EGYPTIAN PHARMACEUTICAL JOURNAL EPJ

Original Article 99

Decorin and meteorin like as differential biomarkers in acute myocardial infarction: diagnostic utility and subtype stratification of ST- elevation and non ST- elevation myocardial infarction

Mohammed Hamid Habeeb AL-Tameemi[1]*, Majid Abdulwahab Matooq[2], Abdulameer Abdulbari Abdulhameed[3], Hassan Abdulrazzaq Swaid[4]

[1,2]College of Health and Medical Techniques/Al-Basrah, Southern Technical University, Ministry of Higher Education and Scientific Research, Iraq
[3]Department of interventional cardiology, University of Basrah-college of medicine: Basrah-Iraq
[4]Al-Sadr Teaching Hospital (Cardiac Center) in Basra Governorate, Iraq – Interventional Cardiologist
*Corresponding Author: Mohammed Hamid Habeeb AL-Tameemi
E-mail: mohammed.habbeb23@fgs.stu.edu.iq
Mobile: 00964-7713166450
Emails
MHHA: mohammed.habbeb23@fgs.stu.edu.iq
MAM: m.maatook@stu.edu.iq
AAA: Abdulameer.hameed@uobasrah.edu.iq
HAS: hasansun84@gmail.com
ORCID
MHHA: https://orcid.org/0009-0001-9775-4145
MAM: https://orcid.org/0000-0002-0301-2459
AAA: https://orcid.org/0000-0003-2156-0632
HSA: https://orcid.org/0009-0004-0437-4787

Received: 18 April 2025
Revised: 21 May 2025
Accepted: 29 May 2025
Published: 3 December 2025

Egyptian Pharmaceutical Journal 2026, 25: 99-108

Background
Myokines are polypeptides or cytokines secreted by the striated muscles. This is because of both abnormal and typical physiological processes. Mounting evidence suggests that myokines are associated with the development of acute myocardial infarction (AMI).
Objectives
To investigate the diagnostic potential of decorin and meteorin-like protein (Metrnl) in distinguishing AMI from healthy controls and assess their ability to stratify AMI subtypes into ST- elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI).
Materials and methods
The 94 patients diagnosed with AMI in the emergency department and 82 healthy controls were included in this study. The levels of Metrnl and decorin were analyzed using a sandwich-based Enzyme-Linked Immunosorbent Assay technique (ELISA) and other basic biochemical parameters in the study groups were analyzed using a sandwich-based electrochemiluminescence immunoassay technique and enzymatic methods.
Results
Serum levels of Metrnl (486.98 ± 22.56 vs. 428.28 ± 7.68 pg/mL; P < 0.001) and decorin (92.68 ± 6.21 vs. 79.60 ± 2.12 ng/mL; P < 0.001) were significantly elevated in AMI patients versus controls. ROC analysis demonstrated high diagnostic accuracy: Metrnl (AUC 0.97, sensitivity 94%, specificity 97% at >439 pg/mL) and decorin (AUC 0.96, sensitivity 90%, specificity 97% at >83.2 ng/mL). STEMI patients exhibited higher Metrnl (492.5 ± 20.43 vs. 481.7 ± 23.45 pg/mL; P = 0.019) and decorin (94.25 ± 5.52 vs. 91.18 ± 6.52 ng/mL; P = 0.015) than NSTEM.
Conclusion
This study revealed elevated serum decorin and Metrnl levels in patients with AMI compared to those in healthy individuals, with distinct differences between ST-elevation and non-ST-elevation subtypes. Higher concentrations were found in STEMI cases, which were linked to the severity of coronary occlusion and inflammation.
Keywords: Acute myocardial infarction, ST-elevated myocardial infarction, non-ST-elevated myocardial infarction, decorin, Metrnl.

Egypt Pharmaceut J 25: 99–108

1687-4315

Introduction

Acute myocardial infarction (AMI) is the leading cause of morbidity and mortality worldwide. The disease affects more than three million people globally [1]. The term AMI is used when myocardial necrosis is caused by ischemic injury [2]. The presence or absence of persistent ST-segment elevation on electrocardiography (ECG) is the fundamental criterion used in the traditional AMI classification to distinguish ST-elevated myocardial infarction (STEMI) from non-ST-elevated myocardial infarction (NSTEMI) [3]. IN STEMI Patients, ST segment of ECG wave elevates in two or more contiguous leads. While in NSTEMI, the ECG waves show ST depression and/or T wave inversion, but without diagnostic ST

elevation shown [4]. STEMI and NSTEMI have the same pathophysiological basis and typically result from acute thrombosis on a culprit coronary atherosclerotic plaque, despite their different ECG presentations. STEMI frequently leads to the complete blockage of the culprit coronary artery. However, in the case of NSTEMI, the angiographic pattern may be more diverse and is typically considered more suspicious of a significant stenosis rather than a complete blockage of the vessel that causes the problem [5]. Remarkably, the clinical manifestations of the two forms of AMI were similar. Acute chest discomfort, characterized by pain, pressure, tightness, and burning, is a common symptom of myocardial ischemia. Dyspnea, epigastric pain, and left arm pain are all possible symptoms comparable to those of chest pain. Less common symptoms such as fatigue, palpitations, syncope, nausea, and vomiting may be present in some patients [6]. AMI is a medical emergency that requires immediate diagnosis and care, and should be distinguished from unstable and stable angina. In addition to clinical presentation, dynamic alterations in troponin (cTnI) levels and ECG findings have been evaluated to aid in the early diagnosis of coronary artery disease (CAD) [7]. Although cTns are the gold standard biomarkers for acute CAD caused by cardiomyocyte necrosis, non-ischemic myocardial injury can cause elevated cTnI levels, which can lead to false-positive results (eg. cardiotoxicity and myocarditis), as well as in conditions involving multifactorial injuries (e.g., pulmonary embolism and congestive heart failure) [8]. In the last few years, investigators have been looking for alternative CAD biomarkers that can be quickly identified in the circulatory system, resulting in a more precise diagnosis [9].

