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Association between Lipoprotein (A) and In-Hospital Outcomes in Patients with Acute ST Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention

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ABSTRACT

Background: Even though lipoprotein (a) has been linked to heart disease, no one knows how it affects people who have had primary percutaneous coronary intervention (PCI) after an ST-elevation myocardial infarction (STEMI).

Objectives: This research aimed to study the relationship between lipoprotein (a) levels and in-hospital outcomes in people who had an acute STEMI after their first percutaneous coronary intervention (PCI).

Methods: In a prospective study, 70 people who were eligible for primary PCI and met the criteria for acute STEMI were enrolled. The COBAS c 501 analyzer was used to measure the levels of lipoprotein (a). Patients were allocated two groups based on their lipoprotein (a) levels. Group I had 49 people with low lipoprotein (a) levels (30 mg/dl), and group II had 21 people with high lipoprotein (a) levels (30 mg/dl). Keeping track of hospital results, especially bad cardiac events that are serious (MACE).

Results: High levels of Lp (a) were linked to lower EF (p=0.014) and a higher wall motion score index (p=0.013). People with high Lp (a) levels were more likely to have no reflow during angiography (p=0.007). The same was true for the number of damaged coronary arteries (p=0.007). When Lp levels were high, bad things happened more often in the hospital, like acute heart failure, reinfarction, and death. An Lp (a) cut off value of 24.55 mg/dl was used to predict in-hospital adverse outcomes with a sensitivity of 90.9%, a specificity of 91.7%, and an accuracy of 91.4%.

Conclusion: In STEMI, high plasma Lp (a) levels can be used to predict severe adverse cardiac events.

Keywords: In-hospital lipoprotein (a) outcomes, Acute myocardial infarction with ST elevation, Primary percutaneous coronary intervention, Association.

INTRODUCTION

Acute myocardial infarction with ST-elevation is a medical condition marked by myocardial ischemia, which leads to transmural myocardial ischemia and myocardial necrosis or death [1]. Reperfusion with early percutaneous coronary intervention (PCI) is the standard of care for ST-elevation myocardial infarction (STEMI) patients who get to a hospital with interventional cardiology expertise (PCIP) [2].

Interheart research shows that almost all MIs are caused by cardiovascular risk factors that can be changed, such as high cholesterol, smoking, high blood pressure, stress, inactivity, poor diet, and not drinking alcohol [1, 3]. Dyslipidemia is a major risk factor for coronary heart disease (CHD), and reducing serum low-density lipoprotein cholesterol (LDL-C) levels with statins may help reduce cardiovascular events after percutaneous coronary intervention (PCI) [4, 5]. Several studies, however, have shown that even when LDL levels are low enough, there is still a big risk of heart disease in people who take lipid-lowering drugs [6].

Lipoprotein (a) (Lp[a]) is one of the independent predictors of the development of more difficult coronary artery lesions in PCI patients, and it is thought that lowering Lp (a) may further lower residual cardiovascular risk. However, statin medications have a small effect on Lp (a) reduction [7]. Lipoprotein (a) is a particle made by the liver that looks like low-density lipoprotein (LDL). It is made of an apo B100 (apo B100) molecule that is chemically bound to apolipoprotein (a), which is a very large glycoprotein (apo[a]) [8]. It is not clear what effects the particle has on the body and the blood vessels, but it has been found that Lp (a) can get to the inner layer of human arteries [9].

Studies on cell cultures and animals suggest that Lp (a) may cause blood clots and foam cells to form [10]. Not many studies have looked at the connection between lipoprotein (a) and cardiovascular events (CVEs) in people who have had PCI [11]. This prospective study aimed to find out if there is a link between Lipoprotein (a) [Lp (a)] and how people do in the hospital after having an acute myocardial infarction with ST elevation.

PATIENTS AND METHODS

Methods:

Patients from the Cardiac Care Unit at Egypt's Menoufia University and Sharm El Sheikh International Hospital took part in this study. STEMI is characterised by persistent chest pain that is consistent with myocardial ischemia and ECG criteria that point to STEMI [ST-segment elevation (measured at the J-point) at least two contiguous leads with ST-segment elevation 2.5 mm in men 40 years or older, 2 mm in men 40 years or younger]. The investigation was done between April and August of 2022.

