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Case Series

Case Series of Acute Myocardial Infraction in Teenagers: Alarming Incidence

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Abstract

We may presume acute myocardial infarction (AMI) is not a likely cause of chest pain in teenagers, but although rare, young adults and even teenagers are also presenting with AMI. Increasing AMI in young age is partly explained conventional risk factors such as smoking dyslipidemia, unhealthy food habits, lack of exercise, but several non-atherosclerotic risk factors are described in adolescents. These are coronary artery ectasia (CAE), spasm, and drug abuse such as cocaine, coronary artery anomalies, coronary embolism, coronary artery dissection, hypercoagulable states, and use of oral contraceptive (OC) pills. Here, we report the two cases of a previously healthy 16-year-old boy and 16 years female of AMI without known risk factors. The possible mechanism for AMI in male is CAE and in female patient OC pills is probably linked with AMI. Our aim is to increase awareness of diagnosis AMI even in adolescents with chest pain so that mishap would not happen.

Key words: Coronary artery ectasia, Coronary heart disease in teenagers, Myocardial infarction, OC pills, Risk factors

INTRODUCTION

Coronary heart disease (CAD) is a major cause of morbidity and mortality with acute myocardial infarction (AMI) being one of the most common presentations of CAD. AMI is rare in teenagers and young adults and accounts for only 3% of all patient with coronary artery disease in individuals younger than 40 years of age. Younger age group has different risk profile as compared with older populations. Non-atherosclerotic causes are usually responsible for AMI in teenagers. Among the many described non-atherosclerotic causes are spontaneous coronary artery dissection, and coronary spasm related to drug use, coronary artery ectasia (CAE), embolism from aortic valve, thrombosis from hypercoagulable states, coronary anomalies, and antiposholipid syndrome are more prevalent in this age group.[1]

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Here, we present two cases of a previously healthy 16-year-old male and female who presented with myocardial infarction without known risk factors.

Here will want to emphasis required to consider diagnosis of AMI in teenagers presenting as chest pain and to discuss different risk profile for AMI in teenagers. Coronary artery disease is alarmly increasing younger population including teenagers too.

CASE REPORTS

Case 1

A 16-year-old male came to our hospital with a complain of chest pain since past 2 days. There was no significant previous medical history and non-addict. On examination, vitals were normal. Respiratory function was normal. Blood investigations revealed that troponin I was positive, but CBC, LFT, and RFT were normal. ECG showed ST elevations in leads V1-5 with consistent with acute ST elevation anterolateral infarction [Figure 1]. Echocardiography showed mild LV dysfunction with the left anterior descending (LAD) artery territory hypokinesia. Coronary angiogram (CAG) showed ecstatic proximal

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left anterior descending artery [Figure 2]. There was no presence of atherosclerotic lesion. Based on above findings, diagnosis of anterior wall myocardial infarction was made. However, no conventional risk factors for this could be elicited.

The patient was given conservative treatment only (dual antiplatelet therapy with Low-Molecular-Weight Heparin [LMWH] and statin). The patient was better after 1 month follow-up and was lost was further follow-ups.

Case 2

A 16-year-old female came to our hospital with a complain of chest pain for an hour. The patient was on oral hormonal medication (Cypoterone acetate 2 mg and E estradiol 0.03 mg in combination) since past 2 months for menorrhagia and had taken 1st dose of covaxin 1 month back. The patient was obese with 27.5 BMI. On examination, vitals were normal. Respiratory function was normal. ECG showed acute anterior wall myocardial infarction [Figure 3]. She was thrombolysed with streptokinase and treated with dual anitiplatelets, statin LMWH, beta-blockers, and antianginals. Echocardiography showed mild LV dysfunction with LAD artery territory hypokinesia. CAG showed normal coronary arteries [Figure 4]. There was no presence of atherosclerotic lesion. Her tests for Protein S, Protein C, antithrombin III, homocysteine, and factor V leiden were negative. The patient was given conservative treatment only (dual antiplatelet therapy with LMWH and statin). The patient was better after treatment and post-treatment echocardiography showed improved LV function with LV apical clot.

