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ORIGINAL ARTICLE

A Comparative Biochemical Study of Oxidative Stress and Lipid Profiles in Patients with Acute Myocardial Infarction

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ABSTRACT

This study aimed to evaluate oxidative stress and lipid profiles in diabetic and non-diabetic acute myocardial infarction (AMI) patients. A cross-sectional analysis of clinical and laboratory data was conducted at Al-Hussein Educational Hospital in Al-Muthanna, Iraq, in collaboration with the College of Science at Al-Muthanna University. Data were collected for 84 subjects divided into three groups: non-diabetic AMI (G_{MI}), diabetic AMI ($G_{MI\text{-}DM}$), and diabetic (G_{DM}). Biochemical parameters measured included malondialdehyde (MDA), ceruloplasmin (Cp), total cholesterol (Cho), triglycerides (TG), low-density lipoprotein (LDL), high-density lipoprotein (HDL), and very low-density lipoprotein (VLDL), along with the patients' age and gender. MI, MIDM, and DM groups exhibited high levels of MDA, Cho, TG, LDL, and VLDL, with low levels of Cp and HDL compared to healthy controls. The study demonstrated increased oxidative stress and dyslipidemia in MI patients compared to controls, regardless of diabetes status.

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1- INTRODUCTION

Acute myocardial infarction continues to be a major cause of mortality and morbidity. Coronary artery disease, a key factor in myocardial infarction and heart failure, accounts for most of the mortalities in the world [1, 2, 3]. AMI is linked to coronary artery obstruction, which causes myocardial ischemia, resulting in myocardial necrosis and production of reactive oxygen species (ROS) [4].

It was well-reported that risk factors include diabetes, dyslipidemia, hypertension, obesity, smoking and family history are associated with development of AMI [5, 6]. Generally acute myocardial infarction occurs when coronary blood flow is suddenly reduced due to thrombotic blockage in a coronary artery that was narrowed by atherosclerosis, leading to improper heart functioning due to in adequate blood flow. Also, AMI occurs when a coronary artery thrombus forms rapidly at the site of vascular damage [7]. Fatty deposits also called plaques are formed when cholesterol and fats accumulated at the inner wall of the arteries. Exposure to substances that promote platelet and thrombin activation, as well as rupture of the lipid plaques result in thrombus. This condition causes an imbalance between oxygen supply and demand, and if this imbalance becomes severe, it results in MI or heart attack [8, 9]. ROS centrally affect myocardial homeostasis.

When ROS are generated in the heart, they can cause contractile dysfunction and structural damage. As a result, ROS may play significant role in the development and aggravation of heart failure [10]. ROS can be produced from several sources, such as the activation of vascular NADPH phosphate oxidase, xanthine oxidase, inducible NO synthase, auto-oxidation of catecholamines, increased levels of angiotensin II and aldosterone, and released proinflammatory cytokines [11]. Proteins are the first biological molecules to experience oxidative damage in cells [12]. Since, phospholipids and proteins are the major components of membranes, therefore, the alterations

in membrane proteins caused by free radicals are involved in the development of myocardial damage. Moreover, accumulation of ROS causes lipid membranes peroxidation and compromises membrane integrity, ultimately leading to necrosis and cell death [13]. Oxidative stress condition occurs when there is an imbalance between ROS generation and body's antioxidant defenses, which in turn causes modifications to cellular structures and damage of macromolecules, membranes, proteins, and DNA [14]. In contrast antioxidants are chemicals whose presence in relatively high quantity significantly reduces the rate of oxidation of lipids, proteins, carbohydrates and DNA [15]. Oxidative stress is a key factor in the development of a variety of cardiovascular disorders such as atherosclerosis, cardiomyopathy, myocardial injury and heart failure [16, 17]. Increased oxidative stress and reduced antioxidant defense are known to play significant role in the pathophysiology of cardiovascular diseases [18]. In addition, oxidative damage of enzymes is the most common effect of elevated oxidative stress which eventually contributes in the cells damage and thereby stimulating apoptosis and necrosis [13, 19].