Each participant gave a detailed medical history that included their name, age, gender, and address. Diabetes mellitus (DM), high blood pressure (HTN), ischemic heart disease (IHD), and smoking were all mentioned in the medical histories of the subjects. Complete clinical evaluation including watching blood pressure and heart rate while the person is at rest. A chest auscultation was done to find out if there was congestion in the lungs. As

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part of the regional cardiac evaluation, auscultation was used to listen for heart sounds and murmurs. The Killip classification was used to evaluate and group people with acute myocardial infarction (MI) based on how bad their heart failure was after the MI (HF). An ECG was done to confirm the diagnosis of STEMI and look for any arrhythmias that might have been caused by STEMI.

At the time of admission, a cubital venipuncture was used to take 10 ml of blood from each participant's vein. Lipoprotein (a), complete blood count (CBC), serum creatinine, and lipid profile tests were done on blood samples (total cholesterol, triglycerides, HDL, LDL, and VLDL). People who needed primary percutaneous coronary intervention were taken to the cath lab. It was written down how many coronary arteries had significant stenosis, where and how long the lesion was, how many stents were put in, and if there were any problems, like no blood flow or coronary artery dissection. It was also written down how much contrast was used.

Along with echocardiography, standard parasternal long-axis and short-axis pictures were taken to look at left ventricular (LV) function, abnormalities in wall motion at rest, and the presence of mechanical problems. To figure out the wall motion score index, each segment of the heart should give number between 1 and 4. The 16-segment model of the heart is put forward. Patients were sent to the cardiac care unit after percutaneous coronary intervention. The participants were subsequently separated into two groups depending on their levels of lipoprotein (a):

Group I: Patients having low levels of lipoprotein (a) (< 30 mg/dL). **Group II:** Patients with high levels of lipoprotein (a) (> 30 mg/dL).

Follow-up

Patients were rigorously evaluated for the following outcomes throughout their hospital stay:

Primary Outcomes:

1. Major unfavorable cardiac outcomes include death, stroke, and myocardial infarction recurrence (MACE).
2. In the hospital, complications include sudden pulmonary edema, cardiogenic shock, and severe arrhythmias.

Secondary Outcomes:

1. Hospitalization time.
2. The appearance of contrast-induced nephropathy (CIN).

Ethical approval: After being told about the study's goals, hypotheses, and methods, all the people who took part gave their informed consents, and The Research Ethics Board of Menoufia University gave the study its go-ahead (approval code: 3/2022 CARD 46). This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

RESULTS

Seventy patients with acute ST-elevation myocardial infarction (STEMI) who were candidates for primary percutaneous coronary intervention were included in this prospective trial (PCI). According to their levels of lipoprotein (a), the participants were split into two categories:

Group I: This sample included 49 individuals with low lipoprotein (a) values (less than 30 mg/dL).

Group II: This sample included 21 individuals with high levels of lipoprotein (a) (> 30 mg/dL).

Compared to patients with low lipoprotein a levels, individuals with high lipoprotein a levels were more likely to have a family history of coronary artery disease (CAD) (4 [19.0%] vs. 1 [2.0%], p = 0.01). In terms of smoking, diabetes, and high blood pressure, there were no statistically significant differences between those with high and low lipoprotein (a) levels. (18 [85.7%] vs. 46 [93.9%], p = 0.264; 3 [14.3%] vs. 12 [24.5%], p = 0.340; 6 [28.6%] vs. 15 [30.6%], p = 0.864, respectively) as shown in (Table 1**)**.

