JBAAR
Journal of Basic and Applied Research in Biomedicine
ISSN 2413-7014

*Original Article*

# Lipid Profile Among Patients with Acute Myocardial Infarction in Iraq: A Cross-Sectional Study

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**HISTORY**
Received: 19-03-2026
Revised: 25-04-2026
Published: 20-05-2026

**FUNDING**
The authors declare that this research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

**CONFLICTS OF INTEREST**
The authors declare no conflict of interest in this study

**DATA AVAILABILITY**
The data that support the findings of this study are available from the corresponding author upon reasonable request.

**ABSTRACT**:
**Background**: Cardiovascular diseases are the global leading cause of morbidity and mortality, with acute myocardial infarction (AMI) being a major clinical manifestation. Dyslipidemia is a modifiable risk factor for atherosclerosis and coronary diseases.
**Objectives**: This study aimed to assess lipid profiles among AMI patients in Duhok City, Iraq.
**Methods**: A cross-sectional study was carried out at Azadi Teaching Hospital and Cardiac Center in Duhok City from June to November 2025. A convenient sample of 80 AMI patients aged ≥18 years was included. Sociodemographic and clinical data were collected through interviews and medical records. Blood samples were analyzed for total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG). Data were analyzed using SPSS version 23, with $p < 0.05$ considered statistically significant.
**Results**: The mean age of patients was 53.0 ± 14.07 years, and 65.1% were aged ≥50 years. AMI was more common among males, urban residents, and individuals with low to moderate socioeconomic status. The lipid profile showed an atherogenic pattern, with elevated triglycerides (212.93 ± 71.91 mg/dL), total cholesterol (210.0 ± 51.68 mg/dL), and LDL-C (125.1 ± 41.24 mg/dL), along with reduced HDL-C levels (39.55 ± 11.51 mg/dL).
**Conclusion**: Dyslipidemia is highly prevalent among AMI patients in Duhok, characterized by elevated atherogenic lipids and low HDL-C. Early screening and lipid-lowering interventions are recommended to reduce cardiovascular risk.

**Keywords:** *Lipid profile, Acute myocardial infarction, Dyslipidemia, Cardiovascular disease, coronary artery diseases.*

Cite this article as: Abdullah, R.Y., Mhammedsadiq, D.F., Mohammedsalih, J.S., Abdullah, A.I. (2026) Lipid Profile Among Patients with Acute Myocardial Infarction in Iraq: A Cross-Sectional Study. Journal of Basic and Applied Research in Biomedicine, 12(1): 7-15



## INTRODUCTION

Coronary Artery Disease (CAD) is one of the most prevalent and life-threatening diseases that affect the global population. In accordance with the data from the World Health Organization (WHO), cardiovascular diseases (CVDs) result in the death of 17.9 million people each year, making them the leading cause of mortality (WHO, 2025a). One of the most severe manifestations of CAD is myocardial infarction (MI), commonly referred to as a "heart attack." MI may occur as a silent event that remains clinically unrecognized or as AMI, which can result in hemodynamic instability, sudden cardiac death, and other life-threatening complications. AMI constitutes a medical emergency in which myocardial tissue begins to undergo necrosis due to inadequate coronary perfusion is caused by a significant reduction or complete cessation of blood flow to a portion of the myocardium, leading to irreversible myocardial injury (Thygesen et al., 2018). Despite substantial advances in diagnostic and therapeutic strategies, AMI remains a leading cause of morbidity and mortality worldwide (WHO, 2023; Mechanic et al., 2015).

Acute myocardial infarction is diagnosed in clinical practice using an integrated approach that includes clinical assessment, electrocardiographic (ECG) evaluation, measurement of cardiac biomarkers, invasive and non-invasive imaging modalities, and, when available, pathological examination. The classic clinical presentation is characterized by chest pain radiating to the left arm and jaw, which occurs more frequently in men. In contrast, women are more likely to present with atypical symptoms, including dyspnea, fatigue, nausea, and epigastric discomfort. Based on ECG presentation, AMI is conventionally classified into ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) (Mechanic et al., 2015), each reflecting distinct pathophysiological mechanisms, therapeutic strategies, and prognostic implications.

Myocardial infarctions are often classified according to ECG results, especially the presence of ST segment elevation, as it determines the degree of urgency with which the condition should be addressed. STEMI is usually caused by a complete occlusion of a medium- to large-sized vessel, whereas NSTEMI is usually caused by a partial occlusion, although it is also possible for complete occlusions to occur without ST segment elevation. AMI, as a clinical entity, is usually characterized by the presence of chest pain with accompanying ST segment elevation, although it is also possible for the condition to be silent without the presence of these characteristic manifestations (Alpert et al., 2000). However, it should be noted that although the ECG is limited, American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines (ACC/AHA), diagnostic criteria for AMI require the presence of at least two of the following for the diagnosis of AMI: the presence of elevated cardiac biomarkers, such as troponin I/T and CK-MB, and ECG changes indicative of myocardial injury (Rao et al, 2025).

The pathogenesis of AMI is strongly linked to atherosclerosis, an inflammatory and metabolic disorder characterized by lipid deposition within the arterial wall, resulting in plaque formation, rupture, and subsequent thrombotic occlusion of coronary vessels (Libby et al., 2019). Dyslipidemia, defined as abnormal

lipid metabolism leading to high cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglyceride (TG) levels, as well as reduced high-density lipoprotein cholesterol (HDL-C) levels, plays a central role in the initiation and progression of atherosclerosis and poses cardiovascular risk (Ballena-Caicedo et al., 2025). Cardiovascular disease remains a leading cause of morbidity and mortality worldwide, with dyslipidemia playing a key role in the development of atherosclerosis. Over the past three decades, its global burden has increased substantially, creating a major public health concern. Atherogenic dyslipidemia refers to a lipid abnormality marked by elevated triglycerides, reduced HDL-C levels, and an increased proportion of small, dense LDL particles, all of which are associated with an elevated likelihood of cardiovascular events. Among these abnormalities, high LDL-cholesterol is a key modifiable determinant of cardiovascular disease, and hypercholesterolemia remains the most common clinical manifestation of dyslipidemia (Pirillo et al., 2021).

