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High-Sensitivity Troponin I, C-reactive Protein, and Hypercholesterolemia as Predictors of Acute Cardiovascular Events in Morocco

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The prevalence of cardiovascular diseases (CVDs) is steadily rising in Morocco and across North Africa, largely linked to dyslipidemia and persistent inflammatory states. This study was conducted to evaluate lipid profiles alongside High-Sensitivity Troponin I (hs-cTnI) and C-Reactive Protein (CRP) levels. A cohort of 240 healthy individuals served as controls, compared with 351 patients diagnosed with acute CVD. Patients exhibited significantly higher levels of LDL-C (1.48 ± 0.45 g/L vs. 1.14 ± 0.24 g/L), hs-cTnI (10.2 ± 5.3 ng/mL vs. 3.1 ± 1.8 ng/mL), and CRP (9.8 ± 7.9 mg/L vs. 2.4 ± 1.6 mg/L), with all differences reaching statistical significance ($p < 0.001$). Multivariate logistic regression analysis identified LDL-C, hs-cTnI, and CRP as independent predictors of acute CVD, with odds ratios (OR) of 1.82 (95% CI: 1.45-2.29), 1.06 (95\% CI: 1.03-1.09), and 1.12 (95\% CI: 1.04-1.21), respectively Receiver Operating Characteristic (ROC) curve analysis showed that hs-cTnI had moderate discriminative ability (AUC = 0.70), whereas CRP displayed limited predictive performance (AUC = 0.62). These findings suggest that hs-cTnI, and to a lesser extent CRP, may enhance risk stratification for acute CVD in Moroccan populations, highlighting the need for biomarker-guided protocols that are both effective and resource-conscious in clinical practice.

Keywords: Cardiovascular Diseases, C-Reactive Protein, Hypercholesterolemia, Morocco, North Africa, Troponin I.

Cardiovascular diseases (CVDs) are a leading global health threat, causing approximately 18 million annual deaths—accounting for 32% of all mortality worldwide—with 75% of these deaths occurring in low- and middle-income countries.[1] This burden is starkly evident in Morocco, where CVDs are the predominant cause of death, responsible for 34% of annual mortality.[1]Nearly most cardiovascular-related mortality, about three out of four, happen in low- and middle-income

regions, highlighting the unequal burden these regions face. [1,2]

Morocco exemplifies this trend, having experienced a 42% increase in CVD prevalence since 2005. Currently, these diseases are responsible for 34% of national mortality.[1,3]This growing health crisis is closely linked to rapid urbanization, aging demographics, and a surge in modifiable risk factors such as hypertension (25.3%), tobacco use (45–50%), and sedentary lifestyles (21.1%).[4]

From a pathophysiological perspective, elevated LDL-C is involved in the disruption of endothelial integrity and the progression of atherogenesis.[5] while systemic inflammation, often reflected by height levels of (hs-CRP), promotes plaque instability.[6] Additionally, troponin I (hs-cTnI) serves as a good marker of myocardial damage/ and is widely employed in detecting acute coronary syndromes.[7]Despite well-established roles for these biomarkers in cardiovascular risk assessment, data from North African populations, including Morocco, remain limited.[8]

The primary objective of this study was to assess the prognostic value of LDL cholesterol (LDL-C), high-sensitivity cardiac troponin I (hs-cTnI), and high-sensitivity C-reactive protein (hs-CRP) in Moroccan patients presenting with acute cardiovascular events. Specifically, the research aimed to establish population-specific cutoff values for these biomarkers, investigate potential sex-related variations in their levels, and contribute to the development of diagnostic strategies tailored to the Moroccan healthcare setting.

MATERIALS AND METHODS

Study Design and Participants

This prospective case–control study included 351 consecutive patients admitted with acute cardiovascular events—such as myocardial infarction, heart failure, or ischemic stroke—at Ibn Rochd University Hospital, Casablanca, Morocco. A control group of 240 apparently healthy volunteers was recruited from the Pasteur Institute of Morocco. Controls were matched to cases by sex and age (±5 years). All participants signed written informed consent prior to enrolment. The study was approved by the institutional ethics committee.

