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Original Article

Association of Serum Adiponectin with Cardiac Enzymes in Diabetic Patients Having Coronary Heart Disease

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Abstract

Objective: To determine serum adiponectin levels and assess their relationship with cardiac enzymes in patients with type 2 diabetes and coronary heart disease.

Methodology: This Case-Control Study was conducted at Khyber Medical College, Peshawar, Khyber Pakhtunkhwa, Pakistan, from October 2025 to March 2026. The study included 60 patients with type 2 diabetes mellitus presenting with a first myocardial infarction and 60 age- and sex-matched healthy subjects. We measured serum adiponectin and troponin I using ELISA kits, and CK, LDH, and ALT using a Micro Lab Analyzer. We analyzed the collected data using SPSS version 21. We used independent-samples t-tests to compare groups and Pearson correlations to assess associations. We considered $p < 0.05$ statistically significant.

Results: Serum adiponectin levels were significantly lower in cases (3.2 ± 1.1 vs. 11.7 ± 2.6 $\mu\text{g/mL}$, $P < 0.0001$). All cardiac enzymes were significantly higher in cases: Troponin I (15.7 ± 14.6 ng/mL vs. 0.014 ± 0.02 ng/mL) and CK (389 ± 43.36 vs. 165 ± 6.78 U/L). LDH (474.54 ± 40.53 vs. 321.59 ± 28.9 U/L) and ALT (115.28 ± 25.63 vs. 45.37 ± 14.6 U/L). We observed a negative correlation between adiponectin and troponin I ($r = -0.62$, $p < 0.001$) and CK ($r = -0.54$, $p < 0.001$).

Conclusion: Serum adiponectin is significantly low in type 2 diabetic patients with coronary heart disease (CHD) and is associated with significantly high cardiac enzymes in these cases with myocardial infarction. Adiponectin may serve as a protective biomarker in diabetic coronary heart disease.

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Introduction

Coronary heart disease [CHD] is the leading cause of mortality among the diabetic population. As the most common complication of type 2 diabetes, CHD accounts for 80% of mortality in this population [1]. Type 2 diabetic patients face a 2- to 4-fold higher risk of cardiac events [2]. Researchers are exploring novel biomarkers that may help diagnose or predict outcomes in these metabolic disorders [3]. Adiponectin is one such biomarker. White adipose tissue secretes this peptide hormone, first identified in 1995. Adiponectin constitutes nearly 10% of serum proteins and typically ranges from 2 to 30 µg/mL. In addition to important functions in the liver and skeletal muscles, adiponectin exerts cardioprotective effects by interacting with its receptor, T-cadherin, on cardiac myocytes [5]. Adiponectin has been proposed to have anti-inflammatory, lipid-regulatory, and anti-atherogenic effects, which may underlie its cardioprotective role. Most of these effects are under investigation to clarify its potential diagnostic, prognostic, and therapeutic functions [6,7,8]. This study aims to determine serum adiponectin and key cardiac enzyme levels, including troponin I, creatine kinase, lactate dehydrogenase, and alanine aminotransferase, in diabetic patients experiencing a first myocardial infarction. It also examines whether adiponectin is associated with these cardiac enzymes in these patients [9]. This study's novelty lies in its simultaneous assessment of serum adiponectin and multiple cardiac enzymes, including troponin I, creatine kinase, lactate dehydrogenase, and alanine aminotransferase, among type 2 diabetic patients experiencing a first myocardial infarction. By examining the relationships between adiponectin levels and these biochemical markers, this study provides locally relevant evidence regarding the potential role of adiponectin as a biomarker of myocardial injury in diabetic patients with coronary heart disease.

Materials and Methods

This Case Control study was conducted at Khyber Medical College, Peshawar, Khyber Pakhtunkhwa, Pakistan. Group A comprised 60 patients with type 2 diabetes who had a first myocardial infarction, while Group B comprised 60 age- and sex-matched healthy controls. All patients were selected randomly. Patients previously diagnosed with CHD and receiving treatment were excluded from the study. All participants with any other major illness, such as liver disease, kidney disease, thyroid disease, endocrine abnormalities, or malignancy, were excluded from both the case and control groups. We conducted the study over six months. We obtained written informed consent from all participants. We recorded a detailed history and demographic characteristics using a structured questionnaire. We also recorded clinical parameters, including blood pressure, height, weight, and BMI.

