Estimation of Myeloperoxidase (MPO), Oxidized LDL and Glutathione (GSH) in Patients with Acute Myocardial Infarction (AMI) in Three Iraqi Hospitals

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ABSTRACT:

BACKGROUND:

Acute myocardial infarction (AMI) is a cardiovascular emergency that needs immediate diagnosis and treatment. Myeloperoxidase (MPO) an enzyme generated by active leukocytes, has been connected to atherosclerosis in a mechanistic way and earlier research has suggested that MPO could be employed as a diagnostic and analytical marker for AMI patients. Many cardiovascular disease (CVD) events have been linked to oxidized-LDL (Ox-LDL) &Glutathione (GSH) .

OBJECTIVE:

Studies a new, rapidly rising, or cost-effective biomarker for the detection of AMI.

PATIENTS AND METHODS:

Eighty (80) patients & (40) control offered with chest pain presented to the Coronary Care Unit looking for medical help about their newly developed symptoms. Serum levels were measured by enzyme-linked immunosorbent assay (ELISA).

RESULT:

The result of this current study showed that a highly significant association between the serum concentration of MPO and the three groups study ($p<0.001$). In this present study, a higher significant difference is noted in the level of serum oxLDL in the STEMI group ($p < 0.001$) and (0.005) in the NSTEMI group. the higher significant difference is noted in the level of serum GSH in NSTEMI & STEMI compared with control ($p < 0.001$) and between the patents groups ($p= 0.019$)

CONCLUSION :

Myeloperoxidase(MPO) & GSH serum level is highly associated with AMI and can be used as a biomarker of early diagnosis, progression, and grading of AMI Increased serum concentrations of oxLDL are analytical of future AMI

KEYWORDS: (MPO) Myeloperoxidase, (NSTEMI) non ST segment elevation myocardial infarction, (STEMI) ST segment Elevation Myocardial infarction.

INTRODUCTION:

Acute myocardial infarction (AMI) is a cardiovascular emergency that needs immediate diagnosis and treatment [(1)] Myocardial infarction is still most commonly caused by plaque rupture, however other syndromes such as plaque destruction, coronary microvascular dysfunction, and drug-induced coronary spasm can also cause MI[(2)]. The scientific community distinguishes between two categories of AMI patients: STEMI is defined as an ECG signal with an ST-segment elevation; NSTEMI is defined as an ECG signal with no ST-segment elevation. This distinction is mostly referencing the fact that in STEMI, the occlusion is complete, resulting in cardiac tissue non-reperfusion, but in NSTEMI, the occlusion is partial, ensuring, at the very least, a blood supply[(3)]. A clinical diagnosis of MI is made by combining elevated cardiac troponin levels with protracted chest pain, ECG recordings, or regional wall signal anomalies indicative of beginning ischemia, or angiographic identification of a coronary thrombus [(4)]. Myeloperoxidase (MPO), an enzyme generated by active leukocytes, has been connected to atherosclerosis in a mechanistic way, and earlier research has suggested that MPO could be employed as a diagnostic and analytical marker for AMI patients [(5)]. Patients with acute myocardial Infarction have a high plasma MPO level, which could be employed as an early diagnostic marker in patients with Chest Pain [(6)]. MPO is a peroxidase enzyme found mostly in neutrophils, which creates hypohalous acids (HOX) to carry out antibacterial activity. MPO was first discovered to be a sensitive predictor of Myocardial Infarction in patients with chest pain in a 2003 study[(7)].

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Myeloperoxidase (MPO) catalyzes H_2O_2 with halide or pseudo-halide ions to produce alternative oxidants of a chemical such as (HOCl) hypochlorous acid, (HOBr) hypobromous acid, and (HOCN) hypothiocyanous acid, all of which are important important in bacterial cell killing and bacterial growth prevention. [8].

Scientists are particularly interested in the oxidation of LDLs since it has been proven that macrophages preferentially absorb oxidized LDLs (rather than native LDLs), which leads to the production of foam cells and the advancement of atherosclerosis plaque. [9].