Sample Size and Sampling Technique

A formal sample size calculation was not performed in advance. Instead, all eligible patients meeting the inclusion criteria during the study period were consecutively enrolled to maximize statistical power and ensure a representative sample. Control participants were selected through simple random sampling and matched to patients based on age and sex.

Data and Sample Collection

Demographic and clinical information—including age, sex, body mass index (BMI), blood pressure, smoking habits, physical activity, and medical history—was collected using standardized questionnaires. After overnight fasting, venous blood samples were drawn into serum-separator tubes, centrifuged at 2,500 rpm for 15 minutes, and analyzed within two hours of collection.

Laboratory Analyses

All assays were performed at the accredited laboratories of the Pasteur Institute of Morocco.

Lipid profile: Total cholesterol, triglycerides, and HDL-C were quantified enzymatically using the VITROS® 5600 platform (Ortho Clinical Diagnostics). LDL-C was calculated using the Friedewald formula.

High-sensitivity C-reactive protein (hs-CRP): Determined via particle-enhanced immunoturbidimetry on the BN ProSpec® analyzer (Siemens).

High-sensitivity cardiac troponin I (hs-cTnI): Measured using a chemiluminescent immunoassay with a detection limit of 1.2 ng/mL.

Apolipoproteins (ApoA1 and ApoB) were measured in a subset of participants due to limited sample availability and logistical constraints. Missing measurements were not replaced or statistically imputed.

Inclusion and Exclusion Criteria

Cases: Patients hospitalized with acute CVD, diagnosed according to the 2020 European Society of Cardiology STEMI/NSTEMI guidelines. Patients with malignancy or autoimmune disease were excluded.

Controls: Healthy individuals without a history of CVD, diabetes, or hypertension, and with normal lipid profiles (total cholesterol <2 g/L; LDL-C <1 g/L).

General exclusion criteria: Pregnancy, incomplete clinical data, or refusal to provide informed consent.

Statistical Analysis

Continuous variables were reported as mean ± standard deviation (SD), and categorical variables as frequencies or percentages. Data normality was tested using the Shapiro–Wilk method; non-normally distributed variables (e.g., hs-cTnI) were log-transformed before analysis. Between-group comparisons used unpaired *t*-tests for continuous variables and Chi-square tests for categorical variables.

Multivariate logistic regression was performed to identify independent predictors of acute CVD, including covariates with $p < 0.05$ in univariate analysis (age, sex, diabetes, smoking, physical activity, lipid parameters, hs-cTnI, and hs-CRP). Multicollinearity was assessed using variance inflation factors (VIF <5).

Receiver operating characteristic (ROC) curves were used to determine optimal biomarker cutoffs, reporting the area under the curve (AUC), sensitivity, and specificity. Bonferroni correction was applied for multiple comparisons. All statistical analyses were conducted using SPSS v26 (IBM Corp.) and Python, with a significance threshold of $p < 0.05$.

RESULTS

Participant Characteristics

A total of 351 patients with acute cardiovascular disease (CVD) and 240 age- and sex-matched controls were included in the analysis. As summarized in Table 1, significant differences were observed in all baseline characteristics. Patients were, on average, older than controls and had a higher proportion of males. Moreover, patients presented with significantly higher systolic and diastolic blood pressure levels, as well as elevated fasting glucose levels compared to the control group (all *p*-values < 0.001).

Lipid Profile

As detailed in Table 2, the lipid profile of patients was markedly atherogenic compared to controls. Patients exhibited significantly higher levels of total cholesterol, LDL-C, and triglycerides, along with significantly lower levels of HDL-C (all *p* < 0.001). In a subgroup analysis, ApoA1 levels were also significantly reduced in patients (*p* < 0.001), while ApoB levels did not differ significantly between the groups (*p* = 0.078).

Clinical Risk Factors

As shown in Table 3, the prevalence of modifiable risk factors was significantly higher in patients than in controls. Smoking demonstrated the strongest association with acute CVD (OR = 14.55, 95% CI: 7.22–29.36, *p* < 0.001), followed by physical inactivity (OR = 2.88, 95% CI: 2.03–4.09, *p* < 0.001). A significant association was also observed for alcohol consumption; however, the odds ratio was extremely high and statistically unstable due to the absence of exposed individuals in the control group (OR = 53.95, 95% CI: 3.29–882.90, *p* = 0.005), and this result should be interpreted with extreme caution.

