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ORIGINAL PAPER

Comparative diagnostic utility of leptin and resistin as inflammatory biomarkers in acute myocardial infarction

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ABSTRACT

Introduction and aim. Acute myocardial infarction (AMI) is a leading cause of mortality. Although traditional risk factors are known, adipokines, such as leptin (LEP) and resistin (RETN), are emerging as potential biomarkers involved in the inflammatory and metabolic processes underlying AMI. This study aimed to evaluate serum LEP and RETN levels in patients with AMI.

Material and methods. This case-control study included 60 patients diagnosed with AMI and 60 healthy controls recruited from the Nasiriyah Heart Hospital, Thi-Qar Province. Serum levels of LEP and RETN were measured using an enzyme-linked immunosorbent assay.

Results. AMI patients exhibited significantly elevated LEP (3.79±2.0 vs. 1.43±0.7 ng/mL, p<0.001) and RETN (606±325 vs. 289±160 ng/L, p<0.001) compared to controls. Both adipokines were positively correlated with high-sensitive troponin I (Hs-TnI), low-density lipoprotein (LDL-C), and triglycerides (p<0.05). ROC analysis demonstrated a high diagnostic accuracy for LEP (AUC=0.964; cutoff >2.23 ng/mL, derived from internal study data) and moderate accuracy for RETN (AUC=0.878; cutoff >305.9 ng/L). The sensitivity and specificity of the LEP were 93% and 92%, respectively.

Conclusion. LEP demonstrated high diagnostic accuracy in our cohort, and its clinical application requires validation in larger prospective studies. The association between RETN and AMI likely reflects inflammatory sequelae rather than predictive utility.

Keywords. acute myocardial infarction, leptin, resistin

Introduction

Acute myocardial infarction (AMI) remains a leading cause of global morbidity and mortality and is driven by complex interactions between metabolic dysregulation, inflammation, and cardiovascular (CV) risk factors.[1-3] While traditional biomarkers, such as troponin are central to the diagnosis of AMI, they lack specificity for the inflammatory and metabolic pathways integral to AMI pathophysiology.[4] This gap underscores the need for novel biomarkers that reflect these mechanisms to improve early detection and risk stratification. Recent projections indicate a substantial increase in CVD burden, with crude mortality expected to increase by 73.4% between 2025 and 2050, culminating in 35.6 million annual CV deaths by 2050.[5] In a published study of 19,781 patients with coronary artery disease (CAD), the prevalence of AMI was 23.3%.[5,6] Contrary to historical perceptions, emerging data highlight that the prevalence of AMI in low-resource settings is now rivaling that of developed nations, driven by urbanization and metabolic risk factors.[5]

Leptin (LEP), an adipocyte-derived hormone[7] was discovered in 1994 as a 16 kDa adipokine.[8] LEP, a 167 amino acids, is mainly synthesized by adipocytes from white adipose tissue and is transported via the circulatory system to the hypothalamus, where it modulates energy homeostasis and regulation of body weight reg-

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ulation.[9] In addition to its metabolic role, LEP exerts immunomodulatory effects by enhancing Th1 cell-mediated immune responses and attenuating Th2-associated inflammatory pathways. Furthermore, it stimulates the secretion of pro-inflammatory cytokines from mononuclear cells. The accumulation of evidence has underscored the involvement of LEP in the pathogenesis of autoimmune disorders.[10]

Resistin (RETN), a pro-inflammatory cytokine, was first identified in 2001 by a research group led by Dr. Mitchell Lazar who proposed its role in bridge the pathophysiological interplay between diabetes and obesity.[11] Originally recognized for its association with the pathological mechanisms of diabetes and its contribution to obesity-related metabolic dysregulation, emerging evidence has since expanded the functional relevance of RETN to include inflammation and immune modulation.[12] Recent studies have shown that RETN regulates the expression of pro-inflammatory mediators and directly stimulates the production of IL-6 and interleukin-8 (IL-8).[13]

