LIPOPROTEIN (A) IN PATIENTS WITH PREMATURE MYOCARDIAL INFARCTION

Toba Kazemi[(1)], Gholamreza Sharifzadeh[(2)], Asghar Zarban[(3)], Azita Fesharakinia[(4)]

Abstract

BACKGROUND: Increased levels of lipoprotein (Lp)(a) is a risk factor for coronary heart disease. In this study we evaluated levels of Lp(a) in patient with acute myocardial infarction (AMI) aged less than 50 years old in comparison to controls.

METHODS: In this case-control study, we compared 98 patients with AMI (case group) and an equal number of age- and sex-matched healthy subjects (control group). Serum Lp(a) level was measured after 12 hours of fasting in both groups.

RESULTS: The mean age of the case and control groups was not significantly different (45.28 ± 5.09 vs. 44.89 ± 5.22 years, respectively P = 0.52).The mean Lp(a) level was significantly higher in the case group than in the control group (32.5 ± 24.5 vs.25.2 ± 22.6 mg/dl, respectively P = 0.04). Prevalence of LP(a) ≥ 30 mg/l was significantly higher in case group than in control group (43.5% vs. 27. %, respectively, P = 0.016, OR = 3.22, 95% CI = 1.24-8.3).

CONCLUSION: Because of high prevalence of high LP(a) in premature AMI, it is necessary to control this disorder in young adults for preventing delayed MI.

Keywords: Highlipoprotein (LP)(a), Premature myocardial infarction, Case- control study

***ARYA Atherosclerosis Journal* 2009, 4(4): 149-151**

Date of submission: 10 August 2008, ***Date of acceptance:*** 15 December 2008

Introduction

Cardiovascular diseases (CVD) are the main causes of death in human. It is estimated CVD would become the main cause of death in 36% of the population up to year 2020.[1] in Iran, CVD is the most common cause of death.[2] 4%-10% of patients with acute myocardial infarction (AMI) are below 45 years of age.[3] Risk factors of AMI differ between young and old patients.[4]

Several epidemiologic and cross-sectional studies, revealed an association between lipoprotein (Lp)(a) CVD.[5,6]

Lp(a) excess, detected in patients with premature CVD. For example, Lp(a) excess was present in 18.6 % of patients with premature coronary heart disease (CHD), 12.7 % of whom had no other dyslipidemia.[7]

In Iran there are little studies about Lp(a) and CVD.[8,9] Because of variability of Lp(a) level between racial groups[10] ,we investigated the level of Lp(a) in an Iranian population under 50 years old.

Materials and Methods

This was a population -based case-control study which carried out during 2005-2007 in Birjand, Eastern of Iran. Cases were 98 young adult patients (< 50 years old) who were admitted to the coronary care units of the Valliassr Hospital with acute myocardial infarction .Since this ward, is the only cardiac ward in Birjand city, our subjects, could be regarded as the representative of all Birjand population. Diagnosis of myocardial infarction (MI) was based on the presence of at least two of the following characteristics:

1-Typical chest pain lasting at least 30 minutes

2-An ECG showing ST elevation of at least 1 mm in two or more contiguous leads, with subsequent evolution of the changes

3-Diagnostic enzyme changes: doubling of creatine kinase with at least 10% MB fraction.[11]

1) Associate Professor of Cardiology, Birjand Cardiovascular Research Center, Birjand, Iran. E-mail: med_847@yahoo.com
2) PhD. Epidemiologist, Birjand Cardiovascular Research center, Birjand, Iran.
3) Associate Professor of Biochemistry, Birjand Cardiovascular Research Center, Birjand, Iran.
4) Associate Professor of Pediatric, Birjand Cardiovascular Research Center, Birjand, Iran.
Corresponding author: *Toba Kazemi*

www.mui.ac.ir

98 healthy subjects (control group) were selected from the neighborhood of each case, after matching age and sex. Informed consent was signed by all members of the two groups.

Ethical approval was obtained from the Ethics Review Committee in Birjand University of Medical Sciences. After being 12 hours fast, a sample of 5cc blood was taken from right brachial vein, in all of the participants and sent to central lab of Birjand University of Medical Sciences. Lp (a) were measured using turbidometry method (Pars Azmon kit, Iran).

Then data were collected and analyzed with χ^2, t-test. SPSS 11.5 software was used for all analyses.

Results

The mean age of cases was 45.25 ± 5.09 years and the mean age of controls was 44.8 ± 5.22 years (P = 0.52). 75 persons (80.9%) in each group were men.

The mean of Lp(a) was significantly higher in cases than controls (Table 1). Prevalence of high Lp(a) (≥ 30 mg/l) was significantly higher in cases (Table 2).

Table 1: Comparison of mean Lp (a) between cases and controls.

	Number	Mean(mg/dl)	Standard Deviation
Cases	98	32.5	24.5
Controls	98	25.2	22.6
P value	0.04*		

* Statistically different (P < 0.05)

Table 2: Comparison of prevalence of high Lp(a) between cases and controls

Lp(a) level	case n = 98	control n = 98	OR CI 95%	P value
<10mg/dl	10(10.6%)	21(21/2%)	1	
10-20mg/dl	25(25.9%)	34(34.2%)	1.52 (0.57-4)	0.4
20-30mg/dl	20(20%)	17(17.6%)	2.27 (0.79-6.5)	0.13
>30mg/dl	43(43.5%)	26(27 %)	3.22 (1.24-8.3)	0.016*

* Statistically different (P < 0.05)

Discussion

In this population-based case-control study, we investigated Lp(a) concentrations in a sample of Iranian subjects with premature AMI.

