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ELEVATED LIPOPROTEIN (a) LEVELS SEEN IN PATIENTS WITH CORONARY ARTERY DISEASE

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ABSRACT
There are a number of risk factors involved in the etiology of coronary artery disease. Lipoprotein (a) [(Lp(a)] is a genetically determined variant of low density lipoprotein (LDL) cholesterol which is highly atherogenic. It has been found to be a common risk factor in families suffering from premature coronary artery disease (CAD). Our study was an analytical cross sectional study, in which Lp(a) levels were measured by ELISA assay in 60 cases of coronary artery disease (CAD), (both sexes, aged 40-60 yrs) and 60 healthy controls. There was a significant difference in serum Lp(a) level between the two with higher levels seen in the CAD cases. It is concluded that it is needed to measure Lp(a) levels as well as the routine lipid profile in CAD patients.

Key Words: *Coronary Artery Disease (CAD), Lipoprotein (a) [Lp (a)].*

INTRODUCTION

In 1963, Berg introduced a new class of lipoproteins named lipoprotein(a) abbreviated as Lp(a). This new lipoprotein appeared to be associated with the occurrence of coronary heart disease in men.[1] Lp(a) can be considered as a genetically determined variant of low density lipoprotein (LDL), which is higher in density and size.[2]

The danger of (LPA) lies in fact that it is 10 times more atherogenic than LDL. Moreover stable lifelong level of Lp(a) is attained in infancy; therefore pathological processes associated with elevated Lp(a) level also begin in infancy, about 20 years earlier than other risk factors for CAD like hypertension, cigarette smoking and other dyslipidaemias.[3]

The gene coding for Lp(a) resides on chromosome 6. It is inherited in a mendelian dominant fashion which means that approximately 50% of offsprings of parents with raised Lp(a) will also have elevated Lp(a).[4] There are more than 25 heritable forms of Lp(a), resulting in a wide variation of plasma levels of this lipoprotein across different populations.[5] Structurally low density lipoprotein (LDL) particles have 1 mole of apolipoproteins B-100 (apo B-100) per particle. In Lp(a) apoB-100 is linked by a disulfide bond to apo(a). Compared to apo B-100, which is relatively constant in weight, the weight of apo (a) moiety varies between 300 and 800 kDa.[6]

There is a close structural homology between Lp(a) and plasminogen. This has raised the possibility that this lipoprotein competes with plasminogen and prevents it's binding to the vascular endothelium thus inhibiting fibrinolysis.[5]

The mean values given for Lp(a) in a Framingham cohort are 14 mg/dL for men and 15 mg/dL for women. Levels above 30 mg/dL are generally considered elevated.[7] Lp(a) levels are affected by a wide variety of factors including age, sex and ethnic background. Older age and female sex are associated with higher Lp(a) levels. Ethnicity has a significant association with Lp(a) level.[8] In Asian Indians, raised Lp(a) levels are a powerful risk factor for premature coronary artery disease, which occurs 3-10 times more in young Asians less than 40 years of age as compared to other populations.[9]

MATERIALS AND METHODS

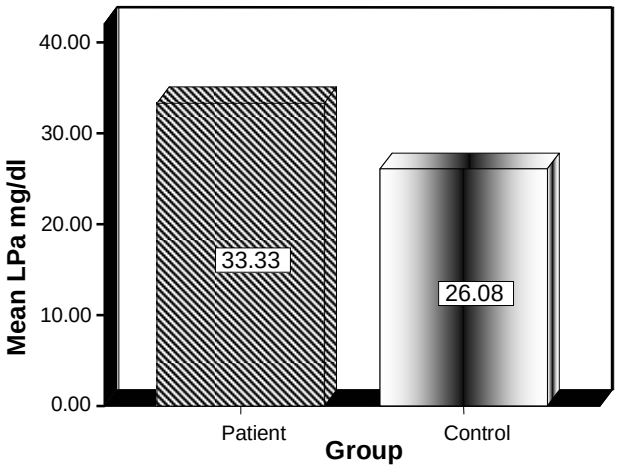
The study was conducted in the University of Health Sciences Lahore. The study population consisted of 60 patients of angiographically proven CAD admitted in PIC and 60 healthy controls without history of angina or myocardial infarction. Subjects suffering from diabetes mellitus, severe liver and renal disease were excluded. Fasting venous blood samples were collected. Lp(a) in the samples was measured using the Hyphen Bio-med 'elitest' Lp(a) CK 103A assay based on sandwich ELISA technique. The data was analysed using SPSS 16 (Statistical Package For Social Sciences).

RESULTS

The serum Lp(a) ranged from 11.24 – 75.32 mg/dl in the cases and from 4.07- 57.66 mg/dl in the controls. The mean serum Lp(a) level was 33.32 mg/dl in the cases and 26.07 mg/dl in the controls. There was significant difference between the two groups (33.32 ± 12.58 vs 26.07 ± 13.39) (p value = 0.03).

DISCUSSION

The mean Lp(a) level was 33.32 ± 12.58 mg/dl in the patients and 26.07 ± 13.39 mg/dl in the controls. There was significant difference between the two groups (p-value 0.03). This is consistent with a number of previous studies. In the various previous studies, mainly in white populations, elevations of plasma Lp(a) levels (usually defined as > 30 mg/dl) were significantly correlated with CAD.[2,10-13] Lp(a) levels were 28.4 versus 16.5 mg/dl in white men with and without CAD respectively in a study by Paultre et al in.[14] The comparatively higher mean value of Lp(a) in our study is in accordance with various studies done on South Asians, which showed a genetic disposition towards higher Lp(a) levels than their western counterparts.[15-18] In a recent study conducted in 2007, elevated Lp(a) levels were related to more severe CAD thus it was concluded that when assessing cardiovascular risk in a CAD patient Lp(a) levels should be considered.[19]



Mean Serum Lp(a) Level in Cases and Controls

It is therefore **concluded** that elevated Lp(a) level is associated with CAD in both males and females. The present study conforms to the previous observation of higher Lp(a) levels compared to the western standards. Lp(a) should be considered in the laboratory work-up of CAD patients.

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