Malondialdehyde (MDA), one of the lipid peroxidation markers is produced when oxygen based free radicals attack and degrade polyunsaturated fatty acids [20]. MDA is a well-known marker for oxidative stress of coronary artery disease [21, 22]. It was reported that AMI patients had increased MDA levels (three fold greater) compared to a control group [23]. The harmful effect of ROS is balanced by the non-enzymatic antioxidant action as well as enzymatic antioxidants [24]. As demonstrated by recent literatures, the atherosclerosis risk factors (hypertension, diabetes, dyslipidemia, smoking, etc.) were found to be associated with oxidative stress. In addition to LDL, plasma lipids are also susceptible to oxidation under oxidative stress [25]. In diabetic patients, the production of ROS increased in hyperglycemia via auto-oxidation of glucose, proteins, and lipoproteins [26]. The incidence of cardiovascular diseases (CVDs) as well as induced mortality was reported to be increased in diabetic subjects compared to healthy subjects [27, 28]. The pathophysiology underlying diabetes is not yet fully understood. However, recent studies highlighted the key role of free radicals, alterations in the composition of some serum proteins, inflammations in the development of diabetes [29, 30].

Increased oxidant-based stress has been involved in the pathogenesis of DM. Protein glycation induced by hyperglycemia facilitates the generation of superoxide free radicals, as a result the levels of plasma peroxide in diabetics are higher than those of non-diabetics [31]. Several studies have shown that antioxidants function is diminished in individual with diabetes, which may further exacerbate the pathogenesis of AMI [32, 33].

Additionally, other studies have indicated that DM reduces the availability of nitric oxide (NO) which plays a crucial role in maintaining the antioxidative capacity of endothelial cells [34]. Ceruloplasmin (Cp) is reported to be a key component of the antioxidant defense system in human plasma. It is a plasma glycoprotein primarily created in the liver and released into the bloodstream. Cp enables the incorporation of iron into transferrin without generating toxic iron byproducts. The crucial role of Cp is to transfer the generated electrons from reducing a variety of substrates with concomitant reduction of O_2 to H_2O, without liberating of harmful intermediates (ROS) [35]. The main physiological function of Cp is facilitating plasma redox reactions. In these reactions, oxygen is directly reduced to water [36]. CP also plays a crucial role in controlling membrane lipid oxidation, likely by directly oxidizing cations, thereby preventing them from catalyzing lipid peroxidation. Therefore, elevated plasma CP levels could indicate abnormally high oxidative stress [37]. The aim of this study was to assess the oxidative stress status in MI patients by measuring markers of free radical production and evaluate the antioxidant protection against ROS, in addition to evaluation the major risk factors, like lipid profiles and conditions such as diabetes mellitus.

2- MATERIALS AND METHODS

2.1 Study Design

The present study was conducted at Al-Hussein Educational Hospital in Al-Muthanna, Iraq, in collaboration with the College of Science at Al-Muthanna University, from January 2024 to July 2024. Among the 84 patients, aged 41–85 years, 40 were men (aged 51–85 years) and 44 were women (aged 41–81 years). There were 48 patients under the age of 60 (aged 41–59 years) (Age 1) and 36 patients aged 60 or older (aged 60–85 years) (Age 2). Participants were randomly selected from individuals who had undergone fasting serum lipid tests at Al-Hussein Educational Hospital. The study included a control group (G_H) of 28 apparently healthy individuals (aged 38–75 years), with 14 men (aged 38–71 years) and 14 women (aged 39–75 years). Additionally, three experimental groups were formed, each consisting of 28 subjects: one with acute myocardial infarction (G_{MI}), one with diabetes mellitus (G_{DM}) and one with both diabetes mellitus and acute myocardial infarction ($G_{MI\text{-}DM}$). The study protocol was approved by the Ethical Committee of the College of Science, Al-Muthanna University. Information about the demographic characteristics of the study population, including the mean age, is presented in Table 1.