The epidemiological and clinical studies have shown that lipid profile abnormalities are related to an increased risk of AMI (Catapano et al., 2019). For example, increased levels of LDL-C and TG accelerate the process of endothelial dysfunction, while decreased levels of HDL-C disrupt the reverse cholesterol transport pathway, which leads to coronary artery disease (Mach et al., 2019). In addition, the lipid profile not only plays a crucial role in the risk assessment of cardiovascular disease but also offers a therapeutic target to reduce the risk of recurrent myocardial infarction with the use of statins (Cholesterol Treatment Trialists, 2019).

Cardiovascular diseases remain a leading cause of morbidity and mortality worldwide and are among the top causes of death in Iraq, including the Kurdistan region (WHO, 2025b). Understanding the contribution of lipid profiles to MI risk is particularly important in regions with distinctive demographic and lifestyle characteristics. The Kurdistan region is characterized by unhealthy lifestyles such as poor dietary habits, physical inactivity, irregular sleep patterns, and smoking along with variable healthcare access, all of which may affect lipid metabolism and cardiovascular health. However, local data remain limited. Investigating lipid profiles in patients with AMI can provide valuable insights into regional risk factors, inform targeted preventive strategies, and improve early diagnosis and secondary prevention. This study aims to assess the lipid profile abnormalities among patients with AMI, contributing evidence to guide clinical management and public health interventions to reduce MI incidence in this population.

## METHODS

### Study design and setting

A hospital-based cross-sectional study was conducted in Duhok city to assess the serum lipid profile in AMI patients. The study was adopted at Azadi Teaching Hospital and the Cardiac Center in Duhok, Kurdistan Region, Iraq. Data were collected between June 4, 2025, and November 12, 2025.

### Study sample and sampling

A convenience sampling technique was used to recruit patients for the study with no present or past history of coronary artery disease or use of lipid-lowering medications. Cases consisted of adult patients (≥18 years) admitted to the coronary care unit (CCU), high-dependency unit (HDU), cardiac center, or emergency department of Azadi Teaching Hospital, Duhok City, within less than 12 hours of symptom onset. The diagnosis of AMI was confirmed at the admission of the patients by a physician based on clinical assessment, ECG changes, and elevated cardiac biomarkers. A total of 80 newly diagnosed AMI patients was recruited during the present study period based on feasibility and availability of eligible patients admitted to the study hospitals.

### Inclusion and exclusion criteria

The study enrolled adult participants (≥18 years) with a confirmed diagnosis of AMI and no prior history of AMI or cardiovascular disease. Eligible participants were free from chronic systemic diseases and were not receiving lipid-lowering therapy. Exclusion criteria included pregnancy, anemia, renal failure, previous coronary artery disease or old myocardial infarction, bleeding or platelet disorders, and current use of antiplatelet or anticoagulant medications. Individuals with chronic conditions that could affect lipid metabolism such as diabetes mellitus, chronic kidney disease, chronic liver disease, active infection, recent major surgery, or hepatic failure were also excluded. Participants using lipid-altering medications (statins or fibrates) or other drugs known to significantly influence lipid profiles within the preceding three months were not included. Written informed consent was obtained from all participants. Lipid profiles were measured using a Cobas Chemistry Analyzer at Azadi Teaching Hospital, Duhok City.

### Data collection

Data were collected through face-to-face interviews with AMI patients to collect the demographic data. Before enrollment, the purpose of the study was explained to all participants, and informed consent was obtained. Regarding lipid profile data were collected in collaboration with the Azadi Teaching Hospital laboratory to check their lipid levels. Cases consisted of patients admitted with AMI to the emergency department of Azadi Teaching Hospital, and the Cardiac Center in Duhok City.

### Tools:

The first part involved obtaining socio-demographic and clinical information, including age, gender, marital status, religion, residence, occupation, economic status, education, and family history of cardiovascular disease. Information was collected using a structured interviewer-administered questionnaire and review of medical records.

The second part of the study consisted of laboratory assessment of lipid profile parameters among AMI patients. Venous blood samples were obtained from all participants using standard phlebotomy techniques. Approximately 3 mL of blood was collected via atraumatic antecubital venipuncture into plain vacutainer tubes, allowed to clot, and subsequently centrifuged to separate the serum. In patients diagnosed with AMI, serum lipid profile parameters, including total TC, TG, HDL-C, and LDL-C, were measured at the time of hospital admission. Biochemical analysis was performed enzymatically using standardized colorimetric methods on a Cobas c501 chemistry analyzer (Roche Diagnostics, Basel, Switzerland) in the accredited central laboratory of Azadi Teaching Hospital. All lipid concentrations were reported in mg/dL, and internal quality control procedures were maintained throughout the analytical process to ensure accuracy and reliability. Dyslipidemia was defined according to established criteria as serum total TC >200 mg/dL, TG >150 mg/dL, LDL >100 mg/dL, and HDL <40 mg/dL in men and <50 mg/dL in women.

### Data management and statistical analysis

The collected data was entered into a secure database. The continuous variables were summarized as the mean ± standard deviation, depending on the distribution of the variables, while the categorical variables were summarized as frequencies and percentages. The lipid profile variables were categorized using cut-off points. The Chi-square test was used to determine the association between the predictor variables and lipid profile categories. The statistical package SPSS version 27 was used for the analysis, and $P < 0.05$ was considered as statistically significant.

### Ethical considerations

The study received initial written approval from the (Nursing Scientific Committee) at the College of Nursing, Azadi teaching hospital (registration number: 54) and the cardiac center (registration number: 151). Subsequently, it was reviewed and approved by the (Research Ethical Committee) at the Directorate of Health in Duhok City (08012025: 8 Jan. 2025). Following by agreement from the Azadi teaching hospital and cardiac center. Informed consent has been obtained from all participants. The confidentiality of the collected data has been maintained.

