

Study of relationship between blood parameters and oxidant-antioxidant status of patients with unstable angina pectoris and myocardial infarctions

Khalid Al-Fartosi* , Raid Al-Salih** , Salih J. Batah***

SUMMARY

The present study investigated the relationship between blood parameters and oxidant-antioxidant parameters of patients with unstable angina pectoris and myocardial infarction in Thi-Qar province / south of Iraq.. The results showed a significant elevation for each one of WBCs count, absolute neutrophil value and ESR in patients with unstable angina(UA) and myocardial infarction(AMI) when compared with control. Also a significant decrease was observed for each one of RBCs count, PCV, and Hb in both patient groups compared with control. The results of biochemical parameters explained a significant increase of malondialdehyde(MDA) and ceruloplasmin(Cp) in patients with UM and AMI compared with control group. Creatin phosphokinase(CPK) and lactate dehydrogenase(LDH) had a significant rising in patients with AMI , while there was a slight and non significant rising for both enzymes in patients with UM compared with control group. Albumin was decreased significantly in blood serum of AMI patients , while albumin level was not effected in blood serum of UA patients compared with control. Also, the results indicated the positive relationship between MDA and ESR,WBCs,absolute neutrophil value and ceruloplasmin in UA patient group , while in AMI patient group , the MDA was positively correlated with ESR,WBCs,absolute neutrophil value,ceruloplasmin ,CPK and LDH.In addition, the was a negative relationship between MDA and albumin in AMI patients.

Key words: Blood parameters , oxidant-antioxidant status , unstable angina , myocardial infraction .

* Department of Biology , College of Education , University of Thi-Qar , Iraq.

** Department of Chemistry , College of Science , University of Thi-Qar , Iraq.

*** Office of Thi-Qar health , Ministry of Health , Iraq

INTRODUCTION

Ischemic heart disease (IHD) in the vast majority of cases, are caused by an imbalance between the myocardial oxygen demand and the blood supply (1,2). The most common cause of ischemic heart diseases is the narrowing of the lumina of the coronary arteries by atherosclerosis

(3,4), and hence (IHD) are often termed coronary heart disease or coronary artery diseases(2) . IHD are the single most common cause of death in economically developed countries of the world (5), where it is responsible for about one third of all deaths (2,6) . Depending on the rate and severity of four syndromes it may develop: (1) various forms of angina

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pectoris (chest pain), (2) acute myocardial infarction, (3) sudden cardiac death, and (4) chronic ischemic heart diseases with congestive heart failure(2) .The term acute coronary syndromes is applied to the spectrum of three acute catastrophic manifestations of IHD: e.g. unstable angina, acute myocardial infarction , and sudden cardiac death. All three cases result from acute changes in the morphology of atherosclerotic plaques (2,7,8) .In most cases of suspected myocardial infarction, measurement of plasma total CK and LD₁ activities together with the clinical and electrocardiograph (ECG) findings are adequate to make a diagnosis (9) . In other side plasma total CK activity alone can be very misleading (10) . Mainly plasma enzyme activities are raised in about 95% of cases of myocardial infarction and are sometimes very high, and the degree of rise is a very rough indicator of the size of the infarct, but is of limited prognostic value (11) . A second rise of plasma enzyme activities after their return to normal may indicate extension of the damage(11) . During ischemia, many mediators or cytokines are secreted from macrophage, endothelial cells, myocardium, and other cells (e.g. IL-1, IL-6, IL-8, TNF- α ,G-CSF and GM-CSF) (12,13) .Myocardial necrosis induces complement activation and free radical generation , triggering a cytokine cascade initiated by tumor necrosis factor (TNF- α) release (13,14) . TNF- α production is induced in ischemic myocardium due to ischemia and several other factors (e.g. mechanical stress deformation of damaged myocytes, reactive oxygen species, auto-regulating self amplification(15) . On other side, TNF promote IL-1 production by mononuclear phagocytes (macrophages) . As a result, the neutrophils increase in number from myeloid precursors in the bone marrow through the combination of influence of (TNF- α , IL-1) and (IL-3, G-