Table (I): Demographic distributions of patients with AMI, MI-DM, DM and Healthy Control Groups

Group	Main group		Males		Females		Age 1		Age 2	
	n	Mean±SD	*n*	Mean±SD	*n*	Mean±SD	*n*	Mean±SD	*n*	Mean±SD
G_{MI}	28	58.8±11.5	14	59.6± 3.3	14	57.7±9.2	13	50.8±5.4	15	69±8.7
G_{MI-DM}	28	60±10.29	16	60.7±11.9	12	59.2±8.7	16	52.6±4.5	12	69.9±6.6
G_{DM}	28	57.8±11.9	12	60.7±11.2	16	56±12.4	15	50.8±8.5	13	68.9±7.2
Healthy	28	53.9±11.3	14	49±10.8	14	55.2±11.6	12	43.5±4.4	16	64.3±3.5

2.2 Analytical methods

2.2.1 Lipid peroxidation (MDA levels)

The serum MDA level was determined using a spectrophotometric method. Briefly, 150 µL of serum sample was mixed with 1 mL of 17.5% trichloroacetic acid (TCA) and 1 mL of 0.66% thiobarbituric acid (TBA). The mixture was incubated in boiling water for 15 minutes and then allowed to cool. After cooling, 1 mL of 70% TCA was added, and the mixture was left to stand at room temperature for 20 minutes. It was then centrifuged at 2000 rpm for 15 minutes, and the supernatant was collected for spectrophotometric measurement at 532 nm [38].

2.2.2 Antioxidant biomarker (ceruloplasmin)

The level of the antioxidant biomarker was measured using manual methods. Ceruloplasmin concentration was determined using a colorimetric method described by Houchin *et al* [39]. In this procedure, 100 µL of serum was added to 1 mL of freshly prepared para-phenylenediamine (PPD) solution dissolved in acetate buffer at 37°C. After 15 minutes of incubation at 37°C, the tubes were placed in an ice bath for 30 minutes. To stop the reaction, 5 mL of sodium azide solution (prepared by dissolving 100 mg of sodium azide in 500 mL) was added to the mixture. The absorbance of both the test and blank samples was measured using a spectrophotometer (Analytik Jena, Specord 200 Plus, and Germany) at 525 nm.b.

2.2.3 Lipid profiles

Serum Cho, TG, HDL, LDL and VLDL concentrations were analyzed using Monarch-240 Biorex spectrophotometer (UK). These assays were carried out within 2 hours after blood was drawn.

2.3 Statistical analysis

Statistical analysis was performed using Microsoft Excel 2010. Data were expressed as means ± standard deviation (SD). Student's t-test was applied to evaluate the mean difference in demographic and biochemical characteristics between patient groups. Descriptive statistics analyzed by using one-way analysis of variance (ANOVA) test was applied to evaluate the mean difference of the data among three groups. Significant variation is considered when P-values are ≤ 0.05.

3- RESULTS AND DISCUSSION

3.1 Lipid peroxidation and antioxidant

Lipid peroxidation is a natural part of normal metabolism. However, increased lipid peroxidation is considered a consequence of oxidative stress. Our results demonstrated an increase in lipid peroxidation levels in patients of AMI, MI-DM and DM compared to the healthy control group. As shown in Table 2, the plasma lipid peroxide (MDA) levels in G_{MI} patients and their subgroups, classified by sex and age, were significantly higher($P<0.05$) than those in the corresponding G_{MI-DM}, G_{DM}, and G_H groups and their subgroups except for the aged 60 years and above. However, the differences in MDA concentrations among the G_{MI-DM}, G_{DM}, and G_H groups and their respective subgroups, based on sex and age, are not statistically significant. Although the results indicate an increase in MDA levels in these three groups and their subgroups compared to the corresponding healthy control groups, this increase did not reach statistical significance. For the Age2 subgroups, the results showed no significant differences in MDA levels between G_{MI} and G_{MI-DM}, or between G_{DM}, and G_H. However, there was a significant increase in MDA levels in G_{MI} and G_{MI-DM} compared to G_{DM}, and G_H. The findings align with other studies reporting a significant increase in the lipid peroxidation products (MDA) in the blood of AMI patients [7].