Oxidized low-density lipoprotein (OxLDL) is involved in the start and progression of atherosclerosis, leading to endothelial dysfunction and plaque destabilization via a variety of pathways. Atherosclerotic plaques contain OxLDL and oxidized lipid byproducts, according to human studies. LDL particles gather in the plasma and penetrate the intima of the arteries, where they are oxidized by oxygen free radicals [10].

Myeloperoxidase (MPO) is an enzyme catalyst for oxidative modification of lipoproteins in the artery wall, according to numerous studies utilizing low-density lipoprotein. The risk of coronary artery disease is inversely related to plasma levels of high-density lipoprotein (HDL) cholesterol and apoA-1, the major apolipoprotein of HDL. HDL is a specific in vivo target for MPO-catalyzed oxidation, which reduces the cardioprotective effects of this antiatherogenic lipoprotein [11].

Glutathione (GSH) is involved in the development of a variety of disorders, including cardiometabolic and cardiovascular diseases (CVD). Significant variations in GSH concentration and/or oxidation states characterize the development and progression of CVD [12].

Myeloperoxidase (MPO) is responsible for the production of hypochlorous acid (HOCl) at areas of harm and inflammation, which is linked to a reduction in GSH concentration. HOCl can rapidly inactivate glutathione peroxidases (GPx), even at low concentrations. H_2O_2 scavenging enzymes, such as catalase or glutathione peroxidases (GPx), in combination with glutathione GSH, can prevent hypochlorous acid formation [13].

Investigation justification Early and accurate diagnosis of AMI remains one of the most difficult difficulties confronting emergency department clinicians.

To investigate a new, rapidly increasing, or cost-effective biomarker for the detection of AMI.

PATIENTS AND METHODS:

Study protocol

The research was planned to run from September 2020 until August 2021. Eighty (80) patients with chest trouble sought medical assistance at the Coronary Care Unit (CCU) of the Ibn Al-Bitar Specialized Center for Cardiac Surgery, Al-Imamian Al-Kadhimiyain Medical City, and Al-Yarmuk General Teaching. The clinical presentation and history were used to make the diagnosis of AMI, which was confirmed by ECG and Troponin concentration.

Study samples were divided into three (3) groups. Group I patients with STEMI, Group II patients with NSTEMI and Group III of control group.

Specimen Collection

Four milliliters of venous blood sample were drawn from each patient (12-48) hours (before the sample is taken) presented to the Coronary Care Unit with acute Chest Pain. The same quantity of blood was drawn from the control group subjects. while blood was transferred into a gel tube, left at room temperature for less than thirty minutes for clotting, then the samples were centrifuged, and the sera were separated and divided into four parts. An aliquot of serum was transferred into a 1.5 ml Eppendorf tube, which was used to measure Human Myeloperoxidase (MPO) concentration level, High sensitive cardiac troponin I (hscTnI), Human oxLDL, and GSH. The tubes were stored at -20 ºC until analysis which was done within 6 weeks after the collection.

Determination of the serum concentration of Myeloperoxidase (MPO) by ELISAHuman

Myeloperoxidase (MPO) ELISA Kit Catalog Number BS175830, Human MPO can be quantified in, serum, plasma, saliva, cell culture supernates, cell lysate, tissue homogenates, and urine.

Determination of the serum concentration of Human High Sensitive Cardiac Troponin (hscTn) by ELISA

Serum Human High Sensitive Cardiac Troponin (HS-cTn) was measured by ELISA Kit Catalog Number MBS165910.

Determination of the serum concentration of Human Oxidized Low Density Lipoprotein (Ox LDL) by ELISA

The ELISA kit (Catalog No: E-EL-H6021) is used to determine the amounts of Human OxLDL in serum and plasma in vitro.

Determination of the serum concentration of Human Glutathione (GSH) by ELISA

The ELISA kit (Catalog Number: E-EL-0026) applies to the in vitro quantitative determination of GSH concentrations in plasma, serum, and other biological fluids.

