Diyala Journal of Medicine

ORIGINAL RESEARCH
Published: 25 April 2026
DOI: 10.26505/djm.v30i1.1357

Early Diagnosis of Myocardial Infarction by Ischemia-Modified Albumin and Antioxidant Markers

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Received: 14 March 2025
Accepted: 23 May 2025
Published: 25 April 2026

Abstract

Background: Myocardial infarction and oxidative stress drive the damage to myocardial tissue, culminating in cell necrosis. The study measured IMA, TAC, DPPH, total protein, and serum albumin in patients with MI.

Objectives: To analyze and diagnose the usefulness of IMA concentration in patients with potential MI signs within six hours.

Patients and Methods: During the period from September 2024 to February 2025, eighty individuals diagnosed with myocardial infarction (MI) who were admitted to Baquba Teaching Hospital in Iraq were enrolled in this study. In addition, twenty healthy individuals were included as a control group. IMA was measured using an ELISA kit, while serum TAC, DPPH, total protein, and albumin were measured spectrophotometrically.

Results: The MI group showed a significantly elevated serum IMA concentration compared to healthy controls ($p < 0.001$). Patients with MI showed lower antioxidant activity and reduced levels of antioxidant molecules. There was no apparent association between serum IMA and DPPH, TAC, total protein, or serum albumin. However, positive correlations emerged between TAC and DPPH (0.678, $p < 0.001$), total protein and serum albumin (0.640, $p < 0.001$), and TAC and serum albumin (0.575, $p < 0.001$).

Conclusion: The aim of the study was to elucidate the crucial role of measuring IMA, TAC, DPPH, total protein, and serum albumin in patients with MI, alongside other oxidative stress indicators, to guide the development of targeted antioxidant treatments for early diagnosis.

Keywords: Myocardial infarction, IMA, Oxidative stress, Antioxidant, Ischemia.

Introduction

Myocardial infarction (MI) arises from atherosclerotic plaques that rupture, ulcerate, fissure, or erode, causing one or more coronary arteries to develop an intraluminal thrombus (ILT). This process leads to reduced blood circulation to the heart muscle and/or distal embolization, ultimately resulting in myocardial necrosis (1). During myocardial ischemia, which sets in almost immediately after a vascular blockage, aerobic glycolysis within the myocytes ceases. As a result, ATP production drops sharply, and creatine phosphate stores are depleted, causing an accumulation of lactic acid and NADH. The accompanying fall in pH leads to progressive dysfunction of cellular proteins and enzymes. This decline in ATP level also hampers the plasma membrane's sodium pump, leading to an influx of sodium while potassium leaves the cell. Likewise, the impaired calcium pump allows excessive calcium to enter, amplifying the tissue damage (2). Lower pH triggers the release of copper and iron previously bound to proteins or stored intracellularly. These metals, in turn, affect the ETC and promote the production of ROS, including superoxide (SOD) (3). Oxidative stress from free radicals targets the histidine

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residues in albumin's amino-terminal region, causing structural alterations. Once the amino terminus is affected, the protein is referred to as IMA (4). (IMA) occurs as soon as myocardial ischemia starts and is a "N-terminal modified" albumin. This region, found at the amino endpoint of HSA (human serum albumin), normally binds to metals in transition, such as Co, Cu, and Ni (5, 6). Albumin stands as the most prevalent protein in the human body, with concentrations of roughly 35–50 g/L. It has numerous physiological and therapeutic roles, including transporting small molecules such as medications, fatty acids, and bilirubin and providing antioxidant protection (7, 8). Human albumin (HA) makes up about half of the total plasma protein content and is essential for maintaining oncotic pressure. Under typical physiological conditions, its buffering capacity primarily stems from the 16 histidine imidazole residues in its structure(9, 10). An antioxidant is a substance that, even in relatively small amounts compared to a component that is reactive with oxygen (such as proteins, lipids, carbohydrates, or DNA), can greatly delay or prevent that material from undergoing oxidation (11, 12). A key role of antioxidants is to shield the body from the harmful effects caused by free radical damage (13). Free radicals can arise within cells and tissues through internal factors like inflammation, diseases, or metabolic processes, as well as from external sources such as radiation, pollution, certain foods, or medications. They may also form when the body's protective mechanisms become insufficient (14). Regardless of the source, heightened free radical production can lead to oxidative damage (15). It further explores the role of these indicators in distinguishing MI prior to the identification of ACS. Another objective is to evaluate IMA's diagnostic capacity, particularly in conjunction with additional biomarkers, for identifying low-risk patients potentially experiencing AMI. IMA determination according to detecting the way it interacts with metal ions, namely Co2+, was initially developed by Bar-Or et al. (16). IMA, Chol, and (LDL) levels were discovered to be positively correlated in individuals with diabetic nephropathy in earlier studies (17). This study aims to analyze the diagnostic (IMA) levels' utility in patients who arrive at the emergency room within six hours with possible MI complaints

