AIM OF THE WORK

The aim of the present work was to determine the level of lipoprotein associated phospholipase A_2 activity in sera of patients with myocardial infarction in comparison with other cardiac markers.

SUBJECTS

The study was conducted on thirty selected patients admitted to Cardiology Department at Alexandria Main University Hospital, whose ages exceeded 30 years and suffered from acute myocardial infarction diagnosed by electrocardiogram. Patients with diabetes mellitus, liver, renal and thyroid diseases were excluded based on clinical examination and clinical history.

Twenty healthy individuals of matched age and sex, with no history of diabetes mellitus, atherosclerosis, cancer or medications, served as a control group.

All subjects included in the study signed a written informed consent before enrollment. The approval of medical Ethics Committee was obtained.

METHODS

All patients and controls (n=50) included in the present study were subjected to the following:

1. Full history taking including:
   - Family history.
   - Smoking habits.
   - Alcohol consumption.
   - Medical history: hypertension and diabetes mellitus.
   - Drug history: statin therapy.
   - Physical activity.
2. Detailed clinical examination including:
   - Blood pressure measurement.
   - Electrocardiogram.
3. Laboratory Investigations including:
   a. Fasting serum glucose. [179]
   b. Lipid profile. [180]
   c. LDH and AST. [180]
   d. High sensitivity C-Reactive protein. [181]
   e. CK- total and CK-MB mass. [182]
   f. Troponin I. [183,184]
   g. Quantitative detection of lipoprotein associated phospholipase A_2 activity with enzymatic asssay. [185]

Specimen Collection and Storage:

1- Five milliliters (5 ml) venous blood sample were drawn from every patient on admission and emptied in a plain tube (red capped vaccutainer) for chemical testing. Blood was allowed to clot for 30 minutes then centrifuged (at 1000-1200 g for 10 minutes, at room temperature). The separated serum was divided into two parts, the first of which was analyzed on Dimension RxL Max analyzer. (Siemens Healthcare Diagnostics, Newark, DE 19714, U.S.A) for chemical tests except hs-CRP was analysed on Cobas c311 (Roche Diagnostics , D1203-03, Germany) . The second part (1.5 ml) was delivered into eppendorf microtube and was kept at – 80 °C for estimation of Lp-PLA_2 activity level.

2- Three milliliters (3 ml) venous blood sample were drawn from every patient after twelve hours (12 hs) fasting and emptied in a plain tube (red capped vaccutainer) for chemical testing. Blood was allowed to clot for 30 minutes then centrifuged (at 1000-1200 g for 10 minutes, at room temperature). The separated serum was analyzed on Dimension RxL Max analyzer (Siemens Healthcare Diagnostics, Newark, DE 19714, U.S.A) for fasting serum glucose and lipid profile.

Principle of tests:

1-CK-MB mass: [182]

The method is one-step enzyme immunoassay based on the "sandwich principle". The sample is incubated with chromium dioxide particles coated with monoclonal antibodies specific for CKB subunit, and conjugate reagent (β-galactosidase labeled monoclonal antibodies specific for CKMB isoenzyme). A particle/CKMB/conjugate sandwich forms during the incubation period. Unbound conjugate is removed by magnetic separation and washing. The sandwich bound β-galactosidase is combined with a chromogenic substrate chlorophenol red-β-d-galactopyranoside (CPRG). Hydrolysis of CPRG releases a chromophore (CPR). The concentration of CKMB present in the patient sample is directly proportional to the rate of color change due to formation of CPR measured at 577 nm. The amount of CKMB protein is measured immunologically and the results are reported in mass units (ng/mL or µg/L).

2-Troponin I: [183,184]

The CTNI method is a one step enzyme immunoassay based on the "sandwich principle". Sample is incubated with chromium dioxide particles coated with a monoclonal antibody specific for the cardiac troponin-I molecule, and a conjugate reagent [alkaline phosphatase (ALP)] labeled monoclonal antibody specific for cardiac troponin-I, to form a particle/cardiac troponin-I/conjugate sandwich. Unbound conjugate is removed by magnetic separation and washing. After separation and washing, the particle/cardiac troponin-I/conjugate sandwich is transferred to the cuvette where the sandwich bound ALP triggers an amplification cascade. ALP dephosphorylates synthetic flavin adenine dinucleotide phosphate (FADP) to produce FAD. FAD binds to apo D-amino acid oxidase and converts it to active holo D-amino acid oxidase. Each molecule of holo D-amino acid oxidase then produces multiple molecules of hydrogen peroxide (H2O2) which, in the presence of horseradish peroxidase (HRP), convert 3,5- dichloro-2-hydroxybenzenesulfonic acid (DCHBS) and 4-aminoantipyrine (4-AAP) to a colored product that absorbs at 510 nm.

The color change measured is directly proportional to the concentration of cardiac troponin-I present in the patient sample.

3-hs-CRP: [181]

The hs-CRP method is a particle enhanced immuniturbidimetric assay.

Human CRP agglutinates with latex particles coated with monoclonal anti-CRP antibodies. The precipitate is determined turbidimetrically.

Determination of Lp-PLA$_2$ activity level [185]

Method:

Lp-PLA$_2$ activity level was determined by using an Enzyme Assay kit (PLAC test), Diadexus Company lot no 1305047, catalog number 10-135 manufactured in South San Francisco,CA 94080 USA.

Principle of the Test:

The PLAC Test for Lp-PLA$_2$ Activity is an enzymatic assay. Lp-PLA$_2$, in serum or plasma, hydrolyzes the sn-2 position of the substrate, 1-myristoyl-2-(4-nitrophenylsuccinyl) phosphatidylcholine, producing a colored reaction product, 4-nitrophenol.The rate of formation of 4-nitrophenol is followed spectrophotometrically and the Lp-PLA$_2$ activity is calculated from the rate of change in absorbance. A set of five Lp-PLA$_2$ calibrators is used to generate a standard curve fit of change in absorbance versus Lp-PLA$_2$ activity level in nmol/min/mL from which the sample Lp-PLA$_2$ activity is derived.

Reagents and materials:

The PLAC Test for Lp-PLA$_2$ Activity was supplied with:

Symbol	Component description
R1	Buffer
R2	Lp-PLA$_2$ Substrate, 1-myristoyl-2-(4-nitrophenylsuccinyl) phosphatidylcholine.
CAL	Calibrator concentrations {0, 50, 100, 250, 400} nmol/ml/min.

Certificate of Analysis – Control Range

The control ranges for the lot were indicated on the Certificate of Analysis :

Control Low ranges: 95.7-129.4 nmol/ml/min with a mean 112.6 nmol/ml/min.

Control High ranges: 243-328.9 nmol/ml/min with a mean 286 nmol/ml/min..

Reagent preparation and storage:

Reagents were provided ready to use. The caps of reagents R1 and R2 was removed and placed on the instrument. Reagents were stable for up to 4 weeks on board the analyzer Beckman Coulter AU 400 .All reagents were stored at 2-8 °C.

Procedure:

Reagent bottles were loaded in the analyzer Beckman Coulter AU 400.

The analyzer used 25 µl of the sample volume +100 µl of R1 (buffer) volume. After incubation till 10 points (188 seconds), 25 µl of R2 (Lp-PLA$_2$ Substrate, 1-myristoyl-2-(4-nitrophenylsuccinyl) phosphatidylcholine are automatically loaded.

The rate of formation of 4- nitrophenol was detected at 12 points (225.6 sec) and 14 points (263.2 sec) at wave length 410 nm.

Another wave length 520 nm was used to decrease interference.

Sensitivity

The clinical sensitivity of the assay is ≤ 10 nmol/min/mL as determined by the limit of quantitation (the lowest concentration with acceptable precision)

Calculation of Results:

Results were calculated from the sample calibration curve:

1. Standard curve was constructed by plotting the concentrations of PLAC test activity calibrators of the standards on the X-axis against the corresponding rate of change of absorbance measurements on the Y-axis on graph paper then the best fit line was drawn.

2. The standard curve was used to determine the concentration of Lp-PLA_2 in the tested samples, including controls and pathological samples.

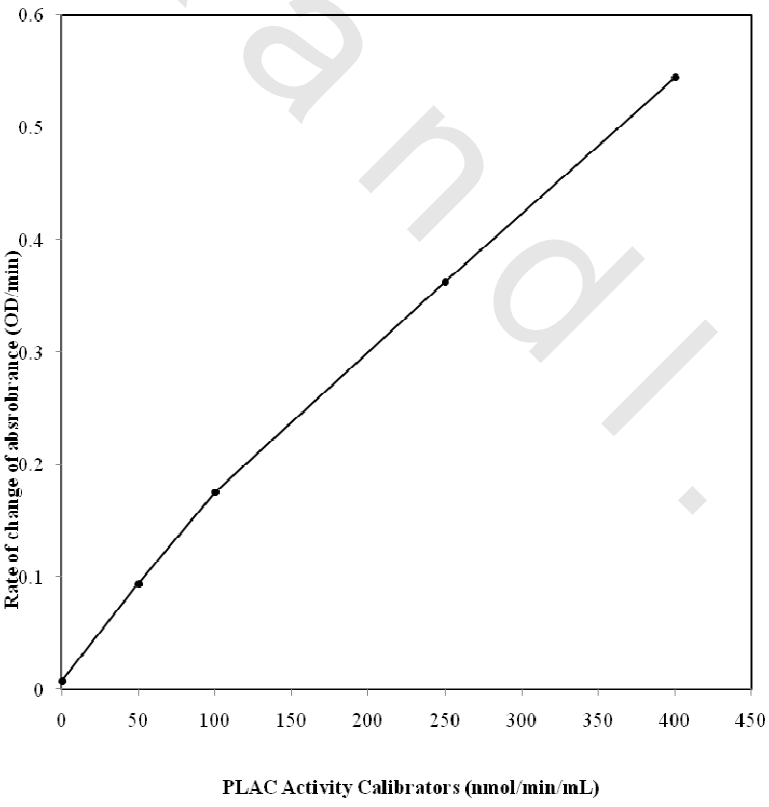

Figure (9): Lp-PLA_2 activity standard curve with rate of change of absorbance plotted on the Y axis and the calibrators concentration on the X axis.

Control results:

Control Low was 109.4 nmol/ml/min.

Control High was 324.1 nmol/ml/min.

The control ranges for the lot were indicated on the Certificate of Analysis **:**

Control Low ranges: 95.7-129.4 nmol/ml/min with a mean 112.6 nmol/ml/min.

Control High ranges: 243-328.9 nmol/ml/min with a mean 286 nmol/ml/min.

Figure (10): Beckman Coulter AU 400, Beckman Coulter Inc, Japan.

