A CASE REPORT OF CORONARY ARTERY DISEASE IN A TEENAGE GIRL

N. Maghami-Pour* and N. Safaie

Department of Cardiac Surgery, Shahid Madani Hospital, School of Medicine, Tabriz University of Medical Sciences, Tabriz, Iran

Abstract- Atherosclerosis is the leading cause of death in most parts of the world. This disorder affects mostly patients above the age 40 years. This case report introduces a 17 years old girl with early development of coronary artery disease who had severe coronary atherosclerosis that did not respond to medical and interventional treatment and underwent surgical operation in cardiac surgery department of Shahid Madani Hospital, Tabriz, Iran. Presence of risk factors for atherosclerosis were evaluated and the only findings were positive family history of cardiac death in her uncle at about 52 years of age and high level of lipoprotein (a) in one of her sisters. In follow up evaluation of this patient, high levels of lipoprotein (a) was documented which was controlled with medical therapy. We concluded that high level of lipoprotein (a) was the probable cause of atherosclerosis in this patient. This case report emphasizes the need to screen siblings of patients with premature myocardial infarction.

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Key words: Atherosclerosis, coronary artery disease, lipoprotein (a)

INTRODUCTION

Atherosclerosis in childhood raises the question of whether the risk factors for adult coronary heart disease (CHD) exist in some forms in children and if they exist, are they associated with the principal arterial lesion of atherosclerosis. Until the middle of the twentieth century, age was considered the major determinant of atherosclerosis and prevention was not emphasized.

This concept has changed. In review of literature, Zeek concluded that atherosclerosis may occur at any age. Study of coronary arteries in autopsy specimens revealed that fatty streaks were present in 8% of the patients aged 10-14 years, but typically clinical disease occur 30 or more years after the process begins as fatty streaks and may be greatly accelerated in persons with cardiovascular risk factors. Major risk factors for atherosclerosis include: hypertension, diabetes mellitus, obesity, gene polymorphism that affect lipid metabolism and smoking. The risk of CHD is higher in peoples with family history of the disease and CHD develops in a younger age in these patients. Serum lipid and lipoprotein are higher in children of parents who had experienced precocious CHD (1, 2). Lipoprotein (a) [Lp(a)] that is a highly polymorphic molecule is structurally similar to plasminogen and prevents fibrinolysis. Lp(a) is a low density lipoprotein (LDL) particle and accumulates in atherosclerotic lesions. Studies have yielded controversial results on the association between Lp(a) level and coronary artery disease. Strong association between cholesterol level in young adult and later risk of cardiovascular disease has been found (3). In a study, the ratio of total cholesterol to HDL-C and high sensitivity (hs)-CRP were the strongest independent predictors of atherosclerotic vascular disease (4).

This case report introduces a 17 years old girl with early development of coronary artery disease who had undergone surgical operation in cardiac surgery department of Shahid Madani Hospital, Tabriz, Iran.

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*** Corresponding Author:**
N. Maghami-Pour, Department of Cardiac Surgery, Shahid Madani Hospital, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran
Tel: +98 411 3309329, Fax: +98 411 3309329
E-mail: dr_maghamipour@yahoo.com

CASE REPORT

The patient was a 17 years old non obese girl with normal general appearance who was referred to cardiologist because of typical exertional chest pain which radiated to her left arm. On primary admission all laboratory data were normal. ETT and myocardial perfusion scan were positive and angiography was done which revealed coronary artery disease (Fig. 1). Attempt to do balloon angioplasty failed, LV gram showed normal ejection fraction and anteroapical hypokinesia.

The patient was discharged with medical follow up. Since patient did not respond to medical treatment, we repeat angiography 10 months later which showed severe diffuse CAD. She was referred for operation.

On surgery with the aid of cardiopulmonary bypass, grafts to all the coronary arteries were done. After operation chest pain improved and patient was followed with medical treatment such as aspirin and lovastatin.

About 20 months after the operation again patient was referred with recurrence of the exertional chest pain. At this time laboratory data were all normal except for high level of Lp(a). ETT and myocardial perfusion scan again showed signs of ischemia so drug therapy for Lp(a) with atorvastatin and gemfibrozil was started. After 3 months, the level of Lp(a) decreased and exertional chest pain improved.

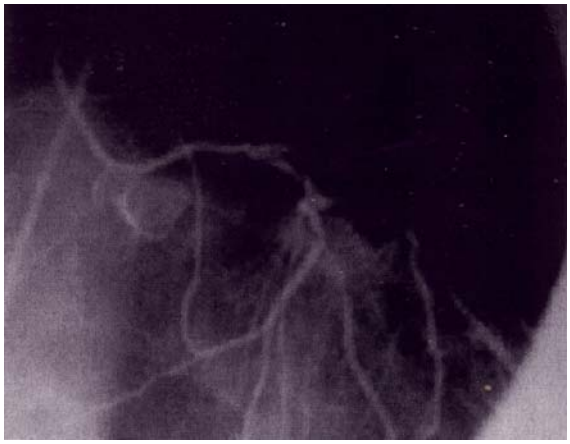

Fig. 1. Coronary angiography of patient, revealing coronary artery disease.