Statistical Analysis

The statistical program for societal sciences (SPSS) version 23 was applied for all statistical analyses. The t-test was used to identify the significant variance between the study groups. It was used to assess the normality of the distribution of all variables. To analyze the sensitivity and specificity of the utilized kits in distinguishing real positive versus false-positive cases in this investigation, the receiver operating characteristics (ROC) curve was constructed. The plots of the ROC curves were also shown. P-values < 0.05 were considered significant, while P-values < 0.01 were considered very significant.[14]

RESULTS:

Serum level of myeloperoxidase (MPO) in AMI patients against the control group

Myeloperoxidase (MPO) was estimated by ELISA technique in the sera of Control & patients at admission (n=120). The mean of MPO (pg/ml) of the control group, the NSTEMI group, and the STEMI group are presented in (table 1). There was a highly significant difference between controls and disease groups (P<0.001). Also, there was a significant variance in the mean between disease groups (p < 0.001), with the highest level seen in the STEMI group.

Serum level of hscTn in AMI patients against the control group

Human High Sensitive Cardiac Troponin (hscTn) was estimated by ELISA technique in the sera of Control & patients at admission (n=120). The mean of hscTn of control, NSTEMI, and STEMI groups are presented in (Table 1). There was highly significant variance between controls and disease groups (P<0.001). Also, there was a highly significant difference in the mean hscTn among disease groups (P=0.001), with the highest level seen in the STMI group.

Serum level of oxLDL in AMI patients against the control group

In this present study, a higher significant difference is noted in the level of serum oxLDL in the STEMI group (p < 0.001) and NSTEMI group (0.005). low concentration of oxLDL level is found in the control group with the Mean ± SE = 139.54 ± 4.34 (pg/ml) compared to the level in NSTENI with the Mean ± SE = 173.14± 10.82 (pg/ml) and compared with high concentration level in patients with STEMI, the Mean ± SE = 188.18 ± 10.75 (pg/ml) table 1. T-Test conducted on the studied group.

serum level of GSH in AMI patients against the control group

According to results demonstrated in Table 1, GSH showed significant differences among all studied groups given that the control samples were obtained from serum. There was highly significant variance between controls and disease groups (P<0.001). Also, there was a significant variance in the mean GSH among disease groups (P=0.019). T-Test conducted on the studied group confirms the significant differences between them (p<0.001).

The sensitivity & specificity between NSTEMI & Control groups

The area under(AUC) the corresponding the receiver operating characteristics (ROC) curve for Serum hsTn ,Myeloperoxidase (MPO) , GTH and oxLDL concentrations were compared between disease group NSTEMI and control group(table 2) for the prediction of type of patient ROC analysis revealed that Serum hsTn , MPO , GTH and oxLDL concentrations had a significant area under the curve (AUC) 0.972,0.902, 0815and 0.65 indicating that a Cutoff point of 162 pg/ml ,1005 pg/ml, 93 (µg/ml) and 148(pg/ml) gave sensitivity of(98%,95 %,85% & 55%) and specificity (98% ,83% , 73% & 60%) respectively .See figure 1 & figure 3

Table 1: Mean ± SE of the biochemical parameters used in the research for control , NSTEMI and STEMI groups.

Groups (n=40)	parameters	Mean	Std. E	Range	p-value	p-value
Control	hsTn (pg/ml)	154.78	5.44	226.43		
	MPO (pg/ml)	938.87	22.92	771.91		
	GSH(Mg/ml)	110.15	4.78	136.79		
	oxLDL(pg/ml)	139.54	4.34	103.33		
NSTEMI	hsTn (pg/ml)	311.31	40.03	1,040.06	<0.001*	
	MPO (pg/ml)	1,143.76	20.51	577.77	<0.001*	
	GSH(Mg/ml)	80.75	2.35	64.65	<0.001*	
	xLDL(pg/ml)	141.34	2.50	67.49	<0.005*	
STEMI	hsTn (pg/ml)	573.51	63.62	1,414.29	<0.001*	0.001**
	MPO (pg/ml)	1,311.60	32.00	858.51	<0.001*	<0.001**
	GSH(Mg/ml)	72.37	2.59	72.39	<0.001*	<0.019**
	xLDL(pg/ml)	188.18	10.75	321.74	<0.001*	<0.001**

* significant difference between controls and disease groups
** significant difference in the mean among disease groups
(STEMI) ST segment elevation Myocardial Infarction, (NSTEMI) non ST segment elevation myocardial infarction, Myeloperoxidase. (MPO) Serum Human High Sensitive Cardiac Troponin (HS-cTn), Glutathione (GSH) . Oxidized Low Density Lipoprotein (Ox LDL).

