (285-728)	450 (205-549)	0.002
Hemoglobin (g/dL)	14.8 (8.0-19.6)	13.1 (5.6-16.7)	0.002
Platelet count (x10^3/µL)	245 (91-523)	193 (84-392)	0.041
Glucose (mg/dL)	132 (85-687)	236 (127-531)	<0.001
Urea (mg/dL)	30.4 (3.3-97.5)	40.8 (8.9-154.8)	0.004
Creatinine (mg/dL)	0.92 (0.37-2.68)	1.26 (0.55-8.50)	<0.001
Sodium (mEq/L)	138 (128-144)	139 (120-149)	0.365
Potassium (mEq/L)	4.2 (3.1-6.0)	4.2 (2.9-9.0)	0.642
Calcium (mg/dL)	9.2 ± 0.5	8.8 ± 0.8	0.038
Chlorine (mg/dL)	100.2 ± 3.1	98.0 ± 7.9	0.229
Albumin (g/L)	41 (27-49) ± 5.6	39 (18-46)	0.003
Aspartate transaminase (IU/L)	29 (5-644)	40 (12-372)	0.101
Alanine transaminase (IU/L)	20 (5-753)	30 (5-517)	0.235
Total bilirubin (mg/dL)	0.49 (0.14-2.18)	0.75 (0.15-1.77)	0.075
Troponin T (pg/mL)	85 (3-10085)	289 (9-10096)	0.056
CK-MB (ng/mL)	9.47 (0.77-300.0)	16.3 (1.6-300.0)	0.237
Total cholesterol (mg/dL)	182 (83-312)	178 (103-366)	0.958
HDL (mg/dL)	39 (20-93)	39 (19-61)	0.976
LDL (mg/dL)	110 (12-250)	100 (48-265)	0.514
Triglycerides (mg/dL)	133 (39-1598)	134 (60-462)	0.650

Table 1. Demographic and laboratory characteristics of study patients

ACE/ARB, Angiotensin-converting enzyme / Angiotensin II receptor blocker; CK-MB, Creatine kinase-muscle/brain, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, PCI: Percutaneous coronary intervention.

Characteristics	Survivors (n = 217)	Nonsurvivors (n = 20)	*p* value
Hemodynamic parameters			
Systolic blood pressure (mm Hg)	131 (58-224)	110 (45-150)	<0.001
Diastolic blood pressure (mm Hg)	83 (40-133)	65 (20-95)	<0.001
Heart rate (beats/min)	80 (36-163)	81 (32-150)	0.913
Cardiac arrest on presentation, n (%)	5 (2.3)	8 (40)	<0.001
LVEF, %	50 (15-65)	35 (15-45)	<0.001
Thrombolytic treatment prior to PCI, n (%)	28 (12.9)	1 (5)	0.482
PCI lesion location, n (%)			
Left anterior descending	160 (73.7)	19 (95)	0.032
Right coronary artery	128 (59)	13 (65)	0.775
Circumflex artery	92 (42.4)	10 (50)	0.674
Multivessel disease	45 (20.7)	8 (40)	0.087
Length of stay, days	3.7 ± 2.6	4.2 ± 5.6	0.263
MELD-XI score	9.44 (9.44-14.47)	10.72 (9.44-20.77)	<0.001

Table 2. Clinical characteristics of study patients

LVEF: Left Ventricular ejection fraction, MELD-XI: Model for end-stage liver disease excluding international normalized ratio, PCI: Percutaneous coronary intervention.

The mean MELD-XI score for the study population was 9.98 ± 1.29. Non-survivors had significantly higher MELD-XI scores compared to survivors, at 10.72 (range: 9.44–20.77) versus 9.44 (range: 9.44–14.47). Spearman correlation analysis revealed a significant negative correlation between MELD-XI scores and LVEF (r =-0.223, P <0.001). As illustrated in Figure. 1, the ROC analysis identified a MELD-XI score of 9.76 as the optimal cutoff for predicting 28-day all-cause mortality, with 80% sensitivity and 70.5% specificity, and an AUC of 0.813 (95% CI, 0.703–0.923; P <0.001). Kaplan-Meier survival analysis further demonstrated that patients with a MELD-XI score above 9.76 had significantly higher 28-day mortality compared to those with lower scores (Log-rank test = 5.43, P <0.001, Figure 2). Independent predictors of mortality were identified as a MELD-XI score ≥9.76, cardiac arrest on admission, age, glucose levels, and hemoglobin levels, as shown in Table 3.

