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Characteristics and clinical predictors of cardiogenic shock among patients presenting with acute ST-segment elevation myocardial infarction

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ABSTRACT

Background: Cardiogenic shock (CS) is the leading cause of mortality in ST-segment elevation myocardial infarction (STEMI). Patients with acute coronary syndrome in Saudi Arabia present a decade younger than Western cohorts and have a higher diabetes burden, yet contemporary local data on CS are limited. We aimed to determine the incidence, characteristics, and predictors of CS in a Saudi STEMI cohort.

Methods: Single-center retrospective cohort study of consecutive adult patients with acute STEMI admitted to King Abdulaziz Cardiac Center, Riyadh, from January 2019 to December 2024. CS was defined by the SHOCK trial criteria. Univariate and multivariate logistic regression identified independent predictors.

Results: A total of 878 patients were included (mean age 57.2 ± 13.6 years; 86.6% male); 80 (9.1%) developed CS. Compared with patients without CS, those with CS had a higher prevalence of dyslipidemia (58.8% *vs.* 44.6%, $p = 0.016$) and ventricular arrhythmia (17.5% *vs.* 5.5%, $p < 0.001$), higher peak troponin and N-terminal pro-B-type natriuretic peptid, and lower hemoglobin, sodium, potassium, and estimated glomerular filtration rate. No differences were observed in age, sex, hypertension, diabetes, or infarct location. In multivariate analysis, independent predictors of CS were female sex odds ratio (OR 0.18, 95% confidence intervals 0.06-0.52), dyslipidemia (OR 2.39, 1.22-4.70), arrhythmia (OR 3.69, 1.63-8.37), peak troponin ($p = 0.040$), peak blood urea nitrogen (OR 1.05, 1.01-1.08), nadir hemoglobin (OR 0.97, 0.96-0.99), and nadir potassium (OR 0.16, 0.07-0.34).

Conclusion: CS occurred in 9.1% of this contemporary Saudi STEMI cohort. The predictors that emerged were not the classical Western ones; larger multicenter studies should confirm these findings and inform regional risk stratification.

Keywords: Cardiogenic shock, acute myocardial infarction, ST-segment elevation myocardial infarction, STEMI, Saudi Arabia, predictors.

Introduction

Cardiogenic shock (CS) is a clinical syndrome of end-organ hypoperfusion caused by primary cardiac dysfunction, most commonly precipitated by acute myocardial infarction [1]. Despite advances in revascularization, pharmacotherapy, and mechanical circulatory support, short-term mortality from acute myocardial infarction-related CS has remained persistently high at approximately 40%-50% [2-4]. CS

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complicates ST-segment elevation myocardial infarction (STEMI) more frequently than other acute coronary syndrome subtypes and remains the leading cause of in-hospital mortality in this population [5,6].

Early identification and prompt reperfusion are central to improving outcomes, as delays in primary percutaneous coronary intervention or coronary artery bypass grafting are associated with worse morbidity and mortality [2]. Traditional clinical predictors of CS in STEMI include older age, anterior infarct location, hypertension, and diabetes mellitus [7]. However, the applicability of these predictors across populations remains uncertain, particularly in regions with different demographic and risk factor profiles.

Patients with acute coronary syndrome in Saudi Arabia have been shown to present approximately a decade younger than patients in Western countries, with a substantially higher prevalence of diabetes mellitus and a different risk factor distribution [8]. A previous single-center Middle Eastern study has also reported a substantial burden of CS among patients presenting with STEMI in the region [9]. The Gulf-CS Registry, which included Saudi participation, reported an overall incidence of acute myocardial infarction-related CS of approximately 4.1%, with STEMI being the predominant presentation [10]. Current Saudi clinical practice guidelines emphasize immediate reperfusion and advise against routine non-culprit-vessel intervention during the index admission [11], and the growing adoption of mechanical circulatory support and dedicated cardiac critical care services in tertiary centers reflects ongoing national efforts to improve outcomes in this high-risk group [12].