Meteorin-like protein (Metrnl) is a newly identified secretory protein that induces multiple intracellular signaling pathways in various cell types, including macrophages, myocytes, cardiomyocytes, and adipocytes [10]. Metrnl has 311 amino acids coded by (936 base pairs) and is found on sections of the human chromosome 17q25.3 [11]. Metrnl, which increases anti-inflammatory cytokine levels, is abundantly expressed in the heart and plays a critical role in the pathogenesis of cardiovascular diseases [12].

Decorin is a member of a growing family of structurally related proteoglycans located in the extracellular matrix and is known as a small leucine-rich proteoglycan (SLRPs). Its apparent molecular weight is approximately 130 kDa [13]. Decorin consists of 10 leucine-rich repeats, with cysteine loops on either side. It is a Class I member of the SLRP [14]. Despite being a scaffolding protein that preserves the structural integrity of the heart, decorin can be liberated in its soluble form in pathological situations, which implies that the release of soluble decorin into the bloodstream is a warning indication [15]. In this study, we aimed to investigate the diagnostic performance of decorin and Metrnl in distinguishing AMI from healthy controls, assess their differential expression between STEMI and NSTEMI subtypes, and evaluate their potential as prognostic tools for infarct severity.

Materials and methods

This case control study included 94 patients (65 males and 29 female) and 82 healthy controls (40 males and 38 female). Patients were divided into two groups based on troponin levels and ECG findings: STEMI and NSTEMI. Patients were selected from individuals who received medical care at the Al-Sader Teaching Hospital and Basra Specialized Hospital for Cardiology and Cardiac Surgery in the Basra governorate between september2024 and january2025. The control group was randomly selected from among the employees and additional healthy volunteers. The current study excluded patients (with cancer, liver diseases, renal diseases, post AMI, heart failure, thyroid diseases, and pregnant women) to avoid confounding effects on biomarker levels, to ensure accurate and generalizable results for the target population. Blood samples were collected from each participant, separated into two parts, and centrifuged to obtain serum. The first sample was used to determine total cholesterol, Low density lipoprotein, high density lipoprotein, triglycerides, high-sensitivity troponin I, and other tests used to apply the exclusion criteria, such as (urea, creatinine, thyroid-stimulating hormone, and liver function tests). The second sample was stored at − 20°C to determine the decorin and Metrnl biomarkers. All tests were performed using a fully automated biochemistry analyzer (Cobas integra 400 system, Cobas E411) and ELISA technique (for Decorin: manufactured by Elabscience, Catalog No: E-EL-H2248) (for Metrnl: manufactured by FineTest, catalog No: EHK2364). The kits used sandwich enzyme-linked immune-sorbent assay technology, with anti-Metrnl and anti-decorin antibodies pre-coated onto 96-well plates. Biotin conjugated antibodies were used as detection antibodies. Standards and pilot samples were added, and unbound conjugates were removed. HRP-Streptavidin was added, and TMB substrates were added to visualize the HRP enzymatic reaction. The blue color product turned yellow after stopping. A total of 94 patients were selected according to the established exclusion criteria, specified timeframe, and conditions prevailing at the sample collection center.

Statistical analysis

Data were entered into an SPSS version 24 spreadsheet for tabulation and analysis. The Kolmogorov-Smirnov test was used to examine the normality of the scale or continuous variables. Descriptive statistics are reported as mean ± standard deviation (SD), frequency, and percentages. Comparisons between cases (myocardial infarction) and controls were performed using the independent sample t-test for normally distributed data and Mann-Whitney U test for non-parametric data. Chi-square (χ^2) tests were used to compare the categorical variables. The association between biomarkers and lipid profiles was assessed using Pearson's correlation. To assess the predictive ability of Metrnl and decorin for myocardial infarction, Receiver Operating Characteristic (ROC) curve analysis was performed, and the area under the curve (AUC) was reported. The sensitivity, specificity, and optimal cut-off values were determined using Youden's index. For all tests, a P-value < 0.05 was deemed statistically significant.

Results

The current study involved 94 individuals known to have myocardial infarction with ST elevation (STEMI) or non-ST-segment elevation (NSTEMI). In addition, 82 healthy individuals were included in the control group. The majority of the patients with AMI were 65 (69.1%) males more than females 29 (30.9%) women. The mean age of cases was 57.96 ± 9.63 years and 55.89 ± 10.76 years for healthy individuals, and there were no significant differences (p>0.05) between the case group and the healthy individual group. Cholesterol and HDL levels were identical in both groups. However, the TG and LDL levels were significantly higher in these cases (p <0.05) (Table 1).

Table 2 shows the biomarkers studied in the case and control groups. AMI cases exhibited markedly elevated serum levels of Metrnl (486.98 ± 22.56 vs. 428.28 ± 7.68 pg/ml, P < 0.001), decorin (92.68 ± 6.21 vs. 79.6 ± 2.12 ng/ml, P < 0.001), and High-sensitivity troponin I (Hs-TnI) (317.46 ± 510.75 vs. 6.0 ± 2.3 ng/L, P < 0.001) compared to controls. All the biomarkers showed statistically significant differences.

Table 3 stratifies AMI cases into STEMI (n = 46) and NSTEMI (n = 48) subgroups. Metrnl levels were significantly higher in STEMI (492.5 ± 20.43 vs. 481.7 ± 23.45 pg/ml, p = 0.019), Figure 1 (A) as were decorin levels (94.25 ± 5.52 vs. 91.18 ± 6.52 ng/ml, p = 0.015) Figure 1 (B). Troponin levels did not differ significantly between subgroups (p = 0.264).

