Vol.19, No.5, Page 341- 353, 2025 http://dx.doi.org/10.59628/jchm.v19i5.1944 ISSN 2958-7476 JMCH

Sana'a University Journal of Medical and Health Sciences

Prediction of Major Adverse Cardiovascular Events Among Yemeni Patients with Acute Coronary Syndrome: A Comparative Study by Gender

Muammar Yahiya Al-Assadi [1] *, Nouradden Noman Aljaber [1], Abdulhafeedh Al-Habeet [2], Osama Al Nono [1] and Ahmed Al-Motarreb[1]

[1]**Department of Cardiology, Faculty of Medicine, Sana'a University, Sana'a City, Yemen,**
[2]**Department of Epidemiology and Biostatistics, Faculty of Medical Sciences, Al-Razi University, Sana'a City, Yemen.**

*Corresponding author:E-mail: Abwabrahymalasdy43@gmail.com

Abstract

Background: Acute coronary syndrome (ACS) poses a significant burden in low- and middle-income countries like Yemen, where delays in care and limited resources elevate the risk of adverse outcomes. Identifying the predictors of in-hospital major adverse cardiovascular events (MACEs) is essential for improving management strategies.

Objective: To assess and compare the predictors of in-hospital MACEs among Yemeni patients diagnosed with ACS, with a specific focus on gender-based differences.

Methods: An observational, retrospective study included 1,743 Yemeni patients aged $\geq$18 years, admitted with ACS to six tertiary hospitals in Sana'a City between January 2020 and December 2023. The primary outcome was in-hospital MACEs including cardiac death, recurrent MI, or revascularization.

Results: MACEs occurred in 12.7% of patients, with a higher occurrence in females (16.5%) and STEMI cases (15.1%). The predictors included older age, STEMI, elevated blood sugar, WBC count, creatinine, low SBP, DBP, and HDL-c. Gender-specific predictors varied, with males showing associations with delayed presentation and prior PCI, and females showing associations with age, STEMI, and increased waist circumference.

Conclusion: Key predictors of MACEs were age, STEMI, elevated creatinine, and waist circumference, with notable gender-based differences suggesting the need for tailored interventions.

Article Info

Keywords:
Major adverse cardiovascular events, coronary artery disease, sex differences, in-hospital outcomes, women's health

Article History:
Received: 17-June-2025,
Revised: 8-August-2025,
Accepted: 28-September-2025,
Available online: 28-October-2025

1. INTRODUCTION

Cardiovascular diseases (CVDs) remain the foremost cause of death worldwide, accounting for approximately 18 million deaths in 2019, representing 32% of global mortality. A substantial majority (exceeding 75 %) of these deaths are concentrated in low- and middle-income countries, underscoring the profound global health disparities and pressing need for equitable access to preventive and therapeutic healthcare services in resource-limited settings [1]. Acute coronary syndrome (ACS), which encompasses ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina, continues to be a leading contributor to global morbidity and mortality. Its clinical burden remains substantial despite significant advancements in diagnostic and therapeutic

strategies, reflecting the ongoing challenges in cardiovascular healthcare delivery [2, 3]. This burden is especially pronounced in low- and middle-income countries, where systemic healthcare disparities, delayed patient presentation, and limited access to advanced cardiac interventions further compound adverse clinical outcomes [4]. In Yemen, a country facing significant socioeconomic and healthcare challenges, ACS continues to pose a major public health concern, with mortality and adverse cardiovascular outcomes frequently reported in both short- and long-term follow-up.

Predicting the risk of major adverse cardiovascular events (MACEs) in patients with ACS is a crucial step in improving patient stratification, guiding therapeutic decisions, and optimizing the use of limited healthcare resources. Numerous risk factors, including age, diabetes mellitus (DM), hypertension (HTN), smoking, and lipid profiles have been studied in relation to ACS outcomes [5, 6]. However, sex-based differences in the clinical presentation, management, and prognosis of ACS have attracted increasing attention in recent years. Evidence suggests that gender may play a pivotal role in influencing both the incidence and outcomes of ACS [7, 8], yet findings remain inconsistent and may vary significantly across different populations.

Sex differences arise from variations in gene expression linked to sex chromosomes as well as from the subsequent effects of sex hormones, which influence gene expression and cardiovascular function. In contrast, gender differences result from sociocultural factors, including the behavioral patterns of women and men, dietary habits, differential environmental exposures, lifestyle, stress, and attitudes toward treatment and prevention. Both sex and gender are important determinants of CVDs [9].

Despite the growing global interest in sex-specific cardiovascular research, there is a paucity of data examining this issue in the Yemeni population. Cultural, social, and healthcare system factors unique to Yemen may contribute to disparities in diagnosis, treatment access, and outcomes between female and male ACS patients.

This study aimed to investigate and compare the predictors of in-hospital MACEs among Yemeni patients diagnosed with ACS, with a specific focus on sex-based differences. By identifying sex-specific risk patterns, this study seeks to contribute valuable insights into improving gender-sensitive strategies for the management of ACS in Yemen and similar settings.

2. METHODS

2.1. Study Design and Population

An observational, retrospective study was conducted between January 2020 and December 2023 to assess the predictors of in-hospital MACEs among Yemeni patients diagnosed with ACS, with a specific focus on gender-based differences, at six tertiary care medical centers in Sana'a City, Yemen (Military Cardiac Center, Azal Hospital, University of Science and Technology Hospital, Hashim Iraqi Hospital, Modern European Hospital, and Al Thawra Modern General Hospital). All patients aged $\geq$ 18 years who were diagnosed with ACS were included in the study, except those for whom hospital records were incomplete or missing.

The study was approved by the Research and Ethics Committees of all six participating hospitals and conducted in accordance with the principles outlined in the 2013 revision of the Declaration of Helsinki. Given that the study involved a retrospective review of medical records, the requirement for informed consent was waived, as previously described [10].

The inclusion and exclusion criteria employed throughout the study period are illustrated in Figure 1, culminating in the determination of the final study cohort subjected to analysis.

2.2. Study Variables and Data Collection

Data for this study were systematically collected from multiple sources, including the hospital's statistics department, detailed patient clinical notes, inpatient care registry, high-dependency unit (HDU) register, catheterization laboratory records, intensive care unit (ICU) register, and discharge registry. To ensure data integrity and reliability, all the information was meticulously cross-verified across these sources. The dataset included demographic, clinical, and laboratory variables such as admission date, sex, age, body mass index (BMI), and key coronary risk factors including smoking status, dyslipidemia, HTN, DM, and blood pressure (BP). Additional clinical information was obtained, including patient comorbidities and medical history, specifically the time elapsed from symptom onset to hospital admission (measured in hours), presence of chronic renal failure (CRF), peripheral arterial disease (PAD), valvular heart disease (VHD), congestive heart failure (CHF), atrial fibrillation (AF), cerebrovascular accident (CVA), and history of cardiac interventions, including coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI).

