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Clinical and Biochemical Characteristics of Patients with Coronary Heart Disease in Baghdad, Iraq

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Abstract

Introduction: Coronary heart disease (CHD) remains a major cause of cardiovascular morbidity and mortality. This study described selected clinical and biochemical characteristics of adults with established CHD receiving specialist care in Baghdad, Iraq.

Methods: A descriptive cross-sectional study included 100 adults with cardiologist-confirmed CHD who attended Ibn Al-Baytar Centre for Heart Surgery between February and June 2025. Sociodemographic and clinical information was obtained from a structured questionnaire and medical records. Total cholesterol, triglycerides, blood glucose, C-reactive protein (CRP), D-dimer, cardiac troponin and blood pressure were summarised descriptively, including sex-stratified categorical distributions.

Results: Fifty participants were male and 50 female; 80% were aged 50–100 years. Family history of cardiovascular disease, hypertension, diabetes mellitus and obesity were reported in 63%, 57%, 47% and 27%, respectively. Lipid-lowering, antihypertensive and antidiabetic treatments were documented in 70%, 53% and 47%. Mean total cholesterol was 232.4 ± 41.6 mg/dL, triglycerides 198.7 ± 52.3 mg/dL, glucose 176.2 ± 48.9 mg/dL, CRP 8.6 ± 3.4 mg/L, D-dimer 612.5 ± 210.7 ng/mL, troponin 0.041 ± 0.018 ng/mL and systolic blood pressure 146.8 ± 18.5 mmHg.

Conclusion: The study describes a substantial burden of cardiovascular risk factors and abnormal biomarker measurements among patients with established CHD. Because there was no independent comparison group and no measure of disease severity or follow-up outcome, the findings do not establish diagnostic performance, causality or incremental prognostic value.

Keywords: Coronary Disease; Biomarkers; C-Reactive Protein; D-Dimer; Blood Pressure

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Introduction

Coronary heart disease (CHD), a clinical manifestation of atherosclerotic cardiovascular disease, remains a major contributor to morbidity and mortality. Contemporary prevention and chronic coronary disease guidance emphasises systematic assessment and management of modifiable cardiovascular risk factors, including blood pressure, lipid abnormalities, diabetes and excess body weight (1, 2). Cardiovascular disease is also increasingly important in low- and middle-income settings, where access to specialist investigations and long-term preventive care may vary across health systems (3). Context-specific evidence is therefore needed to describe how established cardiovascular disease presents and is managed in different Global South settings (4).

Lipid abnormalities have a central role in atherosclerosis. The cumulative burden of low-density lipoprotein (LDL) exposure is causally related to atherosclerotic cardiovascular disease, and lipid assessment remains a core component of cardiovascular prevention and secondary prevention (5). Although the present study collected total cholesterol and triglycerides, LDL cholesterol and high-density lipoprotein cholesterol were not reported in the available dataset. This limits the extent to which the study can characterise the complete lipid phenotype. Atherosclerosis is also an inflammatory disease. Experimental and clinical evidence

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supports an important role for inflammation in plaque development and cardiovascular events, while high-sensitivity C-reactive protein (hs-CRP) has been investigated as a marker of residual inflammatory risk (6, 7, 8). In parallel, thrombotic and fibrinolytic processes interact with atherosclerotic plaque biology. D-dimer reflects fibrin formation and degradation and has been examined as a prognostic biomarker in patients with acute coronary syndromes (9, 10).

Cardiac troponins are established markers of myocardial injury. Their interpretation requires assay-specific upper reference limits and clinical context; an elevated concentration indicates myocardial injury but does not, by itself, establish myocardial infarction (11, 12). High-sensitivity troponin may also have prognostic value in chronic coronary disease, although evidence for routine use outside acute presentations remains an area of continuing evaluation (13).

Ageing, family history, hypertension and diabetes are established components of cardiovascular risk. Age-related vascular and cardiac changes contribute to susceptibility to cardiovascular disease, while inherited susceptibility influences coronary artery disease risk (14, 15, 16, 17, 18, 19).

Despite extensive evidence on individual cardiovascular risk factors and biomarkers, locally generated descriptive data remain useful for understanding the clinical and laboratory profile of patients receiving care in specialist cardiac centres. The present study therefore aimed to describe selected sociodemographic, clinical and biochemical characteristics of adults with established CHD attending a cardiac centre in Baghdad, Iraq. Because all participants had established CHD and no independent control group was available, the study was not designed to estimate the diagnostic performance of the biomarkers or to establish their association with the presence of CHD.

