

Is there a Correlation between Monocyte Chemoattractant Protein-1 with Autotaxin, Azurocidin-1, Apolipoprotein C-III and Elastase-2 in Male Iraqi Acute Myocardial Infraction Patients?

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Acute Myocardial infarction (AMI) is one of the important reasons of loss of life and bodily incapacity global prompt via cardiovascular diseases (CVD). AMI stimulates the innate immune system that is required to employee leukocytes to remove necrotic cells and recruit myocardial healing. To evaluate serum concentrations of Monocyte Chemoattractant Protein-1 (MCP-1), Autotaxin (ATX), Azurocidin-1 (AZU-1), Apolipoprotein C-III (APOC3) and Elastase-2 (ELA-2) in male Iraqi AMI patients and healthy controls (HCs), and explain the association of MCP-1 with ATX, AZU-1, APOC3 and ELA-2 in male Iraqi AMI patients, also explore the best parameter which can use to differentiate between AMI patients. This case-control study included 84 men aged 40–68 years. Waist circumference (WC), hip circumference (HC), thoracic circumference (TC), neck circumference (NC), height, weight, age, and further covariates were obtained via an inquiry form. They were separated into two equivalent groups: the patients group and the healthy group, serum concentrations of MCP-1, ATX, AZU-1, APOC3 and ELA-2 were estimated via ELISA. Serum of MCP-1, ATX, AZU-1, APOC3 and ELA-2 concentrations were importantly greater in patients with AMI than in HCs ($p < 0.001$). MCP-1 concentrations were importantly linked with ATX, AZU-1, APOC3 and ELA-2 in AMI cases ($p < 0.001$). The greater value of MPC-1 association was with ELA-2 ($r = 0.592$). MCP-1 exhibited the maximum value for area under curve ($AUC = 1$) in comparison to other studied biomarkers. The present data explained the role of MCP-1, ATX, AZU-1, APOC3 and ELA-2 in AMI disease; we found they have central functions in this disease, mainly obese patients. Attention must be taken to avoid confusion among risk markers and risk factors. Great serum concentrations of MCP-1 offered important correlations between risk markers and risk factors of AMI disease.

Keywords: MCP-1; Autotaxin; Apolipoprotein C-III; Acute Myocardial Infraction.

Acute Myocardial infarction (AMI), a medical problem of coronary atherosclerosis, mainly grows from the separation of an atherosclerotic plate with formation of thrombus and coronary vessel blocking¹, this leads to a severe decrease in the supply of blood to the heart muscle². Risk factors such as hyperlipidemia hypertension,

smoking, obesity and diabetes mellitus were stated to be related with augmented AMI risk³; nonetheless, the fundamental mechanisms which lead to AMI not yet determined. It's, a complex multifaceted illness, is happened via together genetic tendency and environmental causes⁴.

Previous paper indicated that abnormal immune reply grows through AMI which is harmful and involves to death of cell after the abuses⁵. AMI is described via of adhesion molecules, proinflammatory cytokines expression, and essentially, chemokines that help the leukocytes infiltration into area of infarct, expression of harmful mediators and complement system activation⁶.

Chemokines are inflammatory cytokines subset that contribute in the leukocytes employment to locations of irritation and are assembled into subfamilies: CXC and CC chemokines⁷. MCP-1 is one of the well-studied CC chemokine; it's shown an important relationship with danger elements for CVD like hypertension, renal insufficiency, diabetes and age⁸. MCP-1 may activate the atherosclerosis (AS) progress and development of via monocyte permeation activating and fat-laden foam cells growth⁹. The most important, one research showed that MCP-1 could exert a central function in AMI damage and inverse remodeling by various possible mechanisms¹⁰.

Autotaxin (ATX) which is overexpressed in valves of mineralized aortic is a lysophospholipase-C that hydrolyses ox-Phos into lysophosphatitic acid (LPA)¹¹. LPA is a highly bioactive complex which activates several inflammatory manners involving fibrosis. The most important, one research showed that LPA and ATX are included in the AS pathobiology¹². LPA encourages hypertrophy of cardiomyocyte through encouraging the initiation signals of Rho-intermediated¹³. Nonetheless, whether LPA/ATX signalling is included in the development of obesity-linked AMI is still unidentified, previous paper had established that LPA, manufactured via ATX, stimulates irritation and controls monocytoysis¹⁴.

Azurocidin (AZU) an antimicrobial protein involved in the serprocidin subclass of chymotrypsin-like proteases^{15,16}. AZU is existed in secretory vesicles of the neutrophils and granules of azurophilic¹⁷. Adhesion of Neutrophilic to the endothelium through irritation stimulates the excrete of basal AZU from secretory vesicles and granules of azurophilic^{16,17}. AZU, as a protein with multifunctional, is an essential compound in the reply of host through contagion¹⁵. The physiological roles of AZU involve monocyte

recruitment stimulation to the site of irritation, increase phagocytosis of macrophage, and antimicrobial action¹⁵⁻¹⁷. It's similarly rises effect of macromolecular via cellular barriers breaking and permeability of endothelial¹⁸.

Apolipoprotein C-III (APOC3) was recognized from about six decades as a controller of circulation triglyceride rich lipoproteins (TRLs)¹⁹. Furthermore to possess great ability to prevent mediated lipolysis of LPL; APOC3 similarly been revealed to assist hepatic VLDL emission and assembly. It is not only a main TRLs modulator but similarly participates to the atherogenicity of HDL and LDL molecules²⁰. Clear data show that APOC3 has a number of further roles, involving irritation and endothelial function modulation²¹. The association of APOC3 with high levels of TG and CVD risk has been established in broad humans and animal data²². Additionally, assumed that TRL fragments are currently documented as an underlying danger issue for CVD and that damage APOC3 function mutations which described by a strong dropping of serum TG and decreased the danger of CVD^{23,24}.

Neutrophil elastase (NE) is a powerful serine protease that has specificity of extensive substrate²⁵. Investigational data have possibly dealt with the damaging nature of NE, but a previous paper shows that NE can irritate a diversity of proinflammatory replies, like TGF manufacture in smooth muscle cells of bronchial and IL-8 excrete from epithelium of bronchial, furthermore, removal of NE in mice produce a decline of serum inflammatory parameters like MCP-1, IL-1 and TNF α ,²⁶. Other research has similarly established NE in atherosclerotic plaque shoulders of macrophage-rich human²⁷.

