Lipoprotein A and its Relationship with Other Lipids in Haemodialysis Patients

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Introduction

The prevalence of hyperlipidemia is increased in patients with chronic renal disease (CRD). The type and the severity of lipids abnormalities vary among different CRD patient populations[1]. Lipoprotein (a) (Lp(a)) is a modified form of LDL-cholesterol[2]. Increased serum concentrations of LP(a) have been shown to be a strong , independent risk factor for the development of atherosclerosis in the general population[3] and in haemodialysis patients[4]. Serum Lp(a) levels are predominantly genetically determined, strikingly resistant to environmental influence and they are not associated with other serum lipids, but LDL-cholesterol, in general population[2]. The aim of the present study is to evaluate the serum (Lp(a)) concentration in haemodialysis patients and to examine its relationship with other serum lipid parameters

Patients and methods

We measured Lp(a), total cholesterol, HDL-cholesterol, LDL-cholesterol and trigrycerides in 94 haemodialysis patients (56 males and 38 females, mean age 65±15; range 37-83 years, 19 diabetic), and in 58 healthy controls (36 males and 22 females, mean age 63±13; range 35-81 years, 14 diabetic). Lp(a) concentration was measured using an ELISA assay, while the serum concentrations levels of the other lipids were obtained from the monthly follow-up of these patients for the last 12 months and the mean values were used for statistical analysis. LDL-cholesterol was calculated using the Friedewald formula. A student's two tailed unpaired t test was used to compare values between patients and controls, and a multiple linear regression analysis was applied to determine the correlation of serum Lp(a) with the rest measured serum lipids in the HD patients based on the Wald's statistic and using 5% of the probability for enters and 10% of the probability of removal a variable from the model.. P value of <0.05 was considered statistically as significant.

Results

Mean serum Lp(a) was 65.77±58.79; range 5.60-235 mg/dl in haemodialysis patients, and 23.68±16.82; range 7-92 mg/dl in controls; p<0.005 (Fig. 1). Patients' serum lipids were as follows: Total cholesterol 239.15±51.10, LDL-cholesterol 151.97±40.03, HDL-cholesterol 43.90±9.81 and triglycerides 217.81±10 mg/dl. There was a significant positive correlation of serum Lp(a) with total cholesterol; p<0.01, with LDL-cholesterol; 0.001, with triglycerides; p<0.01 and an inverse correlation with HDL-cholesterol; p<0.05 (Table 1).

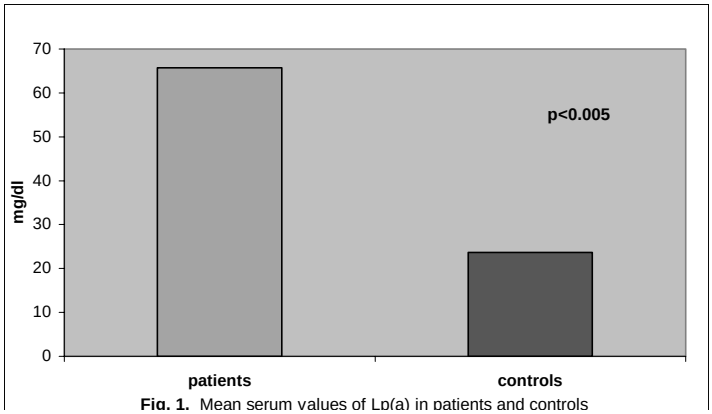

Fig. 1. Mean serum values of Lp(a) in patients and controls

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Table 1. Results of the multiple linear regression analysis between serum Lp(a) and the other lipids

	Serum lipids	P-value
Lp(a)	t. Cholesterol	$p<0.01$
Lp(a)	LDL-Cholesterol	$p<0.001$
Lp(a)	HDL-Cholesterol	$p<0.05$[1]
Lp(a)	Triglycerides	$p<0.01$

[1], inverse correlation

Discussion and Conclusion

The haemodialysis patients we studied had significantly higher levels of serum Lp(a) compared to the healthy controls. This is in accordance with several reports of increased serum concentrations of Lp(a) in chronic renal failure[5]: in predialysis patients, in haemodialysis patients and in CAPD patients as well as in the nephrotic syndrome. Possibly this increase is caused by posttranscriptional change in the phenotypic expression. Furthermore we found significant correlations of Lp(a) serum levels with other serum lipids in the haemodialysis patients. The direct correlation between serum concentrations of Lp(a) and LDL-cholesterol probably reflects the common origin of these lipoproteins. Unexpectedly, we found significant correlations between Lp(a) and total cholesterol and triglycerides, as well as a negative correlation with HDL-cholesterol. Published data regarding to the association of lipoproteins among renal patients are limited. Arnadottir et al. reported significant correlations between Lp(a) and total cholesterol, apolipoprotein B and LDL-cholesterol but no correlation between serum Lp(a) and triglycerides and HDL-cholesterol among patients with moderate renal failure[6].

The present findings suggest that in renal failure there are unknown to the present mechanisms, which superimpose the genetic influence and modulate serum Lp(a) concentration in conjunction with the other lipid parameters.

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