Table (1): Sociodemographic data and risk factors findings between cases with normal and high lipoprotein (a)

Table Demographics	Group (I) Lpa (<30) n= 49, Mean ± SD	Group (II) Lpa (>30) n=21, Mean±SD	test of significance	p-value
Age (years)	52.51 ± 9.66	50.90 ± 9.07	t=0.648	0.519
BMI (Kg/m^2)	27.49±3.78	29.52±3.08	t=2.17	0.03*
	No %	No%		
Sex: Male	44 (89.8)	19 (90.5)		
Female	8 (10.2)	2 (9.5)	X^2=0.008	0.931
Smoking: No	3 (6.1)	3 (14.3)		
Yes	46 (93.9)	18 (85.7)	X2FET=1.25	0.264
Family history of CAD: No	48 (98)	17 (81)		
Yes	1 (2.0)	4 (19.0)	X2FET=6.41	0.01*
DM: No	37 (75.5)	18 (85.7)		
Yes	12 (24.5)	3 (14.3)	X^{2FET}=0.909	0.340
HTN: No	34 (69.4)	15 (71.4)		
Yes	15 (30.6)	6 (28.6)	X^2=0.029	0.864

BMI: body mass index **DM:** diabetes mellitus **HTN:** hypertension

Table (2) showed that there were no statistically significant variations in heart rate or blood pressure between the two groups. However, there were statistically significant differences in the timing of the presentations (p=0.0039).

Table (2): Clinical data findings between cases with normal and high lipoprotein.

	Group (I) Mean± SD	Group (II) Mean± SD	Test of significance	p-value
Heart rate (bpm)	85.31±8.40	86.71±17.34	t=0.459	0.648
Blood pressure (mmhg)	134.79±16.80	142.62±25.96	t=1.50	0.137
Killip classification	1.10±0.36	1.9±0.44	t=2.98	0.0039*

Table (3) demonstrated that there was no difference between individuals with high and low lipoprotein (a) levels in terms of the location of ST elevation on the ECG and the follow-up electrocardiogram outcomes.

Table (3): ECG findings between cases with normal and high lipoprotein

Table Demographics	Group (I) Lpa (<30) n= 49 No %	Group (II) Lpa (>30) n=21 No %	test of significance	p-value
ST elevation				
anterior	29 (59.2)	13 (61.9)	χ^{2MC}=0.128	0.938
Inferior	16 (32.7)	6 (28.6)		
lateral	4 (8.2)	2 (9.5)		
Follow up ECG:				
ST resolution	30 (61.2)	7 (33.3)	χ^{2MC}=5.51	0.064
Persistent ST Elevation	1 (2.0)	2 (9.5)		
Q waves	18 (36.7)	12 (57.1)		

CHB: complete heart block **AF:** Atrial fibrillation **VT:** ventricular tachycardia

Those with high levels of lipoprotein (a), as measured by the Simpson method, had a significantly lower left ventricular ejection fraction (EF) than those with low levels of lipoprotein (a) (45.67±11.07 vs. 51.02±6.47, p = 0.014). People with high lipoprotein (a) levels had a significantly higher wall motion score index than those with low lipoprotein (a) levels (a) (1.68±0.39 vs. 1.48±0.24, p = 0.013). Low-density lipoprotein (LDL) and creatine kinase-MB (CK-MB) levels were significantly higher in persons with high lipoprotein (a) levels after 24 hours (a) (146.09 ± 32.63 vs. 122.14 ± 30.02, p = 0.004; 420 [358 - 502.5] vs. 330 [256 - 410], p = 0.006, respectively) as shown in tables (4 & 5).

Table (4): Echocardiographic findings between cases with normal and high lipoprotein.

Table Demographics	Group (I) Lpa (<30) n= 49 Mean± SD	Group (II) Lpa (>30) n=21 Mean± SD	test of significance	p-value
EF (By simpson method)	51.02±6.47	45.67±11.07	t=2.54	0.014*
Wall motion score index	1.48±0.24	1.68±0.39	t=2.56	0.013*
Mechanical complications	NO %	NO %	P=1.0	
No	49 (100)	21 (100)		
yes	0	0		

EF=Ejection fraction t: Student t test, Z: Mann Whitney test, MC: Monte Carlo test, FET: Fischer exact test *statistically significant

Table (5): laboratory investigations findings between cases with normal and high lipoprotein.

