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## Prevalence and Pattern of Dyslipidemia in Acute Coronary Syndrome Patients Admitted to Medical Intensive Care Unit in Zagazig University Hospital, Egypt

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**Background:** Coronary artery disease (CAD) is the leading cause of mortality in men and women. Acute coronary syndrome (ACS), is a major reason for hospitalization in our country. Dyslipidemia has been identified as one of the most important modifiable risk factors for CAD.

**Aim:** The aim of the study was to determine the prevalence and pattern of dyslipidemia and its relation to other modifiable risk factors in patients admitted with ACS to Medical ICU in Zagazig University Hospital, Egypt within a period of 11 months.

**Subjects and methods:** 170 subjects were included; 150 patients with ACS classified according to clinical presentation, the findings on the admission electrocardiogram (ECG) and the results of serial cardiac troponin levels, into myocardial infarction(MI), either ST-elevation or non ST- elevation MI, and unstable angina(UA) subgroups. The other group included 20 healthy subjects as controls. All subjects were subjected determination lipid profile and lipoprotein (a) [Lp(a)] . Cardiac troponin and ECGs were performed for diagnosis and follow up of the patients.

**Results:** In patients with ACS, high levels of TC ( $> 200$  mg/dl) were found in 60.67% ,high levels of LDL ( $> 130$  mg/dl) were found in 58%, high levels of TG ( $> 150$  mg/dl) were found in 63.33% and high levels of Lp(a) ( $> 30$  mg/dl) were found in 62%, however, low levels of HDL ( $< 40$  mg/dl) were found in 66% . There was a statistically significant elevation in TC, LDL, TG and Lp(a) serum levels in patients with ACS compared to control subjects ( $p < 0.05$ ) while the HDL was significantly low in ACS patient compared to control subjects ( $p < 0.05$ ). TC/HDL  $> 5$  and TG/HDL  $> 4$  were significantly higher in patients with ACS than controls. There was no significant difference between MI and UA patients regarding all lipid profile parameters. TC, LDL, TG and [Lp(a)] were significantly higher in males than in females while HDL was significantly higher in females compared to males. Also TC/HDL and TG/HDL ratios were significantly higher in males compared to females. All lipid components were significantly more prevalent in males than in females except TG where there was no significant difference between males and females. The Lp(a) values were significantly elevated in diabetic and in smoker patients with ACS ( $p < 0.05$ ) while the high values in hypertensive patients were insignificant ( $p = 0.167$ ). Stepwise regression analysis of lipid parameters revealed that TC/HDL and TG/HDL ratios were independent risk factors for ACS.

**Conclusion:** Dyslipidemia is one the major risk factors which is widely prevalent in patients with ACS and is more prevalent in males than in females. High Lp (a) is widely prevalent among patients with ACS, especially those with diabetes mellitus and smokers. We recommend paying more attention to serum lipids and other modifiable risk factors for prevention of ACS and more studies about Lp (a) as a risk factor of atherosclerosis and its impact on other systems is advised.

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### Introduction

Coronary artery disease (CAD) is the leading cause of death in men and women [1]. Regardless of declines in developed countries, both CAD mortality and the prevalence of

CAD risk factors continue to rise rapidly in developing countries [2].

The risk factors of CAD used for the categorization and setting of management targets have been established on the basis of evidence accumulated over a long time [3].

Hypertension, diabetes mellitus and Cigarette smoking have been reported to be risk factors of CAD and stroke through many studies [4, 5, 6], respectively. The risk of CAD was about 4 and 3 times higher in male and female smokers than nonsmokers respectively [6].

Elevated levels of total- and low density lipoprotein cholesterol (TC and LDL-C), elevated levels of triglycerides (TG) and low levels of high density lipoprotein cholesterol (HDL-C) are important risk factors for CAD [7]. LDL-C is considered as 'bad cholesterol' since too high level of this cholesterol is associated with an increased risk of coronary artery disease and stroke. Lp(a) is a LDL particle with apolipoprotein(a) [apo(a)] protein attached loosely to the surface by a disulfide bond linked to the only protein on LDL particle, namely apolipoprotein B (apoB). The same hazard is associated with elevated blood Lp(a) levels [8]. Epidemiologic and prospective studies have revealed elevated levels of Lp (a) in persons with CAD (Mark et al., 2003) [9].

Previous findings suggested that the content of oxidized phospholipids in Lp(a), constitutes an independent predictive marker for cardiovascular (CV) morbidity and mortality [10].