The sensitivity& specificity between STEMI and Control groups

The area under curve (AUC) for Serum hsTn , MPO , GTH and oxLDL concentrations were compared between disease group and control group for the prediction of type of patient . ROC analysis revealed that Serum hsTn, MPO, GTH and oxLDL concentrations had a significant area (table 2) under the curve (AUC) 0.979, 0.932 ,0866 and 0.826 indicating that a Cutoff point of 161 pg 1094 pg/ml, /ml,90 (µg/ml) and 154(pg/ml) gave sensitivity of(95 %,90% 88% & 73%) and specificity (98% ,93% 78% &70%) respectively .See figure2 and figure 4

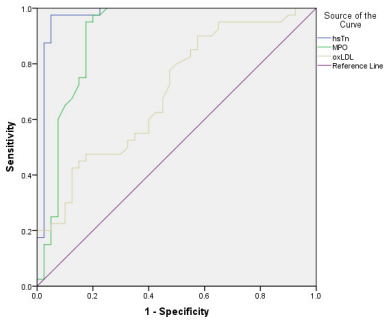

Figure 1: Receiver operating characteristic (ROC) curve for serum hsTn, MPO, and oxLDL in the NSTEMI Group

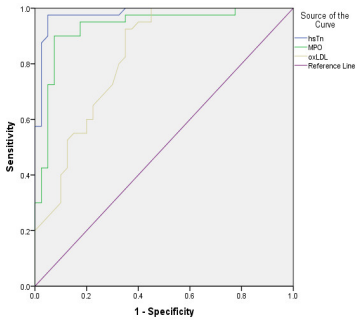

Figure 2: Receiver operating characteristic (ROC) curve for hsTn, MPO, and oxLDL in the STEMI Group

Table 2: Valued Area Under Curve (AUC) and analytical cut-off point values for the serum hsTn , MPO, GSH and oxLDL concentrations with the Receiver Operating Characteristics (ROC) curve examination between patients groups (NSTEMI,STEMI) and Control.

Group	parameters	AUC	Cut off point	sensitivity	specificity	p-value	95% CI
NSTEMI	hsTn (pg/ml)	0.972	162	98%	95%	<0.001*	0.930-1.000
	MPO (pg/ml)	0.902	1005	95%	83%	<0.001*	0.826-0.978
	GSH(µg/ml)	0.815	93	85%	73%	<0.001*	0.715-0.915
	oxLDL(pg/ml)	0.698	142	60%	60%	0.002*	0.584-0.812
STEMI	hsTn (pg/ml)	0.979	161	95%	98%	<0.001*	0.952-1.000
	MPO (pg/ml)	0.932	1094	90%	93%	<0.001*	0.873-0.992
	GSH(µg/ml)	0.866	90	88%	78%	<0.001*	0.784-0.947
	oxLDL(pg/ml)	0.826	154	73%	70%	<0.001*	0.736-0.917

* significant difference between controls and disease groups . (STEMI) ST segment elevation Myocardial Infarction, (NSTEMI) non-ST elevation myocardial infarction, Myeloperoxidase. (MPO) Serum Human High Sensitive Cardiac Troponin (HS-cTn), Glutathione (GSH) . Oxidized Low Density Lipoprotein (Ox LDL).

