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S100A1 downregulation as a calcium-energetic marker in acute myocardial infarction: Correlation with angiogenic and inflammatory pathways

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Abstract

Objective

Acute myocardial infarction (AMI) is a condition of synchronized biological reprogramming characterized by calcium dysregulation, inflammatory activation, and compromised vascular repair. S100A1 is a Ca^{2+}-binding protein abundant in cardiomyocytes that is crucial for excitation-contraction coupling and mitochondrial energetics, thus potentially indicating cardiac functional stability beyond necrotic damage. The aim of this study was to assess S100A1 protein and its mRNA expression, as well as their correlation with inflammatory and vascular mediators in patients with AMI.

Materials and methods

This case-control study (n = 176) assessed circulating S100A1 protein and gene expression in conjunction with TNF-α and VEGF-A levels. Venous blood samples (5 mL) were obtained from patients with acute myocardial infarction (AMI) within 12 hours of symptoms onset and from control participants at the time of enrolment. Quantitative real-time polymerase chain reaction (qPCR) was conducted with SYBR Green dye and the Stratagene Mx3005P platform. Statistical analyses were conducted utilizing SPSS (IBM v25.0) and GraphPad Prism (v9.0).

Results

Patients with acute myocardial infarction showed a marked decrease in S100A1 protein levels, both at the protein level and at the transcriptional level, compared to healthy patients. This decrease was concurrent with a decrease in VEGF-A levels and an increase in TNF-α levels. TNF-α and VEGF-A were shown to be significantly positively correlated ($r = 0.396$, $p = 0.013$; *$p < 0.05$). The expression of S100A1 showed no discernible linear correlation with inflammatory mediators, indicating that the calcium-energetic axis is rather independent. Quantitative real-time PCR analysis revealed a significant downregulation of S100A1 mRNA expression in patients with (AMI) compared with healthy controls. The mean ΔCt difference was approximately 2.1 cycles (95% CI: 1.95-2.25), indicating an estimated 4.3-fold decrease in S100A1 protein mRNA expression in acute myocardial infarction.

Conclusion

Thus, these results support the inclusion of S100A1 in multi-marker frameworks to enhance risk classification in acute myocardial infarction and highlight its significance as a mechanistic biomarker for compromised cardiac function.

Keywords: S100A1 protein; TNF-α; VEGF; S100A1 mRNA expression; Acute myocardial infarction (AMI)

Paper Type: Research Paper.

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Introduction

Acute myocardial infarction (AMI) is a leading cause of morbidity and mortality worldwide despite major advances in reperfusion therapy and antithrombotic strategies (Matsuura et al., 2022; Sia et al., 2025). According to the Fourth Universal Definition of Myocardial Infarction, AMI is defined by acute myocardial injury with a dynamic rise and/or fall of cardiac troponin values, with at least one value above the 99th percentile upper reference limit, in the context of myocardial ischemia (Domienik-Karłowicz et al., 2021). Despite effectively reflecting structural necrosis, cardiac troponins do not give a complete picture of adaptive remodeling mechanisms and intracellular functional integrity. Myocardial necrosis, inflammatory activation, compromised calcium control, mitochondrial energy production, and vascular remodeling are all pathophysiological aspects of acute myocardial infarction (Buja, 2023). Early in ischemia, sarcoplasmic reticulum dysfunction and defective calcium ion reabsorption may cause disruption of the heart muscle's excitation-contraction coupling, which can lead to electrical instability and a decreased systolic reserve (Buja, 2023). The S100A1 protein, which is widely distributed in cardiac muscle cells, controls ryanodine receptors, ATP synthesis in mitochondria, and SERCA2a function (Cheng et al., 2025). Reduced S100A1 gene expression in the myocardium afflicted by ischemia and heart failure is linked to decreased calcium levels, which degrade myocardial contractility, according to experimental and applied research (Noll et al., 2024). At the same time, inflammatory cytokines (TNF-α) play a pivotal role in myocardial injury after infarction via NF-κB-dependent signaling pathways, which promote oxidative stress, mitochondrial dysfunction, and the inhibition of calcium-regulating proteins (Schumacher and Naga Prasad, 2018; Desai et al., 2022). On the other hand, VEGF acts as a central mediator of angiogenesis, endothelial repair after ischemic injury, microvascular integrity, and neovascularization after infarction (Khurana et al., 2005; Frangogiannis, 2014). The interrelated functions of calcium homeostasis, inflammation, and vascular repair in the pathophysiology of acute myocardial infarction (AMI) suggest that assessing circulating S100A1 protein and its gene expression, along with its association with TNF-α and VEGF, may yield mechanistic and predictive insights that surpass conventional biomarkers. The objective of this study was to assess S100A1 protein and its mRNA expression, as well as their correlation with inflammatory and vascular mediators in patients with AMI.

Materials and methods

Sample size determination: The sample size for the 1:1 case-control comparison was calculated based on previously documented and reported reductions in S100A1 protein expression in myocardium affected by ischemia and heart failure, which showed large standardized effect sizes (Cohen's coefficient $d = 0.9\text{-}1.1$) (Most et al., 2001; Pleger et al., 2007; Buja, 2023). Using a conservative estimate of $d = 0.90$, with a two-term significance level $\alpha = 0.05$ and a statistical power of 80%, the minimum sample size required was 20 individuals per group. However, to increase statistical power, and after excluding a number of participants, a much larger cohort was recruited, resulting in 88 participants per group in the final study.

Study population and design: The study carried out at the laboratories of the Department of Clinical Biochemistry at the College of Medicine, University of Babylon, Iraq, along with the Specialized Surgical Center for Cardiac Diseases. 200 sequential adults, aged 45 to 70, received laboratory testing from March to May 2025. Twenty-four participants were excluded due to clinically or laboratory evidence of acute or chronic infection, hence reducing any effect of confounding factors related to inflammation. The final study group consisted of 176 participants: 88 individuals with acute myocardial infarction and 88 seemingly healthy individuals, matched for age and sex in a 1:1 ratio. AMI was diagnosed according to the Fourth International Definition of Myocardial Infarction (FIDIV), current recommendations of the European Society of Cardiology (ESC) (Robert et al., 2023), and the American College of Cardiology/American Heart Association (ACC/AHA) (Rao et al., 2025). Diagnostic criteria included symptoms of ischemia, dynamic ECG changes, and elevated and/or decreased cardiac troponin levels. Meanwhile, the control group had no history of cardiovascular disease or ECG abnormalities, and their cardiac biomarkers were normal at study enrollment. Details of the inclusion and exclusion criteria are shown in Figure 1, while Figure 2 illustrates the study design.