Patients and Methods

Study design: Between September 2024 and February 2025, 80 blood samples were collected from MI patients at Baquba Teaching Hospital in Baquba, Diyala Governorate, who presented within 6 hours of onset. Diagnosis was confirmed by a specialist using the MI criteria (18). (19). Among these patients, 60 males and 20 females, aged 55 to 70 years. Twenty blood samples were obtained from individuals in good health to establish IMA reference ranges. These participants were classified as free from cardiac and renal conditions (no history of myocardial infarction, heart failure, hypertension, or dyslipidemia) and had no record of cancer. Half of the participants were male, and half were female, all between 55 and 70 years of age. Approximately 5 mL of venous blood was drawn using plastic syringes and placed into gel tubes for testing. The serum was separated by centrifugation (5 minutes at 3000 rpm), then divided into 500 µL aliquots in tubes and kept until needed at -20 °C.

Clinical laboratory analysis of groups: An ELISA plate reader was used to determine the IMA, and the ELISA kit from Shanghai Comp. (China) was used to measure IMA concentrations. Serum total antioxidant capacity (TAC) and DPPH were determined through an enzymatic colorimetric assay with kits from Elabscience. In addition, serum total protein and albumin levels were analyzed by enzymatic colorimetry using a kit provided by Clona (USA).

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Statistical Analysis

The statistical analysis was performed using SPSS (version 25). The frequency/percentage was utilized for categorical variables, while the mean and standard deviation were used for numerical variables that were normally distributed. An independent t-test and chi-square test were used to further assess the significance of the difference between the normally distributed numerical variables, with a p-value of 0.05 serving as the significance criterion. IMA, TAC, DPPH, total protein, and serum albumin were tested for their diagnostic or screening potential using receiver operating characteristic (ROC) curve analysis.

Results

Myocardial infarction patient groups and healthy subjects: Statistical analysis revealed no noteworthy differences in female representation between the patient and healthy groups ($P > 0.05$). Likewise, there was no significant variation in the proportion of males when comparing patients to the healthy controls ($P > 0.05$). Table 1 and Figure 1 show the following outcomes.

Table 1. Comparative study of sex and age between study groups calculated by Chi-square and t-test.

Parameter			Groups		Total	P value
			Patient (80)	Healthy control (20)	(100)	
Sex	Male	N	60	10	70	> 0.05
		%	75 %	50 %	70 %	
	Female	N	20	10	30	
		%	25 %	50 %	30 %	
P value			> 0.05	> 0.05	> 0.05	
Age (mean±SD)			61.4±9.44	61.6±7.18		> 0.05

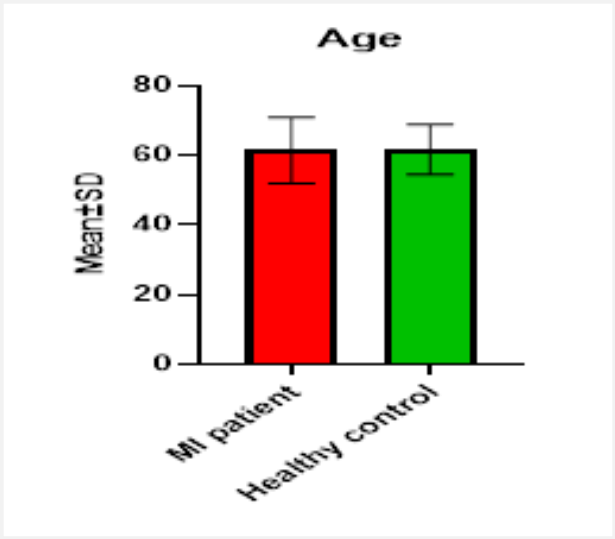

Figure 1. Mean and standard deviation of age.

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Ischemia modified albumin measurements: Serum IMA level increased in patients (1050.5 ± 11.7 ng/mL) in comparison to the healthy control group (455.7 ± 133.9 ng/mL). Additionally, a fairly significant difference was revealed. $p < 0.001$) as depicted in Table 2.

Diagnostic accuracy of ischemic modified albumin: ROC and AUC analyses (see Table 3 and Figure 2) indicated that IMA performed exceptionally well as a diagnostic parameter for distinguishing MI patients from the control group, achieving both 100% sensitivity and 100% specificity. The AUC yielded a p-value below 0.001, affirming its significance as a reliable biomarker.

Table 2. Comparative study between study groups based on ischemia-modified albumin test determined by the t-test

Groups		Numbers	Mean(ng/ml)	SD	*P* value
Ischemia modified albumin	Patients	80	1050.5	11.7	< 0.001**
	Healthy	20	455.7	133.9	

Table 3. Sensitivity and specificity of the IMA test.