The definitions and classification criteria for clinical variables, in-hospital adverse events, and diagnoses of ACS were standardized based on the clinical data standards established by the American College of Cardiology (ACC) across all six participating hospitals [11].

2.3. Operational Definitions

Dyslipidemia was defined according to the Adult Treatment Panel III (ATP III) criteria [12] as the presence of any of the following: triglyceride (TG) level $\leq$150 mg/dL, high

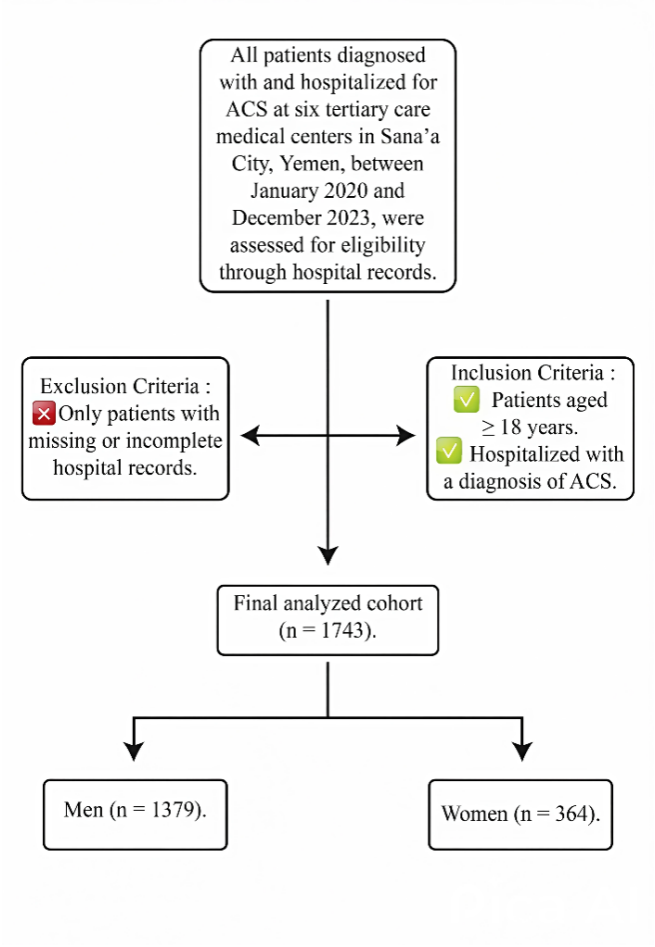

Figure 1. textbf Schematic representation of the patient enrollment process, including the application of inclusion and exclusion criteria leading to the final analytical cohort.

density lipoprotein (HDL)-cholesterol level <35 mg/dL for men or <39 mg/dL for women, total cholesterol level ≥200 mg/dL, low density lipoprotein (LDL)-cholesterol level ≥130 mg/dL, or ongoing treatment for lipid disorders.

DM was defined as a fasting plasma glucose level >126 mg/dL or the use of antidiabetic medications [9].

Smoking status was categorized as never smoker, former smoker (defined as having quit smoking for at least one year), or current smoker. Khat chewing was defined as chewing khat at least three times per week for one hour or more per session during the six months before hospital admission.

Due to the limited number of participants who ceased khat use, former khat chewers were grouped with non-chewers for analytical purposes. Similarly, current and former smokers were combined into a single group and compared to those who had never smoked.

The study's endpoint was defined as the incidence of MACEs, which constituted a composite measure including cardiac mortality, recurrent myocardial infarction (MI), and the necessity for additional revascularization interventions

2.4. Statistical Analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS), version 27. Categorical data were summarized using absolute frequencies and relative percentages to provide a clear description of sample characteristics. The normality of distribution for continuous variables was assessed using the Kolmogorov–Smirnov test, which confirmed that all continuous data followed a normal distribution. Accordingly, these variables were summarized as means with standard deviations (SDs). Comparisons between patients who developed in-hospital MACEs and those who did not were performed using the chi-square test or Fisher's exact test for categorical variables and the independent samples Student's t-test for continuous variables. Furthermore, the Chi-square test was applied to evaluate the unadjusted risk of in-hospital MACEs in the overall cohort and subsequently within sex-specific subgroups (women versus men). To identify independent predictors of in-hospital MACEs, binary logistic regression was performed after adjusting for potential confounders. Variables with a P-value <0.25 in the univariate analysis were considered for entry into the multivariate model. The final

model was adjusted for age, type of ACS, dyslipidemia, BMI, and history of PCI. The results are expressed as odds ratios (ORs) accompanied by their respective 95% confidence intervals (CIs), providing a measure of both the magnitude and direction of the associations observed. A two-tailed P-value of less than 0.05 was used as the threshold to determine statistical significance.

3. RESULTS

3.1. Baseline Demographic and Clinical Characteristics of the Study Cohort

A total of 1,743 patients diagnosed with ACS, including 1,379 males and 364 females. Among the entire cohort, 12.7% (n = 220) (160 males and 60 females) developed in-hospital MACEs.

The baseline clinical and demographic characteristics of the study participants, categorized by the presence or absence of in-hospital MACEs, are presented in Table 1. Patients who developed MACEs were, on average, significantly older than those who did not (mean age: 61.7 ± 12.7 years vs. 57.7 ± 12.3 years; $P < 0.001$). Furthermore, the occurrence of MACEs was significantly higher in female patients (n = 60; 16.5%) than in male patients (n = 160; 11.6%; $P = 0.013$).

Patients presenting with STEMI exhibited a significantly higher MACE rate (n = 184; 15.1%) than those with NSTEMI (n = 36; 6.8%; $P < 0.001$). MACEs were also more frequently observed in patients with comorbid conditions, including dyslipidemia, HTN, DM, CRF, congestive cardiac failure (CCF), CVA, and a history of prior PCI and CABG.

Table 2 provides a summary of the clinical characteristics, biochemical parameters, guideline-directed medical therapy, and procedures among the study participants, categorized according to the presence or absence of MACEs. Patients who developed MACEs had higher levels of random blood sugar (RBS), fasting blood sugar (FBS), white blood cell count (WBC), and serum creatinine, along with reduced levels of SBP, DBP, and HDL-c. Conversely, the use of DAPT upon arrival, in-hospital BB, ACEIs/ARBs (angiotensin-converting enzyme inhibitors/angiotensin receptor blockers), and statins were significantly lower among the patients who developed MACEs.