Apo(a) is a distinctive glycoprotein of Lp(a) and is structurally homologous to plasminogen: a key protein of the fibrinolytic system. Lp(a) does not have fibrinolytic activity and it competes with plasminogen for the binding site on surface of the endothelial cells. Thus it prevents the activation of plasminogen by the tissue plasminogen activator and may, therefore, promote a procoagulant state. Treating dyslipidemia has clear benefits in the primary and secondary

**Table 1: Normal and abnormal levels of lipid profile. [12].**

	Recommended	Borderline	High risk
TC	<200 mg/dl	200-240 mg/dl	>240 mg/dl
HDL	≥40 mg/dl	<40 mg/dl	
LDL	<130 mg/dl	130-160 mg/dl	>160 mg/dl
TG	<150 mg/dl	150-200 mg/dl	>200 mg/dl

prevention of coronary heart disease (CHD) in both sexes [11].

This study focused on dyslipidemia as a risk factor of acute coronary syndrome (ACS). The aim of the study was to determine the prevalence and pattern of dyslipidemia in subjects with acute coronary syndrome, its relation to age, gender and other modifiable risk factors.

## Subjects and Methods

This prospective, descriptive and observational study was carried out in the Medical Intensive Care Unit (ICU) of the Internal Medicine and Biochemistry Departments, Zagazig University Hospital, during the period from September 2012 to August 2013.

The study included two groups:

**Group I:** included 150 patients with ACS with the age ranged from 42 to 81 years with a mean age  $\pm$ SD of 61.50 ys $\pm$ 8.24. Male patients in the study were 91 (60.67% of patients) [mean age  $\pm$ SD (61.35 ys $\pm$ 7.70)] while female patients were 59 (39.33% of patients) [mean age $\pm$ SD (61.39 ys $\pm$ 9.37)]. Regarding risk factors, we found that 94 patients (62.67% of patients) were hypertensive, 79 patients (52.67% of patients) were diabetic and 71 patients (47.33% of patients) were smokers. All smokers were males.

ACS were classified according to clinical presentation, the findings on the admission electrocardiogram (ECG) and the results of serial cardiac troponin levels into;

**-ST-elevation ACS (STE-ACS)** patients presented with acute chest pain, persistent (>20 minutes) ST-segment elevation and a rise in troponin levels [**ST-elevation MI (STEMI)**].

**-Non-ST-elevation ACS (NSTEMI-ACS)** patients presented with acute chest pain but without persistent ST-segment elevation. The ECG shows persistent or transient ST-segment depression or T-wave inversion, flat T waves, pseudo-normalisation of T waves, or no ECG changes at presentation. NSTEMI-ACS is further divided into:

**Unstable angina** normal troponin levels.

**Non-ST-elevation MI (NSTEMI)** with a rise in troponin levels.

**Group II:** included 20 healthy subjects not diabetic, not hypertensive and nonsmokers. Their ages ranged from 49 to 62 years with a mean age of 57ys±3.53 years. They were 11 males and 9 females.

### Exclusion Criteria

Patients with stable angina and those receiving hypolipidemic drugs were excluded from the study.

### Methods

A written consent to participate in the study was taken from each subject. All subjects participated in this study were subjected to the following:

**I-**Thorough history of present illness and history of any other diseases including diabetes mellitus and hypertension. History of previous attacks of acute coronary syndrome and family history of ischemic heart disease were recorded, history of smoking and previous hospital admission were taken in consideration.

**II** Clinical examination with attention to:

- a- blood pressure, measured by a mercury sphygmomanometer with the subject

**Table (2): Disease distribution in the study.**

Disease	Whole study		Gender	
	N	%	Male (n=91)	Female (n=59)
Myocardial infarction	118	78.6%	n=76 (83.52%)	n=42 (71.19%)
Unstable angina	32	21.33%	n=15 (16.48%)	n=17 (28.81%)

recumbent in bed, with the arm supported and positioned at the level of the heart.

b- Pulse

c- Cardiac examination:

### III- Routine investigations

Complete blood count (CBC), lipid profile (**table 1**) [total cholesterol (TC), high density lipoprotein (HDL), low density lipoprotein (LDL) and triglycerides (TG), liver function tests, kidney function tests and fasting blood glucose level. ECG: 12 leads ECGs were performed for diagnosis and follow up of the case.

### Specific investigations

**a** Troponin: Cardiac troponin assay was done using a latex enhanced immunoturbidimetric method [13].

**b** Lipoprotein (a) :Lp(a) were measured by a quantitative turbidimetric test for the measurement of Lp (a) in human serum or plasma. According to the used kits, the normal and abnormal levels of Lp(a) were as the following ; desirable values was < 14 mg/dL, borderline risk values ranged from 14 - 30 mg/dL and high risk values ranged from 31 - 50 mg/dL [14].