DISCUSSION

Earlier AMI was thought a disease of older population but affecting youngers and even teenagers too nowadays. Although it occurs at younger age in India compared to Western population but rare in teenagers. The clinical presentation, risk factors for myocardial infarction in young patient, differs from those in older patients. Coronary arteriography performed in young patients after myocardial infarction has identified a relatively high prevalence of angiographically normal coronary arteries.[2] Furthermore, risk factor analysis in young patients with AMI has shown a particularly high prevalence of smoking compared with that in older patients with higher prevalence for males as compared to females.[2]

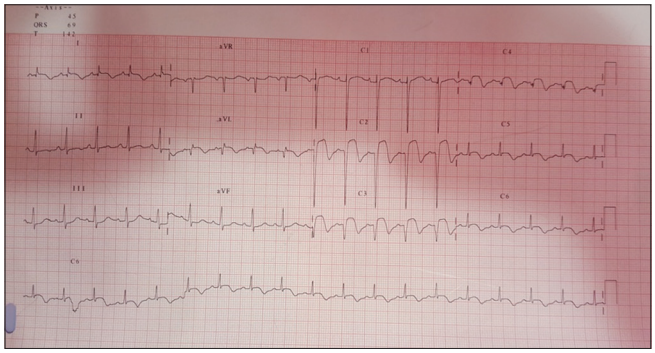

Figure 1: ECG findings

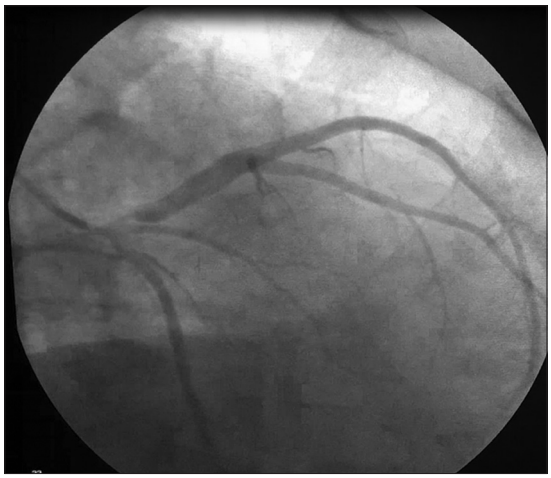

Figure 2: Coronary angiogram showed ecstatic proximal left anterior descending artery

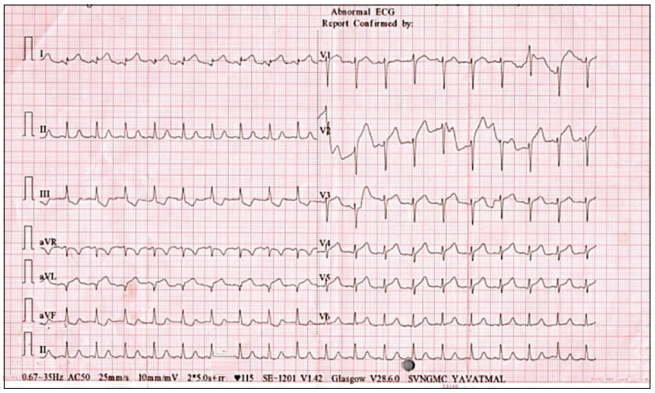

Figure 3: ECG on admission showing anterior wall MI

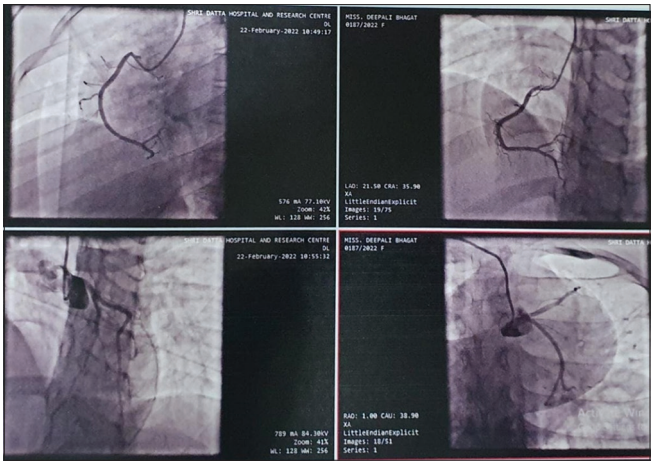

Figure 4: CAG showing normal coronaries

MI in young patients having normal coronary arteries can be due to arteritis, thrombosis, embolization, CAE or spasm, patients on oral contraceptive (OC) pills, coronary artery dissection, and anomalous coronary arteries. As is the case with venous thrombosis, coronary thrombosis can be seen in hypercoagulable states, such as protein C and protein S deficiency, antiphospholipid syndrome, or nephrotic syndrome.[2] Coronary artery spasm can cause MI in patients with cocaine abuse and also in association with alcohol binges. In some cases, the cause of MI in young patients can be the result of atherosclerotic process, which starts in early childhood.