Despite these developments, contemporary single-center data describing the incidence, clinical phenotype, and independent predictors of CS in Saudi patients with STEMI remain limited. Given the younger age at presentation, the higher burden of diabetes, and the evolving local practice patterns, conventional predictors derived from Western cohorts may not adequately reflect the Saudi context. The present study was therefore conducted to determine (i) the incidence of CS in a contemporary cohort of Saudi patients presenting with STEMI, (ii) the clinical and laboratory characteristics that distinguish patients with CS from those without, and (iii) the independent clinical predictors of CS in this population.

Methods

Study design and setting

This was a single-center, retrospective cohort study conducted at the King Abdulaziz Cardiac Center, King Abdulaziz Medical City, Riyadh, Saudi Arabia, a tertiary cardiac referral center that provides 24-hour primary percutaneous coronary intervention services. The study covered consecutive admissions between 1 January 2019 and 31 December 2024. The study was reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement [13].

Study population

All adult patients admitted to the center with a discharge diagnosis of acute STEMI during the study period were screened for eligibility. Patients were identified through the institutional electronic medical record system (BESTCare) using the diagnostic code corresponding to STEMI, and consecutive eligible patients were included.

Inclusion criteria

Patients were included if they (i) were aged 18 years or older, (ii) presented acutely to the emergency department, and (iii) had a confirmed diagnosis of acute STEMI based on current international STEMI guidelines [14], with subsequent primary percutaneous coronary intervention performed on the culprit vessel.

Exclusion criteria

Patients were excluded if they (i) were younger than 18 years, (ii) had late-presentating myocardial infarction precluding primary percutaneous coronary intervention, or (iii) had received thrombolytic therapy prior to or in place of primary percutaneous coronary intervention.

Study definitions

STEMI was defined, in accordance with current international guidelines, as new ST-segment elevation of at least 1 mm in two or more contiguous electrocardiographic leads in a patient presenting with chest pain or an equivalent ischemic symptom [14]. CS was defined, in accordance with the SHOCK trial criteria, as a systolic blood pressure < 90 mmHg sustained for $\geq$ 1 hour, accompanied by signs of tissue hypoperfusion attributable to cardiac dysfunction and unresponsive to intravenous fluid resuscitation [15]. Hypertension, diabetes mellitus, and dyslipidemia were defined by a pre-existing physician-documented diagnosis in the medical record or by ongoing pharmacological treatment for the corresponding condition at admission. Arrhythmia refers to a clinically significant ventricular arrhythmia (sustained ventricular tachycardia or ventricular fibrillation) documented during the index admission. Infarct location categories (anterior, inferior, and lateral) were assigned on the basis of the affected electrocardiographic territories at presentation and were not mutually exclusive: patients with infarction extending across multiple territories (e.g., anterolateral, inferolateral) were counted under each affected category.

Data collection

Demographic, clinical, electrocardiographic, laboratory, and procedural data were extracted from the electronic medical record by trained reviewers using a standardized case-report form. Variables collected included age, sex, height, cardiovascular risk factors, prior cardiovascular history, infarct location (as defined above), admission and peak (or nadir, as appropriate) laboratory values [troponin, creatine kinase-myocardial band (CK-MB)], N-terminal pro-B-type natriuretic peptide (NT-proBNP), estimated glomerular filtration rate (eGFR), blood urea nitrogen (BUN), hemoglobin, glycated hemoglobin

(HbA1c), serum sodium, and serum potassium], and the occurrence of CS during the index admission. The use of mechanical circulatory support was also recorded. All extracted counts and continuous summary statistics were verified against the source records by a second reviewer.

Ethical approval

The study protocol was reviewed and approved by the Institutional Review Board of the King Abdullah International Medical Research Center (KAIMRC), Riyadh, Saudi Arabia (approval no. IRB/2007/23 date of IRB approval 2/8/2023). Given the retrospective design and the use of de-identified data, the requirement for individual informed consent was waived by the Institutional Review Board. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Statistical analysis