**Sample taking** 5ml blood were withdrawn from each case after overnight fasting of 12 hours then centrifuged at 3000 rpm for 10 minutes. 1 ml of serum was kept at -20°C for measurement of lipoprotein (a).

### Statistical Analysis

Statistical presentation and analysis of the collected data were conducted, using the

mean, standard deviation, analysis of variance [ANOVA] test and chi-square test by the SPSS statistical software version 18 for windows [15].

## Results

Myocardial infarction patients included STEMI and NSTEMI Hypertension was found in 94 patients (62.67% of subjects). 79 patients were diabetics (52.67% of patients). Finally, smoking was a habit in 71 patients (47.33% of patients). ). **(Table 3)**

$P < 0.05$  means a significant difference between MI and UA regarding the modifiable risk factors.

Table 5 showed high levels of TC (more than 200 mg/dl) were found in 91 patients (60.67% of patients), high levels of LDL (more than 130 mg/dl) were found in 87 patients (58% of patients), high levels of TG (more than 150 mg/dl) were found in 95 subjects (63.33% of patients) and high levels of Lp(a) (more than 30 mg/dl) were found in 93 patients (62%). However, low levels of HDL (less than 40 mg/dl) were found in 99 patients (66% of patients).

This table showed that there was a statistically significant elevation in TC (total cholesterol), LDL (low density lipoprotein), TG (triglyceride) and Lp(a) [lipoprotein(a)] serum levels in patients with ACS compared to control subjects while the HDL (high density lipoprotein) was significantly low in ACS patients compared to control subjects.  $TC/HDL > 5$  and  $TG/HDL > 4$  were significantly higher in patients with ACS than controls.

There was no significant difference between myocardial infarction and unstable angina regarding all parameters.

TC, LDL, TG and Lp(a) were significantly higher in males than in females while HDL was significantly higher in females compared to males. Also  $TC/HDL$  and  $TG/HDL$  ratios were significantly higher in males compared to females.

**Table (3): Prevalence of the risk factors in the study**

	Number	percentage
Hypertention	94	62.67%
DM	79	52.67%
Smoking	71	47.33%

All components were significantly more prevalent in males than in females except TG where there was no significant difference between males and females

The Lp(a) values were significantly elevated in diabetic and in smoker patients with ACS while the high values in hypertensive patients were insignificant. This table revealed that  $TC/HDL$  and  $TG/HDL$  ratios were independent risk factors for ACS.

## Discussion

CAD is a complex and multifactorial process that manifests as stable angina, unstable angina or myocardial infarction. The atherosclerotic process underlies each of these pathologies. Indeed, clinical symptomatology in CAD is frequently triggered by a thrombus formation on an eroded or ruptured atherosclerotic, lipid-rich plaque characterized by a thin fibrous cap [16].

CAD is the leading cause of death in men and women [1]. Dyslipidemia preponderated among the nine major risk factors (smoking, diabetes, hypertension, visceral obesity, psychosocial stress, sedentary life, low fruit and vegetable consumption and alcohol consumption), and alone accounted for more than 50% of population attributable risk [17]. Regardless of declines in developed countries, both CAD mortality and the prevalence of CAD risk factors continue to rise rapidly in developing countries [2]. Hypertension is a clear risk factor of atherosclerotic CAD [18]. The risk of CAD has been reported to be 2-6 times higher in diabetics than in non-diabetics [4]. Cigarette smoking has been reported to be a risk factor of CAD and stroke through many studies [5]. The risk of CAD was about 4 and 3 times higher in male and

**Table (4): Prevalence of the risk factors in relation to gender and ACS.**

	Hypertention		DM		Smoking	
	Number	%	Number	%	Number	%
Males	65	71.43%	45	49.45%	71	78.02%
Females	39	66.10%	34	57.63%	0	0%
MI	79	66.95%	63	53.39%	59	50%
UA	15	46.88%	16	50%	12	37.50%
P	0.023		0.231		0.037	

female smokers than nonsmokers respectively [6].

Dyslipidemia, manifested by elevated levels of total- and low density lipoprotein cholesterol (TC, LDL-C), low levels of high density lipoprotein cholesterol (HDL-C) and high levels of triglycerides (TG), is an important risk factor for CAD [19]. The relationship of Lp(a) to CAD is considered to be due to interference by Lp(a) with the physiologic roles of plasminogen in the fibrinolytic system. Moreover, Lp(a) is speculated to promote arteriosclerosis by inducing cholesterol deposition in the vascular wall, proliferation of vascular wall cells, and binding of cholesterol to oxidized phospholipids [20].