**Table 1. Sociodemographic characteristics of the participants**

| Characteristics of patients N=80 | | N | % |
|---|---|---|---|
| Age: | 18- 29 | 4 | 5.0 |
| Mean (SD) :53 (14.07) | 30- 39 | 9 | 11.3 |
| | 40- 49 | 15 | 18.8 |
| | 50- 59 | 23 | 28.8 |
| | 60 And Older | 29 | 36.3 |
| Gender | Male | 54 | 67.5 |
| | Female | 26 | 32.5 |
| Marital Status | Single | 4 | 5.0 |
| | Married | 74 | 92.5 |
| | Widowed | 2 | 2.5 |
| Occupation | Employee | 16 | 20 |
| | Unemployed | 29 | 36.2 |
| | Housewife | 26 | 32.6 |
| | Retired | 9 | 11.2 |
| Education | No Formal Education | 31 | 38.8 |
| | Primary School | 22 | 27.5 |
| | Secondary School | 11 | 13.8 |
| | High School | 10 | 12.5 |
| | University Level | 6 | 7.5 |
| Residence | Urban | 49 | 61.3 |
| | Rural | 31 | 38.8 |
| Economic Status | Low | 29 | 36.3 |
| | Moderate | 46 | 57.5 |
| | High | 5 | 6.3 |
| Religion | Muslim | 74 | 92.5 |
| | Yazidi | 6 | 7.5 |
| Family History of MI | Yes | 47 | 58.8 |
| | No | 33 | 41.3 |

SD,Standard Deviasion,N Number,MI Myocardiac Infarction,% Percentage

**Table 2. Lipid profile of Acute MI patients**

| Lipid profile | N | % | Mean (SD) | Minimum | Maximum | Median |
|---|---|---|---|---|---|---|
| Triglycerides Levels | | | | | | |
| **Normal** | 23 | 28.8 | | | | |
| **High** | 57 | 71.3 | 212.93 (71.913) | 92 | 384 | 215 |
| Cholesterol Levels | | | | | | |
| **Low** | 8 | 10.0 | | | | |
| **Normal** | 23 | 28.8 | | | | |
| **High** | 49 | 61.3 | 210 (51.68) | 108 | 302 | 219.5 |
| HDL Levels | | | | | | |
| **Low** | 41 | 51.3 | | | | |
| **Normal** | 31 | 38.8 | | | | |
| **High** | 8 | 10.0 | 39.55 (11.51) | 25 | 70 | 33.50 |
| LDL Levels | | | | | | |
| **Low** | 2 | 2.5 | | | | |
| **Normal** | 21 | 26.3 | | | | |
| **High** | 57 | 71.3 | 125.1 (41.24) | 26 | 220 | 116 |

SD,Standard Deviasion,N Number,% Percentage

## RESULTS

**Table 1** summarizes the sociodemographic characteristics of the 80 participants. The mean age was 53.0 ± 14.07 years, with most participants aged ≥50 years (65.1%). The majority were male (67.5%) and married (92.5%). Unemployed constituted the largest occupational group (36.2 %). Educational levels were generally low, with most participants having no formal or only primary education (66.3%). Most participants lived in urban areas (61.3%) and reported a moderate economic status (57.5%). The sample was predominantly Muslim (92.5%).

**Table 2** summarizes the lipid profile of patients with acute myocardial infarction (AMI) and demonstrates a predominantly atherogenic pattern. Triglyceride levels were elevated (mean 212.93 ± 71.91 mg/dL; median 215 mg/dL), indicating a high prevalence of hypertriglyceridemia. Total cholesterol levels were also increased (mean 210.0 ± 51.68 mg/dL; median 219.5 mg/dL), with many patients falling within borderline-high to high categories. High-density lipoprotein (HDL) cholesterol levels were relatively low (mean 39.55 ± 11.51 mg/dL; median 33.5 mg/dL), reflecting reduced cardioprotective capacity. In contrast, low-density lipoprotein (LDL) cholesterol levels were elevated (mean 125.1 ± 41.24 mg/dL; median 116 mg/dL), exceeding recommended targets for patients at very high cardiovascular risk. Overall, these findings indicate that dyslipidemia is highly prevalent among AMI patients, underscoring its key role in the pathogenesis of acute coronary events and highlighting the importance of early lipid assessment and aggressive lipid-lowering therapy.

**Table 3** summarizes the association between sociodemographic characteristics and triglyceride (TG) levels among patients with AMI. Triglyceride levels varied across age groups; however, no statistically significant association was observed ($p = 0.458$). Similarly, sex and marital status were not significantly related to TG levels ($p = 0.489$ and $p = 0.270$, respectively). In contrast, occupation showed a significant association with triglyceride levels ($p = 0.012$), with higher mean TG values observed among workers and unemployed patients. Educational level was not significantly associated with TG levels ($p = 0.083$), despite observable variations in mean values across categories. Place of residence was significantly associated with triglyceride levels ($p = 0.035$), as urban residents demonstrated higher mean TG levels compared with rural residents. Additionally, income level was significantly related to TG levels ($p = 0.041$), with the highest mean TG levels noted among patients with moderate income.

**Table 4** presents the sociodemographic predictors of total cholesterol levels among patients with acute myocardial infarction. Although variations in mean total cholesterol were observed across age groups, no statistically significant association was found between age and cholesterol levels ($p = 0.359$). Likewise, sex was not significantly associated with total cholesterol levels ($p = 0.077$), despite females demonstrating slightly higher mean values than males. Marital status also showed no significant relationship with cholesterol levels ($p = 0.657$). Occupation, however, was strongly associated with total cholesterol levels ($p < 0.001$). Higher mean cholesterol values were observed among workers and unemployed patients compared with other occupational categories, suggesting a potential influence of employment-related and socioeconomic factors. Educational level demonstrated a statistically significant association with total cholesterol levels ($p = 0.044$), with higher mean cholesterol values noted among patients with lower educational attainment. Place of residence was not significantly associated with total cholesterol levels ($p = 0.310$). Income level showed a significant association with total cholesterol levels ($p = 0.001$), with patients in the low-income category exhibiting higher mean cholesterol levels compared with those in moderate- and high-income groups.