CSF and GM-CSF) (16) . Both IL-1 and TNF- α cause vascular endothelial cells to express adhesion molecules which make the endothelial cells adhesive for neutrophils(17) . After neutrophils adherence to endothelium , it migrates across vessels wall by squeezing between endothelial cells toward cardiomyocytes in a process called transmigration(14) .The complement component C5_a and IL-8 have crucial role in chemotaxis or recruitment of neutrophils into ischemic area along with chemo attractant gradients from injured tissue (14,16) .Also neutrophils make attachment with intracellular adhesion molecules (ICAM -1) expressing cardiomyocytes and attack these myocytes in necrotic area by releasing of proteolytic enzymes and free radicals production (14) . However, the TNF- α promotes intracellular oxidative stress or free radicals production (13) . During ischemia, oxygen-derived free radicals can form as a result of many processes. First, production of superoxide can appear in the mitochondria especially if activity of superoxide dismutase is decreased. Secondly, xanthine oxidase in the coronary endothelium can be activated with formation of superoxide, Thirdly, as a result of catecholamine stimulation arachidonic acid can brokendown with the formation of superoxide ions. Finally, the neutrophils are an important source of free radicals, and neutrophil-dependent phagocytosis is mediated by free radicals (18) . Normally the increase in free radicals production lead to enhancement peroxidation of cell membrane lipids. As a result, antioxidants such as ceruloplasmin albumin,superoxide dismutase (SOD) glutathion peroxidase uric acid, vitamin E and vitamin C, perform their work to scavenge the formed free radicals. Consequently, these antioxidants will increase or decrease in the opposite state of the normal cases.(19) . Ceruloplasmin (*Cp*)

may function as an antioxidant in three different ways: (1) By binding copper, ceruloplasmin prevents free copper ions from promoting oxidative damage.(20) (2) The ferroxidase activity of *Cp* is represented in (oxidation of ferrous ions[Fe²⁺] to the ferric state [Fe³⁺]), makes easily iron loading onto its transport protein, transferrin by incorporating with apotransferrin, resulting in low transferrin antioxidant capacity (21) . (3) Also, *Cp* has an important role in an inhibition of oxidative damage due to its ability to scavenge superoxide (O₂^{•-}) in the same way as the superoxide dismutase (SOD) enzymes. But the *Cp* acts as extracellular (O₂^{•-}) scavenger, while (SOD) acts as intracellular scavenger (22) . As for albumin, it has several biological functions, two of them for antioxidation. However, it acts as chain-breaking antioxidant and a legend binder for free metal ions such as (copper and iron) (23) .These metals can participate in Haber-Weiss reaction, resulting in generation of ·OH which immediately reacts with other molecules causing damage for them, but albumin has the ability to bind copper tightly and iron weakly to its surface(24). Furthermore, there is a large amount of available albumin, it scavenges the generated ·OH by binding to the copper and iron, and results in protecting other protein molecules. Therefore, albumin can be considered (as a sacrificial antioxidant) (25) .

MATERIAL AND METHOD

Selection of Subjects :

The present study was performed in AL. Hussein hospital, especially in the coronary care unit (CCU) and the laboratories during the period from 10/ 9/ 2006 to 31/ 3/ 2007. The study has been

conducted on total number of patients who are divided into 2 groups:-

GROUP 1: 35 patients with unstable angina pectoris [(20)males , (15)females)] with age range (25—83)

GROUP 2: 35 patients with acute myocardial infarction [(26)males, (9)females)] with age range (40—85)

GROUP 3: The control group, consist of 35 healthy subjects [(20) males,(15) females)] with no history of systematic illness.

Sample with drawl :

From the patients with AMI and UA, (10 mL) blood sample was taken from the antecubital vein. 2.5 mL of blood sample was put into a tube that contains EDTA as anticoagulant to perform the hematological analysis, other 7.5 mL of blood sample is put into a glass tube without anticoagulant. then they were centrifuged at 3000 rpm for 5 mins. The serum was separated and stored at (-20c⁰) till the time of the biochemical analysis.

Hematological analysis

The hematological parameters which were studied in this study included the red blood cells count(RBCs) , packed cell volume(PCV) , hemoglobin concentration(Hb.) , erythrocyte sedimentation rate(ESR) , the total leukocytes count(WBC) and leukocytes differential count (neutrophils, acidophils , basophils , lymphocytes and monocytes). These parameters performed according to (26,27).

Biochemical analysis:

The biochemical parameters which were studied in this study included malondialdehyde (MDA) , Creatine phosphokinase (CPK) , Lactate dehydrogenase (LDH) , Ceruloplasmin and albumin which were measured according to (28-30).

Statistical analysis :

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Analyzed data were performed by using SPSS program, a statistically significance of differences of data among studied groups were tested with F- test (ANOVA). The values were given in tables as (mean \pm SD), and were statistically considered significant at probability ($p \leq 0.05$). The Relationships between the parameters in each patient group were analyzed by using Pearson's Correlation coefficient (r).