Variable	AUC	Std. Error[a]	*P* value	Sensitivity %	Specificity %
ischemia modified albumin test	1.000	0.000	< 0.001**	100 %	100 %

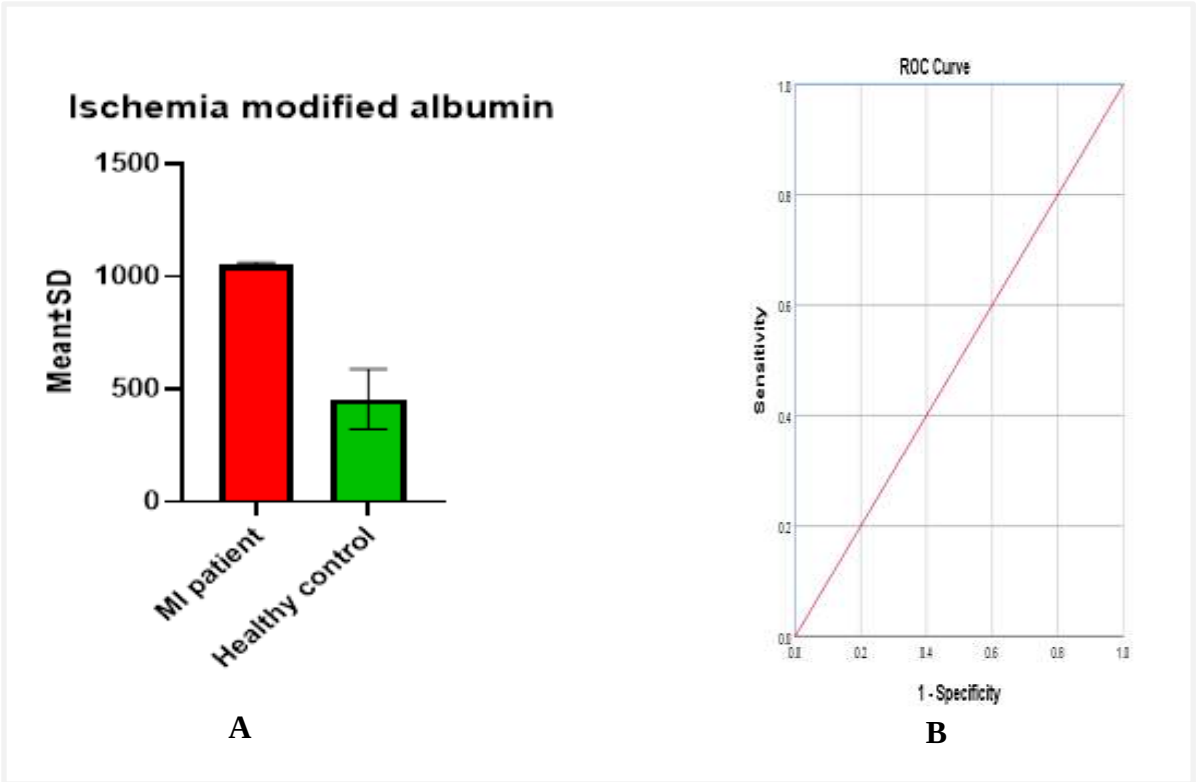

Figure 2. (A) Mean and standard deviation of ischemia-modified albumin. (B) ROC curve of ischemia-modified albumin.

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Antioxidant markers measurements: According to the data in Table 4 and Figure 3, serum DPPH and TAC levels were lower (22.45 ± 9.54 and 16.52 ± 2.44) in patients with MI compared to the healthy control group (40.3 ± 11.49 and 30.3 ± 6.6), respectively; in MI patients, demonstrating a notable distinction (P< 0.001**).

Table 4. Comparative study between study groups based on DPPH and TAC test measured by t-test.

Groups		N	Mean (µmol/L)	SD	P value
DPPH	Patients	80	22.45	9.54	< 0.001**
	Healthy	20	40.30	11.49	
TAC	Patients	80	16.52	2.44	< 0.001**
	Healthy	20	30.30	6.68	