Statistical analysis of the data[168]

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0.[169] Qualitative data were described using number and percent. Quantitative data were described using range (minimum and maximum), mean, standard deviation and median. Comparison between different groups regarding categorical variables was tested using Chi-square test. The distributions of quantitative variables were tested for normality using Kolmogorov-Smirnov test, Shapiro-Wilk test and D'Agstino test, also Histogram and QQ plot were used for vision test. If it reveals normal data distribution, parametric tests was applied. If the data were abnormally distributed, non-parametric tests were used. For normally distributed data, comparison between two independent population were done using independent t-test. Correlations between two quantitative variables were assessed using Pearson coefficient. For abnormally distributed data, comparison between two independent population were done using Mann Whitney test. Agreement of the different Lp-PLA_2 to differentiate between control and cases was expressed in sensitivity, specificity, positive predictive value, negative predictive value and accuracy. Receiver operating characteristic curve (ROC) was plotted to analyse a recommended cutoff, the area under the ROC curve denotes the diagnostic performance of the test. Area more than 50% gives acceptable performance and area about 100% is the best performance for the test.

Significance test results are quoted as two-tailed probabilities. Significance of the obtained results was judged at the 5% level.

RESULTS

The study was carried out on thirty patients suffering from myocardial infarction and twenty healthy individuals, with no history of diabetes mellitus, atherosclerosis and medications as a control group.

Demographic data:

Gender:

Patients included 20 males (66.7%) and 10 females (33.3%), on the other hand control group included 14 males (70%) and 6 females (30%), with no statistical significant difference between the two studied groups (p=0.804). (Table IV, figure 11)

Age:

Patients aged 35-61 years with a mean of 48.53 ± 8.14 years on the other hand control group aged 30-61 years with a mean of 44.85 ± 9.94 years, with no statistical significant difference between the two studied groups (p=0.158). (Table IV, figure 12)

Table (IV): Comparison between the two studied groups according as regards gender and age

	Patients (n=30)		Controls (n=20)		Test of sig.	p
	No.	%	No.	%		
Gender						
Male	20	66.7	14	70	χ^2= 0.061	0.804
Female	10	33.3	6	30		
Age (years)						
Min. – Max.	35 – 61		30 – 61		t = 1.434	0.158
Mean ± SD.	48.53 ± 8.14		44.85 ± 9.94			

p: p value for comparing between the two studied groups
χ^2: Chi square test
t: Student t-test
*: Statistical significant at p ≤ 0.05

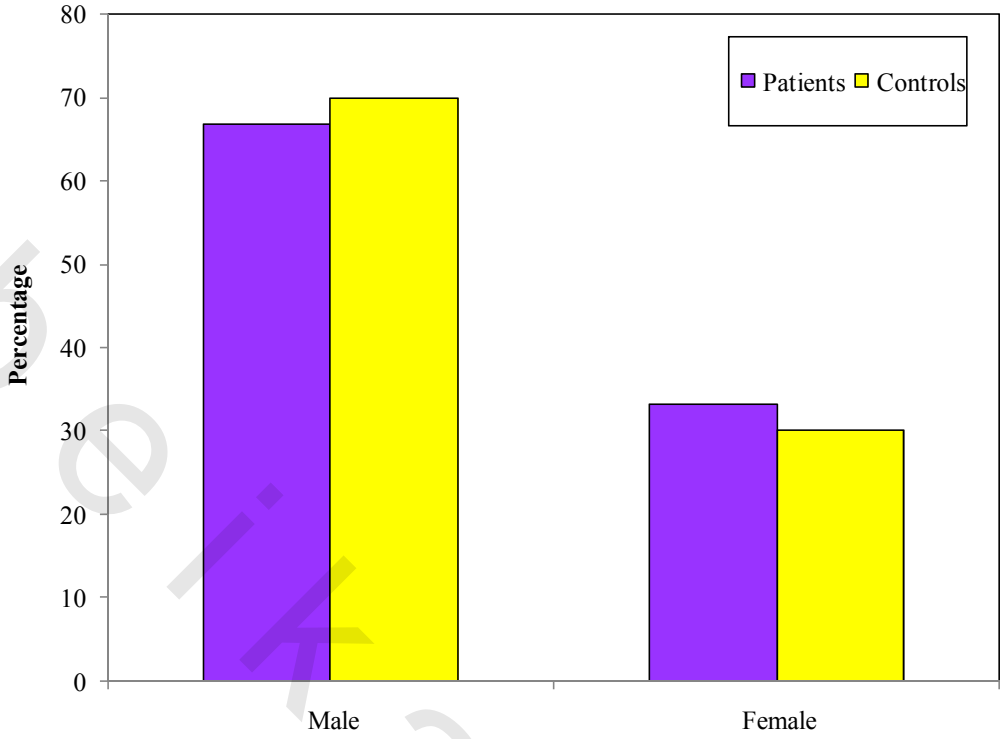

Figure (11): Comparison between the two studied groups according to gender

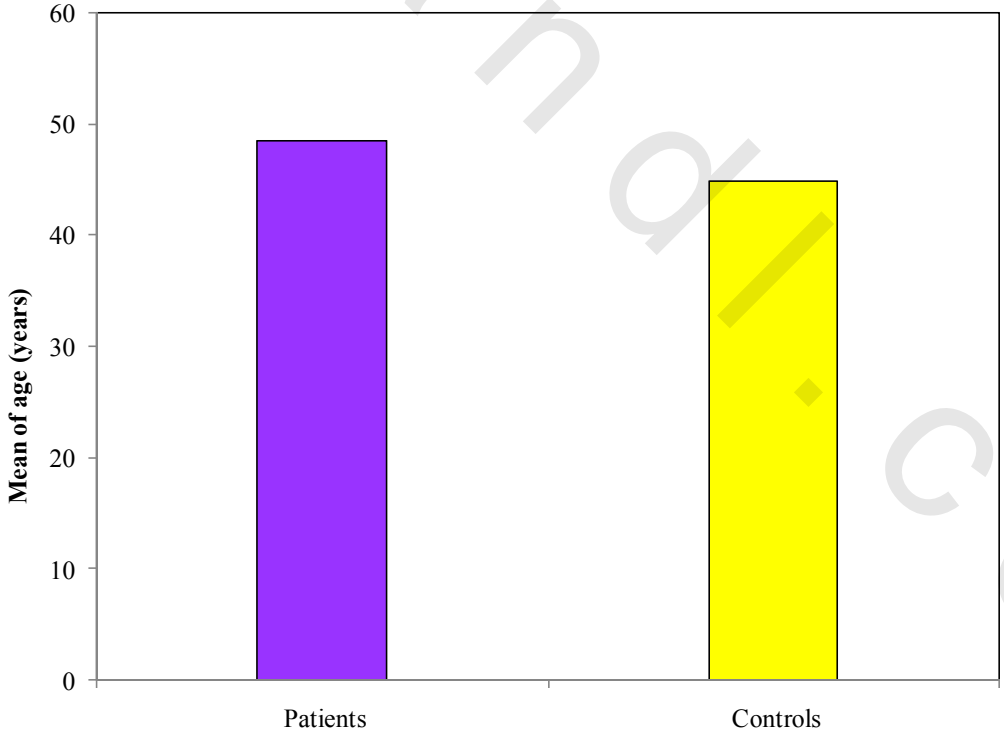

Figure (12): Comparison between the two studied groups according to age

Risk factors:

Smoking:

14 (46.7%) patients and 9 (45%) controls were smokers with no statistical significant difference between the two studied groups (p=0.908). (Table V, figure 13)

Hypertension:

Among patients, 13 (43.3%) had hypertension. On the other hand all controls were non hypertensive. There was a statistical significant difference between the two groups (p=0.001). (Table V, figure 13)

Table (V): Comparison between the two studied groups according to risk factors

	Patients (n=30)		Controls (n=20)		χ^2	p
	No.	%	No.	%		
Smoking	14	46.7	9	45	0.013	0.908
HTN	13	43.3	0	0.0	11.712*	0.001*

HTN: hypertension
χ^2: Chi square test
*: Statistical significant at $p \leq 0.05$

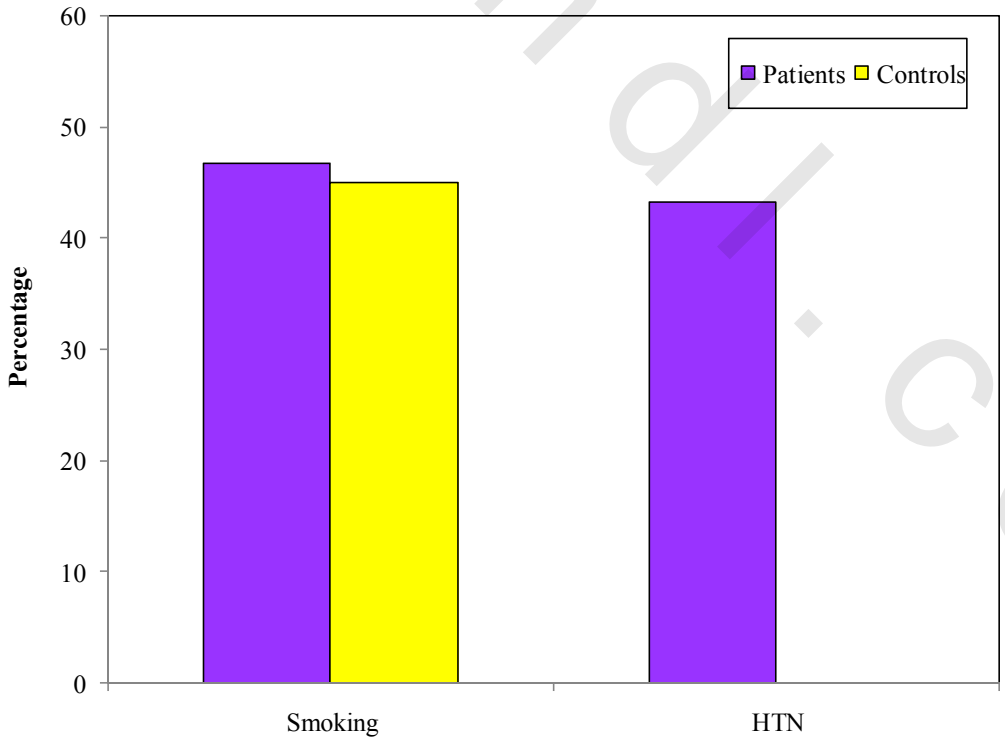

Figure (13): Comparison between the two studied groups according to risk factors

Lipid profile and serum glucose:

1-Triglycerides:

Among patients, triglycerides ranged between 88 and 570 with a median of 150 (mg/dl) whereas in controls triglycerides ranged between 65 and 147 with a median of 122.5 (mg/dl); with statistical significant increase in patients when compared to healthy controls (p=0.010). (Table VI, figure 14)

2-Total cholesterol:

Among patients, total cholesterol ranged between 155 and 492 with a mean of 246.07 ± 72.08 (mg/dl), whereas in controls total cholesterol ranged between 94 and 200 with a mean of 160.05 ± 35.25 (mg/dl); with statistical significant increase in patients when compared to healthy controls (p<0.001). (Table VI, figure 15)

3-LDL-C:

Among patients, LDL- cholesterol ranged between 65 and 339 with a mean of 168.47 ± 59.93 (mg/dl), whereas in controls LDL- cholesterol ranged between 36 and 122 with a mean of 79.23 ± 26.04 (mg/dl); with statistical significant increase in patients when compared to healthy controls (p<0.001). (Table VI, figure 16)

4-HDL-C:

Among patients, HDL- cholesterol ranged between 14 and 67 with a mean of 40.13 ± 12.69(mg/dl), whereas in controls HDL- cholesterol ranged between 36 and 80 with a mean of 53.35 ± 12.10 (mg/dl); with statistical significant increase in patients when compared to healthy controls (p=0.001). (Table VI, figure 17)

5-Glucose:

Among patients, glucose ranged between 63 and 119 with a mean of 103.70 ± 15.54 (mg/dl), whereas in controls glucose ranged between 70 and 110 with a mean of 92.35 ± 12.41 (mg/dl); shows with statistical significant increase in patients when compared to healthy controls (p= 0.009). (Table VI, figure 18)

Table (VI): Comparison between the two studied groups according to lipid profile and serum glucose

	Patients (n=30)	Controls (n=20)	Test of sig.	P
Triglycerides (mg/dl)				
Min. – Max.	88 – 570	65 – 147	Z=2.585*	0.010*
Median	150	122.50		
Total Cholesterol (mg/dl)				
Min. – Max.	155 – 492	94 – 200	t =5.608*	<0.001*
Mean ± SD.	246.07 ± 72.08	160.05 ± 35.25		
LDL-C (mg/dl)				
Min. – Max.	65 – 339	36 – 122	t =7.199*	<0.001*
Mean ± SD.	168.47 ± 59.93	79.23 ± 26.04		
HDL-C (mg/dl)				
Min. – Max.	14 – 67	36 – 80	t = 3.675*	0.001*
Mean ± SD.	40.13 ± 12.69	53.35 ± 12.10		
Glucose (mg/dl)				
Min. – Max.	63 – 119	70 – 110	t=2.734*	0.009*
Mean ± SD.	103.70 ± 15.54	92.35 ± 12.41		

LDL-C: Low density lipoprotein cholesterol, **HDL-C:** High density lipoprotein cholesterol.
p: p value for comparing between the two studied groups
t: Student t-test
Z: Z for Mann Whitney test
*: Statistical significant at $p \leq 0.05$

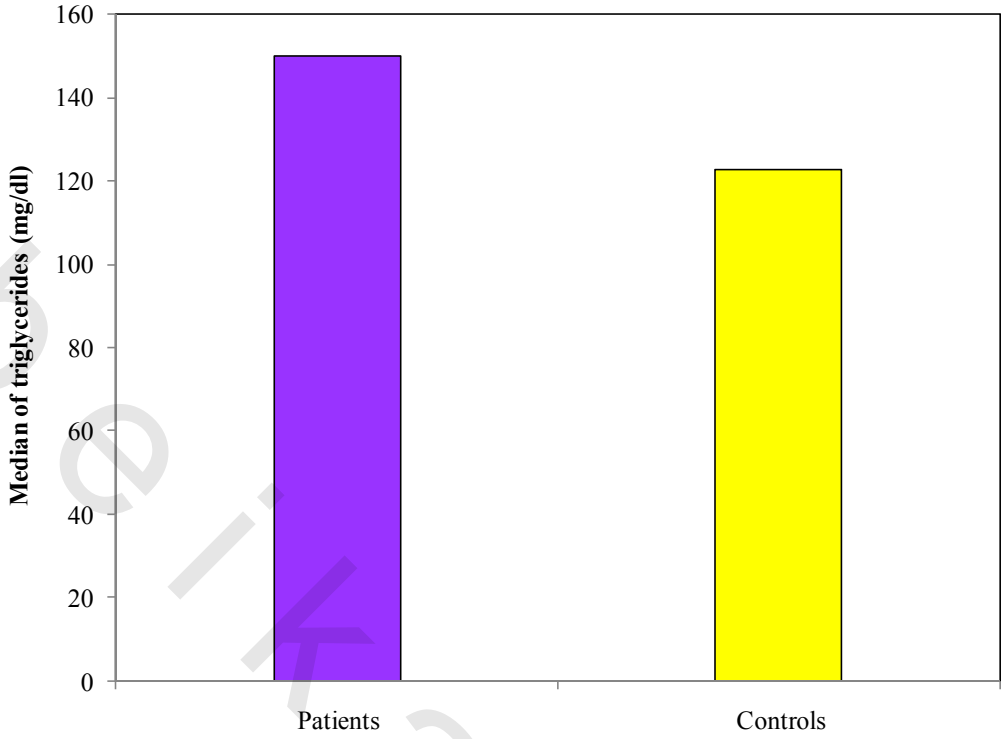

Figure (14): Comparison between the two studied groups according to triglycerides

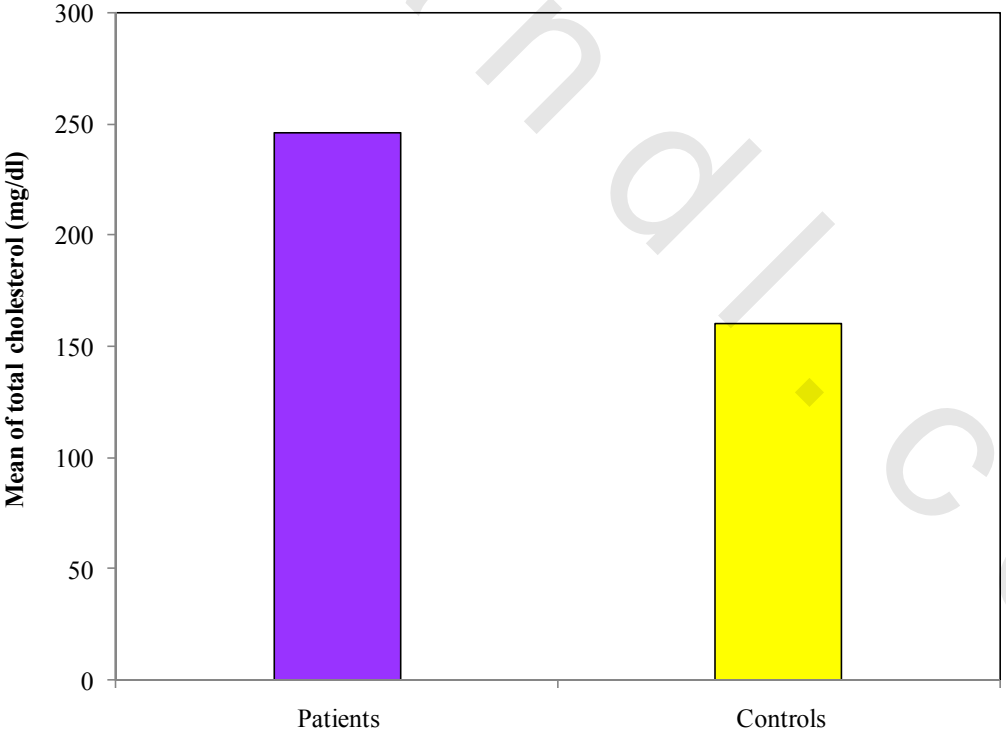

Figure (15): Comparison between the two studied groups according to total cholesterol

