

CASE REPORTS

Acute myocardial infarction in a 17-year-old Bangladeshi boy-possibility of existence of a Bangladeshi paradox

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ABSTRACT

Coronary artery disease (CAD) and acute myocardial infarction (AMI) are diseases of older age. AMI is uncommon in young age and rare in adolescence. A seventeen years old Bangladeshi boy developing acute anterior wall MI is described here. Risk factor analysis for CAD revealed absence of most of the conventional risk factors, like smoking, diabetes mellitus, hypertension. An elevated lipoprotein(a) and a low HDL cholesterol was detected in blood which are emerging risk factors of CAD in young Bangladeshis.

Key Words: Coronary artery disease, Young myocardial infarction, Bangladeshi paradox

1. INTRODUCTION

There is an alarming increase in incidence of Coronary artery disease (CAD) in South Asian countries including Bangladesh, afflicting people here at a relatively younger age.^[1] Incidence of first myocardial infarction (MI) among Asians at age less than 40 years is 9.7% in men and 4.4% in women, which is 2 to 3.5 fold higher than that in the West European populations.^[2] In South Asians about 25% of all cases of AMI occur under the age of 40.^[3] Thus, an increasing number of young patients are being admitted with AMI in Bangladesh. On an average, people here develop MI about 10 years earlier than other populations.^[4] The markedly higher prevalence of MI in young Bangladeshis can not be explained fully by the presence of conventional CAD risk factors alone. Rather, a genetically determined

elevated level of lipoprotein(a) acting in combination with a low HDL-cholesterol related to environmental or lifestyle factors may lead to development of CAD at a much younger age.^[5]

2. CASE REPORT

A 17-year-old male Bangladeshi boy presenting with severe central chest pain, profuse sweating and respiratory distress was admitted to the coronary care unit of Chittagong Medical College Hospital, a tertiary care cardiac center in the port city of Bangladesh. He was normotensive, non diabetic, nonsmoker and was not a cocaine abuser. He had no past history of chest pain. There was no family history of CAD and dyslipidemia. His ECG showed ST-segment elevation in anterior leads(V₁-V₆) and serum troponin I was 8.5 ng/ml.

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His pulse rate was 110/min, BP was 110/75 mmHg. Auscultatory findings revealed tachycardia, no murmur and clear lung bases. His anthropometric parameters were: weight 78 kg, height 5 feet 2 inches, waist circumference 90 cm, and BMI 35.4 kg/m². Hemodynamic and electrocardiographic monitoring started immediately. He was given 300 mg soluble aspirin orally, sublingual nitroglycerine, i.v. morphin 4 mg and i.v. streptokinase 1.5 mg in 100 ml of normal saline for 1 hour, which was administered within an hour of onset of his chest pain symptom. Subsequently, metoprolol 25 mg b.i.d., ramipril 1.25 mg b.i.d. and atorvastatin 20 mg daily were started. The patient became painfree and remained hemodynamically stable. Post thrombolysis followup ECG revealed significant resolution of ST elevation in precordial leads. Bedside echocardiography revealed hypokinesia of anterior wall with an ejection fraction of 50%. Blood biochemical examination revealed a fasting blood glucose of 91 mg/dl and 2 hours post-prandial glucose of 107 mg/dl. Fasting serum lipid profile showed: total cholesterol 224 mg/dl, LDL 133 mg/dl, HDL 32 mg/dl, and triglyceride 133 mg/dl. Serum Lp(a), estimation done by quantitative latex-enhanced immunoturbidimetric test using human Lp(a) kit (Human Gessellschaft, Weisbaden, Germany) was 42 mg/dl, with a normal reference value of 15-20 mg/dl. The patient made a good in-hospital recovery and was discharged after 5 days of admission. Coronary angiography done after 6 weeks of discharge revealed normal epicardial coronary arteries.