Our study revealed that hypertension is the most common risk factor of ACS (62.67%) followed by diabetes mellitus (52.67%). Cigarette smoking came at the last (47.33%). Our results agreed with Sandhu *et al.*, Lahoz *et al.*, and Cooke *et al.*, (2006). [21, 22, 23].

Saito *et al.*, [24] found that the prevalence of hypertension was 45.8%, diabetes mellitus

was 15.8% while cigarette smoking was 16.7% which differs regarding the prevalence from our study. According to Saito *et al.*, [24], hypertension was the commonest risk factor of acute coronary syndrome followed by cigarette smoking, diabetes mellitus came at the last. In our study smoking, as a risk factor for ACS, came after hypertension and diabetes mellitus, probably, because all our female patients (39.33% of our subjects) were nonsmoker.

Our study revealed that myocardial infarction (MI) was found in 118 patients (78.67% of patients) while unstable angina (UA) was found in 32 patients (21.33% of patients). In MI, 79 patients (66.95%) were hypertensive, 63 patients (53.39%) were diabetic and 59 patients (50%) were smokers. On the other hand, in UA, 15 patients (46.88%) were hypertensive, 16 patients (50%) were diabetic and 12 patients (37.50%) were smokers. The increased prevalence of hypertension and smoking were significant in patients with MI ( $p < 0.05$ ) compared to those with UA while it was insignificant regarding diabetes, [table, 5]. Esteghamati *et al.*, [25], in agreement with our results, found that the prevalence of

**Table 5: Prevalence and pattern of dyslipidemia in patients with ACS.**

Type of lipid	Number	Percentage
Total cholesterol [TC](>200 mg/dl)	91	60.67%
Low density lipoprotein [LDL] (>130 mg/dl)	87	58%
High density lipoprotein [HDL] (<40 mg/dl)	99	66%
Triglycerides [TG] (>150 mg/dl)	95	63.33%
Lipoprotein (a) [Lp(a)] (>30 mg/dl)	93	62%

**Table (6): Comparison between lipid profile parameters in patients with ACS and control subjects.**

	Control group (Mean $\pm$ SD)	ACS group (Mean $\pm$ SD)	p-value
TC (mg/dl)	167.45 $\pm$ 3.23	217.87 $\pm$ 43.61	0.023
LDL(mg/dl)	97.67 $\pm$ 6.42	139.25 $\pm$ 38.43	0.02
HDL (mg/dl)	49.65 $\pm$ 2.32	37.88 $\pm$ 4.79	0.034
TG (mg/dl)	137.41 $\pm$ 5.78	174.41 $\pm$ 61.42	0.043
Lp(a) (mg/dl)	22.65 $\pm$ 4.87	53.26 $\pm$ 41.59	0.008
TC/HDL	3.37	5.75	0.015
TG/HDL	2.77	4.604	0.032

**Table 7: Comparison between lipid profile parameters in MI and UA patients**

	Myocardial Infarction (Mean ± SD)	Unstable Angina (Mean ± SD)	P
TC (mg/dl)	217.84±44.99	217.97±38.75	NS
LDL (mg/dl)	139.34±39.94	138.91±32.82	NS
HDL (mg/dl)	37.80±4.77	38.15±4.91	NS
TG (mg/dl)	176.45±66.04	175.75±52.95	NS
Lp(a) (mg/dl)	56.19±43.29	42.44±32.96	NS
TC/HDL	5.74	5.71	NS
TG/HDL	4.72	4.60	NS

\*p < 0.05 means significant.

hypertension and smoking were significantly higher in patients with MI compared to those with UA ( 96% vs 89.2% for hypertension and 52.8% vs. 38.6% for smoking) while diabetes mellitus was significantly higher in patients with MI compared to patients with UA which was different from our results (44.6% vs 25.2% ). Also our results revealed that there was no significant difference between patients with MI and UA regarding all lipid profile parameters (Table, 7) which did not agree with that of Guler *et al.*, [26], and Esteghamati *et al.*, [25] who reported that Low levels of HDL were significantly low in subjects with MI compared to those with UA.