Biomarker Distribution

The analysis revealed distinct sex-specific and age-specific patterns in biomarker levels.

For high-sensitivity cardiac troponin I (hs-cTnI), the distribution was significantly influenced by sex. Women exhibited notably lower median values compared to men, although with

Table 1. Baseline demographic and clinical characteristics of study participants

Characteristic	Patients (n = 351)	Controls (n = 240)	*p*-value
Age, years	59.5 ± 10.4	54.8 ± 6.2	<0.001
Sex, male	212 (60.4%)	116 (48.3%)	0.003
Systolic BP, mm Hg	131.3 ± 19.9	121.1 ± 14.5	<0.001
Diastolic BP, mm Hg	75.5 ± 6.8	72.4 ± 6.8	<0.001
Fasting glucose, g/L	1.50 ± 0.11	0.90 ± 0.14	<0.001

Data are presented as mean ± standard deviation or n (%).
BP, blood pressure.
p-values were derived from independent samples t-test for continuous variables and Chi-square test for categorical variables (sex).

considerable overlap in the interquartile ranges between the two groups (Figure 1).

For high-sensitivity C-reactive protein (hs-CRP), a pronounced age and sex-specific pattern was observed. Analysis of age-stratified medians revealed that men experienced a relatively stable, gradual increase in hs-CRP levels with advancing age. In stark contrast, women demonstrated a sharp and significant peak in hs-CRP levels specifically within the 60-69 age group, a trend not observed in their male counterparts (Figure 2).

The chart compares average levels of total cholesterol, LDL, HDL, and triglycerides in patients using bars (n=351) and controls (n=240). Error bars represent standard deviations, "This figure emphasizes a marked increase in CT, bad cholesterol LDL-C, and triglycerides, and a significant decrease in good cholesterol HDL-C among patients (all P < .001)."

Lipid profiles, particularly HDL-mediated lipid transfers, are essential for evaluating cardiovascular risk, as they are closely linked to atherosclerosis, a primary cause of CVDs such as myocardial infarction and stroke.[9] Elevated LDL-C is a key atherogenic factor, with strong evidence from observational, genetic, and clinical studies supporting its role in CVD pathogenesis.[10]

Substantial LDL-C lowering achieved through PCSK9 inhibition has been consistently associated with reduced cardiovascular event rate; reinforcing the principle of minimizing LDL-C levels.[11]HDL-C is traditionally associated with reduced CVD risk; however, interventions to increase HDL-C have not consistently lowered event rates, suggesting that HDL functionality may be more critical than its absolute levels.[12]Elevated triglycerides, often seen in insulin resistance and type 2 diabetes, promote atherosclerosis via Triglyceride-enriched lipoproteins such as VLDL and chylomicrons.[13] Non-HDL cholesterol, which includes all atherogenic lipoproteins, is a useful marker, particularly in hypertriglyceridemic conditions.[14]

Table 2. Comparison of lipid profiles between patients and controls

Parameter	Patients (n = 351)	Controls (n = 240)	*p*-value
Total cholesterol, g/L	1.86 ± 0.43	1.56 ± 0.42	<0.001
LDL-C, g/L	1.48 ± 0.45	1.14 ± 0.24	<0.001
HDL-C, g/L	0.40 ± 0.14	0.49 ± 0.10	<0.001
Triglycerides, g/L	1.80 ± 0.77	0.90 ± 0.34	<0.001
ApoA1, g/L [a]	1.20 ± 0.30 (n=190)	1.45 ± 0.19 (n=140)	<0.001
ApoB, g/L [a]	0.95 ± 0.22 (n=204)	0.90 ± 0.10 (n=140)	0.078

[a] ApoA1 and ApoB analyses were performed in a subset of participants due to sample availability.
Data are presented as mean ± standard deviation. p-values were derived from independent samples t-test.
LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B.
ApoA1 and ApoB analyses were performed in a subset of participants (ApoA1: 190 patients, 140 controls; ApoB: 204 patients, 140 controls) due to sample availability.