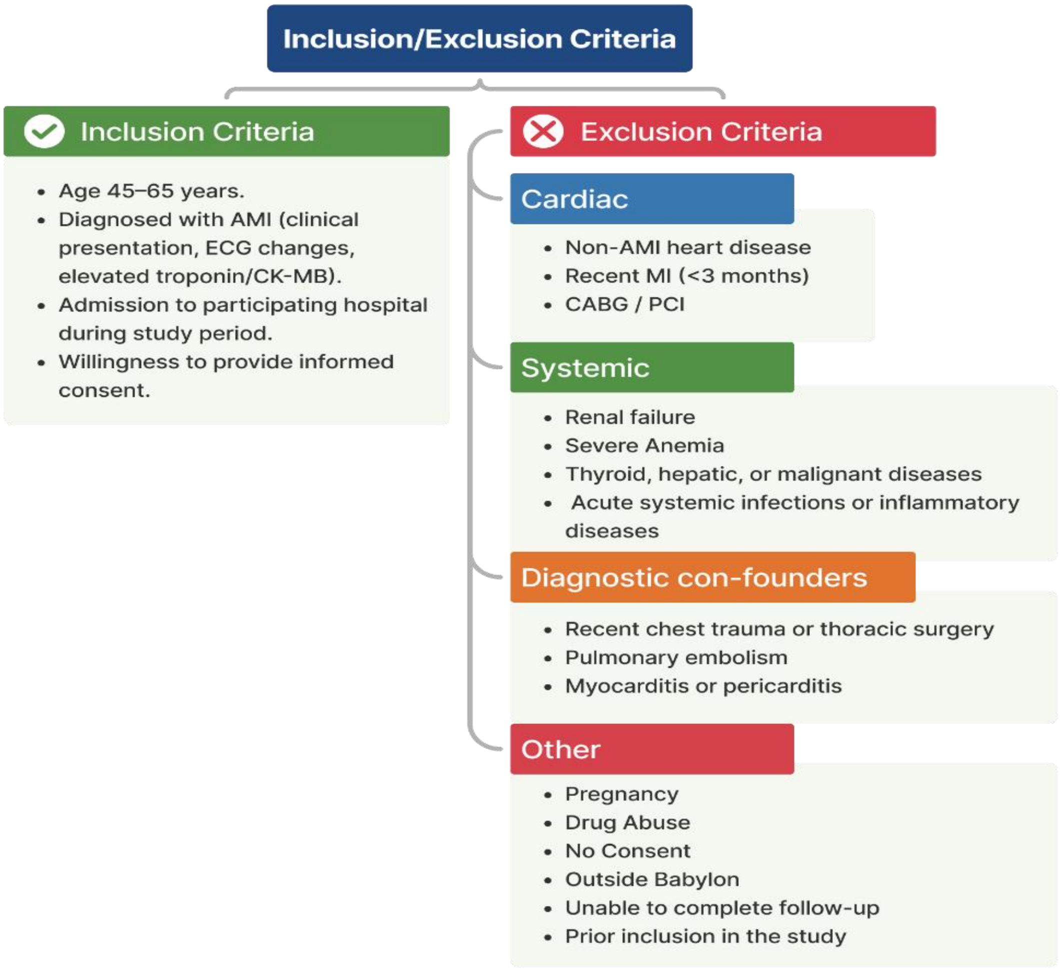


Figure 1. Flowchart showing the rules of this research for both including and excluding participants.

Biochemical and molecular analyses: Venous blood samples (5 mL) were obtained from patients with acute myocardial infarction (AMI) within 12 hours of symptoms onset and from control participants at the time of enrolment. From the whole fluid, 3 mL were put into plain tubes, allowed to coagulate, then centrifuged at 2000×g for 15 minutes to isolate serum. Serum aliquots were preserved at −80 °C until biochemical examination was conducted. The circulating concentrations of S100A1, TNF-α, and VEGF-A were measured using commercially available sandwich ELISA kits (Bioassay Technology Laboratory, China) in accordance with the manufacturer's instructions. All samples underwent double analysis, and mean values were utilized for statistical evaluation. Two milliliters of whole blood were placed in special cryo-resistant tubes and immediately frozen in liquid nitrogen (-196°C) until use for molecular analysis. This analysis was performed in a series of steps, beginning with the PureLink™ RNA Mini Kit (Thermo Fisher Scientific, USA) to separate the total RNA spectrophotometer was then used to verify the purity and concentration of the RNA and Samples with absorbances between 1.8 - 2.0 at wavelengths between 260 - 280 nm were considered acceptable. The High-Capacity cDNA Reverse Transcription Kit (Applied Biosystems) was utilized to generate complementary DNA (cDNA) from RNA. Quantitative real-time polymerase chain reaction (qPCR) was conducted with SYBR Green dye and the Stratagene Mx3005P platform. All reactions were conducted in triplicate with a final volume of 20 µL, and no-template controls were included in each run to prevent contamination.