Elastase-2 (ELA-2), an angiotensin-II (Ang-2) generating chymotrypsin serine protease elastase family member 2A, is extensively originate in many tissues, like the liver, heart, pancreas, kidney of rodents, lung and blood vessels (mesenteric and carotid arteries)²⁸. The significance of this purposeful alternate pathway for Ang-2 reproduction was confirmed in carotid, heart, spontaneously hypertensive (SHR) and mesenteric arteries from normotensive rats²⁵. Furthermore, prolonged treatment by enalapril, an inhibitor of ACE, raises ELA-2 involvement to Ang-2 reproduction in the normotensive and SHR carotid

artery of rats²⁹. Therefore, AMI may be importantly rises reproduction of Ang-2; we wanted to explore what if ELA-2 participates to vascular reproduction of Ang-2 and perhaps to cardiac injury by AMI.

Therefore, the main goals of this research were to explore the correlation of MCP-1 with ATX, AZU-1, APOC3 and ELA-2 in Male Iraqi MI Patients and to determine the significance of these risk markers in AMI disease, also to estimate the role of these variables as a predictors of the diagnosis of AMI disease in men Iraqi patients.

METHOD AND MATERIALS

The current case-control study employed 42 patients with AMI disease from several private laboratories in Al-Fallujah, Iraq, between July 2018 and April 2019. The same number of healthy persons matched age; gender and ethnic background were selected for comparison. The patients recruited all met the following inclusion criteria: 40 years < age < 68 years, we excluded patients with any of the following criteria: prior history of AMI, rescue angioplasty, previous history of coronary artery bypass graft, severe acute heart failure, also patients who had infectious diseases were omitted from the study.

Height in centimeters (cm), total body weight in kilograms (kg) and also WC, HC, TC and NC in cm were determined by an anthropometric scale, with a precision of 500 g and 1 cm,

accurately standardized. Body mass index (BMI) was calculated via body weight (kg) divided on the square of length (m²).

Blood samples (5 ml) were collected in plain tubes. The samples were centrifuged at a speed of 3000 xg at 250C for 20 minutes to isolate the blood cells and the serum. Then the serum was pipetted and kept at -200C till use. Serum concentrations of MCP-1, ATX, and AZU-1, APOC3, ELA-2 and hs-cTnT were determined by enzyme-linked immunosorbent assay (ELISA). Kits were obtained from Mybiosource Inc (Southern California, San Diego, USA). The manufacturer's guidelines were followed. Ethical approval was gotten from the Ethics Committee and Medical Research Committee from university Of Anbar. All participants gave their printed informed agreement.

Statistical analysis

The results of this paper were stated as the mean, standard deviation (SD) and standard error of mean (SEM). The difference among variables was verified through an unpaired t-test. The correlations between variables were confirmed by Spearman correlation analysis. The Receiver Operating Characteristics (ROC) curve investigation was used to explore the best variable which can be used to identification of AMI disease through Area under curve (AUC) of the ROC curve for logistic regression which offered along with 95% confidence intervals (CI). All the statistical

Table 1. Baseline characteristics and clinical outcomes in AMI patients and controls

Parameter	Healthy controls			Patients (MI)			p-value
	Mean	SD	SEM	Mean	SD	SEM	
Age years	55.96	10.21	1.921	57.45	10.53	2.121	0.546
WC cm	84.55	6.22	1.27	109.7	9.995	1.29	<0.0001
HC cm	104.8	9.194	1.877	116.4	9.782	1.263	<0.0001
TC cm	99.69	7.885	1.61	106.4	8.354	1.078	0.0010
NC cm	40.22	3.146	0.642	42.98	3.551	0.458	0.0013
W/H	0.808	0.0286	0.006	1.036	0.124	0.016	<0.0001
W/T	0.849	0.0339	0.007	1.131	0.128	0.017	<0.0001
W/N	2.106	0.127	0.026	2.801	0.315	0.041	<0.0001
BMI Kg/m ²	24.49	2.399	0.490	32.75	4.054	0.523	<0.0001
MCP-1 ng/mL	31.31	5.422	0.817	70.32	17.72	2.672	<0.0001
ATX ng/mL	364.4	135.9	20.49	823	356	53.67	<0.0001
AZU-1 pg/mL	13.98	3.544	0.534	23.85	8.531	1.286	<0.0001
APOC3 pg/mL	212.2	86.79	13.08	388.9	153.3	23.1	<0.0001
ELA-2 ng/mL	16.39	7.247	1.092	47.57	18.67	2.814	<0.0001
hs-cTnT ng/L	0.500	0.173	0.026	9.246	3.426	0.517	<0.0001

analyses were done with GraphPad Prism version 7.04 (GraphPad Software, San Diego, CA). A p-value below 0.05 was appointed to be important.

