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Original Article

Others Section

Oxidant-antioxidant Status Discrepancy between Patients with ST Segment Elevation and Non ST Segment Elevation Myocardial Infarction: A Case-Control Study

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ABSTRACT

Introduction: Depending on the degree of coronary artery occlusion and the damage to the myocardium, two types of myocardial infarction can be distinguished: non ST Elevation Myocardial Infarction {NSTEMI type (without ST segment elevation)} and ST Elevation Myocardial Infarction {STEMI type (with ST segment elevation)}. Consequently, this may lead to diverse levels of Reactive Oxygen Species (ROS) and dissimilar oxidative profile between the two conditions.

Aim: To assess the oxidant-antioxidant status in STEMI versus NSTEMI patients and healthy controls.

Materials and Methods: This case-control study was carried out from September 2018 to March 2019. A total of 67 Acute Myocardial Infarction (AMI) patients, categorized into two groups: those with STEMI (Group 1) and those with NSTEMI (Group 2), were enrolled into the study, and were compared with 75 healthy controls. Oxidant-antioxidant markers, lipid profile and uric acid were quantified for all participants. Statistical comparison between all groups was performed by one-way Analysis of Variance (ANOVA) test followed by Tukey post-hoc test.

Results: The findings showed a significant increase in the level of oxidative markers as determined by serum Malondialdehyde (MDA), Carbonyl Proteins (CP), Nitric Oxide (NO•) and superoxide radicals (p-value<0.001) in AMI patients particularly in those with STEMI. Concomitantly, there was a notable decrease in the level of antioxidants such as Glutathione (GSH) and catalase (p-value<0.001). A noteworthy raise was also noticed in the serum uric acid concentration in patients with AMI as compared to healthy subjects. However, no statistically significant difference between the two cases groups was revealed.

Conclusion: The magnitude of imbalance between oxidant and antioxidant markers is greater in STEMI patients, most likely because of the expanse of myocardial necrosis and superior occlusion of their coronary arteries.

Keywords: Acute myocardial infarction, Oxidative stress, Reactive oxygen species, Serum uric acid

INTRODUCTION

The Acute Myocardial Infarction (AMI) is a serious cardiovascular event and one of the most prevailing cause of mortality and morbidity worldwide [1]. AMI can be clinically divided into NSTEMI or STEMI depending on the absence or presence of ST segment elevation on the Electrocardiogram (ECG) [2]. Their pathophysiological genesis and clinical presentations are comparables, but they diverge in severity. STEMI often presents with total thrombotic occlusion in an epicardial coronary vessel and the expanse of myocardial damage or necrosis is prominent. In contrast, patients with NSTEMI disclose a partial vessel narrowing leading to a limited and transitory blockage of the coronary artery [3].

The pathogenesis of AMI is very intricate and not wholly elucidated. The underlying causes are diverse; amongst others, a pivotal role is carried out by the oxidative stress that arises from an excessive production of Reactive Oxygen Species (ROS), overpowering endogenous antioxidant mechanism [4]. Oxidative stress is the major mechanism in development and evolution of atherosclerosis [5]. It has been usually regarded as a contributor to different stages in the syndrome progression and is closely linked to ischaemic myocardial damage and necrosis [6]. Indeed, following ischaemia, ROS are generated during the reperfusion phase [7]. Greater amounts of ROS are produced after a consecutive cascade of thrombotic episodes and atherosclerotic plaque rupture that cause occlusion of the coronary artery; disrupting blood flow and oxygen supply to myocardium and promotes ischaemia of contiguous tissues, leading to cell injury and necrosis. The duration of ischaemic determines the intensity of damage to the myocardium and connected tissues [8].

ROS, such as superoxide anion (O2•-) and nitric oxide (NO•), are highly reactive molecules implicated in cell damage, necrosis and cell apoptosis due to their direct oxidising effects on all types of biological molecules as proteins, lipids and Deoxyribonucleic Acid (DNA) [9]. However, proteins are likely to be the most immediate vehicle to undergo oxidative damage in cells and their side chains can be carbonylated by reactive carbonyl compounds [10]. Protein Carbonyl (PC) is oxidatively modified protein with diagnostic potential for AMI. In patients with AMI assayed within 24-96 hour after the acute event, grand levels of PC have been detected [11].