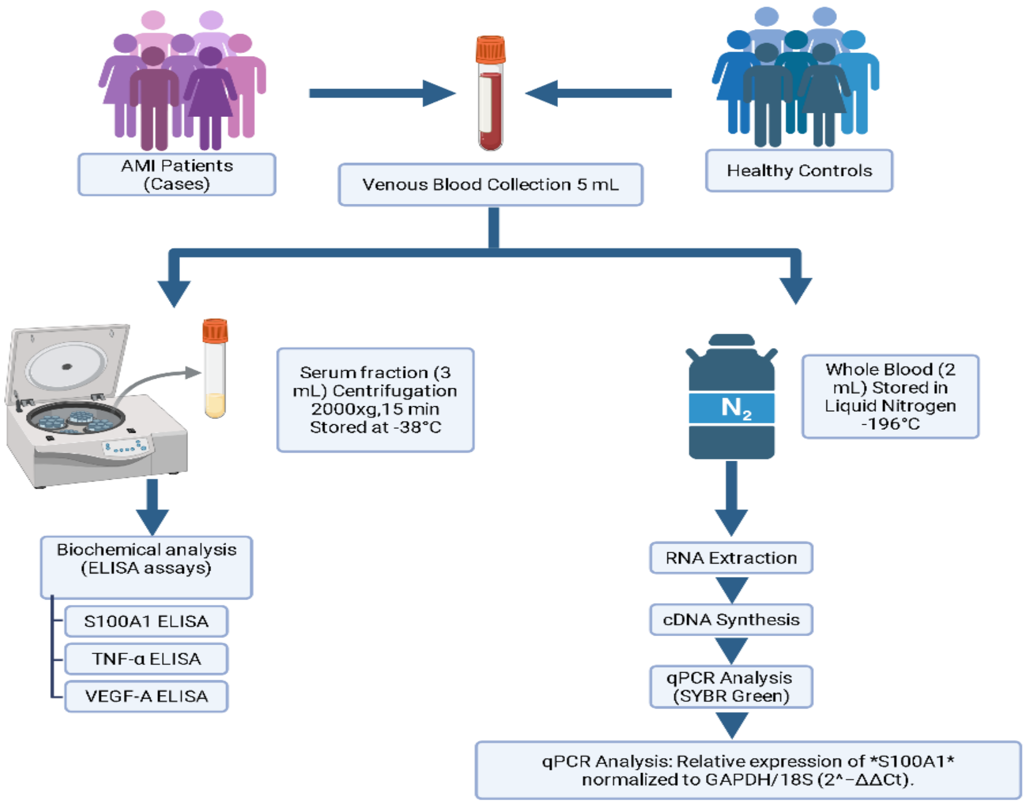


Figure 2. Diagram of the research design and analytical framework.

The primer's specificity was validated using melt-curve analysis, which displayed a single amplification peak without the formation of primer-dimers. The amplification efficiencies for S100A1 and the reference genes (GAPDH and 18S rRNA) were evaluated using standard curves, falling within the acceptable range of 90-110%. This confirmed the validity of the comparative Ct approach. The relative expression of the S100A1 gene was measured using the comparative Ct ($2^{-\Delta\Delta Ct}$) method. ΔCt values were calculated by normalizing the S100A1 Ct values to the geometric mean of GAPDH and 18S rRNA. The control group served as the reference for ΔΔCt calculations. Finally, the statistical analyses were carried out.

Ethical considerations: This research was conducted in accordance with the ethical principles adopted for human studies. Written informed consent was obtained from all participants prior to their enrollment, in addition to approvals from the ethics review committees of the Iraqi Ministry of Higher Education and Scientific Research and the Ministry of Health. All procedures were carried out in accordance with national regulations and international ethical standards relevant to biomedical research.

Statistical analysis: Statistical analyses were conducted utilizing SPSS (IBM v25.0) and GraphPad Prism (v9.0). Normality was evaluated with the Shapiro-Wilk test. Continuous variables are expressed as mean ± SD for regularly distributed data or as median (IQR) where applicable. Comparisons between groups were performed using independent-samples t-tests or Mann-Whitney U tests, where applicable. Categorical variables were examined with the Chi-square test. Pearson or Spearman correlation coefficients were computed based on the data distribution. Receiver operating characteristic (ROC) curve analysis was conducted to assess diagnostic performance, with the area under the curve (AUC) presented alongside 95% confidence intervals. Statistical comparisons for gene expression were conducted on ΔCt values, whereas fold changes ($2^{-\Delta\Delta Ct}$) were utilised for graphical representation. A two-tailed p-value of less than 0.05 was deemed statistically significant.

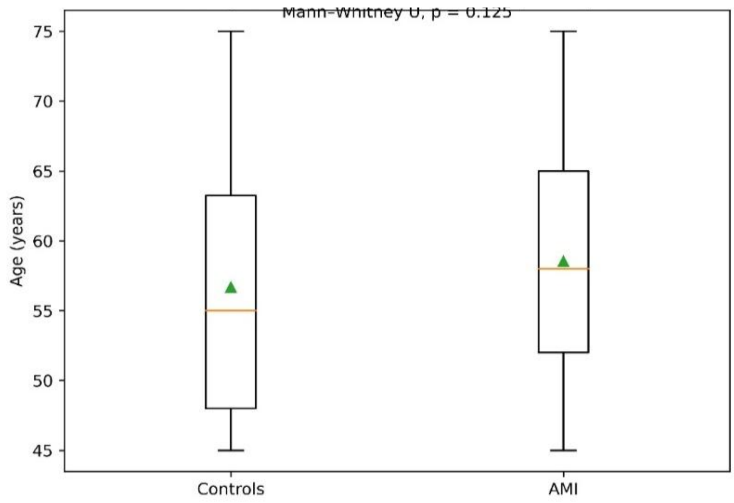
Results

Demographic characteristics of the studied groups (age, BMI and sex): The baseline demographic characteristics are presented in Table 1. There was no significant age difference between controls (56.68 ± 9.02 years) and AMI patients (58.52 ± 8.58 years) (Mann-Whitney U = 4390.0, p = 0.125). The sex distribution was uniform across groups, with 50.0% male and 50.0% female in both cohorts (Chi-square = 0.00, p = 1.000), demonstrating perfect equilibrium in sex allocation. The distribution of BMI categories varied considerably between groups (Chi-square = 12.71, p = 0.005). Normal BMI was more common among controls (70.1%) than in AMI patients (54.5%), while obesity (10.2%) and underweight status (2.3%) were solely found in the AMI group. The prevalence of overweight individuals was similar between the groups (29.9% vs 33.0%). These data indicate a notable alteration in BMI distribution towards higher-risk categories within the AMI group (Figure 3).