Discussion

In this retrospective cohort study, the prognostic significance of the MELD-XI score in predicting 28-day mortality among STEMI patients presenting to the ED was explored. The findings demonstrated that the MELD-XI score is a robust predictor of mortality in this population, with higher scores significantly correlating with an increased risk of mortality. By extending the use of the MELD-XI score to STEMI patients, this study highlights the potential benefits of integrating hepatic and renal function metrics into routine cardiovascular risk assessments, facilitating a more comprehensive approach to patient management in acute care settings.

LVEF is a well-established prognostic marker in STEMI, with lower values commonly associated with adverse outcomes and elevated mortality risk (11). In this study, LVEF at presentation was notably lower among no survivors compared to survivors, underscoring the association between compromised LVEF and increased mortality. Çelik et al. similarly identified LVEF as an independent predictor of in-hospital mortality, reinforcing its role in risk stratification for STEMI patients. Furthermore, a significant negative correlation between MELD-XI scores and LVEF (r =-0.223) was found, closely aligning with Çelik et al.'s findings (r =-0.232) (12). These results highlight a consistent relationship between elevated MELD-XI scores and reduced LVEF, emphasizing the prognostic value of the MELD-XI score as an indicator of cardiac impairment severity in STEMI patients. Elevated MELD-XI scores are associated with multi-organ stress and metabolic dysregulation, contributing to the observed decline in cardiac performance and worse outcomes in myocardial infarction cases. This inverse relationship between MELD-XI and LVEF, consistently observed across studies, supports the MELD-XI score as not only a reliable risk stratification tool but also a complementary measure of left ventricular function, enhancing multidimensional assessment in emergency settings.

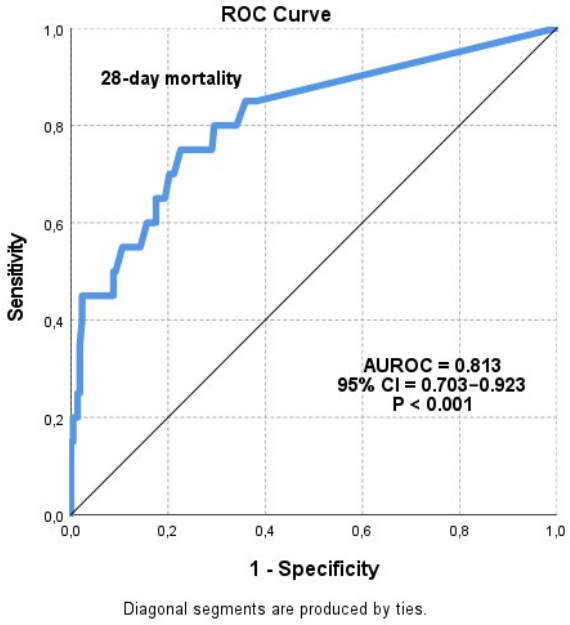

Figure 1. Receiver Operating Characteristic (ROC) Curve for MELD-XI Score in Predicting 28-Day All-Cause Mortality