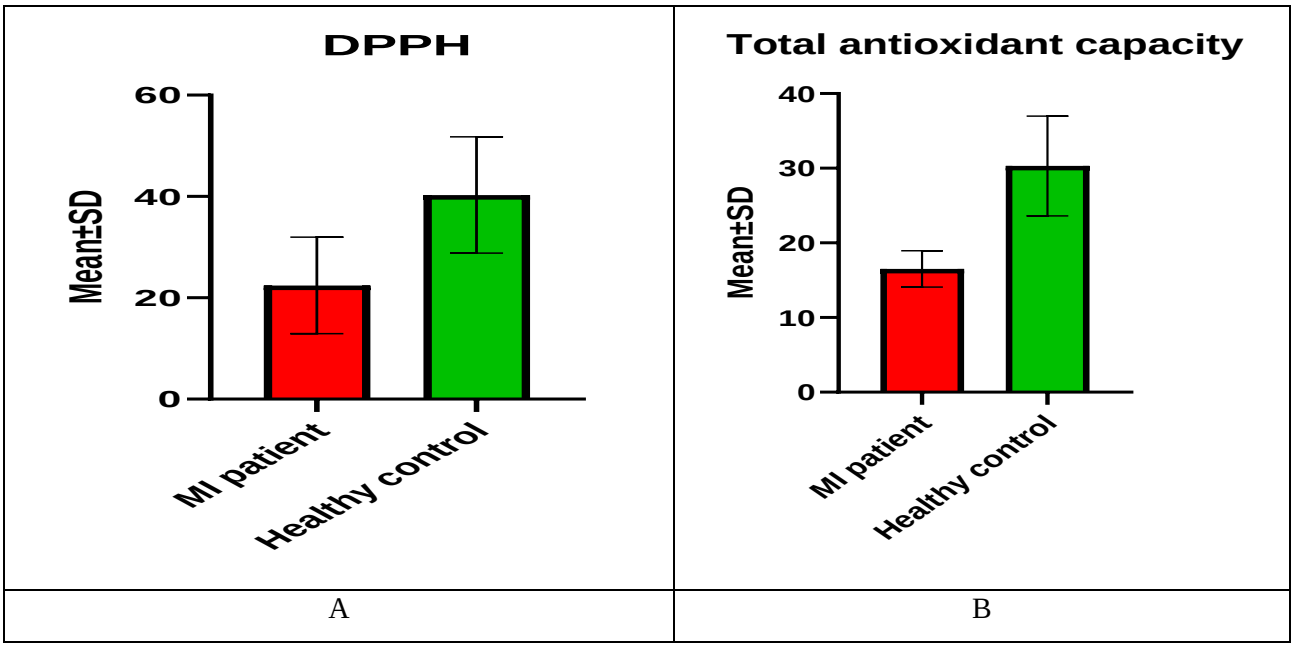

Figure 3. (A) Mean ± SD of DPPH and(B) Mean ± SD of TAC.

Diagnostic accuracy of DPPA and TAC: ROC and AUC evaluations for DPPH and TAC revealed promising diagnostic performance in differentiating MI patients from controls (Table 5, Figure 4). DPPH showed 80% accuracy and 90% specificity, while TAC achieved 100% sensitivity and 95% specificity. Both p-values were below 0.001, underscoring their statistical significance. These high sensitivity and specificity rates highlight the potential of DPPH and TAC as reliable & safe approaches for MI diagnosis. Additionally, the p-value < 0.001 for DPPH confirms that antioxidant capacity can serve as an effective biomarker.

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Table 5. Sensitivity and specificity of TAC and DPPH test.

Variables	AUC	Std. Error[a]	*P* value	Sensitivity %	Specificity %
DPPH	0.906	0.057	< 0.001**	80 %	90 %
TAC	0.996	0.005	< 0.001**	100 %	95 %

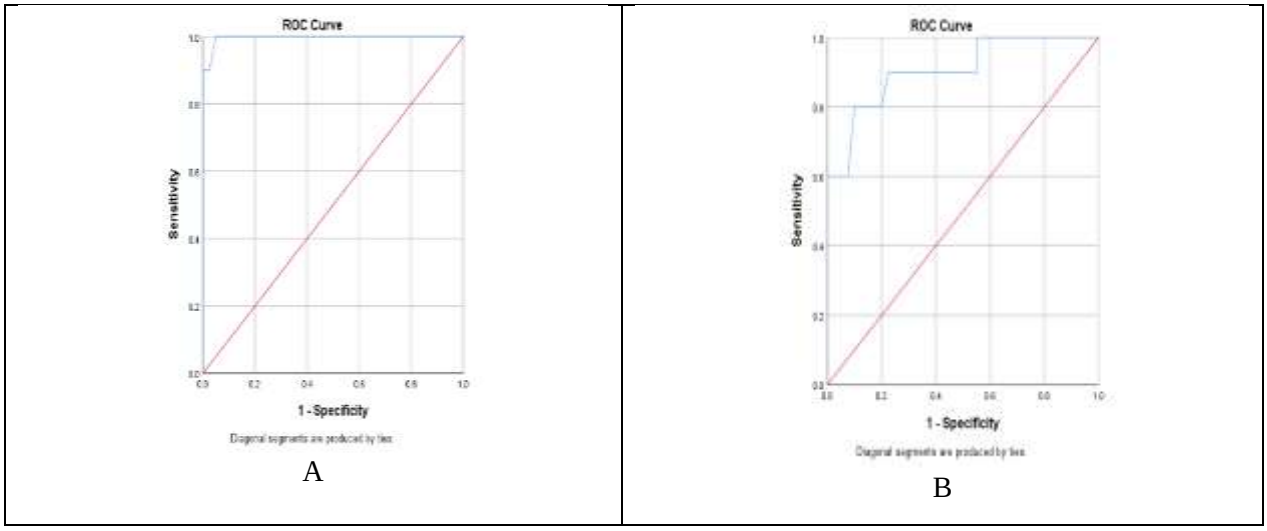

Figure 4. (A) ROC curve of DPPH and (B) ROC curve of TAC.