Table 3. Association between modifiable risk factors and acute cardiovascular disease

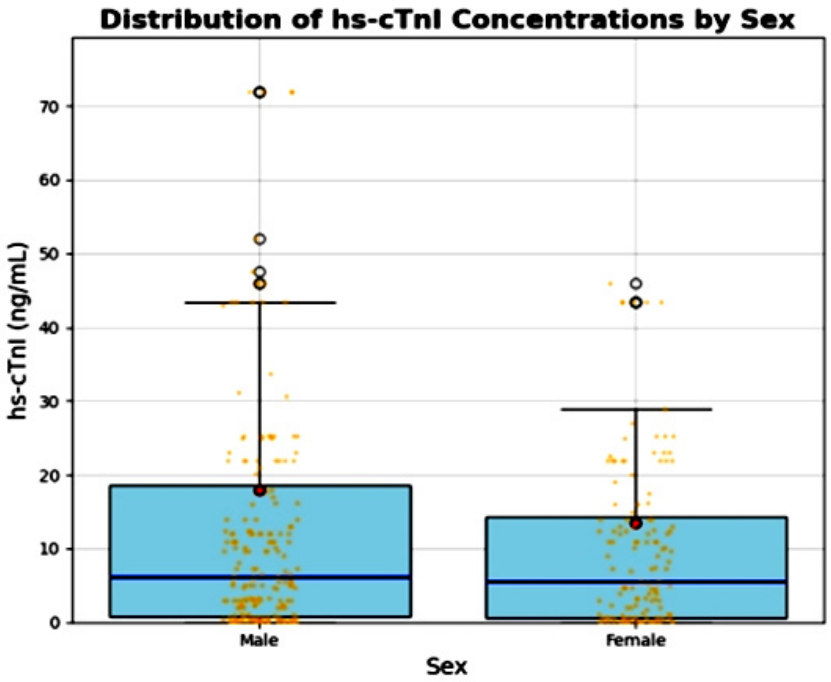
Risk Factor	Patients (n=351) n (%)	Controls (n=240) n (%)	Odds Ratio (OR)	95% CI	*p*-value
Smoking	127 (36.2)	9 (3.8)	14.55	7.22 – 29.36	<0.001
Physical inactivity	266 (75.8)	125 (52.1)	2.88	2.03 – 4.09	<0.001
Alcohol consumption z	35 (10.0)	0 (0.0)	53.95	3.29 – 882.90	0.005

[a] Haldane correction applied due to zero cell count in controls.

The lipoprotein subclass known as Lp(a) is considered a genetically influenced, independent risk factor for the development of cardiovascular disease ; with genetically determined plasma levels that are minimally affected by standard lipid-lowering therapies.[15,16] Guidelines recommend Lp(a) measurement for risk stratification, particularly in intermediate-risk or secondary prevention settings.[17]Lipid parameters are incorporated into risk assessment tools, such as the updated SCORE2 algorithm, to predict 10-year CVD risk and inform clinical management.[18] Novel biomarkers, including Lp(a) and non-HDL cholesterol-derived metrics, are under investigation to enhance residual risk prediction.[19]

Modifiable risk factors, including smoking, physical inactivity, hypertension, and hyperglycemia, significantly contribute to CVD risk.[20] Meta-analyses confirm structured exercise reduces systolic BP by 5-8 mmHg in hypertensives ,[21] [22] reinforcing physical activity's role in Moroccan CVD prevention.[23,24] In Moroccan women, obesity and hypertension are major contributors to the growing CVD burden, exacerbated by an epidemiological transition.[23,25] "T2DM, the most prevalent type of diabetes, how is a critical driver of CVD, accounting for 30-40% of cardiovascular mortality in LMICs , through mechanisms like chronic hyperglycemia, insulin resistance, endothelial dysfunction, inflammation, and dyslipidemia.[26,27] Recent therapeutic advances, Therapies such as SGLT2 inhibitors and GLP-1 receptor agonists not only improve glycemic control but also confer cardioprotective effects in patients with type 2 diabetes.[28,29] Cardiac troponins (cTnI and cTnT) are specific markers of myocardial injury, released upon cardiac cell damage. [30]

This figure displays a box plot of hs-cTnI levels by sex, showing median values, interquartile ranges, and outliers. Women exhibit lower troponin levels than men, consistent with prior studies.[31] Age and sex influence troponin levels, with evidence suggesting age-specific cutoffs may enhance diagnostic accuracy of high-sensitivity assays.[32]Sex-specific cutoffs are proposed by some manufacturers, but their diagnostic benefit