Regarding prevalence and pattern of dyslipidemia in our study, [table, 6], we found that high levels of TC (more than 200mg/dl) were found in 91 patients (60.67% of patients) [mean±SD (217.87 mg/dl±43.61)]. High levels of LDL (more than 130 mg/dl) were found in 87 patients (58% of patients) [mean±SD (139.25 mg/dl±38.43)]. Low levels of HDL (less than 40 mg/dl) were found in 99 patients (66% of patients) [mean±SD (37.88 mg/dl±4.79)]. High levels of TG (more than 150 mg/dl) were found in 95 patients (63.33% of patients) [mean±SD (174.41 mg/dl±61.42)]. Also, our results revealed that the TC/HDL ratio was more than five (TC/HDL>5) and TG/HDL ratio was more than four (TG/HDL>4). . According to the

**Table 8: Pattern of dyslipidemia in ACS patients in relation to gender**

	Male ( Mean ± SD)	Female (Mean ± SD)	P
TC (mg/dl)	225.17±43.90	206.61±41.02	0.01
LDL (mg/dl)	146.18±40.14	128.56±33.19	0.006
HDL (mg/dl)	36±4.64	42.91±4.86	0.034
TG (mg/dl)	183.54±66.18	163.64±57.37	0.012
Lp(a) (mg/dl)	60.26±44.53	42.46±34.23	0.007
TC/HDL	6.19	4.81	0.037
TG/HDL	5.04	3.85	0.029

\*p < 0.05 means significant.

American Heart Association, the goal is to keep TC/HDL ratio < 5 and TG/HDL < 4. A higher ratios indicates a higher risk of heart disease; a lower ratio indicates a lower risk.

Finally, high levels Lp(a) (more than 30 mg/dl) were found in 93 patients (62% of patients).

Assessment of lipid profile parameters revealed that there was a statistically significant elevation in serum levels of TC, LDL, TG, TC/HDL, TG/HDL and Lp(a) in ACS patients compared with the control subjects while regarding HDL it was significantly low in ACS patients compared to the control subjects (p < 0.05) [Table 8]. Our results were in agreement with that of Kamariya *et al.*, [27] and Yadav and Bhagwat [19] who reported increased TC, TG, LDL, Lp(a) and decreased HDL levels in patients with ACS than controls.

The prevalence of MI was higher in male gender than females (83.52 % vs 71.19 % respectively) [table 2]. This can be explained by our finding that hypertension and smoking were more prevalent in males than in females. Smokers were only males. Regarding the prevalence of diabetes, there were 45 diabetic males (49.45 %) vs 34 diabetic females (57.63%) which was statistically insignificant [table, 4]. Another factor which can explain occurrence of MI in males than females was the more prevalent dyslipidemia

**Table 9: Prevalence of dyslipidemia in ACS patients in relation to gender.**

	Male (n=91)		Female (n=59)		p-value
TC (>200 mg/dl)	60	65.93%	31	52.54%	0.023
LDL (>130 mg/dl)	60	65.93%	27	45.76%	0.042
HDL (<40 mg/dl)	65	71.43%	34	57.63%	0.011
TG (>150 mg/dl)	59	64.84%	36	61.01%	0.053
Lp(a) (> 30 mg/dl)	62	68.13%	31	52.54%	0.008

\*p < 0.05 means significant.

in male than female patients [Table, 7]. Our results agreed with that of Leebmann *et al.*, [28], El-Menyal *et al.*, [29], Youssef *et al.*, [30] and Nouredine *et al.*, [31] who reported that MI was more prevalent in males than females.

The prevalence of dyslipidemia and its pattern in patients with ACS were more significant in males than females (p<0.01) [tables 8 & 10].

The same findings were present for Lp(a) which were significantly more prevalent in male patients compared to females (p<0.05) while Low levels of HDL (<40 mg/dl) were significant in male patients compared to female patients (p=0.034), (71.43% of male patients with mean±SD of HDL of 37mg/dl±4.64) vs. (65.63% of female patients with the mean±SD of HDL of 42.91 mg/dl±4.86)

A higher levels of TG [mean ±SD (174.41mg/dl±61.42)] were found in males compared to that in females [mean ±SD (173.41mg/dl±5, 78)], which was insignificant (p=0.172). These results were the same results of Jacob *et al.*, [32] who reported that men had higher TG and TC levels and lower HDL-levels compared to women (P < 0.001). On the other hand, Esteghamati *et al.*, [25] found that mean levels of TG were lower in male patients [170.6±97.3 mg/dl] than in female patients [188.4±88.3 mg/dl]. That would be due to differences in genetics, body fat distribution, life styles and dietary habits between different countries where studies were carried out.

