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Adiponectin as a Potential Biomarker in Acute Myocardial Infarction and Association with Renal Dysfunction

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Abstract

Background: The association between serum adiponectin levels and acute myocardial infarction, particularly how adiponectin predicts the development of acute myocardial infarction in patients, remains unresolved. Hence, we aimed to determine whether higher adiponectin levels predict cardiovascular events and mortality in these patients.

Methods: At Al-Nasiriyah Heart Hospital, 60 patients diagnosed with acute myocardial infarction (AMI) and 60 healthy controls participated in a case-control study. ELISA was used to measure serum ADIPOQ levels, and fluorescent immunoassay was used to measure Hs-TnI levels. Serum creatinine, blood urea, estimated glomerular filtration rate (eGFR), and fasting blood glucose were also measured.

Results: When comparing AMI patients with the control group, the mean body mass index (BMI), glomerular filtration rate, urea, creatinine, and Hs-TnI levels were significantly higher. On the other hand, adiponectin levels and estimated glomerular filtration rate were considerably lower in patients who had experienced an acute myocardial infarction.

Conclusion: Patients with MI have significantly lower serum AdipoQ levels, which are linked to elevated cardiac injury markers (Hs-TnI) and compromised renal function.

1. Introduction

Myocardial infarction (MI) is one of the main causes of death. Nine treatable risk indicators, according to international studies, include weight gain, high cholesterol, and hypertension, which can reach 90%. [1] A susceptible atherosclerotic plaque ruptures to cause coronary artery occlusion, or blockage, which is the most frequent cause of MI. [2] It has been reported that the major portion of deaths (>50%) in patients with diabetes mellitus (DM) are due to cardiovascular (CV) causes. [3] A higher risk of cardiovascular and metabolic disorders, including hypertension, coronary atherosclerosis, myocardial hypertrophy, diabetes, dyslipidemia, and elevated cardiovascular morbidity and mortality, is linked to obesity. [4]

An estimated 200,000 recurrent AMIs and 605,000 incident AMIs happen annually in the US. [5] Even though the prognosis is better and the incidence is relatively low, AMI can be deadly and cause long-term harm at a young age. [6] Even though the global prevalence of MI-associated mortality has decreased by [7], the prevalence of heart failure is still significant worldwide. Approximately 290 million Chinese people have cardiovascular disease, with 11 million of them having coronary atherosclerotic heart disease, of which myocardial infarction is the most severe and fatal condition. [8]

In 1995, adiponectin also referred to as ADIPOQ, Acrp30, GBP-28, and apM1—was initially identified. [9, 10] Adipocytes in white adipose tissue release ADIPOQ, a single-chain adipokine consisting of 244 amino acids and with a molecular weight of roughly 26 kDa. However, osteoblasts, fetal tissue, myocytes, cardiomyocytes, and bone marrow also produce it. [11, 12] It is present in serum in three main forms: hexamer, trimer, and high molecular weight (HMW) multimer. [13] Because of its cardioprotective, anti-inflammatory, and anti-atherosclerotic properties, ADIPOQ is crucial for cardiovascular disorders. [14]

One cardiac-specific biomarker for the early identification and assessment of myocardial injury is high-sensitivity troponin I (hs-TnI). [15] The troponin complex, which controls cardiac muscle contraction, includes troponin I. It has very little bloodstream presence under physiological conditions. Nevertheless, troponin I is released into the bloodstream when cardiomyocytes are damaged by ischemia or other types of stress. [16] Hs-TnI assays are a promising approach for cardiovascular risk assessment in the general population because they can identify remarkably low levels of cardiac troponin in individuals who seem healthy and asymptomatic. [17] The purpose of the study is to assess how ADIPOQ levels in AMI patients vary and how these variations relate to biochemical markers such as GFR and Hs-TnI.

2. Materials and Methods

Samples were taken from patients with acute myocardial infarction (MI) at Al-Nasiriyah Heart Hospital in Thi-Qar Governorate between December 2024 and March 2025, 8 to 12 hours after the onset of symptoms, and from 60 patients (29 males and 31 females) were included in the first group and 60 healthy people were assigned to the second group. The study only included patients with acute MI; patients with a history of cancer, autoimmune diseases, or chronic kidney disease were not included, nor were those taking drugs that affect adipokines, such as corticosteroids. Participants who were pregnant or nursing were also not allowed to take part.

