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Case Report

Management of ST Elevation Myocardial Infarction in a 17 years Girl in A Tertiary Care Hospital.

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Abstract

Background: *Worldwide, myocardial infarction (MI) is an important cause of death. Number of AMI among young adult is increasing day by day in Bangladesh. Acute MI occurs most commonly at an older age. However, the incidence of acute MI in adolescents is increasing. This is partly due to an increase in cardiovascular risk factors (e.g. smoking, unhealthy diet), which might lead to premature atherosclerosis. However, several non-atherosclerotic causes of MI in adolescents are also described in the literature, such as vascular spasm due to the use of cocaine, amphetamine etc. We may assume that acute MI is not considered to be the most likely cause of chest pain in adolescents. Therefore, the risk of a dramatic outcome in this patient category may be significant. Myocardial infarction (MI) in the young (age < 45 years) is a significant problem; however, there is a scarcity of data on premature coronary heart disease and MI in the adolescent patients. MI in adolescents (age between 10–19 years) is extremely rare.Premature AMI, particularly in the setting of obstructive CAD and/or female sex, is an aggressive disease with high rates of recurrence and mortality, attributed largely to suboptimal control of modifiable risk factors.1 Collet et al2 reported that 1 in 3 patients with premature (≤45 years of age) CAD, of whom the majority experienced AMI, had at least 1 recurrent event over a follow-up period of 20 years. Strong independent predictors for recurrent events were persistent smoking, diabetes, hypertension. We present a case of the 17-year-old girl with extensive ST-segment elevated anterior wall myocardial infarction and found to have complete thrombotic occlusion of proximal left anterior descending coronary artery.*

Keywords: *ST-elevation myocardial infarction, Young MI, Premature coronary artery disease, Acute coronary syndrome, Primary percutaneous coronary intervention, Risk factors.*

Introduction: Number of AMI among adolescence is increasing day by day in Bangladesh.The earlier age of AMI in South Asians can be largely explained by higher risk factor levels at younger ages. Acute myocardial infarction (MI) is rare in teenagers and young adults. The pathophysiology of their infarcts is varied, which not usually due to atherosclerotic plaque rupture except for those genetically predetermined or familial hyperlipidemias. There are many non-atherosclerotic causes of premature coronary artery disease like the coronary spasm, coronary embolization, coronary dissection, anomalous coronary origin, coronary aneurysm, antiphospholipid syndrome, myocardial bridges and very rarely hypercoagulable states.

The South Asian countries of India, Pakistan, Bangladesh, Sri Lanka, and Nepal account for about a quarter of the world's population and contribute the highest proportion of the burden of cardiovascular diseases compared with any other region globally.[3-5] South Asian migrants living in several countries have higher death rates from coronary heart disease (CHD) at younger ages compared with the local population despite apparently lower levels of conventional risk factors.[4-8] Deaths related to cardiovascular disease also occur 5 to 10 years earlier in South Asian countries

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than they do in Western countries.[9,10] This has raised the possibility that South Asians exhibit a special susceptibility for acute myocardial infarction (AMI) that is not explained by traditional risk factors.

Among individuals living in the United Kingdom, the earlier onset of CHD among South Asian migrants is not an artifact of differences in the population distribution because the higher incidence of CHD is most marked in those younger than age 40 years (about a 3-fold difference), whereas it is less marked in those older than 60 years (about a 1.5-fold difference) based on an analysis of UK mortality data.[8] Despite documenting the higher rates of earlier CHD in South Asians, few studies have been able to shed light on its reasons. Most studies do not include information on diet, physical activity, abdominal obesity, psychosocial factors or apolipoprotein levels, and do not have sufficiently large numbers of clinical events to reliably assess the comparative effects of the various risk factors at various ages in South Asians compared with other ethnic groups.

Within the 5 South Asian countries studied, Bangladeshis had the highest prevalence of most risk factors. Similar observations also have been made in migrant Bangladeshis living in the United Kingdom. Whether this is related to lower income and educational levels in Bangladesh compared with other South Asian countries is unclear and needs to be examined. The striking variation observed in the lower age of presentation of first AMI in South Asians, with Bangladeshis being the youngest and Nepalese the oldest, indicates that the onset of AMI could be delayed by modifying these risk factors.

