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Lipoprotein (a) level in acute myocardial infarction: comparison with healthy subjects.

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Abstract:

Increased lipoprotein (a) [LP (a)] concentration was reported to be an independent risk factor for coronary heart disease (CHD).

This study was performed to determine the level of Lp(a) and other lipids in patients with acute myocardial infarction and comparing them with matched healthy subjects. We studied a total of 43 cases who admitted to coronary care unit (CCU) for acute myocardial infarction (MI) and also 43 healthy subjects. Data collection comprised Lp (a), total cholesterol, LDL-cholestrol (LDL-C), HDL-cholestrol (HDL-C) level and medical history. The mean Lp(a) level in acute MI cases was 49.18mg/dl, and in the control group was 37.95mg/dl. There was significant difference between two groups (p=0.018).

The mean total cholesterol, LDL-C and HDL_C, level in acute MI cases were 183.9, 106.5 and 58.12 mg/dl respectively and in control cases were 227,145.8 and 51mg/dl respectively. The mean level of Lp(a) in women (case& control) was 50mg/dl that was higher than men (37.1mg/dl). Also Lp(a) was independent of other lipids. As one of the first studies about Lp(a) in Acute MI patients, this study shows that average Lp(a) level in patients with acute MI is higher than control patients. Another interesting finding is that level in women is higher than men. We recommend complementary epidemiologic study to evaluate this finding.

Key Words: lipoprotein (a), acute myocardial infarction,cholesterol.

Introduction:

Lipoprotein (a) was first described by Berg in 1963(1). Its biological role has attracted the attention of many investigators since 1987 when Mclean et al determined the amino acid sequence of apo (a), and suggested a structural homology between apo(a) and plasminogen(2). Lipoprotein (a) is a cholesterol–rich lipoprotein particle composed of an LDL particle and a large glycoprotein, apolipoprotein (a) [apo (a)].

Apo (a) is highly variable in size because of length polymorphism in the apo (a) gene. It

was recognized that high concentrations of Lp(a) in plasma are associated with coronary heart disease and early myocardial infarction (3, 4). This observation was confirmed by numerous studies in different ethnic groups, Using different endpoints for definition of coronary artery disease, and different methods#In the name of God to measure Lp(a) in plasma(5). It has been suggested that it is a coronary risk factor independent of increase in other serum lipids (eg.cholesterol and triglycerides), hypertension, smoking obesity and a family history of IHD (6).

Aim of our study is to determine the level of Lp(a) in patients with acute myocardial infarction and compare it with matched healthy control group.

Materials and Methods:

Patients: Forty – three patients with acute myocardial infarction were selected from a series of consecutive patients admitting the coronary care unit (CCU) of Ghaem hospital, Mashad, Who had the complete data including family history, laboratory findings and clinical data. All patients and control subjects were older than 40 years old. The healthy controls were selected from subjects who underwent routine laboratory examination for checkup.

Inclusion criteria in this group were: absence of a history of smoking, cardiovascular disease and diabetes and the age over 40 years old. Forty three subjects with these criteria were chosen.

Blood sampling and assay: Fasting venous blood sample from all patients (the day after admission to CCU) and control subjects were collected. Blood was centrifuged for 10 minutes and the serum stored at -20°c until analyzed.

Lp(a) was quantified by immunoturbidiometric method (Pars azmoon Co). Total cholesterol, HDL – cholesterol and triglyceride were determined by enzymatic methods (Pars azmoon Co). Friedewald formula was used to calculate the LDL – cholesterol level. Samples with severe hemolysis or TG more than 2000 mg/dl, were excluded. The Lp(a) samples of patients and controls were unknown for technician who measured them.

Statistical Analysis: All biochemical and clinical data were recorded prospectively. We compared the Lp(a) level of acute MI patients with those of age and sex–matched controls. Based on manufactures instructions, Lp(a) > 35 mg / dl is the threshold value linked to its pathologic effects. We defined subjects with > 35 mg / dl as those with high Lp(a) and examined its frequency in acute myocardial infarction .Continuous variables were reported as mean ±1 standard deviation. Differences in mean group variables were analyzed by Mann – whitney test for non-parametric data. A value of $P < 0.05$ was considered significant.