Table (2): Comparison of lipid peroxidation products among the control group and patients with AMI, MI-DM, and DM.

Group	MDA levels (µMol/L) mean±SD				LSD
	G_{MI}	G_{MI-DM}	G_{DM}	G_H	
Main group	9.8±4.7[a]	5.2±3.7[b]	6.1±3.4[b]	4.5±1.3[b]	2.1
Males group	7.7±6.1[a]	3.2±1.6[b]	4.6±1.6[b]	4.4±1.1[b]	3.0
Females group	11.1±3.3[a]	6±2.7[b]	5.2±2.1[b]	4.6±1.5[b]	2.3
Age1 group	11.1±5.4[a]	3.7±2.4[b]	6.7±4.2[b]	4.1±1.2[b]	3.0
Age2 group	8.9±2.5[a]	7.3±4.1[a]	4.7±2.2[ab]	5.2±1.2[ab]	2.6

Superscripts (eg: a, b, ab) indicate statistical comparisons among groups

On the contrary, the results indicated that plasma Cp levels, a marker of antioxidation, increased significantly in G_{DM} and its subgroups as compared to G_{MI}, G_{MI-DM}, and G_H, and their respective subgroups, with the exception of the group aged 60 years and above (Age2), where the increase in Cp levels in G_{DM} did not reach statistical significance as compared to G_{MI} group. Recent studies have demonstrated an association between increased oxidative stress and diabetes which are well known risk factors for atherosclerosis [40]. The present study found that patients with AMI, MIDM and DM exhibited either significant or non-significant increase in MDA levels, a marker of lipid peroxidation, and elevation of Cp levels, an enzymatic antioxidant defenses marker, compared to healthy controls. The rise in MDA levels likely reflect increased ROS production during ischemic/reperfusion events associated with AMI. On the other hand, Cp may act as a protective response to the increased levels of unbound Fe^{+2}, which can catalyze further free radical-induced lipid peroxidation through the formation of hydroxyl radicals ($OH^{\bullet}$) from H_2O_2. Antioxidants are the first line of defense against free radical toxicity, and it is well-established that plasma antioxidant capacity declines with the oxidative/antioxidative balance shifting toward oxidative stress in patients with AMI. Our findings are consistent with previous studies that report elevated lipid peroxidation and plasma Cp levels in individuals with DM [41]. Notably, our study observed increased lipid peroxidation (MDA) and diminished antioxidant enzymatic defenses (Cp) in the AMI group and its subgroups compared to the DM and healthy groups (Table 3). However, the changes in Cp levels were less pronounced than the increase in MDA levels. This observation may be attributed to the fact that the damage caused by AMI is more severe than caused by DM. It highlights significant impairment of the antioxidant system in the pathogenesis of AMI, which appears inadequate in combating the heightened oxidative stress.

Table (3): Comparison of Ceruloplasmin Levels among the control group and patients with AMI, MI-DM, and DM.

Group	Cp levels (mg/dL) mean±SD				LSD
	G_{MI}	G_{MI-DM}	G_{DM}	G_H	
Main group	37.2±15.7[b]	36±11.2[b]	48.9±12.7[a]	32±7.5[b]	7.4
Males group	30.9±16[b]	33.3±9.9[b]	44.8±10[a]	30.8±6.9[b]	10.7
Females group	45.8±11.7[a]	38.9±12.4[ab]	52.3±12.7[a]	32.2±6.9[ab]	10.2
Age1 group	40.4±16.4[a]	37.3±12.4[ab]	49±12.1[a]	32.3±7.4[ab]	10.3
Age2 group	33.3±16.8[b]	34.2±9.8[b]	51±6.9[a]	31.7±8.1[b]	10.7