RESULTS

Study group comprised from 42 patients diagnosed with AMI disease. Patients with

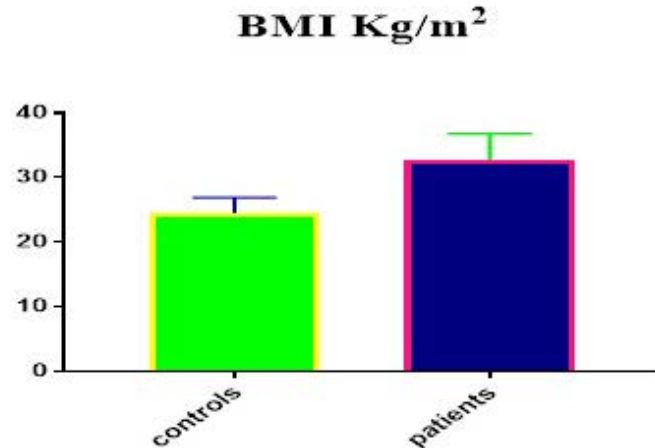


fig. (1): mean+ S.D for BMI in control and cases

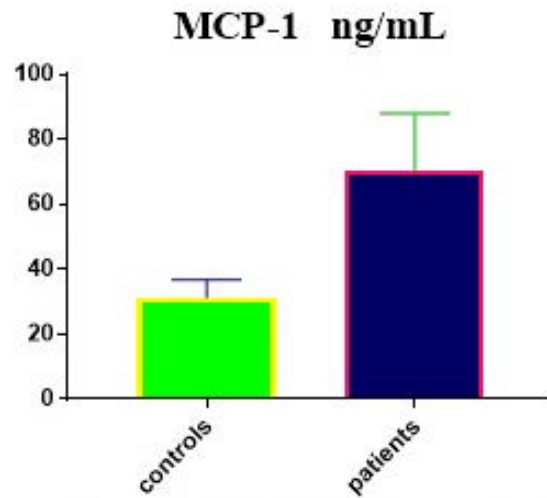


fig. (2): mean+ S.D for MCP-1 in control and cases

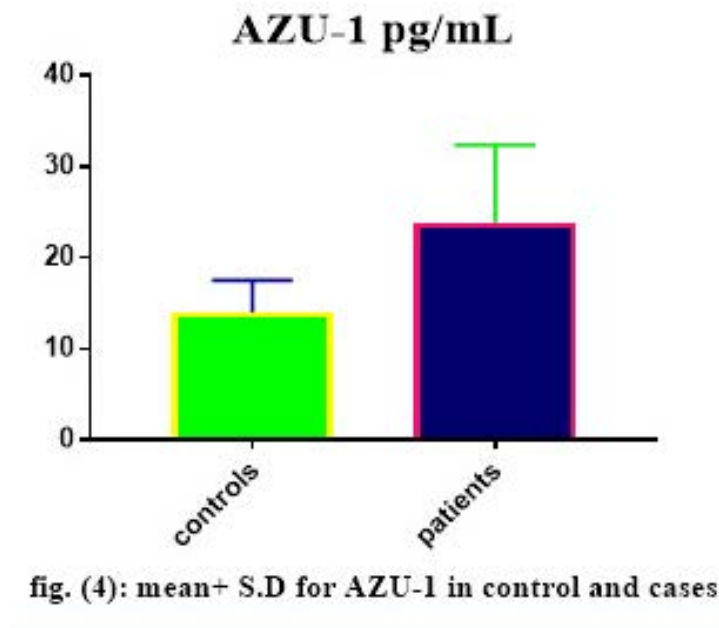
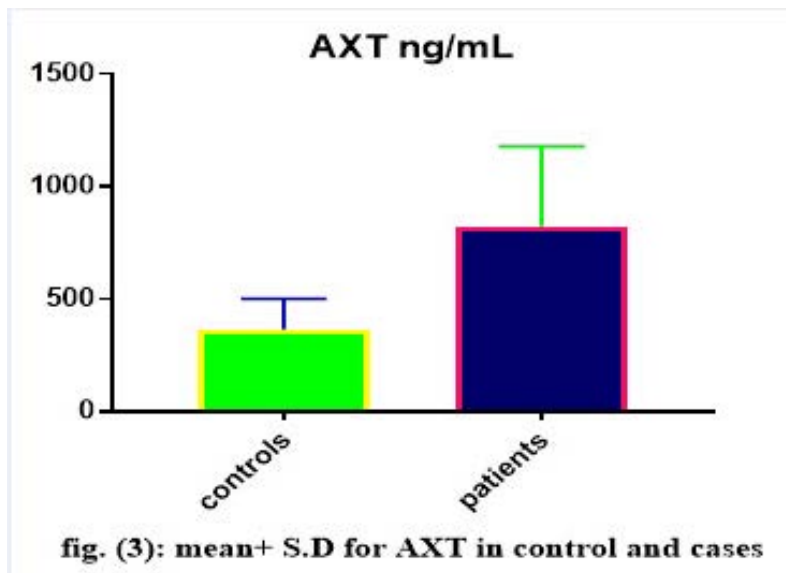
Table 2. Pearson's correlation coefficient of MCP-1 with studied parameters

	MCP-1	ATX	AZU-1	APOC3	ELA-2	BMI	hs-cTnT ng/L
MCP-1 pg/ml		0.530	0.472	0.489	0.592	0.461	0.686
ATX ng/ml			0.295	0.390	0.483	0.349	0.628
AZU-1 pg/ml				0.351	0.453	0.415	0.415
APOC3 pg/ml					0.463	0.422	0.563
ELA-2 ng/ml						0.333	0.666
BMI Kg/m2							0.440

P-values for all parameters less than 0.01

identified risk elements like hypertension, diabetes mellitus, and smoking were excepted from this research. WC, HC, TC, NC, W/H, W/T, W/N and BMI of these patients were calculated. These data were compared with 42 healthy men (control group). This study involved men aged 40 years and over. The results for anthropometric measurements (AMs) and BMI (Kg/m²) are presented in Table 1. As shown in this table, the mean age (years) of the men was 57.45 (10.53) in the AMI group and

55.96 (10.21) in the control group. The mean WC, HC, TC, NC, W/H, W/T and W/N were found to be more in the AMI group when compared that of the control group ($p < 0.01$). The BMI in the study group was greater than that of the control group ($p < 0.001$) (Table 1; fig. 1). On the other hand, Serum MCP-1, ATX, AZU-1, APOC3, ELA-2 and hs-cTnT concentrations, as shown in Table 1 (figure 2-7 respectively) increased significantly ($P < 0.0001$) in AMI group than in HCs group.