Results:

Summary of patients and control subjects data have been presented in table 1. The mean serum Lp(a) concentration in control group was 37.94 ± 49 with maximum level of 328 mg/dl. In the case group, the mean serum Lp(a) level was 49.18±47.44 with maximum level of 280 mg/dl. There is a significant difference between two groups ($P = 0.018$)

Based on more than 35 mg/dl threshold, 21/43(48%) of cases and 14/43 (32%) of control subjects had hig Lp(a) .

The mean of Lp(a) level in women (case & Control) was 50 mg/dl that is higher than men (37.1 mg/dl).

The female controls had mean serum Lp(a) level equal to 42.65 (10.8 – 328) mg/dl and the female patients had mean serum Lp(a) level equal to 61.31 18.7 – 280) mg/dl. There is also a significant difference between two groups ($P = 0.033$).

The male controls had mean serum Lp(a) concentration equal to 30.75 (8.6 – 108) mg/dl. The male patients had mean serum Lp(a) concentration equal to 41.25 (14.5 – 131) mg/dl. There is no significant

difference between two groups (P = 0.196). Also, the Lp(a) level was independent of the level of other lipids. There is no correlation between Lp(a) and Hypertension, hyperlipidemia, diabetes and smoking. The mean cholesterol, Triglyceride, LDL – C, HDL – C level in case and control group are presented in table 2.

Table 1: Demographic data of patients and controls

	Case	Control
No	43	43
Age(mean)	56	61
F/M	17/26	26/17
Hypertensin	19	------
NIDDM	13	-------
IDDM	0	-------
Smoking	17	10

Table2: summary of lipid values in patients and controls

	Case	Control
Cholestreal total *	183±47	227±40
LDL –C *	106±46	145±35
HDL –C *	58±68	51±8
Triglyceride*	130±82	149±77
Lp(a)	49±47	37±49

Discussion:

In this study, serum Lp(a) concentration were compared in acute MI patient and healthy control subjects. We showed that in average, Lp(a) level in patients with acute MI is higher than control persons. Another findings are that the level in women is higher than men and the Lp(a) level is independent from the level of other lipids .

An interesting point of our study is the selection of patients who present with Acute MI. As far as we know, there is few studies that have been done in patients with acute MI.

The total cholesterol and LDL level in patients were lower than control group, which is due to post MI period. But, Lp(a) level even in this period in these patients were higher than controls . It means that the Lp(a) level in patients could be higher , if acute MI has similar effect on Lp(a) level. It needs to be confirmed by another research.

David .J. Moliterno et al showed that Elevated plasma concentration of Lp(a) are associated with coronery atherosclerosis in Caucasians. They also showed that although African - Americans have a higher median plasma Lp(a) concentration than Caucasians, they do not have a greater incidence of coronary atherosclerosis (7).

In a study by Abraham A.Ariyo, It was shown that among older adults in the United states, an elevated Lp(a) lipoprotein is an independent predictor of stroke, death from vascular disease, and death from any cause in men but not in women. These data support the use of Lp(a) Lipoprotein levels in predicting the risk of these events in older men (8).

In Akihisa's study it was concluded that Lp(a) is an independent potential and modifiable coronary risk factor, and that reduction of serum Lp(a) is important in the clinical management of patients with IHD (9) .

Laron Z. et al in their investigation determined the effect of human growth hormone (hGH) and Insulin like growth factor -1(IGF-1) on circulating Lp (a): long term GH treatment increase and IGF-1 decrease circulating levels of Lp(a) (10) .

It seems that Lp(a) is specifically an independent risk factor in diabetics (11-12) .

Nogues X .et al suggested a discriminant cut off of Lp(a) concentration equal to 20 mg/dl or 30 mg/dl in enzyme immunoassay (13). In the future there may be therapeutic methods to reduce Lp(a) levels which may be proven to be useful in preventing myocardial infarction .

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