Identification of effective biomarkers for AMI is crucial for improving patient outcomes and reducing mortality.[8] LEP and RETN, adipokines primarily secreted by adipose tissue, have been linked to CVDs through their roles in inflammation, insulin resistance, and lipid metabolism.[14] Elevated levels of both adipokines have been observed in coronary artery disease (CAD) and AMI, suggesting their involvement in atherosclerosis and plaque rupture.[15] While LEP and RETN have been linked to inflammatory and metabolic pathways, the exact mechanisms by which they contribute to myocardial injury and plaque rupture have not been fully elucidated.[16] However, prior studies have largely evaluated LEP and RETN in isolation, with limited comparative analyses of their diagnostic utility or adjustments for confounding factors such as body mass index (BMI).[17]

This study addresses these gaps by providing a head-to-head evaluation of LEP and RETN in patients with AMI, including ROC analysis to assess diagnostic accuracy, and adjusting for BMI to isolate its independent prognostic value. Furthermore, we explored their correlation with lipid profiles and high-sensitivity troponin I (Hs-TnI), offering novel insights into their synergistic contributions to the pathophysiology of AMI. By integrating these adipokines with established biomarkers, our findings aimed to refine risk stratification and highlight their potential as complementary diagnostic tools in the management of AMI.

Aim

This study aimed to evaluate and compare the diagnostic and predictive efficacy of LEP and RETN levels in patients with AMI and to assess their correlation with conventional biochemical markers, particularly Hs-TnI.

Material and methods

In this study a case-control study design was used. Samples were collected at Nasiriyah Heart Hospital in Thi-Qar province from individuals diagnosed with AMI, and samples were collected 8–12 h after symptoms appeared. The study population consisted of 60 patients divided into two categories: 29 male patients and 31 female patients. In addition, control group: healthy volunteers (n=60) were randomly recruited from the same community as the patients, without matching for age or sex. Sample collection was carried out between December 2024 and March 2025, and the study protocol was approved by the IRB (287/2024–December 11, 2024).

Inclusion and exclusion criteria

Patients were eligible for inclusion in the AMI group if they had a confirmed diagnosis of AMI, based on clinical presentation, electrocardiographic findings, and elevated levels of Hs-TnI equal to or exceeding 14 ng/L. Only people 18 years or older were included. Furthermore, blood samples were required to be collected between 8 to 12 hours after the onset of symptoms.

Participants were excluded if they had a history of chronic kidney disease, autoimmune disorders, or cancer. Further exclusion criteria included the use of corticosteroids or other medications known to affect adipokine levels. Pregnant or lactating women were also excluded from the study.

Sample collection

AMI patient sample collection consisted of performing a venous puncture to obtain 5 mL of peripheral blood, allowing coagulation at room temperature, and the collected sample was placed in a gel tube. The tubes were then centrifuged at 3000 rpm for 10 min to facilitate serum separation. The separated serum was then transferred to multiple Eppendorf tubes and stored at -20°C for further examination. Various factors, such as LEP, RETN, and Hs-TnI

Biomarker quantification methodologies

Human LEP (E-EL-H6017) and RETN (E-EL-H1213) concentrations were quantified using enzyme-linked immunosorbent assay kits (Biotek, Winooski, Vermont, USA) employing a sandwich methodology. Hs-TnI levels were analyzed using the AFIAS 6 fluorescence immunoassay system (South Korea), with a diagnostic threshold of 0.3 ng/L (negative: <0.3 ng/L; positive: 0.3 ng / L). The AFIAS 6 system facilitated ambient-temperature assembly and incorporated individual solid-phase receptacles, test strips, calibrators, and controls per sample. Lipid profiles were measured using a Mindray BS-230 clinical chemistry analyzer (Shenzhen Mindray Bio-Medical Electronics Co., Ltd, Shenzhen, China). The calibration protocols specified a two-point adjustment with a Min-

dray Human Multi-Calibrator and 9 g/L NaCl, traceable to the manufacturer's standards. Calibration was performed on the basis of reagent lot changes or as mandated by the internal quality control (QC) protocols. QC procedures require the analysis of two-tiered control materials (Mindray Human Assayed Control recommended) per sample batch, following calibration updates, reagent cartridge replacement, or maintenance interventions. The system automates all procedural steps to ensure standardized processing.