Among the 1,743 patients diagnosed with ACS, 1,019 (58.5%) underwent coronary angiography (CAG) within the first 24 hours of hospital admission. The procedure was performed more frequently in patients who subsequently experienced MACEs. compared to those who did not (65.0% vs. 57.5%). Likewise, PCI was more commonly performed in patients who developed MACEs than in those without MACEs (52.7% vs. 42.7%).

3.2. Analysis of the Relationship Between Baseline Clinical and Biochemical Risk Factors and the Occurrence of MACEs

Analysis of the relationship between baseline clinical and biochemical risk factors and the occurrence of MACEs using univariate and multivariate regression analyses is presented in Tables 3 and 4. In the univariate regression analysis, baseline clinical and biochemical variables found to be significantly associated with MACEs included older age, female sex, STEMI, elevated BMI, dyslipidemia, HTN, DM, CRF, CCF, CVA, history of PCI or CABG, increased heart rate (HR), high FBS, elevated WBC, and elevated serum creatinine levels. Additionally, lower SBP, DBP, FBS, and HDL-c levels were also associated with MACEs.

In the multivariate regression analyses, all variables that were significantly associated with MACEs in the univariate analysis remained significant, except for prior PCI. In addition, prior MI emerged as an independent predictor of MACEs with an adjusted odds ratio (AOR) of 2.499 (95% CI: 1.627–3.837, $P < 0.001$).

3.3. Subgroup Analysis by Gender

In the gender-stratified analysis, the following variables were associated with MACEs in males: old age, STEMI, higher BMI, dyslipidemia, HTN, DM, CRF, CCF, CVA, and previous CABG. In females, old age, STEMI, and elevated waist circumference (WC) were associated with MACEs.

In addition to the variables that showed a significant association with MACEs in the univariate regression analysis in males, five variables emerged as statistically significant independent predictors of MACEs in the multivariate regression model for males. These included elevated WC (AOR, 1.01; 95% CI, 1.00-1.02; P = 0.049), lower HR (AOR, 0.95; 95% CI, 0.91-0.98, P = 0.005), elevated TG (AOR, 1.01; 95% CI, 1.00-1.02; P <0.001), WC (AOR, 1.01; 95% CI, 1.00-1.02, P = 0.049), previous PCI (AOR, 4.31; 95% CI, 1.48-12.5; P = 0.007), and time from symptom onset to admission (AOR, 1.03; 95% CI, 1.00-1.06; P = 0.031). (See Tables 5 and 6).

Table 7 presents the results of the univariate and multivariate regression analyses of the GDMT associations. and invasive procedures for MACEs. in the overall study population, and stratified according to sex. In the univariate analysis, DAPT, BB, ACEIs/ARBs, and statins were significantly associated with a lower risk of MACEs in the overall population and in women; in men, this association persisted for BB, ACEIs/ARBs, and statins, but not for DAPT. Conversely, CAG and PCI were significantly associated with a higher risk of MACEs, except CAG in men.

In the multivariate analysis, these associations remained

Table 1. Baseline Characteristics of Study Participants Stratified by the Occurrence of Major Adverse Cardiovascular Events

Variables	Overall (n= 1743)	With MACEs (n = 220)	Without MACEs (n = 1523)	P
Age, mean ± SD	58.2 ± 12.5	61.7 ± 12.7	57.7 ± 12.3	0.000*
Gender, n (%)				
Male	1379 (79.1)	160 (11.6)	1219 (88.4)	0.000*
Female	364 (20.9)	60 (16.5)	304 (83.5)	
ACS type, n (%)				
STEMI	1216 (69.8)	184 (15.1)	1032 (84.9)	0.000*
NSTEMI*	527 (30.2)	36 (6.8)	491 (93.2)	
Smoking status, n (%)				
Smoker	1258 (72.2)	155 (12.3)	1103 (87.7)	0.543
Nonsmoker	485 (27.8)	65 (13.4)	420 (86.6)	
Khat chewing status, n (%)				
Chewer	1348 (77.3)	162 (12.0)	1186 (88.0)	0.161
Nonchewer	395 (22.7)	58 (14.7)	337 (85.3)	
Medical history, n (%)				
Dyslipidemia, n (%)	230 (13.2)	51 (22.2)	179 (77.8)	0.000*
HTN, n (%)	603 (34.6)	95 (15.8)	508 (84.2)	0.004*
DM, n (%)	403 (23.1)	67 (16.6)	336 (83.4)	0.006*
CRF, n (%)	9 (0.5)	5 (55.6)	4 (44.4)	0.003*
VHD, n (%)	15 (0.9)	3 (20.0)	12 (80.0)	0.292
AF, n (%)	37 (2.1)	6 (16.2)	31 (83.8)	0.508
Prior Angina, n (%)	500 (28.7)	72 (14.4)	428 (85.6)	0.156
Prior MI, n (%)	263 (15.1)	41 (15.6)	222 (84.4)	0.116
Prior CCF, n (%)	69 (3.9)	18 (26.1)	51 (73.9)	0.001*
Prior CVA, n (%)	71 (4.1)	15 (21.1)	56 (78.9)	0.028*
PAD, n (%)	32 (1.8)	6 (18.8)	26 (81.3)	0.292
Previous PCI, n (%)	91 (5.2)	5 (5.5)	86 (94.5)	0.019*
Previous CABG, n (%)	19 (1.1)	6 (31.6)	13 (68.4)	0.012*
FH, n (%)	111 (6.3)	13 (11.7)	98 (88.3)	0.765
Time from symptom onset to admission (hours), mean ± SD	12.6 ± 6.4	13.1 ± 6.2	12.5 ± 6.4	0.141

ACS, acute coronary syndrome; STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; BMI, body mass index; WC, waist circumference; HTN, hypertension; DM, diabetes mellitus; CRF, chronic renal failure; VHD, valvular heart disease; AF, atrial fibrillation; MI, myocardial infarction; CCF, congestive cardiac failure; CVA, cerebral vascular accident; PAD, peripheral artery disease; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; FH, family history.