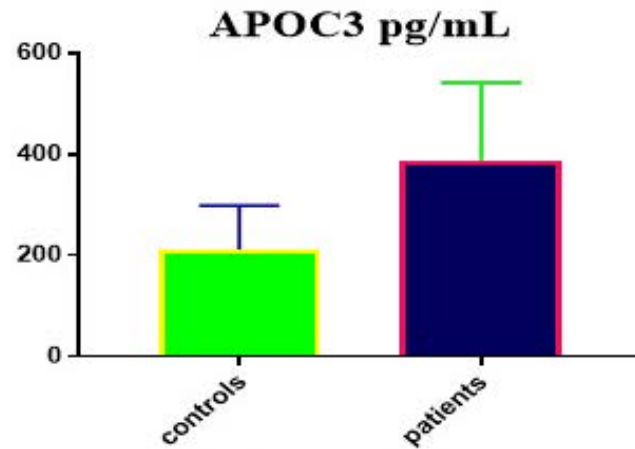


fig. (5): mean+ S.D for APOC3 in control and cases

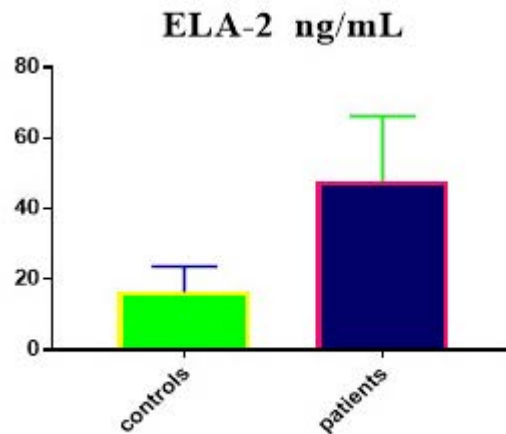


fig. (6): mean+ S.D for ELA-2 in control and cases

Table 3. Diagnostic Criteria of the ROC Curve for Tested Parameters in MI Patients

Parameter	AUC	Std. Error	95% confidence interval	P-value
WC cm	0.9972	0.0033	0.9907 to 1.004	<0.0001
HC cm	0.8014	0.0474	0.7085 to 0.8943	<0.0001
TC cm	0.6990	0.05849	0.5843 to 0.8136	0.0046
NC cm	0.7017	0.0585	0.5871 to 0.8164	0.0040
W/H	0.9903	0.0072	0.9762 to 1.004	<0.0001
W/T	0.9917	0.0087	0.9746 to 1.009	<0.0001
W/N	1	0	1 to 1	<0.0001
BMI kg/m ²	0.9757	0.0169	0.9426 to 1.009	<0.0001
hs-cTnT ng/L	1	0	1 to 1	<0.0001
MCP-1 ng/mL	1	0	1 to 1	<0.0001
ATX ng/mL	0.8982	0.0321	0.8354 to 0.9611	<0.0001
AZU-1 pg/mL	0.8580	0.0389	0.7817 to 0.9342	<0.0001
APOC3 pg/mL	0.8301	0.0438	0.7443 to 0.9158	<0.0001
ELA-2 ng/mL	0.9685	0.0147	0.9397 to 0.9972	<0.0001

Correlation coefficients (r) of MCP-1 with ATX, AZU-1, APOC3-, ELA-2, BMI and hs-cTnT in AMI group (Table 2 and figures 8-13, respectively) show the following values of r (0.530, 0.472, 0.489, 0.592, 0.461 and 0.686 respectively, with p-values less than 0.001 for all), hs-cTnT and ELA-2 showed the strongest correlation value with MCP-1, while AZU-1 and BMI presented the weakest correlation value with MCP-1, but all these parameters exhibited positive correlations of moderate strength.

ROC curve examination showed that the best biomarkers were able to discriminate patients with AMI from HCs with AUC=1; SE=0, 95% CI=1 to 1 and p<0.0001 are hs-cTnT and MCP-1 as shown in table 3 (figures 14 and 15), also the anthropometric parameter W/N display the same

result above (table 3: fig. 16), Likewise, the other AMs presented in descending order WC, W/T, W/H, BMI, HC, NC and TC with the following values ??for each variable (AUC=0.9972; SE=0.0033 and 95% CI=0.9907 to 1.004, AUC=0.9917; SE=0.0087 and 95% CI=0.9746 to 1.009, AUC=0.9903; SE=0.0072 and 95% CI=0.9762 to 1.004, AUC=0.97571; SE=0.0169 and 95% CI=0.9426 to 1.009, AUC=0.8014; SE=0.0474 and 95% CI=0.7085 to 0.8943, AUC=0.7017; SE=0.0585 and 95% CI=0.5871 to 0.8164 and AUC=0.6990; SE=0.05849 and 95% CI=0.5843 to 0.8136 with p-values less than 0.01 for all, respectively), as shown in table 3.

The remaining variables studied presented in descending order ELA-2, ATX, AZU-1 and APOC3 with the following values ??for each