Likewise, increasing ROS leads to the peroxidation of lipid membranes and loss of membrane integrity, resulting in necrosis and cell death. Lipid peroxidation is reported to play a major pathogenic role in the genesis of coronary atherosclerosis. The best evidence of lipid peroxidation is the increased formation of MDA which is one of the principal breakdown products by the action of endoperoxidase. Many studies have ascertained the elevated level of serum MDA in heart diseases, demonstrating a link between oxidative stress and AMI [12,13].

Multiple antioxidant defensive systems exist to counteract ROS accumulation by scavenging and converting ROS to non toxic molecules. These systems include catalase, Glutathione Peroxydase (GSHPx), and reduced Glutathione (GSH) [14]. Reduced GSH constitutes the most pertinent natural antioxidant. It acts as a scavenger of electrophilic and oxidant species either in a direct way or through enzymatic catalysis, since GSH is the co-substrate of GSHPx and enables the reduction of peroxides [15].

Moreover, catalase is supposed to play a major role in the first line of enzymatic antioxidant defense. It plays a prominent role in maintaining cell membrane integrity by interacting with membrane phospholipids and thereby, limiting lipid peroxidation by ROS [16].

Excessive production of ROS or insufficient antioxidant protection generates a condition known as oxidative stress, which can lead to different degrees of damage [10]. In view of the above established facts, the present study was conducted to assess and compare the levels of oxidative stress markers and antioxidants in STEMI with regard to NSTEMI patients by measuring plasma O2•-, NO•, MDA, carbonyl proteins, catalase, and reduced GSH levels. Changes in serum lipid levels and uric acid were also determined.

MATERIALS AND METHODS

This study was a hospital-based, case-control study carried out from September 2018 to March 2019, in adult patients with a diagnosis of first incident of AMI admitted to the Department of Cardiology, Tlemcen University Hospital Center (Algeria), within 24 hours from symptoms' initiation. Preceding to the beginning of the study ethical approval from Institutional Ethics Committee was acquired (D01N01UN1301202007). Informed consent was obtained from all participants who were recruited after an entire elucidation of the purpose of the study.

Inclusion criteria

For cases: Patients admitted with chest pain relative to first episode of AMI, typical symptoms and ECG signs compatible with AMI and significant rise of serum cardiac enzyme concentration (troponin I) were included.

For controls: Healthy volunteer's subjects with no history of chest pain, no clinical evidence of heart disease and no history of AMI were included as controls.

Exclusion criteria

For cases: Patients with prior history of chronic angina or AMI, valvular disease, heart failure and malignancy were excluded from the study.

For controls: Subjects with history of heart disease or taking medication that could influence oxidative status were excluded from the study.

Sample size calculation: Using power and sample size calculator (Statistical solutions, Sigma), a sample size of 75 controls and 67 AMI patients, with 39 STEMI and 28 NSTEMI was obtained using 80% power of detecting a 20% difference in measurements.

Patients were subdivided according to the presence or absence of ST segment elevation in the initial ECG into the STEMI (n=39) or NSTEMI (n=28) group. Data regarding demographic characteristics, laboratory analysis and clinical profile of patients was collected for all cases.

Procedure

Blood samples: Blood samples were obtained from the antecubital vein using a clean venipuncture under controlled venous stasis within the first 24 hours after hospital admission following overnight fasting in different containers.

Ethylenediamine Tetraacetic Acid (EDTA) vial: A 5 mL of blood was taken. Plasma was separated by centrifugation for 15 min at 3000 rpm and was used for dosage of Nitric Oxide (NO•). The remaining red cells were rinsed with isotonic saline and haemolysed with glacial distilled water, afterward the cell debris were isolated by centrifugation for 10 minutes at 4000 rpm and the haemolysate was recuperated and exploited for assessment of reduced GSH, catalase, Malondialdehyde (MDA), Carbonyl Proteins (CP) and Superoxide Anion (O2•-) levels [17].

Plain vial: Blood was allowed to clot and a serum was separated by centrifugation for 5 minutes at 5000 rpm and used for determination of biochemical parameters.

Biochemical Parameters

Lipid profile: A full fasting lipid profile, comprising of the Total Cholesterol (TC), Triglycerides (TG), and High-Density Lipoprotein (HDL) colorimetric methods using the kits obtained from Bioassay Systems, Hayward, CA, USA was done.

Plasma Low-Density Lipoprotein Cholesterol (LDL-C) was determined from the values of TC and HDL-C via the Friedewald formulae: LDL-C=TC-(TG/5)-HDL-C (mg/dL) [18].