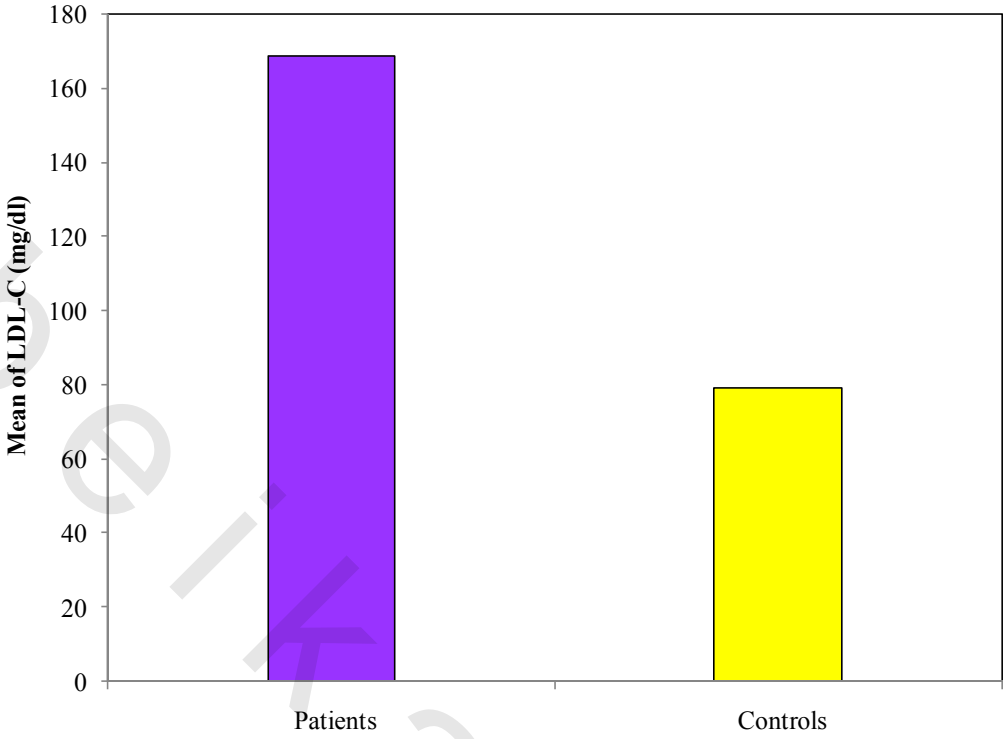

Figure (16): Comparison between the two studied groups according to LDL-C

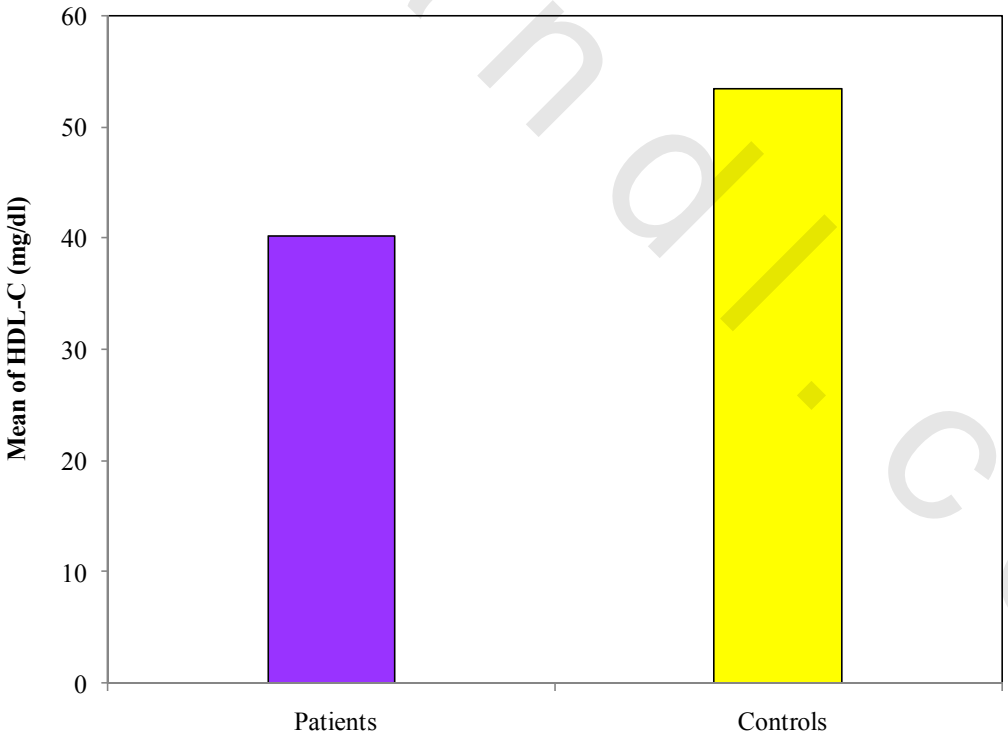

Figure (17): Comparison between the two studied groups according to HDL-C

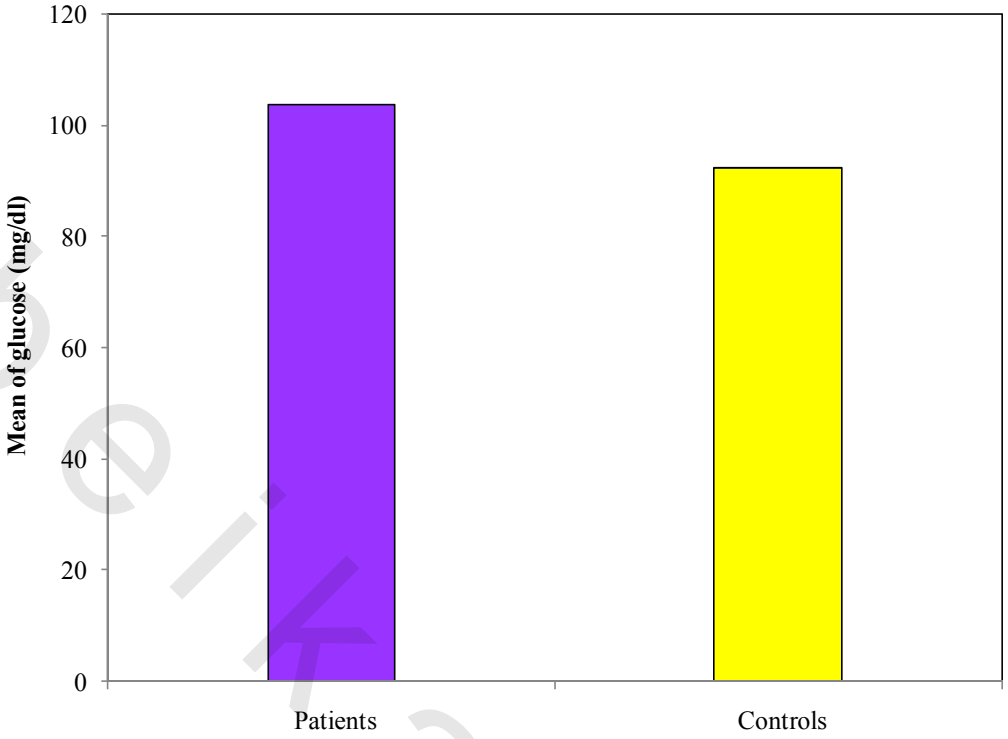

Figure (18): Comparison between the two studied groups according to serum glucose

Cardiac biomarkers:

1- CK:

Among patients, CK ranged between 100 and 3218 with a median of 1082.5 (U/L), whereas in controls CK ranged between 28 and 193 with a median of 80(U/L); with statistical significant increase in patients when compared to healthy controls ($p<0.001$). (Table VII, figure 19)

2- CK-MB mass:

Among patients, CK-MB mass ranged between 1.1 and 954 with a median of 120.3 (ng/ml), whereas in controls CK-MB mass ranged between 0.1 and 0.8 with a median of 0.5 (ng/ml); with statistical significant increase in patients when compared to healthy controls ($p<0.001$). (Table VII, figure 20)

3- Troponin I:

Among patients, troponin I ranged between 0.06 and 318 with a median of 25.21(ng/ml), whereas in controls troponin I ranged between 0.0 and 0.01 with a median of 0.0 (ng/ml); with statistical significant increase in patients when compared to healthy controls ($p<0.001$). (Table VII, figure 24)

4- AST:

Among patients, AST ranged between 15 and 383 with a median of 131.5 (U/L), whereas in controls AST ranged between 11 and 47 with a median of 21 (U/L); with statistical significant increase in patients when compared to healthy controls ($p<0.001$). (Table VII, figure 21)

5- LDH:

Among patients, LDH ranged between 197 and 1756 with a median of 618 (U/L), whereas in controls LDH ranged between 95 and 198 with a median of 144 (U/L); with statistical significant increase in patients when compared to healthy controls ($p<0.001$). (Table VII, figure 22)

6- hs-CRP:

Among patients, hs-CRP ranged between 2.6 and 462.6 with a median of 52.85 (mg/l), whereas in controls hs-CRP ranged between 0.5 and 3 with a median of 1.65 (mg/l); with statistical significant increase in patients when compared to healthy controls ($p<0.001$). (Table VII, figure 23)

Table (VII): Comparison between the two studied groups according to cardiac biomarkers

	Patients (n=30)	Controls (n=20)	Z	p
CK (U/L)				
Min. – Max.	100 – 3218	28 – 193	5.803*	<0.001*
Median	1082.50	80		
CK-MB mass (ng/ml)				
Min. – Max.	1.10 – 954	0.10 – 0.80	5.949*	<0.001*
Median	120.30	0.50		
Troponin I (ng/ml)				
Min. – Max.	0.06 – 318	0 – 0.01	6.110*	<0.001*
Median	25.21	0		
AST (U/L)				
Min. – Max.	15 – 383	11 – 47	5.210*	<0.001*
Median	131.50	21		
LDH (U/L)				
Min. – Max.	197 – 1756	95 – 198	Z=5.921*	<0.001*
Median	618	144		
hs-CRP (mg/L)				
Min. – Max.	2.60 – 462.60	0.50 – 3	Z = 5.863*	<0.001*
Median	52.85	1.65		

CK: Creatine kinase,
CK-MB mass: Creatine kinase muscle brain isoform,
AST: Aspartate transaminase,
LDH: Lactate dehydrogenase,
hs-CRP: High sensitivity c-reactive protein.
Z: Z for Mann Whitney test
*: Statistical significant at $p \leq 0.05$