2.1. Sample Collection

Five ml of venous blood was collected from the patients and healthy people in gel and EDTA tubes; one ml of blood was used with EDTA to make the complete blood picture; the other four ml of blood were put in a gel tube and then centrifuged at 3000 rpm for 10 min to obtain serum and the separated serum was then transferred into multiple Eppendorf tubes to avoid errors and compensate if an error occurs and store at -20°C until used for quantification of immunological parameters (ADIPOQ and Hs-TnI). The Mindray BS-230 clinical chemistry analyzer (China) was used to measure the results of glucose and renal function tests. Using a sandwich methodology and enzyme-linked

immunosorbent assay (ELISA) kits from Biotek, the concentrations of human ADIPOQ (ADIPOQ; E1550Hu) were measured. The AFIAS 6 fluorescence immunoassay system (South Korea) was used to measure the levels of Hs-TnI.

2.2. Statistical Analysis

This study employed an extensive statistical approach, using Excel for data management and GraphPad Prism 9. Descriptive statistics summarized categorical variables as frequencies/percentages and numerical variables using means with standard deviations (SD) and medians. Proportions and means were reported, and inferential analyses were performed (Chi-square or Fisher's Exact tests for categorical associations and independent samples t-tests for numerical comparisons) between AMI patients and the healthy group.

3. Results

In this study, the intricate interactions among cardiac biomarkers, lipid profiles, and adipokines in acute myocardial infarction (AMI) are examined. Insights into the pathophysiology of AMI and the possible function of adipokines as therapeutic targets and diagnostic indicators in cardiovascular disease are provided by this thorough analysis.

The demographic details of both the healthy group and the acute myocardial infarction (AMI) patients are compiled in Table 1. Body mass index (BMI), age, and sex were compared between the two groups. The findings revealed that the mean age of AMI patients was higher (55.4 ± 7.1 years) than that of the healthy group (48.3 ± 9.8 years), with a non-significant difference at the significance level ($p \geq 0.05$). The AMI group had a greater percentage of men (60% vs. 48%) and a lower percentage of women (40% vs. 52%), according to the gender distribution; however, these differences were not statistically significant. The BMI of AMI patients was found to be significantly higher than that of the healthy group, with mean values of 30.0 ± 4.2 kg/m², compared to 25.3 ± 3.1 kg/m².

Table 1. Comparison of Demographic Characteristics Between Patients with Acute Myocardial Infarction and the Healthy Group

Characteristic	Healthy *n* = 60	AMI *n* = 60	*p*
Age (years)			
Mean ±SD	**48.3 ± 9.8**	**55.4 ± 7.1**	**0.1 I NS**
Gender			
Male, *n* (%)	**29 (48%)**	**36 (60%)**	**0.2 C^{NS}**
Female, *n* (%)	**31 (52%)**	**24 (40%)**	
BMI (kg/m²)			
Mean ±SD	**25.3 ± 3.1**	**30 ± 4.2**	**<0.001 I*** **

n: number of cases; SD: standard deviation; Fisher's Exact Test.; I: independent samples t-test; NS: not significant (p

Table 2 compares glucose levels and kidney function parameters between acute myocardial infarction (AMI) patients and healthy controls. The results showed a significant increase ($p < 0.001$) in the levels of fasting blood glucose (FBS: 167 ± 75.7 vs. 106 ± 18.44 mg/dL), blood urea (51.5 ± 36.4 vs. 29.6 ± 6.5 mg/dL), and serum creatinine (1.46 ± 1.0 vs. 0.7 ± 0.2 mg/dL) in the AMI group compared to the healthy control group. The results also showed a significant decrease in the estimated glomerular filtration rate (eGFR: 58.4 ± 20.0 vs. 151 ± 42.9 ml/min/1.73 m²) compared to the healthy control group. These findings indicate a marked impairment of glucose regulation and renal dysfunction in patients with myocardial infarction, highlighting the metabolic and systemic disturbances associated with myocardial infarction.

Table 2. Comparison of Renal Function and Glucose Homeostasis in Acute Myocardial Infarction Patients and the Healthy Group

Characteristic	Healthy *n* = 60	AMI *n* = 60	*p*
FBS (mg/dL)			
Mean ±SD	**106 ± 18.44**	**167 ± 75.7**	**<0.001 I*** **
Blood urea (mg/dl)			
Mean ±SD	**29.6 ± 6.5**	**51.5 ± 36.4**	**<0.001 I*** **
Serum creatinine (mg/dl)			
Mean ±SD	**0.7 ± 0.2**	**1.46 ± 1.0**	**<0.001 I*** **
eGFR (ml/min/1.73 m²)			
Mean ±SD	**151 ± 42.9**	**58.4 ± 20.0**	**<0.001 I*** **

The symbol *** indicates a highly significant difference ($p < 0.001$) FBS: fasting blood sugar, eGFR: Estimated Glomerular Filtration Rate, I: independent samples t-test.