Colorimetric method [19]- Uric acid is oxidised by uricase enzyme to allontoine and hydrogen peroxide, which under the influence of peroxydase, 4-aminophenazone and 2-4 dichloro sulfonate forms a red coloured compound (quinoneimine) quantified spectrophotometrically at 520 nm (SPINREACT kit).

Determination of Oxidant Status

Measurement of O2•-: Erythrocyte superoxide anion was determined by a spectrophotometric method involving the reduction of Nitro Blue Tetrazolium (NBT) in the presence of O2•-. The reaction end product was assayed at 550 nm [20].

Measurement of NO•: Fresh plasma nitric oxide level was evaluated by the technique of Guevara I et al., [21] after deproteinisation using methanol: diethyl ether (3:1, v/v). Nitrite and nitrate levels were measured together nitrate being formerly converted to nitrite by cadmium reduction. Nitrite was measured directly by spectrophotometry at 540 nm, using the colourimetric procedure of Griess.

Measurement of Malondialdehyde (MDA): Erythrocyte MDA intensity, as a main indicator of lipid peroxidation, was estimated by its interaction with Thiobarbituric Acid (TBA). The absorbance of the reaction end product was measured by spectrophotometry at 532 nm [22].

Measurement of Carbonyl Proteins (CP): Erythrocyte CP concentration, as a chief indicator of protein oxidation, was determined by the derivatisation of PC groups with 2,4- Dinitro Phenyl Hydrazone (2,4-DNPH) generating the creation of stable DNPH adducts [23].

Determination of Antioxidant Status

Measurement of Glutathione (GSH): Erythrocyte reduced GSH level was analysed by a colourimetric method [24], founded on the reduction of 5,5′-Dithiobis (2-nitrobenzoic acid) by GSH leading to the development of yellow coloured compound (2-nitro-5-thiobenzoicacid). This reaction product was followed spectrophotometrically at 412 nm (Sigma Aldrich kit; St. Louis, MO, USA).

Measurement of Catalase activity: Erythrocyte catalase activity was measured, via the technique of Aebi [25], through the spectrophotometric assessment of the rate of hydrogen peroxide decomposition at 420 nm. The results are expressed as unit/gram of haemoglobin (U/g Hb).

STATISTICAL ANALYSIS

All statistical analyses were performed using the Statistica software (version 4.1; Stat soft, Paris, France) and values were expressed as Mean±standard deviation (SD). Statistical comparison between all groups was performed by one-way ANOVA test followed by Tukey post-hoc test. All p-values less than 0.05 were considered as statistically significant.

RESULTS

Socio-demographic and clinical data of cases and control groups are mentioned in [Table/Fig-1]. In this study, case group males out

numbered females one by around a 3.2:1 ratio. The high number of AMI was among males (76.1%). In controls, the sex ratio was about 1.1:1.

[Table/Fig-1] shows also that females with AMI were obese (BMI >30) compared with males and controls.

Biochemical analysis: As expected, TC, LDL-C and TG concentrations were significantly superior in cases groups in comparison to the control group, whilst HDL-C levels were lower (p-value <0.001) [Table/Fig-2]. Amongst cases, higher means of lipid profile (TC, LDL-C and TG) were found in STEMI patients compared to those with NSTEMI. Mean HDL-C level was feebly highest in NSTEMI cases without reaching significance. There was statistically significant difference in mean concentrations of LDL-C and TC in both male and female cases whereas TG levels were only significantly elevated in males with STEMI as compared to NSTEMI (p<0.05). With regard to uric acid, it was significantly higher in cases relatively to controls. Whereas STEMI group had higher levels than NSTEMI group, the two patients groups were not found to be significantly different.

Oxidant status: In this study, pattern of increase of all oxidant markers is as follows: STEMI > NSTEMI >Control, with a noticeable elevation in females compared to males in all the study groups [Table/Fig-3]. Compared to healthy controls, AMI patients displayed an extremely significant increase in oxidant markers (p-value <0.001). However, there was no statistical difference in O2•-, NO• levels among patients with STEMI and NSTEMI unlike MDA and CP levels.

Antioxidant status: [Table/Fig-4] illustrate that all MI patients have a significant decrease in erythrocyte GSH and catalase concentrations compared to their respective controls, regardless of gender. When considering separately the two case groups, blood GSH and catalase levels were found significantly inferior (p-value <0.01 and p-value <0.001, respectively) in STEMI groups especially in males.