Legend as in Table 1

3.2 Lipid profile

The results indicated that the differences in Cho levels among G_{MI}, G_{MIDM} and G_{DM} were not significant, whereas the GH group showed a marginally significant difference (Table 4). However, the G_{MI} groups exhibited the highest mean Cho levels, while the G_H groups had the lowest values. Notably, females and older patients (Age2 group) demonstrated distinct pattern compared to males and younger patients (Age1 group). Specifically, Cho levels in the G_{MI} group were significantly higher in females and the Age2 group compared to other groups, whereas this elevation was not significant in males or the Age1 group. Additionally, the results indicated to significant decrease in Cho levels observed in certain conditions like GMIDM, GDM and GH, particularly in females and older individuals. This suggests that gender, age and specific health conditions are significantly influence Cho levels in patients with AMI. Our findings align with previous research, which

reported significantly higher total Cho levels ($p < 0.05$) in AMI patients with and without DM compared to the control group [16].

Table (4): Comparison of Cholesterol Levels among the control group and patients with AMI, MI-DM, and DM.

Group	Cho. levels (mg/dL) mean±SD				LSD
	G_{MI}	G_{MI-DM}	G_{DM}	G_{H}	
Main group	202.2±51.2[a]	172.1±67.6[a]	184.6±47.9[a]	168.4±25.3[ab]	30.9
Males group	191.2±44. 6[a]	178.6±55.7[a]	172±23.9[a]	163.2±32.4[a]	39.3
Females group	214.6±56.6[a]	158±84.4[b]	160.9±49[b]	161.2±25[b]	52.4
Age1 group	197.3±57.5[a]	196.3±68.5[a]	175.3±24.1[a]	172.6±44.7[a]	42.3
Age2 group	213.7±37.7[a]	137.4±52.3[b]	168.9±34.1[b]	159.2±25.2[b]	37.1

Legend as in Table 1

As is well known, an increase in plasma lipid concentration is frequently observed in patients with AMI, contributing to the development of vascular disease. In this study the findings indicated that, except for participants under 60 years (Age1 group) there was no statistical significant difference in the mean serum levels of TG and VLDL among patient groups. However, these levels were significantly elevated when compared with those of a healthy control group. In the Age1 group, as detailed in Table 5, TG and VLDL levels were notably higher in the G_{MI-DM} group than in the other groups. Our findings revealed a significant increase of TG and VLDL levels in AMI patients compared to healthy controls, regardless of age. This supports the understanding that lipid metabolism abnormalities are a key factor in AMI, emphasizing the importance of lipid profile monitoring in cardiovascular risk assessments, particularly among older adults.

Table (5): Comparison of Triglyceride and Very Low Density Lipoprotein Levels among the control group and patients with AMI, MI-DM, and DM.

Biochemical parameter	Group	(mean±SD)				LSD
		G_{MI}	G_{MI-DM}	G_{DM}	G_{H}	
TG (mg/dL)	**Main group**	243.2±114.2[a]	264.7±183.6[a]	190.6±115[a]	127.5±64.1[ab]	77.7
	Males	288.4±123.9[a]	285.9±118.3[a]	185.7±116[a]	142.2±67.6[ab]	104.7
	Females	203.6±83.9[a]	280.2±234.5[a]	160.4±79[a]	126.6±58.9[ab]	121.5
	Age1 group	224.8±121.4[b]	352.7±181.5[a]	143.3±55[b]	154.8±59.5[b]	95.9
	Age2 group	233.3±115.2[a]	147.4±108.8[a]	179.4±106[a]	91.1±53.1[ab]	95.2
VLDL (mg/dL)	**Main group**	48.6±22.9[a]	52.8±36.7[a]	38.9±23[a]	25.5±12.8[ab]	15.6
	Males	53.3±23.5[a]	50.1±26.4[a]	37.1±21.1[a]	28.4±13.5[ab]	21
	Females	39.1±16.8[a]	56.1±43.9[a]	32.1±20.5[a]	25±11.8[ab]	23.1
	Age1 group	47.1±23.2[b]	70.5±36.3[a]	30.3±12.7[b]	31±11.9[b]	19.4
	Age2 group	43.5±23.4[a]	29.5±21.8[a]	34.9±20.8[a]	20.3±10.9[ab]	19.9