Statistical analysis

This study used an extensive statistical approach using Excel for data management and GraphPad Prism 9 (GraphPad Software, LLC, Boston, Massachusetts, USA). Descriptive statistics summarized categorical variables as frequencies/percentages and numerical variables as 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 sample t-tests for numerical comparisons) between AMI patients and controls. The evaluated parameters included demographics, lipid profiles, hematological measures, cardiac biomarkers (Hs-TnI), and adipokines (LEP and RETN). The diagnostic performance of adipokines in distinguishing AMI from non-AMI cases was assessed using the ROC curve analysis.

Table 1. Comparative analysis of demographic characteristics between control subjects and patients with AMI*

Characteristic	Control (n=60)	AMI (n=60)	p
Age (years)			
Mean±SD	48.3±9.8	55.4±7.1	0.1 I
Range	28–62	39–77	
Gender			
Male, n (%)	29 (48%)	36 (60%)	0.2 C
Female, n (%)	31 (52%)	24 (40%)	
BMI (kg/m^2)			
Mean±SD	25.3±3.1	30±4.2	<0.001 I
Range	20–33	21.9–33	

* n – number of cases, SD – standard deviation, C– Fisher's exact test, I – independent samples t-test

Results

This study investigated the complex interplay between adipokines, lipid profiles, and cardiac biomarkers in patients with AMI. This study compared various parameters between AMI patients and healthy controls, including demographic characteristics, biochemical markers, hematological profiles, and adipokine levels. The findings revealed significant differences in these parameters between the two groups and in the metabolic and systemic disturbances associated with AMI. This study also examined the diagnostic efficacy of adipokine markers for AMI using ROC curve analysis. This comprehensive analysis provides valuable information on the pathophysiology of AMI and the potential role of adipokines as diagnostic markers and therapeutic targets in CVDs.

Table 2 shows that patients with AMI have significantly higher levels of total cholesterol, LDL-C, triglycerides (TG) and VLDL, with lower HDL-C compared to the control group. Patients with AMI also exhibited wider ranges in TG and LDL-C, indicating greater variability. These results highlight dyslipidemia in AMI patients, consistent with known CV risk factors.

Table 2. Comparison of mean values of lipid profile among control group and patients with acute myocardial infarction*

Characteristic		Control (n=60)	AMI (n=60)	P
Cholesterol (mg/dL)				
	Mean±SD	184.1±45.3	244±65.7	<0.001 I
	Range	112–298	129–429	
HDL (mg/dL)				
	Mean±SD	47.9±9.7	36.8±12.5	<0.001 I
	Range	33.5–71.6	14.2–69.7	
LDL (mg/dL)				
	Mean±SD	103.1±47.4	158.3±60.7	<0.001 I
	Range	42.5–137	76.53–195	
TG (mg/dL)				
	Mean±SD	154.6±60.6	191.6±86.4	0.02 I
	Range	57.3–280	72–448	
VLDL (mg/dL)				
	Mean±SD	30.7±11.7	37.4±17.8	0.02 I
	Range	11.4–56	14.4–89.6	

* I – independent samples t-test

Table 3. Comparative analysis of adipokine levels in control and AMI patients*

Characteristic		Control (n=60)	AMI (n=60)	P
Hs-TnI (ng/L)				
	Mean±SD	6.0±2.3	39.2±12.8	<0.001 I
	Range	1.9–11.8	17.5–68.9	
LEP (ng/mL)				
	Mean±SD	1.43±0.7	3.79±2.0	<0.001 I
	Range	0.5–3.9	2.0–12.1	
RETN (ng/L)				
	Mean±SD	289±160	606±325	<0.001 I
	Range	83–615	177–1197	