DISCUSSION

The AMI is the foremost cause of death and disability worldwide. Quite a number of factors take part in the genesis and the progression of AMI. Oxidative stress is a predictable contributor to myocardial infarction, and several previous studies have spotlighted alteration in the oxidant-antioxidant status in patients with AMI [8,16]. This study aimed to investigate the magnitude of changes in oxidant-antioxidant balance in STEMI versus

Groups	Controls (n=75)		Cases (Myocardial Infarction)			
			STEMI (n=39)		NSTEMI (n=28)	
	Males (n=35)	Females (n=40)	Males (n=29)	Females (n=10)	Males (n=22)	Females (n=6)
Age (years)	50±3	48±4	59±13	61±5	62±11	66±9
Weight (kg)	75.94±2.11	65.33±3.14	82.86±7.43	79±14.26	81.5±6.32	77.17±8.84
Height (m)	1.80±0.33	1.68±0.27	1.78±0.04	1.61±0.07	1,77±0.05	1.59±0.06
BMI (kg/m^2)	23.44±0.62	23.15±0.52	25.99±1.86	30.39±3.95	25.95±1.78	30.48±2.44

[Table/Fig-1]: Socio-demographic and clinical characteristics of the study groups.
Values are presented as means±SD; BMI: body mass index (weight/height^2)

Groups	Controls		Cases (Myocardial Infarction)				p-value
			STEMI		NSTEMI		
	Males	Females	Males	Females	Males	Females	
TC (mmol/L)	4.86±0.26	4.44±0.18	5.80±0.41***	5.95±0.38***	5.34±0.27**‡	5.12±0.24**‡	<0.001
HDL-C (mmol/L)	1.52±0.22	1.68±0.27	1.13±0.09**	1.11±0.11**	1.22±0.10**ns	1.23±0.12**ns	<0.01
LDL-C (mmol/L)	2.47±0.21	2.50±0.24	3.53±0.35***	3.61±0.29***	3.03±0.27***†	3.04±0.25*†	<0.001
TG (mmol/L)	1.19±0.15	1.25±0.08	2.55±0.38 ***	2.42±0.25 ***	2.28±0.21***†	2.27±0.19*** ns	<0.001
Uric acid (µmol/L)	257.15±49.48	277.15±46.18	484.48±71.89***	494.24±136.24***	409.83±80.62***ns	439.91±95.65***ns	<0.001

[Table/Fig-2]: Biochemical parameters of the study groups.
TC: Total cholesterol; HDL-c: high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TG: triglyceride; Values are means±SD; Statistical comparison between all groups was performed by one-way ANOVA test followed by Tukey post-hoc test; Cases versus controls:* Significant at p-value <0.05; ** Significant at p-value<0.01; ***Significant at p-value<0.001; STEMI versus NSTEMI: †for p-value<0.05, ‡for p-value<0.01; ns: not significant at p-value >0.0.5

Groups	Controls		Cases (Myocardial Infarction)				p-value (ANOVA)
			STEMI		NSTEMI		
	Males	Females	Males	Females	Males	Females	
O2•- (µmol/L)	2.36±0.33	2.42±0.32	5.15±0.97 ***	5.72±0.69 ***	4.63±1.20***ns	4.95±0.36***ns	<0.001
NO• (µmol/L)	16.18±2.56	18.51±3.56	31.70±3.41***	32.66±3.12***	26.95±4.80***ns	28.63±3.19***ns	<0.001
MDA (µmol/L)	2.93±0.44	3.32±0.31	6.37±0.70***	6.40±0.47***	5.21±0.73***+++	5.03±0.59***‡	<0.001
CP (µmol/L)	0.98±0.05	1.12±0.06	4.09±0.43***	4.34±0.25***	3.21±0.41***+++	3.13±0.36***+++	<0.001

[Table/Fig-3]: Oxidant markers of the study groups.
CP: Carbonyl proteins; MDA: Malondialdehyde; NO•: nitric oxide; O2•: superoxide anion; Values are means±SD; Statistical comparison between all groups was performed by one-way ANOVA test followed by Tukey post-hoc test; Cases versus controls:* Significant at p-value<0.05; ** significant at p-value<0.01; *** significant at p-value<0.001, ns = not significant at p-value>0.0.5. STEMI versus NSTEMI: †for p-value<0.05, ‡for p-value<0.01, +++for p-value<0.001