Legend as in Table 1

It is well-known that high levels of LDL are associated with an increased risk of cardiovascular diseases, such as heart attacks and strokes. Elevation LDL levels lead to excess Cho building up in the walls of arteries, causing atherosclerosis, which can eventually result in AMI. Table 6 demonstrated a smaller difference in means of LDL across all patients and control groups and subgroups, which is statistically not significance. This indicated that LDL levels are relatively consistent in most patient groups and subgroups, although they remain higher than those in the respective control groups. However, these elevations were not statistically significant. Our findings revealed that the highest LDL levels in the main patient groups were noted in the G_{DM}, whereas, the most pronounced elevations were noted in all subgroups belonging to G_{MI}. The results also showed a marginally

significant decrease in LDL levels in the GH subgroup of females compared to other groups. Further explanation and investigation are needed to clarify this observation. Notably, our findings suggest that reduction of LDL levels from GMI to GH in younger patients (Age1) was relatively more pronounced than in older patients Age2.

Table (6): Comparison of Low Density Lipoprotein Levels among the control group and patients with AMI, MI-DM, and DM.

Group	LDL levels (mg/dL) mean±SD				
	G_{MI}	G_{MI-DM}	G_{DM}	G_H	LSD
Main group	89.2±35.6[a]	77.6±36.8[a]	91±27.4[a]	72.1±28.2[a]	19.8
Males group	90.2±30.8[a]	85.6±38.9[a]	76±19.1[a]	67.6±32.4[a]	29.9
Females group	95.3±40.6[a]	72.6±39.4[a]	83.4±32.5[a]	62±23.4[ab]	31.4
Age1 group	87.7±36.5[a]	82±44.6[a]	86±29.2[a]	73.6±25.7[a]	20.2
Age2 group	92.8±37.8[a]	71.8±24.1[a]	76.7±16.1[a]	70±32.7[a]	27.7

Legend as in Table 1

High-density lipoprotein (HDL) is a protein-rich lipoprotein known for its protective role, primarily due to its active involvement in the reverse transport of Cho from extra hepatic tissues to the liver for biliary excretion [42]. A decrease in HDL levels is the most common lipoprotein abnormality observed in patients with AMI and DM. Notably a significant reduction in HDL in these patients serves as a predictive marker for future disease events, even when total cholesterol levels are within the normal range [43]. In our study (Table 7), the mean serum HDL levels showed either a significant or non-significant decrease in patients with AMI, MI-DM and DM compared to healthy control subjects. Additionally, we observed elevated levels of Cho, LDL, TG and VLDL, along with decreased HDL levels in MI patients with and without DM compared to the control group. These findings are consistent with previous studies, which also reported significant increase the concentrations of Cho, TG, LDL and VLDL, along with significant lowering HDL levels compared to the control group among diabetics [42]. However, they differ from other reports, which noted that changes in HDL concentration were not significant in patients without DM or dyslipidemia and in those with DM compared to controls [16].

Table (7): Comparison of High Density Lipoprotein Levels among the control group and patients with AMI, MI-DM, and DM.

Group	HDL levels (mg/dL) mean±SD				
	G_{MI}	G_{MI-DM}	G_{DM}	G_H	LSD
Main group	54.8±19.0b	49.4±21.8b	58±26.7b	71.3±12.3a	12.67
Males group	47.9±12.8ab	47.8±12.7ab	58.8±30.9a	67.8±11.4a	18
Females group	62.3±21.3a	51.8±29.9ab	48.9±17.8ab	74.2±13.1a	19.42
Age1 group	53.3±20.5ab	60.8±20.4a	60.8±29a	71.3±10a	17.36
Age2 group	56.4±18.3a	55.8±23.1a	56.6±29a	71.3±15.5a	21.2