Table 3 compares troponin I levels (ng/L). The results showed a significant increase ($p < 0.001$) in the mean troponin I level in patients with myocardial infarction (39.3 ± 12.9 ng/L) compared to the healthy control group (6.1 ± 2.4 ng/L).

Table 3. Comparison of High-Sensitivity Troponin I Levels Between Acute Myocardial Infarction Patients and the Healthy Group

Characteristic	Healthy *n* = 60	AMI *n* = 60	*p*
High-sensitivity troponin I (ng/L)			
Mean ±SD	**6.1 ± 2.4**	**39.3 ± 12.9**	**<0.001 I*** **

Statistical significance was indicated by ***p<0.001. I: independent samples t-test.

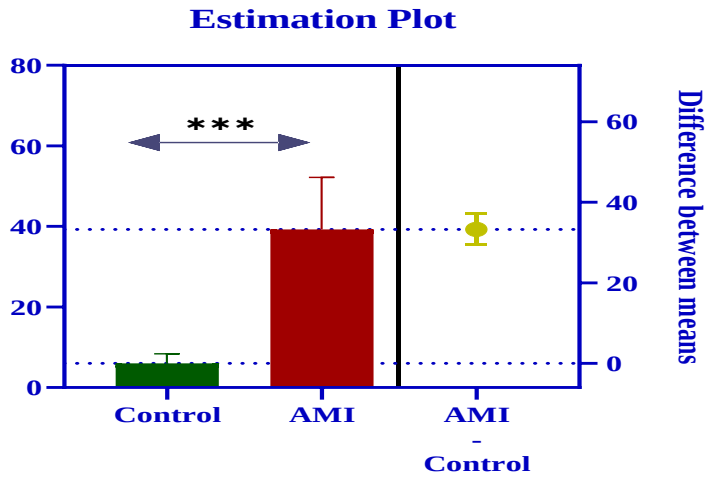

Fig. 1. Bar chart showing comparison of High-sensitivity troponin I among Acute Myocardial Infarction Patients and the Healthy Group

Table 4 provides a comparative analysis of ADIPOQ levels in the serum of patients with acute myocardial infarction (AMI) and healthy controls. The results showed a significant decrease ($p<0.001$) in ADIPOQ levels in patients with acute myocardial infarction (3.88 ± 1.8 mg/L) compared to healthy controls (8.34 ± 4.4 mg/L). This highlights the dysregulation of adipokine levels in the pathophysiology of acute myocardial infarction.

Table 4. Comparative ADIPOQ Levels in Acute Myocardial Infarction Patients and the Healthy Group

Characteristic	Healthy *n* = 60	AMI *n* = 60	*p*
Adiponectin (mg/L)			
Mean ±SD	**8.34 ± 4.4**	**3.88 ± 1.8**	**<0.001 I*** **

Statistical significance was indicated by ***p<0.001. I: independent samples t-test.

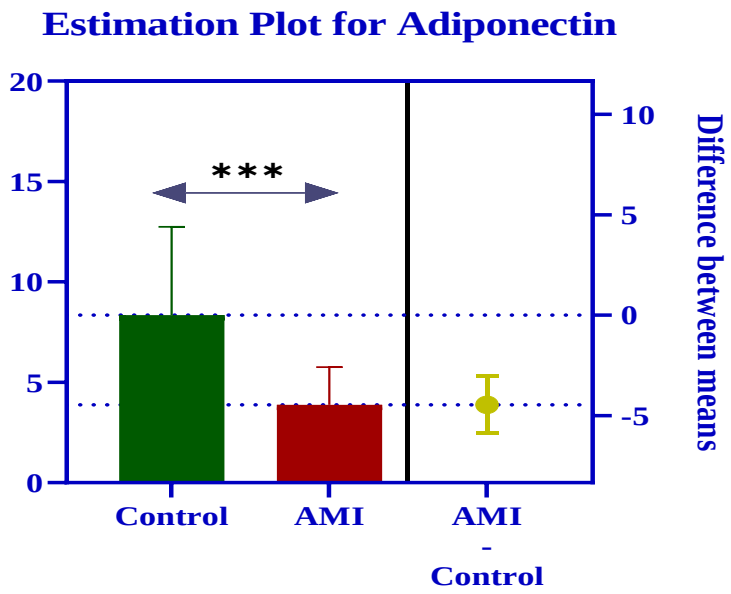

Fig. 2. Bar chart showing comparison of ADIPOQ among Acute Myocardial Infarction Patients and the Healthy Group