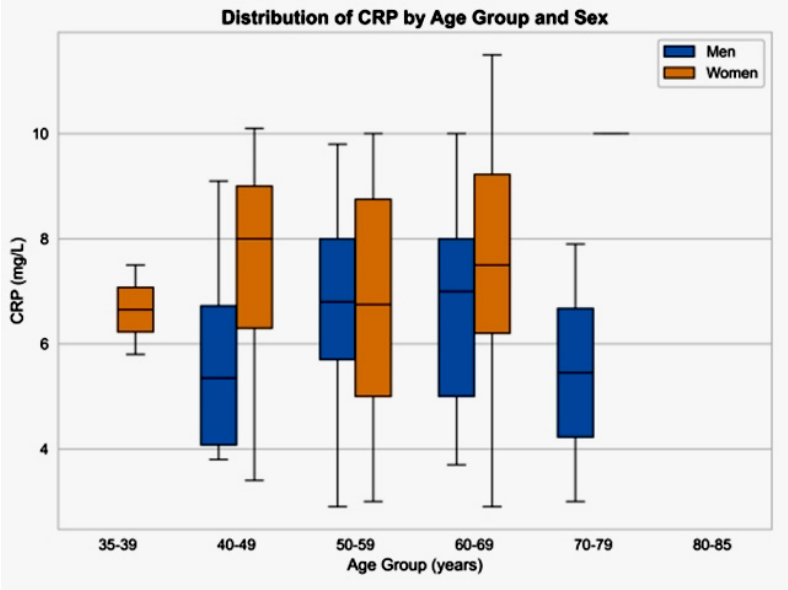


Box plot comparing serum hs-cTnI concentrations (in ng/L) between men and women. The box represents the interquartile range (IQR), the horizontal line inside the box indicates the median, and the whiskers extend to the minimum and maximum values within 1.5 * IQR. Circles represent outliers

Fig. 1. Sex-specific distribution of high-sensitivity cardiac troponin I (hs-cTnI) levels

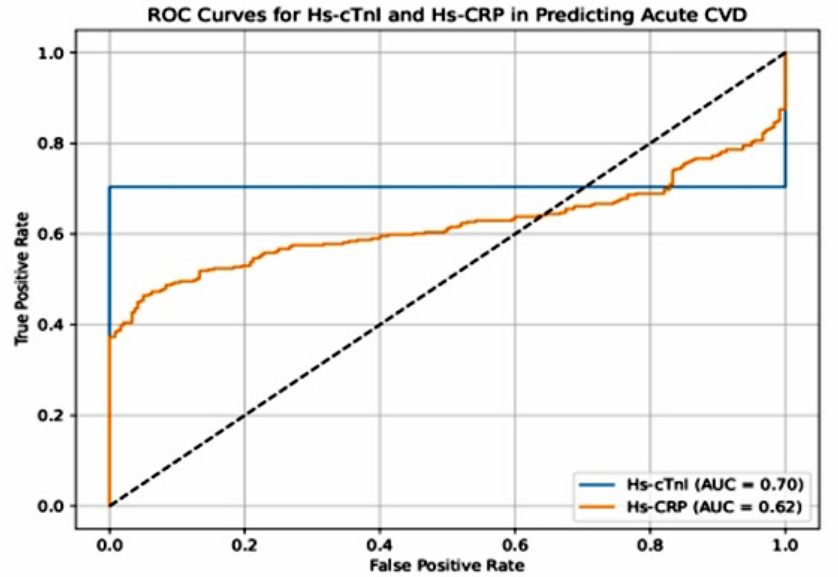
remains debated, and current European Society of Cardiology (ESC) guidelines do not endorse them.[31,32] Clinicians should consider confounders like age, sex, and renal function when interpreting hs-cTn results, which are reliable for ruling out myocardial infarction.[33]Hs-CRP, a marker of systemic inflammation, displays distinct patterns based on sex differences.

hs-CRP levels across age groups (<50 vs e+50 years) and sexes, illustrating median values



Bar graph displaying median serum hs-CRP levels (in mg/L) with error bars (representing interquartile range or standard deviation) across different age strata for men and women. The plot highlights the distinct peak in median hs-CRP levels among women in the 60-69 age group.