* I – independent sample t-test

Table 3 compares serum hs-TnI levels between control subjects and patients with AMI. AMI patients had significantly higher levels (39.2±12.8 ng/L) compared to the control group (6.0±2.3 ng/L), with a highly significant

difference (p<0.001), indicating a marked increase in Hs-TnI in AMI cases. Adipokine levels (LEP and RETN) in control subjects and patients with AMI AMI patients had significantly higher levels of LEP (3.79±2.0 ng/mL) and RETN (606±325 ng/L) levels compared to controls, with broader ranges in AMI. All comparisons are statistically significant (p <0.001), as shown in Figure 1.

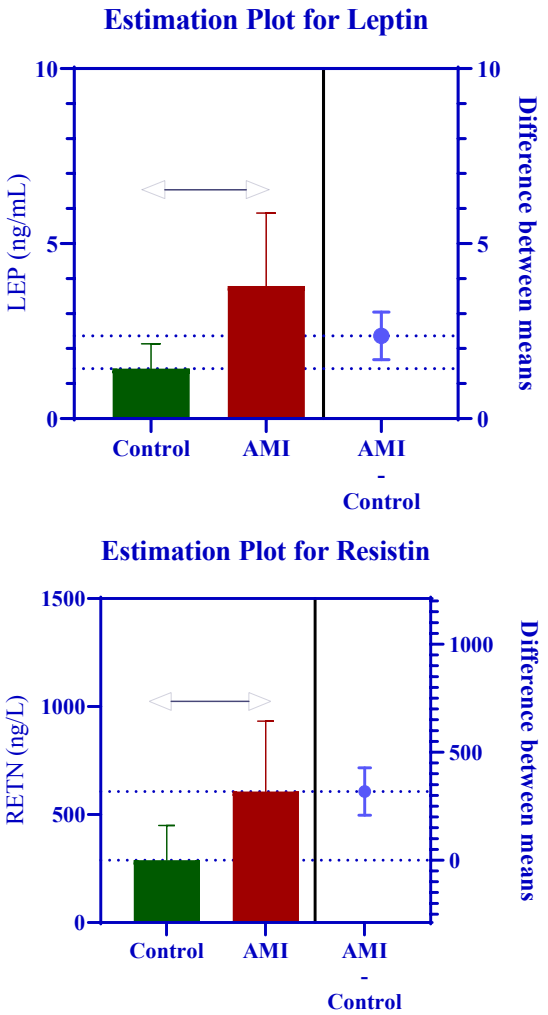

Fig. 1. Bar chart showing comparison of LEP and RETN between the control group and patients with AMI

Table 4. Diagnostic performance of LEP and RETN in AMI: a ROC curve analysis*

Variables	Cut-off value	Sens%	Spec%	PPV	NPV	Accuracy	AUC%	95% CI	p (AUC= 0.05)
LEP (ng/mL)	>2.23	93	92	93	92	85	96	0.89 to 0.99	0.001
RETN (ng/L)	> 305.9	93	70	79	89	70	87	0.78 to 0.94	0.001

* Sens – sensitivity, Spec – specificity, PPV – positive predictive value, NPV – negative predictive value, accuracy [(Sensitivity + Specificity) - 1], AUC – area under the curve, CI – confidence interval

Table 4 summarizes the receiver operating characteristic (ROC) curve analysis to evaluate the diagnostic efficacy of LEP and RETN in AMI (Fig. 2). LEP (cut-off >2.23 ng/mL) demonstrated the highest accuracy (85%) and area under the curve (AUC: 96%), with 93% sensitivity and 92% specificity, while RETN (cut-off >305.9 ng/L) had the lowest accuracy (70%) and AUC (87%). All biomarkers exhibited statistically significant discriminative power (p=0.001). In particular, LEP and RETN had high sensitivity (93%).