Correlation analyses between lipid parameters (cholesterol, TG, HDL, LDL) and biomarkers (Hs-TnI, decorin, Metrnl) in patients with AMI. Significant positive correlations were observed between cholesterol and TG (r = 0.363, p < 0.001) and LDL (r = 0.850, p < 0.001) levels. HDL levels exhibited a weak positive correlation with troponin levels (r = 0.316, p = 0.002). Metrnl showed a moderate positive correlation with decorin levels (r = 0.426, p < 0.001; Figure 2). No significant correlations were found between TG, LDL, decorin, and the other biomarkers (p > 0.05). Notably, the strongest association was observed between cholesterol and LDL levels, indicating a robust linear relationship in the AMI cohort.

Receiver Operating Characteristic (ROC) curves were plotted for Metrnl and decorin to examine their predictive ability. Diagnostic performance of Metrnl and decorin using ROC curve analysis. Metrnl (cutoff >439 pg/ml) achieved 94% sensitivity, 97% specificity, and 97% AUC (p < 0.001). Decorin (cut-off >83.2 ng/ml) showed 90% sensitivity, 97% specificity, and 96% AUC (p < 0.001). Both biomarkers demonstrated high accuracy (91% and 88%, respectively), supporting their utility as non-invasive diagnostic tools for AMI (Figure 3 [A,B]). In the subgroup analysis distinguishing STEMI from NSTEMI, Metrnl (cutoff >497 pg/mL) yielded an AUC of 0.71 (p < 0.001), with 67% sensitivity and 85% specificity. Decorin (cutoff >92 ng/mL) showed an AUC of 0.70 (p < 0.001), with 71% sensitivity and 63% specificity (Figure 4 [A,B]). While these results reflect lower discriminatory performance compared to the primary cohort, both biomarkers retained clinical relevance in differentiating STEMI from NSTEMI.

Table 1 Demographic and clinical characteristics of acute myocardial infarction cases and controls.

Characteristic			Cases *n* = 94	Control *n* = 82	P-value
Age (years)		Mean ±SD	57.96 ± 9.63	55.89 ± 10.76	0.186 [a NS]
Sex	Male	*n* (%)	65 (69.1%)	44 (53.7%)	0.05 [b NS]
	Female	*n* (%)	29 (30.9%)	38 (46.3%)	
Age	35-45 years	*n* (%)	13(13.8%)	13(15.9%)	0.845[b NS]
	46-55 years	*n* (%)	24(25.5%)	25(30.5%)	
	56-65 years	*n* (%)	34 (36.2%)	25(30.5%)	
	66-77 years	*n* (%)	23(24.5%)	19(23.2%)	
Lipid profile	Cholesterol (mg/dl)	Mean ±SD	184.88 ± 56.18	173.61 ± 23.46	0.09 [a NS]
	TG (mg/dl)	Mean ±SD	169.83 ± 74.86	106.62 ± 22.93	<0.001[a] **
	HDL (mg/dl)	Mean ±SD	35.40 ± 46.76	44.22 ± 6.28	0.09[a NS]
	LDL (mg/dl)	Mean ±SD	118.46 ± 48.10	86.84 ± 8.43	<0.001[a] **

n: number of cases; SD: standard deviation; * P <0.05: statistical significance. a: Independent sample t-test. b: Chi-square. NS: Not Significant.

Table 2 Serum biomarker levels in acute myocardial infarction cases and controls.

Biomarker		Cases *n* = 94	Control *n* = 82	P-value
Metrnl (Pg/ml)	Mean ±SD	486.98 ± 22.56	428.28 ±7.68	<0.001 I***
Decorin (ng/ml)	Mean ±SD	92.68 ± 6.21	79.6 ± 2.12	<0.001 I***
Hs-TnI (ng/L)	Mean ±SD	317.46 ± 510.75	6.0 ± 2.3	<0.001 I***

n: number of cases; SD: standard deviation; I: independent samples t-test; *** P <0.001: statistical significance.

Table 3 Biomarker profiles in ST-elevated myocardial infarction and non-ST-elevated myocardial infarction subgroups.

Biomarker		STEMI *n* = 46	NSTEMI *n* = 48	P value
Metrnl **(pg/ml)**	Mean ±SD	492.5 ± 20.43	481.7 ± 23.45	0.019 I*
Decorin **(ng/ml)**	Mean ±SD	94.25 ± 5.52	91.18 ± 6.52	0.015 I*
Hs-TnI **(ng/L)**	Mean ±SD	377.85 ± 532.68	259.59 ± 487.35	0.264 I[NS]

n, number of cases; SD, standard deviation; I, independent samples t-test; NS: Not Significant; * P <0.05, statistical significance.

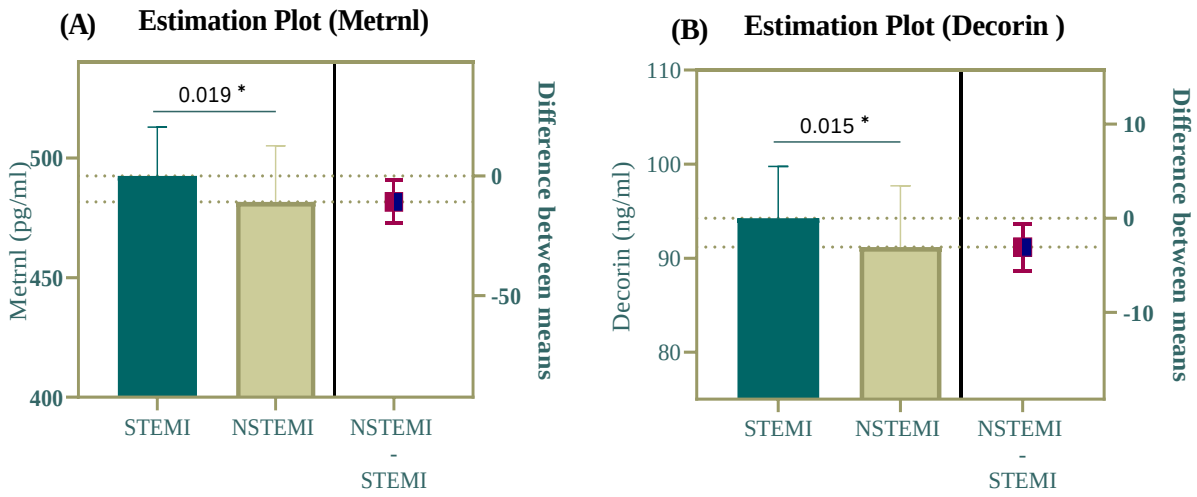

Fig. 1 The bar charts for metrnl (A) and decorin (B).