Table 1. Demographic characteristics of the studied groups (AMI patients vs healthy controls)

Variable		Control (n=88)	AMI (n=88)	Test	Statistic	P value
Age (years)		56.68 ± 9.02	58.52 ± 8.58	Mann-Whitney U	4390.0	0.125
Sex:	Male	44 (50.0%)	44 (50.0%)	Chi-square	0.00	1.000
	Female	44 (50.0%)	44 (50.0%)			
BMI	Normal	61 (70.1%)	48 (54.5%)	Chi-square	12.71	0.005
	Overweight	26 (29.9%)	29 (33.0%)			
	Obese	0 (0.0%)	9 (10.2%)			
	Underweight	0 (0.0%)	2 (2.3%)			

Age is reported as mean ± SD. Categorical variables are reported as n (%).



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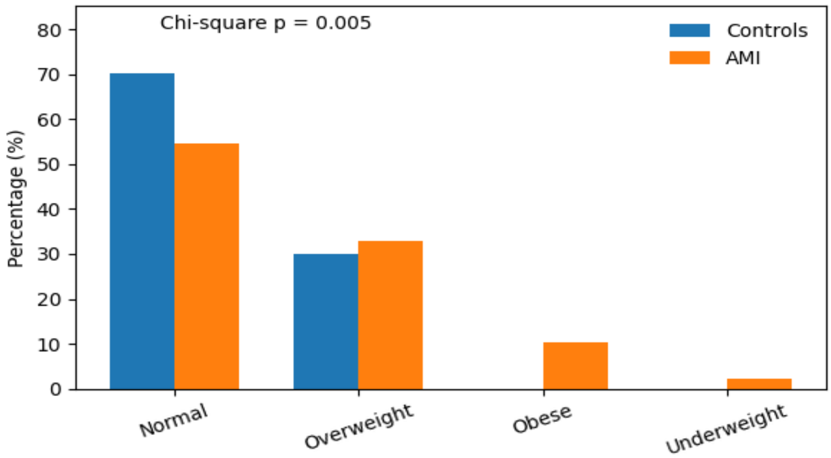

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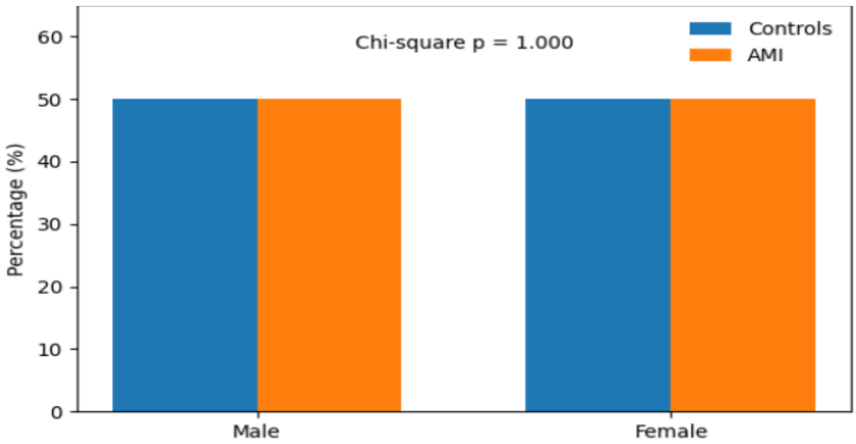

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Figure 3. Demographic characteristics of the studied groups (age, BMI and sex), A. Age comparison between study groups, B. BMI category distribution between study groups, C. Sex distribution between study groups

Pearson correlation analysis: Pearson correlation analysis demonstrated significant positive associations between TNF-α and VEGF-A ($r = 0.396$, $p = 0.013$) No significant linear association was observed between S100A1 fold change and inflammatory mediators.

Conversely, TNF-α exhibited a notable positive association with VEGF-A ($r = 0.396$, $p = 0.013$)), indicating synchronised inflammatory activation (Figures 4 and 5).

Table 2. Pearson correlation matrix of circulating biomarkers and S100A1 expression

Variables	S100A1 FC**	TNF-α	VEGF-A	S100A1 (protein)
S100A1FC	1.00	0.032	0.30	-0.309
TNF-α	0.032	1.00	0.396*	-0.282
VEGF-A	0.30	0.396*	1.00	0.050
S100A1 (protein)	-0.309	-0.282	0.050	1.00

FC**S100A1 fold change ($2^{-\Delta\Delta Ct}$), *$p < 0.05$, TNF-α=Tumor Necrosis Factor-alpha, VEGF-A=Vascular Endothelial Growth Factor.