Proteins parameters: Table 6 and Figure 5 demonstrate a substantial rise in blood protein levels in MI patients. (4.57 ± 1.29 g/dl) in contrast to the group of healthy subjects (3.98 ± 0.61mg/dl). Additionally, MI patients had much lower serum levels. (5.26 ± 1.75) mg/dl compared to the healthy group (3.75 ± 1.81) mg/dl, as displayed in Table 6; the concentration of serum T.P and S.ALB. in the I group was compared with the healthy group ($p < 0.05$).

Table 6. Comparative study between study groups based on the S.TP and S.ALB test determined by the t-test.

Groups		Numbers	Mean(mg/dl)	SD	P value
Total protein	Patients	80	4.57	1.29	< 0.05
	Healthy	20	3.98	0.61	
Groups		N	Mean	SD	*P* value
ALB	Patients	80	5.26	1.75	< 0.05
	Healthy	20	3.75	1.81	

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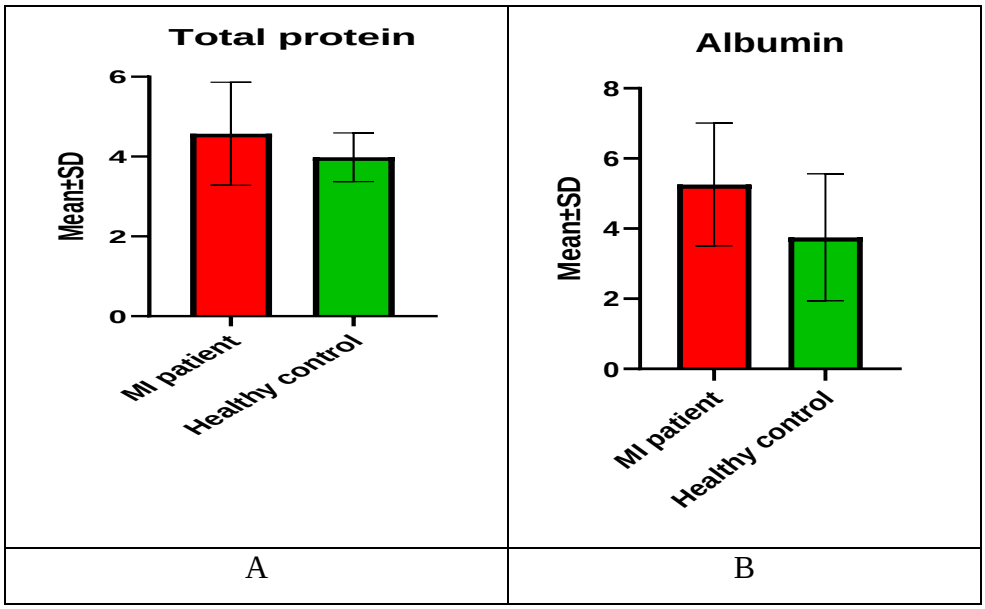

Figure 5. (A) Mean and standard deviation of T.P and(B) Mean and standard deviation of S.ALB.

Diagnostic accuracy of T.P and S.ALB: ROC and AUC evaluations (Table 7, Figure 6) showed that T.P and serum albumin (S.ALB) serve as useful diagnostic tools for distinguishing MI patients from healthy controls. T.P showed 72.55% sensitivity and 80% specificity ($p < 0.05$), while S.ALB achieved 87.5% sensitivity and 70% specificity ($p < 0.001$). These findings indicate that T.P and S.ALB can be reliably employed to diagnose MI, with albumin in particular exhibiting robust diagnostic potential. On the other hand, the T.P p-value under 0.05 suggests moderate diagnostic accuracy.