It is important to consider an acute MI as a diagnosis in adolescents with chest pain. Acute MI can present with or without ST-segment elevations on the electrocardiogram: STEMI or non-STEMI [4,5]. Prior to acute MI, a phase of unstable angina pectoris may occur. Unstable angina pectoris means chest pain at rest, de novo or with a crescendo pattern. Unstable angina pectoris, STEMI and non-STEMI are part of the acute coronary syndrome[4,5]. It is important to realize that acute MI presents differently over time on an electrocardiogram. At first, there is a hyperacute phase with large symmetrical or peaked T waves. These are followed by convex ST-segment elevation, which represents a completely developed ischaemic phase. Finally, a chronically stabilized phase develops, in which negative T waves are replaced by positive T waves. A STEMI in adolescents may be the result of premature atherosclerosis or have an underlying non-atherosclerotic cause.

Case Report:

A 17 years old, poorly controlled known diabetic girl, height-5-1 inches, weight-48 kgs, admitted in CCU of Rangpur Medical College Hospital with 48 hours history of retrosternal, compressive chest pain, profuse sweating, shortness of breathing, palpitation, nausea, vomiting, pain radiate to left upper limb. She was drowsy, her pulse was non-palpable, BP was non-recordable. Immediately ECG was done, her 12-lead ECG shows ST elevation from Lead V1 to V6, heart rate-146 beat/mi, rhythm-regular, her serum troponin-I was-10.76ng/ml (Reference value<0.06ng/ml). She was diagnosed as acute extensive anterior MI with uncontrolled Type 2 diabetes mellitus & managed conservatively by standard protocol, comprising-oxygen inhalation, Ecosprin, inj-Pathedine, inj-Low-mollicular weight Heparin, B-Blocker, Atorvastatin, inj-Dopamin, Inj-Nor-adrenalin, ACE-inhibitor, Nicorandil, Ivabradin, Frusemide, subcutaneous regular insulin, etc. She was not thrombolysed by streptokinase due to delayed arrival in hospital. There was no history of premature coronary artery disease in the family or familiahypercholesterolemia (FH) and she denied any history of smoking and drug abuse, analgesic abuse, athletic training or vigorous physical exercise. There was no history of any previous episode of arterial or venous thrombosis. On examination, her BMI was 15.74 and there were no tendon xanthomas, no arcus senilis, no carotid or renal bruit. At CCU, her bed side echocardiogram was done on day of admission & found-global hypokinesia of LV except basal segment of postero-lateral wall, moderate LV systolic dysfunction (LV EF-34%), Trace TR, no pericardial effusion. After all initial management, at 9 days, she was referred to National Institute of Cardiovascular Disease (NICVD), Dhaka for further specific management.

On September 28, 2022, she got admitted in NICVD, Dhaka for coronary angiogram. During preparing her for coronary angiogram, she was found Covid-19 positive by rapid antigen test. Then she was isolated & treated conservatively for 8 days. After 8 days, she was again tested & found covid-19 negative, then she was transferred from covid isolation ward to cardiology unit for coronary angiogram.

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On 12.10.2022, after maintaining all standard protocol, her coronary angiogram was done by right sided radial route & found-LAD-Type 111 vessel & Proximal LAD had 90-99% stenosis, Diagonal-Normal, LMCA-Normal, LCX-Dominant vessel, normal, RCA-Non-dominant vessel, normal, PDA & PLB-Normal. During coronary angiogram, she suddenly developed cardiac arrest. Immediately CPR was started, IV Nor-adrenalin, IV Dopamine started, IV atropine given, cardiac anesthetic was called immediately, then promptly she was intubated & then she was put in Ventilator. Inspite of applying all life-saving medicine, her heart was non-responsive for about 7 minutes, then after applying repeated Intracardiac-Nor-adrenalin, her heart started beating. Then suddenly ventricular fibrillation developed & managed immediately by applying repeated DC Cardio version following standard protocol. Immediately she developed respiratory acidosis & managed by repeated IV Sodium bicarbonate injection, oxygen inhalation, etc. She was still then in cardiogenic shock. With all supportive measures, then immediately stenting was done at Proximal LAD, using one drug-eluting stents-Orsiro 2.75x30 mm (At 10 ATM), G. catheter was-EBU 6Fx3.5, G. Wire was-Asahi Rinato. Then after completion of CAG & PCI, she was sent to CCU for further management. At CCU, repeated ECG was done & found no further development of new MI. Immediately blood was sent for urgent Blood Gas Analysis, Serum Electrolyte, Serum Creatinine, RBS, Complete Blood Count & found respiratory acidosis, Hypokaelemia, Hyponatremia, Leukocytosis & managed accordingly. Next day, at CCU, bed sided Echocardiogram was done to see whether any pericardial effusion. Echocardiogram revealed no pericardial effusion, LV-Antero-septum, apex, mid to apical anterior wall was hypokinetic, moderate LV systolic dysfunction (LVEF-35-40%), good RV systolic function. Next day, she complaints of pain & swelling of right thigh & femoral access site with haematoma formation. We examined & found that she developed haematoma but her distal right leg pulse was normally present. Then we referred her to vascular surgery department for Duplex Study of right leg swelling & for its further specific management. Duplex Study report was found normal. Her pain & haematoma subsided with conservative management. Before discharged, we've completed a further echocardiogram & found-whole septum, apex, mid to apical segment of anterior wall of LV was found hypokinetic, mild LV systolic dysfunction with LV EF-47%, No Diastolic Dysfunction, No Pericardial Effusion, No pulmonary Hypertension. We further check her all vital & biochemical parameter & when found all normal, then we discharged her with all optimal drugs treatment, life style modification advices & further follow-up advices.