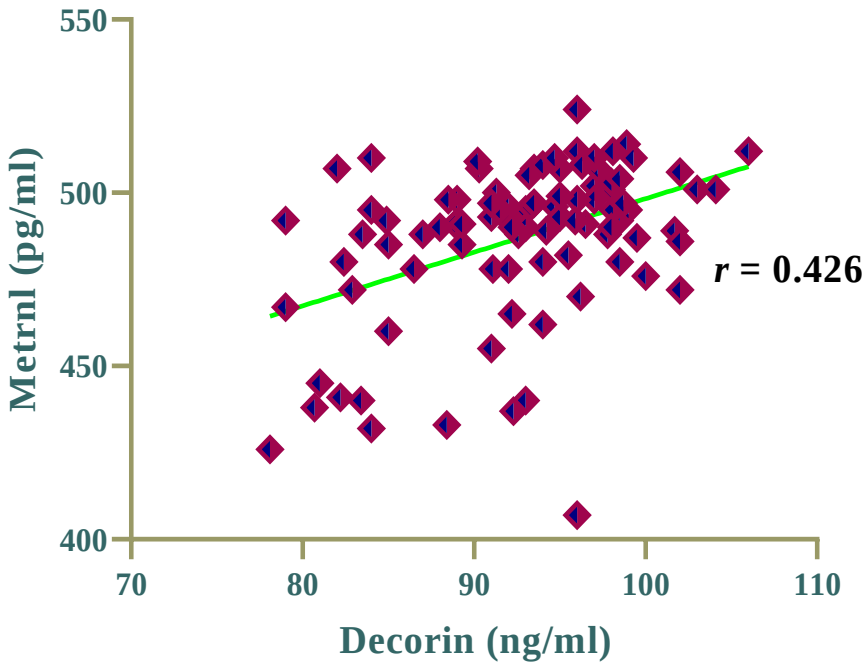

Fig. 2 Chart for positive correlation of metrnl with decorin.

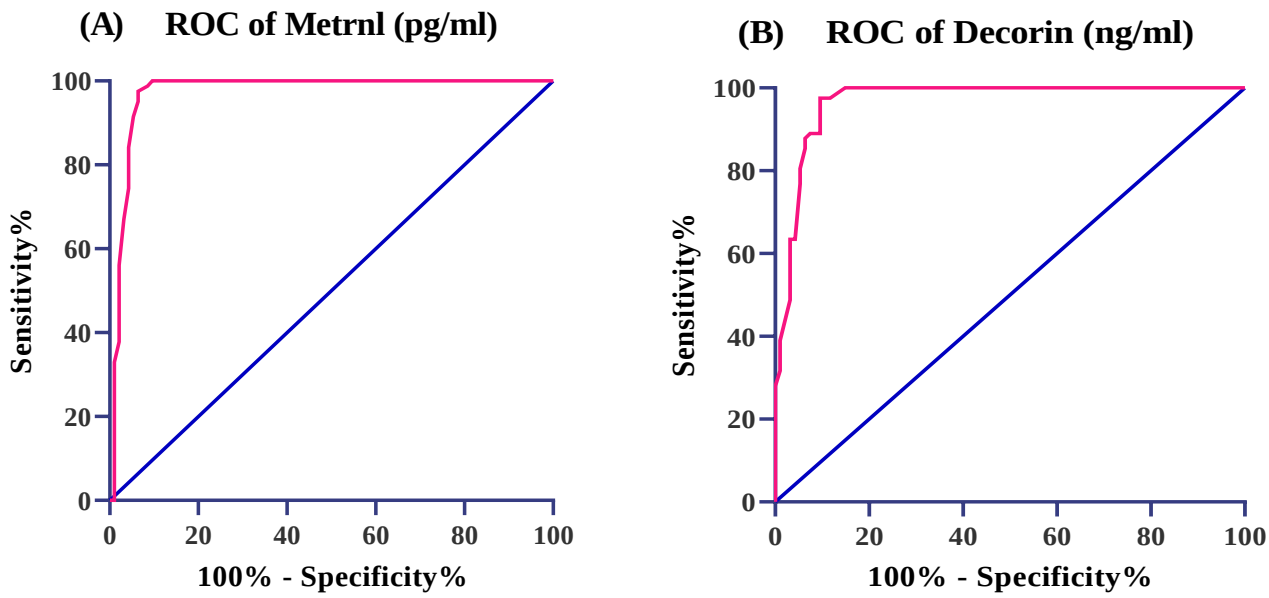

Fig. 3 ROC curve of metrnl (A) and decorin (B).

