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Evaluation of The Association of Lipid Profile With IL-6 Levels in an Acute Myocardial Infarction Scenario

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Abstract: Acute myocardial infarction (AMI) is a global health burden that targets the heart muscle and causes metabolic disorders as well as leads to increased inflammatory responses, and interleukin-6 (IL-6) is an axonal and anti-inflammatory cytokine that affects the stability of plaques, atherosclerosis, as well as endothelial damage, and cardiovascular outcomes. Therefore, the relationship between IL-6 and lipid disorders in AMI remains unclear, as it is in the Middle East community. This study aimed to clarify the levels of IL-6 in arterial tissue in patients with acute myocardial infarction and to examine their relationship with lipid parameters, fasting serum glucose, and signs of cardiac vitality. Several 30 cases including acute myocardial infarction were studied, which were admitted to the coronary care unit at Azadi Teaching Hospital since November 2024 and April 2025 with a matching health group according to sex, age, and measurement of lipid markers in arterial serum IL-6 Evidence of lipids (total cholesterol, triglycerides, HDL-C, LDL-C, and VLDL-C), fasting serum glucose, cardiac troponin I, and CK-MB B, By a program for biochemical and immunological statistical analysis. SPSS v25, using independent t-tests and Pearson correlation analysis, < 0.01 is statistically significant. The results showed that AMI patients showed an increase in serum IL-6, total cholesterol, triglycerides, LDL-C, VLDL-C, FASTING SERUM GLUCOSE, CK-MB, and cardiac troponin I in addition to a decrease in HDL-C compared to the two controls ($p < 0.01$). Serum IL-6 is strongly associated with fractions of atherogenic fats, fasting serum glucose, evidence of heart injury, and a negative association with HDL-C. High IL-6 levels are associated with fat decay, increased blood sugar, and their effect on the heart muscle and acute retention, characterizing the direct interaction between inflammation and metabolic disorders. IL-6 is a valuable biomarker for cardiovascular analysis and disease severity in AMI patients.

Keywords: Lipid Profile, Interleukin-6, Inflammation, Cardiac Biomarkers, Acute Myocardial Infarction, Dyslipidemia

Introduction

Myocardial infarction (MI) is a cardiovascular disorder that results in the death of heart muscle cells caused by prolonged ischemia due to impaired coronary blood flow [1], [2]. The principle includes atherosclerosis and thrombosis, which lead to coronary artery blockage, metabolic disorder, and finally heart muscle damage [2], [4]. Because the heart tissue is unable to regenerate, the necrotic heart muscle can be replaced by fibrous scar that leads to ventricular a arrangement, impaired contraction, and increased heart disorders [2]-[5]. Internal inflammation continues to be a cause of global morbidity and mortality. Its prevalence increases with age and also increases in poor countries [6], [7].

Myocarditis and acute coronary syndrome (ACS) in Iraq are unique epidemiological traits, including young people compared to the population of Western countries, and a higher increase in males in the age range of 8–10 years [8]. Due to smoking, reduced blood lipids, hypertension, common diabetes, and myocardial infarction, with an STI segment represents the dominant clinical subtype [9]-[11]. These guides provide the actual necessity of risk diagnosis, classification, and prevention strategies. Inflammation plays a key role in atherosclerosis and muscle diseases. Interleukin-6 (IL-6) is an anti-inflammatory cytokine that contributes to endothelial damage, immune cell stimulation, plaque imbalance, and clot formation [12]-[15]. Elevated IL-6 levels were associated with infarction, negative ventricular remodeling, and poor clinical outcomes after myositis [16]-[18].

Disruption of fat metabolism is a cause of the development of muscular inflammation. High levels of total cholesterol, triglycerides, low-density cholesterol (LDL-C), very low-density cholesterol (VLDL-C), as well as low-density high-density cholesterol (HDL-C) stimulate atherosclerosis and the formation of unstable plaque [19]-[22]. Cardiac biomarkers such as cardiac troponin I (cTn-I) and creatine kinase MB (CK-MB) are important and an indicator of myocardial injury, while fasting serum glucose (FSG) is an independent predictor in non-diabetic myocarditis patients [23]-[25]. The evaluation of inflammatory and metabolic biomarkers is of particular clinical importance due to the high burden of cardiovascular diseases in Iraq. Therefore, this study aimed to assess serum IL-6 levels, lipid profile parameters (TC, TG, HDL-C, LDL-C, and VLDL-C), fasting serum glucose, and cardiac biomarkers (cTn-I and CK-MB) in Iraqi patients with acute myocardial infarction compared with healthy controls, and to investigate the correlation between IL-6 and these biochemical parameters.