Methods

Study design and setting

A hospital-based descriptive cross-sectional study was conducted at Ibn Al-Baytar Centre for Heart Surgery in Baghdad, Iraq. Data were collected between February and June 2025. The centre provided the clinical setting in which participants with established CHD were assessed and treated.

Study population and participant selection

The study included 100 adults aged 18–100 years with a cardiologist-confirmed diagnosis of CHD and complete clinical and laboratory records at the time of data collection. The study documentation states that participants were recruited consecutively and comprised 50 men and 50 women. The exact sampling process by which this sex balance was achieved was not described. No independent healthy or non-CHD comparison group was included.

Data collection

Sociodemographic and clinical information was obtained from a structured questionnaire and patient medical records. The reported variables included age, sex, residence, family history of cardiovascular disease, hypertension, diabetes mellitus, obesity, hospital admission related to cardiovascular symptoms, and use of antihypertensive, lipid-lowering and antidiabetic medication. The study documentation described the questionnaire as validated but did not provide details of its development, validation process or psychometric performance.

Clinical and biochemical measurements

Physiological measures included systolic and diastolic blood pressure. In the available report, blood pressure measurements were extracted from clinical records; the timing, number of readings, patient position and device used were not specified. Biochemical measures included total cholesterol, triglycerides, blood glucose, CRP, D-dimer and cardiac troponin. The study documentation states that laboratory investigations were performed using standard procedures routinely applied in the hospital laboratory, but does not identify assay manufacturers, analytical platforms, sampling conditions or assay-specific upper reference limits. The reported laboratory or clinical limits were: total cholesterol <200 mg/dL; triglycerides <150 mg/dL; blood glucose 70–110 mg/dL; CRP <5 mg/L; D-dimer <500 ng/mL; cardiac troponin <0.04 ng/mL; and systolic blood pressure <120 mmHg. These values are presented as the laboratory or clinical limits reported for the study; they are not a control group and do not provide evidence of a difference from a population of healthy individuals.

Statistical analysis

Data were analysed using IBM SPSS Statistics version 26. Categorical variables were summarised using frequencies and percentages, while continuous variables were summarised using means and standard deviations. Sex-stratified distributions were presented for selected categorical biomarkers. Because the available study population consisted entirely of participants with established CHD and no independent comparator or disease-severity outcome was available, the revised analysis is descriptive and does not make claims about diagnostic accuracy, causality or independent prognostic value. The inferential p-values reported in the original tables were not retained where they could not be reconciled from the displayed summary data.

Results

Eighty of the one hundred participants (80%) were aged 50–100 years and 20 (20%) were aged 18–49 years. The sample contained 50 men and 50 women. Sixty-three participants (63%) reported a family history of cardiovascular disease, while hypertension and diabetes mellitus were documented in 57 (57%) and 47 (47%), respectively. Table 1 summarises the demographic and clinical characteristics.

Table 1: Demographic and clinical characteristics of patients with established coronary heart disease (n = 100)

Variable	Category	n (%)
Age group	18–49 years	20 (20.0)
	50–100 years	80 (80.0)
Sex	Male	50 (50.0)
	Female	50 (50.0)
Family history of cardiovascular disease	Yes	63 (63.0)
	No	37 (37.0)
Hypertension	Yes	57 (57.0)
	No	43 (43.0)
Diabetes mellitus	Yes	47 (47.0)
	No	53 (53.0)
Obesity	Yes	27 (27.0)
	No	73 (73.0)
Hospital admission due to CVD-related symptoms	Yes	77 (77.0)
	No	23 (23.0)

CVD: cardiovascular disease. Age was available only as two categories; individual-level ages were not supplied

Figure 1 provides a visual summary of the main demographic, clinical and treatment characteristics. The measures are not mutually exclusive; for example, participants receiving more than one class of medication are represented in each relevant category.