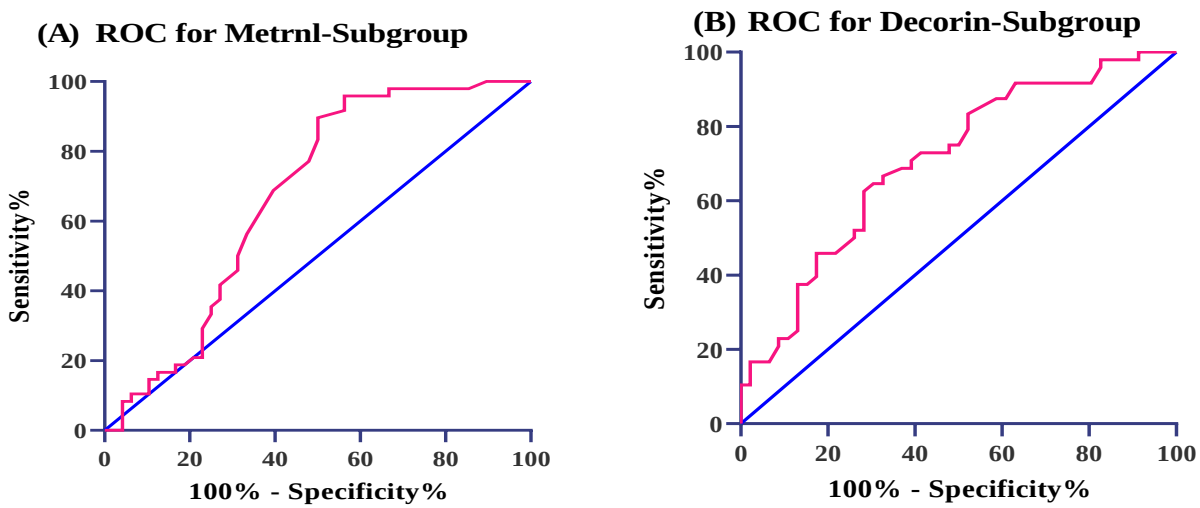

Fig. 4 ROC curve of metrnl (A) and decorin (B) in subgroups

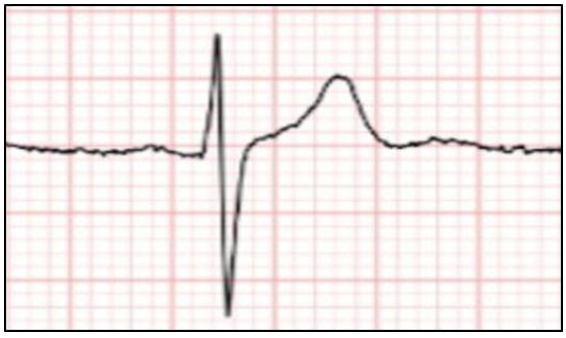

Fig. 5 Normal ECG wave.

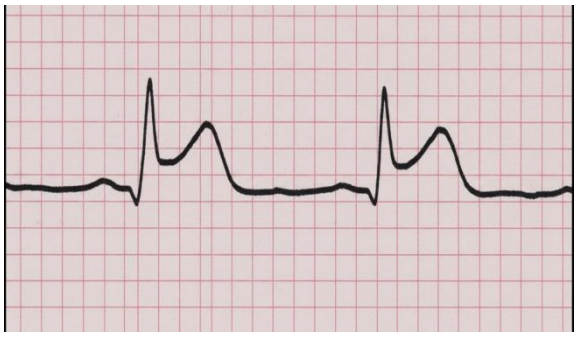

Fig. 6 ST-segment elevation.

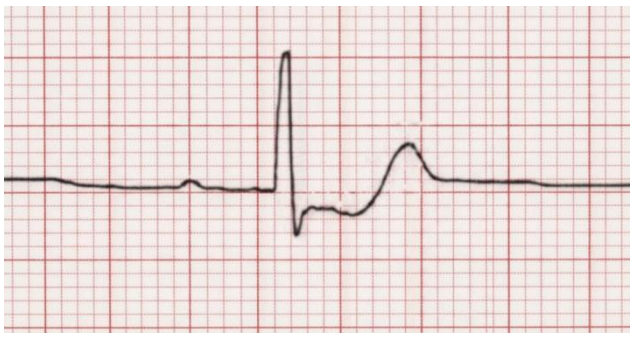

Fig. 7 ST-segment depression (NSTEMI)

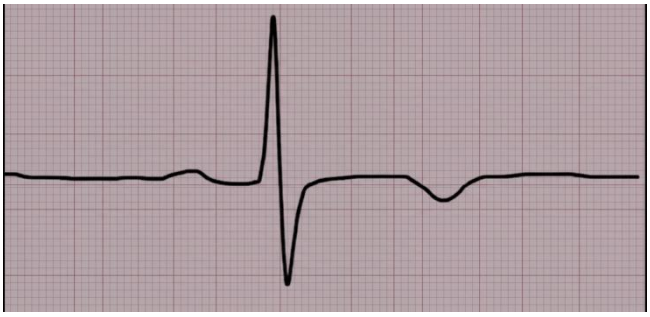

Fig. 8 T-wave inversion (NSTEMI).

Discussion

Acute myocardial infarction has been reported to happen ten years earlier in developing nations than in developed nations, where it typically strikes men in their mid-sixties and women in their early seventies [16, 17]. The current investigation determined the mean age of patients with AMI in southern Iraq to be lower (57.96 ± 9.63 years), which is consistent with previous regional studies in northern Iraq, Saudi Arabia, and Iran, which revealed a lower mean age of AMI [18, 19]. This age discrepancy compared to developed nations may reflect differences in social welfare systems, cultural practices, lifestyle patterns, and access to preventive healthcare between high- and low-resource settings [20] [19].

The study findings indicate that males (69.1%) are significantly more likely than females (30.9%) to experience AMI, which is consistent with prior studies that attributed the higher incidence in males to a combination of biological and lifestyle factors [21-23]. Estrogen is thought to have a protective effect against cardiovascular events in premenopausal women, thereby reducing the risk of AMI. Furthermore, men tend to exhibit a higher prevalence of cardiovascular risk factors, including smoking, hypertension, and dyslipidemia, which contribute to their increased susceptibility to AMI [24].