**Table 10: Prevalence of lipoprotein (a) in relation to risk factors**

	Lp(a) > 30 mg/dl		P < 0.05 = significant
Hypertension	61 patients	64.90%	0.167
DM	54 patients	68.35%	0.045
Smoking	53 patients	74.65%	0.021

Our results revealed that high Lp(a) values were more prevalent in males than females, (68.13% of male patients vs 52.54% of female patients). Also Lp(a) values were significantly higher in male than female patients with a mean±SD of 60.26mg/dl ±44.53 vs in males vs a mean±SD of 42.46mg/dl±34.23 in females respectively (p < 0.007). Guler *et al.*, [26] and Lima *et al.*, [33] reported the same findings

In relation to risk factors, we found that Lp(a) was significantly higher in diabetics and smokers than non diabetics and nonsmokers but there was no significant difference regarding hypertension in our patients. Jatav *et al.*, [34] found that levels of lipoprotein (a) were significantly higher in diabetic patients (87%) than in non-diabetic ones (61%) (p<0.05), which agreed with our results.

Although lipoprotein (a) was significantly prevalent in diabetics which is mildly more common in females, smoking increases levels of lipoprotein (a) which might be the cause that levels of lipoprotein (a) were significantly higher in males than in females, as smoking was prevalent in our males patients only [35]. Using stepwise regression of lipid profile parameters we found that TC/HDL and TG/HDL ratios were independent risk factors for ACS. [Table 11]

## Conclusions

In conclusion, this study revealed that hypertension was the most common risk factor followed by diabetes mellitus. Smoking was the last risk factor. Hypertension and smoking were more prevalent in males than in females. Regarding dyslipidemia, we found that dyslipidemia was prevalent in ACS patients compared to control. Low level of

**Table 11: Stepwise regression analysis of dyslipidemia in relation to ACS**

	B	Std. Error for B	$\beta$	Sig.	95% Confidence Interval for B	
					Lower	Higher
TC	.000	.003	-.034	NS	-.007	.006
LDL	.000	.003	.032	NS	-.006	-.006
HDL	.003	.008	.040	NS	-.012	.019
TG	.000	.001	-.141	NS	-.002	.002
TC/HDL	2.435	1.231	0.031	Sig.	1.549	78.86
TG/HDL	2.201	0.768	0.41	Sig.	1.386	56.12
Lp(a)	-.001	.001	-.141	NS	-.003	.000
(Constant)	1.133	.364	---	0.002	---	---

HDL was the most common, followed by high TG, high Lp(a), high TC then high level of LDL, TC/HDL and TG/HDL ratios. Dyslipidemia was significantly related to gender. TC, LDL, TG and Lp(a) were significantly higher in males than in females. There was no significant difference between patients with MI and those with UA regarding all lipid profile parameters. We also found that HDL was significantly lower in male than females. High levels of Lp(a) were prevalent in 62% of patients with ACS. It was significantly higher in males than in females. We also found that high level of Lp(a) was significantly higher in diabetics than non-diabetics. Generally, except HDL, levels of all types of dyslipidemia were higher in males than in females. Using backward stepwise logistic regression analysis of dyslipidemia, we found that TC/HDL and TG/HDL ratios were independent risk factors for ACS.

Based on these results, we can recommend to pay more attention to serum lipids for prevention of acute coronary syndrome, periodic check of fasting lipid profile and enriching the people culture about dyslipidemia, its hazards and how to avoid. Further studies about LP (a) as a risk factor of atherosclerosis and its impact on other systems.

### Conflict of Interests

The authors declare that there are no conflicts of interests

### Authors' Contribution

**AA:** Preparation of the draft manuscript and literature search

**MSF:** Concept and design, editing of the manuscript.

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None declared

### Ethical Considerations

The study was approved by the Institute Ethics Committee.

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### References

1. Thom T, Haase N, Rosamond W, et al.: Heart Disease and Stroke. Statistics-2006 Update. A Report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*. 2006; 113:e85. [[Pubmed](#)]
2. Okrainec K, Banerjee D, Kand Eisenberg MJ.: Coronary artery disease in the developing world. *Am Heart J*. 2003; 148:7-15. [[Pubmed](#)]
3. Ueshima H, Choudhury SR, Okayama A, Hayakawa T, Kita Y, Kadowaki T, Okamura T, Minowa M, Iimura O; NIPPON DATA80 Research Group: Cigarette Smoking as a Risk Factor for Stroke Death in Japan: NIPPON DATA80. *Stroke*, 2004; 35:1836-1841. [[Pubmed](#)]
4. Haffner SM, Lehto S, Rönemaa T, Pyörälä K, Laakso M: Mortality from coronary heart disease in subjects with type 2 diabetes and in