Table 2. Baseline Clinical Characteristics, Biochemical Parameters, Guideline-Directed Medical Therapy, and Procedures among Study Participants Stratified by the Occurrence of Major Adverse Cardiovascular Events

Variables (mean ± SD)	Overall (n= 1743)	With MACEs (n = 220)	Without MACEs (n = 1523)	P
BMI (kg/m^2), mean ± SD	25.5 ± 5.1	26.9 ± 5.8	25.3 ± 4.9	0.000*
WC (cm), mean ± SD	90.6 ± 13.5	91.0 ± 18.4	90.6 ± 12.6	0.788
HR, bpm	89.2 ± 21.3	92.2 ± 28.8	88.7 ± 20.0	0.086
SBP, mmHg	129.0 ± 31.6	112.2 ± 43.5	131.4 ± 28.7	0.000*
DBP, mmHg	80.8 ± 20.0	69.4 ± 28.9	82.5 ± 17.8	0.000*
RBS, mg/dl	180.0 ± 94.5	194.0 ± 104.9	178.1 ± 92.9	0.001*
FBS, mg/dl	125.1 ± 57.2	140.5 ± 75.1	123.3 ± 54.6	0.020*
HbA1c, %	8.0 ± 2.7	8.8 ± 3.3	7.9 ± 2.6	0.157
TC, mg/dl	221.3 ± 60.2	221.2 ± 60.4	221.3 ± 60.3	0.997
TGs, mg/dl	188.0 ± 96.5	176.0 ± 77.8	189.5 ± 98.6	0.199
LDL-c, mg/dl	125.3 ± 44.1	122.9 ± 45.1	125.5 ± 44.0	0.905
HDL-c, mg/dl	44.2 ± 18.0	43.77 ± 17.6	47.8 ± 20.2	0.046*
WBC, × 10^9/L	9.1 ± 3.6	10.6 ± 5.1	8.9 ± 3.3	0.000*
Serum creatinine, mg/dl	1.22 ± 0.95	1.59 ± 1.11	1.17 ± 0.80	0.002*
In-hospital GDMT and procedures, n (%)				
DAPT	1533 (88.0)	178 (80.9)	1355 (89.0)	0.001*
BB	1104 (63.3)	80 (36.4)	1024 (67.2)	0.000*
ACEIs/ARBs	1332 (76.4)	113 (51.4)	1219 (80.0)	0.000*
Statins	1630 (93.5)	185 (84.1)	1445 (94.9)	0.000*
CAG	1019 (58.5)	143 (65.0)	876 (57.5)	0.035*
PCI	766 (43.9)	116 (52.7)	650 (42.7)	0.003*

ACEIs/ARBs, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers; BB, beta blocker; BMI, body mass index; CAG, coronary angiography; cm, Centimeter; kg/m, kilograms per meter; L, liter; mg/dl, milligrams per deciliter; mmHg; millimetres of mercury; DAPT, dual antiplatelet therapy; DBP, diastolic blood pressure; FBS, fasting blood sugar; GDMT, guideline-directed medical therapy; HbA1c, glycated hemoglobin; HDL-c, high-density lipoprotein cholesterol; HR, heart rate; LDL-c, low-density lipoprotein cholesterol; MACEs, major adverse cardiac events; PCI, percutaneous coronary intervention; RBS, random blood sugar; SBP, systolic blood pressure; TC, total cholesterol; TGs, triglycerides; WBC, white blood cell count; WC, waist circumference.

Table 3. Univariate and multivariate regression analyses of baseline clinical risk factors associated with major adverse cardiovascular events among all study participants

predictors	Univariate analysis		Multivariate model*	
	COR (95% CI	P	AOR (95% CI)	P
Age	1.026 (1.015-1.038)	0.000*	1.028 (1.016-1.040)	0.000*
Female gender	1.504 (1.089-2.075)	0.003*	1.508 (1.082-2.103)	0.015*
STEMI	2.432 (1.675-3.531)	0.000*	2.412 (1.645-3.539)	0.000*
Current or ex-smoker	0.908 (0.666-1.239)	0.543		
Khat chewer	0.794 (0.574-1.097)	0.161	0.961 (0.673-1.372)	0.826
Dyslipidemia	2.266 (1.597-3.215)	0.000*	2.076 (1.425-3.023)	0.000*
HTN	1.514 (1.138-2.021)	0.004*	1.442 (1.066-1.948)	0.017*
DM	1.546 (1.130-2.112)	0.004*	1.535 (1.107-2.129)	0.010*
CRF	8.834 (2.355-33.147)	0.001*	11.344 (2.848-45.185)	0.001*
VHD	1.740 (0.481-6.219)	0.393		
AF	1.349 (0.552-3.273)	0.507		
Prior Angina	1.248 (0.913-1.687)	0.157	1.686 (1.215-2.338)	0.002*
Prior MI	1.346 (0.920-1.934)	0.117	2.499 (1.627-3.837)	0.000*
Prior CCF	2.573 (1.476-4.490)	0.001*	4.297 (2.307-8.003)	0.000*
Prior CVA	1.9125 (1.062-3.451)	0.030*	1.977 (1.066-3.666)	0.031*
PAD	1.618 (0.654-3.968)	0.297		
Previous PCI	0.387 (0.150-0.962)	0.042*	0.568 (0.222-1.455)	0.239
Previous CABG	3.252 (1.224-8.656)	0.018*	5.598 (1.972-15.894)	0.001*
FH	0.915 (0.501-1.653)	0.765		
Time from symptom onset to admission	1.016 (0.993-1.048)	0.141	1.015 (0.992-1.039)	0.198

COR, crude odds ratio; AOR, adjusted odds ratio; STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; HTN, hypertension; DM, diabetes mellitus; CRF, chronic renal failure; VHD, valvular heart disease; AF, atrial fibrillation; MI, myocardial infarction; CCF, congestive cardiac failure; CVA, cerebral vascular accident; PAD, peripheral artery disease; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; FH, family history.
*Model was adjusted for age, type of ACS, dyslipidemia, BMI, and previous PCI.

significant, except for PCI in the overall population and in both women and men, and CAG in the overall population and in women. In addition, the association between DAPT and MACEs in men remains insignificant.

4. DISCUSSION

This study aimed to identify the predictors of in-hospital MACEs among Yemeni patients diagnosed with ACS. The findings revealed that advanced age, female sex, STEMI, elevated BMI, dyslipidemia, HTN, DM, CRF, CCF, CVA, prior PCI or CABG, elevated HR, FBS, WBC, serum creatinine, and reduced SBP, DBP, and HDL-c levels were significantly associated with in-hospital MACEs in the entire study population. Gender-stratified analysis revealed that among male patients, older age, STEMI, elevated BMI, dyslipidemia, HTN, DM, CRF, and a history of CABG were significant predictors of in-hospital MACEs. In contrast, among female patients, older age, STEMI, and increased WC were primary factors associated with MACEs. To the best of our knowledge, this is the first observational clinical study to explore independent risk factors for in-hospital MACEs in a Yemeni cohort whose clinical profiles and disease presentations differ significantly from those typically reported in Western populations. One of the unique contributions of this study is its focus on the Yemeni cohort, a population underrepresented in cardiovascular research. Sociopolitical instability, limited healthcare infrastructure, and high rates of untreated chronic conditions pose significant challenges for optimal ACS management in Yemen.

The observed overall MACE rate of 12.7% aligns with previously reported ranges in regional and international studies of ACS patients, though some variability is expected due to differences in population characteristics, healthcare access, and clinical practices [13–15].