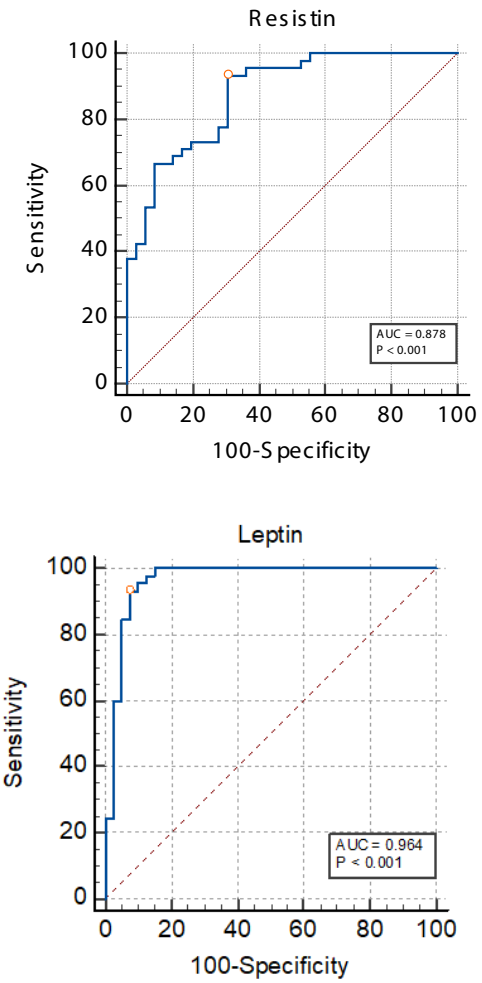

Fig. 2. ROC Curve chart of LEP, and RETN for diagnosing AMI in a sample of 60 patients and 60 controls

The Pearson correlation matrix that evaluates associations between lipid profiles and adipokines highlights patients with AMI-specific correlations of adipokines, demonstrating significant positive relationships between LEP and total cholesterol TC (r=0.428, p<0.001) and low-density lipoprotein (LDL: r=0.456, p<0.001). LEP was strongly and positively correlated with Hs-TnI (r=0.494, p<0.001). Although there was a significant positive correlation between RETN and LDL (r = 0.256, p = 0.032) and Hs-TnI (r = 0.527, p<0.001), RETN was positively correlated with LEP (r=0.525, p<0.001) and negatively correlated with HDL (r = 0.256, p = 0.026). The correlations between RETN and TC (r = 0.208,

p=0.062) and between HDL and LEP (r=-0.171, p=0.117) were not statistically significant.

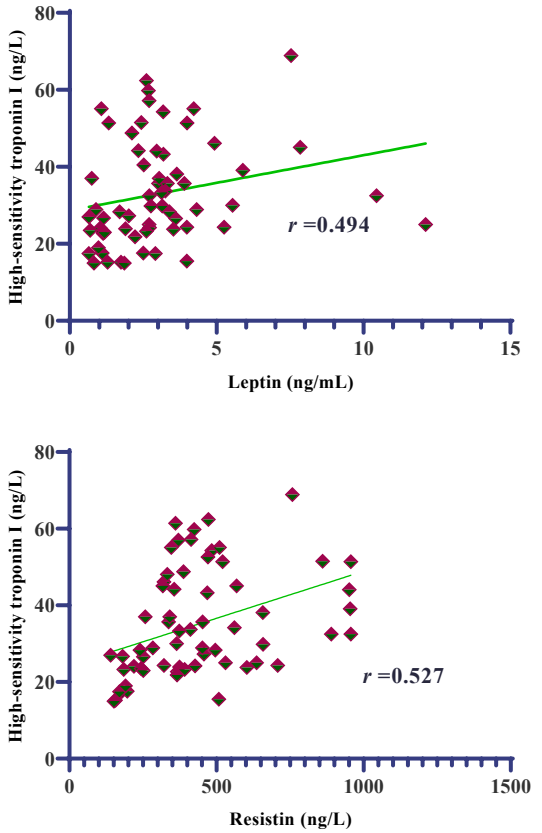

Fig. 3. Correlation chart of LEP and RETN with Hs-TnI in patients with AMI (n=60)