RESULTS AND DISCUSSION

Lipid peroxidation:

The statistical data were reported in table (1), indicated to the present of significant increase in MDA concentrations of patient groups as compared with the healthy control group. No significant alteration between these two patient groups (Unstable angina pectoris and acute myocardial infarction) may be detected in the mentioned table. When the oxidative stress happens with or in lipids, called lipid peroxidation. The malondialdehyde is a known stable end product of lipid peroxidation (31). Therefore, the estimation of the MDA by the thiobarbituric acid reaction may be used to know whether a process of lipid peroxidation has happened (32).

In this study, the levels of serum MDA increases significantly because during acute coronary syndromes (UA and AMI), many processes are thought to be involved, (e.g. leukocytosis). It seems reasonable to expect a raised MDA conc. by action of ROS during the phagocytosis process. Aznar (1983) (38) and Muzakova (2000) showed that within few hours of the appearance of chest pain, the serum MDA conc. in the AMI begins to raise gradually, however, after AMI reaches maximum during (6–8) days (32,33). While Dubois-Rande (1994) and Natalia (2005) have showed the MDA concentration increases in serum of patients with the acute

coronary syndromes reaching the maximum during the third day (34,35).

Erythrocyte parameters : Routine erythrocyte parameters for determination of anemia :

The tables (2) and (3) explained that the (RBCs count, PCV, and Hb) have significant decrease in the two patient groups when compared with their normal control values which are specific for each one. Despite no significant differences in these parameters between patient groups in each sex. Also, these data did not give any relationship or significant correlations between each one of these above parameters with MDA measurements for both patient groups (UA and AMI). There are some parameters that reflect the structure and function of RBC such as PCV, Hb, and RBCs count. These tests are done together to aid the diagnosis and classification of anemia. The significant decrease for (RBCs count, PCV, and Hb) in males and females of (UA or AMI) in this study, may be happened due to the dropping of iron (Fe) especially during the first three days, due to some factors: (1) extensive neurohormonal effect, (2) utilization of serum iron in an immunologic inflammatory process by producing free radicals which play a role in the pathogenesis of IHD (36) and (3) an increased uptake of iron in the reticulo-endothelial system for synthesis of ferritin (37). Fitzsimons (1980) and Van Der Schouw (1990) referred to that AMI is similar to the chronic inflammatory diseases, accompanied by a significant decrease of Fe and total iron binding capacity (TIBC) during the first days, more than angina pectoris. As a result of the decreasing in (Fe and TIBC), the Hb and MCV fall (37,38). One study showed a statistically significant elevation in the

mean erythrocyte count of most male groups while the females showed a decreased values. and the mean hemoglobin content of male groups expressed a statistically increased value but the females were statistically observed with decreased value(39). While other researchers explained that PCV can raise in the early days after MI as a result of hem concentration.(40,41)

Erythrocyte sedimentation rate :

Table (4) demonstrated a significant rising in ESR values in two patients groups, compared with the healthy control values. Whereas no significant difference in ESR values between UA and AMI groups, can be observed. Figure (1) showed a positive correlation coefficient explaining a relationship between MDA and ESR measurements in both groups of UA and AMI patients ESR is a non specific test , routine analysis mainly used to screen for the presence of hidden inflammation, which measures the tendency of red blood cells to aggregate (42) .

The ESR was elevated in acute coronary syndromes (ACS), and this is due to increasing in α 2-globulin fibrinogen level (43).The increasing of these proteins results in a decrease of the zeta potential (negative charge) surrounding the erythrocytes. With a lower zeta potential, the erythrocytes are able to join together in rouleaux formation and settle from the plasma at a faster rate in the westergreen tube which is used for ESR measurements(44). Also, we found a positive relationship between MDA and ESR in both patient groups, we thought the cause return to that ischemia /reperfusion in ACS, induce the releasing of some cytokines such as (TNF- α and IL-6), however, TNF- α stimulate the production of ROS by leukocytes which are increased. And at the same time IL-6 induce creation of α 2-globulin and fibrinogen, so these proteins have direct effect to increase ESR. That

association between (TNF- α and IL-6) events makes the above relationship found .Natali (2003) found that ESR was significantly rising in both acute coronary syndrome patients (UA and AMI) (43) .

White Blood Cell Parameters WBCs Count :

Table (5) showed a significant elevation in WBCs count of these two patient groups in comparison with normal control values, also explained a significant elevation when the comparison between two patient groups (UA and AMI) was established.

Absolute Neutrophil Values :

The statistical analysis illustrated in table (5) that there is a significant increasing for absolute neutrophil values in both patients groups in comparison with normal control values. Also, a significant increase was observed in the absolute neutrophil values in AMI group when compared with UA group. We showed in Figures (2) and (3) good relationships between each one of (WBCs , ab. neutrophil values) and MDA levels in both patient groups (UA and AMI).