In our male patient, CAG showed proximal LAD ectasia. The various studies by Befeler *et al.* and Alford *et al*., have defined CAE as an abnormal enlargement of one part of the coronary artery to 1.5–2 times more than the diameter of an adjacent normal segment is defined as CAE, and further, enlargement is defined as coronary artery aneurysm. CAE may be congenital or acquired. The associated diseases reported in its etiology are 50%atherosclerosis, 20–30% congenital diseases, and 10–20% inflammatory or connective tissue diseases.[3] The thinning of the vascular media layer is remarkable in the pathogenesis of atherosclerosis and ectasia. Thin vascular wall and an increased wall stress result in a vicious cycle, thus a diffuse ectasia or local aneurysm develops as a result of progressive dilatation in the coronary. CAE is mostly asymptomatic. Symptomatic cases usually emerge in the form of effort angina or rest angina. Some studies reported that ectasia caused slow blood flow, thrombus formation, and vasospasm in coronary arteries; and it could lead to an ischemic episodes without obstructive CAD.

The ischemic mechanism in patients with CAE though has not been fully understood, it is accepted that the leading cause of ischemia and angina is the impaired microvascular perfusion. It has been reported that a slow or turbulent flow in the dilated vessels resulted in thrombosis or ischemia at the ecstatic segment, leading to embolism in the distal coronary artery.[3] In our male patient, CAE is a possible mechanism for AMI.

Our second case (female patient) was taking OC pills since 1 month. Her CAG was normal, no detectable hypercoagulable state. Although lowering the dose of estrogen in combined hormonal contraceptives has reduced the risk of thrombosis, it has not eliminated it. With regard to arterial thromboembolism, Lidegaard *et al.* found relative risks of stroke of 1.60 (95% confidence interval [CI] 1.37–1.86; NNH 29 762), 1.75 (95% CI 1.61–1.92; NNH 23 810), and 1.97 (95% CI 1.45–2.66; NNH 18 409) among patients taking combined hormonal contraceptives containing EE at a dose of 20 μg, 30–40 μg and 50 μg, respectively, compared with non-users. The corresponding relative risks for myocardial infarction were found to be 1.40 (95% CI 1.07–1.81; NNH 357 143), 1.88 (95% CI 1.66– 2.13; NNH 162 338), and 3.73 (95% CI 2.78–5.00; NNH 52 329).[4]

It has been proposed that the observed difference in thrombosis risk between agents containing different progestogens is the total estrogenicity of the combined product. Estrogenicity is determined by both the dose of EE and the type of progestogen used. Sex hormone-binding globulin can be used as a marker of estrogenicity, because the hormone levels rise in states of high estrogen. Cyproterone acetate-ethinyl estradiol raises the levels of sex hormone-binding globulin by 300–400% compared with a 50% increase caused by levonorgestrel preparations, which are known to have the lowest risk of thrombosis.[4] This excessive rise in estrogenicity caused by CPA-EE likely increases the risk of thrombosis through various effects on the coagulation pathway, including reduction of the activity of various coagulation inhibitors (e.g., antithrombin, protein C, and tissue factor pathway inhibitor); increased levels of coagulation factors, including II, VII, VIII, and X; and increased platelet aggregation.[5]

Various studies of young AMI by Duvernoy *et al.* who described acute MI in two adolescents' males (14 years, 15 years) and studies by Miyayama *et al*., Maghaieth *et al*., Shahsavariet *et al*., Abid *et al*., Jaymali *et al*., Yildiz *et al*., and Chen *et al.* have described non atherosclerotic cases of AMI. All these patients are older than 20 years and only one female patient of 30 years by Abid *et al*.[2]

However, prognosis seemed to be also affected by angiographic picture and by the occurrence of revascularization. The absence of significant coronary stenosis and presence of healthy coronary arteries was associated with a better prognosis.

CONCLUSION

Although rare, considering diagnosis of AMI in teenagers presenting with chest pain can avoid catastrophe. Risk profile is different in young AMI. Our two cases have two different etiologies for AMI. One is with CAE and another with OC pills. Arterial thrombosis is less common than venous with OC pills. Our female case had suffered AMI possibly secondary to arterial (coronary) thrombosis with OC pills and to the best of our knowledge youngest female patient with AMI.

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