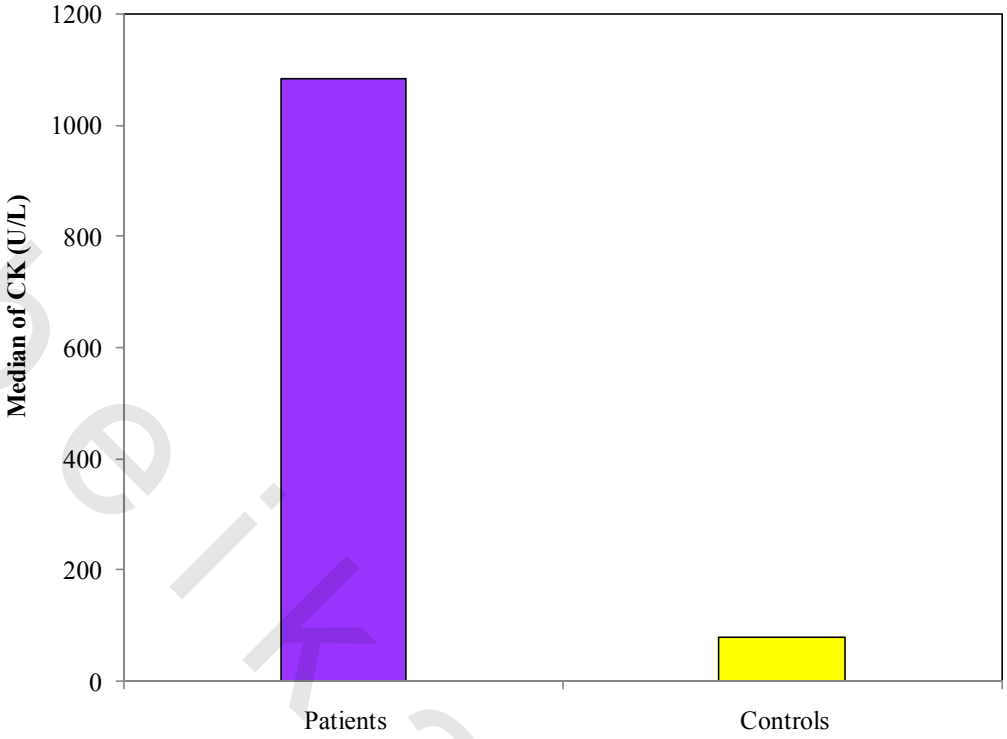

Figure (19): Comparison between the two studied groups according to CK

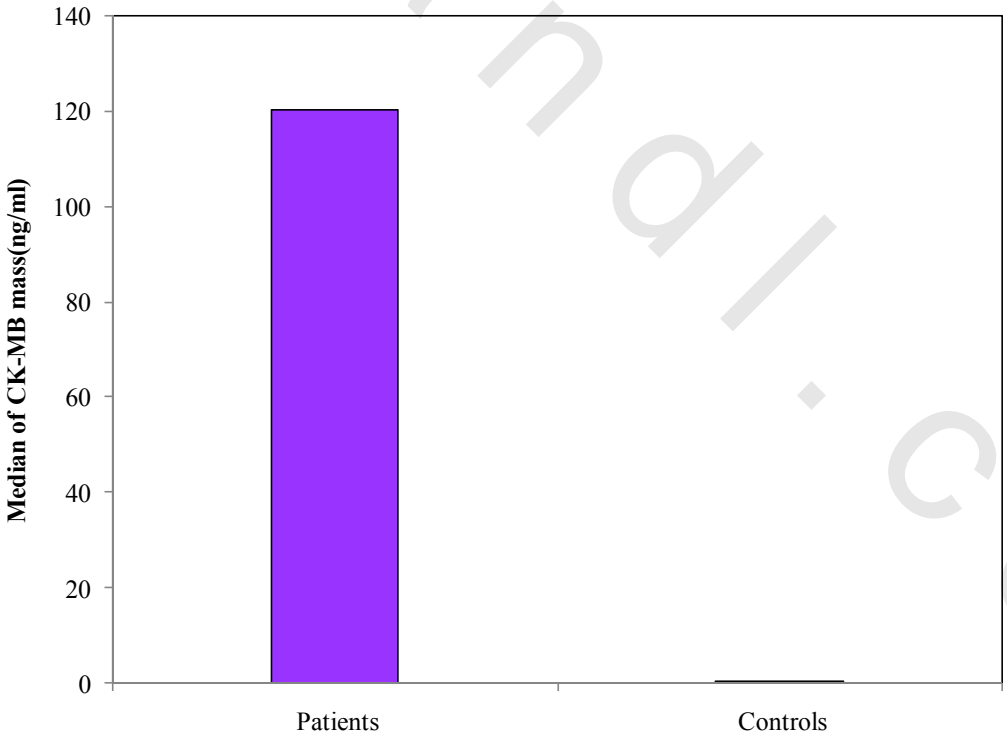

Figure (20): Comparison between the two studied groups according to CK-MB mass

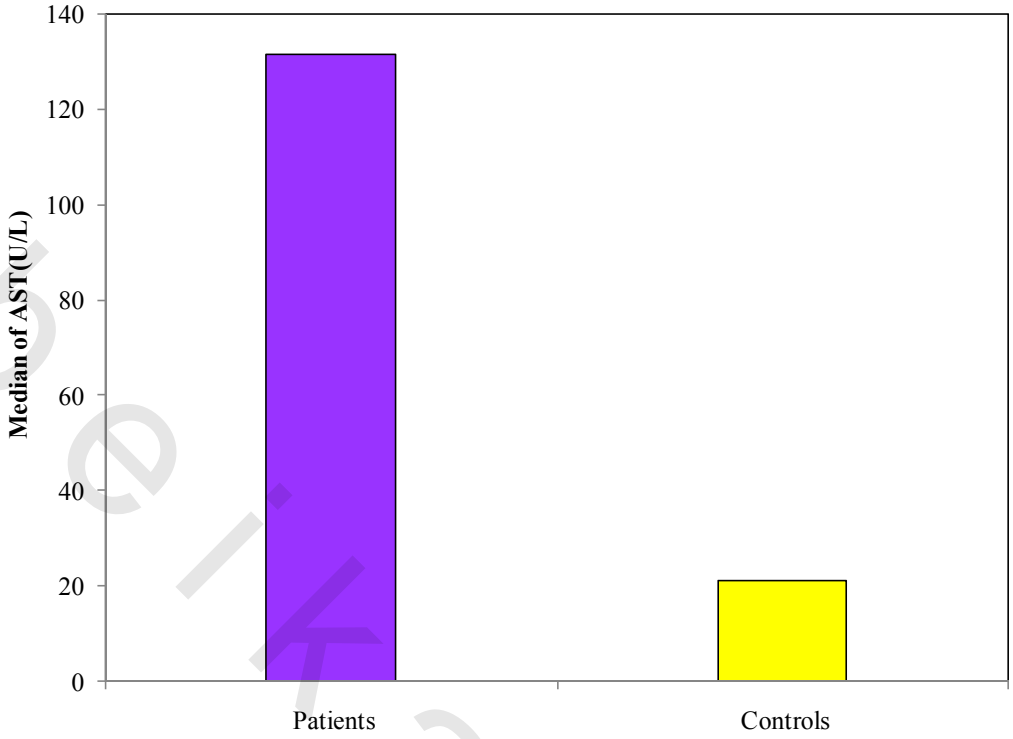

Figure (21): Comparison between the two studied groups according to AST

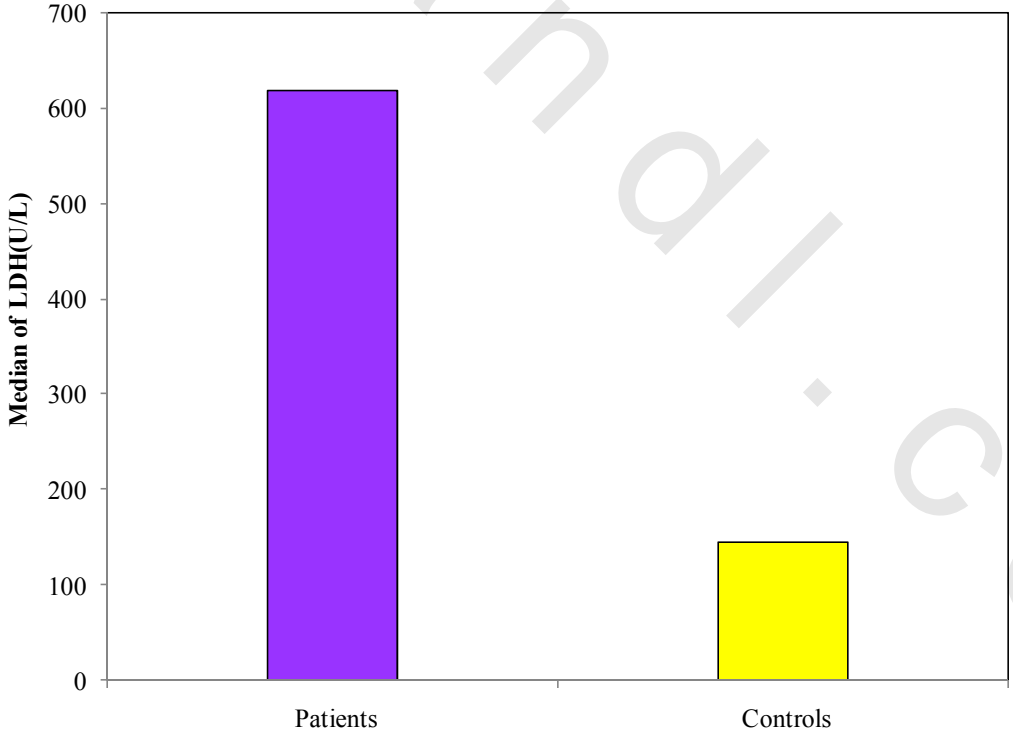

Figure (22): Comparison between the two studied groups according to LDH

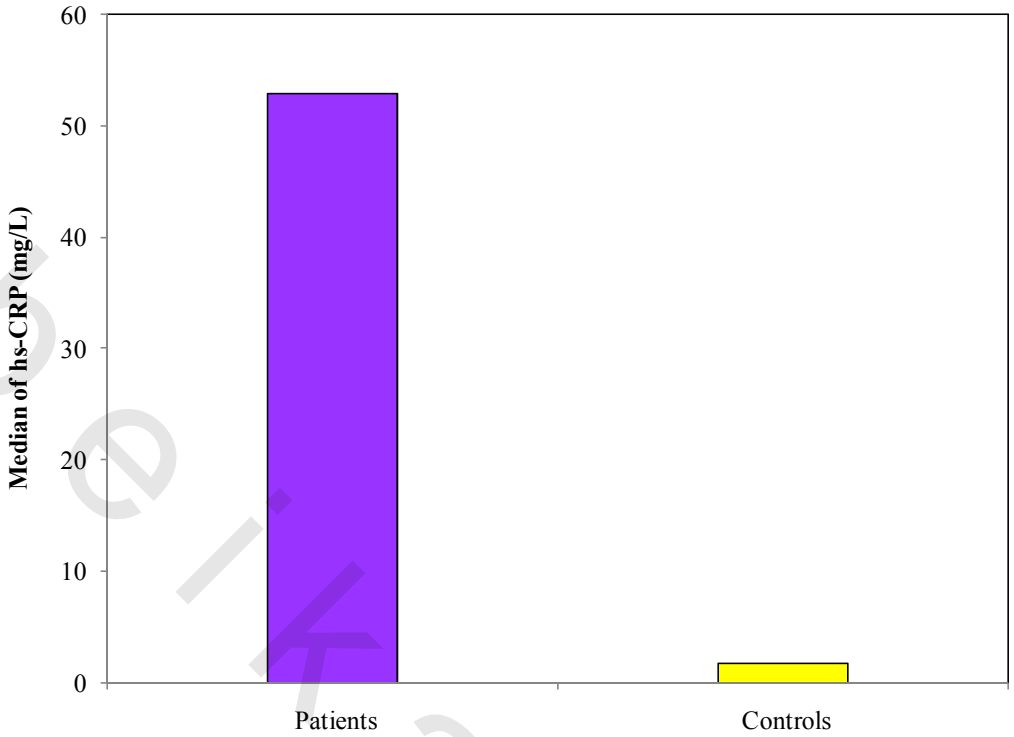

Figure (23): Comparison between the two studied groups according to hs-CRP

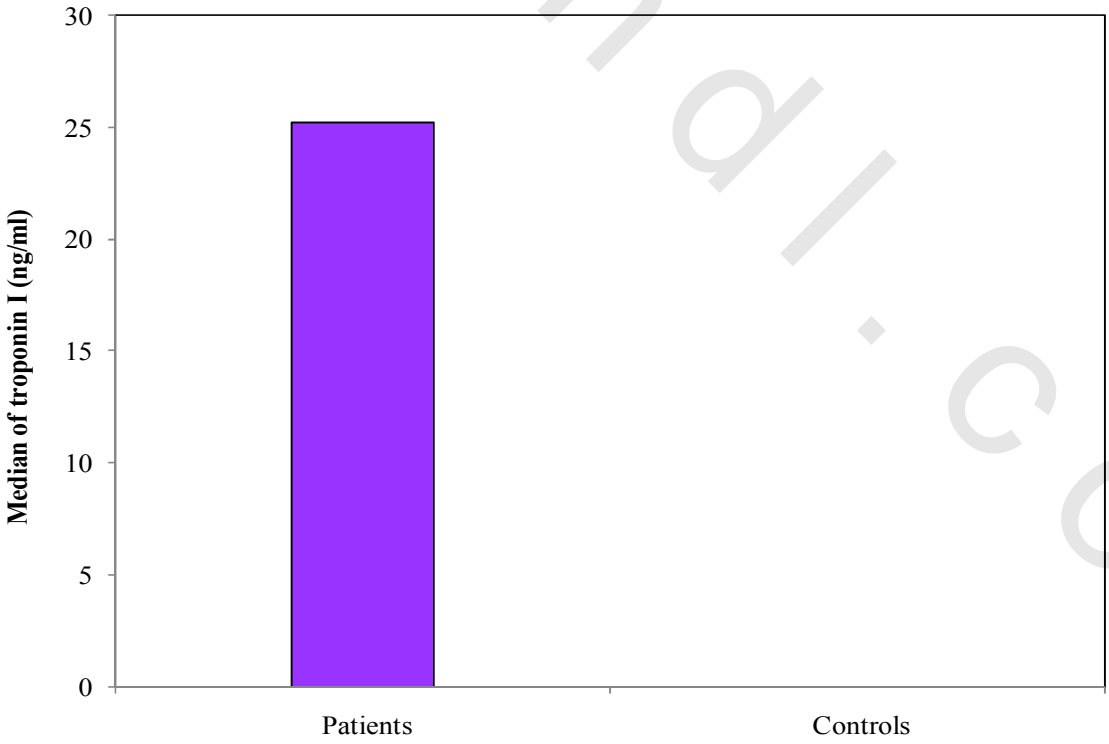

Figure (24): Comparison between the two studied groups according to troponin I (ng/ml)

Lipoprotein associated phospholipase A_2 activity:

Among patients, Lp-PLA_2 ranged between 101 and 381.6 with a mean of 238.75 ± 61.70 (nmol/min/ml), whereas in controls Lp-PLA_2 ranged between 80 and 240 with a mean of 150.27 ± 54.19 (nmol/min/ml); with statistical significant increase in patients when compared to healthy controls ($p<0.001$). (Table VIII, figure 25)

Table (VIII): Comparison between the two studied groups according to Lp-PLA_2

	Patients (n=30)	**Controls (n=20)**	**t**	**p**
Lp-PLA_2 (nmol/min/mL)				
Min. – Max.	101 - 381.60	80 – 240	5.208*	<0.001*
Mean ± SD.	238.75 ± 61.70	150.27 ± 54.19		

Lp-PLA_2: Lipoprotein associated phospholipase A_2 activity.
t: Student t-test
*: Statistical significant at $p \leq 0.05$

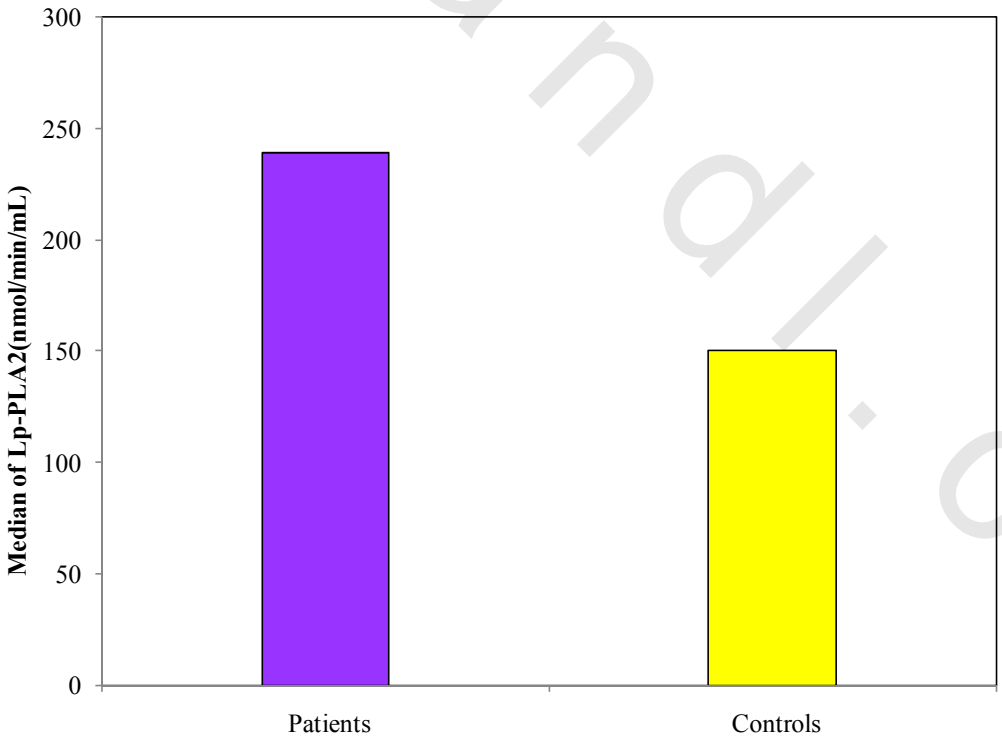

Figure (25): Comparison between the two studied groups according to Lp-PLA_2

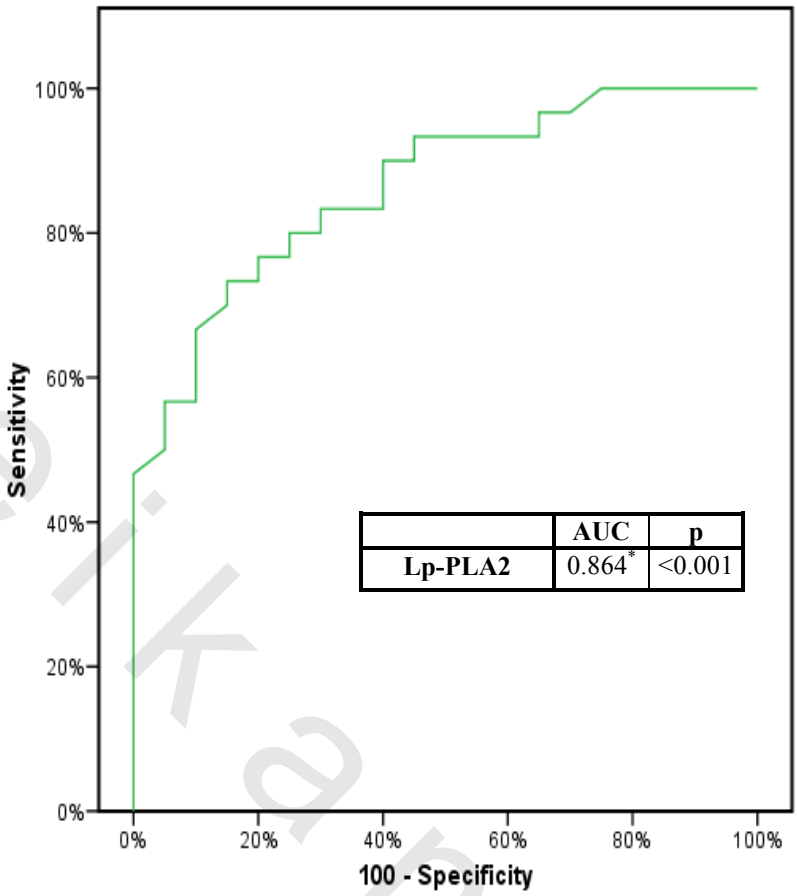

Figure (26): ROC curve for Lp-PLA$_2$

ROC curve for Lp-PLA$_2$ activity:

The cutoff value for Lp-PLA$_2$ activity was 204.6 nmol/min/ml, with 76.67% sensitivity, 75% specificity and 76% accuracy with significant p-value (<0.001)

Among patients, 7 had cutoff value ≤ 204.6 nmol/ml/min and 23 had cutoff value > 204.6 nmol/ml/min, whereas in controls 15 had cutoff value ≤ 204.6 nmol/ml/min and 5 had cutoff value > 204.6 nmol/ml/min. (Table IX), (Figure 26).

Table (IX): Diagnostic performance (sensitivity, specificity and accuracy) for Lp-PLA$_2$ activity in cases of myocardial infarction.

		Controls	Patients	Sensitivity %	Specificity %	PPV	NPV	Accuracy
Lp-PLA$_2$ (nmol/ml/min)	≤ 204.6	15	7	76.67	75	82.14	68.18	76
	> 204.6	5	23					

Relation between Lp-PLA$_2$ with gender among patients:

There were 20 males with Lp-PLA$_2$ ranged between 108.1 and 305.8 with a mean of 244.39 ± 52.17 (nmol/min/ml), and 10 females with Lp-PLA$_2$ ranged between 101.0 and 381.6 with a mean of 227.47 ± 79.44 (nmol/min/ml); with no statistical significant difference between Lp-PLA$_2$ activity levels and gender (p=0.489). (Table X, figure 27)

Relation between Lp-PLA$_2$ with smoking among patients:

There were 16 non-smoker patients with Lp-PLA$_2$ ranged between 101.0 and 381.0 with a mean of 225.0 ± 72.40 (nmol/min/ml), and 14 smoker patients with Lp-PLA$_2$ ranged between 181.4 and 305.8 with a mean of 254.4 ± 44.13 (nmol/min/ml); with no statistical significant difference between Lp-PLA$_2$ activity levels and smoking in patients group (p=0.198). (Table X, figure 28)

Relation between Lp-PLA$_2$ with hypertension among patients:

There were 17 non-hypertensive patients with Lp-PLA$_2$ ranged between 108.1and 305.8 with a mean of 232.9 ± 49.41 (nmol/min/ml), and 13 hypertensive patients with Lp-PLA$_2$ ranged between 101.0 and 381.6 with a mean of 246.4 ± 76.38 (nmol/min/ml); with no statistical significant difference between Lp-PLA$_2$ activity levels and hypertension in patients group (p=0.562). (Table X, figure 29)

Table (X): Relation between Lp-PLA$_2$ with different studied parameters in patients group

	N	Lp-PLA$_2$(nmol/ml/min)		t	p
		Min. – Max.	Mean ± SD.		
Gender					
Male	20	108.1 – 305.8	244.39 ± 52.17	0.702	0.489
Female	10	101 – 381.6	227.47 ± 79.44		
Smoking					
No	16	101 – 381	225 ± 72.40	1.319	0.198
Yes	14	181.4 – 305.8	254.4 ± 44.13		
Hypertension					
No	17	108.1 – 305.8	232.9 ± 49.41	0.587	0.562
Yes	13	101 – 381.6	246.4 ± 76.38		