**Table 3. Sociodemographic predictors of triglyceride levels among acute Myocardial infarction patient**

| Characteristics | Categories | Triglycerides levels | | Mean | SD | *P* |
|---|---|---|---|---|---|---|
| | | Normal N (%) | High N (%) | | | |
| Age | 18- 29 | 0 (0.0) | 4 (100) | 177.25 | 62.345 | 0.458 |
| | 30- 39 | 1 (11.1) | 8 (88.9) | 242.11 | 77.757 | |
| | 40- 49 | 5 (33.3) | 10 (66.7) | 202.67 | 67.903 | |
| | 50- 59 | 7 (30.4) | 16 (69.6) | 210.48 | 69.081 | |
| | 60 and older | 10 (34.5) | 19 (65.5) | 215.52 | 81.126 | |
| Gender | Male | 15 (27.8) | 39 (72.2) | 211.44 | 68.230 | 0.489 |
| | Female | 8 (30.8) | 18 (69.2) | 215.42 | 85.207 | |
| Marital status | Single | 0 (0.0) | 4 (100) | 177.25 | 62.345 | 0.27 |
| | Married | 23 (31.1) | 51 (68.9) | 211.61 | 72.743 | |
| | Widowed | 0 (0) | 2 (100) | 325.50 | 3.536 | |
| Employment status | Employee | 4 (25) | 12 (75) | 194.75 | 48.627 | 0.936 |
| | Unemployed | 9 (31) | 20 (69) | 215.28 | 77.832 | |
| | Housewife | 8 (30.8) | 18 (69.2) | 215.42 | 85.207 | |
| | Retired | 2 (22.2) | 7 (77.8) | 228.78 | 65.247 | |
| Educational | No formal education | 13 (41.9) | 18 (58.1) | 204.26 | 86.903 | 0.083 |
| | Primary school | 4 (18.2) | 18 (81.8) | 231.64 | 66.826 | |
| | Secondary school | 4 (36.4) | 7 (63.6) | 238.20 | 73.341 | |
| | High school | 0 (0) | 10 (100) | 188.91 | 41.803 | |
| | University level | 2 (33.3) | 4 (66.7) | 188.50 | 56.472 | |
| Residency | Urban | 10 (20.4) | 3 9(79.6) | 229.76 | 75.270 | 0.035 |
| | Rural | 13 (41.9) | 18 (58.1) | 185.84 | 63.231 | |
| Economic status | Low | 12 (41.4) | 17 (58.6) | 232.60 | 95.770 | 0.041 |
| | Moderate | 9 (19.6) | 37 (80.4) | 222.30 | 71.677 | |
| | High | 2 (40) | 3 (60) | 194.14 | 71.819 | |

SD, Standard Deviation, % Percentage, *P* Probability value using Chi Square or Fisher's exact test, N number,

**Table 4. Sociodemographic predictors of total cholesterol levels among acute Myocardial infarction patient**

| Characteristics | Categories | Cholesterol levels | | | Mean | SD | *P* |
|---|---|---|---|---|---|---|---|
| | | Low N (%) | Normal N (%) | High N (%) | | | |
| Age | 18- 29 | 0 (0) | 2(50) | 2(50) | 201.00 | 21.245 | 0.359 |
| | 30- 39 | 1(11.1) | 2(22.2) | 6(66.7) | 211.89 | 45.993 | |
| | 40- 49 | 3(20) | 6(40) | 6(40) | 185.40 | 56.733 | |
| | 50- 59 | 0(0) | 5(21.7) | 18(78.3) | 218.13 | 27.813 | |
| | 60 and older | 4(13.8) | 8(27.6) | 17(58.6) | 210.34 | 59.932 | |
| Gender | Male | 8(14.8) | 13(24.1) | 33 (61.1) | 205.74 | 51.956 | 0.077 |
| | Female | 0 (0) | 10(38.5) | 16(61.5) | 211.50 | 43.716 | |
| Marital status | Single | 0(0) | 2(50) | 2(50) | 201.00 | 21.245 | 0.657 |
| | Married | 8(10.8) | 21(28.4) | 45(60.8) | 206.59 | 50.291 | |
| | Widowed | 0(0) | 0(0) | 2(100.0) | 258.50 | 3.536 | |
| Employment status | Employee | 4(25) | 7(43.8) | 5(31.3) | 171.13 | 39.941 | 0.000 |
| | Unemployed | 0(0) | 6(20.7) | 23(79.3) | 230.38 | 40.253 | |
| | Housewife | 0(0) | 10(38.5) | 16(61.5) | 211.50 | 43.716 | |
| | Retired | 4(44.4) | 0(0) | 5(55.6) | 187.89 | 65.314 | |
| Educational | No formal education | 1(3.2) | 8(25.8) | 22(71) | 222.55 | 48.937 | 0.044 |
| | Primary school | 2(9.1) | 8(36.4) | 12(54.5) | 209.90 | 38.240 | |
| | Secondary school | 2(18.2) | 2(18.2) | 7(63.6) | 205.09 | 53.172 | |
| | High school | 0(0) | 4(40) | 6(60) | 199.45 | 45.543 | |
| | University level | 3(50) | 1(16.7) | 2(33.3) | 161.17 | 51.277 | |
| Residency | Urban | 5(10.2) | 17(34.7) | 27(55.1) | 202.18 | 46.031 | 0.310 |
| | Rural | 3(9.7) | 6(19.4) | 22(71) | 216.19 | 53.541 | |
| Economic status | Low | 2(6.9) | 6(20.7) | 21(72.4) | 214.69 | 44.199 | 0.001 |
| | Moderate | 3(6.5) | 17(37) | 26(56.5) | 207.39 | 48.479 | |
| | High | 3(60) | 0(0) | 2(40) | 168.60 | 74.002 | |

SD, Standard Deviation, % Percentage, *P* Probability value using Chi Square or Fisher's exact test, N number

**Table 5** summarizes the association between selected sociodemographic characteristics and HDL cholesterol levels among patients with acute myocardial infarction. Overall, age, gender, marital status, employment status, educational level, and economic status were not significantly associated with HDL levels ($p > 0.05$). Although variations in mean HDL levels were observed across age groups, with relatively higher means among patients aged 40–49 years and those aged ≥60 years, these differences did not reach statistical significance ($p = 0.347$). Similarly, females exhibited a slightly higher mean HDL level than males, but this difference was not significant ($p = 0.524$). Marital status and employment status also showed no meaningful association with HDL levels. Educational attainment demonstrated modest differences in mean HDL values, with university-educated participants having the highest mean, yet this association remained non-significant ($p = 0.48$). In contrast, residency was significantly associated with HDL levels ($p = 0.008$), with urban residents showing higher mean HDL levels compared with rural residents. Economic status was not significantly related to HDL levels, despite slightly higher mean values among participants with high economic status.