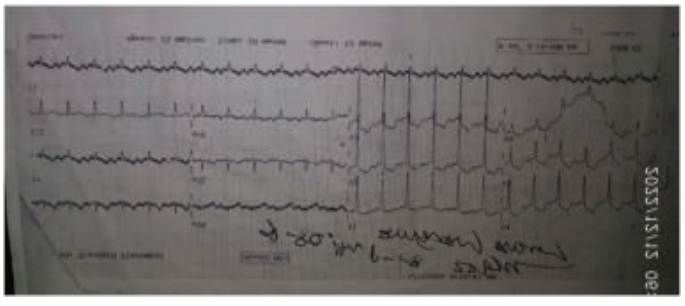

Figure 1: ECG on 21.09.2022.

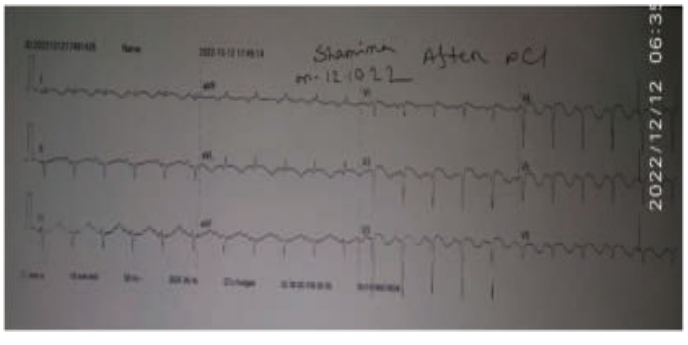

Figure 2: ECG on 12.10.2022 after PCI.

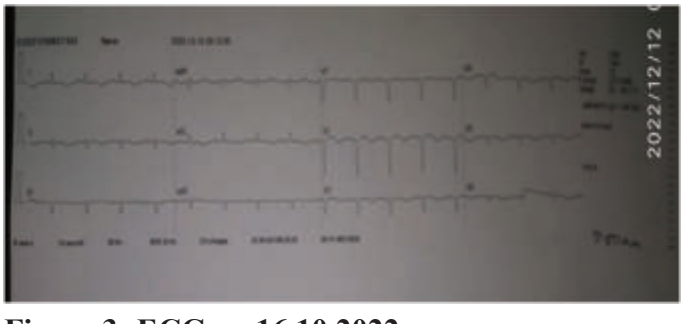

Figure 3: ECG on 16.10.2022.

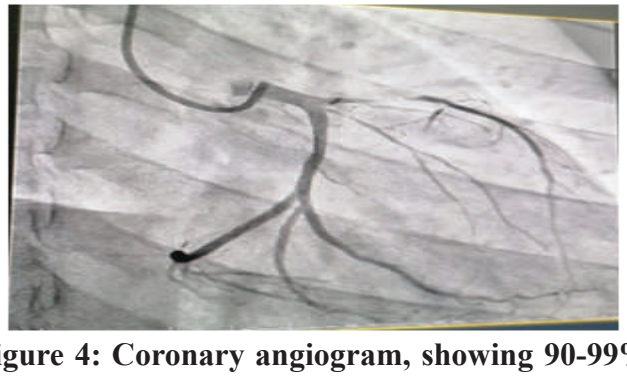

Figure 4: Coronary angiogram, showing 90-99% block at Proximal LAD before Stenting.