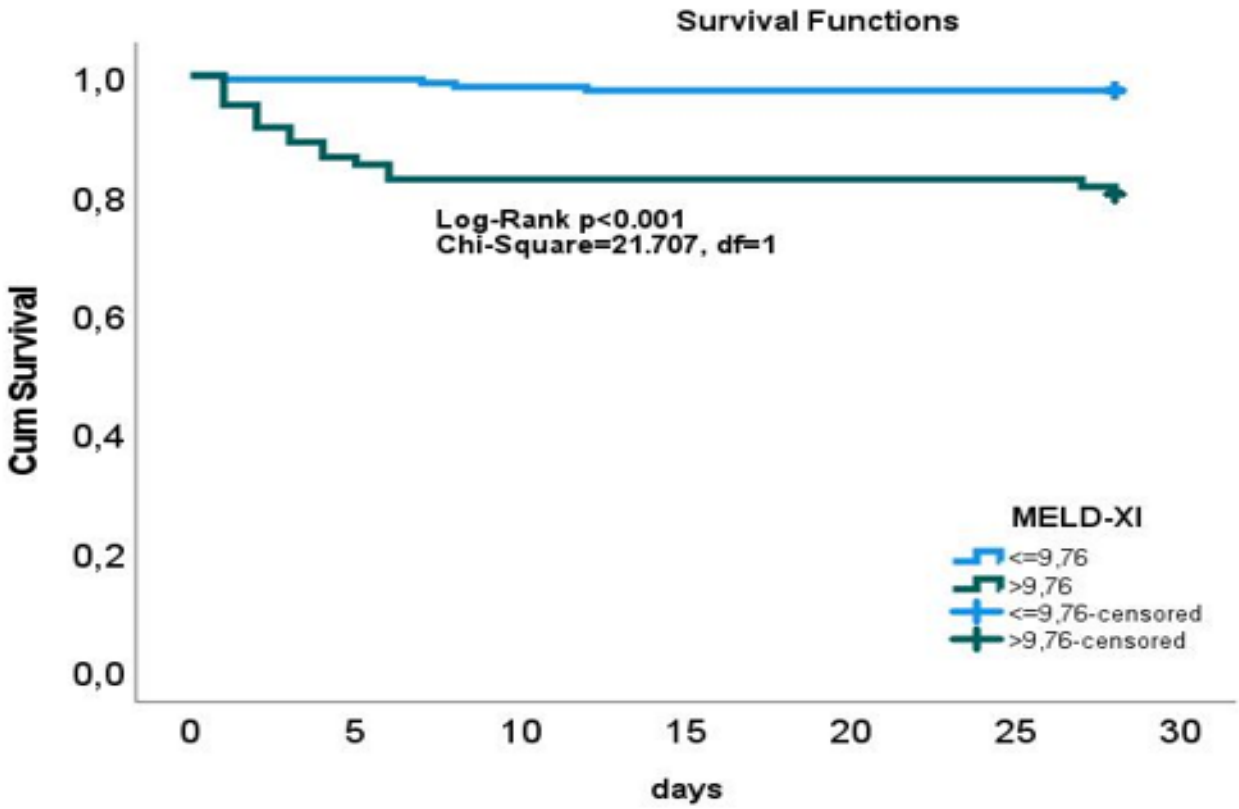

Figure 2. Kaplan–Meier Survival Curves for 28-Day Mortality Stratified by MELD-XI Score Threshold of 9.76

Variables for 28 d	Odds ratio	95% CI	*p* value
MELD-XI score ≥9.76	5.132	1.548-17.020	0.007
Cardiac arrest on admission	9.455	3.266-27.367	<0.001
Age, years	1.053	1.005-1.104	0.030
Glucose, g/dL	1.007	1.003-1.010	<0.001
Hemoglobin, g/dL	0.781	0.616-0.990	0.041

Table 3. Cox regression analysis for the prediction of 28-day mortality

CI: Confidence interval, MELD-XI: Model for end-stage liver disease excluding international normalized ratio.

Zhang et al. identified a MELD-XI threshold of >9.78 as an optimal predictor of short-term mortality in STEMI patients undergoing PCI (13), which closely corresponds to the threshold of >9.76 identified in this study. The minimal variation between these cutoff values underscores the consistency and reliability of the MELD-XI score across different cohorts. This supports the clinical utility of the MELD-XI score in ED settings, providing timely identification of patients at elevated risk. The concordance between these findings and those of Zhang et al. further validates the prognostic value of the MELD-XI score, suggesting its potential as a standardized metric to guide clinical interventions.

Similarly, Çelik et al. investigated the prognostic utility of the MELD-XI score in predicting in-hospital mortality among STEMI patients and found a significant association between elevated MELD-XI scores and higher mortality rates (12). Their study identified a threshold of 10, closely aligned with the threshold of 9.76 in this study. The consistency of these results corroborates the value of the MELD-XI score in risk stratification and patient management in the ED. Both studies support the use of the MELD-XI score for identifying patients vulnerable to adverse outcomes, optimizing the allocation of critical resources in ED.

In addition, He et al. focused specifically on elderly patients (≥60 years) with STEMI undergoing PCI and examined the prognostic role of the MELD-XI score (14). Their findings showed that a MELD-XI score ≥13 was significantly associated with poorer outcomes, suggesting that the MELD-XI score offers substantial prognostic value beyond the traditional TIMI risk score, particularly in older patients. Elderly populations often present with a higher burden of comorbidities and physiological decline, which may exacerbate STEMI outcomes. The higher MELD-XI scores reported in He et al.'s study, compared to this study, may be attributable to the inclusion of older patients with greater susceptibility to hepatorenal dysfunction. The additive value of the MELD-XI score, as shown in their investigation, supports current literature advocating for risk stratification tools that consider multi-organ involvement, particularly in complex cardiovascular cases. Such tools are essential for improving clinical decision-making and patient outcomes in

high-risk populations, enhancing the management of patients in EDs.