Table Demographics	Group (I) Lpa (<30) n= 49 Mean± SD	Group (II) Lpa (>30) n=21 Mean± SD	test of significance	p-value
Hb (g/dL)	13.87±1.69	14.13±1.93	t=0.567	0.572
PLT (x10^3/mm^3)	223.45±51.38	233.14±62.67	t=0.677	0.501
TLC (x10^3/mm^3)	9.17±2.45	9.44±2.60	t=0.415	0.679
LDL (mg/dL)	122.14±30.02	146.09±32.63	t=2.98	0.004*
TG (mg/dL)	181.45±42.33	196.81±59.60	t=1.23	0.225
CK-MB (on admission) (ng/mL)	45 (38-60)	51 (39-60)	z=0.205	0.837
Ck- MB (after 24 hours) (ng/mL)	330 (256-410)	420 (358-502.5)	z=2.75	0.006*
Troponin (on admission) (ng/mL)	0.15 (0.095-0.365)	0.190 (0.1-0.80)	z=1.02	0.310
Troponin (After 24 hours) (ng/mL)	2.3 (1.3-4.5)	2.8 (1.6-5.15)	z=0.712	0.477
Creatinine (mg/dL)	1 (0.9-1.1)	1 (0.87-1.1)	z=0.335	0.737

CK-MB: Creatine Kinase MB Hb: hemoglobin PLT: platelets TLC: total leucocytic count
LDL: low density lipoprotein TG: triglyceride

Regarding other lab tests, table (6) showed that there were no any statistically significant differences between the two groups.

Table (**6):** angiographic data findings between cases with normal and high lipoprotein.

Table Demographics	Group (I) Lpa (<30) n= 49 No%	Group (II) Lpa (>30) n=21 No%	Test. of significance	p-value
Culprit artery			χ^{2MC}=0.664	0.956
LAD	25 (51)	11 (52.4)		
LCX	7 (14.3)	2 (9.5)		
RCA	11 (22.4)	5 (23.8)		
diagonal	1 (2.0)	1 (4.8)		
OM	5 (10.2)	2 (9.5		
Number of stents			χ^{2MC}=2.85	0.415
1	35 (71.4)	18 (85.7)		
2	12 (24.5)	2 (9.5)		
3	1 (2.0)	1 (4.8)		
4	1 (2.0)	0		
Coronary artery dissection			0.0	1.0
No	49 (100)	21 (100)		
yes	0	0		
No reflow			9.89	0.007*
No	49 (100)	17 (81.0)		
Yes	0	4 (19.0)		
Number of vessels affected			χ^{2MC}=9.98	0.007*
1	34 (69.4)	7 (33.3)		
2	12 (24.5)	8 (38.1)		
3	3 (6.1)	6 (28.6)		

LAD: left anterior descending artery LCX: left circumflex artery RCA: right coronary artery

Patients with high lipoprotein (a) levels had a much higher rate of no reflow on angiography than those with low lipoprotein (a) levels. (4 [19.0 %] vs. 0, p = 0.007). Patients with high levels of lipoprotein (a) had much more damage to their coronary arteries than those with low levels of lipoprotein (a) (p = 0.007). In terms of the culprit artery, coronary artery dissection, and number of stents used during primary PCI, there were no big differences between the two groups (p = 0.956, 1.0, and 0.415, respectively). More people with high lipoprotein (a) levels got atrial fibrillation while they were in the hospital than those with low lipoprotein (a) levels. (p = 0.001).

During hospitalization, people with high lipoprotein (a) levels were also much more likely to have acute cardiac failure than those with low lipoprotein (a) levels (p = 0.03). Also, people with high lipoprotein (a) levels had a much higher chance of having another heart attack and dying in the hospital than people with low lipoprotein (a) levels (2 [9.5%] vs. 0, p = 0.001; 1 [4.8%] vs. 0, p = 0.026, respectively). There was no big difference between the two groups in how often strokes happened while they were in the hospital Table 7).

In the univariate analysis, troponin (at admission), heart rate, persistent ST elevation, EF (according to the Simpson technique), wall motion score index, and lipoprotein (a) were found to be significant independent predictors of poor in-hospital outcomes. Also, table the multivariate analysis showed that troponin (on admission) and lipoprotein (a) were the best predictors of bad outcomes in the hospital (Table 8).