The WBCs increase in count as a result of stimulation of immune system. During the ischemia /reperfusion, some changes happen. However the ischemic or necrotic area in myocardium secretes some mediators or cytokines with a huge amounts and the important cytokine in this inflammatory process is TNF- α (45), which stimulates with another two cytokines (G-CSF and GM-CSF) the proliferation of neutrophils from bone marrow; therefore, the total WBCs count will increase (46). TNF- α has another important role, it is a stimulation of neutrophils to produce ROS , a powerful killing factors in phagocytosis. It is clear that leukocytosis and high ROS production are associated with the increase of TNF- α secretion, and this reason is behind the positive correlation between (WBCs count and Ab. Neutr. Count) and

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MDA levels in the present study. Though some researches illustrated that the leukocytes may raise in numbers with three folds about in the normal conditions during the acute coronary syndromes, as a result to the elevation of neutrophil proliferation rate, and principally ACS include many inflammatory processes, leukocytosis is one of them (47).

Myocardial enzymes :

CPK :

The results in Table (6) showed that CPK levels of AMI group are significantly increasing in comparison with the normal control CPK values, and slightly increase was observed in the CPK values of the UA group but it is not considerable .A significant rising was found in the AMI group when compared with the UA group.

LDH :

Data in Table (6) demonstrated that the LDH concentration is significantly elevating in the AMI group only when compared with the healthy control values, but that was not found in case of UA group. So, there was a significant elevation in the LDH concentration for the AMI group rather than UA. Either the myocardial markers (CPK , LDH) were positively correlated with elevation of MDA in serum of AMI patients; while in the UA patients group, no correlation was found between those parameters (CPK , LDH) and MDA levels. Figures (4) , (5). The CPK and LDH are found in the heart with large quantities which are needed for providing the heart muscles with a high energy that is enough for muscles contraction (48). However the blood supply from the heart requires the fast movement (muscles contraction) .

Generally the death for ischemic myocytes converting to necrotic cells which release its contents and enzymes such as (CPK and LDH), due to the destruction of cell membrane by three factors that are formed

because of the lower oxygen in cells : (1) production of internal free radicals, (2) phospholipase activation and (3)releasing of lysosome enzymes (49,50). While in UA, small numbers of myocytes die and may cause slight increase in (CPK and LDH) levels or may not . In addition, we concluded, there is a positive relationship between MDA levels and myocardial enzymes (CPK and LDH) in case of AMI. The (CPK and LDH) rise heavily in AMI, due to the increasing of ischemic period over that is enough to occur UA, which cause death of tissue (necrosis of myocardium) and releasing for these enzymes, thus this prolong ischemic period is also considered the first reason for high MDA raising, as that is previously mentioned as inflammation and role of leukocytes in ROS generation.

Antioxidants :

Ceruloplasmin :

The marked values which are recorded in table (7) referred to a significant elevation for *Cp* levels in the groups of UA and AMI in condition of the comparison to the values of normal control groups , but we observed no significant differences between UA and AMI groups in *Cp* levels . The correlation coefficient values in Figure (6) refer to a positive relationship between MDA and *Cp* levels in two patients groups. Also, we got another positive relationship between *Cp* levels and ESR levels in each patient group. Figure (7). Ceruloplasmin is an α_2 -globulin containing copper, an important extracellular antioxidant (51). *Cp* functions as an antioxidant (host defense mechanism) through ferroxidase activity, superoxide radical scavenging and copper donor activity (52). *Cp* is an important intravascular antioxidant and it protects tunica intima against free radical injury. Also, incorporating ceruloplasmin, produced a positive effect on central

haemodynamics and contractile pump capacity of the myocardium (53). *Cp* exhibits a cardioprotective effect and prevents oxygen free radical induced release of noradrenaline, a powerful vasoconstrictor (54). *Cp* synthesis and /or secretion is altered by inflammation, hormones, and copper. So the physiological factors like cancer, exercise, chronic inflammation, pregnancy increase its level (54). Here, in this study, a clear and significant rising for *Cp* conc. was gotten in UA and AMI patients. And the cause behind this result, that ceruloplasmin is an acute phase protein and is synthesized by the liver in response to tissue damage and inflammation (55). The positive correlation between MDA values and *Cp* conc. for both UA and AMI groups, is found because the increased plasma *Cp* levels are associated with the generation of oxidation products, i.e. $\cdot O_2^-$ and H_2O_2 (56). Also the positive correlation between serum *Cp* and ESR in each patient group, came from that *Cp* is α_2 - globulin in nature and the later participate in an elevation of ESR. (Reunanen,1984) and (Natalia,2005) showed high conc. of serum *Cp* in patients with AMI and with other forms of coronary heart diseases, and *Cp* decreases slowly and reaches the baseline within a month (34,57).