**Table 6** presents the relationship between sociodemographic characteristics and LDL cholesterol levels among patients with acute myocardial infarction. Overall, age, marital status, employment status, residency, and economic status were not significantly associated with LDL levels ($p > 0.05$). Although LDL levels tended to increase with advancing age, particularly among patients aged 50–59 years and those aged 60 years and older, these differences were not statistically significant ($p = 0.133$). Educational status was also significantly associated with LDL levels ($p = 0.015$), as participants with no formal education demonstrated the highest mean LDL levels, while those with university-level education had comparatively lower mean values. In contrast, no significant differences in mean LDL levels were found between urban and rural residents ($p = 0.493$) or across economic status categories ($p = 0.810$).

**Table 5. sociodemographic predictors of HDL levels among acute Myocardial infarction patient**

| Characteristics | Categories | HDL levels | | | Mean | SD | *P* |
|---|---|---|---|---|---|---|---|
| | | Low N (%) | Normal N (%) | High N (%) | | | |
| Age | 18- 29 | 2(50) | 2(50) | 0(0) | 35.00 | 11.576 | 0.347 |
| | 30- 39 | 7(77.8) | 0(0) | 2(22.2) | 39.67 | 17.270 | |
| | 40- 49 | 7(46.7) | 6(40) | 2(13.3) | 43.73 | 13.472 | |
| | 50- 59 | 13(56.5) | 7(30.4) | 3(13) | 37.70 | 10.416 | |
| | 60 and older | 15(51.7) | 13(44.8) | 1(3.4) | 39.41 | 9.128 | |
| Gender | Male | 31(57.4) | 19(35.2) | 4(7.4) | 38.59 | 10.641 | 0.524 |
| | Female | 13(50) | 9(34.6) | 4(15.4) | 41.54 | 13.146 | |
| Marital status | Single | 2(50) | 2(50) | 0(0) | 35.00 | 11.576 | 0.668 |
| | Married | 40(54.1) | 26(35.1) | 8(10.8) | 40.05 | 11.580 | |
| | Widowed | 2(100) | 0(0) | 0(0) | 30.00 | .000 | |
| Employment status | Employee | 12(75) | 2(12.5) | 2(12.5) | 36.37 | 13.391 | 0.121 |
| | Unemployed | 16(55.2) | 11(37.9) | 2(6.9) | 38.07 | 9.308 | |
| | Housewife | 13(50) | 9(34.6) | 4(15.4) | 41.54 | 13.146 | |
| | Retired | 3(33.3) | 6(66.7) | 0(0) | 44.11 | 8.085 | |
| Educational | No formal education | 17(54.8) | 12(38.7) | 2(6.5) | 38.87 | 9.878 | 0.48 |
| | Primary school | 10(45.5) | 10(45.5) | 2(9.1) | 40.50 | 10.331 | |
| | Secondary school | 6(54.5) | 4(36.4) | 1(9.1) | 40.18 | 12.023 | |
| | High school | 8(80) | 0(0) | 2(20) | 36.90 | 17.572 | |
| | University level | 3(50) | 2(33.3) | 1(16.7) | 42.83 | 13.467 | |
| Residency | Urban | 31(63.3) | 17(34.7) | 1(2) | 36.94 | 9.467 | 0.008 |
| | Rural | 13(41.9) | 11(35.5) | 7(22.6) | 43.65 | 13.321 | |
| Economic status | Low | 13(44.8) | 14(48.3) | 2(6.9) | 40.07 | 10.484 | 0.39 |
| | Moderate | 28(60.9) | 13(28.3) | 5(10.9) | 38.89 | 11.952 | |
| | High | 3(60) | 1(20) | 1(20) | 42.40 | 14.993 | |

SD, Standard Deviation, HDL High density of lipoprotein % Percentage, *P* Probability value using Chi Square or Fisher's exact test, N number