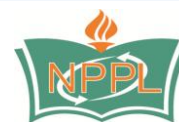


- nondiabetic subjects with and without prior myocardial infarction. *N Engl J Med*, 1998; 339:229-234. [[PubMed](#)]
5. Wolf PA, D'Agostino RB, Kannel WB, Bonita R, Belanger AJ: Cigarette smoking as a risk factor for stroke: the Framingham Study. *JAMA*, 1988; 259:1025-1029. [[PubMed](#)]
  6. Baba S, Iso H, Mannami T, Sasaki S, Okada K, and Konishi M; Shoichiro Tsugane; JPHC Study Group: Cigarette smoking and risk of coronary heart disease incidence among middle-aged Japanese men and women; the JPHC Study Cohort. *Eur J Cardiovasc Prev Rehabil*, 2006; 13:207-213. [[PubMed](#)]
  7. Yadav AS and Bhagwat VR: Lipid Profile Pattern in Anginal Syndrome Patients From Marathwada Region of Maharashtra State *Journal of Medical Education & Research*, 2012; Vol. 2, No.2, 12-15. July-Dec 2012
  8. Emerging Risk Factors Collaboration, Erqou S, Kaptoge S, Perry PL, Di Angelantonio E, Thompson A, White IR, Marcovina SM, Collins R, Thompson SG, Danesh J. Lipoprotein(a) concentration and the risk of coronary heart disease, stroke, and nonvascular mortality. *JAMA*. 2009 Jul 22; 302(4):412-23. [[PubMed](#)]
  9. Mark AH, Marcovina SM, et al.: Inhibition of Plasminogen Activation by Lipoprotein(a). *Journal of Biological Chemistry*. 2003; June 27, 23260 – 69. [[PubMed](#)]
  10. Kiechl S, Willeit J, Mayr M, et al.: Oxidized phospholipids, lipoprotein(a), lipoprotein-associated phospholipase A2 activity, and 10-year cardiovascular outcomes: prospective results from the Bruneck study. *ArteriosclerThromb Vasc Biol*. 2007; 27(8):1788-1795. [[PubMed](#)]
  11. Kakafika AI, Athyros VG, Tziomalos K, Karagiannis A, Mikhailidis DP: Primary and secondary coronary heart disease prevention using statins: is targeting Adam or Eve equally effective? *Expert Opin Pharmacother* 2008; 9:1437–40. [[PubMed](#)]
  12. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults: Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP), Adult Treatment Panel III (ATP III). *JAMA* 2001; 285(19):2486-2497. [[PubMed](#)]
  13. Thygesen K, Mair J, Katus H, Plebani M, Venge P, Collinson P, Lindahl B, Giannitsis E, Hasin Y, Galvani M, Tubaro M, Alpert JS, Biasucci LM, Koenig W, Mueller C, Huber K, Hamm C, Jaffe AS: Study Group on Biomarkers in Cardiology of the ESC Working Group on Acute Cardiac Care. Recommendations for the use of cardiac troponin measurement in acute cardiac care. *Eur Heart J* 2010; 31:2197 – 2204.
  14. Ryan GM, Torelli J: Beyond cholesterol: 7 life saving heart disease tests that your doctor may not give you. New York: St. Martin's Griffin. 2005; p. 91.
  15. Yadolah: The Oxford Dictionary of Statistical Terms. Oxford University Press, 2003. ISBN 0-19-920613-9.
  16. Libby P: The molecular mechanisms of the thrombotic complications of atherosclerosis. *J Intern Med* 2008; 263, 517–527. [[PubMed](#)]
  17. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al.: Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet* 2004; 364, 937–952. [[PubMed](#)]
  18. Kannel WB: Fifty years of Framingham Study contributions to understanding hypertension. *J Hum Hypertens*, 2000; 14:83-90. [[PubMed](#)]
  19. Yadav AS, Bhagwat VR: Lipid Profile Pattern in Anginal Syndrome Patients From Marathwada Region of Maharashtra State. *Journal of Medical Education & Research*, 2012; Vol. 2, No.2, 12-15. July-Dec 2012
  20. Tsimikas S, Brilakis ES, Miller ER, McConnell JP, Lennon RJ, Kornman S, Witztum JL, and Berger PB: Oxidized phospholipids, Lp(a) lipoprotein, and coronary artery disease. *N Engl J Med*, 2005; 353:46-57. [[PubMed](#)]
  21. Sandhu K, Singh A, Nadar S: Management of elderly patients with troponin positive chest pain in a District General Hospital. *Cardiol J* 2012; 19, 4: 395–401. [[PubMed](#)]
  22. Lahoz C, Mostaza J, Tranche S Martin-Jadraque R21-Tsimikas S, Brilakis ES, Miller ER, McConnell JP, Lennon RJ, Kornman, Mantilla M, Lopez-Rodriguez I, Monteiro B, Sanchez-Zamorano M, Taboada M: Atherogenic dyslipidemia in patients with established coronary artery disease. *Nutrition, Metabolism & Cardiovascular Diseases* 2012; 22, 103e108
  23. Cooke C, Hammerash W: Retrospective review of sex differences in the management of dyslipidemia in coronary heart disease: an analysis of patient data from a maryland-based health maintenance organization. *Excerpta Medica*, 2006; 28: 591-599. [[PubMed](#)]
  24. Saito Y, Kita T, Mabuchi H, Matsuzaki M, Matsuzawa Y, Nakaya N, Oikawa S, Sasaki J, Shimamoto K, Itakura H: Obesity as a risk factor for coronary events in japanese patients with hypercholesterolemia on low-dose simvastatin therapy. *J Atheroscler Thromb*, 2010; 17:270-277. [[PubMed](#)]
  25. Esteghamati A, Abbasi M, Nakhjavani M, Yousefzadeh A, Basa A, Afshar H: Prevalence of diabetes and other cardiovascular risk factors in an Iranian population with acute coronary syndrome. *Cardiovascular Diabetology* 2006; 5:15. [[PubMed](#)]
  26. Guler E, Gecmen C, Guler G, Karaca1 O, Agus H, Gunes H, Batgerel U, Elveran A, Esen A: Adding lipoprotein(a) levels to the GRACE score to predict prognosis in patients with non-ST elevation acute coronary syndrome. *Kardiologia Polska* 2013; 71, 7: 695–701;