Lp(a) is consisted of two apolipoproteins. A large glycoprotein, apolipoprotein(a) [apo(a)] is covalently bound to apo B by a disulfide bridge. Lp(a) has a structural similarity with plasminogen[12] and interfere with plasma fibrinolysis by inhibiting the generation of plasmin.[12]

Lp(a) is a modified form of LDL .Because of similariy of Lp(a) with LDL, It has been suggested that Lp(a) may display atherogenic capacity by bounding with macrophages.[13,14]

Several epidemiological studies suggested that Lp(a) is an independent risk factor for CVD and premature AMI.[5,6]

We found in our study that the mean of Lp(a) as significantly higher in infracted patients. Prevalence of high Lp(a)(≥ 30 mg/l) was 43.5% in case group and 27% in control group,in this study. Risk of developing of AMI is 3.22 times more in those with high Lp(a) compared with healthy persons.

Isser et al. assessed Lp(a) levels in young patients with myocardial infarction, their first-degree relatives, and age- and sex-matched controls The mean Lp(a) level was 22.28 mg/dl in patients, 13.88mg/dl in their first degree relatives and 9.28 mg/dl in controls. Very high levels of Lp(a) (> 30 mg/dl) were found in 10% of young patients with MI, 3.2% of their first-degree relatives and none of the controls.[15]

In another case-control study (111 infarcted men and 99 men free from disease), Calmarza et al reported that infarcted patients had significantly higher Lp(a) concentrations than noninfarcted subjects (P = 0.001). Infarcted patients had also greater proportion of Lp(a) > 30 mg/dl (37.8% in case, 20.2% in control P = 0.01).

Fazlinezhad et al in Iran (compared Lp(a) in 43 patients with AMI with 43 healthy subjects.Mean Lp(a) level in patients with AMI was 49.18 mg/dl and in controls was 37.95 mg/dl (P = 0.018).[9]

www.mui.ac.ir

Conclusion

Our results indicateed that Lp (a) is a risk factor for premature AMI. In a primary prevention setting, it may be useful to measure Lp (a) levels especially in the population with high risk for an early onset AMI.

Acknowledgements

This study was supported by grants from the Birjand University Medical sciences.

References

1. WHO. World report on violence and health. Geneva: World Health Organization; 2002.
2. Naghavi M. Transition in health status in Islamic Republic of Iran. Iranian Journal of Epidemiology 2006; 1(3): 13-25.
3. Chan MY, Woo KS, Wong HB, Chia BL, Sutandar A, Tan HC. Antecedent risk factors and their control in young patients with a first myocardial infarction. Singapore Med J 2006; 47(1): 27-30.
4. Chen L, Chester M, Kaski JC. Clinical factors and angiographic features associated with premature coronary artery disease. Chest 1995; 108(2): 364-9.
5. Bennet A, Di Angelantonio E, Erqou S, Eiriksdottir G, Sigurdsson G, Woodward M, et al. Lipoprotein(a) levels and risk of future coronary heart disease: large-scale prospective data. Arch Intern Med 2008; 168(6): 598-608.
6. Danesh J, Collins R, Peto R. Lipoprotein(a) and coronary heart disease. Meta-analysis of prospective studies. Circulation 2000; 102(10): 1082-5.
7. Genest JJ, Jr., Martin-Munley SS, McNamara JR, Ordovas JM, Jenner J, Myers RH, et al. Familial lipoprotein disorders in patients with premature coronary artery disease. Circulation 1992; 85(6): 2025-33.
8. Yousefi AA, Givtaj N, Zavareh A, Sabouri-zadeh N, Chizaree MR. Lipoprotein(a) (LPA), Fibrinogen, and Homocysteine in Patients with Coronary Artery Disease and without Major Risk Factors. Iranian Heart Journal 2006; 7(1): 37-9.
9. Fazlinezhad A, Shakeri MT, Taghavi S, Ziallhagh R, Abbaszadegan MR, Ghafarrzadegan K. Lipoprotein(a) level in acute myocardial infarction: comparison with healthy subjects. Shiraz E-Medical Journal 2005; 6(1-2).
10. Sandholzer C, Saha N, Kark JD, Rees A, Jaross W, Dieplinger H, et al. Apo(a) isoforms predict risk for coronary heart disease. A study in six populations. Arterioscler Thromb 1992; 12(10): 1214-26.
11. Libby P, Bonow Ro, Mann DL, Zipes DP, FACC. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 8th ed. Philadelphia: W.B. Saunders; 2007.
12. Loscalzo J, Weinfeld M, Fless GM, Scanu AM. Lipoprotein(a), fibrin binding, and plasminogen activation. Arteriosclerosis 1990; 10(2): 240-5.
13. Tsimikas S, Brilakis ES, Miller ER, McConnell JP, Lennon RJ, Kornman KS, et al. Oxidized phospholipids, Lp(a) lipoprotein, and coronary artery disease. N Engl J Med 2005; 353(1): 46-57.
14. Poon M, Zhang X, Dunsky KG, Taubman MB, Harpel PC. Apolipoprotein(a) induces monocyte chemotactic activity in human vascular endothelial cells. Circulation 1997; 96(8):2514-9.
15. Isser HS, Puri VK, Narain VS, Saran RK, Dwivedi SK, Singh S. Lipoprotein(a) and lipid levels in young patients with myocardial infarction and their first-degree relatives. Indian Heart J 2001; 53(4): 463-6.
16. Calmarza P, Cordero J, Santos V, Vella JC. Apolipoprotein(a) isoforms in infarcted men under 60 years old. Clin Biochem 2004; 37(10): 911-8.

www.mui.ac.ir