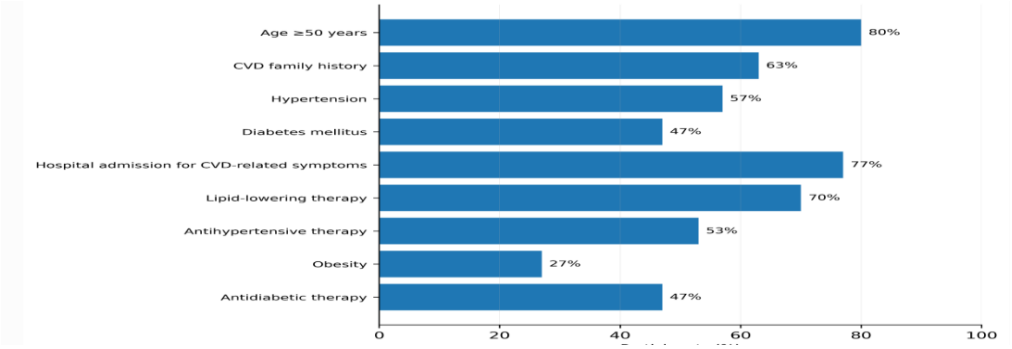


Figure 1: Selected demographic, clinical and treatment characteristics of patients with established coronary heart disease (n = 100)

Abnormal biomarker categories were common. Among men, elevated total cholesterol, D-dimer, CRP and cardiac troponin were documented in 30 (60.0%), 33 (66.0%), 20 (40.0%) and 17 (34.0%), respectively. Among women, the corresponding proportions were 37 (74.0%), 20 (40.0%), 27 (54.0%) and 10 (20.0%). Overall, elevated total cholesterol was present in 67% of participants, elevated D-dimer in 53%, elevated CRP in 47% and elevated cardiac troponin in 27% (Table 2).

Table 2: Sex-stratified distribution of selected biochemical markers

Biochemical marker	Sex	Elevated n (%)	Normal n (%)
Total cholesterol	Male	30 (60.0)	20 (40.0)
	Female	37 (74.0)	13 (26.0)
D-dimer	Male	33 (66.0)	17 (34.0)
	Female	20 (40.0)	30 (60.0)
CRP	Male	20 (40.0)	30 (60.0)
	Female	27 (54.0)	23 (46.0)
Cardiac troponin	Male	17 (34.0)	33 (66.0)
	Female	10 (20.0)	40 (80.0)

CRP: C-reactive protein

Clinical and treatment characteristics showed that 77% of participants had been admitted because of cardiovascular disease-related symptoms. Obesity was documented in 27%, while 53% were receiving antihypertensive medication, 70% lipid-lowering therapy and 47% antidiabetic medication (Table 3).

Table 3: Clinical and treatment characteristics of patients with established coronary heart disease (n = 100)

Variable	Yes n (%)	No n (%)
Hospital admission due to CVD-related symptoms	77 (77.0)	23 (23.0)
Obesity	27 (27.0)	73 (73.0)
Antihypertensive medication	53 (53.0)	47 (47.0)
Lipid-lowering therapy	70 (70.0)	30 (30.0)
Antidiabetic medication	47 (47.0)	53 (53.0)

CVD: cardiovascular disease

The reported mean values of the selected biochemical and physiological measures were above the laboratory or clinical limits reported for the study. Mean total cholesterol was 232.4 ± 41.6 mg/dL and triglycerides 198.7 ± 52.3 mg/dL. Mean blood glucose was 176.2 ± 48.9 mg/dL, CRP 8.6 ± 3.4 mg/L, D-dimer 612.5 ± 210.7 ng/mL and cardiac troponin 0.041 ± 0.018 ng/mL.

Mean systolic blood pressure was 146.8 ± 18.5 mmHg. These findings are summarised in Table 4.

Table 4: Reported mean clinical and biochemical values and corresponding laboratory or clinical thresholds

Parameter	Mean ± SD	Reported threshold/interval	Units
Total cholesterol	232.4 ± 41.6	<200	mg/dL
Triglycerides	198.7 ± 52.3	<150	mg/dL
Blood glucose	176.2 ± 48.9	70–110	mg/dL
CRP	8.6 ± 3.4	<5	mg/L
D-dimer	612.5 ± 210.7	<500	ng/mL
Cardiac troponin	0.041 ± 0.018	<0.04	ng/mL
Systolic blood pressure	146.8 ± 18.5	<120	mmHg

CRP: C-reactive protein. Thresholds/intervals are the limits reported for the study

Discussion

This study provides a descriptive profile of 100 adults with established CHD attending a specialist cardiac centre in Baghdad. The study population was predominantly aged 50 years or older, and hypertension, diabetes, obesity and family history of cardiovascular disease were common. Many participants were already receiving cardiovascular therapies, particularly lipid-lowering medication. The available biochemical measurements showed mean total cholesterol, triglycerides, blood glucose, CRP, D-dimer and cardiac troponin values above the thresholds reported in the study documentation. These findings describe the clinical complexity of the study population but, because the study did not include a non-CHD comparison group or measures of coronary disease severity, they should not be interpreted as estimates of risk attributable to individual biomarkers or as evidence of diagnostic performance.