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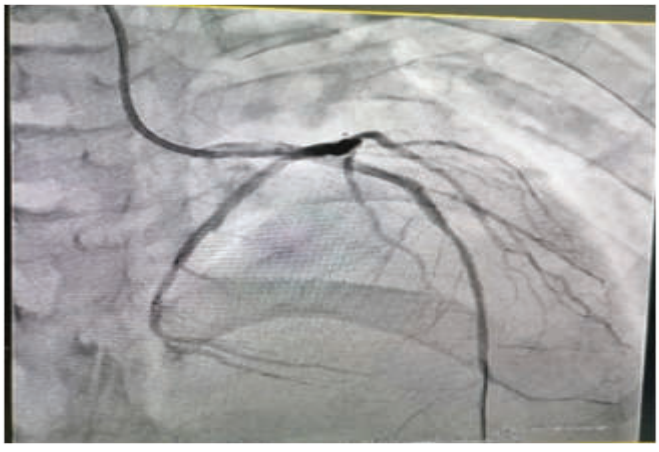

Figure 5: Coronary angiogram-after stenting at Proximal LAD.

Discussion:

Epidemiology

The prevalence of CAD varies considerably by populations, may be up to 10 folds.[3] South Asians are unduly prone to develop CAD.[4] Most notable features of CAD in this population are the extreme prematurity and severity; 2–4-fold higher prevalence, incidence, hospitalization and mortality; 5–10 years earlier onset of first myocardial infarction (MI) and 5–10-fold higher rates of MI and death before the age of 40 years.[4] The exact prevalence of CAD in Bangladesh is not known. Only a limited number of small-scale epidemiological studies are available. Probably the prevalence of IHD was first reported in 1976, which was 0.33%.5 More recent data indicates CAD prevalence between 1.85%6 and 3.4%7 in rural and 19.6% in an urban sample of working professionals.[8, 9] Despite marked disparity in values, there seems to be a rising prevalence of CAD in Bangladesh.

A recent study from rural Bangladesh demonstrated a dramatic increase in CVD from 1986 to 2006. The age-standardized CVD mortality rates increased by 30-fold (from 16 deaths per 100,000 to 483 deaths per 100,000) among males and 47-fold (from 7 deaths per 100,000 to 330 deaths per 100,000) in females.10 A nation-wide survey is needed to find out the current epidemiological aspects of CAD in the country.

Coronary artery disease (CAD) is an increasingly important medical and public health problem, and is the leading cause of mortality in Bangladesh. Like other South Asians, Bangladeshis are unduly prone to develop CAD, which is often premature in onset, follows a rapidly progressive course and angiographically more severe. The underlying pathophysiology is poorly understood. Genetic predisposition, high prevalence of metabolic syndrome and conventional risk factors play important role. Lifestyle related factors, including poor dietary habits, excess saturated and trans-fat, high salt intake, and low-level physical activity may be important as well. Some novel risk factors, including hypovitaminosis D, arsenic contamination in water and food-stuff, particulate matter air pollution may play unique role. At the advent of the new millennium, we know little about our real situation. Large scale epidemiological, genetic and clinical researches are needed to explore the different aspects of CAD in Bangladesh.

Among patients presenting with acute myocardial infarction (AMI), the proportion of young individuals has increased in recent years. Although coronary atherosclerosis is less extensive in young patients with AMI, with higher prevalence of single-vessel disease and rare left main involvement, the long-term prognosis is not benign. Young patients with AMI with obstructive coronary artery disease have similar risk factors as older patients except for higher prevalence of smoking, lipid disorders, and family history of premature coronary artery disease, and lower prevalence of diabetes mellitus and hypertension. Smoking cessation is by far the most effective secondary preventive measure. Myocardial infarction with nonobstructive coronary arteries is a relatively common clinical entity (10%-20%) among young patients with AMI, with intravascular and cardiac magnetic resonance imaging being key for diagnosis and potentially treatment. Spontaneous coronary artery dissection is a frequent pathogenetic mechanism of AMI among young women, requiring a high degree of suspicion, especially in the peripartum period.