In this study, advanced age was identified as an independent predictor of in-hospital MACEs. This trend was observed not only in the overall population but also consistently in both male and female subgroups. This finding is consistent with those of previous studies that have highlighted the strong link between age and cardiovascular risk [16, 17]. Advanced age is associated with endothelial dysfunction, increased arterial stiffness, and a higher prevalence of comorbidities, all of which contribute to poorer outcomes in ACS patients [18–20].

Similarly, the identification of female gender as an independent risk factor for in-hospital MACEs is consistent with a growing body of literature that highlights sex-based disparities in cardiovascular outcomes [18]. Biological, clinical, and sociocultural factors may have contributed to this observation. Women with ACS often present

Table 4. Univariate and multivariate regression analyses of baseline clinical and biochemical risk factors associated with major adverse cardiovascular events among all study participants

Predictors	Univariate analysis		Multivariate model*	
	COR (95% CI	P	AOR (95% CI)	P
BMI	1.056 (1.031-1.082)	0.000*	1.061 (1.034-1.087)	0.000*
WC	1.002 (0.991-1.013)	0.732		
HR	1.017 (1.109-1.044)	0.023*	1.008 (1.001-1.014)	0.028*
SBP	0.983 (0.970-0.985)	0.000*	0.977 (0.973-0.982)	0.000*
DBP	0.972 (0.967-0.972)	0.000*	0.967 (0.960-0.974)	0.000*
RBS	1.019 (1.004-1.020)	0.051	1.001 (0.999-1.003)	0.335
FBS	1.015 (1.003-1.024)	0.003*	1.005 (1.001-1.008)	0.004*
HbA1c	1.110 (0.961-1.28)	0.160	1.109 (0.950-1.295)	0.190
TC	1.002 (0.993-1.011)	0.997		
TGs	0.998 (0.992-1.004)	0.149	0.995 (0.993-0.998)	0.001*
LDL-c	0.990 (0.997-1.003)	0.548		
HDL-c	0.996 (0.985-1.009)	0.027*	1.007 (0.997-1.017)	0.197
WBC	1.113 (1.076-1.150)	0.000*	1.122 (1.082-1.164)	0.000*
Serum creatinine	1.314 (1.149-1.511)	0.000*	1.257 (1.078-1.466)	0.003*

COR, crude odds ratio; AOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; DBP, diastolic blood pressure; FBS, fasting blood sugar; HbA1c, glycated hemoglobin; HDL-c, high-density lipoprotein cholesterol; HR, heart rate; LDL-c, low-density lipoprotein cholesterol; MACEs, major adverse cardiac events; OR, odds ratio; RBS, random blood sugar; SBP, systolic blood pressure; TC, total cholesterol; TGs, triglycerides; WBC, white blood cell count; WC, waist circumference.
* The model was adjusted for age, type of ACS, dyslipidemia, BMI, and previous PCI.

with atypical symptoms, are less likely to receive early invasive interventions, and tend to have a higher burden of comorbidities, which may collectively lead to delayed diagnoses and suboptimal management [10, 21, 22]. Furthermore, the significant association between increased WC and MACEs among women suggests a possible role of central obesity and metabolic dysfunction as sex-specific contributors to cardiovascular risk in this population [23, 24].

The observation that STEMI is more frequently associated with MACEs than NSTEMI corresponds with existing literature, as STEMI typically reflects a complete coronary occlusion, leading to more extensive myocardial damage and complications [25, 26]. Moreover, the association of traditional cardiovascular risk factors, such as DM, HTN, and dyslipidemia, with adverse outcomes is well documented and reflects the cumulative burden of atherosclerosis and metabolic dysfunction [27]

In this study, HTN emerged as an independent risk factor for in-hospital MACEs, consistent with prior findings that link a history of HTN to increased cardiovascular risk [28, 29]. For instance, Madan et al. reported that HTN independently predicted the adverse outcomes of PCI in a cohort of 9,494 patients [30]. Chronic HTN is a well-established contributor to the development of atherosclerosis [31] and is associated with an elevated incidence of CAD [32], chronic kidney disease [33], PAD [34], and CVA [31]. Moreover, it is a major risk factor for MI, heart failure, stroke, and cardiovascular disease [35, 36].

Lower SBP and DBP at admission in patients with ACS are both associated with an increased risk of MACEs during hospitalization. This relationship exists because low SBP often reflects poor cardiac output and impaired tissue perfusion, which may indicate severe cardiac dysfunction such as heart failure or cardiogenic shock [37, 38]. At the same time, low DBP, especially values below 60 mmHg, can compromise coronary perfusion because the heart muscle receives most of its blood supply during diastole [39, 40]. When both SBP and DBP are low, the risk of inadequate oxygen delivery to the myocardium increases, further increasing the likelihood of ischemia, arrhythmias, and adverse outcomes [37, 40, 41]. Studies have demonstrated a U- or J-shaped relationship, where both very low and very high blood pressure values are linked to worse outcomes; in particular, SBP < 110 mmHg and DBP < 60 mmHg are associated with significantly higher rates of in-hospital complications and mortality in ACS patients [37, 39, 40]. Therefore, monitoring and maintaining adequate SBP and DBP are critical for reducing the risk of in-hospital MACEs in patients with ACS.

Table 5. Gender-stratified univariate and multivariate regression analyses of baseline clinical predictors for in-hospital major adverse cardiovascular events.