Table 5 explains the ROC curve study used to assess adiponectin's diagnostic effectiveness in AMI. ADIPOQ (cut-off ≤4.52 µg/mL) showed moderate accuracy (73%) and AUC (91%). Biomarkers exhibited statistically significant discriminative power (p=0.001). Notably, ADIPOQ had higher specificity (82%).

Table 5. ROC Curve Analysis of Adiponectin's Diagnostic Performance in Acute Myocardial Infarction

Variables	Cut-off value	Sens**%	Spec%	PPV**	NPV	Accuracy	AUC%	*P*-value (AUC= 0.05)
Adiponectin (µg/mL)	**≤ 4.52**	**87**	**82**	**83**	**87**	**73**	**91**	**0.001***

Sens: Sensitivity; Spec: Specificity; PPV: positive predictive value; NPV: negative predictive value; Accuracy [(Sensitivity + Specificity) - 1]; AUC: area under curve;

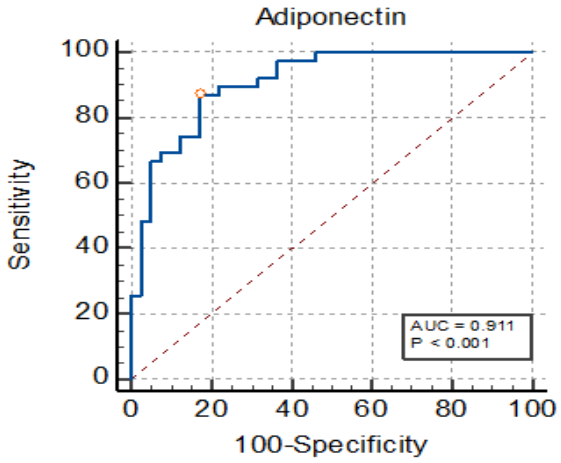

Fig. 3. ROC Curve chart of ADIPOQ in Acute Myocardial Infraction

4. Discussion

The research found that there was a significant difference in mean BMI between AMI patients and the healthy group. This demonstrates how obesity has been linked to an increased risk of cardiovascular disease, especially in AMI. These results are corroborated by numerous studies, which strengthen the established link between a higher BMI and a higher risk of cardiovascular events. [18, 19] Obesity, most of which is measured by BMI and is caused by excess body fat, especially visceral lipid (lipid surrounding the organs), causes inflammatory changes and metabolic disturbances that negatively affect heart health. This fat accumulates on the walls of the arteries, leading to narrowing of the artery walls and reducing blood flow, which increases the risk of MI. [20]

The study found a significant difference in FBS and RFT between AMI patients and controls, with AMI patients showing higher mean FBS, BU and CREA values and reduced eGFR. These findings are consistent with previous study by Viveca Ritsinger (2021) on 45,468 patients with AMI, but without diabetes in SWEDEHEART (Swedish Web System for Enhancement and Development of Evidence Based Care in Heart Disease Evaluated According to Recommended Therapies) concluded that patients without diabetes are identified by elevated levels of glucose at AMI admission as being at higher risk for long-term consequences, specifically hospitalization for heart and kidney failure. [21] Because glucose is a widely accessible and reasonably priced biomarker that is elevated as a result of the body's stress response to AMI, it may be useful for early detection. [22] Acute hyperglycemia is a normal response of the body when exposed to severe stress such as MI, where the body secretes hormones such as cortisol and adrenaline that elevated blood glucose levels. [23]

The study showed a decrease in eGFR. [24] GFR declines as a result of hemodynamic abnormalities brought on by a decline in cardiac output and venous return to the body. Additionally, the system known as the renin-angiotensin system (RAAS) and the sympathetic nervous system are activated in AMI patients, which might worsen kidney

injury and cause vasoconstriction. [25] Decreased GFR after MI occur as a result of

several interconnected mechanisms, the most prominent causes are: Decreased renal perfusion after an MI, the heart pumps less blood, leading to decreased renal ischemia and decreased GFR, elevated blood pressure in the blood vessels due to heart failure impedes blood flow to the kidneys and reduces GFR, activation of the sympathetic nervous system and RAAS this activation leads to renal vasoconstriction, increasing the workload on the kidneys and reducing GFR and inflammatory response resulting from an MI leads to damage to kidney cells. [26]