Groups	Controls		Cases (Myocardial Infarction)				p-value (ANOVA)
			STEMI		NSTEMI		
	Males	Females	Males	Females	Males	Females	
Catalase (U/g Hb)	92.13±4.01	100.68±6.05	62.32±6.29 ***	60.80±7.42***	71.78±3.53†**	71.78±2.32†***	<0.001
GSH (mmol/L)	4.45±0.32	6.46±0.42	2.60±0.47**	2.76±0.47 ***	3.36±0.52†**	3.30±0.44†***	<0.01

[Table/Fig-4]: Erythrocyte GSH and Catalase levels of the study groups.
GSH: Reduced glutathione; U/g Hb: unit/gram of haemoglobin; Values are means±SD; Statistical comparison between all groups was performed by one-way ANOVA test followed by Tukey post-hoc test. Cases versus controls: *Significant at p-value<0.05; **significant at p-value<0.01; ***significant at p-value<0.001; STEMI versus NSTEMI: †for p-value<0.05

NSTEMI patients. Predominance of AMI was noted in males than females, which was in accordance with previous studies [7,12]. The findings clearly demonstrated that oxidative stress is more prominent in STEMI patients compared with NSTEMI patients. The present study substantiates what had been reported in the study of Serdar Z et al., but contradicts the results of the study of Lavall MC et al., suggesting that the oxidative profile produced during STEMI and NSTEMI is comparable regardless of the size of arterial occlusion generated by thrombus [26,27].

Indeed, the results revealed a significant increase in MDA levels (p-value<0.001), a characteristic end product of lipid peroxidation, in all cases groups which is indicative of noteworthy oxidative stress in AMI patients. These results are reliable with other researchers' findings. [Table/Fig-5] showed an important raise in lipid peroxidation products (MDA, TBA reactive substances (TBARS, predictor of MDA) in following AMI [7,10,12,28]. Khan HA et al., have revealed that the serum MDA concentration begins to rise gradually after acute event of MI, reaching a maximum 6-8 days later [7]. Likewise, Bagatini MD et al., and Al-Fartosi K et al., have demonstrated a significant increase in MDA level which is positively correlated with serum levels of biomarkers of myocardial infarction [10,28].

Author's name and year	Place of study	Number of subjects	Parameters assessed	Conclusion
Al-Fartosi K et al., 2010 [28]	Thi-Qar province/ South of Iraq	35 AMI patients/35 with unstable angina pectoris/ 35 controls	MDA	MDA level increased significantly in AMI patient as compared to controls. There was a positive relationship between MDA levels and myocardial enzymes (CPK and LDH) in case of AMI
Bagatini MD et al., 2011[10]	Brazil	40AMI patients/20 risk group/20 controls	MDA	MDA level increased significantly in AMI patient as compared with the risk group and the healthy control group. MDA was positively correlated with troponin I levels in AMI patients
Khan HA et al., 2013 [7]	Riyadh, Saudi Arabia	128 AMI patients/121 controls	MDA	MDA levels raised gradually after acute event of MI and reached a maximum 6-8 days later.
Ismail MK et al., 2018[12]	Mosul, Iraq	161 AMI patients/156 controls	MDA	Higher MDA levels in AMI patients compared to controls
Present study	Tlemcen, Western region of Algeria	67 AMI patients/75 controls	MDA	MDA levels increased significantly in AMI patient as compared with controls

[Table/Fig-5]: Comparative evaluation between similar studies.

In this respect and by a cursory glance through our data, it is worthy to note that the highest levels of MDA were particularly founded in patients with STEMI, in agreement with another study [26]. It was observed that MDA levels were elevated more than two times in STEMI group as compared to controls. The intense generation of lipid peroxides during STEMI seems to be linked to the extension of myocardial damage. It may be an important trigger of intense coronary thrombosis and total vessel coronary occlusion that lead to an abrupt and sudden blockage of the coronary artery [29].

Likewise, it was noticed a marked elevation in CP concentration following AMI, in agreement with previous studies [11,26]. Indeed, CP levels were elevated more than four times in STEMI group and three times in NSTEMI group as compared to control. This dissimilarity between the two cases groups is statistically significant as evidenced by prior study [26]. This may disclose severe oxidative damage to proteins as a consequence of the widespread of myocardial injury.