Correlation between the parameters: The correlation matrix (Table 8) displays Pearson correlation coefficients among various biological markers, age, IMA, DPPH, TAC, T.P, and S.ALB, as an assessment of antioxidant activity. These coefficients vary from -1 to +1, indicating both the strength and direction of any linear relationships. A positive coefficient points to a direct relationship, whereas a negative one signals an inverse correlation. Statistical significance is marked with asterisks. Serum IMA levels were not correlated with DPPH, TAC, T.P, or S.ALB. Nonetheless, there were positive associations found between TAC and DPPH. (0.678, $p < 0.001$), T.P and S.ALB (0.640, $p < 0.05$), and TAC and S.ALB (0.575, $p < 0.001$), suggesting that higher TAC correlates with improved free radical scavenging. Additionally, elevated IMA levels may indicate increased albumin modification resulting from ischemic events. A further finding included a significant inverse correlation between DPPH percentage quenching and protein levels.

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Table 7. Sensitivity and specificity of T.P and S.ALB test.

Variables	AUC	Std. Error[a]	*P* value	Sensitivity %	Specificity %
Total protein	0.714	0.081	< 0.05	72.5 5	80 %
Albumin	0.800	0.097	< 0.01*	87.5 %	70 %

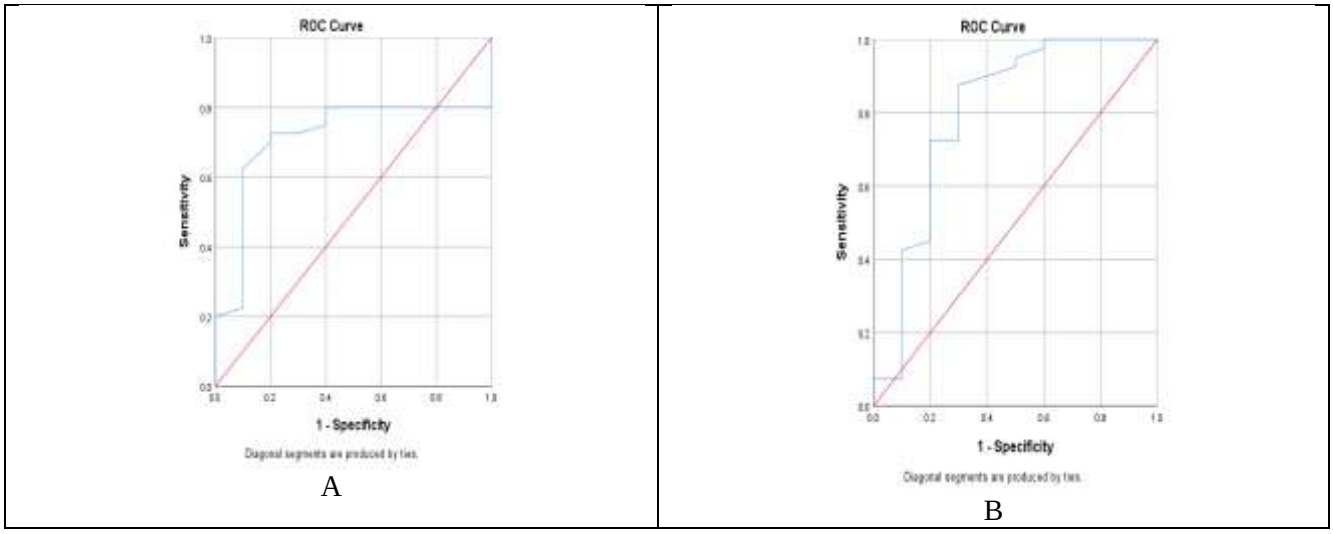

Figure 6. (A)ROC curve of total protein and (B) ROC curve of albumin.

Table 8. The correlation among biomarkers.

Correlations							
		Age	IMA	TP	DPPH	ALB	TAC
Age	Pearson Correlation	1	.031	-.041	-.106	-.056	-.210
	Sig. (2-tailed)		.850	.800	.516	.733	.193
IMA	Pearson Correlation	.031	1	-.221	-.108	-.179	-.091
	Sig. (2-tailed)	.850		.171	.507	.269	.576
TP	Pearson Correlation	-.041	-.221	1	.070	.640**	.305
	Sig. (2-tailed)	.800	.171		.668	.000	.055
DPPH	Pearson Correlation	-.106	-.108	.070	1	.312*	.678**
	Sig. (2-tailed)	.516	.507	.668		.050	.000
ALB	Pearson Correlation	-.056	-.179	.640**	.312*	1	.575**
	Sig. (2-tailed)	.733	.269	.000	.050		.000
TAC	Pearson Correlation	-.210	-.091	.305	.678**	.575**	1
	Sig. (2-tailed)	.193	.576	.055	.000	.000	
** Correlation is significant at the 0.01 level (2-tailed).							
* Correlation is significant at the 0.05 level (2-tailed).							

