APOLIPOPROTEINS AND LIPOPROTEIN (a) IN PATIENTS WITH PREMATURE MYOCARDIAL INFARCTION AND THEIR CHILDREN

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Abstract

INTRODUCTION: A positive family history of premature coronary artery disease can prompt the assessment of children of the family for coronary risk factors like hypertension, obesity and dyslipidemia. This study was performed to determine the relationship between lipoprotein (a), apolipoprotein A1 and B100 in patients with a positive history of premature myocardial infarction and their offspring.

METHODS: This cross-sectional study was conducted on 91 parents and their offspring (91 children). The parents were randomly selected from among patients with premature myocardial infarction hospitalized in the critical care unit of Vali-e-Asr hospital (Birjand, Iran). Lipoprotein (a), apolipoprotein A1 and B100 were measured in both groups.

RESULTS: Our study showed a significant relationship between lipoprotein (a) levels in parents and their children. High lipoprotein (a) and high apolipoprotein B100 in parents and their children were also significantly related.

CONCLUSIONS: We recommend that lipoprotein (a) and apolipoprotein B100 be measured in children with familial history of premature myocardial infarction.

Keywords: Premature myocardial infarction, lipoprotein (a), risk factors, offspring, apolipoproteins.

***ARYA Atherosclerosis Journal* 2007, 3(1): 26-28**

Date of submission: 30 Oct 2006, ***Date of acceptance:*** 16 Apr 2007

Introduction

Cardiovascular disease aggregates in families. This is probably due in part to familial aggregation of important cardiovascular risk factors.[1,2] Hence, early screening and control of risk factors in offspring of high-risk families may help in efforts to prevent cardiovascular disease.[3] This is of particular importance for individuals with family history of premature coronary artery disease (CAD).[4] The importance of cardiovascular risk factors like hypertension, obesity and dyslipidemia in predicting CAD is well known.[5-7]

New risk factors, such as fibrinogen, apolipoproteins, homocysteine and lipoprotein (a) have been identified and are under further investigation.[6,8,9]

This study was performed in the city of Birjand, Northeastern Iran, to characterize the relationship of CVD risk factors, namely lipoprotein (a), apolipoprotein A1 and B100 in parents with positive history of premature myocardial infarction (MI) and their children.

Materials and methods

The study population consisted of 91 parents, randomly selected from among patients who had suffered premature MI (<55 years) and were hospitalized in the coronary care units (CCU) of the Vali-e-Asr Hospital affiliated to Birjand University of Medical Sciences. Only one child (age 2-14 years) was randomly selected from every family.

Subjects had been instructed to fast for 12-14 hours. Antecubital venous blood was collected. Biochemical tests including measurement of lipoprotein (a) and apolipoprotein were carried out. An Elan autoanalyzer was used to measure lipoprotein (a).

Apolipoproteins (Apo B100, Apo A1) were measured by spectrophotometry using the turbidimetry method.

Statistical analysis was performed with SPSS statistical package using independent t-test and partial Pearson correlation coefficients. P values less than 0.05 were considered as significant.

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TABLE 1. Mean level of Lp (a), Apo A1 and Apo B100 in parents and their children and the relationship between them

Variable	Children (n= 91) Mean ± SD	Parents (n= 91) Mean ± SD	Partial correlation ®	Pearson coefficients (P)
Apo A1	105.3 ± 17.9	101.3 ± 16.9	0.07	0.57
Apo B100	73.5 ± 22	88.1 ± 20.2	0.1	0.36
Lp (a)	32 ± 24.6	30.9 ± 29.4	0.36	0.001 *

Results

Ninety-one parents (85 fathers, 6 mothers) and 91 offspring (45 girls, 46 boys) were studied. Parents and children had mean ages of 44.4±4.5 and 11.2±2.6 years, respectively.

Our study showed a significant relationship between lipoprotein (a) levels in parents and their children (Table 1). Lp (a) (values >0.3 g/l) and high Apo B100 (values >1 g/l) (10) were significantly related in parents and their children (P= 0.003 for both).

Discussion

Familial aggregation of cardiovascular risk factors including hypertension, obesity and dyslipidemia has been extensively investigated.[1,2]

It has been demonstrated that both genetic and environmental factors contribute to the variability of risk factors and their familial aggregation.[10,11]

The aim of this study was to characterize the relationship between Lp (a), Apo A1 and Apo B100 in parents with history of premature MI and their children. Lp (a) levels, as well as high Lp (a) levels were significantly related in patients and their children.