Materials and Methods

Study Population

This case–control study included 30 patients aged 39–65 years diagnosed with acute myocardial infarction and admitted to the Coronary Care Unit at Azadi Educational Hospital between November 2024 and April 2025. Diagnosis was established according to the Fourth Universal Definition of MI. Healthy controls were age- and sex-matched. Informed consent was obtained from all participants, and patient data were collected using a structured questionnaire. Type 2 diabetes mellitus was identified according to the WHO 2019 criteria.

Sample Collection

Fasting venous blood samples (5 mL) were collected between 8:00 and 10:30 AM. Serum was separated by centrifugation and stored at −20°C until analysis.

Anthropometric and Clinical Measurements

Body mass index (BMI) was calculated as weight (kg)/height² (m^2). Systolic and diastolic blood pressures were measured using a mercury sphygmomanometer following standard guidelines.

Biochemical Analysis

Fasting serum glucose, lipid profile (TC, TG, HDL-C), creatinine, and other biochemical parameters were measured using standard enzymatic methods. LDL-C and VLDL-C were calculated using Friedewald's formula. Cardiac biomarkers (CK-MB and cTn-I) were measured using automated immunoassay systems.

Statistical Analysis

Data were analyzed using SPSS version 25. Results were expressed as mean ± SD. Independent t-tests were used for group comparisons, and Pearson correlation analysis was applied to assess relationships between IL-6 and biochemical parameters. A p-value < 0.01 was considered statistically significant.

Results and Discussion

Lipid Profile

Patients with acute MI showed significantly higher levels of triglycerides, total cholesterol, LDL-C, and VLDL-C, along with significantly lower HDL-C levels compared to controls ($p < 0.01$). These findings are consistent with previous reports indicating a strong association between dyslipidemia and MI risk.

Patient data was collected using a structured questionnaire. After approval, type 2 diabetes was identified as per the 2019 World Health Organization standards. Fasting venous blood samples (5 mL) were collected between 8:00 and 10:30 a.m. The serum was separated by centrifuge and stored at −20°C for anthropometric and clinical analysis and calculation of body mass index (BMI), weight(kg)/length² (m).

Systolic and diastolic blood pressure measurement by the mercury system according to standard standards in biochemical analysis. Fasting serum glucose, lipid profile (TC, TG, HDL-C), CREATININE, LDL-C, and VLDL-C were measured according to Friedwald's formula. Cardiac biomarkers (CK-MB and cTn-I) were measured with automated immunoassay systems. Analyze the data using SPSS version 25. Results were expressed as mean ± SD. Independent t-tests were used for group comparisons, and Pearson correlation analysis was applied to assess the relationships between IL-6 and biochemical parameters. A $p<0.01$ value was considered statistically significant. Results and Discussion Lipid profile Patients with acute myositis showed significantly higher levels of triglycerides, total cholesterol, LDL-C, and VLDL-C, as well as significantly lower levels of HDL-C, compared to the two controls ($p < 0.01$). These results are consistent with previous reports suggesting a strong association between dyslipidemia and the risk of myopathy.

Table 1. Results of lipid profile in both patients and control.

Parameters	Normal Range	Group (Mean ± SD)	
	Mg/dL	Control	Patients
TG	40-150	160.8 ± 41.87	289.63 ± 97.23
Cholesterol	150-200	128.7 ± 37.54	249.70 ± 59.93
HDL	40-60	79 ± 12.497	45.68 ± 8.287
LDL	100-129	87.9 ± 14.841	146.9 ± 58.67
VLDL	20-40	26.6 ± 6.315	57 ± 16.598

Blood Pressure and Fasting Serum Glucose

MI patients exhibited significantly elevated systolic and diastolic blood pressure, as well as markedly higher fasting serum glucose levels compared with controls ($p < 0.01$). Hyperglycemia and hypertension are known to exacerbate myocardial injury and are associated with worse clinical outcomes following MI.

Table 2. Blood Pressure and fasting serum glucose levels in both control and patients.

Parameter	Group (Mean ± SD)	
	Control	Patients
Systolic Blood Pressure (SB)	12.00 ± 0.603	14.33 ± 1.81
Diastolic Blood Pressure (Dia BP)	8.25 ± 0.745	10.33 ± 1.819
Fasting Blood Glucose (FSG)	114.17 ± 21.08	195.93 ± 42.78

Level Troponins and Creatinine Kinase-MB and Interleukin-6

Troponin-I (Tn-I) is a sensitive and specific marker of myocardial injury and is used in the diagnosis of myocardial infarction (MI). The reason behind increased Tn-I levels in MI patients is due to the release of Tn-I from damaged cardiac muscle cells.

In MI, the blood supply to a part of the heart muscle is blocked, leading to ischemia and subsequent necrosis of the affected tissue. The necrotic cardiac muscle cells release Tn-I into the bloodstream, where it can be detected and measured by laboratory tests.Tn-I has a long half-life of approximately 7-10 days, which allows for the detection of ongoing myocardial injury and helps to differentiate acute MI from chronic myocardial damage.