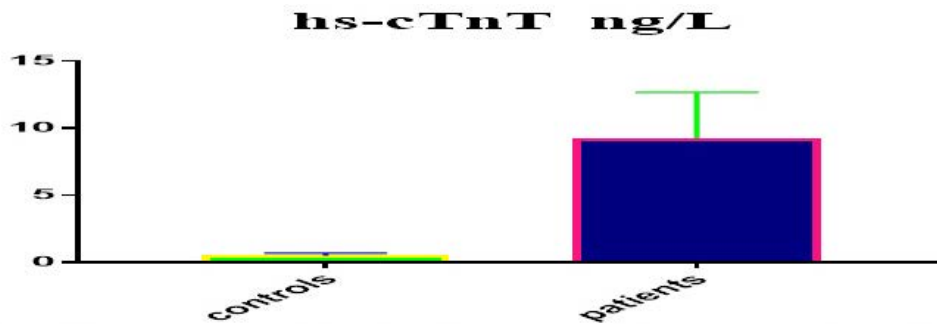


fig. (7): mean+S.D for hs-cTnT in control and cases

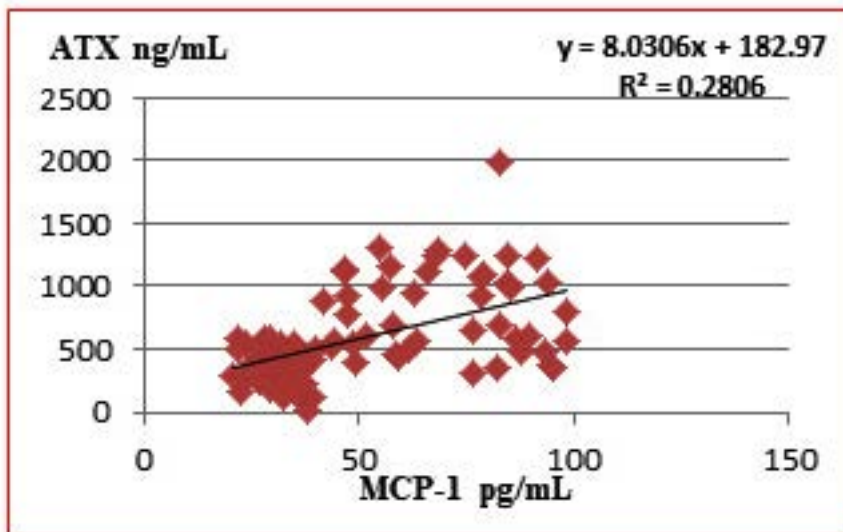


Fig. 8: Association of MCP-1 with ATX

variable (AUC=0.9685; SE=0.0147 and 95% CI=0.9397 to 0.9972, AUC=0.8982; SE=0.0321 and 95% CI=0.8354 to 0.9611, AUC=0.8580; SE=0.0389; 95% CI=0.7817 to 0.9342 and AUC=0.8301; SE=0.0438; 95% CI=0.7443 to 0.9158, with $p < 0.0001$ for all, respectively), as shown in table 3 (figures 17-20).

DISCUSSION

The data of the current study shown that AMI group had importantly greater serum MCP-1, ATX, AZU-1, APOC3 and ELA-2 concentrations when compared to the HCs group; this may

show that the concentrations of these biomarkers gradually rise with severity of the AMI disease.

AMI activates native inflammatory replies which yield the employment of leukocytes and then damage of myocardial and healing. MCP-1, exert a central function in the inflammatory reply and injury of myocardial after reperfusion/ischemia [30]. It is mainly included in the AS pathogenesis and in the AS pathological results, may participates to plate deterioration, and it is a essential facilitator in opposing rebuilding after AMI injury³¹.

Signals from investigational and medical papers shown that C-C chemokines, particularly MCP-1, exert critical pathogenic functions in