Predictors	Univariate analysis		Multivariate model*	
	COR (95% CI)	P	AOR (95% CI)	P
Men				
Age	1.023 (1.016-1.035)	0.000*	1.027 (1.014-1.041)	0.000*
STEMI	2.515 (1.608-3.950)	0.000*	2.394 (1.500-3.822)	0.000*
Smoker	1.24 (0.788-1.987)	0.353		
Khat chewer	0.952 (0.617-1.482)	0.839		
Dyslipidemia	2.674 (1.804-3.974)	0.000*	2.251 (1.475-3.435)	0.000*
HTN	1.626 (1.162-2.283)	0.005*	1.553 (1.092-2.208)	0.014*
DM	1.583 (1.083-2.290)	0.016*	1.641 (1.117-2.409)	0.012*
CRF	5.801 (1.281-26.174)	0.022*	7.672 (1.595-36.896)	0.011*
VHD	2.564 (0.685-9.59)	0.160	3.624 (0.908-14.460)	0.068*
AF	1.320 (0.508-3.471)	0.568		
Prior Angina	1.339 (0.945-1.909)	0.103	1.694 (1.167-2.460)	0.006*
Prior MI	1.431 (0.940-2.177)	0.091	2.016 (1.274-3.195)	0.003*
Prior CCF	2.974 (1.608-5.501)	0.001*	4.251 (1.475-3.435)	0.000*
Prior CVA	2.608 (1.366-4.980)	0.004*	2.744 (2.384-9.437)	0.021*
PAD	2.425 (0.879-6.717)	0.088	2.473 (0.851-7.191)	0.096
Previous PCI	0.370 (0.132-1.034)	0.059	1.196 (0.846-1.691)	0.310
Previous CABG	4.271 (1.566-11.731)	0.005*	5.642 (1.921-16.568)	0.021*
FH	0.745 (0.350-1.58)	0.449		
Time from symptom onset to admission in hours	1.018 (0.996-1.047)	0.187	1.019 (0.993-1.046)	0.160
Women				
Age	1.036 (1.002-1.051)	0.012*	1.032 (1.007-1.058)	0.012*
STEMI	2.465 (1.251-4.846)	0.004*	2.365 (1.022-5.471)	0.044*
Smoker	0.940 (0.519-1.720)	0.848		
Khat chewer	0.789 (0.458-1.377)	0.400		
Dyslipidemia	1.324 (0.626-2.812)	0.472		
HTN	1.116 (0.630-1.934)	0.713		
DM	1.295 (0.727-2.305)	0.384		
CRF	NA			
VHD	NA			
AF	2.558 (0.220-28.62)	0.446		
Prior Angina	0.989 (0.547-0.180)	0.968		
Prior MI	1.130 (0.518-2.473)	0.756		
Prior CCF	1.546 (0.413-5.791)	0.517		
Prior CVA	0.626 (0.131-2.774)	0.532		
PAD	0.491 (0.066-3.961)	0.510		
Previous PCI	0.628 (0.078-5.108)	0.663		
Previous CABG	NA			
FH	1.449 (0.516-4.059)	0.485		
Time from symptom onset to admission in hours	1.011 (0.960-1.053)	0.682		

COR, crude odds ratio; AOR, adjusted odds ratio; STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; HTN, hypertension; DM, diabetes mellitus; CRF, chronic renal failure; VHD, valvular heart disease; AF, atrial fibrillation; MI, myocardial infarction; CCF, congestive cardiac failure; CVA, cerebral vascular accident; PAD, peripheral artery disease; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; FH, family history.
*Model was adjusted for age, type of ACS, dyslipidemia, BMI, and previous PCI.

Table 6. Gender-stratified univariate and multivariate regression analyses of baseline clinical and biochemical predictors for in-hospital major adverse cardiovascular events.

Predictors	Univariate analysis		Multivariate model*	
	COR (95% CI	P	AOR (95% CI)	P
Men				
BMI	1.0780 (1.03-1.10)	0.000*	1.076 (1.044-1.109)	0.000*
WC	1.016 (1.005-1.023)	0.055	0.995 (0.980-1.012)	0.577
HR	1.003 (0.991-1.016)	0.101	1.005 (0.997-1.013)	0.239
SBP	0.989 (0.970-0.993)	0.000*	0.977 (0.972-0.983)	0.000*
DBP	0.971 (0.964-0.988)	0.000*	0.970 (0.962-0.978)	0.000*
RBS	1.001 (0.995-1.014)	0.199	1.001 (0.998-1.003)	0.619
FBS	1.013 (1.005-1.019)	0.011*	1.005 (1.001-1.009)	0.006*
HbA1c	1.103 (0.926-1.328)	0.271		
TC	1.001 (0.990-1.013)	0.749		
TGs	0.995 (0.983-1.008)	0.134	0.994 (0.991-0.997)	0.000*
LDL-c	1.001 (0.992-1.016)	0.758		
HDL-c	1.010 (1.004-1.023)	0.024*	1.009 (0.997-1.021)	0.148
First WBC	1.105 (1.062-1.159)	0.000*	1.118 (1.072-1.166)	0.000*
Serum creatinine	1.324 (1.110-1.157)	0.001*	1.240 (1.034-1.486)	0.020*
Women				
BMI	1.023 (0.972-1.070)	0.309		
WC	1.940 (1.905-1.993)	0.043*	0.976 (0.953-1.000)	0.049*
HR	1.015 (0.994-1.026)	0.107	1.011 (0.997-1.026)	0.132
SBP	0.974 (0.960-0.989)	0.000*	0.977 (0.968-0.986)	0.000*
DBP	0.956 (0.942-0,977)	0.000*	0.958 (0.944-0.973)	0.000*
RBS	1.003 (0.994-1.016)	0.140	1.002 (0.999-1.005)	0.261
FBS	1.005 (0.991-1.013)	0.168	1.004 (0.998-1.010)	0.238
HbA1c	1.090 (0.847-1.426)	0.502		
TC	0.992 (0.986-1.006)	0.461		
TGs	0.993 (0.986-1.009)	0.728		
LDL-c	0.995 (0.989-1.002)	0.057	0.991 (0.982-1.001)	0.066
HDL-c	1.004 (0.987-1.028)	0.556		
WBC	1.128 (1.056-1.210)	0.001*	1.142 (1.059-1.231)	0.001*
Serum creatinine	1.309 (1.019-1.688)	0.042*	1.331 (0.987-1.795)	0.061

AOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; DBP, diastolic blood pressure; FBS, fasting blood sugar; HbA1c, glycated hemoglobin; HDL-c, high-density lipoprotein cholesterol; HR, heart rate; LDL-c, low-density lipoprotein cholesterol; MACEs, major adverse cardiac events; OR, odds ratio; RBS, random blood sugar; SBP, systolic blood pressure; TC, total cholesterol; TGs, triglycerides; WBC, white blood cell count; WC, waist circumference.
*Model was adjusted for age, type of ACS, dyslipidemia, BMI, and previous PCI.

Table 7. Univariate and Multivariate Regression Analyses of Guideline-Directed Medical Therapy and Procedures in Relation to Major Adverse Cardiovascular Events among the Overall Study Population and Stratified by Gender