Low-density lipoprotein and triglyceride levels were observed to increase more significantly in patients with AMI than in controls, consistent with previous studies highlighting elevated LDL and TG levels as key contributors to AMI [25, 26]. Atherosclerosis, the primary pathological process underlying AMI, is strongly associated with circulating LDL, one of the earliest recognized factors in its progression [27, 28]. Genetic and

epidemiological research has identified elevated TG levels as a major residual cause of atherosclerotic cardiovascular disease (ASCVD) [29, 30]. These findings underscore the complex interplay between lipid metabolism and cardiovascular risk in AMI patients.

Myokines are polypeptides or cytokines secreted from striated muscles during exercise [31]. This is because of both abnormal and typical physiological processes. Increasing evidence suggests that myokines are associated with the development of cardiovascular disease [32]. A previous study reported higher levels of Metrnl in patients with AMI. [33] In Spain, increased blood Metrnl levels have been observed in STEMI patients. In Parallel with these observations, the current study revealed a significantly higher level of Metrnl in the AMI group than in the healthy control group. Although the exact mechanism by which higher Metrnl levels occur in AMI is unknown, Metrnl secreted from monocytes and macrophages is a high-affinity ligand for the stem cell factor receptor KIT, a receptor tyrosine kinase, and a stimulator of post-infarction angiogenesis, which is involved in tissue repair after MI [34, 35]. In a mouse model of MI, Metrnl plays an important role in the attenuation of inflammation to reduce infarct size and improve cardiac function by decreasing pro-inflammatory markers (e.g., IL-1β and TNF-α) and increasing anti-inflammatory cytokines such as IL-10 [36]. Elevated Metrnl levels in patients with AMI may reflect a compensatory anti-inflammatory response to myocardial injury, potentially mediated through its role in post-infarction angiogenesis and modulation of cytokine dynamics, and could position Metrnl as both a prognostic biomarker and a potential therapeutic target in AMI management.

In this study, we found that decorin levels were remarkably higher in the serum of patients after a heart attack than in that of the healthy group, and they were shown to be reliable indicators for diagnosing AMI. The results of this study are consistent with a Chinese study that indicated that individuals with acute coronary syndrome had higher levels of decorin than those in the control group [37]. While calcification of vessels is a common feature of advanced atherosclerosis [38], it stimulates calcification in arterial smooth muscle cells (SMCs) and colocalizes with mineral deposits in human atherosclerotic plaques, which are often associated with unstable plaques and increased cardiovascular risk [39, 40]. Moreover, decorin plays a crucial role in inflammation and tissue remodeling by modulating various signaling pathways and interacting with extracellular matrix components. It regulates inflammation through mechanisms involving TLR2/4 signaling and PDCD4, which influences the expression of pro-inflammatory cytokines, such as TNF-α and IL-12p70, while suppressing anti-inflammatory mediators, such as IL-10 [41, 42]. Thus, according to these results, decorin can serve as a potential diagnostic biomarker for AMI and reflects the role of decorin in the mechanisms underlying inflammation, vascular calcification, and tissue remodeling in cardiovascular disease. Recognizing these associations could contribute to advancements in the diagnosis and treatment of patients with myocardial infarction.

Furthermore, individuals with STEMI exhibited higher levels of decorin and Metrnl compared to those with NSTEMI. This finding for decorin is consistent with previous studies that have also reported elevated levels in STEMI patients[37]. In contrast, [33] reported that Metrnl was increased in the STEMI group compared to the NSTEMI group. Typically, STEMI is caused by the total occlusion of the coronary artery, leading to transmural ischemia and a larger infarct size. Furthermore, the acute inflammatory response is more obvious in patients with STEMI because of greater myocardial injury [43]. Extensive necrosis of STEMI and microvascular obstruction leads to more severe structural remodeling compared with NSTEMI [44]. The results of this study can help refine the diagnostic and prognostic approaches for myocardial infarction and provide valuable clinical insights.

ROC curves were plotted for Metrnl and decorin to examine their predictive ability. Diagnostic performance of Metrnl and decorin using ROC curve analysis. Metrnl (cutoff >439 pg/ml) achieved 94% sensitivity, 97% specificity, and 97% AUC ($p < 0.001$). Decorin (cut-off >83.2 ng/ml) showed 90% sensitivity, 97% specificity, and 96% AUC ($p < 0.001$). Both biomarkers demonstrated high accuracy (91% and 88%, respectively), supporting their utility as non-invasive diagnostic tools for AMI.

The biomarkers demonstrated exceptional discriminatory capacity in the ROC curve analysis), with an AUC of 0.96 for decorin and 0.97 for meteorin-like, indicating near-perfect diagnostic performance [45]. At the optimal cut-off of (in decorin > 83.2 and > 439 in Metrnl), the biomarkers achieved a sensitivity of (>96.0%), which is critical for minimizing false negatives in disease detection. Accuracy reflects balanced performance across true positives and negatives, although this metric is prevalence-dependent and less informative than the AUC in isolation. These metrics collectively position these biomarkers as promising candidates for clinical validation, particularly in triage protocols that require high negative predictive values [46].

Study limitations and future directions

This study has several limitations. Two-center recruitment from the Basra Governorate limits geographic diversity, raising questions about its applicability to broader populations. Additionally, biomarker levels were measured at a single time point, omitting insights into temporal dynamics after post-AMI. Further validation through multicenter studies is essential to confirm their reliability and explore their synergistic use with established biomarkers, particularly in regions with limited healthcare resources where the cardiovascular disease burden remains high. This study advances the pursuit of novel biomarkers to refine diagnostic strategies and improve patient outcomes in acute coronary syndrome.

Conclusion

This study established that serum decorin and Metrnl levels are significantly elevated in patients with AMI compared with healthy individuals, with distinct differences observed between ST-elevation and non-ST-elevation myocardial infarction subtypes. Both biomarkers demonstrated pronounced increases in patients with AMI, with higher concentrations detected in STEMI cases, linking the severity of coronary occlusion and inflammatory pathophysiology. These findings underscore the role of decorin and Metrnl in AMI pathogenesis and their capacity to differentiate between subtypes, which may enhance clinical decision making. However, further studies are warranted to validate its clinical application and to explore its integration with other biomarkers for enhanced diagnostic precision.