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Discussion

Research emphasized assessing levels of the new marker, ischemia-modified albumin. The results revealed that IMA values were markedly increased in MI patients compared to healthy individuals, with MI levels at 1050.5 ± 11.7 ng/mL vs. 455.7 ± 133.9 ng/mL in the control group, patients who had an oxidative stress test and CAD, blood IMA showed an increase within 6–10 minutes, remained elevated for around 6–12 hours (20-22), and then back to normal levels after about 12–24 hours (21, 23). Under ischemic conditions, oxidative stress underlies IMA formation. Tissue hypoxia and the switch to anaerobic glycolysis drive acidosis, causing copper-bearing proteins like ceruloplasmin to release copper ions. The conversion of Cu^{2+} to Cu^{+} in the presence of reducing substances promotes the production of superoxide anions. SOD then catalyzes the conversion of $O_2\bullet^-$ into (H_2O_2). When combined with Cu^{2+}, H_2O_2 undergoes the Fenton reaction and yields hydroxyl radicals (•OH). These highly reactive species contribute to structural alterations that result in IMA (24). Numerous studies have indicated that IMA is recognized as an early marker in a wide range of conditions linked to ischemia and oxidative stress. This finding concurs with that of Lee and Wu (2015), who discovered a substantial correlation between IMA levels and MI (25). The N-terminal region of human serum albumin (Aspartic acid1–Alanine2–Histidine3–Lysine4) is highly prone to oxidative stress-induced molecular modification and breakdown. As a result, its ability to bind transition metals, particularly cobalt, diminishes. Ischemia-modified albumin is the name given to this type of albumin (26). Shortly after myocardial ischemia begins, cells experience reduced oxygen, free radical formation, and metabolic disorders, which are followed by disruptions in the Na and Ca ion pumps within cell membranes. Research suggests that ischemia–reperfusion can alter albumin by exposing it to A lack of oxygen in the extracellular space and endothelium, acidic conditions, and injury from free radicals, along with disturbances in ATP-dependent sodium and calcium transport—ultimately leading to the release of free iron and copper ions (27). Research findings suggest that IMA concentrations rise rapidly once ischemia begins and remain elevated for around 6–12 hours thereafter (28). These findings align with earlier work by Ertekin et al. (2013), who highlighted IMA's high sensitivity and specificity as well as its affordability, rapid results, and ease of measurement, which collectively underscore its promise as a potent diagnostic tool in clinical practice, particularly within emergency settings. (29). Table 4 shows that the total antioxidant capacity (TAC) readings were notably different between MI patients and the control group. In MI patients, the mean ± SD of TAC was 16.52 ± 2.44 U/mL; in the control group, it was 30.30 ± 6.68 U/mL. This finding ($p < 0.01$) indicates significantly lower TAC values among those with MI. Consistent with these results, Fazendas et al. reported reduced plasma total antioxidant capacity in MI patients, a decrease that has been considered a potential risk factor for coronary heart disease (30). Nojiri et al. (2001) reported a notably reduced total antioxidant status (TAS) in patients with coronary artery disease (CAD) compared with controls (31). In contrast, Berg et al. (2004) observed elevated TAS levels in two patient groups during percutaneous coronary interventions and coronary angiography (32). Furthermore, multiple investigations have recorded significantly diminished blood antioxidant levels and TAC among individuals with coronary heart disease (CHD). Long-term oxidative stress can deplete the antioxidant defenses, potentially explaining the reduced TAC levels (Opara, Abdel-Rahman et al. 1999; Chrzczanowicz, Gawron-Skarbek et al. 2012) (33). Table 4

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presents the DPPH measurements in MI patients and controls, revealing a mean (± SD) of 22.45 ± 9.54 µmol/L in the MI group and 40.3 ± 11.49 µmol/L in the controls—a significant decrease in the MI group ($p < 0.01$). According to Kedare and Singh (2011), the DPPH assay is one of the most widely used methods to evaluate oxidative stress inhibition (34). Its core principle involves tracking the color reduction of DPPH, measured at 517 nm, where the extent of discoloration indicates antioxidant potency. In line with this mechanistic rationale, the current study employed the DPPH radical-scavenging assay to assess oxidative stress in MI patients. Janaszewska and Bartosz (2002) were the first to apply this assay to human plasma, capitalizing on the stable free radical nature of DPPH (35). Our findings reveal that DPPH levels decline significantly in MI groups, paralleled by a reduction in plasma antioxidant capacity. We observed noticeably higher T.P and serum albumin (S.ALB) levels in MI patients than in controls, with mean values of 4.57 ± 1.29 mg/dL (T.P) and 5.26 ± 1.75 mg/dL (S.ALB), compared to 3.98 ± 0.69 mg/dL (T.P) and 3.75 ± 1.81 mg/dL (S.ALB) in the healthy group ($P < 0.05$). Under ischemic conditions, albumin and other proteins undergo various alterations due to elevated oxidative stress, increased ROS generation, and acidosis. In particular, thiol group modifications can heighten oxidative stress in MI patients (36). Furthermore, protein carbonyl—a key indicator of protein oxidation—was considerably higher among MI patients. It typically forms when proteins interact with lipid peroxidation products or undergo oxidation of amino acid residues, reflecting the extent of ROS-driven protein damage from MI (37).