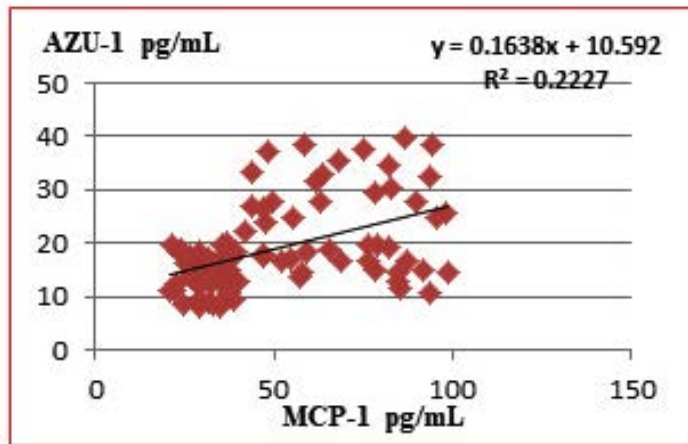


Fig. 9: Association of MCP-1 with AZU-1

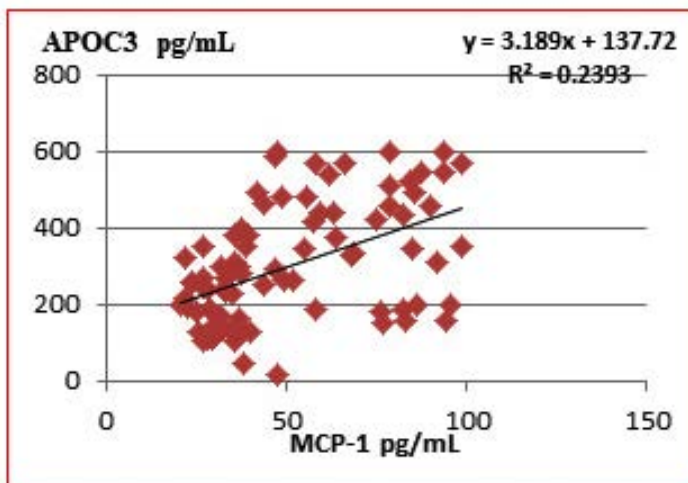


Fig. 10: Association of MCP-1 with APOC3

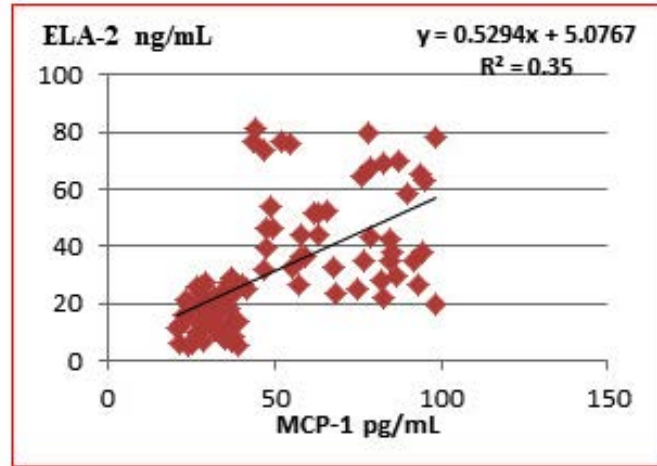


Fig. 11: Association of MCP-1 with ELA-2

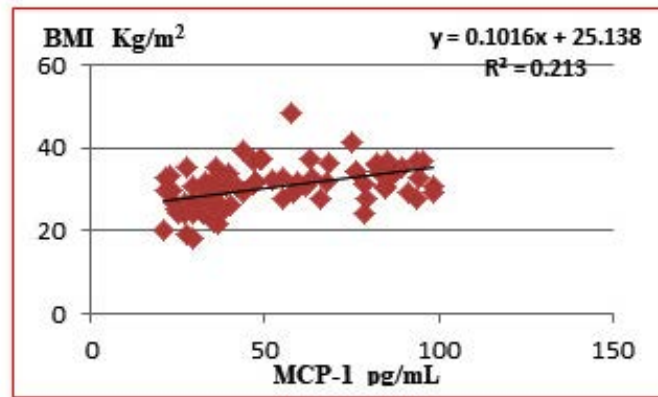


Fig. 12: Association of MCP-1 with BMI

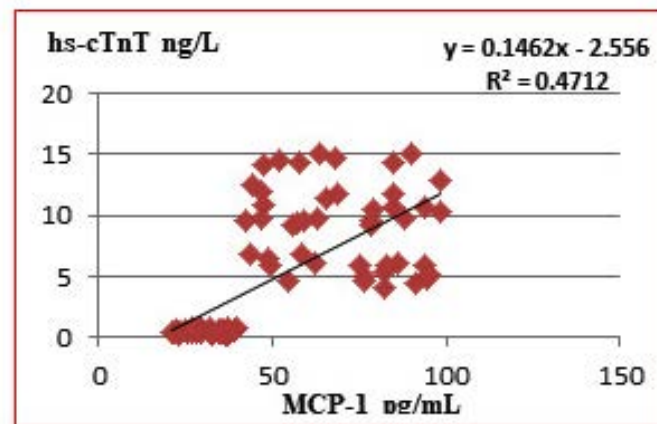


Fig. 13: Association of MCP-1 with hs-cTnT

CVD³². Previous study showed that serum levels of MCP-1 were constantly raised in AMI patients during a 7-day hospitalization period in compared to HCs³³. Furthermore, low concentrations of MCP-1 may inhibit AMI and as a result inverse rebuilding by the inhibition of oxidative stress related to macrophage³⁴. The reducing of macrophage stimulation, reduced myofibroblast penetration and inhibition of synthesis of proinflammatory

markers, may explanation reducing AMI disease³⁵. There are signs showing that low concentrations of MCP-1 may inhibit apoptosis of myocardial³⁶. Our data produce further documents for the irritation theory of pathogenesis of AS and for additional study to examine MCP-1 function as a proinflammatory danger element; we suggest that higher concentrations of MCP-1, may serving as a biomarker of inflammatory action, similarly may be

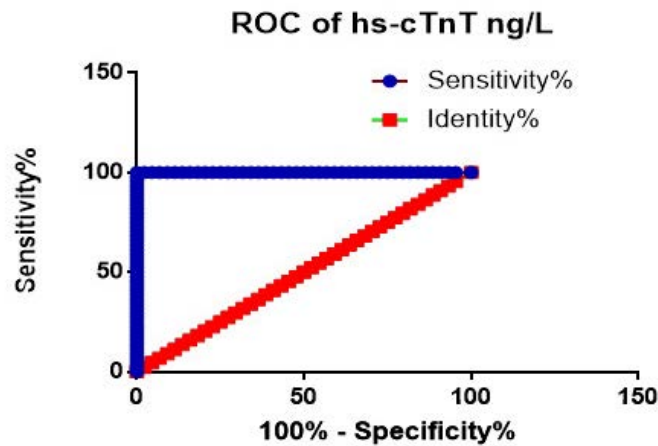


Fig. 14: ROC curve displaying AUC of hs-cTnT in MI patients

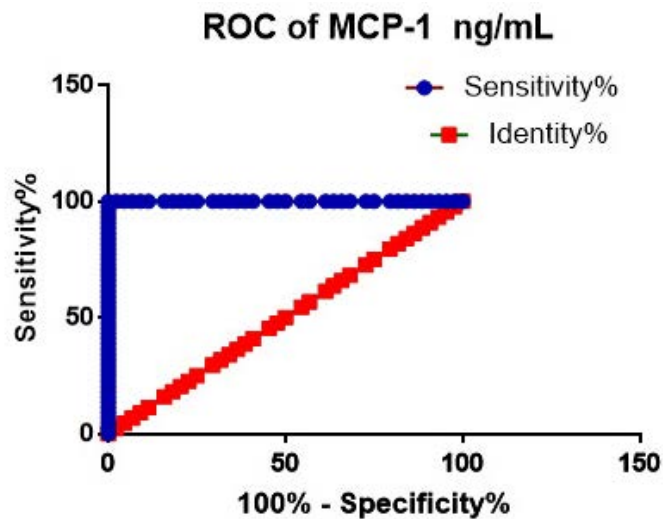


Fig. 15: ROC curve displaying AUC of MCP-1 in MI patients

a risk marker of AMI disease. Our data suggest that MCP-1 may be an initial predictor of AMI disease and an initial marker of its normal growth. MCP-1 sources are macrophage and endothelia like cells³⁷, which are identified to exert a important function in progress of AMI and plate development. In vitro data have discovered that endothelial cells are

able to yield MCP-1 in reply to LDL a significant activating element of AS³⁸.

Present study found important positive associations among serum levels of MCP-1 with ATX, AZU-1, APOC3 and ELA-2 in male Iraqi AMI patients. In the current study, MCP-1 was positively associated with markers of AMI like