This study demonstrates a significant elevation of Hs-TnI levels in patients with AMI compared to controls. The marked increase in Hs-TnI underscores its critical role in detecting even minor myocardial injury, aiding in the early diagnosis of AMI, particularly in cases with unclear clinical presentations. [27] The researchers observed that the quantity of vesicles at the surface of the cardiomyocytes rises when ischemia is induced and that these vesicles are reabsorbed into the cytoplasm when ischemia is removed. [28] When an MI occurs, myocardial tissue is exposed to oxygen deprivation, leading to rupture of myocardial cell membranes and the release of troponin into the bloodstream and severe ischemia triggers an inflammatory response, leading to increased vascular permeability and troponin accumulation in the bloodstream. [29]

This study shows that there is a significant decrease in serum ADIPOQ levels between control subjects and AMI patients compared to the healthy group. ADIPOQ seems to be a symptom of compromised metabolic signaling, which is connected to the development of heart failure in patients with CVD undergoing cardiovascular surgery. AMPK acts as a master regulator of cellular energy homeostasis. When activated by energy stress or adiponectin signaling via AdipoR1/R2, AMPK promotes ATP-generating processes (such as fatty acid oxidation and glucose uptake) and inhibits ATP-consuming pathways (such as lipogenesis and inflammation). In cardiomyocytes, AMPK activation during ischemia plays a pivotal role in protecting cells from apoptosis and maintaining energy balance. Reduced adiponectin levels impair AMPK activation, exacerbating oxidative stress and myocardial injury. [30] This includes inflammation, inadequate nutrition, and muscle atrophy. [31] Another study indicates that an elevated inflammatory response to AMI is linked to hypoadiponectinemia. [32] Adiponectin exerts anti-inflammatory effects partly by inhibiting COX-2 expression via AMPK and PPAR-α pathways. In the context of myocardial infarction, reduced adiponectin levels may lead to upregulated COX-2 activity, thereby enhancing inflammatory responses and contributing to myocardial injury. [33]

5. Study Limitations and Future Directions

This study has limitations, including a modest sample size due to recruitment and resource limitations, which may limit generalizability. In addition, this study single-center design and recruitment from one hospital may limit the generalizability of the findings to broader, diverse populations, while the cross-sectional nature precluded tracking temporal changes in adipokine levels post-AMI onset, obscuring insights into their dynamic roles in the acute versus chronic phases. Future multicenter longitudinal studies should investigate temporal fluctuations in ADIPOQ levels post-AMI, correlate these changes with long-term clinical outcomes (e.g., heart failure and mortality), and incorporate comprehensive clinical covariates (e.g., BMI, age, sex, diabetes, hypertension) to enable multivariable risk stratification. The reasons for excluding pregnant and lactating women from this study are due to the physiological changes during pregnancy and lactation that affect blood dynamics and hormones, including ADIPOQ, as well as ethical considerations and ethical restrictions on the participation of pregnant women in studies that do not directly benefit them. The 8-12-hour window after the onset of myocardial infarction symptoms was chosen for sample collection to ensure Hs-TnI elevation was sufficient for diagnosis and to achieve temporal consistency among participants, reducing variability in results. This period also allows for reliable assessment of early changes in ADIPOQ and Hs-TnI before the effects of therapeutic agents or complications. It is also the most clinically practical, given that most patients arrive several hours after symptom onset.

6. Conclusion

While this study provides valuable insights, reduced AdipoQ is a significant feature in AMI patients, associated with cardiac damage and renal impairment, positioning it as a potentially valuable biomarker in the assessment of AMI.

7. Acknowledgements

We thank the management and staff of Nasiriyah Heart Hospital in Thi-Qar Province for their assistance in diagnosing patients and obtaining blood samples. We also thank the deanship of the College of Science, University of Thi-Qar, for the facilities provided to us in conducting research analyses.

8. Ethical approval

This study was conducted according to the ethical rules of medical research at the University of Thi-Qar, College of Science. Before sampling, the consent of the patient or their companion was taken. The study protocol and subject information and approval form were reviewed and approved by the main laboratory in Nasiriyah Heart Hospital-Iraq by Document No. 287/2024 dated (11/12/2024) to obtain this approval.

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