Biochemical Analysis

We collected fasting blood [5 mL] from all participants. We centrifuged the blood at 4500 rpm for 5 minutes to obtain clear serum. We determined adiponectin and troponin I using ELISA kits from BioVendor, Germany, and the Metrolab Clinical Chemistry Analyzer. We analyzed other cardiac enzymes, including CK, LDH, and ALT, using the Micro Lab Chemistry Analyzer.

Participants

The study included 60 type 2 diabetic patients with a first episode of myocardial infarction (Group A) and 60 age- and sex-matched healthy controls (Group B), giving a total sample of 120 participants. Patients with previously diagnosed coronary heart disease and those receiving treatment were excluded. Participants with major comorbidities, including liver disease, kidney disease, thyroid disease, endocrine disorders, or malignancy, were also excluded. We randomly selected all participants who provided written informed consent.

Statistical Analysis

We analyzed data using SPSS version 21. We compared continuous variables with normal distribution between groups using the independent-samples t-test, and used the Mann-Whitney U test for non-normally distributed variables, including troponin I. We assessed the association between serum adiponectin and cardiac enzymes using Pearson's correlation coefficient (r). We considered P <0.05 statistically significant.

Results

The groups were similar in age (56.4±8.2 vs. 55.9±7.9 years; P=0.73) and sex distribution (62% male in each group). Table 1 shows the biochemical parameters of the two groups. Adiponectin levels were markedly lower in patients with MI than in the control group [3.2±1.1 vs. 11.7±2.6 µg/mL, P < 0.0001]. Cardiac enzymes, including troponin I, creatine kinase, lactate dehydrogenase, and alanine aminotransferase, were significantly higher in group A with MI than in the control group (p < 0.0001). We used Pearson's correlation coefficient [r] to assess the association between adiponectin and cardiac enzymes, as shown in Table 2. We observed a significant negative correlation between adiponectin and troponin I [r = -0.62, p < 0.001], CK [r = -0.54, p < 0.001], and LDH [r = -0.31, p = 0.016], but no significant association with ALT.

Table 1. Comparison of biochemical parameters in the study groups

Variable	Group A Cases means±SD	Group B Control means±SD	P value
Adiponectin µg/ mL	3.2±1.1	11.7±2.6	<0.0001
Troponin I ng/mL	15.68 ± 14.57	0.014 ± 0.02	<0.0001
CK U/L	389.0 ± 43.4	165.0 ± 6.8	<0.0001
LDH U/L	474.5 ± 40.5	321.6 ± 28.9	<0.0001
ALT U/L	115.3 ± 25.6	45.4 ± 14.6	<0.0001

Values are presented as mean ± standard deviation (SD). Group A included 60 type 2 diabetic patients with a first myocardial infarction, and Group B included 60 age- and sex-matched healthy controls. We considered P < 0.05 statistically significant. CK, creatine kinase; LDH, lactate dehydrogenase; ALT, alanine aminotransferase.

Table 2. Association of adiponectin with cardiac enzymes.

Variables	r	p
Troponin I	-0.62	<0.0001
CK	-0.54	<0.0001
LDH	-0.31	0.016
ALT	-0.22	0.10

We used Pearson's correlation coefficient (r) to assess the association between serum adiponectin and cardiac enzymes. A negative r value indicates an inverse association. We considered p < 0.05 statistically significant. CK, creatine kinase; LDH, lactate dehydrogenase; ALT, alanine aminotransferase.