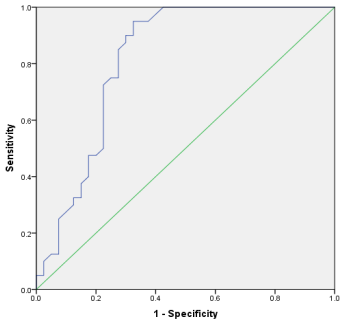

Figure 3: Receiver operating characteristic (ROC) curve for GSH in the NSTEMI Group

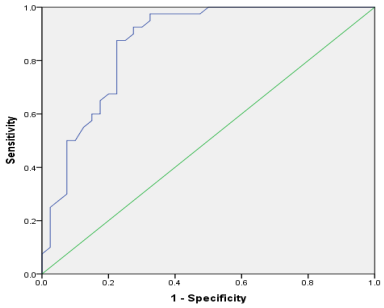

Figure 4: Receiver operating characteristic (ROC) curve for GSH in the STEMI Group

DISCUSSION:

Early diagnosis of Acute Myocardial Infarction (AMI) patients with clinical signs suggestive of coronary heart disease (CHD) is a difficult task for physicians. However, because of its extremely low detection limits, the new biomarkers assay has substantially enhanced the early detection of mild cardiac damage. Admission for patients with a diagnosis of AMI without a thorough study with cardiac biomarkers frequently results in overcrowding and high hospital expenditures. One possible benefit of a marker strategy is the ability to diagnose or rule out Acute Myocardial Infarction (AMI) quickly. In all patients who report chest discomfort in the cardiac care unit(CCU), hcTn values are required for the diagnosis of AMI. We further compared the diagnostic utility of hs-cTnT with MPO, GSH, and oxLDL tests for detecting AMI in patients with Coronary Heart Disease who presented with chest pain.

Myeloperoxidase (MPO) serum level and Acute Myocardial Infarction (AMI)

The result of this current study showed that a highly significant association between the serum concentration of MPO and the three groups study ($p<0.001$). low concentration of MPO level is found in control group with the Mean ± SE = 938.87 ± 22.92 (pg/ml) *Table1* compared to the level in NSTENI with the Mean ± SE = 1143.76 ± 20.51(pg/ml) ($p<0.001$) and compared with highly concentration level in patients with STEMI, the Mean ± SE = 1311.60 ± 32.00 (pg/ml) ($p<0.001$). Many previous studies would agree with the finding of this study and the more relevant ones were reviewed below.

MPO is produced locally by neutrophils, and high levels of MPO in the blood have been linked to acute STEMI. MPO levels were raised in AMI patients even two hours before the onset of myocardial infarction [15].

A study from China showed that Plasma MPO has been revealed to be an independent risk factor for plaque erosion and to be a strong predictor of plaque erosion. Although MPO's predictive value for plaque erosion isn't high enough to make it a stand-alone biomarker, it could be useful in risk stratification and establishing a suitable treatment plan for patients with AMI [16].

Myeloperoxidase (MPO) and CRP levels were greater in rats with myocardial infarction, which had been identified from the first 24 hours. This indicates that the rats in this group were inflamed [6].

Another study demonstrated that In AMI patients, high MPO plasma levels have been demonstrated to have predictive significance for death and major adverse cardiovascular events (MACE) [17]. Also, a study suggested that MPO is a marker that is raised in CHD, particularly in cases of acute myocardial infarction (AMI), because it acts as an enzyme in the production of a variety of reactive oxygen species (ROS) by catalyzing the conversion of hydrogen peroxide (H_2O_2) to species such as hydroxide ion(OH^-), peroxynitrite ONOO, hypochlorous acid (HOCl), and NO_2 [18]. Myeloperoxidase (MPO) is a possible biomarker for risk stratification connected to the development of CHD and atherosclerotic plaque, and it is receiving a lot of interest [19].

Oxidized Low-density lipoprotein (oxLDL) in Acute Myocardial Infarction (AMI)

According to results demonstrated in *Table 1* oxLDL showed significant differences among all studied groups given that the control samples were obtained from serum. oxLDL score of STEMI patients showed the highest level which is significantly ($p<0.001$) also of NSTEMI patients who showed significantly ($p=0.005$) higher scores when compared with control samples. There was no significant variance between disease groups ($P=0.327$). Many previous studies would agree with the finding of this study and the more relevant ones were reviewed below.