The predominance of participants aged 50–100 years is consistent with the well-established relationship between ageing and cardiovascular disease. Ageing is accompanied by changes in arterial structure and function, including increasing arterial stiffness and altered vascular compliance, which can increase susceptibility to cardiovascular disease (14, 15). The broad age categories available in the dataset prevent a more detailed examination of age gradients. Future studies should report continuous age data or narrower age categories to permit more informative comparisons.

A positive family history of cardiovascular disease was reported by 63% of participants. Family history is a clinically useful marker of inherited and shared environmental susceptibility to coronary artery disease, and genomic studies have identified multiple genetic determinants of coronary risk (16, 17). In the present study, however, the frequency of family history cannot demonstrate a genetic contribution within this CHD study population because no comparison group was available. Family history should therefore be treated as a descriptive risk characteristic rather than an outcome attributable to a measured genetic mechanism.

Hypertension and diabetes mellitus were documented in 57% and 47% of participants, respectively. Both conditions are well-established cardiovascular risk factors, and current clinical guidance recommends systematic management of blood pressure and cardiometabolic risk in people with cardiovascular disease (1, 18, 19, 20). The mean systolic blood pressure of 146.8 ± 18.5 mmHg suggests that elevated blood pressure remained common in the study population despite 53% reporting antihypertensive treatment. Because treatment type, dose, duration and adherence were not available, the study cannot determine why blood pressure remained elevated or assess treatment effectiveness.

Total cholesterol and triglycerides were also elevated relative to the thresholds reported in the study documentation. Extensive clinical and genetic evidence supports a causal role for LDL exposure in atherosclerotic cardiovascular disease (5). The current dataset did not provide LDL cholesterol or high-density lipoprotein cholesterol, so the observed total cholesterol and triglyceride values should be interpreted as partial lipid characterisation rather than a complete lipid profile. The finding that 70% of participants were receiving lipid-lowering therapy also suggests that treatment exposure needs to be considered when interpreting lipid concentrations in this population.

CRP provides a readily available marker of systemic inflammation, although its interpretation depends on the assay used. Inflammatory pathways are integral to atherosclerotic plaque biology, and hs-CRP has been investigated as a marker of residual inflammatory risk (6, 7, 8). The mean CRP of 8.6 ± 3.4 mg/L in the present study indicates that inflammatory marker concentrations were often above the reported laboratory threshold. Because the study does not establish whether conventional CRP or hs-CRP was measured, no inference should be made about residual inflammatory risk or the potential effect of inflammation-targeted therapy. The mean D-dimer concentration was 612.5 ± 210.7 ng/mL, and 53% of participants had values categorised as elevated in the dataset. D-dimer reflects fibrin turnover and is influenced by multiple acute and chronic conditions; an elevated value should therefore not be equated with a hypercoagulable state without additional clinical and laboratory information. Previous observational work has associated elevated D-dimer with adverse outcomes after acute coronary syndrome and percutaneous coronary intervention (9). More recently, a large prospective cohort study of 5,139 patients with acute coronary syndrome undergoing percutaneous coronary intervention found that higher baseline D-dimer independently predicted 5-year all-cause and cardiac mortality and improved discrimination when added to the PARIS thrombotic risk score (10). Those findings support continued investigation of D-dimer as a prognostic marker, but they cannot be directly applied to the present cross-sectional study, which was not designed to assess subsequent clinical outcomes.

Cardiac troponin was elevated in 27% of participants according to the threshold reported for the study, while the overall mean was 0.041 ± 0.018 ng/mL. Interpretation requires particular caution because myocardial injury is defined using an assay-specific 99th percentile upper reference limit and should be distinguished from acute myocardial infarction, which also requires clinical evidence of myocardial ischaemia and appropriate troponin dynamics (11, 12). High-sensitivity troponin has demonstrated prognostic associations in patients with chronic coronary disease, but such evidence arises from studies with defined clinical outcomes and standardised assays (13). The present study did not provide the assay type, sampling time, 99th percentile reference limit or serial measurements, and therefore cannot determine whether the reported elevations represented acute or chronic myocardial injury.