Fig. 2. Age-stratified distribution of high-sensitivity C-reactive protein (hs-CRP) levels by sex.



ROC curves illustrating the diagnostic performance of hs-cTnI and hs-CRP in discriminating between patients with acute CVD and healthy controls. The area under the curve (AUC) is shown for each biomarker.

Fig. 3. Receiver operating characteristic (ROC) curves for hs-cTnI and hs-CRP

and distribution shapes. Women show higher hs-CRP levels than men, possibly due to estrogen-mediated interleukin-6 (IL-6) production.[34]

hs-CRP distribution by age and sex, showing early increases in men, indicative of premature inflammatory activation, and a significant rise in women around age 50, likely linked to menopausal hormonal changes. These patterns suggest estrogen's protective anti-inflammatory role pre-menopause.[35] In older adults, Higher hs-CRP levels are predictive of an heightened likelihood of cardiovascular outcomes; and functional decline, requiring cautious interpretation.[36]

Multivariate Logistic Regression Analysis

In multivariate logistic regression analysis adjusted for age, sex, diabetes, and other lipid parameters, hs-cTnI, hs-CRP, and LDL-C were independently associated with acute CVD. Smoking and physical inactivity also remained significant predictors after adjustment for covariates (Table 4).

DISCUSSION

This case-control study provides novel insights into the predictive role of LDL-C, hs-cTnI, and hs-CRP for acute CVD in a Moroccan population. Our key findings are threefold:
(1) a pronounced atherogenic lipid profile characterized by elevated LDL-C and reduced HDL-C; (2) a strong association of modifiable lifestyle risk factors, notably smoking; and (3) clinically informative sex- and age-specific distributions for hs-cTnI and hs-CRP that could refine risk stratification.

Table 4. Multivariate logistic regression analysis of biomarkers and lifestyle factors

Parameter	OR	95% CI	p-value
Hs-cTnl	1.06	1.03-1.09	<0.001
Hs-CRP	1.12	1.04-1.21	0.003
LDL-C	1.82	1.45-2.29	<0.001
Smoking	14.55	7.22 – 29.36	<0.001
Physical Inactivity	2.88	2.03 – 4.09	<0.001

CI, confidence interval; LDL-C, low-density lipoprotein cholesterol; hs-cTnI, high-sensitivity cardiac troponin I; hs-CRP, high-sensitivity C-reactive protein.

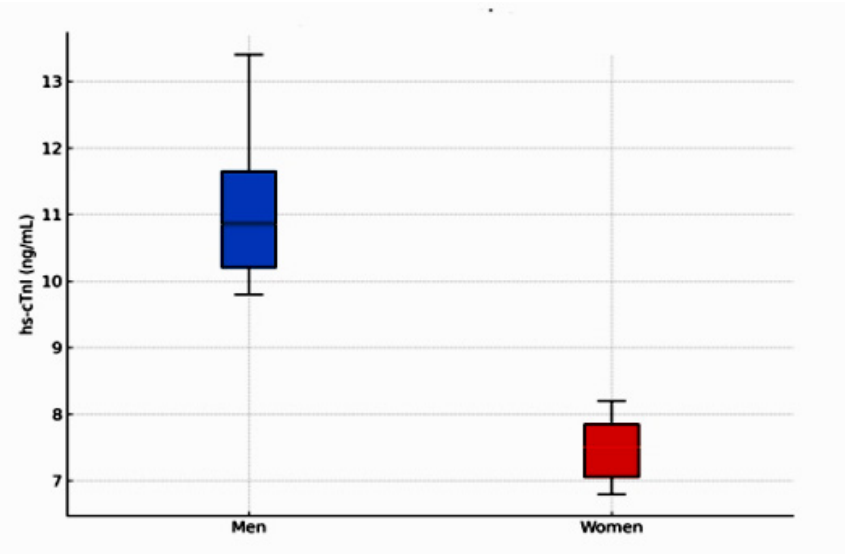


Fig. 4. Sex-Specific Distribution of Troponin Levels

Comparative analyses of lipid profiles in diverse populations, including Morocco's evolving CVD landscape, reveal varying patterns of dyslipidemia associated with cardiovascular disease (CVD).[37] The observed dyslipidemic profile—marked by significantly higher LDL-C and lower HDL-C in patients—aligns with established evidence on lipid-related atherogenesis.[5,10] The magnitude of LDL-C elevation in our cohort is comparable to Tunisian data[38] and exceeds reports from Brazilian and Japanese cohorts,[39,40] underscoring a particularly pronounced dyslipidemic risk profile in North African populations that warrants targeted public health interventions.