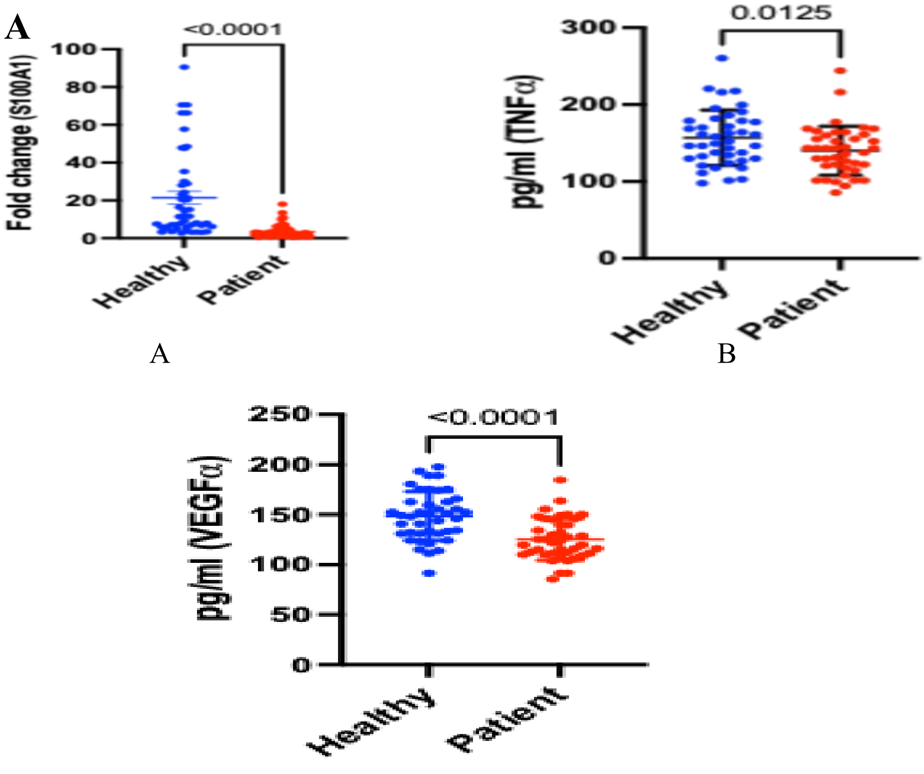

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Figure 4. Circulating biomarker concentrations in AMI patients and controls. A. Serum S100A1 levels were significantly reduced in AMI patients compared with controls. B. Serum TNF-α levels were significantly elevated in the AMI group. C. Serum VEGF-A concentrations were significantly decreased in AMI patients. Data are presented as mean ± SD. Statistical comparisons were performed using independent-samples tests. *$p < 0.05$; ****$p < 0.0001$

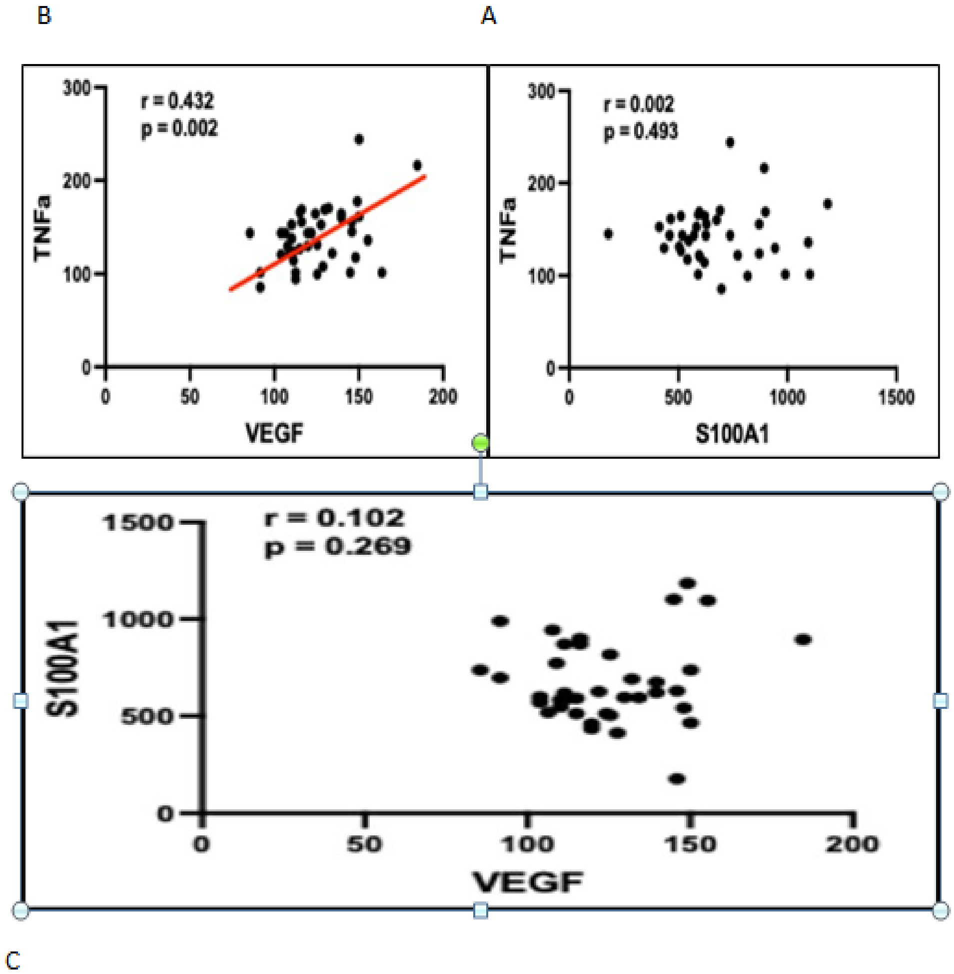

Figure 5. Correlations among circulating S100A1, TNF-α, and VEGF-A. (A) S100A1 vs TNF-α, (B) VEGF-A vs TNF-α, (C) S100A1 vs VEGF-A

Correlation structure of circulating biomarkers: The mechanistically structured Pearson correlation matrix is orientated from inflammatory (TNF-α) and angiogenic (VEGF-A) mediators to calcium signaling (S100A1 protein and S100A1 mRNA fold change). Correlation coefficients are visualised in the heatmap. TNF-α and VEGF-A were shown to be significantly positively correlated ($r = 0.396$, $p = 0.013$; $^*p < 0.05$). The expression of S100A1 showed no discernible linear correlation with inflammatory mediators, indicating that the calcium-energetic axis is rather independent (Figure 6).

S100A1 mRNA Expression in Acute Myocardial Infarction: Quantitative real-time PCR analysis revealed a significant downregulation of S100A1 mRNA expression in patients with (AMI) compared with healthy controls. In this case-control study, real-time PCR amplification kinetics revealed distinct and biologically relevant variations in S100A1 transcription between the groups. In healthy controls (Figure 7-A), amplification curves commenced the exponential phase earlier (about cycles 20-23), with the majority of responses surpassing the fixed fluorescence threshold within a limited Ct range (≈23-25 cycles). The graphs had steep, virtually parallel slopes during exponential amplification, signifying good reaction efficiency, optimal assay performance, and uniform transcript abundance among control samples.