Discussion

Serum adiponectin levels vary widely in health and disease. A research gap remains regarding adiponectin levels in diabetic subjects with CHD, especially MI, and their association with cardiac enzymes in our region. In the present study, we found significantly lower adiponectin in diabetic cases with myocardial infarction. This is the first prospective cohort study to report hypoadiponectinemia with CHD [10]. In the present

study, serum adiponectin was significantly lower in type 2 diabetic patients with a first myocardial infarction than in healthy controls. These findings are consistent with previous reports linking hypoadiponectinemia with cardiovascular disease, type 2 diabetes mellitus, coronary heart disease, and metabolic syndrome [11]. A study described adiponectin as a potential therapeutic target because of its association with metabolic and cardiovascular disorders [12]. Several mechanisms may contribute to adiponectin's cardioprotective effects, including improved insulin sensitivity, increased endothelial nitric oxide production, reduced macrophage foam-cell formation, and inhibition of vascular smooth-muscle proliferation and calcification [13]. The marked reduction in adiponectin observed in patients with myocardial infarction may also reflect inflammatory responses and increased interaction of adiponectin with T-cadherin receptors on injured cardiomyocytes [14,15]. We observed significant inverse correlations between adiponectin and troponin I, CK, and LDH, indicating an association between lower adiponectin levels and greater biochemical evidence of myocardial injury. A similar inverse relationship between adiponectin and CK-MB has been reported in patients with ST-elevation myocardial infarction [16,17]. An association between lower adiponectin levels and higher troponin I concentrations has also been reported in STEMI patients [18]. However, because this study was cross-sectional, these associations should not be interpreted as evidence of causality. The non-significant correlation with ALT suggests that the observed relationship was more closely linked to cardiac injury than to ALT levels in this study. We observed a significant negative association between adiponectin levels and cardiac enzymes, including troponin I, CK, and LDH [19]. This strong correlation further supports our findings by ruling out confounding effects from tissue injury and strengthens the possible causal relationship between decreased adiponectin and myocardial injury. The non-significant correlation between adiponectin and ALT suggests preserved liver integrity in our cases; this further indicates that low adiponectin is more likely a specific response to cardiac myocyte injury rather than a chance finding [20]. A study of ST-elevation myocardial infarction [STEMI] patients reported a rapid decline in adiponectin levels after reperfusion, with the lowest level observed at 24 hours. This reduction was also inversely correlated with CK-MB [$p=0.013$] [21]. A study of 73 STEMI patients reported similar results: an adiponectin level ≤ 1.8 ng/mL was significantly associated with higher troponin I levels [45.01 vs. 5.46 ng/mL, $p=0.03$]. This study identified a cut-off value of 1.8 ng/mL for labeling patients at risk, consistent with the markedly reduced levels [3.2 ± 1.1 $\mu\text{g/mL}$] reported in the present study [22]. Single time-point measurement and lack of long-term follow-up may be limitations. Future large-sample prospective studies are recommended to further confirm the relationship between adiponectin and cardiac enzymes.

Conclusion

Adiponectin is markedly reduced in type 2 diabetic subjects having CHD and is negatively associated with cardiac enzymes. These findings help identify the possible preventive, diagnostic, and prognostic value of adiponectin in patients with myocardial infarction.

Authors' Contributions

Sadaf Durrani: Conceptualization, study design, data acquisition, data analysis, manuscript drafting, and critical revision.

Ubaid Ur Rahman: Conceptualization, study design, supervision, interpretation of findings, critical revision of the manuscript, and final approval of the version to be published.

Ubaidullah: Data acquisition, data analysis, literature review, and manuscript revision.

Naheed Khattak: Data acquisition, data analysis, preparation of tables and figures, and critical revision of the manuscript.

Safiyya Ubaidullah: Data acquisition, data analysis, literature review, manuscript drafting and revision, and reference verification.

All authors: Read and approved the final manuscript and agreed to be accountable for all aspects of the work.

Conflict of interest: None

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Ethical Approval

The Institutional Research and Ethical Review Board (IREB) of Khyber Medical College, Peshawar, approved the study on 08 January 2026 (Approval No. 16/IREB/DME/KMC).

Strength: The study's well age-matched controls and highly significant p -values for the correlation between adiponectin and cardiac enzymes are strengths.

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