Acknowledgements

The authors would like to thank the staff of AL-Sader Teaching Hospital and Basra-specialized Hospital for Cardiology and Cardiac Surgery in the Health Department of Basrah for contributing to the success of this work, which is part of the requirement for obtaining a master's degree.

Competing interests

The author declare that they have no competing interests

Author contribution

The authors contributed equally to the conceptualization of the research, collected data, and participated in data analysis and write-up, editing, and review.

Ethical approval

An agreement to conduct this study was signed before the start of the sample collection process. The Southern Technical University and Basra Health Department collaborated to conduct this study. The study was approved by the research committee (number 970/2024) on September 3, 2024, in accordance with the Helsinki Declaration. The participants provided written consent to participate in the study.

References

1. Salari N, Morddarvanjoghi F, Abdolmaleki A, Rasoulpoor S, Khaleghi AA, Hezarkhani LA, et al. The global prevalence of myocardial infarction: a systematic review and meta-analysis. BMC Cardiovascular Disorders. 2023;23(1):206.
2. Bhatt DL, Lopes RD, Harrington RA. Diagnosis and Treatment of Acute Coronary Syndromes: A Review. JAMA. 2022;327(7):662-75.
3. Collet J-P, Thiele H, Barbato E, Barthélémy O, Bauersachs J, Bhatt D, et al. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation.
4. Hu T. Electrocardiographic Interpretation and Abnormalities: A Comprehensive Review of Clinical, Technical, and Pathophysiological Insights. 2025.
5. Khan S, Chaudhry R, Mahmood A, Masmoum M, Altaf SA, Mirza MSS. Myocardial Infarction: Causes, Symptoms, and Emergency Response. Journal of Advances in Medicine and Medical Research. 2024;36(8):72-83.
6. Raat W, Nees L, Vaes B. Diagnostic accuracy of signs and symptoms in acute coronary syndrome and acute myocardial infarction: A diagnostic meta-analysis. Scandinavian Journal of Primary Health Care. 2025;43(1):111-9.
7. Du X, Su X, Zhang W, Yi S, Zhang G, Jiang S, et al. Progress, opportunities, and challenges of troponin analysis in the early diagnosis of cardiovascular diseases. Analytical Chemistry. 2021;94(1):442-63.
8. Hartikainen T, Westermann D. Advances in rapid diagnostic tests for myocardial infarction patients. Expert Review of Molecular Diagnostics. 2023;23(5):391-403.
9. Abubakar M, Irfan U, Abdelkhalek A, Javed I, Khokhar MI, Shakil F, et al. Comprehensive quality analysis of conventional and novel biomarkers in diagnosing and predicting prognosis of coronary artery disease, acute coronary syndrome, and heart failure, a comprehensive literature review. Journal of Cardiovascular Translational Research. 2024;17(6):1258-85.
10. Altintas N, Fazlioglu N, Guzel S, Yilmaz A, Aydın C. Is meteorin-like (Metrnl) a novel biomarker to distinguish patients with obstructive sleep apnea (OSA) and patients with OSA at vascular risk. Sleep and Breathing. 2023;27(5):1865-74.
11. AKDAG T. Novel Candidate Biomarkers for Diabetes Mellitus. NEW FRONTIERS IN HEALTH SCIENCES.161.
12. Li Z, Gao Z, Sun T, Zhang S, Yang S, Zheng M, et al. Meteorin-like/Metrnl, a novel secreted protein implicated in inflammation, immunology, and metabolism: A comprehensive review of preclinical and clinical studies. Frontiers in immunology. 2023;14:1098570.
13. Carvalho MS, Cabral JM, da Silva CL, Vashishth D. Bone matrix non-collagenous proteins in tissue engineering: Creating new bone by mimicking the extracellular matrix. Polymers. 2021;13(7):1095.
14. Mochida Y, Miguez P, Yamauchi M. Small Leucine-Rich Proteoglycans (SLRPs) and Biomineralization. Extracellular Matrix Biomineralization of Dental Tissue Structures. 2021:125-55.
15. Dong Y, Zhong J, Dong L. The role of decorin in autoimmune and inflammatory diseases. Journal of immunology research. 2022;2022(1):1283383.
16. Samad Z, Noorali AA, Farhad A, Awan S, Qureshi NQ, Mawani M, et al. Leveraging Clinical Digitized Data to