**Table 6. Sociodemographic predictors of LDL levels among acute Myocardial infarction patient**

| Characteristics | Categories | LDL levels | | | Mean | SD | *P* |
|---|---|---|---|---|---|---|---|
| | | Low N (%) | Normal N (%) | High N (%) | | | |
| Age | 18- 29 | 0(0) | 1(25) | 3(75) | 107.75 | 10.905 | 0.133 |
| | 30- 39 | 0(0) | 3(33.3) | 6(66.7) | 110.89 | 17.525 | |
| | 40- 49 | 2(13.3) | 5(33.3) | 8(53.3) | 101.33 | 34.539 | |
| | 50- 59 | 0(0) | 3(13) | 20(87) | 129.30 | 31.498 | |
| | 60 and older | 0(0) | 9(31) | 20(69) | 128.90 | 41.941 | |
| Gender | Male | 0(0) | 17(31.5) | 37(68.5) | 120.57 | 33.463 | 0.06 |
| | Female | 2(7.7) | 4(15.4) | 20(76.9) | 121.15 | 41.014 | |
| Marital status | Single | 0(0) | 1(25) | 3(75) | 107.75 | 10.905 | 0.946 |
| | Married | 2(2.7) | 19(25.7) | 53(71.6) | 121.96 | 36.930 | |
| | Widowed | 0(0) | 1(50) | 1(50) | 102.50 | 6.364 | |
| Employment status | Employee | 0(0) | 4(25) | 12(75) | 111.75 | 10.408 | 0.303 |
| | Unemployed | 0(0) | 9(31) | 20(69) | 127.34 | 41.407 | |
| | Housewife | 2(7.7) | 4(15.4) | 20(76.9) | 121.15 | 41.014 | |
| | Retired | 0(0) | 4(44.4) | 5(55.6) | 114.44 | 29.164 | |
| Educational | No formal education | 0(0) | 8(25.8) | 23(74.2) | 125.58 | 33.902 | 0.015 |
| | Primary school | 0(0) | 6(27.3) | 16(72.7) | 127.68 | 41.102 | |
| | Secondary school | 2(18.2) | 1(9.1) | 8(72.7) | 119.20 | 28.039 | |
| | High school | 0(0) | 2(20) | 8(80) | 105.36 | 41.707 | |
| | University level | 0(0) | 4(66.7) | 2(33.3) | 101.33 | 7.891 | |
| Residency | Urban | 2(4.1) | 12(24.5) | 35(71.4) | 128.26 | 35.818 | 0.493 |
| | Rural | 0(0) | 9(29) | 22(71) | 116.02 | 35.111 | |
| Economic status | Low | 1(3.4) | 9(31.0% | 19(65.5) | 127.20 | 50.712 | 0.810 |
| | Moderate | 1(2.2) | 10(21.7% | 35(76.1) | 120.15 | 36.015 | |
| | High | 0(0) | 2(40.0% | 3(60) | 120.62 | 34.083 | |

SD, Standard Deviation, LDL Low density of lipoprotein % Percentage, N number, *P* Probability value using Chi Square or Fisher's exact test

## DISCUSSION

The present study demonstrated that the majority of patients presenting with AMI exhibited abnormal and unhealthy serum lipid profiles, characterized predominantly by elevated total TC, LDL-C, TG, and/or HDL-C. These findings reinforce the well-established role of dyslipidemia as a major modifiable risk factor in the pathogenesis of CADs and AMI.

The findings from the present study indicate that a significant majority of patients presenting with AMI exhibit elevated and unhealthy lipid profiles, this study demonstrate a significant dyslipidemia among patients presenting with AMI, with the majority exhibiting an elevated and atherogenic lipid profile. This observation aligns strongly with established literature recognizing dyslipidemia as a primary and modifiable risk factor for cardiovascular diseases, including AMI (Abera et al., 2024; Pappan et al.,2024). The findings of this study are also in agreement with prior regional and international studies reporting a high prevalence of lipid abnormalities among patients with AMI (Abera et al., 2024; Pappan et al., 2024; Kumar and Sinha, 2020). This observation reinforces the cornerstone pathophysiological model of coronary artery disease, wherein lipid accumulation, particularly within the intimal layer of coronary arteries, initiates and propagates atherosclerotic plaque formation, the principal substrate for most AMI events (Libby et al., 2019). The prevalence of dyslipidemia, characterized by high levels of TC, LDL-C, and triglycerides, often coupled with reduced HDL-C, is well-documented in patients experiencing acute coronary events (Khan et al.,2013, Zuo et al., 2025). Our results are consistent with a substantial body of epidemiological and clinical evidence confirming that disturbances in serum lipids specifically elevated TC, LDL-C, and TG, along with depressed HDL-C are potent, independent risk factors for acute coronary syndromes (Cholesterol Treatment Trialists, 2019).

Serum lipid abnormalities play a central role in the pathophysiology of AMI, and findings across multiple studies consistently demonstrate the presence of atherogenic dyslipidemia among affected patients (Libby et al., 2009; Ference et al., 2017). Low-density lipoprotein cholesterol remains the most critical lipid fraction implicated in atherosclerotic cardiovascular disease. Elevated LDL-C promotes endothelial dysfunction, oxidative modification, and foam cell formation, ultimately leading to plaque rupture and acute coronary occlusion (Ference et al., 2017). The persistently high LDL-C levels reported in patients with AMI align with extensive evidence identifying LDL-C as a primary therapeutic target in both primary and secondary prevention strategies

(Catapano et al., 2020). In contrast, HDL-C, which exerts anti-atherogenic, anti-inflammatory, and antioxidative effects, was consistently reduced, potentially diminishing vascular protection during the acute ischemic phase (Libby et al., 2009; Ridker, 2014). Elevated levels of TC, LDL-C, and TG, together with reduced HDL-C, underscore the contribution of lipid derangements to coronary plaque formation, instability, and thrombosis (Ference et al., 2017; Guijarro C, Cosin-Sales; Madsen et al., 2017; Kosmas et al., 2023). Elevated LDL-C is a central contributor to atherosclerotic plaque formation through its accumulation within the arterial intima, where it undergoes oxidative modification and triggers inflammatory cascades that promote endothelial dysfunction and plaque instability. Rupture of these vulnerable plaques is a key mechanism underlying acute myocardial infarction (Libby, 2021). Hypertriglyceridemia was also a notable finding in several studies. Elevated triglyceride levels are increasingly recognized as an independent cardiovascular risk factor, particularly through their association with triglyceride-rich lipoproteins and small dense LDL particles, which possess enhanced atherogenic and pro-inflammatory properties (Madsen et al., 2017).

Serum lipid levels undergo significant and dynamic alterations following MI, largely due to acute stress, inflammatory responses, and myocardial necrosis. These transient changes have important implications for accurate diagnosis, cardiovascular risk assessment, and prognostic evaluation. Evidence indicates that acute myocardial infarction can rapidly affect lipid parameters, potentially leading to misinterpretation if measured later in the clinical course. Therefore, current clinical guidelines recommend obtaining lipid profiles within the first 24 hours of hospital admission or after clinical stabilization to ensure reliable assessment (Aubiniere-Robb et al., 2019; Catapano et al., 2019). Lipid measurements obtained within 24–48 hours of symptom onset are generally considered to reasonably reflect baseline lipid status and chronic cardiovascular risk (Jain et al., 2017). However, beyond this period, more pronounced metabolic changes occur, typically characterized by reductions in TC, LDL-C, and HDL-C, along with a concomitant increase in TG levels (Pitt et al., 2008).