A

	S100A1 Protein	S100A1 FC (mRNA)	TNF-α	VEGF-A
S100A1 Protein	1.000	-0.309	-0.282	0.050
S100A1 FC (mRNA)	-0.309	1.000	0.032	0.300
TNF-α	-0.282	0.032	1.000	0.396
VEGF-A	0.050	0.300	0.396	1.000

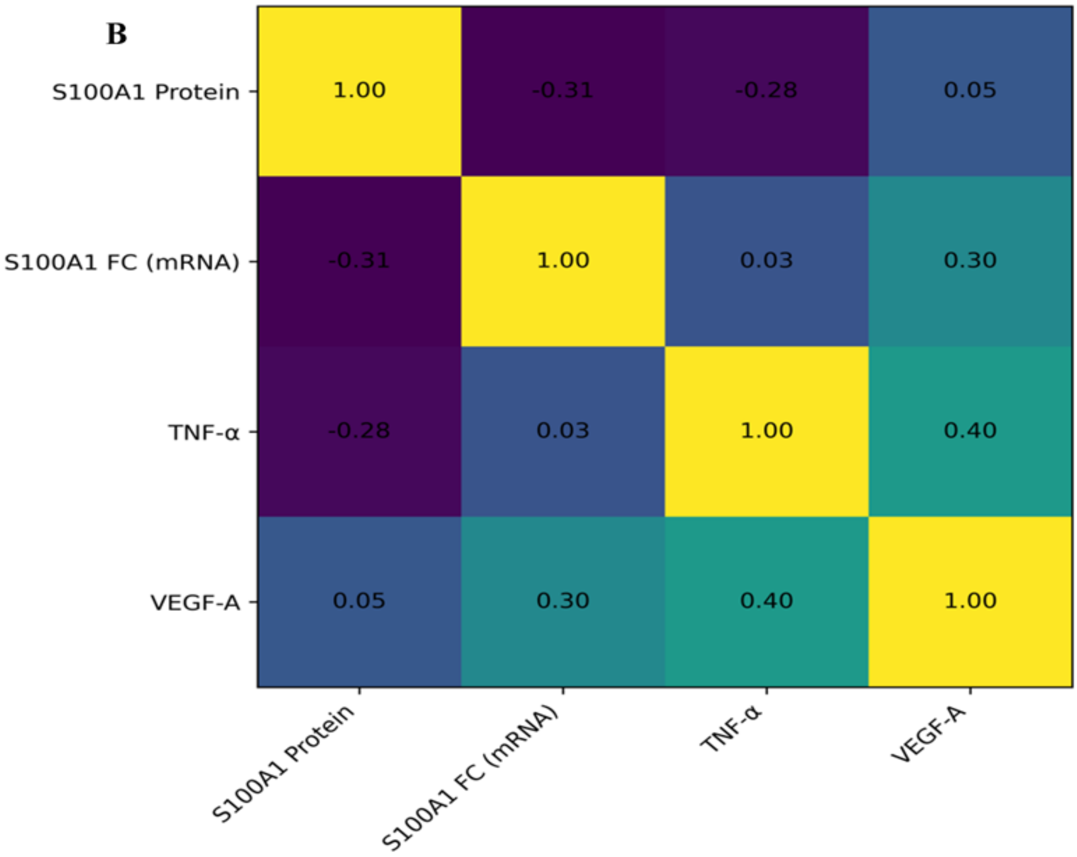

Figure 6. A. Mechanistically Ordered Correlation Matrix, B. Heatmap of Correlation Coefficients

The slight variation between samples reinforces biological consistency within the healthy group. In contrast, patients with acute myocardial infarction (Figure 7-B) exhibit a marked rightward shift in the amplification curves, characterized by a delayed exponential onset (approximately 23-27 cycles) and thresholding at much higher Ct values. This confirms the preservation of the x-shape and parallel gradients, indicating the adequacy of the test specificity and amplification efficiency; however, the wider curve dispersion in patients suggests increased biological variability associated with acute ischemic injury. Real-time quantitative polymerase chain reaction (qPCR) analysis showed a marked increase in S100A1 protein Ct levels in patients with acute myocardial infarction compared to the control group (25.73 ± 0.47 vs. 23.63 ± 0.46; $p < 0.0001$). The mean ΔCt difference was approximately 2.1 cycles (95% CI: 1.95-2.25), indicating an estimated 4.3-fold decrease in S100A1 protein mRNA expression in acute myocardial infarction (relative change was 0.23) (Table 3).

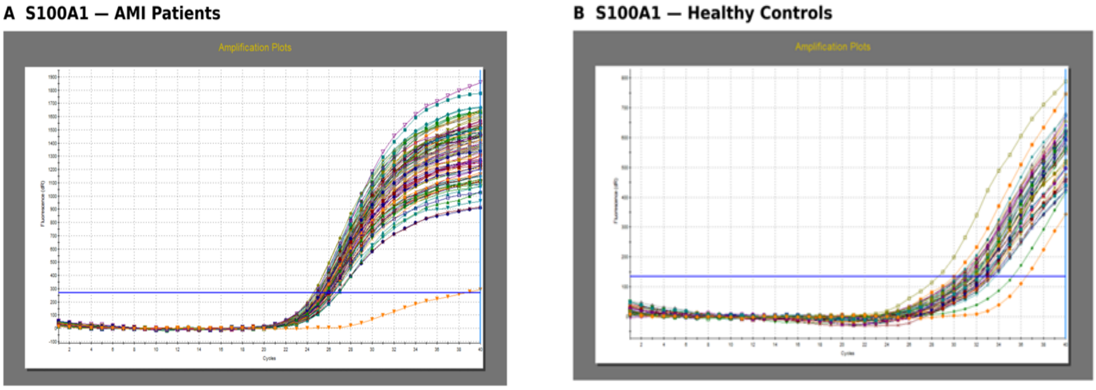

Figure 7. S100A1 expression patterns in acute myocardial infarction using real-time polymerase chain reaction amplification. (A) Amplification plots from AMI patients. (B) Amplification plots from healthy control samples

Table 3. The S100A1 Ct values for each of the study groups

Variable	Cohen's d	p-value	Controls (n=88)	AMI Patients (n=88)
S100A1 Ct (Mean ± SD)	4.47	<0.0001	23.63 ± 0.46	25.73 ± 0.47
95% Confidence Intervals			23.50 - 23.76	25.63 - 25.83

Mean difference 95% CI: 1.95 - 2.25.