t: Student t-test

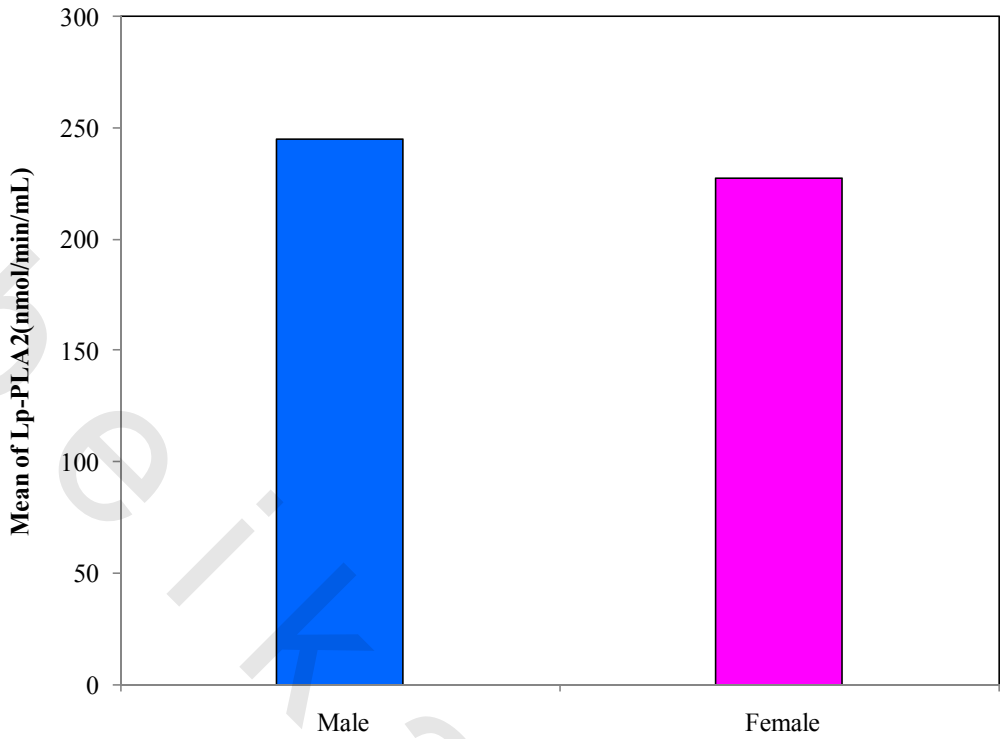

Figure (27): Relation between Lp-PLA$_2$ with gender in patients group

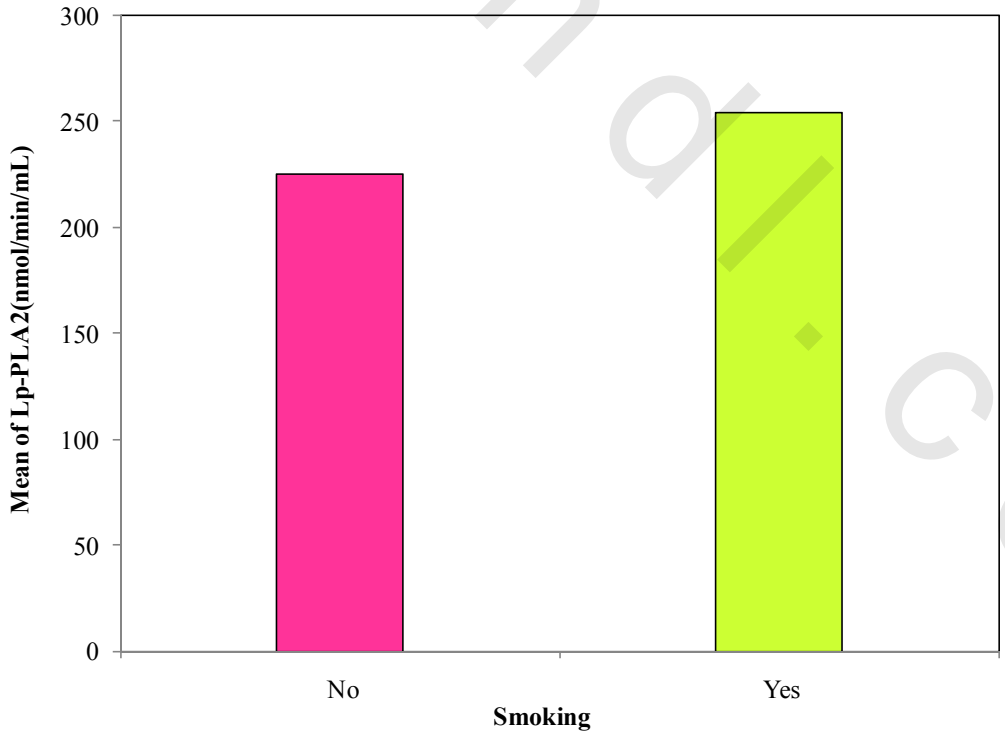

Figure (28): Relation between Lp-PLA$_2$ with smoking in patients group

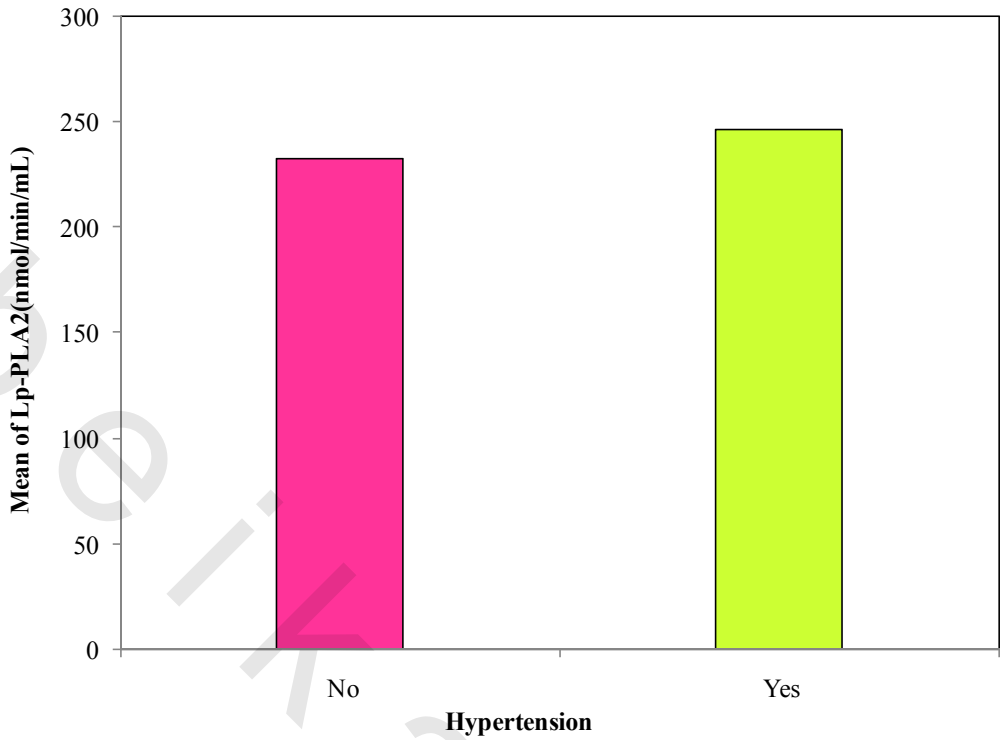

Figure (29): Relation between Lp-PLA$_2$ with hypertension in patients group

Relation between Lp-PLA$_2$ with gender among controls group:

There were 14 males with Lp-PLA$_2$ ranged between 80.0 and 230.0with a mean of 139.63 ± 50.61 (nmol/min/ml), and 6 females with Lp-PLA$_2$ ranged between 95.0 and 240.0 with a mean of 175.10 ± 58.68 (nmol/min/ml); with no statistical significant difference between Lp-PLA$_2$ activity levels regarding gender in healthy controls (p=0.187). (Table XI, figure 30)

Relation between Lp-PLA$_2$ with smoking among controls group:

There were 11 non-smoker controls with Lp-PLA$_2$ ranged between 95.0 and 240.0 with a mean of 153.45 ± 55.54 (nmol/min/ml), and 9 smoker controls with Lp-PLA$_2$ ranged between 80.0 and 230.0 with a mean of 146.39 ± 55.58 (nmol/min/ml); with no statistical significant difference between Lp-PLA$_2$ activity levels regarding smoking in healthy controls (p=0.781). (Table XI, figure 31)

Table (XI): Relation between Lp-PLA$_2$ with different studied parameters in controls group

	N	Lp-PLA$_2$(nmol/ml/min)		t	p
		Min. – Max.	Mean ± SD.		
Gender					
Male	14	80 – 230	139.63 ± 50.61	1.372	0.187
Female	6	95 – 240	175.10 ± 58.68		
Smoking					
No	11	95 – 240	153.45 ± 55.54	0.283	0.781
Yes	9	80 – 230	146.39 ± 55.58		

t: Student t-test

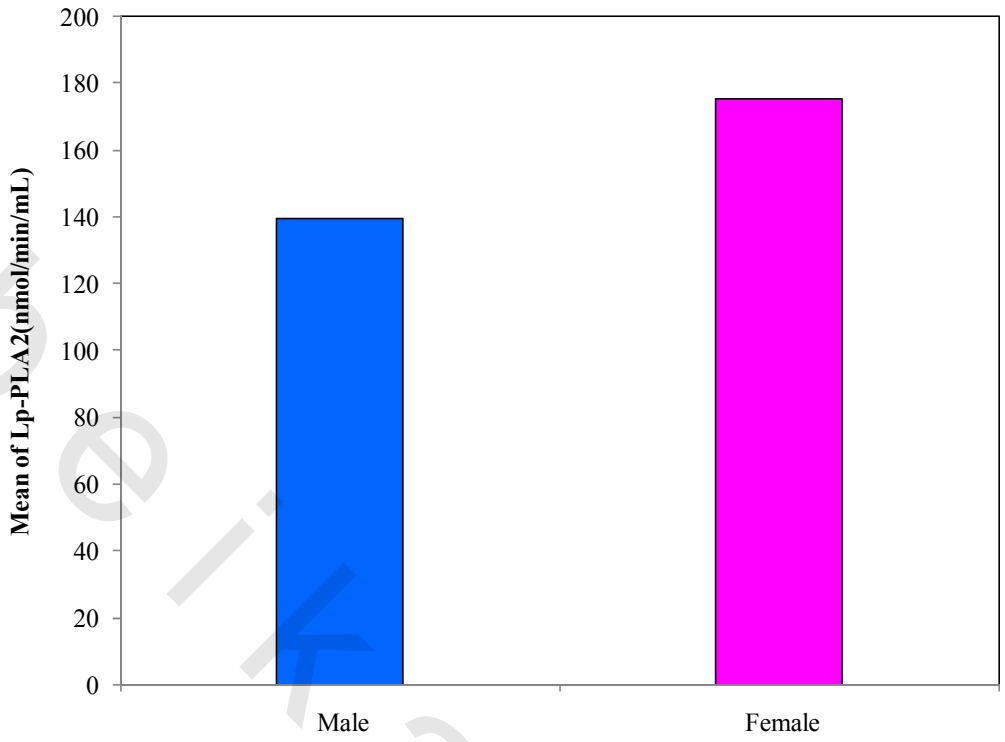

Figure (30): Relation between Lp-PLA$_2$ with gender in controls group

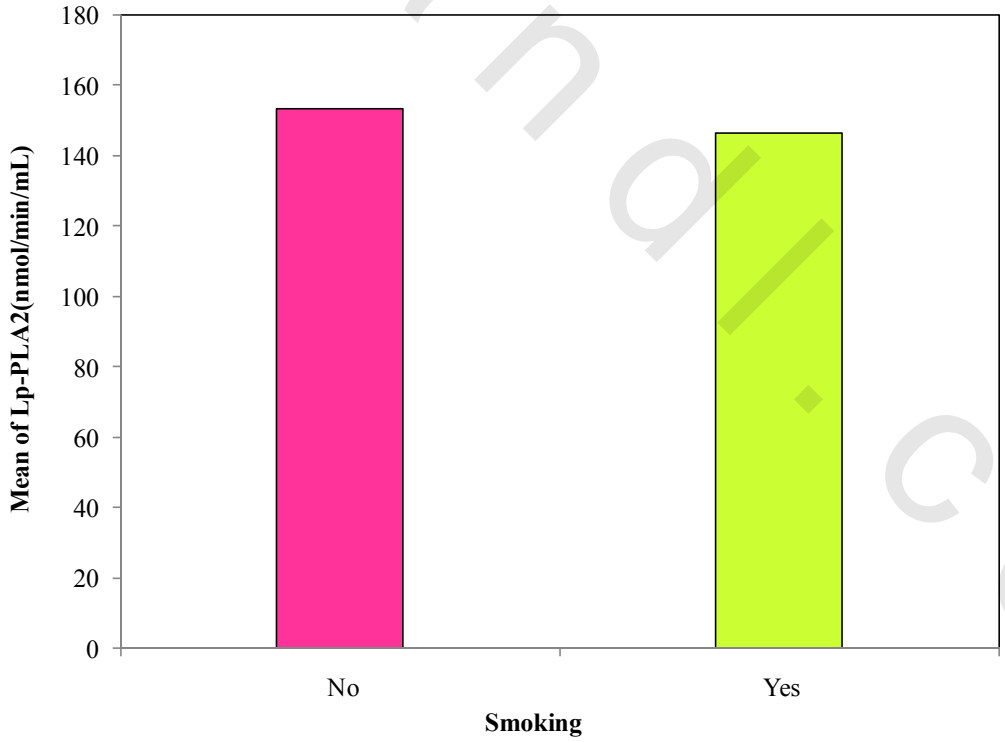

Figure (31): Relation between Lp-PLA$_2$ with smoking in controls group

Correlation between Lp-PLA_2 with different parameters in cases and control group:

There were a significant positive correlation of Lp-PLA_2 activity with triglycrides (p=0.017, r=0.431), and LDL-C (p=0.003, r=0.523) and there was no correlation of age, total cholesterol, creatine kinase, CK-MB, troponin I, LDH, AST, hs-CRP or glucose with Lp-PLA_2 activity level in cases, Also there were inverse correlation of HDL-C with Lp-PLA_2 activity level in cases (p=0.035, r=-0.386). (Table XII, figure 32,33,34)

No correlations were observed between Lp-PLA_2 activity and any of the studied parameters in the control group. (Table XII)

Table (XII): Correlation between Lp-PLA_2 with different parameters in cases and control group

Sig	Lp-PLA_2(nmol/ml/min)			
	Patients		Controls	
	r	p	r	p
Age (years)	0.062	0.745	-0.102	0.670
TG	0.431*	0.017	0.131	0.583
CHOL	-0.003	0.989	0.082	0.730
LDL-C	0.523*	0.003	0.253	0.282
HDL-C	-0.386*	0.035	-0.095	0.689
CKI	-0.223	0.237	-0.004	0.985
CKMB	-0.192	0.309	0.108	0.651
TROP	-0.146	0.442	0.281	0.230
AST	0.040	0.832	-0.276	0.239
LDH	0.015	0.936	0.175	0.461
GLUC	-0.070	0.714	0.284	0.226
hs-CRP	0.052	0.786	-0.340	0.143

r: Pearson coefficient

LDL-C: Low density lipoprotein cholesterol, HDL-C: High density lipoprotein cholesterol, CK-MB: Creatine kinase muscle brain isoform, AST: Aspartate aminotransferase, LDH: lactate dehydrogenase, hs-CRP: High sensitivity c-reactive protein

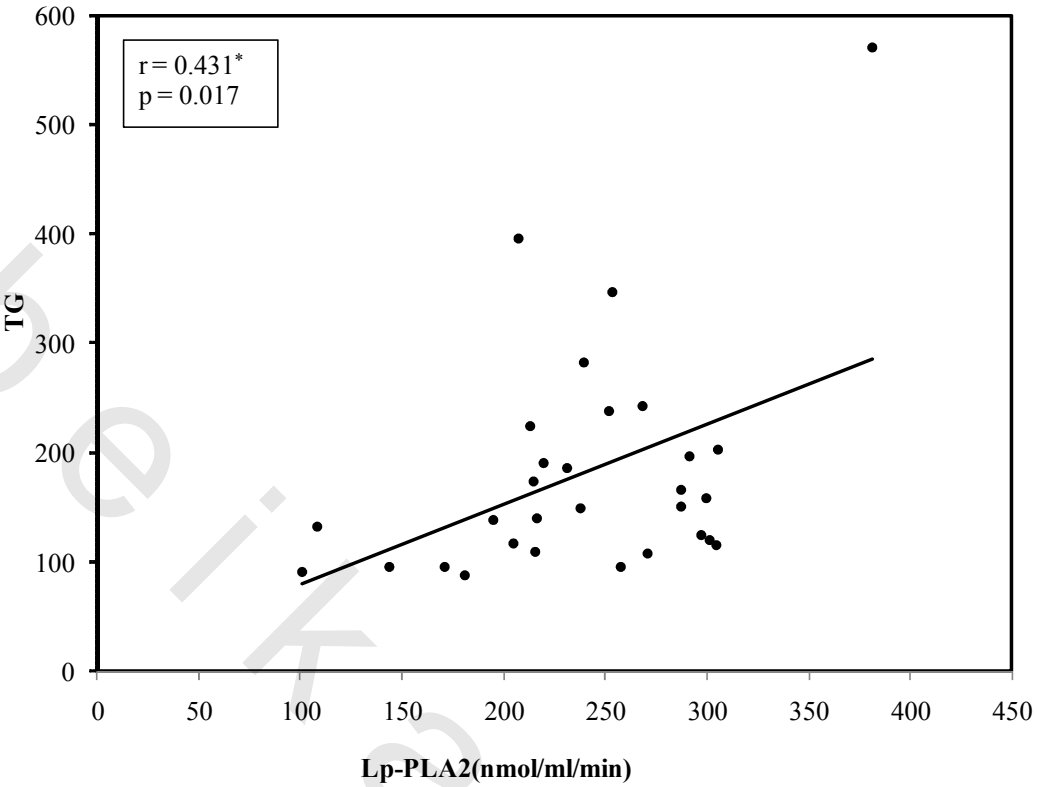

Figure (32): Correlation between Lp-PLA$_2$ with TG in cases group

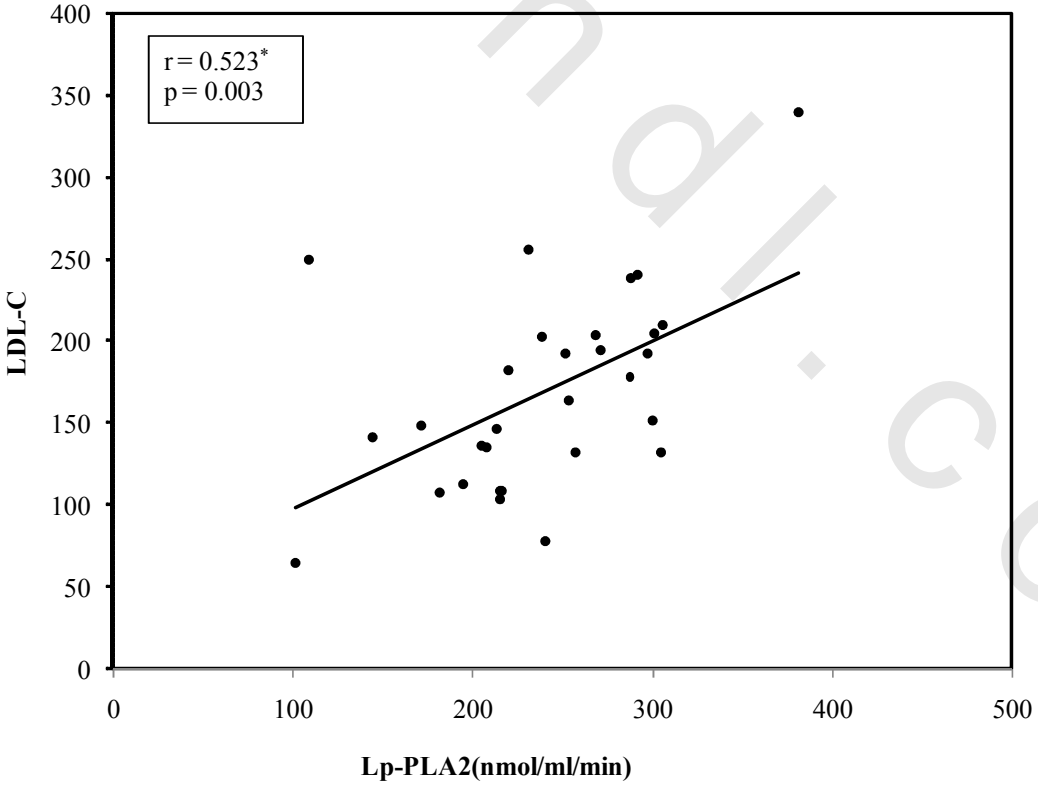

Figure (33): Correlation between Lp-PLA$_2$ with LDL-C in cases group

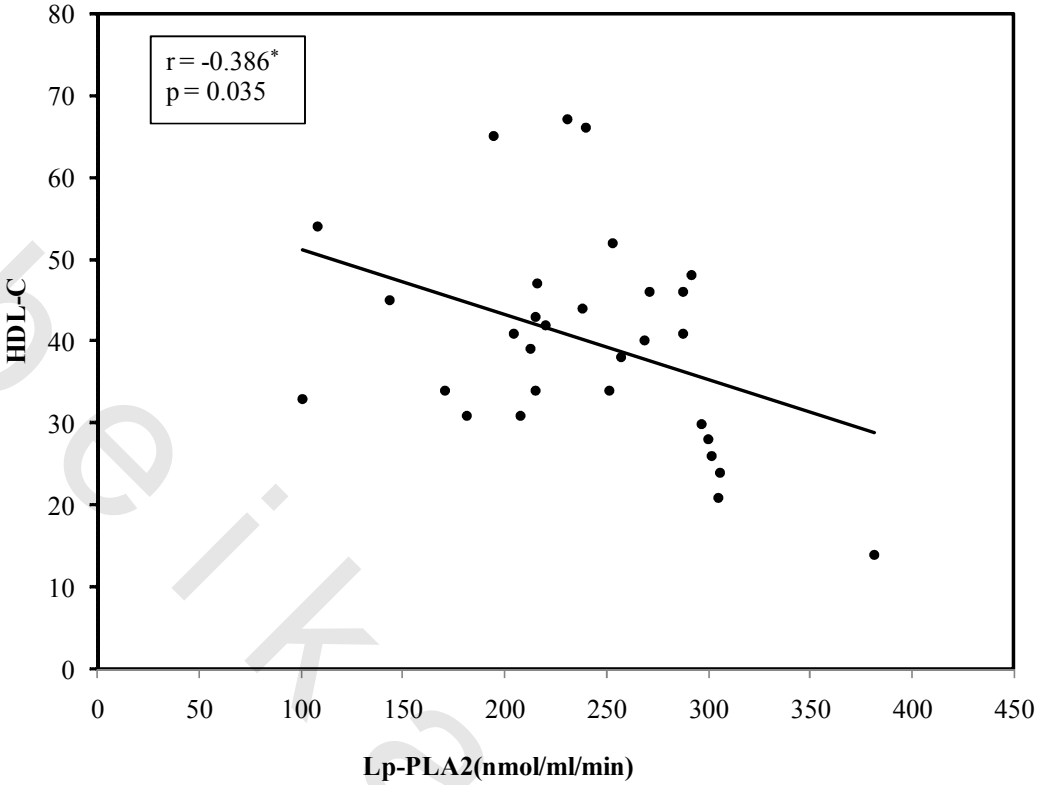

Figure (34): Correlation between Lp-PLA$_2$ with HDL-C in cases group

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