Therapy/ procedure	Univariate analysis		Multivariate model*	
	COR (95% CI	P	AOR (95% CI)	P
Overall				
DAPT	0.525 (0.362-0.763)	0.001*	0.270 (0.128-0.573)	0.001*
BB	0.278 (0.207-0.374)	0.000*	0.330 (0.181-0.600)	0.000*
ACEIs/ARBs	0.263 (0.197-0.353)	0.000*	0.172 (0.093-0.316)	0.000*
Statins	0.285 (0.186-0.437)	0.001*	0.221 (0.094-0.517)	0.001*
CAG	1.372 (1.021-1.842)	0.036*	1.266 (0.932-1.719)	0.132
PCI	1.498 (1.128-1.989)	0.005*	1.288 (0.954-1.740)	0.099
Men				
DAPT	0.639 (0.408-0.1002)	0.051*	0.664 (0.405-1.026)	0.064
BB	0.268 (0.190-0.377)	0.000*	0.250 (0.175-0.356)	0.000*
ACEIs/ARBs	0.294 (0.209-0.413)	0.000*	0.277 (0.194-0.395)	0.000*
Statins	0.325 (0.195-0.540)	0.000*	0.313 (0.184-0.533)	0.000*
GAG	1.123 (0.797-1.581)	0.507	0.989 (0.695-1.408)	0.952
PCI	1.421 (1.021-1.978)	0.037*	1.190 (0.842-1.683)	0.325
Women				
DAPT	0.304 (0.151-0.614)	0.001*	0.270 (0.128-0.573)	0.001*
BB	0.332 (0.196-0.593)	0.000*	0.330 (0.181-0.600)	0.000*
ACEIs/ARBs	0.185 (0.103-0.332)	0.000*	0.172 (0.093-0.319)	0.000*
Statins	0.208 (0.092-0.471)	0.001*	0.221 (0.094-0.517)	0.001*
GAG	2.812 (1.560-5.069)	0.001*	2.449 (1.337-4.487)	0.004*
PCI	2.090 (1.192-3.665)	0.010*	1.660 (0.910-3.2029)	0.098

ACEIs/ARBs, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers; BB, beta blocker; CAG, coronary angiography; DAPT, dual antiplatelet therapy; GDMT, guideline-directed medical therapy; PCI, percutaneous coronary intervention.
*Model was adjusted for age, type of ACS, dyslipidemia, BMI, and previous PCI.

Biochemical markers such as elevated WBC, FBS, and serum creatinine also demonstrated strong associations with MACEs. An elevated WBC indicates a heightened inflammatory state, which is known to contribute to plaque instability and thrombosis [42]. Similarly, hyperglycemia, even in the absence of overt DM, worsens ischemic outcomes and promotes endothelial injury [43]. Elevated serum creatinine levels reflect underlying renal dysfunction, a known independent predictor of adverse cardiovascular outcomes owing to its association with systemic inflammation, oxidative stress, and impaired clearance of vasoactive substances [44].

In the gender-stratified multivariate analysis, unique predictors of MACEs emerged among male patients, including elevated WC, lower HR, higher TG levels, prior PCI, and delayed hospital presentation. Central obesity, indicated by increased WC, is strongly linked to insulin resistance, inflammation, and atherogenesis [45]. The association of lower HR with worse outcomes may reflect autonomic imbalance or underlying heart failure [46]. Prior PCI may be a marker of severe or recurrent coronary artery disease, whereas delayed presentation contributes to a larger infarct size and more complications [47].

LIMITATIONS OF THE STUDY

Despite the unique contributions of this study, several limitations should be acknowledged when interpreting the findings. First, its hospital-based design may restrict the generalizability of the results to other regions, particularly rural areas or countries with different health care systems. The retrospective and observational nature of the study also introduces potential biases, including selection bias and unmeasured confounding, which limits the ability to make causal inferences. Additionally, certain important treatment-related variables, such as the timing of interventions and adherence to guidelines, were not captured, despite their known impact on in-hospital outcomes. The underrepresentation of female patients may have reduced the statistical power to detect sex-specific associations, especially in the multivariate models. Moreover, the analysis was confined to in-hospital events, without follow-up data on long-term outcomes such as mortality or recurrent ischemic events.

5. CONCLUSIONS

This study provides important insights into the predictors of in-hospital MACEs among Yemeni patients with ACS,

with a particular emphasis on gender-related differences. Advanced age, STEMI, and elevated WC and serum creatinine levels consistently emerged as key predictors across both sexes, while male patients exhibited additional independent predictors, such as elevated TGs, previous PCI, and delayed hospital presentation. These findings underscore the need for tailored risk-stratification approaches and gender-sensitive management strategies to improve short-term outcomes in patients with ACS in resource-limited settings. Future research should focus on prospective studies that include long-term follow-up and incorporate socioeconomic, behavioral, and healthcare access variables. In addition, sex-specific strategies for prevention, diagnosis, and treatment should be further explored to improve outcomes for both male and female patients with ACS in Yemen and similar settings.

6. ACKNOWLEDGMENT

The authors sincerely acknowledge the administrative offices of all six participating hospitals, the data collection teams, and the hospital records department staff for their invaluable time and support in facilitating the data acquisition process.

REFERENCES

[1] WHO. *Cardiovascular diseases (CVDs)*. 2021. URL: https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds).

[2] B. A. Bergmark et al. “Acute coronary syndromes”. In: *The Lancet* 399.10332 (2022), pp. 1347–1358.

[3] A. Timmis et al. “Global epidemiology of acute coronary syndromes”. In: *Nat. Rev. Cardiol.* 20.11 (2023), pp. 778–788.

[4] L. Beza et al. “Acute coronary syndrome treatment delay in low to middle-income countries: a systematic review”. In: *IJC Heart & Vasc.* 35 (2021), p. 100823.

[5] V. Notara et al. “Diabetes mellitus, hypertension and hypercholesterolemia in relation to the 10-year ACS prognosis; the GREECS study”. In: *Curr. Vasc. Pharmacol.* 14.3 (2016), pp. 295–301.

[6] T. Varghese and A. Kumar. “Predisposing risk factors of acute coronary syndrome (ACS): A mini review”. In: *J. Pharm. Sci. Res.* 11.5 (2019), pp. 1999–2002.

[7] R. Pelletier, B. Ditto, and L. Pilote. “A composite measure of gender and its association with risk factors in patients with premature acute coronary syndrome”. In: *Biopsychosoc. Sci. Med.* 77.5 (2015), pp. 517–526.

[8] A. Angarita-Fonseca, A. Peebles, and L. Pilote. “Gender-related factors associated with outcomes of acute coronary syndrome in young female patients”. In: *CJC Open* 6.2 (2024), pp. 370–379.

[9] C. Y. Lee et al. “Sex and gender differences in presentation, treatment and outcomes in acute coronary syndrome: a 10-year study from a multi-ethnic Asian population”. In: *PLoS One* 16.2 (2021), e0246474.

[10] M. Y. Al-Assadi et al. “Sex-related differences in acute coronary syndrome: insights from an observational study in a Yemeni cohort”. In: *Front. Cardiovasc. Med.* 12 (2025), p. 1481917.

[11] G. J. Dehmer et al. “2020 AHA/ACC key data elements and definitions for coronary revascularization: a report of the American College of Cardiology/American Heart Association task force on clinical data standards”. In: *J. Am. Coll. Cardiol.* 75.16 (2020), pp. 1975–2088.

[12] Adults ToHBCi Detection NCEPEPo. *Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III)*. The Program, 2002.