Legend as in Table 1

Our study examined the effect of gender on biochemical parameters in patients with MI, MI-DM and DM. The results are presented in Table 8. The patients were divided into two subgroups: male and female. The study demonstrated that gender differences were evident in oxidative stress markers, as the mean Cp values in the female groups of G_{MI} and G_{DM} were significantly higher than those in the corresponding male groups. A similar trend was observed in the G_{MI-DM} group, where mean Cp values were higher in females than in males; however, this difference did not reach statistical significance. Notably, the mean MDA values in the female groups across all patient categories were higher than in the corresponding male groups, but this difference was not statistically significant. These findings demonstrated that the pathological condition in AMI is responsible for increase oxidative stress levels and the mobilization of the antioxidant enzymatic system.

Table (8): The effect of gender on oxidative stress in patients with MI, MI-DM and DM.

Biochemical parameter	Group	Mean±SD		
		Male	Female	P value
MDA (µMol/L)	G_{MI}	8.34±5.01	11.13±3.16	0.12
	G_{MI-DM}	4.55±4.38	5.97±2.68	0.39
	G_{DM}	4.48±2.01	6.12±3.53	0.1
Cp (mg/dL)	G_{MI}	28.29±14.75	47.18±11.99	0.002
	G_{MI-DM}	33.36±9.83	38.9±12.40	0.27
	G_{DM}	43.25±10.35	53±10.97	0.01

Regarding the lipid profile, our study showed a non-significant fluctuation in the mean serum lipid values between male and female patient groups, presented in Table 9. Specifically, we observed a non-significant decrease in Cho and LDL in G_{MI}, and in TG and VLDL in G_{MI-DM}, as well as in Cho, TG, LDL and VLDL in G_{DM} for males compared to females. Conversely, there was a non-significant increase in other lipid parameters in males compared to females. These contrasting findings prevented us from conclusively assessing the effect of gender on dyslipidemia as a risk factor in patients with MI and DM. However, the study highlighted an increase level of HDL in females compared to males across all patient groups, suggesting that women may be less susceptible to AMI than men. Additionally, the strong effect of estrogen in females likely provides a protective effect on heart health. Our results align with previous studies suggesting that HDL appears to be a key lipid risk factor in patients presenting with AMI.

Table (9): The effect of gender on lipid profile in patients with MI, MI-DM and DM.

Biochemical parameter	Group	Mean±SD		
		Male	Female	P value
Cho. (mg/dL)	G_{MI}	198.14±48.08	211.91±54.48	0.5
	G_{MI-DM}	171.64±52.28	170.5±84.37	0.97
	G_{DM}	174.75±33.83	177.37±51.87	0.9
TG (mg/dL)	G_{MI}	266.64±117.54	195.55±83.98	0.1
	G_{MI-DM}	250.64±131.97	280.2±234.53	0.73
	G_{DM}	168.5±105.52	178.58±102.6	0.8
LDL (mg/dL)	G_{MI}	83.14±28.5	97.18±39.04	0.31
	G_{MI-DM}	82.18±35.57	72.6±39.37	0.56
	G_{DM}	82.25±24.5	86.32±27.53	0.6
HDL (mg/dL)	G_{MI}	49.57±15.28	62±20.18	0.09
	G_{MI-DM}	47.18±11.50	51.8±29.93	0.66
	G_{DM}	56.25±27.79	59.89±26.67	0.7
VLDL (mg/dL)	G_{MI}	53.33±23.51	39.11±16.8	0.25
	G_{MI-DM}	50.13±26.39	56.1±43.94	0.69
	G_{DM}	33.7±21.10	35.72±20.52	0.8

The study also evaluated the association between age and oxidative stress, as a risk factor. A notable change in MDA levels was observed in the G_{MI-DM} group, where the mean MDA levels in older patients were significantly higher than in younger patients, as presented in Table 10. Although the difference in mean Cp levels between these two groups was not significant, the findings suggest that oxidative stress increases with age in this group. However, in the G_{MI} and G_{DM} patient groups, oxidative stress did not appear to be significantly affected by age, as no significant differences were observed in the mean MDA and Cp levels between younger and older groups.