The normality of continuous variables was assessed using the Shapiro-Wilk test and visual inspection of histograms. Normally distributed continuous variables are presented as mean ± standard deviation (SD), and right-skewed laboratory variables (cardiac biomarkers and BUN) are presented as mean ± SD for descriptive consistency with the source database, while between-group comparisons for these variables were performed using the Mann-Whitney *U* test. Categorical variables are presented as frequencies and percentages. Between-group comparisons used Student's independent-samples *t*-test for normally distributed continuous variables, the Mann-Whitney *U* test for non-normally distributed continuous variables, and the chi-square or Fisher's exact test (for expected cell counts <5) for categorical variables. Predictors of CS were assessed using univariate logistic regression. Age, sex, diabetes mellitus, hypertension, and infarct location were forced into the multivariate model a priori on clinical grounds; additional variables were entered if they reached $p < 0.10$ in the univariate analysis. All candidate variables were entered simultaneously, without stepwise selection. Results are reported as odds ratios (OR) with 95% confidence intervals (CI). A two-sided *p*-value < 0.05 was considered statistically significant. All analyses were performed using Stata (version 14; StataCorp LLC, College Station, TX) [16].

Results

Baseline characteristics

A total of 878 patients with STEMI were included in the analysis. The mean age was 57.2 ± 13.6 years, and 760 (86.6%) were male. Cardiovascular risk factors were prevalent: 494 patients (56.3%) had diabetes mellitus, 447 (50.9%) had hypertension, and 403 (45.9%) had dyslipidemia. Anterior STEMI was the most frequent presentation (n = 447; 50.9%), followed by inferior STEMI (n = 394; 44.9%) and lateral STEMI (n = 99; 11.3%); because patients with multi-territory infarction were counted under each affected category, the three categories sum to more than the total cohort (Table 1, footnote †). The mean peak troponin was 102,447 ± 116,821 ng/l, and the mean peak NT-proBNP was 110.5 ± 260.9 pg/ml. All patients presented acutely to the emergency department and underwent primary percutaneous coronary intervention to the culprit vessel.

CS cohort

CS developed in 80 patients, corresponding to an overall incidence of 9.1% (Table 1). Compared with patients without CS, those who developed CS had a higher prevalence of dyslipidemia (58.8% *vs*. 44.6%; $p = 0.016$) and a substantially higher prevalence of ventricular arrhythmia during admission (17.5% *vs*. 5.5%; $p < 0.001$) (Figure 1). No mechanical complications of myocardial infarction were documented in the CS group. Notably, no patient in this cohort received mechanical circulatory support, reflecting local practice patterns during the study period rather than an absence of indication; this point is discussed further below.

Patients with CS had significantly higher admission NT-proBNP and BUN levels and significantly lower eGFR and hemoglobin (all $p \leq 0.012$). Admission troponin did not differ between groups, whereas peak troponin and peak NT-proBNP were significantly higher in patients with CS (both $p < 0.001$). Patients with CS also had a higher prevalence of hypokalemia, hyponatremia, and anemia at their nadir values. No significant differences were observed between groups in age, sex, prevalence of diabetes mellitus or hypertension, or infarct location (Table 1).

Continuous variables are presented as mean ± SD and categorical variables as frequencies (percentages). Between-group comparisons were performed using Student's independent-samples *t*-test for normally distributed continuous variables, the Mann-Whitney *U* test for non-normally distributed continuous variables, and the chi-square or Fisher's exact test for categorical variables. † Infarct location categories are not mutually exclusive; patients with multi-territory infarction are counted in each affected category, so the percentages sum to more than 100%. ‡ Right-skewed laboratory variables; the mean ± SD is shown for descriptive consistency with the source database, but the between-group comparison and the corresponding p-value are based on the Mann-Whitney U test. BUN, blood urea nitrogen; CK-MB, creatine kinase-myocardial band; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; NT-proBNP, N-terminal pro–B-type natriuretic peptide; STEMI, ST-segment elevation myocardial infarction.