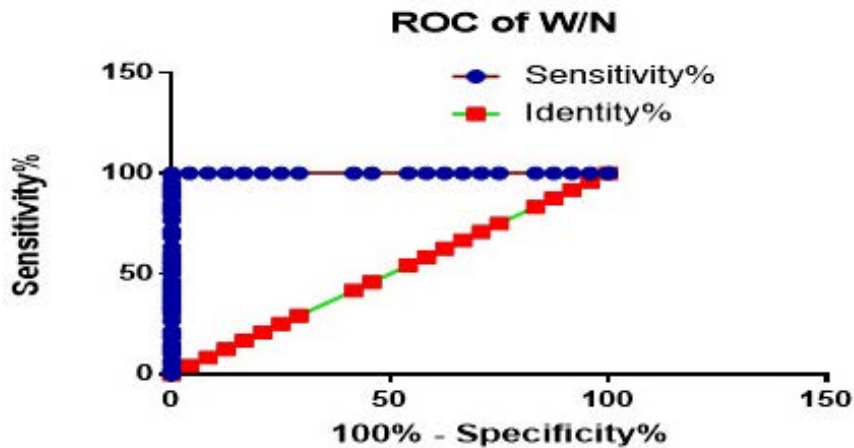


Fig. 16: ROC curve displaying AUC of W/N in MI patients

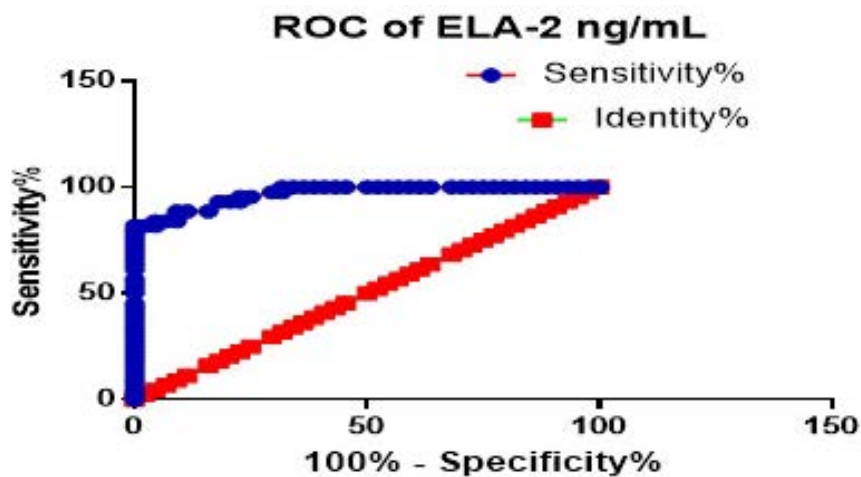


Fig. 17: ROC curve displaying AUC of ELA-2 in MI patients

hs-cTnT and BMI³⁹, clarified these results via the augmented action of circulating macrophage and endothelia like cells (which excrete MCP-1) that is linked to more wide necrosis of myocardial and then product in worse diagnosis of AMI, so, we look for MCP-1 as a dependable danger biomarker in AMI patients.

The strong association among BMI and danger of occurrence AMI was detected in this study which has led to rising discriminate of the autonomous structural and functional effects of obesity itself on the heart muscle⁴⁰. This study indicated that greater BMI was associated with

augmented AMI risk. BMI growths with together lean and fat weight, so is an alternate, instead of a precise measure, of accurate body obesity. AMs, like WC or direct body lipid assessing modalities, are possible to further precisely reveal accurate fatness weight.

Current data show that ATX exerts a central function in facilitating heart muscle morbidity in men Iraqi AMI patients. The raised concentrations of ATX were related with morbidity of heart muscle, and we revealed that ATX encouraged cardiac damages in these patients. Previous paper had established that ATX is

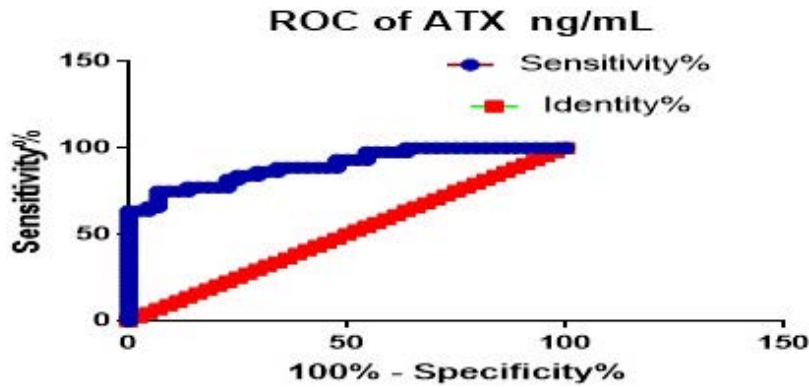


Fig. 18: ROC curve displaying AUC of ATX in MI patients

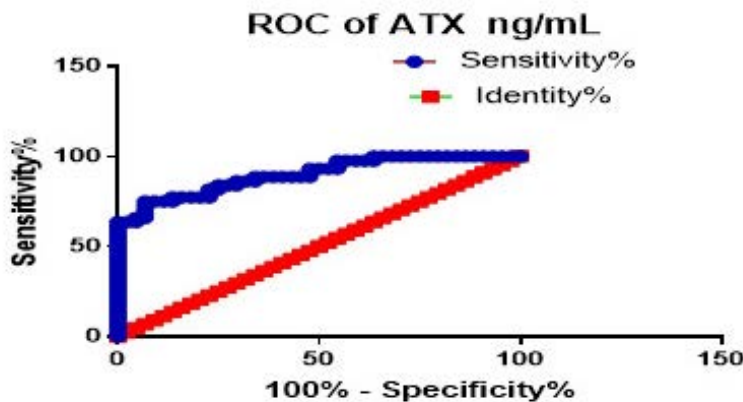


Fig. 19: ROC curve displaying AUC AUZ-1 in MI patients

essentially manufactured in reply to irritation and the great concentrations of ATX continue in relationship with inflammatory illnesses⁴¹. The manufacture of inflammatory markers, like and IL-1 β and TNF-a, in irritated and injured cell is a indicator for augmented expression of ATX⁴². Nonetheless, whether its impact inflammatory reply or action is still unidentified, with this fatty tissues are main source of ATX and other inflammatory markers in obese individuals⁴³. Together human

and mouse data establish the close link among ATX concentration and biomarker of CVD.

The present data consistent with the fact that irritation exerts a big role in the AMI pathophysiological mechanisms⁴⁴. Recent research confirmed the function of neutrophils and irritation in the atherosclerotic development, cardiac rebuilding and healing after AMI⁴⁵. The positive important association of AZU-1 concentration with the MCP-1 in the present paper may show