Table 5 presents the results of the logistic regression analysis evaluating the association of the adipokines LEP and RETN with AM. The model demonstrated (R^2=0.93) and discrimination, as evidenced by a near-perfect ROC AUC of 0.995 (95% CI: 0.942–1.000). LEP emerged as a statistically significant predictor, with a coefficient (B) of 9.676 (Wald=4.07, p<0.01) and an odds ratio of 43.7 (95% CI: 6.85–279.9), indicating a strong positive association with AMI risk. In contrast, RETN was not statistically significant in the model. ROC analysis demonstrated exceptional discriminative performance with an AUC of 0.995 (95% CI: 0.942-1,001). The classification accuracy was 94.5%, with 93.7% accuracy for negative cases and 95.1% for positive cases.

Table 5. Logistic regression analysis of the association of LEP and RETN with AMI (R^2=0.93)

Variables	B (coef)	Wald	Odds ratio	95% CI for odds ratio	p
LEP (ng/mL)	9.676	4.07	43.7	6.85 to 279.9	<0.01
RETN (ng/L)	Not significant in the model				
ROC AUC	0.995 (95% CI: 0.942 to 1.000)				
Classification accuracy	Negative cases: 93.7%, positive cases: 95.1%, overall: 94.5%				

Discussion

The study revealed significantly higher mean BMI values in patients with AMI than in controls, strengthening the established link between obesity and CV risk. This finding underscores the well-established association between obesity and increased cardiovascular risk in patients with AMI. Several studies support these findings, strengthening the well-documented relationship between higher BMI and an increased risk of CV events. A study conducted in the United States found that obesity is significantly associated with an increased risk of mortality in AMI patients.[18] Similarly, in Western Norway, obese individuals had an increased risk of AMI and CV death, which is consistent with previous results.[19] Furthermore, a study in Romania revealed that higher baseline BMI values, along with increased epicardial adipose tissue in specific coronary arteries, are strong predictors of sudden cardiac death and body fat distribution in CV health.[20] A study conducted in the United States identified what has been termed the "obesity paradox." Contrary to expectations, some studies have reported an obesity paradox in which a higher BMI correlates with a lower mortality from AMI. However, our findings align with the majority of evidence that underscores obesity as a risk enhancer.[21] Lipid abnormalities further differentiated AMI patients from controls, with significantly elevated levels of TC, LDL-C, TG, and VLDL and reduced HDL-C. These findings reinforce the well-established relationship between lipid abnormalities and the development of AMI. In Copenhagen, elevated LDL cholesterol levels were significantly associated with a markedly increased unconditional risk of MI and atherosclerotic CVD.[22] Furthermore, it was also confirmed in their study that elevated LDL levels were robustly associated with an increased risk of ASCVD, and lipid abnormalities, particularly elevated LDL-C and TG levels, contributed to a higher risk of CV events.[23] Another study by Ravnskov argued that high blood cholesterol levels are not the primary cause of CVD.[24]

The results of this study showed a significant increase in Hs-TnI levels in patients to AMI compared with controls. The pronounced elevation in Hs-TnI levels highlights its critical role in detecting subtle myocardial injuries, facilitating early diagnosis of AMI, especially in clinically ambiguous cases. The findings of this study highlight the enhanced sensitivity of modern Hs-TnI assays compared to traditional diagnostic methods.[25] This increase in the sensitivity of Hs-TnI assays has made them an invaluable tool in clinical practice, as it enhances diagnostic accuracy, even in cases where clinical symptoms may not be immediately apparent. Similarly, a study by Boeddinghaus (2020) supported the findings of this research, as it concluded that Hs-TnI offers high diagnostic accuracy in patients suspected of having MI, with clinical performance comparable to or

even exceeding that of traditional central laboratory assays.[26] Raber (2021) further substantiated these findings by reporting that higher concentrations of Hs-TnI were more strongly associated with AMI.[27]