Albumin :

The albumin concentration was noticed as a significant lower in serum of AMI patients group table (8), when we try to compare it with the control values. Also the AMI group has significant differences against UA group in albumin

concentration. But there was no significant marker in the comparison of UA group values with normal control values. There was a negative correlation between MDA and the albumin in the group of AMI patients, while in opposite side there was no relationship between MDA and the albumin in the group of UA patients [Figure (8)]. There is an evidence for a significant antioxidant activity of the represent the major and predominant circulating antioxidant in plasma known to be exposed to continuous oxidative stress(25,58). The present study give a picture for lowering of the serum albumin significantly in patients of AMI but we did not observe that in the patients of UA. The depression, which happened in serum albumin levels in case of AMI, may be according to some causes: (1) increasing of the albumin excretion by kidney (59), (2) diffusing of the albumin into damaged tissues by means of increased permeability of blood vessels (60), (3) inflammation is considered the principle cause of a decrease in the serum albumin, however, IL-6 directly decreases the expression of albumin messenger RNA (61), and finally (4) its antioxidant function. According to the points (3) and (4): (stopping for the albumin production and consumption of the albumin to scavenge free radicals with continuously), this study proved a negative correlation between MDA levels and albumin concentration in serum of AMI patients. However, clearly that relationship occur with more time of ischemia. So more free radicals cause less albumin.

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Table (1): Serum MDA concentration as an Index of lipid peroxidation in the studied groups

Group	No.	Serum MDA (nmol/L)*
Control	35	12.87 ± 2.72 ^a
UA	35	80.94 ± 27.8 ^b
AMI	35	98.13 ± 41.17 ^b

* Each data refer to mean ± S.D. values with non identical superscripts (a, b, c) where considered significantly different ($p \leq 0.05$).

No.: number of subjects

UA: Unstable angina pectoris patients

AMI: Acute myocardial infarction patients

Table (2): Erythrocyte parameters of males for each studied group

Groups (Male)	No.	RBCs (cell/cmm)*	PCV (%)*	Hb (g/dl)*
Control	20	560.12 ± 90.27 ^a	44.04 ± 3.17 ^a	14.5 ± 0.5 ^a
UA	19	459.5 ± 51.80 ^b	40.4 ± 3.85 ^b	13.17 ± 1.25 ^b
AMI	26	515.29 ± 126.2 ^b	42.07 ± 6.29 ^b	13.77 ± 1.98 ^b

Comment as in table (1)

Table (3): Erythrocyte parameters of females for each studied group

Groups (Female)	No.	RBCs (cell/cmm)*	PCV (%)*	Hb (g/dl)*
Control	15	490.44 ± 80.23 ^a	39.3 ± 4.76 ^a	13.6 ± 0.45 ^a
UA	16	445.64 ± 58.94 ^b	36.57 ± 5.40 ^b	11.75 ± 1.79 ^b
AMI	9	434.13 ± 99.85 ^b	34.67 ± 5.31 ^b	11.14 ± 1.65 ^b

Comment as in table (1)

Table (4): Erythrocyte Sedimentation Rate For Each Studied Group

Group	No.	ESR (mm/1hr)*
Control	35	13.94 ± 6.936 ^a
UA	35	36.38 ± 20.38 ^b
AMI	35	31.69 ± 30.39 ^b

Comment as in table (1)

Table (5): White Blood Cells Count and Absolute neutrophil values For Each Studied Group

Group	No.	WBCs count (cell/cmm)*	Ab. neutr. Values (cell/cmm)*
Control	35	7435.29 ± 2178.95 ^a	4896.77 ± 1766.95 ^a
UA	35	11287.9 ± 4423.99 ^b	8655.4 ± 4237.3 ^b
AMI	35	14000 ± 5830.23 ^c	11000.16 ± 5580.12 ^c

Comment as in table (1)

Table (6): Creatine phosphokinase and lactate dehydrogenase concentrations for each patients group

Group	No.	CPK (IU/L)*	LDH (IU/L)*
Control	35	98.23 ± 22.78 ^a	106.6 ± 23.07 ^a
UA	35	130 ± 18.3 ^a	116.7 ± 23.58 ^a
AMI	35	631.08 ± 298.19 ^b	159.74 ± 20.84 ^b

Comment as in table (1)

Table (7): Ceruloplasmin concentration for each patients group

Group	No.	Ceruloplasmin (mg/L)*
Control	35	173.72 ± 86.10 ^a
UA	35	367.18 ± 120.16 ^b
AMI	35	337.07 ± 68.15 ^b