Discussion

Acute myocardial infarction (AMI) is acknowledged not merely as an ischemic event but also as a systemic disturbance characterized by the dynamic reprogramming of cardiomyocyte calcium homeostasis, innate immune activation and vascular repair mechanisms (Most et al., 2001). Our findings position S100A1 at the intersection of these processes and suggest that its depletion represents more than a mere passive indicator of injury and it may signify a failure of myocardial energy resilience. S100A1 is a cardiomyocyte Ca^{2+} sensor that precisely regulates excitation -contraction coupling by modulating SERCA2a, ryanodine receptors, and mitochondrial Ca^{2+} flow in a coordinated manner (Byrne et al., 2023; Buja, 2023). This signaling network keeps the muscles working well and protects the body's energy balance in normal conditions (Schumacher and Naga Prasad, 2018; Morciano et al., 2022) (Figure 8). The experimental restoration of S100A1 improves post-ischemic function, underscoring its importance as a key regulator of myocardial performance (Gopal et al., 2013; Desai et al., 2022; Robert et al., 2023). The observed reduction in circulating S100A1 in our AMI sample likely signifies a disruption of adaptive calcium - energetic coupling during peak metabolic demand and oxidative stress.

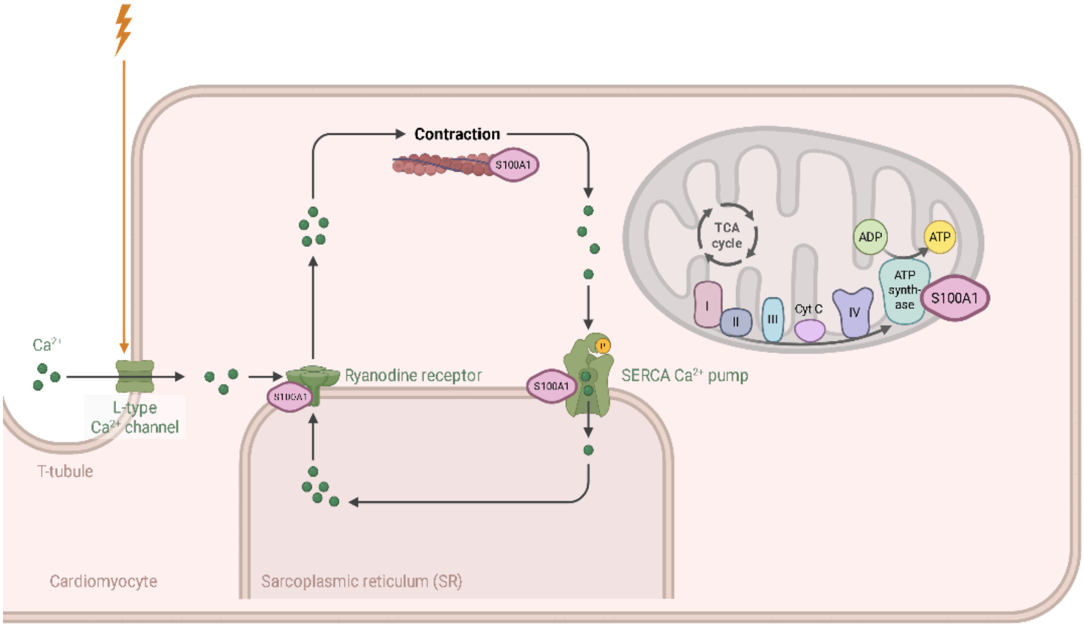

Figure 8. Schematic diagram illustrating the molecular function of the S100A1 protein in calcium regulation in cardiac muscle cells. Created using https://BioRender.comn

The decline in S100A1 mRNA levels is associated with a reduction in myocardial S100A1 levels during heart failure and ischemia, indicating that transcriptional and translational remodeling has been influenced by the stress response (Desai et al., 2022). However, the peripheral transcription measurements are significantly affected by the dynamics of inflammatory cells. S100A1 expression is low in leukocytes, which causes neutrophils and monocytes to multiply quickly after an acute myocardial infarction. This, in turn, reduces the quantity of myocardial-derived transcripts in whole blood (Cheng et al., 2025). Therefore, the decreased circulating mRNA level of the S100A1 protein indicates a complex of biological signals, including myocardial cell death, altered cellular energy utilization, and changes in the types of inflammatory cells present. This interpretation is consistent with the idea that the molecular markers circulating in the blood during acute myocardial infarction point to systemic reorganization rather than mere gene suppression (Sun et al., 2024). Concurrently with S100A1 deletion, TNF-α levels were markedly increased, underscoring the pivotal role of innate immune activation in infarct progression (Noll et al., 2024). TNF-α compromises calcium-handling proteins, diminishes contractility, and induces mitochondrial oxidative dysfunction (Frangogiannis et al., 2002; Sun et al., 2024). Its signaling is primarily facilitated by NF-κB activation, which coordinates the transcriptional reprogramming of inflammatory networks (Liu et al., 2017; Morciano et al., 2022). Additionally, S100A8/A9 released as damage-associated molecular patterns can enhance TNF-α production through TLR4 and RAGE interaction, hence exacerbating NF-κB-dependent inflammatory pathways (Khurana et al., 2005; Ehrchen et al., 2009; Sun et al., 2024). These pathways create a self-perpetuating cycle that could intensify cardiac stress if not properly addressed. Despite this mechanistic feasibility, we found no significant linear association between S100A1 and TNF-α. This evident separation holds conceptual significance. It indicates that calcium-energetic failure and cytokine-mediated inflammation, although interacting, are not necessarily linked at the systemic level. Instead, they may signify concurrent stress axes functioning on different temporal and cellular scales. TNF-α primarily indicates systemic inflammation, while S100A1 assesses the integrity of cardiac cell function. The independence of these two markers underscores the complex biology of myocardial infarction and cautions against reducing cardiac injury to a single inflammatory model. Meanwhile, decreased VEGF-A levels support the hypothesis that VEGF-A is essential for endothelial cell survival, angiogenesis, and microvascular repair (Frangogiannis, 2014; Sreejit et al., 2019). Reduced VEGF-A levels in acute myocardial infarction could indicate defective adaptive neovascularization, prolong microvascular hypoxia and delay functional recovery (Livak and Schmittgen, 2001; Frangogiannis, 2014). The reduction in VEGF-A, coupled S100A1 depletion and TNF-α raised, indicates a maladaptive remodeling environment characterized by energy deficits, prolonged inflammation, and impaired reparative signaling. This study had several limitations as Some patients were excluded due to incomplete data, and all data were obtained from a single center, limiting the generalizability of the findings in addition to Challenges in patient recruitment that arose from participants' fear of blood collection. The absence of detailed clinical outcome measures, such as comorbidities, restricted correlations with biochemical parameters. Moreover, subsequent studies ought to incorporate larger cohorts of patients from various centers, encompassing thorough clinical and biochemical evaluations, as well as lifestyle assessments, to augment the validity and applicability of the findings.