Conclusion

In summary, our findings showed a strong association between serum IMA, ATC, DPPH, T.P, and S.ALB levels and myocardial infarction. We proposed these markers as potential early diagnostic tools for MI. MI patients exhibited significantly elevated IMA, T.P, and S.ALB levels, alongside markedly lower DPPA and TAC activities, suggesting a pronounced state of oxidative stress combined with insufficient antioxidant defenses. It further explores the role of these molecules in MI differentiation to detect ACS earlier. Another objective is to evaluate IMA's diagnostic capacity, particularly in conjunction with additional biomarkers, for identifying low-risk patients who may be experiencing AMI.

Source of funding: No source of funding.

Ethical clearance: This study received approval from the Ethics Committees of Baquba Teaching Hospital and the University of Diyala. Written informed consent was obtained from each participant prior to enrollment, and all procedures were conducted in accordance with the principles outlined in the Helsinki Declaration.

Conflict of interest: None.

Use of Artificial Intelligence (AI): The authors state they did not use any generative AI tools for creating or editing the manuscript's language.

Acknowledgments: The authors would like to thank the staff of Baquba Teaching Hospital for their support and cooperation. We also express our gratitude to all participants and laboratory personnel for their valuable contributions to this study.

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ORIGINAL RESEARCH
Published: 25 April 2026
DOI: 10.26505/djm.v30i1.1357

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Diyala Journal of Medicine

ORIGINAL RESEARCH
Published: 25 April 2026
DOI: 10.26505/djm.v30i1.1357

التشخيص المبكر لاحتشاء عضلة القلب باستخدام الألبومين المعدّل بنقص التروية ومؤشرات مضادات الأكسدة

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الملخص

الخلفية: يؤدي احتشاء عضلة القلب والإجهاد التأكسدي إلى إحداث ضرر في نسيج عضلة القلب، مما ينتهي بنخر الخلايا. قامت الدراسة بقياس الألبومين المعدّل بنقص التروية (IMA)، والسعة الكلية لمضادات الأكسدة (TAC)، واختبار DPPH، والبروتين الكلي، وألبومين المصل لدى مرضى احتشاء عضلة القلب.

الأهداف: تحليل وتقييم الفائدة التشخيصية لتركيز IMA لدى المرضى الذين تظهر عليهم علامات محتملة لاحتشاء عضلة القلب خلال الست ساعات الأولى.

المرضى والطرق: خلال الفترة من أيلول ٢٠٢٤ إلى شباط ٢٠٢٥، تم إشراك ثمانين مريضًا مشخصين باحتشاء عضلة القلب (MI) والراقدين في مستشفى بعقوبة التعليمي في العراق ضمن هذه الدراسة. بالإضافة إلى ذلك، تم تضمين عشرين شخصًا سليمًا كمجموعة ضابطة. تم قياس IMA باستخدام عدة ELISA، في حين تم قياس TAC وDPPH والبروتين الكلي وألبومين المصل باستخدام الطرق الطيفية (الامتصاصية).

النتائج: أظهرت مجموعة مرضى احتشاء عضلة القلب ارتفاعًا معنويًا في تركيز IMA في المصل مقارنةً بالأصحاء ($p < 0.001$). كما أظهر المرضى انخفاضًا في النشاط المضاد للأكسدة وتراجعًا في مستويات جزيئات مضادات الأكسدة. ولم تُلاحظ علاقة واضحة بين IMA في المصل وكل من DPPH وTAC والبروتين الكلي وألبومين المصل. ومع ذلك، ظهرت علاقات ارتباط موجبة بين TAC وDPPH (0.678، $p < 0.001$)، وبين البروتين الكلي وألبومين المصل (٠,٦٤٠، $p < 0.001$)، وكذلك بين TAC وألبومين المصل (٠,٥٧٥، $p < 0.001$).

الاستنتاج: هدفت الدراسة إلى توضيح الدور المهم لقياس IMA وTAC وDPPH والبروتين الكلي وألبومين المصل لدى مرضى احتشاء عضلة القلب، إلى جانب مؤشرات الإجهاد التأكسدي الأخرى، بما يسهم في توجيه تطوير علاجات مضادة للأكسدة موجهة للتشخيص المبكر.

الكلمات المفتاحية: احتشاء عضلة القلب، IMA، الإجهاد التأكسدي، مضادات الأكسدة، نقص التروية.

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تاريخ الاستلام: ١٤ اذار ٢٠٢٥

تاريخ القبول: ٢٣ أيار ٢٠٢٥

تاريخ النشر: ٢٥ نيسان ٢٠٢٦

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