The present findings indicated the existence of an abnormal balance between the oxidative and protective mechanisms in AMI patients, mostly in patients with STEMI rather than those with NSTEMI. Diverse previous results suggested antioxidant consumption during AMI, particularly during myocardial reperfusion injury [30,31]. In particular, different vitamins, as well as antioxidant enzymes, have been found noticeably dropped in ischaemic disease [31]. Accordingly, the findings confirmed the chronic fall and severe acute damage to the antioxidant system in AMI patients. Indeed, it was noticed that there was a significant drop in catalase activity, the first well- known line enzymes of the antioxidant defense against ROS. This is in line with other works suggesting that this decrease in catalase activity in AMI patient is related to accumulation of hydrogen peroxide under ischaemia reperfusion, inhibiting afterwards catalase activity [32]. This provided evidence of a severe damage of antioxidant system, which is unable to fight oxidative stress and inflammation [33]. Catalase levels tended to be significantly inferior in STEMI groups especially in males. This may reflect the extensive tissue damage in this group.

Beyond that, GSH system, which is the vital defensive system against oxidative damage [34], is sternly depleted in both cases groups. These results are consistent with others studies demonstrating a considerable decrease in reduced GSH level in AMI patients [7,35]. This may imply that decreased GSH levels is probably associated with enhanced protective mechanisms intending to scavenge and detoxify oxygen free radicals implicated in atherosclerotic plaque rupture that cause occlusion of the coronary artery [7]. So, it is conceivable that the observed decrease in total GSH in patients is in response to the excessive ROS generation after myocardial injury. Thus, the present findings confirmed that the extent of drop in GSH level is affected by the expanse of myocardial necrosis and deterioration of myocardial function as demonstrated by the gradual decrease in going from NSTEMI to STEMI. Among patients with STEMI, males were more likely than females to report significantly reduced levels of GSH as compared to NSTEMI group.

Furthermore, the present data showed that levels of free radicals such superoxide anion (O2•-) and nitric oxide (NO•) were significantly higher in AMI patients as compared with controls (p-value<0.001), which is similar to preceding studies [7,9]. Although STEMI patients had a tendency toward higher O2•-, NO• levels, this did not reach statistical significance when compared to NSTEMI patients.

Dyslipidemia, as defined by increased TC, TG and LDL along with decreased HDL, is obvious in this study in both STEMI and NSTEMI patients [Table/Fig-2] compared to controls, in agreement with previous studies [26,36]. However, there was no statistical significant difference in the serum levels of HDL between the two patients groups. Authors noticed significant high TG blood levels in AMI patients which are concordant with previous reports [11]. It has been suggested that hypertriglyceridemia predisposes to thrombosis by increasing factor VII coagulant activity [37]. When comparing the two patients groups. TG levels seem to be notably more pronounced in STEMI patients particularly in males.TG levels in women with STEMI were not statistically different from those with NSTEMI.

Lipids are one of the main targets of ROS (oxidation of LDL-C), thus play a central role in the pathogenesis of AMI. Dyslipidemia alongside with oxidative stress is a key process in the formation and development of atheromatous lesions, inducing stenosis or occlusion of arterial lumens [38].

With regard to serum uric acid, there was an increased level in AMI patients as compared to controls (p-value<0.001). This result was consistent with previous studies which showed that hyperuricaemia was common in patients with AMI [39,40]. Nevertheless, the two groups of patients were not found to be significantly different in terms of the uric acid levels. This issue needs more assessments in future studies. It was believed that uric acid exhibits pro-oxidant activity and causes oxidative modification of LDL. Available data suggest that uric acid can induce intracellular stress and inflammation which may provoke endothelial injury and enhancement of vasoconstrictor effects [41]. This indicates that uric acid may act as an oxidative stress indicator, though mechanism remains uncertain [42].

Limitation(s)

The present study had some limitations. It is worthy to mention mainly that the sample size was relatively small due to constraints of time and resources. A further limitation was lack of follow-up of the patients to determine the prognostic value of uric acid and oxidative stress markers in AMI patients.

CONCLUSION(S)

Overall, the results of the present study support the concept that oxidative stress is associated with AMI. Hence proved, this study indicates an imbalance between oxidant and antioxidant markers in both STEMI an NSTEMI patients. Moreover, STEMI patients showed a more deteriorated oxidant-antioxidant status compared with NSTEMI patients and healthy controls, most likely as a consequence of the widespread of myocardial injury and larger inflammation in those patients. The divergence in the oxidant-antioxidant status in STEMI versus NSTEMI patients forecasts the importance of measuring the level of serum oxidative stress markers as a diagnostic tool for the medical management of AMI. Finally, a continued investigation in this field is required to appraise the prognostic value of these markers in AMI patients.

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