The strength of the associations revealed by multivariate analysis underscores the multifactorial nature of CVD pathogenesis in our cohort. The markedly elevated risk associated with smoking (OR: 14.55) aligns with its well-documented role in promoting endothelial dysfunction and systemic inflammation, which are key drivers of atherosclerotic plaque formation and instability.[41,42] Moreover, the independent predictive value of both LDL-C and hs-CRP reinforces the intricate link between dyslipidemia and inflammatory pathways in driving atherogenesis.[5,6] While the independent association of hs-cTnI with acute CVD events confirms its role as a crucial marker of subclinical myocardial injury, its moderate discriminative capacity (AUC = 0.70) suggests it should be interpreted in conjunction with other clinical findings rather than used in isolation.[7,31]

The sex and age-specific patterns we observed add a crucial layer to risk stratification. The lower hs-cTnI values in women are consistent with established biological variations,[31] while the sharp peak in hs-CRP levels among women aged 60-69 likely reflects the pro-inflammatory state associated with postmenopausal hormonal changes and the loss of estrogen's cardioprotective effects.[34,35] This distinct inflammatory trajectory in women suggests a potential window for targeted anti-inflammatory interventions and underscores the necessity of sex-specific risk assessment algorithms.

Our findings are consistent with regional studies investigating cardiac biomarkers. For instance, a Moroccan study defined a specific troponin cutoff for diagnosing myocardial infarction post-cardiac surgery, underscoring the importance of context-specific thresholds.[44] Furthermore, reinforcing the clinical value of our biomarker-focused approach, research from the same institution emphasized the critical need for the appropriate use of troponin testing in emergency departments to optimize patient care and resource utilization.[45]

Several limitations of our study warrant consideration. The case-control design precludes the establishment of causality. The single-center recruitment may limit the generalizability of our findings to the entire Moroccan population. Although apolipoproteins were analyzed, missing data in a subset of participants precluded a comprehensive analysis of all lipoprotein subfractions. Despite these limitations, our results provide a robust foundation for future prospective studies aimed at validating population-specific cutoff values and developing cost-effective, biomarker-guided risk stratification protocols for Morocco and similar resource-limited settings in North Africa.[49,50,54]

CONCLUSION

Cardiovascular diseases (CVDs) represent a major and growing contributor to mortality in Morocco, a trend propelled by urbanization, an aging population, and shifting lifestyles. [46]This study identifies elevated LDL-C, hs-cTnI, and hs-CRP as significant and independent predictors of acute CVD events in the Moroccan population, underscoring the synergistic role of dyslipidemia, myocardial injury, and inflammation in the region's risk profile.[47,48]

Beyond confirming a pronounced atherogenic dyslipidemia, our analysis revealed critical sex- and age-specific patterns for both hs-cTnI and hs-CRP, suggesting that men and women may experience divergent pathophysiological pathways to acute CVD.[49,50]The superior discriminative performance of hs-cTnI (AUC = 0.70) supports its potential utility for early risk stratification in acute clinical settings, while the distinct trajectory of hs-CRP, particularly its sharp increase in postmenopausal women, provides valuable insight for guiding long-term, sex-specific prevention strategies.[51,52]

These findings strongly advocate for the integration of these biomarkers, particularly hs-cTnI, into national CVD diagnostic and risk-stratification protocols, alongside conventional risk factor assessment.[53]We therefore recommend the development and validation of biomarker-guided clinical algorithms. Future large-scale, prospective studies are essential to establish population-specific cutoff values and to rigorously assess the cost-effectiveness of their implementation within resource-conscious healthcare systems like Morocco's.[49,50,54]

Ultimately, adopting such a tailored, evidence-based approach is imperative to improve early detection, refine risk prediction, and effectively alleviate the escalating burden of CVD in Morocco and the broader North African region.[55]

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Conflict of interest

No conflict of interest to declare.

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