[13] J. Wang et al. “Prediction of major adverse cardiovascular events in patients with acute coronary syndrome: development and validation of a non-invasive nomogram model based on autonomic nervous system assessment”. In: *Front. Cardiovasc. Med.* 9 (2022), p. 1053470.

[14] A. Ulvenstam et al. “Incidence and predictors of cardiovascular outcomes after acute coronary syndrome in a population-based cohort study”. In: *Sci. Reports* 13.1 (2023), p. 3447.

[15] H. Al-Furasi et al. “Predictors of major adverse cardiac events after percutaneous coronary intervention in Sana’a City-Yemen (single center study)”. In: *Al-Razi Univ. J. for Med. Sci.* 5.2 (2021).

[16] M. H. Bae et al. “Usefulness of surgical parameters as predictors of postoperative cardiac events in patients undergoing non-cardiac surgery”. In: *Circ. J.* 78.3 (2014), pp. 718–723.

[17] I-T. Tsai et al. “The burden of major adverse cardiac events in patients with coronary artery disease”. In: *BMC Cardiovasc. Disord.* 17 (2017), pp. 1–13.

[18] A. J. Donato, D. R. Machin, and L. A. Lesniewski. “Mechanisms of dysfunction in the aging vasculature and role in age-related disease”. In: *Circ. Res.* 123.7 (2018), pp. 825–848.

[19] A. A. Damluji et al. “Management of acute coronary syndrome in the older adult population: a scientific statement from the American Heart Association”. In: *Circulation* 147.3 (2023), e32–e62.

[20] M. Oberoi et al. “Age Considerations in the Invasive Management of Acute Coronary Syndromes”. In: *US Cardiol. Rev.* 16 (2022), e14.

[21] J. C. McSweeney et al. “Reliability of the McSweeney Acute and Prodromal Myocardial Infarction Symptom Survey among black and white women”. In: *Eur. J. Cardiovasc. Nurs.* 12.4 (2013), pp. 360–367.

[22] J. G. Canto et al. “Symptom presentation of women with acute coronary syndromes: myth vs reality”. In: *Arch. Intern. Med.* 167.22 (2007), pp. 2405–2413.

[23] K. M. Rexrode et al. “Abdominal adiposity and coronary heart disease in women”. In: *JAMA* 280.21 (1998), pp. 1843–1848.

[24] S. Yusuf et al. “Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study”. In: *The Lancet* 366.9497 (2005), pp. 1640–1649.

[25] ESC Task Force. “2017 ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation”. In: *Russ. J. Cardiol.* 5 (2018), pp. 103–158.

[26] I. Poudel et al. “Major adverse cardiovascular events: an inevitable outcome of ST-elevation myocardial infarction? A literature review”. In: *Cureus* 11.7 (2019).

[27] S. Yusuf et al. “Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study”. In: *The Lancet* 364.9438 (2004), pp. 937–952.

[28] Å. Omstedt et al. “Hypertension predicts major adverse cardiac events after discharge from the emergency department with unspecified chest pain”. In: *Eur. Heart Journal: Acute Cardiovasc. Care* 5.5 (2016), pp. 441–448.

[29] S. Howell et al. “Risk factors for cardiovascular death after elective surgery under general anaesthesia”. In: *Br. J. Anaesth.* 80.1 (1998), pp. 14–19.

[30] P. Madan et al. “Predicting major adverse cardiac events after percutaneous coronary intervention: the Texas Heart Institute risk score”. In: *Am. Heart J.* 155.6 (2008), pp. 1068–1074.

[31] V. J. Dzau. “Atherosclerosis and hypertension: mechanisms and interrelationships”. In: *J. Cardiovasc. Pharmacol.* 15 (1990), S59–S64.

[32] D. Levy et al. “Stratifying the patient at risk from coronary disease: new insights from the Framingham Heart Study”. In: *Am. Heart J.* 119.3 (1990), pp. 712–717.

[33] M. Adamczak et al. “Kidney and hypertension”. In: *Kidney Int.* 61 (2002), S62–S67.

[34] J. M. Murabito et al. “Intermittent claudication: a risk profile from the Framingham Heart Study”. In: *Circulation* 96.1 (1997), pp. 44–49.

[35] A. M. Richards et al. “Antecedent hypertension and heart failure after myocardial infarction”. In: *J. Am. Coll. Cardiol.* 39.7 (2002), pp. 1182–1188.

[36] K. Miura et al. “Relationship of blood pressure to 25-year mortality due to coronary heart disease, cardiovascular diseases, and all causes in young adult men: the Chicago Heart Association Detection Project in Industry”. In: *Arch. Intern. Med.* 161.12 (2001), pp. 1501–1508.

[37] J. Pei et al. “Association between admission systolic blood pressure and major adverse cardiovascular events in patients with acute myocardial infarction”. In: *PLoS One* 15.6 (2020), e0234935.

[38] S. Haryuni et al. “Systolic blood pressure, blood glucose, and mortality of patients with acute coronary syndrome”. In: *Open Access Maced. J. Med. Sci.* 10.B (2022), pp. 1–4.

[39] J. W. McEvoy et al. “Diastolic blood pressure, subclinical myocardial damage, and cardiac events: implications for blood pressure control”. In: *J. Am. Coll. Cardiol.* 68.16 (2016), pp. 1713–1722.

[40] W. L. Chang et al. “Cardiovascular risk in patients with treated isolated diastolic hypertension and isolated low diastolic blood pressure”. In: *J. Am. Heart Assoc.* 13.8 (2024), e032771.

[41] J. Sun, J. Yuan, and B. Li. “SBP is superior to MAP to reflect tissue perfusion and hemodynamic abnormality perioperatively”. In: *Front. Physiol.* 12 (2021), p. 705558.

[42] P. Libby. “Inflammation in atherosclerosis”. In: *Arter. Thromb. Vasc. Biol.* 32.9 (2012), pp. 2045–2051.

[43] S. E. Capes et al. “Stress hyperglycaemia and increased risk of death after myocardial infarction in patients with and without diabetes: a systematic overview”. In: *The Lancet* 355.9206 (2000), pp. 773–778.

[44] A. S. Go et al. “Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization”. In: *New Engl. J. Med.* 351.13 (2004), pp. 1296–1305.

[45] J. P. Després. “Body fat distribution and risk of cardiovascular disease: an update”. In: *Circulation* 126.10 (2012), pp. 1301–1313.

[46] A. Hjalmarson. “The role of heart rate in cardiovascular disease”. In: *Dialogues Cardiovasc. Med.* 15.3 (2010), pp. 179–191.

[47] C. J. Terkelsen et al. “System delay and mortality among patients with STEMI treated with primary percutaneous coronary intervention”. In: *JAMA* 304.7 (2010), pp. 763–771.