The findings of the present study indicate that AMI was more prevalent among males and older patients, those patients with lower educational attainment, and lower socioeconomic status. The mean age of patients with AMI in the present study was 53 years. This finding is broadly consistent with previous research conducted in the Kurdistan Region of Iraq, which reported a mean age of 56.2 years (Allami, 2024), as well as another Iraqi study that documented a comparable mean age of 55.5 years (Mohammad et al., 2021). Similarly, data from countries in the Arabian Peninsula have shown a nearly identical mean age of 56 years among patients presenting with ACS (Alhabib et al., 2019). The similarity in age distribution across these regional studies suggests a consistent pattern in the onset of AMI and ACS within Middle Eastern populations. This pattern may be attributed to shared demographic characteristics, a high prevalence of cardiometabolic risk factors, and common lifestyle influences.

Advancing age is widely recognized as a major non-modifiable determinant of CVD and AMI, and the findings of the present study are consistent with this established paradigm. The increased susceptibility to AMI observed with advancing age can be attributed to progressive age-related vascular and myocardial changes, including endothelial dysfunction, arterial stiffening, and reduced vascular compliance. These structural and functional alterations, together with prolonged cumulative exposure to traditional cardiovascular risk factors such as hypertension, dyslipidemia, and diabetes mellitus, accelerate atherogenesis and increase the likelihood of plaque instability and thrombosis (Benjamin et al., 2019; Curtis et al., 2018). Furthermore, our results support previous evidence demonstrating a direct and progressive relationship between age and AMI risk. This association reflects the chronic and cumulative nature of atherosclerotic disease, which evolves over decades and becomes clinically manifest more frequently in older individuals (Libby et al., 2019). In addition, the coexistence of multiple cardiometabolic comorbidities including hypertension, diabetes mellitus, and obesity further amplifies cardiovascular risk in this population (Garcia et al., 2016; Costantino et al., 2016; Mechanic et al., 2015). Findings in the present study indicate that AMI frequently occurs during late middle age in this population, underscoring the need for early risk assessment and timely implementation of preventive strategies to reduce cardiovascular morbidity and mortality. From a clinical and public health perspective, these findings underscore the critical importance of prioritizing aggressive risk factor modification and preventive strategies in older adults. Although age itself cannot be modified, early identification and optimal management of modifiable cardiovascular risk factors may mitigate the overall burden of AMI and improve cardiovascular outcomes in aging populations.

In the present study, gender-based differences were evident in the present study, males were significantly had a higher risk of AMI compared with females. This finding is consistent with regional data from the Kurdistan Region of Iraq, where a male-to-female ratio of 2.5:1 was reported among patients with acute coronary syndrome (Allami, 2024), and supports the well-established observation that men experience AMI more frequently than women, particularly during middle and later adulthood (EUGenMed Cardiovascular Clinical Study Group et al., 2016). Previous studies have further demonstrated that men are approximately three to four times more likely to develop STEMI or NSTEMI, underscoring the magnitude of this sex disparity (Suman et al., 2023) Moreover, longitudinal cohort data indicate that men remain at nearly twice the risk of cardiovascular events even after adjustment for traditional cardiovascular risk factors, suggesting that male sex represents an independent predictor of adverse cardiovascular outcomes (Albrektsen et al., 2016).

The mechanisms underlying this association are multifactorial and involve both biological and behavioral determinants. From a biological perspective, endogenous estrogen is believed to exert cardioprotective effects in premenopausal women through favorable influences on endothelial function, lipid metabolism, and vascular inflammation, thereby delaying the progression of atherosclerosis (Maas and Appelman, 2010; Vaccarino et al., 2011). In addition, differences in gene expression related to sex chromosomes may further contribute to sex-specific cardiovascular risk profiles (Suman et al., 2023). Conversely, men are more frequently exposed to adverse lifestyle and environmental risk factors, including higher rates of smoking and occupational stress, which may accelerate atherosclerotic processes and increase the likelihood of earlier cardiovascular events (Maas and Appelman, 2010). This is consistent with epidemiological evidence showing that men typically present with AMI at younger ages, whereas women tend to present later in life, particularly after menopause, when the protective effects of estrogen decline and cardiovascular risk increases (Vaccarino et al., 2011; Pana et al., 2024). From a clinical perspective, these findings highlight the importance of incorporating sex-specific considerations into cardiovascular risk assessment and prevention strategies. The higher and earlier risk observed among men underscores the need for early identification and aggressive management of modifiable risk factors in this group. At the same time, increased awareness and preventive efforts remain essential for women, particularly in the postmenopausal period when their cardiovascular risk rises substantially. Addressing these sex-specific differences may contribute to improved prevention, earlier intervention, and ultimately better cardiovascular outcomes.

Educational level showed a significant inverse association with AMI risk in the present study, with individuals of lower educational attainment being more affected. Education is a key indicator of health literacy and is strongly linked to health-related behaviors, access to healthcare services, and adherence to treatment and preventive measures. Individuals with lower

education levels are more likely to engage in unhealthy behaviors such as smoking, physical inactivity, and poor dietary practices, all of which increase cardiovascular risk (Dienhart et al., 2023; Stringhini et al., 2017). The findings of this study support the growing recognition that improving educational opportunities and health literacy may contribute to reducing the burden of AMI.

Our findings on education levels and economic status are consistent with extensive research on socioeconomic determinants of health. Lower educational attainment and reduced economic status are consistently linked to an elevated risk of AMI (Kelli et al., 2019). This inverse relationship is often dose-dependent, where higher levels of education correlate with a decreased risk of AMI (Sztaniszlav et al., 2024). The mechanisms underlying this association are multifaceted. Individuals with lower socioeconomic status often face barriers to healthcare access, including preventative care and timely treatment for acute conditions. Furthermore, socioeconomic disadvantage is frequently associated with a higher prevalence of traditional cardiovascular risk factors, such as smoking, poor diet, and physical inactivity. Psychosocial stress, a common consequence of financial strain and limited resources, also plays a significant role by activating neuroendocrine and immune responses that can damage arterial walls and the myocardium (Kelli et al., 2019). Financial constraints can limit access to preventive healthcare, medications, and healthy lifestyle options, while chronic psychosocial stress related to economic hardship may exacerbate atherosclerotic processes and trigger acute coronary events (Schultz et al., 2018). The observed association underscores the role of social inequality as a major contributor to cardiovascular disease and highlights the need for public health policies aimed at reducing socioeconomic disparities.