Univariate and multivariate logistic regression

In univariate logistic regression analysis, dyslipidemia (OR 1.77, 95% CI 1.11-2.82; $p = 0.017$) and arrhythmia (OR 3.64, 95% CI 1.89-6.98; $p < 0.001$) were significantly associated with CS. Among laboratory parameters, higher peak troponin ($p < 0.001$), higher peak NT-proBNP (OR 1.001, 95% CI 1.001-1.002; $p < 0.001$), and higher peak BUN (OR 1.09, 95% CI 1.06-1.11; $p < 0.001$) were associated with increased odds of CS, whereas lower nadir hemoglobin (OR 0.96, 95% CI 0.95-0.97; $p < 0.001$), lower nadir potassium (OR 0.09, 95% CI 0.05-0.16; $p < 0.001$), and lower admission eGFR (OR 0.99, 95% CI 0.98-0.99; $p = 0.001$) were

Table 1. *Baseline characteristics of the study population, overall and stratified by the occurrence of CS.*

Variable	Total (*n* = 878)	CS (*n* = 80; 9.1%)	No CS (*n* = 798; 90.9%)	*p*-value
Age, years	57.16 ± 13.59	59.66 ± 13.56	56.91 ± 13.58	0.084
Female sex, *n* (%)	118 (13.44)	9 (11.25)	109 (13.66)	0.547
Height, cm	165.64 ± 11.18	165.45 ± 9.20	165.66 ± 11.36	0.872
Cardiovascular risk factors				
Hypertension, *n* (%)	447 (50.91)	41 (51.25)	406 (50.88)	0.949
Diabetes mellitus, *n* (%)	494 (56.26)	50 (62.50)	444 (55.64)	0.238
Dyslipidemia, *n* (%)	403 (45.90)	47 (58.75)	356 (44.61)	0.016
Past cardiovascular history				
Prior myocardial infarction, *n* (%)	119 (13.55)	10 (12.50)	109 (13.66)	0.773
Arrhythmia, *n* (%)	58 (6.61)	14 (17.50)	44 (5.51)	< 0.001
Stroke, *n* (%)	50 (5.69)	5 (6.25)	45 (5.64)	0.822
Infarct location †				
Anterior STEMI, *n* (%)	447 (50.91)	47 (58.75)	400 (50.13)	0.141
Inferior STEMI, *n* (%)	394 (44.87)	31 (38.75)	363 (45.49)	0.248
Lateral STEMI, *n* (%)	99 (11.28)	7 (8.75)	92 (11.53)	0.454
Laboratory values on admission				
Troponin, ng/l ‡	17,474.5 ± 53,484.1	18,035.6 ± 62,509.0	17,418.0 ± 52,534.2	0.922
CK-MB, ng/ml ‡	138.98 ± 1,137.73	106.43 ± 178.16	142.27 ± 1,192.57	0.790
NT-proBNP, pg/ml ‡	74.01 ± 180.80	143.75 ± 342.70	67.02 ± 154.26	< 0.001
eGFR, ml/min/1.73 m²	79.97 ± 27.15	70.28 ± 28.79	80.93 ± 26.81	0.001
BUN, mmol/l ‡	6.17 ± 3.70	7.16 ± 4.32	6.07 ± 3.62	0.012
Hemoglobin, g/l	148.18 ± 22.28	141.60 ± 28.17	148.84 ± 21.51	0.005
HbA1c, %	7.65 ± 2.38	7.81 ± 2.40	7.64 ± 2.38	0.555
Sodium, mmol/l	135.96 ± 7.04	136.29 ± 7.60	135.93 ± 6.98	0.662
Potassium, mmol/l	4.36 ± 1.33	4.26 ± 0.68	4.36 ± 1.38	0.509
Laboratory values at peak (maximum) or nadir (minimum)				
Peak troponin, ng/l ‡	102,447.2 ± 116,820.8	152,980.3 ± 159,350.3	97,329.9 ± 110,468.2	< 0.001
Peak CK-MB, ng/ml ‡	193.95 ± 260.77	233.65 ± 275.89	189.93 ± 259.03	0.156
Peak NT-proBNP, pg/ml ‡	110.49 ± 260.91	249.01 ± 556.22	96.52 ± 204.84	< 0.001
Peak BUN, mmol/l ‡	9.68 ± 7.88	17.29 ± 12.32	8.92 ± 6.85	< 0.001
Nadir hemoglobin, g/l	123.67 ± 24.79	100.40 ± 25.22	126.00 ± 23.52	< 0.001
Nadir sodium, mmol/l	133.03 ± 6.14	131.75 ± 8.35	133.16 ± 5.86	0.051
Nadir potassium, mmol/l	3.87 ± 1.39	3.42 ± 0.38	3.91 ± 1.45	0.003

associated with reduced odds of CS. Age, hypertension, diabetes mellitus, infarct location, CK-MB, HbA1c, and admission sodium were not significantly associated with CS.