The results of this study revealed significant differences in serum LEP adipokine levels between patients with AMI and control subjects. These findings suggest that both adipokines, which are involved in inflammation and metabolic regulation, may play an important role in the pathophysiology of AMI. The findings of this study were consistent with those of previous studies. For example, in Saint-Petersburg, Russia, elevated levels of LEP in obese patients were found to mediate the mechanisms of atherogenesis, metabolic damage, arrhythmogenesis, and myocardial ischemic damage in both experimental animal models and humans.[28] Similarly, in Pakistan, a positive correlation was found between serum LEP levels and AMI, particularly in smokers and obese patients; elevated LEP stems from the increased adipose tissue secretion as the amount of adipose tissue in the body increases, as does the level of LEP in the blood. LEP resistance also occurs in cases of obesity, and the brain may become resistant to the effects of LEP, leading to persistent feelings of hunger despite sufficient stored fat and metabolic disorders. High levels of LEP may be an indicator of disorders such as metabolic syndrome, in which LEP is produced in greater quantities by adipose tissue.[29] Another case-control study compared 40 patients with CAD and 40 healthy controls. Our study demonstrated a significant increase in LEP levels in CAD patients. Researchers noted a significant association between serum LEP level, BMI, and waist circumference, suggesting that it is an independent risk factor for cardiac disorders that are largely dependent on obesity.[30] A prospective study has provided evidence supporting the role of LEP as a risk marker, particularly in metabolic syndrome and inflammatory comorbidities. Although its diagnostic utility remains secondary to that of conventional biomarkers, its prognostic value for recurrent events and metabolic complications warrants further investigation.[31]

A study in Egypt found that serum RETN levels are elevated in patients with acute STEMI, further supporting the role of RETN in AMI.[32] Elevated LEP levels can help identify patients at higher risk of AMI and guide clinical decisions regarding treatment strategies.[33] A prospective case-control study demonstrated increased RETN levels in patients with AMI and established the prognostic significance of RETN for recurrent acute coronary syndrome (ACS). RETN levels are related to the extent of inflammation, highlighting their potential role as inflammatory markers in CV pathology.[34] A cross-sectional investigation involving 77 Saudi patients experiencing hypoxia classified participants into three cohorts: a control group, patients with normal BMI, and patients with AMI patients with heterogeneous BMI profiles. This study demonstrated significantly elevated serum RETN levels in patients to AMI compared with controls. Furthermore, under hypoxic conditions, RETN concentrations are markedly higher in obese patients with AMI than in non-obese AMI counterparts.[35] A meta-analysis of clinical investigations supports a link between elevated circulating RETN concentration and progressive severity of CAD. The study found a gradual increase in RETN levels in different CAD subtypes, with the lowest concentrations in unstable angina, intermediate levels in unstable angina pectoris, and the highest levels in acute myocardial infarction. Comparative analyses revealed a significant increase in standardized mean differences, indicating a proportional relationship between RETN elevation and CAD manifestation severity.[36] Increased serum RETN levels and a strong association with CAD severity showed a similar relationship with myocardial inflammation in RETN.[37]

In Abha, Saudi Arabia, serum LEP and RETN levels were independently associated with an increased risk of AMI. Both LEP and RETN levels are significantly elevated in patients with AMI. These results suggest that LEP and RETN could be important biomarkers to assess CV risk and may provide insights into the mechanisms underlying AMI.[38] There are several biological mechanisms by which LEP and RETN contribute to myocardial injury and inflammation. After MI, the innate immune system is activated, leading to the release of inflammatory cytokines, which improve LEP production from white adipose tissue, leading to elevated levels in circulation.[39] Infarction leads to ischemia/reperfusion, which increases the formation of reactive oxygen species (ROS) and induces further oxidative stress in cardiac cells, leading to a negative feedback loop that increases inflammation and cell damage. Furthermore, there is a direct effect on the myocardium, which binds to its receptors in cardiac cells (Ob-R), leading to hypertrophy of cardiac cells and increased proteolytic activity, contributing to impaired cardiac function.[40]