Table (10): The effect of age on oxidative stress in patients with MI, MI-DM and DM.

Biochemical parameter	Group	Mean±SD		
		Age1	Age2	P value
MDA (µMol/L)	G_{MI}	10.16±5.58	8.82±2.42	0.43
	G_{MI-DM}	3.68±2.43	7.28±4.11	0.02
	G_{DM}	6.15±3.52	4.43±1.99	0.09
Cp (mg/dL)	G_{MI}	39.93±16.2	32.36±16.44	0.26
	G_{MI-DM}	37.33±12.39	34.22±9.83	0.5
	G_{DM}	49.16±13.33	49.33±8.76	0.97

In examining the impact of age on lipid profiles, our findings revealed notable patterns in the MI-DM patient group, as presented in Table 11. The results showed that younger patients had significantly higher levels of Cho, TG and VLDL compared to elderly patients, with non-significant differences observed in LDL levels. Although HDL levels were lower in younger patients, the difference was not statistically significant. These findings suggest that dyslipidemia may be a more prominent risk factor in younger MI-DM patients compared to their older counterparts. Our study is also in good agreement with similar study that have shown dyslipidemia to be more prevalent in non-elderly male patients compared to their elderly counterparts [44]. In the AMI patient group, our study observed a non-significant decrease in Cho, LDL and HDL levels and a non-significant increase in TG and VLDL levels in younger patients compared to older patients. These mixed results prevent us from drawing definitive conclusion about the impact of age on lipid profile changes and consequently, the role of dyslipidemia as a risk factor in this patient group. For the DM patient group, the data showed a similar downward trend in lipid parameters with age. Specifically, LDL levels decreased significantly in older patients, while declines in other lipid parameters did not reach statistical significance this indicates that dyslipidemia may also pose a risk factor for younger DM patients.

Table (11): The effect of age on lipid profile in patients with MI, MI-DM and DM.

Biochemical parameter	Group	Mean±SD		
		Age1	Age2	P value
Cho (mg/dL)	G_{MI}	193.5±54.86	217.82±42.59	0.24
	G_{MI-DM}	196.33±68.54	137.44±52.30	0.04
	G_{DM}	185.74±50.22	161.5±32.12	0.15
TG (mg/dL)	G_{MI}	235.71±115.66	234.91±103.62	0.98
	G_{MI-DM}	352.67±181.52	147.44±108.82	0.007
	G_{DM}	174.95±103.75	174.25±103.99	0.99
LDL (mg/dL)	G_{MI}	86.64±34.33	92.73±33.85	0.66
	G_{MI-DM}	82±44.61	71.78±24.13	0.5
	G_{DM}	92.16±28.61	73±16.32	0.02
HDL (mg/dL)	G_{MI}	52.5±19.45	58.27±17.14	0.44
	G_{MI-DM}	44.58±20.42	55.78±23.1	0.25
	G_{DM}	61.37±25.71	53.92±28.74	0.46
VLDL (mg/dL)	G_{MI}	47.14±23.19	43.55±23.44	0.24
	G_{MI-DM}	70.53±36.3	30.67±20.97	0.008
	G_{DM}	34.99±20.75	34.85±20.8	0.84

4- CONCLUSION

The findings of our study confirm that oxidative stress is higher in AMI patients than in those with MI-DM and DM, regardless of patient sex and age. This suggests significant impairment of the antioxidant system in the pathogenesis of AMI, rendering it unable to effectively contract oxidative stress. Furthermore, the damage caused by AMI appears to be more severe than that caused by DM. The data indicate that women with AMI may be less susceptible to oxidative stress as a risk factor compared to men. Similarly, women with AMI may also be less affected by dyslipidemia as a risk factor than men. However, the contradictory findings regarding lipid profiles and age prevent us from making definitive conclusions about the impact of age on lipid profile changes and, consequently, on the role of dyslipidemia as a risk factor in this patient group.

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