Comment as in table (1)

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Table (8): Albumin concentration for each patients group

Group	No.	Albumin (g/l) [*]
Control	35	39.7 ± 2.59 ^a
UA	35	39.17 ± 11.37 ^a
AMI	35	32.52 ± 3.82 ^b

Comment as in table (1)

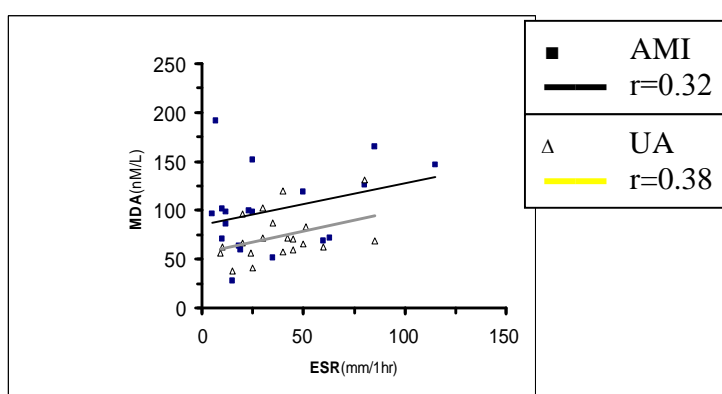


Figure (1): Relationship between serumMDA and ESR measurements in both patient groups

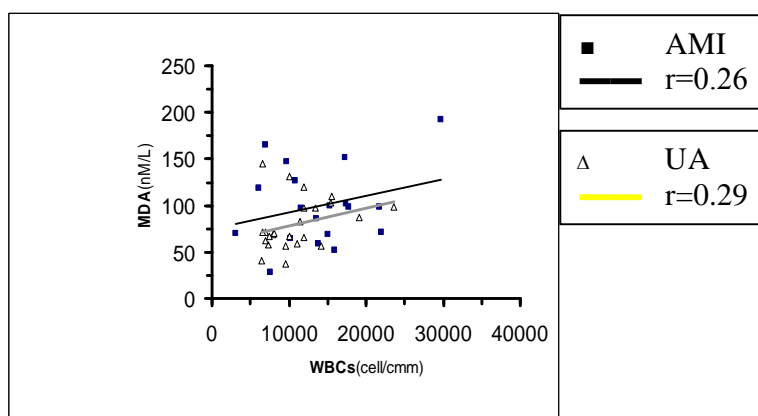


Figure (2): Relationship between serum MDA and WBCs count in both patient groups

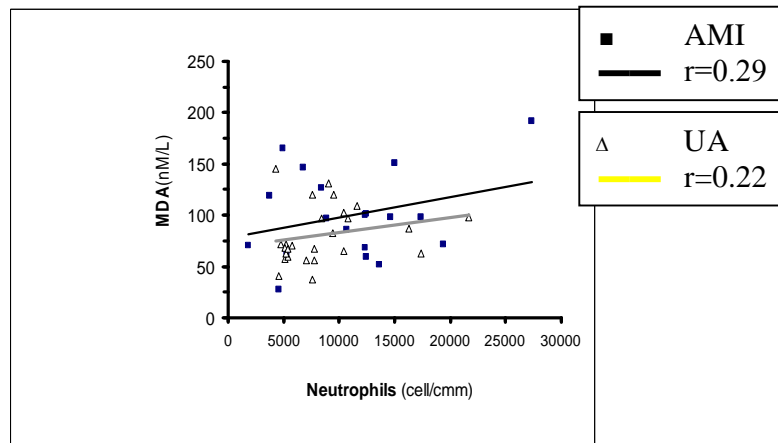


Figure (3): Relationship between serum MDA and Absolute neutrophil values in both patient groups