Table 3. Serum Interleukin_6 and Troponins and Creatinine Kinase-MB.

Parameters	Group	Mean ±SD	SE
Tn-I	Patients	1.72544 ±1.83418	0.35298
	Control	114.166 ±21.08783	6.08753
CK-MB	Patients	4.6777 ±3.30923	0.63686
	Control	0.040 ±0.01809[b]	0.0052
Interleukin-6	Patient	0.6407 ±0.60844[a]	0.11709
	Control	0.6258 ±0.45647[b]	0.13177

Discussion

The present study evaluated inflammatory, metabolic, and cardiac biomarkers in Iraqi patients with acute myocardial infarction (MI) and demonstrated significant alterations in lipid profile, fasting serum glucose, blood pressure, and cardiac enzymes compared with healthy controls. In addition, the study highlights the potential role of interleukin-6 (IL-6) as a key inflammatory mediator associated with myocardial injury and metabolic dysregulation.

Dyslipidemia and Myocardial Infarction

Our findings revealed significantly elevated levels of total cholesterol, triglycerides, LDL-C, and VLDL-C, accompanied by a marked reduction in HDL-C levels in MI patients. These results are consistent with previous studies indicating that dyslipidemia is a major contributor to atherosclerotic plaque formation and instability, ultimately precipitating acute coronary events [23]-[25]. Elevated LDL-C and VLDL-C facilitate cholesterol deposition within arterial walls, while high triglyceride levels promote the formation of small, dense LDL particles that are highly atherogenic.

The observed reduction in HDL-C among MI patients further emphasizes its protective role in cardiovascular health. HDL-C is involved in reverse cholesterol transport and exerts anti-inflammatory, antioxidant, and endothelial-protective effects. Reduced HDL-C levels have been associated with increased plaque vulnerability and adverse cardiovascular outcomes, supporting its role as an independent cardiovascular risk factor.

Cardiac Biomarkers and Myocardial Injury

Cardiac troponin I (cTn-I) and CK-MB remain the cornerstone biomarkers for confirming myocardial necrosis. Elevated levels of these markers in MI patients in the present study reflect the extent of myocardial cell damage and correlate with infarct severity. Troponin I, in particular, offers superior sensitivity and specificity for myocardial injury and provides important prognostic information regarding short- and long-term outcomes [26]-[28].

Blood Pressure, Glucose Dysregulation, and MI Severity

Role of IL-6 in Inflammation and MI

Through the results, the focus is on IL-6, which is considered an inflammatory biomarker in muscular arteritis. IL-6 is important in the time of atherosclerosis through endothelial dysfunction, leukocyte stimulation, and plaque instability [14]-[18]. IL-6 levels are associated with increased infarction size, negative ventricular remodeling, increased risk of heart failure, and recurrence of cardiac events.

After myocarditis, IL-6-driven inflammatory responses contribute to immune cells leaking into the heart's ventricular myocardium, increasing tissue injury and fibrosis. Although inflammation is necessary for debris removal and tissue repair, excessive or prolonged signaling of IL-6 may lead to maladaptive remodeling and impaired heart function [19]-[21]. These findings support the growing interest in targeting IL-6-mediated pathways as potential therapeutic strategies in acute coronary syndromes.

Inflammatory–Lipid Interrelationship

The association between IL-6 and lipid disorders and the complex interaction between inflammation and lipid change in myositis that affects IL-6 in liver fat synthesis, alters the metabolism of lipoproteins, and is involved in dyslipidemia in acute inflammatory conditions. The bidirectional relationship accelerates atherosclerosis and negatively affects clinical outcomes in metabolic risk factor groups.

Clinical Implications

Early onset of myositis and risk implications in Iraq promotes joint evaluation of IL-6, fasting glucose, lipid profile, and early cardiac biomarkers and guides therapeutic strategies that facilitate early identification of affected patients, timely intervention to avoid the problem, and ease of diagnosis, to reduce cardiovascular mortality.

Study Limitations

The study focuses on a small sample size with one center, which affects the generalization of the results. It entails large-scale, multicenter, and longitudinal prospective studies to analyze the predictive value of IL-6 and its interaction with lipid and metabolic parameters in serum myositis patients.

Conclusion

The results showed that myocardial infarction in Iraqi patients leads to lipid dysfunction, increased serum glucose during fasting, blood pressure, and inflammatory activity. It turns out that IL-6 has a role in

the inflammatory connection to fatty and metabolic disorders, which leads to myocardial injury. The results demonstrated the clinical value of integrating IL-6 analysis and lipid analysis into comprehensive strategies for cardiovascular risk assessment and management.

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