3. DISCUSSION

CAD is emerging as a new epidemic in developing countries including Bangladesh, afflicting the young peoples more and more. The INTERHEART study showed that the mean age of first MI in South Asian (Bangladesh, India, Pakistan, Sri Lanka and Nepal) population is 53 years, whereas, that in Western Europe, China and Hong Kong it is 63 years.^[2] This increase in MI in the early age group population may be due to an increase in various CAD risk factors among them.^[6] A genetically elevated level of lipoprotein(a) has been found to produce premature atherosclerosis in South Asians leading to its higher prevalence among populations under age 40.^[7] The atherogenicity of LP(a) is 10 times higher than LDL cholesterol. It is structurally homologous to circulating plasminogen, thus having thrombogenic and antifibrinolytic property in addition to its high atherogenicity. Genetically determined elevated LP(a) play an important role in accelerating atherosclerosis that leads to premature CAD and MI. A combination of high Lp(a) and low HDL cholesterol confers a very high risk of CAD. Such a combination was found to be present in 42% of Asian Indians by Enas *et al.*^[8] A high prevalence of CAD was found among physicians of Indian

origin working in U.S. , in whom most of the conventional CAD risk factors except diabetes were absent.^[9] This high CAD rates despite low level of risk factors was leveled as Asian Indian paradox.^[10]

A raised level of Lp(a) along with a slightly raised LDL and markedly low HDL was the only risk factor in this boy with anterior wall MI. The patient was obese and had a waist circumference of 90 cm, and a BMI of 35.4 kg/m². South Asians have more abdominal obesity and a higher percentage of body fat compared with other populations. Here, a person with a BMI > 25 kg/m² is considered to be obese, whereas, the same is > 35 kg/m² for Western populations.^[11] Similarly, the recommended desirable value for waist circumference is 8-10 cm lower than that for Western populations. Thus an early institution of weight management program is recommended for these peoples. Serum lipid profile is influenced by environmental and lifestyle factors. A study done on urban Bangladeshi population showed a significantly higher levels of total cholesterol, LDL and triglyceride among younger peoples aged 30-39 years compared with older peoples regardless of sex.^[12] The prevalence of obesity is increasing in children and adolescents in Bangladesh. Increased use of atherogenic diet coupled with inadequate consumption of fruits and vegetables and lack of physical exercise are responsible for childhood obesity here. A genetically raised Lp(a), in combination with an altered lifestyle factors leading to obesity and dyslipidemia are both responsible for MI in this case. The boy presented to CCU early (within 1 hour of onset of chest pain). Hence, thrombolysis with i.v. streptokinase was preferred to primary percutaneous coronary intervention. Coronary angiography done 6 weeks after discharge from hospital revealed normal coronary arteries. The boy made a good in-hospital recovery. Thrombus in the infarct related artery underwent a complete lysis accounting for absence of residual thrombus and findings of normal coronaries on CAG. Any possible source of thrombus with subsequent embolization into coronary artery were excluded by doing Doppler echocardiographic study. Normal coronary arteries on CAG following an attack of MI in youngs were also reported in CASS Registry.^[13]

Profile of young Bangladeshis developing AMI were also published by Fazilatunnesa M *et al.*^[14] and Das P K *et al.*^[15] who reported the youngest age of their subjects as 17.5 years and 21 years respectively. Thus, the present one is the youngest patient with AMI reported in Bangladesh. Moreover, we looked for Lp(a) level, which is one of the emerging risk factors incriminated for premature CAD in South Asian populations. A high CAD rate in presence of low conventional risk factor raises the possibility of a CAD risk paradox in our population. Like Indian paradox, as de-

scribed in CADI study, this may be a Bangladeshi paradox as well. This urges searching for high Lp(a) level among Bangladeshis at an early age, preferably soon after birth and controlling lifestyle factors to prevent childhood obesity.

4. CONCLUSION

A genetically determined elevated Lp(a) along with a low HDL cholesterol related to lifestyle factors are responsible for AMI in this case. Controlling lifestyle factors by dietary intervention and regular physical exercise may prevent childhood obesity. Considering the alarming increase of CAD and MI at a much early age in Bangladeshis in absence of most

of conventional risk factors, the possibility of a Bangladeshi paradox of CAD should be considered and studied further.

Recommendation

Screening for serum lipoprotein(a) may identify those at risk of developing MI at a very young age. Subjects with an elevated LP(a) should receive potent high dose statin along with niacin to prevent development of CAD and subsequent MI.

CONFLICTS OF INTEREST DISCLOSURE

The authors have declared no conflicts of interest.

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