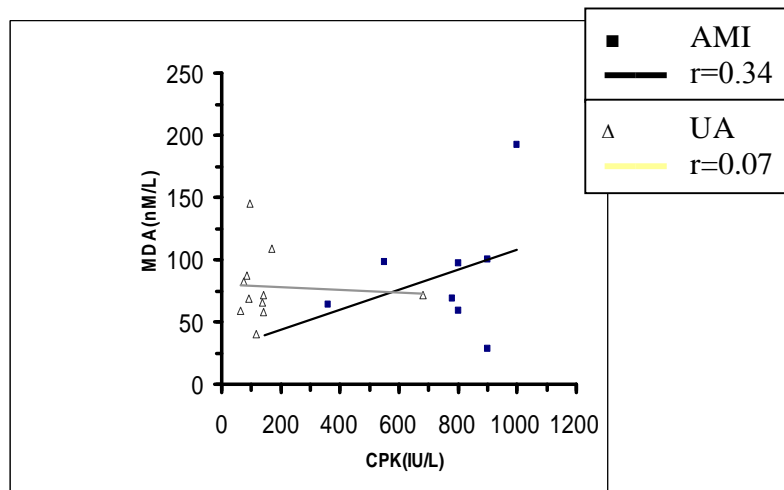
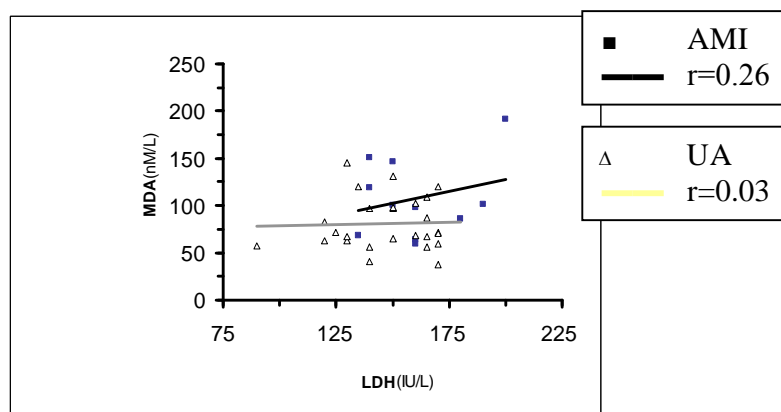


Figure (4): Relationship between serum MDA and CPK concentration in both patient groups



Figure(5): Relationship between serum MDA and LDH concentration in both patient groups

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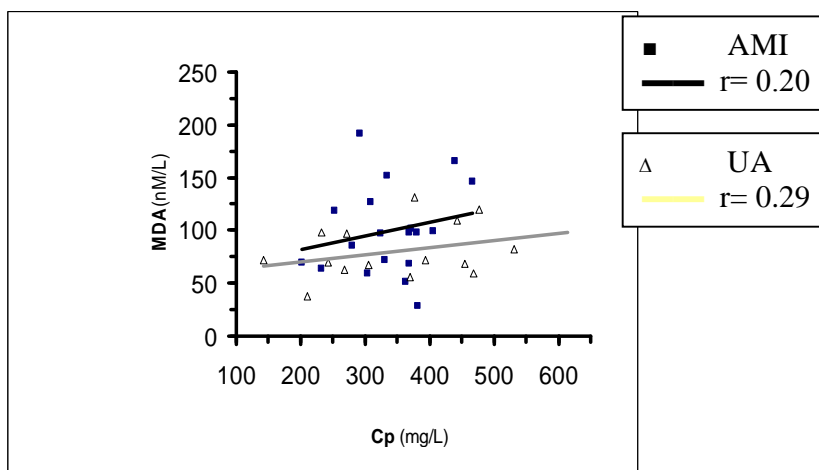


Figure (6): Relationship between serum MDA and Ceruloplasmin concentration in both patient groups

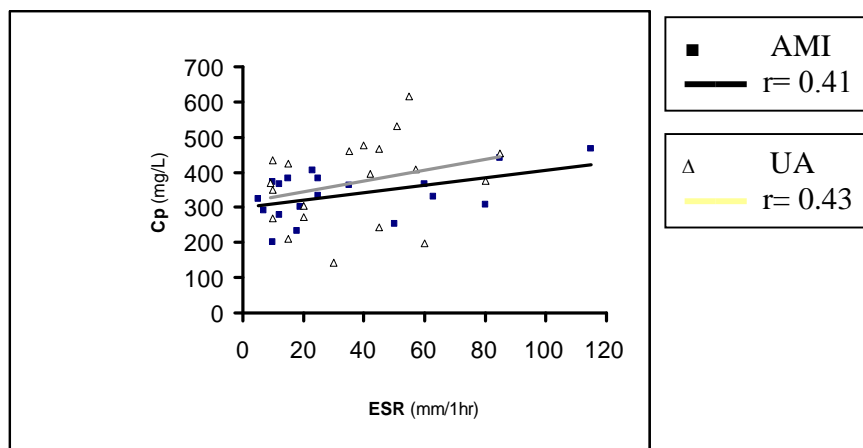


Figure (7): Relationship between serum Ceruloplasmin and ESR measurements in both patient groups