As shown in the table (9), ROC curve analysis came up with a lipoprotein (a) cutoff value of 24.55 mg/dL for predicting the occurrence of adverse outcomes in the hospital with a sensitivity of 90.9 %, a specificity of 91.7 %, and an accuracy of 91.4 % for MACE prediction.

Table (7): The in-hospital outcome of the studied cases

Table Demographics	**Group (I) Lpa (<30) n= 49 No %**	**Group (II) Lpa (>30) n=21 No %**	**Test. of significance**	**p-value**
In hospital serious arrhythmia			χ^{2MC}=14.31	0.001*
No	48 (98.0)	14 (66.70)		
AF	1 (2.0)	6 (28.6)		
VT	0 (0.0)	0 (0.0)		
CHB	0 (0.0)	1 (4.8)		
in hospital Heart Failure			χ^{2MC}=5.54	0.03*
No	40 (81.6)	11 (52.4)		
Acute pulmonary edema	8 (16.4)	9 (42.8)		
Cardiogenic shock	1 (2.0)	1 (4.8)		
in hospital cerebrovascular stroke				
No	49 (100)	21 (100)		
Yes	0	0		
in hospital reinfarction			χ^{2MC}=9.97	0.001*
No	49 (100)	19 (90.5)		
Yes	0	2 (9.5)		
in hospital mortality			χ^{2MC}=4.92	0.026*
No	49 (100)	20 (95.2)		
yes	0	1 (4.8)		

AF: atrial fibrillation CHB: complete heart block

Table (8): Univariate and multivariate analysis for prediction of in hospital adverse outcome

Univariate and multivariate analysis for prediction of in hospital adverse outcome					
	Univariate analysis		**Multivariate analysis**		
	p value	Crude Odds ratio (95%CI)	β	p value	Adjusted Odds ratio (95%CI)
Age (years)	0.246	0.967 (0.914-1.02)	0.643	0.308	1.456 (0.551-6.587)
Sex **Male (r)** **Female**	0.496	1.74 (0.354-8.52)	1.021	0.144	0.953 (0.693-1.072)
Smoking **No (r)** **Yes**	0.916	0.909 (0.154-5.38)	2.82	0.247	2.753 (1.30-5.82)
Family history **No (r)** **Yes**	0.176	3.63 (0.561-23.5)	0.018	0.076	1.025 (0.999-1.041)
DM **No (r)** **Yes**	0.655	0.747 (0.209-2.68)	0.034	0.890	1.256 (0.584-1.596)
HTN **No (r)** **Yes**	0.372	0.588 (0.184-1.88)	1.838	0.856	22.743 (0.944-41.895)
Number of stents	**0.272**	**0.553 (0.192-1.59)**	0.028	0.268	1.046 (0.978-1.084)
Culprit artery **LAD (r)** **LCX** **RCA** **diagonal** **OM**	.435 .734 .695 .282	 1 0.505 (0.09-2.8) 0.804 (0.229-2.8) 1.77 (0.102-30.7) 0.295 (0.032-2.73)	 0.340 **1.838** 1.100 0.998 2.144	 0.091 **0.056** 0.072 **0.912** 0.173	1.356 (0.946-2.093) 22.864 (0.944-41.895) 1.157 (0.991 – 1.221) 1.007 (0.980 – 1.019) 2.356 (0.713 – 6.456)
LDL (mg/dL)	.062	1.021 (0.999-1.04)	0.011	0.962	1.256 (0.646-1.583)
TG (mg/dL)	.240	1.009 (0.994-1.02)	0.880	0.316	1.467 (0.074-2.323)
Creatinine (mg/dL)	.195	.078 (0.002-3.69)	0.140	0.560	1.185 (0.543-1.393)
ST elevation **anterior (R)** **Inferior** **lateral**	0.291 0.909	1 0.529 (0.163-1.72) 0.90 (0.147-5.51)	0.868 0.242	0.406 0.525	1.854 (0.054-3.266) 1.257 (0.370-1.661)
Follow up ECG **ST resolution (R)** **Presistant ST Elevation** **Q waves**	0.073 0.009*	1 10.33 (0.803-132.96) 4.52 (1.46-14.01)	1.35 0.871	0.642 0.391	1 3.86 (0.013-150.69) 2.39 (0.327-17.45)
EF (By M-mode)	0.001*	0.872 (0.803-0.947)	0.277	0.672	1.32 (0.365-4.76)
Wall motion score index	0.001*	40.77 (4.77-69.72)	10.98	0.522	58.58 (14.58-69.28)
In hospital serious arrhythmia VT, AF CHB	0.07 1.0	1 20.57 (0.28-185.49) Undefined	0.513 0.101	0.694 0.061	2.858 (0.046-7.824) 0.963 (0.812-1.005)
LPA **Normal (R)** **high**	<0.001*	67.5 (13.72-89.58)	7.97	0.003 *	60.25 (14.58-90.56)
Overall % predicted=90% **Model χ^2=33.85, p<0.001***					