Recent studies have further underscored the prognostic relevance of the MELD-XI score across various cardiovascular populations. In a retrospective cohort of patients with chronic heart failure, Lin et al. demonstrated that a high MELD-XI score (median: 12.71 [IQR 10.88–15.44]) was independently associated with increased 3-year all-cause mortality, even after adjusting for confounders such as chronic kidney disease (15). Similarly, Chen et al. found that in patients with acute myocardial infarction undergoing coronary artery stenting, those with higher MELD-XI scores (mean: 11.98 ± 2.34) had significantly lower LVEF and higher rates of heart failure (24.5%) and mortality (5.7%) during 1-year follow-up compared to those with lower scores (16). Complementing these findings, Curcio et al. conducted a prospective longitudinal study in 93 patients with advanced heart failure and reported that patients who experienced adverse outcomes—including death, urgent heart transplant, or LVAD implantation—had significantly higher MELD-XI scores (mean: 16.3 ± 4.0) compared to event-free survivors (mean: 12.5 ± 4.4, P < .001) (17). Collectively, these findings support the broader applicability of MELD-XI as an integrative biomarker reflecting hepatic-renal dysfunction and its emerging role in prognostic models for cardiovascular risk stratification, particularly in populations with overlapping systemic comorbidities.

Beyond cardiac conditions, recent studies have expanded the prognostic scope of the MELD-XI score to include acute respiratory pathologies. In a large ICU cohort of patients with respiratory failure, Arslan et al. found that MELD-XI scores ≥11 were strongly associated with increased mortality across multiple etiological subgroups—including COPD exacerbation, cardiogenic pulmonary edema, and pneumonia—with a hazard ratio of 2.6 (95% CI: 2.4–2.9, P < .001). The mean MELD-XI score among non-survivors was notably elevated (13.6 ± 5.4), reinforcing its role as an independent predictor of ICU mortality (18). Similarly, in a study of normotensive patients with acute pulmonary embolism, Jiao et al. reported that the MELD-XI score was associated with in-hospital adverse events—including shock, catecholamine requirement, and mechanical ventilation—although its predictive capacity was slightly lower than the original MELD score (AUC: 0.618 vs. 0.731) (19). In an even larger multinational ICU cohort involving 11,091 mechanically ventilated patients, Wernly et al. demonstrated that a MELD-XI score >12 was independently associated with both hospital mortality (46% vs. 27%) and 28-day mortality (39% vs. 22%), even after adjusting for disease severity and ventilatory parameters (HR: 1.04; 95% CI: 1.03–1.05; P < .001) (20). These findings emphasize that the MELD-XI score may serve as a pragmatic, organ dysfunction–oriented tool across cardiopulmonary emergencies, including those not classically hepatic in origin, and underscore its potential integration into multidisciplinary risk stratification strategies.

However, it is important to note that the generalizability of our findings is limited to a specific subset of STEMI patients. Given that individuals with advanced hepatic or renal dysfunction, malignancy, or prior organ transplantation were excluded from this study, the prognostic performance of the MELD-XI score in such populations remains uncertain. Future studies are needed to evaluate the applicability and calibration of MELD-XI in broader, more heterogeneous STEMI cohorts, particularly those with coexisting multi-organ pathology.

Limitations

This study has several limitations that warrant consideration. First, the retrospective design, single-center setting, small sample size, and short follow-up period may limit the generalizability of the findings. Furthermore, patients with advanced liver or kidney failure, malignancy, or prior transplants were excluded to reduce confounding; as a result, the prognostic utility of MELD-XI in these higher-risk groups remains untested and should be investigated in future studies. Additionally, socioeconomic factors, which can influence patient prognosis and access to healthcare, were not included in this analysis, potentially introducing biases in evaluating the predictive value of the MELD-XI score for mortality. Furthermore, variability in the timing of patient presentation to the ED following STEMI, and the absence of door-to-balloon time data, may have introduced confounding factors. These limitations highlight the need for additional research involving larger, multicenter cohorts to validate the prognostic value of the MELD-XI score in STEMI patients in EDs.

Conclusion

The MELD-XI score is a crucial prognostic indicator for predicting 28-day all-cause mortality in STEMI patients upon presentation to the ED. Integrating this score into clinical practice may enhance risk stratification, enabling timely identification of high-risk individuals who could benefit from expedited interventions. By systematically incorporating MELD-XI assessments into routine evaluations, emergency physicians can refine management strategies and optimize patient outcomes. This study emphasizes the importance of hepatic and renal function metrics in informing clinical decision-making in acute care settings, particularly in selected STEMI patients without advanced hepatic or renal dysfunction. Broader validation studies are warranted to assess its performance across more diverse clinical subgroups.

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Authors' Contribution: ŞEE: ŞEE: Designing the study, access to study data, statistical analysis, analysis and interpretation of data, drafting the manuscript, proofreading the

manuscript. **YG:** Designing the study, access to study data, statistical analysis, analysis and interpretation of data, drafting the manuscript, proofreading the manuscript.
All authors read and approved the final submitted version of the manuscript.
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