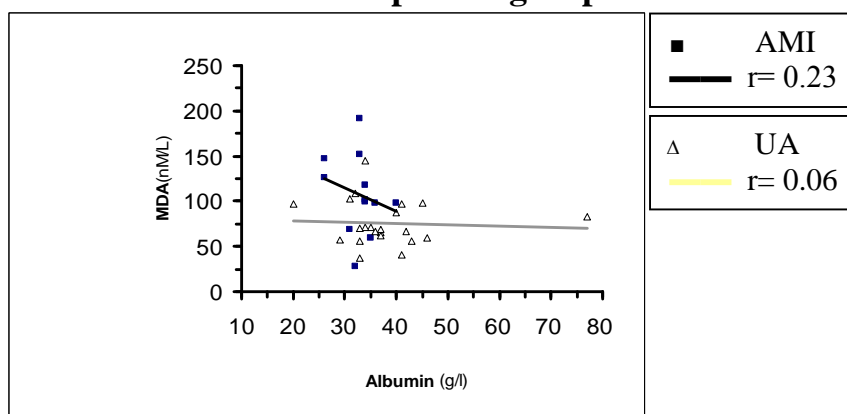


Figure (8): Relationship between serum MDA and Albumin concentration in both patient groups

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دراسة العلاقة بين المعايير الدموية وحالة المؤكسدات ومضادات الأكسدة لمرضى الذبحة الصدرية غير المستقرة واحتشاء القلب الحاد

خالد كاظم الفرطوسي* ، راند معك الصالح** ، صالح جبار بطاح***

الخلاصة:

هدفت الدراسة الحالية لبحث العلاقة بين بعض المعايير الدموية وحالة الأكسدة ومضادات الأكسدة لمرضى الذبحة الصدرية غير المستقرة واحتشاء القلب الحاد. شملت الدراسة (١٠٥) شخص موزعين إلى ثلاث مجاميع تشمل كل من مجموعة مرضى الذبحة الصدرية غير المستقرة ومجموعة مرضى احتشاء القلب الحاد ومجموعة الأصحاء (مجموعة سيطرة) وبواقع ٣٥ عينة لكل مجموعة. أظهرت نتائج الدراسة الحالية ارتفاعا معنويا في معدل ترسيب كريات الدم الحمر ، العدد الكلي لخلايا الدم البيض وعدد الخلايا العدلة المطلق لدى مرضى الذبحة الصدرية غير المستقرة واحتشاء القلب الحاد مقارنة مع مجموعة السيطرة. كما لوحظ انخفاضاً معنوياً لكل من عدد كريات الدم الحمر ، حجم كريات الدم المضغوط وخضاب الدم للمجموعتين المرضيتين مقارنة مع السيطرة. أظهرت النتائج كذلك ارتفاعاً معنوياً في الموالون داي الديهايد والسريولوبلازمين لمرضى الذبحة الصدرية غير المستقرة واحتشاء القلب الحاد مقارنة مع الأصحاء ، في حين اقتصر الارتفاع المعنوي لكل من كرياتين فوسفوكاينيز واللاكتيت ديهيدروجينيز على مرضى احتشاء القلب الحاد ، فيما كان الارتفاع طفيفاً أو غير معنوياً لكلا الإنزيمين لدى مرضى الذبحة الصدرية غير المستقرة مقارنة مع مجموعة السيطرة. انخفض مستوى الألبومين معنوياً في حالة احتشاء القلب الحاد ، في حين لم يتأثر مستواه في حالة الذبحة الصدرية غير المستقرة. أظهرت النتائج بعض العلاقات الإيجابية بين الموالون الديهايد وكل من معدل ترسيب الكريات الحمر ، عدد خلايا الدم البيض ، عدد الخلايا العدلة المطلق ، والسريولوبلازمين لدى مرضى الذبحة الصدرية ، فيما ارتبط الموالون الديهايد إيجابياً مع كل من معدل ترسيب كريات الدم الحمر ، عدد الكريات البيض ، عدد الخلايا العدلة المطلق ، السريولوبلازمين ، كرياتين فوسفوكاينيز واللاكتيت ديهيدروجينيز في حالة مرضى احتشاء القلب الحاد. كما لوحظ أن هنالك علاقة سلبية بين الموالون الديهايد والألبومين لدى مرضى احتشاء القلب الحاد.

الكلمات المفتاحية: المعايير الدموية ، حالة المؤكسدات ومضادات الأكسدة ، الذبحة الصدرية غير المستقرة ، احتشاء العضلة القلبية .

- البحث مستل من رسالة ماجستير .

* قسم علوم الحياة / كلية التربية / جامعة ذي قار / العراق

** قسم الكيمياء / كلية العلوم / جامعة ذي قار / العراق

*** دائرة صحة ذي قار / وزارة الصحة / العراق