In several studies Lp (a) level was measured in school children and the parents were questioned about past history of heart attack. There was a significant relationship between high Lp (a) in students and family history of premature CVD.[7,12]

Different studies have showed significantly higher levels of Lp (a) in offspring of parents with past history of premature MI compared to controls.[13-15]

High Apo B100 levels in patients and their children were significantly related. Two earlier studies have demonstrated a significant relationship between high Apo B100 level in school children and family history of premature CAD.[7,12]

Sniderman et al.[16] reported higher Apo B100 levels in children of patients with premature MI, a finding supported by several other studies.[6,15,16]

Epidemiologic studies have suggested that multiple risk factors increase the probability of cardiovascular events, since CVD risk factors tend to reinforce each other and the influence they have on morbidity and mortality. A family history of premature MI is a risk factor for CVD and detecting other risk factors such as hypertension, obesity and dyslipidemia is very important.[5-7]

This study showed a significant relationship between high Lp (a) and Apo lipoprotein B100 in parents with a positive history of premature MI and their offspring; we therefore recommend measurement of Lp (a) and Apo B100 as new cardiovascular risk factors in these children.

Acknowledgment

We thanks the Research Center of Birjand University of Medical Sciences for supporting this study.

References

1. Colditz GA, Stampfer MJ, Willett WC, et al. A prospective study of parental history of myocardial infraction and coronary heart disease in women, Am J Epidemiol. 1986;123:48-58.
2. Myers RH, Kiely DK, Cupples LA, Kannel WB. Parental history is independent risk factors for coronary artery disease (the Framingham study), Am Heart J. 1990;120:963-969.
3. Szamosi T, Murber A, Szamosi T Jr, et al. Atherosclerosis risk factors in children of high-risk families. Acta Physiol Hung. 1999;86(93-4):185-90.
4. De Bacquer D, De Backer G, Kornitzer M, Blackburn H. Parental history of premature coronary heart disease mortality and signs of ischemia on the resting electrocardiogram. J Am Coll Cardiol. 1999;33:1491-1498.
5. Zinner SH, Levy PS, Kass EH. Familial aggregation of blood pressure in childhood. N Engl J Med. 1971;284:401-404.
6. Kelishadi R, Sarrafzadegan N, Nadery Gh. A, et al. Atherosclerosis risk factors in children and adolescents with or without family history of premature coronary artery disease. Med Sci Monit. 2002;8(6):425-429.
7. Glowinska B, Urban M, Koput A. Cardiovascular risk factors in children with obesity, hypertension and diabetes: lipoprotein (a) levels and body mass index correlate with family history of cardiovascular disease. Eur J Pediatr. 2002;161(10):511-518.
8. Beigel Y, George J, Leibovici L, et al. Coronary risk factors in children of parents with premature coronary artery disease. Acta Paediatr. 1993;82(2):162-165.

9. Kishore JH. Potential new cardiovascular risk factors: left ventricular hypertrophy, homocysteine, lipoprotein (a), triglycerides, oxidative stress and fibrinogen. Annals of internal medicine. 1999;131(5):376-386.
10. Sveger T, Flodmark CE, Nordborg K, et al. Hereditary dyslipidemias and combined risk factors in children with a family history of premature coronary artery disease. Arch Dis Child. 2000;82:292-296.
11. Breslow JL. Genetics of lipoprotein disorders. Circulation.1993;87(III):16-21.
12. Wilcken DE, Wang XL, Greenwood J, Lynch J. Lipoprotein (a) and apolipoprotein B and A-1 in children and coronary vascular events in their grand parents. J Pediatr. 1993; 123(4):519-526.
13. Salma R, Abdus sattar Kh, Rahim B. Comparison of lipoprotein (a) and serum cholesterol in offspring of patients diagnosed as myocardial infarction. J Postgrad Med Inst. 2004; 18(3):477-484.
14. Pay S, Ozcan N, Tokgozoglu SL. Elevated Lp(a) is the most frequent familial lipoprotein disorder leading to premature myocardial infarction in a country with low cholesterol levels. Int J Cardiol. 1997 Aug 8;60(3):301-305.
15. Marquez A, Mendoza S, Carrasco H, et al. High lipoprotein (a) in children from kindred with parental premature myocardial infarction. Pediatr Res. 1993;34(5):670-674.
16. Sniderman A, Teng B, Genest J, et al. Familial aggregation and early expression of hyper-apobetalipoproteinemia. Am J Cardiol. 1985;55(4):291-295.