In multivariate logistic regression, female sex emerged as an independent predictor of lower odds of CS (OR 0.18, 95% CI 0.06-0.52; $p = 0.002$), despite not being significant in the univariate analysis. Dyslipidemia (OR 2.39, 95% CI 1.22-4.70; $p = 0.011$) and arrhythmia (OR 3.69, 95% CI 1.63-8.37; $p = 0.002$) remained independently associated with higher odds of CS, as did peak troponin ($p = 0.040$; per-unit OR rounds to 1.000 because the unit is small, Table 2 footnote ‡) and peak BUN (OR 1.05, 95% CI 1.01-1.08; $p = 0.013$). Lower nadir hemoglobin (OR 0.97, 95% CI 0.96-0.99; $p < 0.001$) and lower nadir potassium (OR 0.16, 95% CI 0.07-0.34; $p < 0.001$) were also independently associated with the development of CS. Age, hypertension, diabetes mellitus, infarct location, NT-proBNP, CK-MB, sodium, eGFR, and HbA1c were not independently associated with CS after adjustment (Table 2, Figure 2). The multivariate OR for the infarct location categories should be interpreted with caution because of their wide CI (see Strengths and limitations).

Variables forced into the multivariate model a priori on clinical grounds were age, sex, diabetes mellitus, hypertension, and infarct location. Remaining variables were entered if they reached $p < 0.10$ in the univariate analysis. All candidate variables were entered simultaneously; no stepwise selection was applied. Female sex is reported against male sex as the reference category. OR are reported per 1-unit increase in the corresponding measurement unit. † Infarct location categories are not mutually exclusive (Table 1, footnote †). ‡ Because the unit (ng/l for troponin; pg/ml for NT-proBNP) is small relative to the observed range, the per-unit OR rounds to 1.000; statistical inference is based on the p-value, and the corresponding per-SD interpretation is provided in the text. BUN, blood urea nitrogen; CI, confidence interval; CK-MB, creatine kinase–myocardial

Table 2. *Univariate and multivariate logistic regression analyses of clinical and laboratory predictors of CS among patients with STEMI.*

Variable	Univariate			Multivariate		
	OR	95% CI	*p*-value	OR	95% CI	*p*-value
Age, per year	1.015	0.998-1.031	0.085	1.000	0.974-1.026	0.979
Female sex (*vs.* male)	0.801	0.389-1.650	0.548	0.176	0.060-0.516	0.002
Cardiovascular risk factors						
Hypertension	1.015	0.641-1.607	0.949	0.749	0.356-1.574	0.445
Diabetes mellitus	1.329	0.827-2.134	0.239	1.098	0.501-2.407	0.815
Dyslipidemia	1.768	1.109-2.819	0.017	2.394	1.219-4.704	0.011
Past cardiovascular history						
Prior myocardial infarction	0.903	0.452-1.805	0.773	0.896	0.358-2.242	0.814
Arrhythmia	3.635	1.894-6.976	< 0.001	3.688	1.625-8.372	0.002
Stroke	1.116	0.430-2.896	0.822	0.265	0.064-1.103	0.068
Infarct location †						
Anterior STEMI	1.417	0.889-2.259	0.143	2.894	0.622-13.472	0.176
Inferior STEMI	0.758	0.473-1.214	0.249	2.915	0.610-13.934	0.180
Lateral STEMI	0.736	0.329-1.646	0.455	0.744	0.275-2.014	0.560
Laboratory values at peak or nadir						
Peak troponin, per ng/l ‡	1.000	1.000-1.000	< 0.001	1.000	1.000-1.000	0.040
Peak CK-MB, per ng/ml	1.000	0.999-1.001	0.174	0.999	0.998-1.001	0.276
Peak NT-proBNP, per pg/ml ‡	1.001	1.001-1.002	< 0.001	1.000	0.999-1.001	0.744
Peak BUN, per mmol/l	1.088	1.063-1.113	< 0.001	1.046	1.010-1.084	0.013
Nadir hemoglobin, per g/l	0.961	0.952-0.971	< 0.001	0.973	0.960-0.987	< 0.001
Nadir sodium, per mmol/l	0.978	0.952-1.004	0.092	1.021	0.962-1.084	0.486
Nadir potassium, per mmol/l	0.087	0.048-0.155	< 0.001	0.156	0.071-0.343	< 0.001
Admission laboratory values						
eGFR, per ml/min/1.73 m²	0.986	0.978-0.994	0.001	1.002	0.990-1.014	0.754
HbA1c, per %	1.029	0.935-1.134	0.555	0.972	0.847-1.115	0.688

band; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; NT-proBNP, N-terminal pro-B-type natriuretic peptide; OR, odds ratio; STEMI, ST-segment elevation myocardial infarction.

Discussion

In this contemporary single-center cohort of 878 Saudi patients presenting with STEMI, CS occurred in 9.1% of patients and remained a high-risk complication despite universal access to primary percutaneous coronary intervention. Female sex (which emerged only after multivariate adjustment), dyslipidemia, ventricular arrhythmia during admission, higher peak troponin and BUN, and lower nadir hemoglobin and potassium were independent predictors of CS. Importantly, classical Western predictors such as advanced age, anterior infarct location, hypertension, and diabetes mellitus were not independently associated with CS in this cohort. CS is the most lethal complication of STEMI, with short-term mortality persisting at approximately 40% despite recent advances in care [15], underscoring the need for vigilant early identification of patients at risk.

The independent association of female sex with lower adjusted odds of CS in our cohort merits comment. Female sex was not significant in the univariate analysis and only emerged after multivariate adjustment, a change that may reflect a suppressor effect once other predictors were held constant but could equally reflect a sparse-data artifact: only 9 of the 118 female patients in the cohort developed CS, and the resulting wide multivariate CI (0.06-0.52) is consistent with such an artefact. This contrasts with prior reports that women are more prone to CS in the setting of STEMI and tend to have worse short-term outcomes [17]. The finding should therefore be interpreted with caution and confirmed in larger samples. Ventricular arrhythmia, a recognized cause of hemodynamic instability in STEMI, impairs cardiac output and may precipitate or aggravate CS; hypokalemia is a well-documented driver of ventricular arrhythmia, particularly ventricular fibrillation, in the setting of STEMI [18], which is in keeping with our finding that lower nadir potassium was an independent predictor of CS. Higher peak troponin reflects a larger infarct size, which in turn is associated with greater left ventricular systolic dysfunction and a higher risk of CS [19]. Higher low-density lipoprotein cholesterol (LDL-C) levels have been associated with larger infarct size in patients with STEMI [20], providing a biological rationale for the independent association between dyslipidemia and CS observed in our cohort. Treatment of dyslipidemia remains challenging in clinical practice, with many patients failing to reach guideline-recommended LDL-C targets. Anemia is common in patients with STEMI and