Understand Temporal Characteristics and Outcomes of Acute Myocardial Infarctions at a Tertiary Care Medical Centre in Pakistan from 1988–2018 – Methods and Results. Global Heart. 2022.
17. Oberoi M, Ainani N, Abbott JD, Mamas MA, Velagapudi P. Age Considerations in the Invasive Management of Acute Coronary Syndromes. US Cardiology Review. 2022;16:e14.
18. Kinsara AJ, Alsaleh A, Taher ZA, Alshamiri M, Elshaer F, Sr. The Primary Management Strategies for ST-Elevation Myocardial Infarction Patients in Saudi Arabia: A Sub-Study of the Saudi Acute Myocardial Infarction Registry. Cureus. 2020;12(11):e11783.
19. Sharif Nia H, Sivarajan-Froelicher E, Haghdoost AA, Moosazadeh M, Huak-Chan Y, Farsavian AA, et al. The estimate of average age at the onset of acute myocardial infarction in Iran: A systematic review and meta-analysis study. ARYA atherosclerosis. 2018;14(5):225-32.
20. Amen SO, Baban ST, Yousif SH, Hawez AH, Baban ZT, Jalal DMF. Prevalence of the most frequent risk factors in Iraqi patients with acute myocardial infarction. Medical Journal of Babylon. 2020;17(1):6-18.
21. Woodward M. Cardiovascular disease and the female disadvantage. International journal of environmental research and public health. 2019;16(7):1165.
22. Taqueti VR. Sex differences in the coronary system. Sex-Specific Analysis of Cardiovascular Function. 2018:257-78.
23. Earle NJ, Doughty RN, Devlin G, White H, Riddell C, Choi Y, et al. Sex differences in outcomes after acute coronary syndrome vary with age: a New Zealand national study. European Heart Journal Acute Cardiovascular Care. 2023;13(3):284-92.
24. Kim H-L. Differences in risk factors for coronary atherosclerosis according to sex. Journal of Lipid and Atherosclerosis. 2024;13(2):97.
25. Balling M, Afzal S, Davey Smith G, Varbo A, Langsted A, Kamstrup PR, et al. Elevated LDL triglycerides and atherosclerotic risk. Journal of the American College of Cardiology. 2023;81(2):136-52.
26. Farnier M, Zeller M, Masson D, Cottin Y. Triglycerides and risk of atherosclerotic cardiovascular disease: an update. Archives of cardiovascular diseases. 2021;114(2):132-9.
27. Zhang B-H, Yin F, Qiao Y-N, Guo S-D. Triglyceride and triglyceride-rich lipoproteins in atherosclerosis. Frontiers in Molecular Biosciences. 2022;9:909151.
28. Al-Rubaye AAH. The correlation between type 2 diabetes mellitus and hypertension in Iraqi patients. J Med Chem Sci. 2023;6:778-88.
29. Toth PP, Fazio S, Wong ND, Hull M, Nichols GA. Risk of cardiovascular events in patients with hypertriglyceridaemia: A review of real- world evidence. Diabetes, Obesity and Metabolism. 2020;22(3):279-89.
30. Si S, Hou L, Chen X, Li W, Liu X, Liu C, et al. Exploring the causal roles of circulating remnant lipid profile on cardiovascular and cerebrovascular diseases: Mendelian randomization study. Journal of epidemiology. 2022;32(5):205-14.
31. Al-Rubaye AAH, Walaa EJ, Ahmed AM. Myonectin, Irisin, Apelin-13 and Elabela hormones levels as biomarkers for type 2 diabetes mellitus: A systematic review. Ukr Biochem J. 2024;96(4):17-24.
32. Mancinelli R, Checcaglini F, Coscia F, Gigliotti P, Fulle S, Fanò-Illic G. Biological aspects of selected myokines in skeletal muscle: focus on aging. International journal of molecular sciences. 2021;22(16):8520.
33. Ferrer-Curriu G, Rueda F, Revuelta-López E, García-García C, Codina P, Gálvez-Montón C, et al. Meteorin-like protein is associated with a higher risk profile and predicts a worse outcome in patients with STEMI. Revista Española de Cardiología (English Edition). 2023;76(11):891-900.
34. Reboll MR, Klede S, Taft MH, Cai C-L, Field LJ, Lavine KJ, et al. Meteorin-like promotes heart repair through endothelial KIT receptor tyrosine kinase. Science. 2022;376(6599):1343-7.
35. Huynh K. Meteorin-like protein repairs the ischaemic heart via receptor KIT in endothelial cells. Nature Reviews Cardiology. 2022;19(9):575-.
36. Ferrer-Curriu G, Soler-Botija C, Monguio-Tortajada M, Poblador F, Navarro Gascon A, Blasco Roset A, et al. Protective role of Metrnl on macrophage recruitment during the acute inflammatory phase in a mouse model of myocardial infarction. European Heart Journal. 2024;45(Supplement_1).
37. Zhuang L, Ge Y, Zong X, Yang Q, Zhang R, Fan Q, et al. High Proteoglycan Decorin Levels Are Associated With Acute Coronary Syndrome and Provoke an Imbalanced Inflammatory Response. Frontiers in physiology. 2021;12:746377.
38. Li M, Wang Z-W, Fang L-J, Cheng S-Q, Wang X, Liu N-F. Programmed cell death in atherosclerosis and vascular calcification. Cell death & disease. 2022;13(5):467.
39. Saba L, Nardi V, Cau R, Gupta A, Kamel H, Suri JS, et al. Carotid artery plaque calcifications: lessons from histopathology to diagnostic imaging. Stroke. 2022.
40. Fischer JW, Steitz SA, Johnson PY, Burke A, Kolodgie F, Virmani R, et al. Decorin Promotes Aortic Smooth Muscle Cell Calcification and Colocalizes to Calcified Regions in Human Atherosclerotic Lesions. Arteriosclerosis, Thrombosis, and Vascular Biology. 2004;24(12):2391-6.
41. Vu TT, Jubert M, Thanh LL, Tuyet NAT, Kyu KH, and Han J. The role of decorin in cardiovascular diseases: more than just a decoration. Free Radical Research. 2018;52(11-12):1210-9.
42. Dong Y, Zhong J, Dong L. The Role of Decorin in Autoimmune and Inflammatory Diseases. Journal of immunology research. 2022;2022:1283383.
43. Matter MA, Paneni F, Libby P, Frantz S, Stähli BE, Templin C, et al. Inflammation in acute myocardial infarction: the good, the bad and the ugly. European heart journal. 2024;45(2):89-103.
44. Kingma J. Myocardial infarction: An overview of STEMI and NSTEMI physiopathology and treatment. World Journal of Cardiovascular Diseases. 2018;8(11):498.
45. Roumeliotis S, Schurgers J, Tsalikakis DG, D'Arrigo G, Gori M, Pitino A, et al. ROC curve analysis: a useful statistic multi-tool in the research of nephrology. International urology and nephrology. 2024;56(8):2651-8.
46. Çorbacıoğlu ŞK, Aksel G. Receiver operating characteristic curve analysis in diagnostic accuracy studies: A guide to interpreting the area under the curve value. Turkish journal of emergency medicine. 2023;23(4):195-8.