HTN: hypertension DM: diabetes mellitus CHD: coronary heart disease AF: Atrial fibrillation EF=Ejection fraction LAD: left anterior descending artery LCX: left circumflex artery RCA: right coronary artery LDL: low density lipoprotein TG: triglyceride.

Table (9): ROC curve using Lpa as continuous variable in prediction of MACE.

AUC (95% CI)	**0.925 (0.844-1.01)**
P value	<0.001*
cut off point	24.55
Sensitivity	90.9%
Specificity	91.7%
PPV	83.3%
NPV	95.7%
Accuracy	91.4%

AUC: Area undercurve, PPV: Positive predictive value, NPV: Negative predictive value.

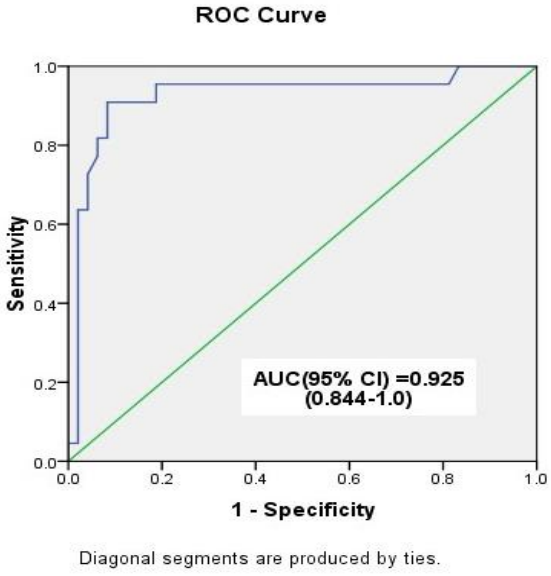

Figure (1): ROC Curve

DISCUSSION

Endothelial dysfunction, macrophage migration and proliferation, foam cell buildup, and necrotic core expansion are all caused by Lp (a) inflammatory and oxidative effects. It is a risk factor for atherosclerosis and blood clots. Lipoprotein (a) is an independent predictor of the development of more problematic coronary artery lesions in PCI patients and reducing Lp (a) may decrease cardiovascular risk further. Many individuals worldwide are killed or disabled by acute myocardial infarction (AMI) and coronary artery disease (CAD). The objective of AMI therapy is to immediately restore blood supply to the injured myocardium. Primary percutaneous coronary intervention (PPCI), which is performed immediately and by a specialist, may assist with acute ST-elevation myocardial infarction (STEMI). When done swiftly and well by a competent crew [12].

The current research sought to determine if there was a connection between Lp (a) levels and how patients fared in the hospital after a primary percutaneous coronary intervention for an acute ST-elevation myocardial infarction (PCI).

Acute cardiac failure was substantially more common in individuals with high Lp (a) than in those with low Lp (a) (p-value 0.03). Individuals with high Lp (a) had substantially higher rates of reinfection and death in hospitals than those with low Lp (a), with p-values of 0.001 and 0.026, respectively. According to our findings, individuals with high Lp had considerably lower EF (as measured by the Simpson technique) than those with low Lp (a) (45.67±11.07 vs 51.02±6.47) p-value 0.014. The high Lp group (a) had a considerably higher wall motion score index than the low Lp group (b) (1.68±0.39 vs 1.48±0.24) p-value 0.013.