According to their current findings, the oxLDL concentration in the LDL was nearly 3 fold higher in patients with AMI than in control participants, as assessed by sandwich ELISA. During catheter treatments, blood samples from AMI patients were taken from the infarct locations [20].

The plasma oxLDL level increased transiently after Myocardial Infarction or soon after vascular damage produced by percutaneous transluminal coronary angioplasty (PTCA) treatment, according to a recent study. The oxLDL accumulated in ruptured atherosclerotic lesions is the source of oxLDL in the rapid rise in plasma. Because oxLDL clearance from the circulation is so quick [21].

Another study showed that the degree of oxidation of LDL-c is related to the severity of the condition. This process causes a series of vascular alterations with clinical consequences, such as artery narrowing and related clinical symptoms (angina pectoris), as well as ACS of various forms depending on the stability of the atherosclerotic plaque. ACS is a kind of coronary artery disease (CAD) that has distinct symptoms and is frequently linked to AMI [22]. Other recent study showed that because the vessels that oxidized LDL damage transmits blood to all of your organs and tissues, inflammation in the arteries creates complications. Atherosclerosis is assumed to be aided by oxidized LDL, which raises your chances of suffering a heart attack or stroke [23].

A Study from Russia presented that Their findings show that increasing the amount of hydrogen peroxide H_2O_2 utilized to alter LDL via hypochlorous acid (HOCl) generated in an MPO catalyzed process increases the affinity of the resulting oxLDL [24].

Glutathione (GSH) in Acute Myocardial Infarction (AMI)

In this present study, the higher significant difference is noted in the level of serum GSH in NSTEMI & STEMI compared with control ($p < 0.001$) and between the patents groups ($p= 0.019$). High concentration of GST level is found in the control group with the Mean ± SE = 110.51 ± 4.78 (µg/ml) compared to the level in NSTENI with the Mean ± SE = 80.75 ± 2.35 (µg/ml) and compared with low concentration level in patients with STEMI, the Mean ± SE = 72.37 ± 2.59 (µg/ml) *table1*. Many previous studies would agree with the finding of this study and the more relevant ones were reviewed below.

A study from India showed that Antioxidant indicators such as glutathione (GSH) level and catalase activity were found to be considered minor in the high risk of Atherosclerotic Cardiovascular Disease (ASCVD) compared to the low-risk group in the study [25].

Another study demonstrated that the link between reactive species(ROS & RNS) generation and the growth of cardiovascular diseases Because cardiovascular diseases are linked to a rise in reactive oxygen and nitrogen species (ROS and RNS), and because glutathione (GSH) is directly involved in dropping the damaging effects of both the disease and the oxidative properties of ROS and RNS, it will be possible to develop therapeutic strategies to help restore normal physiological conditions (26).

Also, a study from Serbia suggested in individuals with asymptomatic and symptomatic CVD, the amount of GSH was reduced by 21% and 40%, respectively. The authors concluded that decreases in GSH levels are closely associated with cardiac anomalies in individuals with CVD based on these findings. Furthermore, because these findings reveal that GSH levels were also reduced in patients with undiagnosed CVD [(12)].

Other study showed that the glutathione (GSH) concentrations were estimated in the filters of samples from the heart of rats that had preserved with changing times of Myocardial Ischemia. It is identified that the concentrations of GSH can keep reducing with long ischemia time. Also, reperfusion next Ischemia can reduction the GSH level in Myocardial cells [(27)]

High sensitive Cardiac Troponin (hscTn) in AMI

In this present study, the higher significant difference is noted in the level of serum hscTn in three study groups ($p < 0.001$). Low concentration of hscTn level is found in control group with the Mean ± SE = 154.78± 5.44 (pg/ml) compared to the level in NSTENI with the Mean ± SE = 331.31± 40.03 (pg/ml) ($p<0.001$) and compared with highly concentration level in patients with STEMI, the Mean ± SE = 573.51± 63.62 (pg/ ml) ($p<0.001$) *table1*. Previous studies would agree with the finding of this study and the more relevant ones were reviewed below.