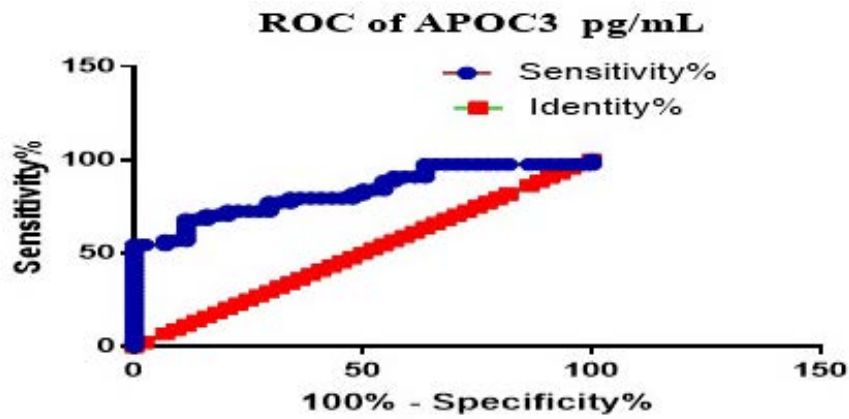


Fig. 20: ROC curve displaying AUC of APOC3 in MI patients

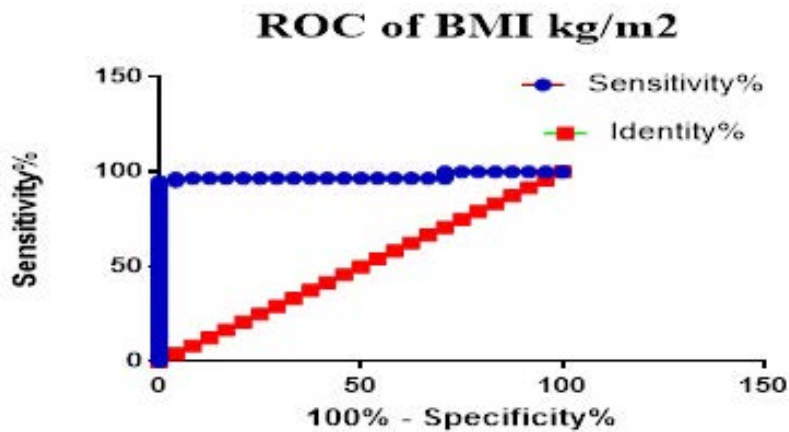


Fig. 21: ROC curve displaying AUC of BMI in MI patients

the state of persistent inflammatory as the illness severity rises. The present results revealed that AZU-1 concentrations were positively associated with BMI values. This finding can lead scientists to propose new research to present AZU-1 to cardiology practice as a predictive parameter in the next years. The site of AZU-1 in the secretory granules, which are the first to be mobilized upon stimulation of neutrophil, may clarify this increase of AZU-1 levels in AMI patients than in HCs⁴⁶.

This study designed to determine high levels of APOC3 that could stimulate the further danger elements in AMI patients. Main role of APOC3 has been identified to the surface of certain complexes of lipoproteins that consecutively stimulated an inflammatory incident which absorbs a deposit of atherosclerotic⁴⁷. In consistent with our data, previous study on APOC3 levels has revealed that greater concentrations have linked to a number of pathological situations such as CVD⁴⁸. A molecular disorder on APOC3 is exposed to outcome in receptor facilitated cracking of TRLs which may grow AMI⁴⁹. From our data, in addition to other data, could be established that reduce the amount of APOC3 is a dependable variable for level of lipoprotein related CAD danger which causes AMI⁵⁰.

Heart failure is related with renin-angiotensin-system (RAS) increase effectiveness, where AMI importantly raises the expression of a number of RAS elements, involving Ang-2 concentrations in the heart muscle⁵¹. Because of AMI causes strong RAS stimulation and ELA-2 are augmented in CVD like arterial hypertension of arterial²⁹, we wanted to explore whether ELA-2 participates to rise AMI disease in Iraqi male. Our finding shows that ELA-2 changes cardiac job and may cause AMI. Additional support to this finding originates from documents from recent study⁵², displaying that ELA-2 KO mice show declined cardiac autonomic destabilize and cardiac rate described by augmented parasympathetic manner and decreased cardiac sympathetic. Current results show that ELA-2 may exert a main function on peripheral resistance and essential heart function.

Identification of a new biomarker indicating AMI before the rise of troponins could be of important medical value due to the narrow troponins sensitivity in the initial stage of AMI. Our results showed that the MCP-1 and W/N are

the best sensitive markers of risk for repeated AMI, they have the same diagnostic power as hs-cTnT (AUC=1), In second place and descending WC, W/T, W/H and BMI, this unequivocally establishes that AMs are dependable danger elements for identification and treatment of AMI disease. We establish the measures of central fatness (W/N, WC, W/T and W/H) to be better to BMI in predicting clinical severity of AMI, while NC and TC have no influence on it.

Central fatness is extra powerfully related with AMI danger than common fatness. The adipose tissue deposition is related with systemic irritation, which has a straight influence on AMI danger. When calculated alone, BMI is insufficient for detecting persons at augmented danger of AMI as it does not distinguish among and fat-free weight and obese. Nonetheless, AMs of central fatness have greater specificity and sensitivity. AMs are similarly extra sensitive to alterations linked to way of life and should be merged into the calculation of AMI danger elements, mainly as soon as evaluating the danger in elderly men.

Several limitations are found in this paper. First, our subjects were joined from private test centers which may give choice bias. Second, the absence of long-term follow up and in-hospital, also the absence of concurrent determination of additional inflammatory parameters and compare them with calculated biomarkers. Third, the absence of pharmacological examination which confirm the influences of suppression for studied biomarkers in treatment of AMI disease. Fourth, adipose tissue data was not obtainable for all AMI participants; as a result, statistics on distribution of body fat was built on AMs basically. Fifth, a small sample size of this case control study which may be not success to explain the pivotal correlation among MCP-1 concentrations and further calculated variables in AMI disease.

In conclusion, there was a direct positive relationship among MCP-1 serum levels with ATX, AZU-1, APOC3 and ELA-2 in male Iraqi AMI patients. Therefore, these parameters may be used as biomarkers for danger extrapolation for AMI disease and additional studies of these parameters as healing marks are required. The investigated AMs may be used in the future as free predictive and/or diagnostic indicators especially if they are assessed and confirmed in papers with

a bigger sample size. The data submit that there is a positive relationship of obesity linked AMIs with AMI danger elements. W/N seemed to be a superior indicator of AMI danger than W/N, WC, W/T W/H, and BMI. We established that serum concentration of MCP-1 had a positive relationship with hs-cTnT, and so, several biomarker of heart-tissue source have been tested in the identification of AMI from past to current day. Depending on the results of this study, we recommend the following variables: hs-cTnT, MCP-1 and W/N for accurate identification of AMI disease, which may be the gold-standard biomarkers for diagnosing AMI with high precision. Lastly, these data should be established in prospective papers through together prolonged and AMI disease individuals. Further cardiac-specific results, involving progress of arrhythmias and heart failure, should be investigated.

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