There is most likely a lot going on here. Patients with high Lp, for example, may have greater congestive heart failure because ischemia damage is severe (a). High Lp (a) patients had 70% greater coronary stenosis, were more likely to have type C lesions and left anterior descending coronary stenosis, and had reduced coronary perfusion before and after PCI treatment. Those with high Lp (a) had a higher unadjusted risk of acute stent thrombosis than those with low Lp (a). This might be because TIMI flow was less optimal in these ACS groups following PCI [13].

According to a recent study, the amount of Lp (a) in the blood was shown to be associated to the severity of coronary artery disease (CAD), but not to events in patients with stable CAD. **Peng *et al.*** [14] observed that plasma Lp (a) concentration was linked to severe insight on the possible reasons of no-reflow in persons who have had PCI for a long period. Ischemia and reperfusion may damage the tiny blood arteries in the heart, resulting in no-reflow. We assumed that arrhythmias in STEMI patients who were hospitalized.

In comparison to patients with low Lp (a), patients with high Lp (a) had no significant increase in reflow. (4 (19.0%) vs 0) (p-value= 0.007). The number of coronary arteries impacted in the high Lp group was significantly greater than in the low Lp group (a) (4 (19.0 %) vs 0), (p-value 0.007). Many researches have given that Lp (a) is associated with distal embolization, making it more probable that there would be no reflow. There are several methods to explain this connection. The existence of a lipid-rich plaque and the size of the thrombus are both associated with micro-embolization in the coronary arteries. Prior research by **Lima *et al.*** [15] and **You *et al.*** [16] found that high Lp (a) was associated with more severe coronary artery stenosis as detected by angiography, as well as less coronary collateral circulation in patients with acute myocardial infarction; and high Lp (a) was associated with a more severe coronary artery lesion as reflected by an increased number of vessels affected and an increased number of vessels with no reflow.

Individuals with high Lp (a) were hospitalised with considerably more AF than those with low Lp (a) in our research (p-value 0.001). Another investigation, which supports our findings, revealed that plasma Lp (a) concentration was related with in-hospital severe arrhythmias in STEMI patients. Lp is one-of-a-kind molecule possessing pro-atherogenic, pro-thrombotic, and pro-inflammatory characteristics, which explains its role in atrial fibrillation (AF). Because acute coronary

syndrome (ACS) increases endothelial permeability, Lp (a) may enter atrial tissue and cause microcalcifications, electrical remodeling, and local cell death [17].

According to our findings, those with high Lp (a) had considerably higher LDL levels than those with low Lp (a) (146.09 ± 32.63vs 122.14 ± 30.02) (p-value = 0.004). This is consistent with the findings of **Gencer *et al.*** [18] who observed that higher Lp (a) plasma levels were associated with higher LDLC values. **Afshar *et al.*** [19] found that the risk of Lp (a) in individuals with premature ACS was greater in those with concurrently increased LDLC values.

Because Lp (a) levels are heavily controlled by genetic factors, it is considered a hereditary risk factor for atherosclerotic cardiovascular disease. When these pathophysiological pathways are linked, those with high Lp (a) have a substantially greater residual cardiovascular risk than people with low Lp (a) [20].

In the current investigation, Lp (a) had a sensitivity of 90.9%, a specificity of 91.7% and an accuracy of 91.4% at a threshold of 24.55 mg/dl for predicting MACE. The definition of the Lp (a) limit for vulnerable populations is disputed [21].

CONCLUSION

According to the results of this investigation, higher plasma Lp (a) levels might be employed as a surrogate marker for the prediction of severe adverse cardiac events with great sensitivity and specificity. When people present with acute myocardial infarction with ST elevation, a high plasma Lp (a) level is a strong predictor of hospital death. Because of this, determining an individual's Lp (a) levels provides a novel way to more aggressive therapy for those who are at a high risk.

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