27. Kamariya CP, Gorasia JH, Vachhani U, Gohel M: Evaluation of serum lipoprotein (a) in young patients with myocardial infarction. *International Journal of Medicine and Public Health*, 2014; Vol (4 ) Issue 1.107-109.
28. Leebmann J, Roeseler E, Julius U, Heigl F, Spitthoever R, Heutling D, Breitenberger P, Maerz W, Lehmacher W, Heibges A, Klingel R: Lipoprotein apheresis in patients with maximally tolerated lipid-lowering therapy, lipoprotein(a)-hyperlipoproteinemia, and progressive cardiovascular disease: prospective observational multicenter study. *Circulation*. 2013; 128(24):2567-76.
29. El-Menyar A, Ahmed E, Albinali H, Al-Thani H, Gehani A, Singh R, Al Suwaidi J: Mortality trends in women and men presenting with acute coronary syndrome: insights from a 20-year registry. *PLOS ONE* 2013; 8(7): e70066. [[PubMed](#)]
30. Youssef A, Dimitry S, Saweris M: Clinical presentation of acute coronary syndrome in women and its difference from that in men. *Heart Mirror J* 2012; 6(2): 111-114
31. Nouredine S, Arevian M, Adra M, Puzantian H: Response to signs and symptoms of acute coronary syndrome: differences between Lebanese men and women. *Am J Crit Care* 2008; 17:26-35. [[PubMed](#)]
32. Seidell JC, Cigolini M, Charzewska J, et al.: Fat distribution and gender differences in serum lipids in men and women from four European communities. *Atherosclerosis* 1991; 87:203-210. [[PubMed](#)]
33. Lima L, Carvalho M, Loures-Vale A, Fernandes A, Mota A, Neto C, Garcia J, Saad J, Souza M: Increased serum levels of lipoprotein(a) correlated with the severity of coronary artery disease in patients submitted to angiography. *Arquivos Brasileiros de Cardiologia*, 2005; 87.
34. Jatav O P, Agrawal N, Raghuvanshi A, Tiwari D: Serum lipoprotein(a) an independent risk factor of acute coronary syndrome. *International Journal of Clinical Cases and Investigations*. 2010; 1, 20-26
35. Swierszcz J, Dubiel JS, Milewicz T, et al (): Smoking, increase in plasma lipoprotein (a) and triglycerides, as well as decrease in plasma HDL-cholesterol concentrations seem to be linked with aortic valve stenosis and its progression. *Przegl Lek*; 2009; 66(4):159-65. Polish [[PubMed](#)]



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