A previous study presented that The adjudication committee did not evaluate the diagnostic of myocardial injury; only the diagnosis of Acute Myocardial Infarction was careful Many clinicians in the United States are concerned about the use of hs-cTn assays to diagnose acute myocardial infarction [(28)].

Another study showed that after accounting for traditional risk variables, CTnT had no connection with MI or CHD, whereas CTnI did. As a result, CTnI was found to be more significantly linked to the major CVD outcome. CTnT was linked to non-CVD death, whereas CTnI was not [(29)]. A preceding study indicated that High-sensitivity (hscTn) assays are defined as those that can detect cTn in healthy people. By definition, diagnostic tests must be able to detect cTn levels in more than half of a healthy population (hence, 50 percent of the population is above the lower limit of detection). As a result, the term "high sensitivity" relates to analytical sensitivity rather than clinical sensitivity [(30)].

Sensitivity and Specificity of parameters

The cut-off values for all biomarkers were calculated using the point on the ROC curve with the smallest distance from the plot's left upper corner. This method yields a cut-off concentration that has the best sensitivity and specificity while also yielding the most objective outcome. A more subjective method could be utilized in future studies or clinical practice, emphasizing either specificity or sensitivity after deciding which is more important. Serum hsTn ,MPO,GSH and oxLDL were suitable for NSTEMI diagnostic providing sensitivities of 98%,95%,85% and 60% *table2* and specificities 95%,83%,73%, and 60%, respectively, with significant AUCs 0.972, 0.902, 0.815 and 0.654,respectively. Serum biomarkers were appropriate for STEMI diagnostic provided that sensitivities of 95%,90%,88%and 73% and specificities 98%,93%,78%, and 70%, with significant AUCs 0.979, 0.932, 0.866 and 0.826, respectively. The hs-cTnT assay's sensitivity and specificity were excellent for detecting AMI early. The results of this investigation are really useful. MPO is an excellent diagnostic for diagnosing AMI, and increased serum MPO activities are linked to AMI prevalence and high oxLDL levels. Low levels of GSH, on the other hand, were associated with AMI. These findings are consistent with many previous studies mentioned below.

A study showed that At the time of admission to CCU, the AUC values for hs-cTnT in our AMI patients were identical to those reported by[(31)].

Other study presented that several studies have shown that MPO plays a function in the atherosclerotic process at all stages. Due to the rapid spike of MPO levels after myocardial necrosis, MPO is an early diagnostic biomarker in individuals with suspected AMI. This evidence from a previous study revealed that MPO might be used to categorize AMI patients in patients hospitalized soon after the onset of chest pain and that its sensitivity could be comparable to the gold standard troponin I in this group of patients[(32)].

Another study found that MPO serum levels in patients with ACS accurately predicted an increased risk of future cardiovascular events, adding to the prognostic data [33].

A study showed that MPO had an area under the curve ROC = 0.906 (0.870–0.934; 95 percent CI; P = 0.0001), whereas cTnI had an area under the curve ROC= 0.922 (0.889–0.948; 95 percent CI; P = 0.0001)[34].

A study presented that oxLDL is not a laboratory biomarker that can be used in clinical trials. Before oxLDL to be accepted as a therapeutically relevant screening tool, its sensitivity, specificity, positive or negative predictive value and cost-benefit concerns must all be determined [35].

CONCLUSION:

Myeloperoxidase(MPO)serum level is highly associated with AMI and can be used as a biomarker of early diagnosis, progression, and grading of AMI**.** Glutathione ((GSH) serum level is associated with AMI and can be used as a biomarker of diagnosis of AMI**.** Increased serum concentrations of oxLDL are predictive of future AMI events in Iraqi patients. The estimation of oxLDL may improve the prediction of atherosclerotic AMI complications. The mechanisms of inhibiting MPO's activity and function should be studied to prevent MPO expression.

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