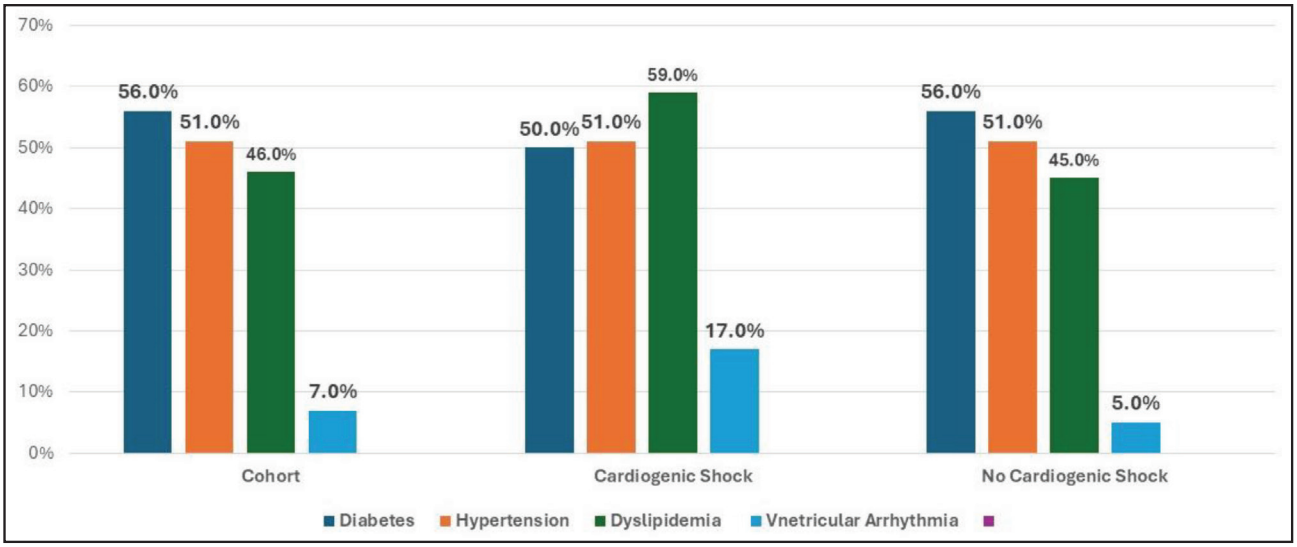

Figure 1. *Prevalence of the principal clinical predictors of CS in the study cohort, comparing patients with and without CS.*

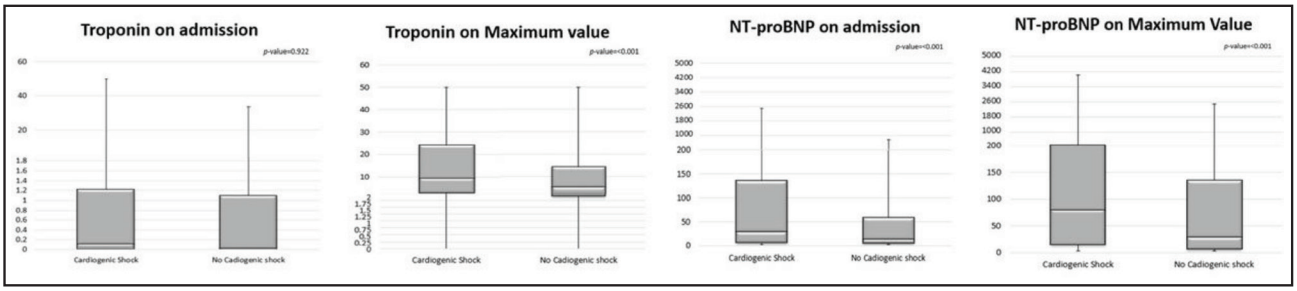

Figure 2. *Admission and peak troponin and NT-proBNP levels, stratified by the occurrence of CS.*

has been shown to increase the risk of heart failure and adverse outcomes in this setting [21], consistent with the independent association between lower nadir hemoglobin and CS in our analysis.

Multiple studies have reported clinical, angiographic, and echocardiographic predictors of short- and long-term outcomes in patients with CS, and emerging machine-learning approaches may further support the early identification of high-risk patients before overt hemodynamic deterioration. In contrast to prior reports, age, infarct location, and diabetes mellitus did not predict CS in our cohort. A plausible explanation is the broad adoption of contemporary STEMI care pathways, including rapid recognition and timely primary percutaneous coronary intervention, which together reduce the incidence of complications. Furthermore, advances in diabetes therapy have improved glycemic control and reduced macrovascular complications; the mean HbA1c in our cohort was 7.65% ± 2.38%, suggesting reasonably good metabolic control overall.

The complete absence of mechanical circulatory support in our cohort, despite a 9.1% incidence of CS, also deserves comment. During the study period, device availability and institutional practice favored pharmacological and revascularization-based management of CS, with mechanical circulatory support reserved for refractory cases that were not represented in this consecutive sample. This reflects regional practice patterns at the start of the study window and is consistent with the gradual national roll-out of mechanical support programs described in recent reviews [12].