The high prevalence of cardiovascular medication use has practical implications for interpreting all measured biomarkers. Lipid-lowering therapy, antihypertensive treatment and antidiabetic medication can alter biochemical and physiological measurements. Without information on drug class, dose, duration and adherence, observed values cannot be interpreted as untreated disease characteristics. This is particularly relevant to secondary prevention, where contemporary guidelines recommend integrated management of lipids, blood pressure, diabetes, lifestyle and other modifiable risk factors (1, 2, 19, 20).

The study also has relevance for health systems in which basic biochemical testing is more accessible than advanced imaging or invasive assessments. The findings suggest that routine measurements can provide a useful descriptive picture of patients already receiving specialist cardiovascular care. However, the present data do not demonstrate that a combined panel of cholesterol, CRP, D-dimer, troponin and blood pressure improves diagnosis, risk stratification or clinical outcomes. Claims of this type require comparative and preferably prospective designs with prespecified outcomes, standardised assays and multivariable adjustment.

Study limitations

Several limitations should be considered. First, there was no independent non-CHD control group. The study therefore cannot estimate the diagnostic discrimination of the biomarkers or determine whether the observed abnormalities are more common than in people without CHD. Second, there was no measure of angiographic or clinical disease severity, preventing assessment of whether biomarker concentrations varied with coronary disease burden. Third, recruitment occurred at a single specialist centre, and the study population may not represent all patients with CHD in Baghdad or Iraq. Fourth, the source report describes an exactly balanced sex distribution but does not explain whether a sex quota was used, which may affect representativeness. Fifth, assay details were not provided for CRP, D-dimer or cardiac troponin, and the troponin upper reference limit was not identified as assay-specific.

Sixth, the source dataset was presented with broad age categories and did not provide individual-level ages, so a mean age could not be calculated from the supplied material. Seventh, treatment exposure and adherence were incompletely characterised, and no multivariable analysis was possible from the reported aggregate data. Finally, the timing and physiological context of blood sampling were not described, which is relevant to glucose, CRP, D-dimer and troponin.

Future directions

Future studies should use multicentre designs and recruit an appropriate comparison group matched or adjusted for age

and sex. Biomarker assays should be reported with manufacturer, analytical platform, units and assay-specific reference limits, particularly for cardiac troponin and CRP. Studies of CHD severity should incorporate reproducible measures such as the number of affected vessels, Gensini score or SYNTAX score where angiographic data are available. Prospective follow-up would permit evaluation of whether D-dimer, CRP or cardiac troponin adds prognostic information beyond established risk factors and validated clinical scores. Multivariable models should account for age, sex, diabetes, blood pressure, obesity, medication use and other clinically relevant confounders. Research should also assess the feasibility, affordability and incremental value of biomarker testing within the local cardiac care pathway.

Conclusion

Among adults with established CHD attending a specialist cardiac centre in Baghdad, older age, hypertension, diabetes, family history of cardiovascular disease and obesity were common, and many participants were receiving cardiovascular medications. Mean total cholesterol, triglycerides, blood glucose, CRP, D-dimer, cardiac troponin and systolic blood pressure were above the laboratory or clinical thresholds reported in the study documentation. These findings describe the clinical and biochemical profile of this study population but do not establish diagnostic accuracy, causal relationships or incremental prognostic value. Comparative, multicentre and longitudinal studies with standardised measurements are needed to determine the clinical utility of these biomarkers.

Abbreviations

CHD: coronary heart disease
CRP: C-reactive protein
CVD: cardiovascular disease
hs-CRP: high-sensitivity C-reactive protein
LDL: low-density lipoprotein
SPSS: Statistical Package for the Social Sciences

Declarations

Ethics Approval and Consent to Participate: Ethical approval was obtained from the Review Committee, Department of Pathological Analysis, University of Samarra, Iraq (ECM/2026/341) before data collection, with patient confidentiality maintained. The responsible research ethics committee, approval/reference number and informed-consent procedure were not supplied in the study documentation and must be added before submission.

Data Availability: The underlying dataset was not supplied with the manuscript revision. The authors should provide a study-specific statement indicating whether the de-identified dataset is available from the corresponding author or through a repository, subject to ethics and institutional requirements.

Competing Interests: The author declares no competing interests.

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Author contributions: MSS conceived and designed the study, developed the methodology, collected and analysed the data, interpreted the findings, drafted and critically revised the manuscript, and approved the final version for publication. MSS accepts responsibility for the integrity and accuracy of the work.

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Use of Artificial Intelligence: Generative artificial intelligence was used to assist with manuscript restructuring and language revision. The authors are responsible for verifying all scientific content, numerical results, references and declarations and for the final version submitted to the journal.

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