Elevated RETN levels in patients with AMI may indeed result from AMI-induced inflammation induced by AMI rather than serve as a precursor. RETN is secreted by macrophages and adipocytes in response to inflammatory signals (eg IL-6 and TNF-α) post-infarction. Furthermore, RETN impairs endothelial function (endothelial dysfunction) and increases vascular permeability, which promotes immune infiltration and increases the severity of local inflammation in the myocardium.[41] Regression models prioritize variables that explain unique variance, and LEP, with a stronger association with AMI, probably overshadowed the contribution of RETN. Additionally, the elevation of RETN may reflect systemic inflammation secondary to AMI rather than being an independent causal factor. This aligns

with studies suggesting that RETN acts as a reactive biomarker of inflammation (Tripathi et al., 2020; Zhang et al., 2017), while the mechanistic role of LEP in metabolic dysregulation may confer greater predictive utility. The ROC curve analysis conducted in this study suggests that LEP has considerable potential as a diagnostic biomarker for AMI, due to its high sensitivity and specificity. Furthermore, the combination of LEP and RETN can improve diagnostic accuracy, offering a more robust approach. Future investigations are required to assess the utility of adipokines in the stratification and management of risk of AMI patients.[42]

Study limitations and future directions

This study has limitations, including a modest sample size due to recruitment and resource limitations, which can limit its generalizability. Furthermore, the single-center design and recruitment from one hospital in this study can limit the generalizability of the findings to broader and diverse populations, while its cross-sectional nature precluded tracking temporal changes in adipokine levels after onset of AMI, obscuring insights into their dynamic roles in acute versus chronic phases. Future multicenter longitudinal studies should investigate temporal fluctuations in LEP and RETN levels after AMI, correlate these changes with long-term clinical outcomes (eg, heart failure and mortality), and incorporate comprehensive clinical covariates (e.g., BMI, age, sex, diabetes, and hypertension) to enable multivariate risk stratification.

Conclusion

This study revealed significantly elevated serum levels of LEP and RETN in AMI patients, which strongly correlated with dyslipidemia, systemic inflammation, and markers of myocardial injury such as Hs-TnI. In particular, LEP demonstrated superior diagnostic accuracy compared to RETN, closely aligned with Hs-TnI, and emerged as a promising complementary biomarker to enhance early detection of AMI and risk stratification. Although RETN elevation likely reflects inflammatory sequelae rather than predictive utility, both adipokines deserve further exploration in multicenter cohorts. Despite the diagnostic potential of LEP, its clinical translation requires validation through larger prospective studies to confirm its role alongside established cardiac biomarkers. These findings underscore the importance of integrating novel adipokine profiles into AMI diagnostics, while emphasizing the need for rigorous population-diverse research to refine their clinical applicability.

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Declarations

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Author contributions

Conceptualization: A.K.H. and B.R.A.; Methodology, A.K.H.; Software, A.K.H.; Validation, A.K.H., and B.R.A.; Formal Analysis, A.K.H.; Investigation, A.K.H.; Resources, A.K.H.; Data Curation, A.K.H.; Writing – Original Draft Preparation, A.K.H.; Writing – Review & Editing, A.K.H., and B.R.A; Visualization, A.K.H.; Supervision, B.R.A.; Project Administration, B.R.A., and A.K.H.; Funding Acquisition, A.K.H.

Conflicts of interest

The authors have nothing to disclose.

Data availability

All data supporting the study findings were obtained from the Nasiriyah Heart Hospital in the Thi-Qar province.

Ethics approval

The study protocol, subject information, and approval form were reviewed and approved by the main laboratory in Nasiriyah Heart Hospital, Iraq, in accordance with Document No. 287/2024 dated (11/12/2024) to obtain this approval.

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