Conclusion: Acute myocardial infarction is not merely an ischemic occurrence but a synchronized failure of calcium signaling, inflammatory activation, and vascular adaptation. Our findings identify S100A1 as central to this disturbance. The significant decrease in circulating S100A1 at both protein and mRNA levels indicates a dysfunction in cardiomyocyte calcium-energetic coupling at peak metabolic demand. Importantly, this depletion appears biologically dissociated from NF-κB-dependent cytokine amplification and VEGF-driven angiogenic responses, suggesting that contractile dysfunction, inflammation, and repair are mechanistically parallel rather than linearly coupled processes. Within this paradigm, S100A1 serves as a molecular marker of myocardial energy resilience and a possible target for therapeutic intervention in ischemic heart disease.

Author contributions

Conceptualization: A. H. A. and A. A., Methodology: A. A. and E. F. H., Data collection and analysis: A. H. A., A. A., and E. F. H., Manuscript preparation: E. F. H. and A. H. A.

Data availability statement

The data of this research article are available from the authors upon reasonable request.

Acknowledgments

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Ethical considerations

This research was conducted in accordance with the ethical principles adopted for human studies. Written informed consent was obtained from all participants prior to their enrollment, in addition to approvals from the ethics review committees of the Iraqi Ministry of Higher Education and Scientific Research and the Ministry of Health. All procedures were carried out in accordance with national regulations and international ethical standards relevant to biomedical research.

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Conflict of interest

The authors declare no conflict of interest.

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کاهش بیان S100A1 به‌عنوان یک نشانگر کلسیمی-انرژتیکی در انفارکتوس حاد میوکارد: ارتباط با مسیرهای آنژیوژنیک و التهابی

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چکیده

هدف: انفارکتوس حاد میوکارد (AMI) وضعیتی است که با بازبرنامه‌ریزی هماهنگ زیستی مشخص می‌شود و شامل اختلال در تنظیم کلسیم، فعال‌شدن مسیرهای التهابی و اختلال در ترمیم عروقی است. پروتئین S100A1 یک پروتئین متصل‌شونده به Ca^{2+} است که در کاردیومیوسیت‌ها فراوان بوده و نقش مهمی در کوپلینگ تحریک-انقباض و متابولیسم انرژی میتوکندریایی دارد؛ بنابراین می‌تواند نشانگر پایداری عملکرد قلب فراتر از آسیب نکروتیک باشد. هدف این مطالعه بررسی سطح پروتئین S100A1 و بیان mRNA آن و همچنین ارتباط آن با میانجی‌های التهابی و عروقی در بیماران مبتلا به AMI بود.

مواد و روش‌ها: این مطالعه (n=176) سطح پروتئین S100A1 در گردش خون و بیان ژنی آن را همراه با سطوح TNF-α و VEGF-A ارزیابی کرد. نمونه‌های خون وریدی (۵ میلی‌لیتر) از بیماران مبتلا به انفارکتوس حاد میوکارد طی ۱۲ ساعت اول پس از شروع علائم و از افراد سالم در زمان ورود به مطالعه جمع‌آوری شد. واکنش زنجیره‌ای پلیمراز کمی در زمان واقعی (qPCR) با استفاده از SYBR Green و دستگاه Stratagene Mx3005P انجام شد. تحلیل‌های آماری با نرم‌افزارهای SPSS (IBM v25.0) و GraphPad Prism (v9.0) صورت گرفت.

نتایج: بیماران مبتلا به AMI کاهش قابل‌توجهی در سطح پروتئین S100A1 هم در سطح پروتئینی و هم در سطح رونویسی نشان دادند. این کاهش همزمان با کاهش سطح VEGF-A و افزایش سطح TNF-α بود. بین TNF-α و VEGF-A همبستگی

مثبت معنی‌داری مشاهده شد ($r = 0.396, p = 0.013; *p < 0.05$). با این حال، بیان S100A1 همبستگی خطی مشخصی با میانجی‌های التهابی نشان نداد که بیانگر استقلال نسبی محور کلسیم–انرژی است. نتایج qRT-PCR نشان داد که بیان mRNA ژن S100A1 در بیماران مبتلا به AMI به‌طور معنی‌داری کاهش یافته است. اختلاف میانگین ΔCt حدود ۲/۱ سیکل (95% CI: 1.95-2.25) بود که نشان‌دهنده کاهش تقریبی ۴/۳ برابری بیان mRNA پروتئین S100A1 در انفارکتوس حاد میوکارد است.

نتیجه‌گیری: نتایج این مطالعه از گنجاندن S100A1 در چارچوب‌های چندنشانگری برای بهبود طبقه‌بندی خطر در انفارکتوس حاد میوکارد حمایت می‌کند و اهمیت آن را به‌عنوان یک بیومارکر مکانیستی برای اختلال عملکرد قلب نشان می‌دهد.

کلمات کلیدی: انفارکتوس حاد میوکارد (AMI)، بیان mRNA ژن S100A1، پروتئین S100A1، TNF-α، VEGF

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