Myocardial Infarction with Obstructive Coronary Arteries:

Pathology of atherosclerotic plaque in young individuals:

Premature atherosclerosis: Development of coronary atherosclerosis at a young age is correlated with the presence of conventional cardiovascular risk factors. Examples are smoking, hypertension, dyslipidaemia, overweight, inactivity and stress [1]. Smoking is one of the most important risk factors.

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Frequent exposure to cigarette smoke stimulates the release of catecholamines, which cause damage to the endothelial cells. This can lead to vascular intima dysfunction already at a young age. This process involves alternating low-level cholesterol deposition and platelet aggregation (plaque formation). The lipid core within this plaque formation may rupture. This results in release of vasoactive factors leading to acute vascular occlusion. Genetic predisposition also increases the risk of premature atherosclerosis, such as in the case of a mutation in the factor V Leiden gene or genetic hypercholesterolemia [7]. Treatment of acute MI due to premature atherosclerosis consists of protocolized revascularization by means of primary PCI, followed by cardiovascular risk management on risk factors.

Atherosclerosis is a progressive disease that starts during the early years of life. It can be considered a "childhood disease" with an "incubation period" of a few decades.11,12 Age-associated changes in the cardiovascular system accelerate atherosclerosis; hence atherosclerotic CAD is expected to be less extensive in young individuals. Indeed, angiographic studies in patients with CAD that used intravascular ultrasound-virtual histology have demonstrated that the burden of atherosclerosis is increasing day by day.

Myocardial Infarction with Nonobstructive Coronary Arteries:

Non-atherosclerotic causes: In MI of non-atherosclerotic causes the mechanism of coronary occlusion differs [1, 2]. This may result from various underlying disorders that are accompanied by unconventional risk factors in some cases, such as pregnancy and direct contact sports. For example, the occlusion can be induced by coronary spasms, embolization through coronary arteries due to endocarditis or secondary thrombus formation. Development of secondary thrombosis could be explained by the Virchow triad. This theory describes that development of thrombosis that can be triggered by three factors: stasis of blood flow, endothelial damage and hypercoagulability or abnormal blood composition. For example, patients with the nephrotic syndrome have an increased risk of developing thrombosis because of an increased coagulation state. Coronary spasms can also cause an increased risk of secondary thrombosis because these spasms induce minor damage to the vascular endothelium and activate coagulation. Both cocaine and binge drinking can cause coronary spasms.

We should address the pathophysiological process and subsequent diagnostic approach in adolescents with MI resulting from either premature atherosclerosis or of non-atherosclerotic causes. Insight into the potential operational mechanisms of the coronary artery incident may have a major impact on the clinical course following admission. We would like to underline that a personalized clinical approach remains of utmost importance in each patient treated by protocolized medicine. This is particularly true when acute MI occurs at a young age, since the underlying cause more frequently differs from the conventional atherosclerotic process in this patient category.

Acute MI in adolescents may be caused by premature atherosclerosis or have non-atherosclerotic causes (e.g. vascular spasms during the use of cocaine)[2]. We should address the pathophysiological process and the diagnostic approach in adolescents with MI resulting from either premature atherosclerosis or non-atherosclerotic causes. We would like to mention that our description of non-atherosclerotic causes extends beyond the current clinical conception of MINOCA (myocardial infarction in non-obstructive coronary artery disease) [3].

Prognosis:

Premature AMI, particularly in the setting of obstructive CAD and/or female sex, is an aggressive disease with high rates of recurrence and mortality, attributed largely to suboptimal control of modifiable risk factors. Collet et al5 reported that 1 in 3 patients with premature (≤45 years of age) CAD, of whom the majority experienced AMI, had at least 1 recurrent event over a follow-up period of 20 years. Strong independent predictors for recurrent events were persistent smoking, diabetes, hypertension.

Conclusion:

This case illustrates that MI in adolescent population can occur in the absence of: smoking, family history of premature CAD, atherosclerotic risk factors, drug abuse, coronary anomalies, systemic illness, antiphospholipid syndrome and hypercoagulable states. Adolescent (≤18 year) patients presenting with symptoms and ECG changes suggestive of myocardial ischemia or infarction should receive

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appropriate treatment, including the immediate administration of thrombolytic drugs or coronary angioplasty if indicated.

Conflict of interest: There is no conflict of interest.

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