Strengths and limitations

The principal strengths of this study include the contemporary recruitment window (2019-2024), the relatively large single-center sample of consecutive Saudi patients with STEMI, and the standardized electronic record-based data collection in a high-volume tertiary cardiac center with 24-hour primary percutaneous coronary intervention capability. The study also adheres to the STROBE reporting framework for observational research [13].

Several limitations should be acknowledged. First, this was a single-center study, and the findings may not be fully generalizable to other Saudi or regional populations, particularly those treated at centers without round-the-clock primary percutaneous coronary intervention. The availability of timely reperfusion at our center may underestimate the true regional incidence of CS. Second, data on ischemia time, reperfusion time, and right ventricular involvement were not available, and we were unable to adjust for left main or multivessel coronary artery disease in the multivariate model - variables that are known to influence the risk of CS. Third, because of the retrospective design, some data points may be missing or subject to documentation bias, and unmeasured

confounding cannot be excluded. Fourth, the multivariate model included approximately 20 candidate covariates against 80 CS events, giving an events-per-variable ratio of approximately 4 - below the conventional threshold of 10 - which may produce wide CI (notably for the infarct location categories) and a degree of model instability; results should therefore be interpreted as hypothesis-generating. Fifth, several laboratory variables (troponin, CK-MB, NT-proBNP, and BUN) are right-skewed and are presented descriptively as mean ± SD; the between-group comparisons and the corresponding *p*-values shown in Table 1 are based on the Mann-Whitney *U* test. The per-unit OR for troponin and NT-proBNP in Table 2 round to 1.000 because the reporting unit (ng/l for troponin; pg/ml for NT-proBNP) is small relative to the observed range, so statistical inference for these variables is based on the *p*-value rather than the point estimate. Finally, in-hospital and longer-term mortality were not analyzed in this manuscript and will be the focus of future work.

Future research directions

Larger, prospective, multicenter studies enrolling consecutive Saudi and regional patients with STEMI are needed to confirm the independent predictors of CS identified here, to evaluate their prognostic value for in-hospital and longer-term mortality, and to support the development of region-specific risk-stratification tools. Future work should also incorporate angiographic and echocardiographic variables, ischemia and reperfusion times, anthropometric measures, and the use of mechanical circulatory support, and could explore the role of machine-learning models in identifying patients at risk of CS before overt hemodynamic deterioration.

Conclusion

In this contemporary single-center Saudi cohort, CS occurred in approximately 1 in 11 patients presenting with STEMI. The predictors that emerged - most notably ventricular arrhythmia, dyslipidemia, and lower nadir potassium and hemoglobin - were not the classical Western predictors of older age, anterior infarct, hypertension, or diabetes. Larger multicenter studies are warranted to confirm these findings and to inform region-specific risk-stratification and management strategies for CS in STEMI.

List of Abbreviations

BUN	Blood urea nitrogen
CI	Confidence interval
CK-MB	Creatine kinase-myocardial band
CS	Cardiogenic shock
eGFR	Estimated glomerular filtration rate
HbA1c	Glycated hemoglobin
IRB	Institutional review board
KAIMRC	King Abdullah International Medical Research Center
LDL-C	Low-density lipoprotein cholesterol
NT-proBNP	N-terminal pro–B-type natriuretic peptide
OR	Odds ratio
PCI	Percutaneous coronary intervention
SD	Standard deviation
STEMI	ST-segment elevation myocardial infarction
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology

Conflict of interest

The authors declare that they have no competing interests.

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Consent for publication

Not applicable.

Ethical approval and consent to participate

The study was approved by the Institutional Review Board of King Abdullah International Medical Research Center (KAIMRC), Riyadh, Saudi Arabia (Approval No. IRB/2007/23). The requirement for individual informed consent was waived in view of the retrospective design and the use of de-identified data. The study was conducted in accordance with the Declaration of Helsinki.

Availability of data and materials

The de-identified datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request and subject to institutional data-sharing policies.

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Supplementary content (If any) is available online.

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