These behavioral and biological risks are not merely individual choices but are structured by socioeconomic status (SES) through mechanisms such as: Health Literacy and Access to Information: Lower educational attainment can limit understanding of preventive health messages and navigation of complex healthcare systems. Material Deprivation: Lower income constrains access to healthy food options (e.g., fruits, vegetables), safe environments for physical activity, and quality healthcare, including preventive screenings and medications. Psychosocial Stress: Chronic stress associated with financial insecurity, job strain, and lower perceived social status can lead to dysregulation of neuroendocrine pathways (e.g., increased cortisol and catecholamines), promoting inflammation, endothelial dysfunction, and hypertension (Steptoe and Kivimäki, 2013).

In the present study, urbanization was significantly associated with unfavorable lipid profiles, specifically HDL cholesterol and triglyceride levels. Patients living in urban areas exhibited lower HDL and higher triglyceride levels compared with patients residing in rural areas. Systematic reviews and meta-analyses show that urban residents tend to have higher TC, LDL cholesterol, and TG, alongside lower HDL cholesterol, compared with rural populations. These patterns are largely attributed to the urban nutrition transition, reduced physical activity, and increased consumption of processed foods rich in refined sugars and fats (de Groot et al., 2019). Socioeconomic status and education are also strongly linked to dyslipidemia. Individuals with lower education and income levels are more likely to exhibit elevated total cholesterol, LDL-C, and triglycerides, as demonstrated by studies from NHANES, EPIC-Norfolk, and other international cohorts (Shohaimi et al., 2014; Ahmed et al., 2025). While some middle-income countries still report higher lipid levels among high-SES groups, evidence from the PURE study indicates a shift toward greater dyslipidemia burden among lower-SES populations, driven by reliance on inexpensive, energy-dense, ultra-processed foods (Rosengren et al., 2019). Overall, low education, low socioeconomic position, and urban residence are well-established determinants of dyslipidemia. These factors operate through interconnected pathways involving limited health literacy, unhealthy dietary patterns, physical inactivity, chronic psychosocial stress, and restricted access to preventive healthcare, ultimately contributing to more atherogenic lipid profiles (Stringhini et al., 2017; Brunner et al., 2017).

Because convenience sampling was used, the sample may not be fully representative of the underlying population in Duhok, and findings should be interpreted with caution regarding generalizability. Additionally, the cross-sectional measurement of lipids at or near the time of AMI may be affected by the acute phase; this was addressed through recording fasting status and conducting sensitivity analyses restricted to fasting samples. Potential confounders included age, sex, smoking status, hypertension, diabetes mellitus, body mass index (BMI), and family history of cardiovascular disease.

## CONCLUSION

This cross-sectional study comprehensively investigated the lipid profiles of patients with acute myocardial infarction in Duhok City, Iraq; a high burden of dyslipidemia was evident. The results demonstrate a clear lipid atherogenic profile in AMI patients showing increased triglyceride, total cholesterol, and low-density lipoprotein cholesterol levels with significantly lowered high-density lipoprotein. These findings highlight the critical role of dyslipidemia as a modifiable risk factor in the pathogenesis of AMI within this specific population. The pattern of AMI was more frequent among males, urban residents, older age (≥ 50 years), those with low educational status, and moderate levels of socioeconomic status, suggesting potential socio-demographic disparities in cardiovascular health. Based on these findings, specific cardiovascular prevention strategies are recommended in the Kurdistan Region of Iraq, such as early screening for dyslipidemia for high-risk groups, use of effective lipid-lowering therapies, and encouraging healthy lifestyle modifications. Additional multicenter longitudinal studies with larger populations are needed to define regional lipid reference patterns, assess long-term cardiovascular outcomes, and identify genetic and environmental factors contributing to lipid abnormalities. Routine lipid profile assessment at hospital admission along with adequate counseling and evidence-based management should be strengthened to improve AMI prevention and patient outcomes.

### ETHICS APPROVAL

Ethical approval for this study was granted by the Research Ethics Committee at the Directorate of Health in Duhok City (Ref: 08012025, Date: January 8, 2025). Initial institutional permissions were obtained from the Nursing Scientific Committee, Azadi Teaching Hospital (Reg. No. 54), and the Cardiac Center (Reg. No. 151). Formal administrative agreement and facility access were approved by the administrative directorates of both Azadi Teaching Hospital and the Cardiac Center. Informed consent was obtained from all study participants prior to enrollment. Data privacy and confidentiality were strictly maintained; all collected data were anonymized and used exclusively for research purposes.

### DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES

The authors acknowledge that AI-assisted technologies were partially used in the preparation of this manuscript. Specifically, the generative AI tool ChatGPT was used to support language refinement and grammar correction. All content was critically reviewed, edited, and approved by the authors to ensure accuracy, originality, and alignment with academic standards. No AI tools were used to generate data, interpret results, or replace the authors' intellectual contribution.

### DATA AVAILABILITY

The data that support the findings of this study are available from the corresponding author upon reasonable request.

### CONFLICT OF INTEREST

The authors declare no conflict of interest for this study.

### FUNDING

The authors declare that this research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

### AUTHOR'S CONTRIBUTION

R.Y. conceptualized and designed the study. D.F., J.S, and A.I. performed the experiments and data collection. R.Y. conducted the formal statistical analysis. R.Y. and A.I. wrote the original draft of the manuscript. R.Y., D.F., and J.S. reviewed and edited the manuscript. R.Y. supervised the project and acquired funding. All authors have read and agreed to the published version of the manuscript.

### ACKNOWLEDGMENTS

We would like to express our sincere gratitude to all the patients who participated in this study, whose cooperation and contribution made this research possible.

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