DISCUSSION

Previous studies have shown that atherosclerosis begins at childhood with fatty streaks in vessels and risk factors if present may increase the risk of coronary artery disease but it takes time for the true atherosclerosis to develop (5, 6).

The association between CAD risk factors and atherosclerosis suggest that serum lipoprotein cholesterol concentration, smoking, diabetes, obesity and hypertension are important determinants of atherosclerosis in adolescents and young adults and must be controlled to prevent the progression of the disease.

Lp(a) is a LDL particle and accumulates in atherosclerotic lesions. Lp(a) has a structure similar to plasminogen and prevents fibrinolysis in patients with myocardial infarction. Previous studies suggested that Lp(a) is an independent risk factor and there is a positive correlation between the Lp(a) plasma level with cardiac events (7, 8). Myocardial infarction studies have shown that the level of Lp(a) was high (9). But some other studies have not found this association with Lp(a), and there remain doubt as to whether Lp(a) is a risk factor for CAD.

This case report presents a rare form of atherosclerosis in young age. In our patient no risk factor except Lp(a) was detected. Lp(a) was not measured until recent follow up, and patient did not use any medication for this high level of Lp(a). High level of Lp(a) (103 mg/dl) in our patient caused severe coronary artery disease, but we do not know why this process has occurred so fast in young age.

Lp(a) is produced by the liver and is not controlled with diet. Drug such as atorvastatin and gemfibrozil have been shown to decrease the concentration of Lp(a) in blood (10, 11).

We believe that there is much more to do to prevent CHD among the young than among the older adults. Primary prevention must begin prior to 15-34 years age group if we want to reduce the prevalence and extent of atherosclerosis and ultimately reduce the risk of atherosclerosis disease later in life. In our patient if we could diagnose the high level of Lp(a) with the onset of the symptom, early therapy could have reverted the progression of CAD. We should add that due to rarity of CAD in this age group

especially in Iran, this study could not be compared with other centers. Available papers and articles only point to the risks and manifestations of the disease in this age group and do not mention the prevalence of CAD in youth.

We conclude that elevated Lp(a) level is the most important cause of premature CAD in this patient, and emphasize the need to screen siblings of patients with premature CAD. We recommend that in patients with CAD in young age complete laboratory examination be done and risk factors be modified to prevent progression of CAD.

REFERENCES

1. Hawe E, Talmud PJ, Miller GJ, Humphries SE; Second Northwick Park Heart Study. Family history is a coronary heart disease risk factor in the Second Northwick Park Heart Study. Ann Hum Genet. 2003 Mar; 67(Pt 2):97-106.
2. Krauss RM. Atherogenecity of triglyceride-rich Lipoprotein. Am J Cardiol. 1998; 81: 13b-17b.
3. Braunwald E, Zipes DP, Peter L, Bonow R, editors. Braunwald's heart diseases. 6th ed. Philadelphia: W B Saunders; 2003. p.1637-1639.
4. Ridker PM, Stampfer MJ, Rifai N. Novel risk factors for systemic atherosclerosis: a comparison of C-reactive protein, fibrinogen, homocysteine, lipoprotein(a), and standard cholesterol screening as predictors of peripheral arterial disease. JAMA. 2001 May 16; 285(19):2481-85.
5. Oalmann MC, Strong JP, Tracy RE, Malcom GT. Atherosclerosis in youth: are hypertension and other coronary heart disease risk factors already at work? Pediatr Nephrol. 1997 Feb; 11(1):99-107.
6. Tribble DL, Rizzo M, Chait A, Lewis DM, Blanche PJ, Krauss RM. Enhanced oxidative susceptibility and reduced antioxidant content of metabolic precursors of small, dense low-density lipoproteins. Am J Med. 2001 Feb 1; 110(2):103-110.
7. Schettler G, Schwarz S, Benderly M, Brunner D. Subfractions of high density lipoprotein in CHD patients compared to healthy individual and their correlation with other risk factor for CHD. Atherosclerosis. 1995; 115: S78.
8. 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.
9. Kinlay S, Dobson AJ, Heller RF, McElduff P, Alexander H, Dickeson J. Risk of primary and recurrent acute myocardial infarction from lipoprotein(a) in men and women. J Am Coll Cardiol. 1996 Oct; 28(4):870-875.
10. Gonbert S, Malinsky S, Sposito AC, Laouenan H, Doucet C, Chapman MJ, Thillet J. Atorvastatin lowers lipoprotein(a) but not apolipoprotein(a) fragment levels in hypercholesterolemic subjects at high cardiovascular risk. Atherosclerosis. 2002 Oct; 164(2):305-311.
11. Ramires JA, Mansur AP, Solimene MC, Maranhao R, Chamone D, da Luz P, Pileggi F. Effect of gemfibrozil versus lovastatin on increased serum lipoprotein(a) levels of patients with hypercholesterolemia. Int J Cardiol. 1995 Feb; 48(2):115-120.