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Case Report

Acute Myocardial Infarction in a Juvenile Patient: A Case Report

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Abstract

This case report involves a 14 years old boy who was diagnosed with Acute Myocardial Infarction (AMI). He developed acute chest pain while in his sleep, which persisted for more than 20 minutes, and upon examination ST elevation in ECG and signs of positive of myocardial injury were found. However, coronary angiography showed normal results. This paper aims to emphasize the significance of clinical examination in diagnosing AMI in juvenile patients thereby reducing adverse cardiac events.

Keywords: Juvenile; Acute Myocardial Infarction; Coronary Spasm

Case Report

Acute Myocardial Infarction (AMI) is the most serious disease threatening human life [1]. It is most prevalent among males in middle age, although females become more susceptible than males after menopause. There are now increasing reports of AMI in younger subjects, which suggests the possible involvement of changing lifestyle and increasing social and psychological burden. In adolescents, AMI is quite rare but the present report on a 14 years old who presented to our hospital with signs of what appeared to be AMI in August 2016, indicates that it may be on the increase.

The 14 years old (born in June 2002) presented with a history of sudden and intermittent chest pain that started 3 days prior while in his sleep and progressively increased in severity with subsequent episodes. The last episode was experienced in his sleep 4.5 hours before presentation. Each episode lasted about 40 mins and spontaneously subsided. No referred pain, sweating or dizziness. The patient's ECG results from a local hospital showed: Sinus rhythm and ST segment elevation (0.1 mv to 0.2 mv) in lead II, III, avF and V7-V9, and positive cardiac Troponin T (TnT). The patient was then transferred to the emergency department of our hospital, where tests showed the same ST segment elevation in lead II, III, avF and V7-V9 on ECG, and TnT of 1.124 ng/ml (over the threshold of 0.1 ng/ml, and almost reaching the maximal level of 2 ng/ml). An emergency CAG was done on the patient, who had had a history of viral myocarditis four years earlier that was treated successfully, but the result were normal. The patient had no other disease, nor did he have allergy to any food or medicine. He had no history of smoking and alcohol consumption, and no family history of coronary heart disease and myocarditis. Physical examination revealed: T: 36.4°C P: 69 b/min R: 18 b/min BP: 139/77 mmhg BMI: 21.5, conscious and fully oriented and normal head, neck, heart, abdomen and pulmonary and nervous systems. ECG showed sinus rhythm, with ST elevation (0.1 mv to 0.2 mv) in lead II, III, avF and V7-V9, while routine and biochemical examination results showed WBC 9.15 × 10^9/L, NEUT 81.11%, RBC 4.69 × 10^{12}/L, PLT 255 × 10^9/L, HGB 135 g/L, Bun 4.04mmol/L, Cr 56.70mmol/L, UA 400.10mmol/L. Myocardial injury biomarkers: 4.5 hours after last episode of chest pain: CK 853.00 U/L (normal range 50-170 U/L), CK-MB 81.00 U/L (normal range 0-16 U/L), AST 71.00 U/L (normal range 0-60 U/L), TNT 1.124 ng/ml; 12 hours after last episode of chest pain: CK 770.00 U/L, CK-MB 55.00 U/L, AST 90.00 U/L; 17 hours after last episode of chest pain: CK 683.00 U/L, CK-MB 41.00 U/L, AST 57.00 U/L. Echocardiography done 12 hours after last episode of chest pain showed normal heart chambers, ventricular wall thickness and movement, heart valves, aorta and pulmonary artery diameters. However, ventricular septum basal segment myocardial echo was found to be uneven, can explore mottled strong echo. Left Ventricular End-Diastolic Diameter (LVEDD) was 34 mm, and the Left Ventricular Ejection Fraction (LVEF) was 57%. Conclusion: ventricular septum echo abnormalities. After 10 days of hospital

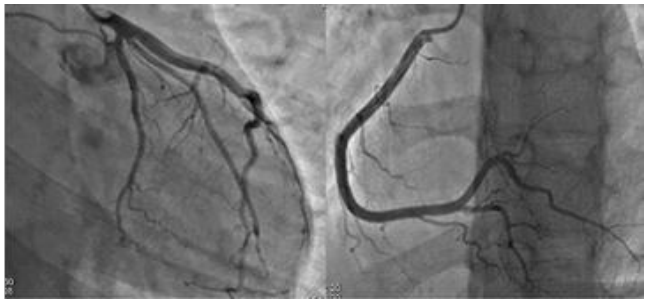

Figure 1: Coronary angiography result (left coronary artery and right coronary artery).

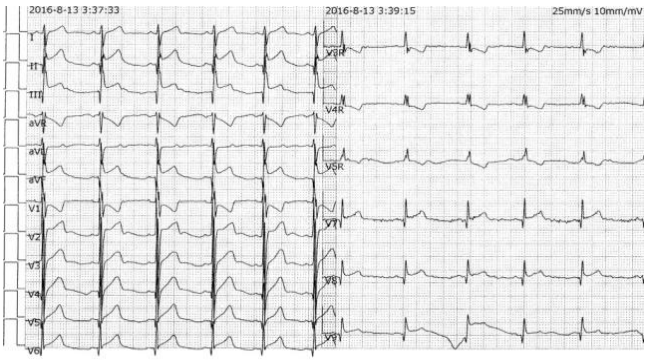

Figure 2: ECG 4.5 hours after last episode of chest pain.

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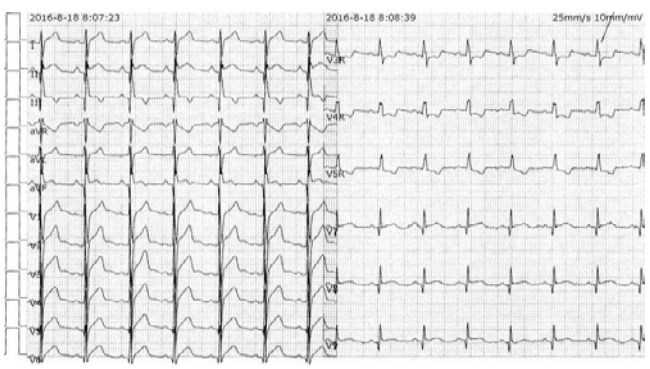

Figure 3: ECG 6 days after last episode of chest pain.

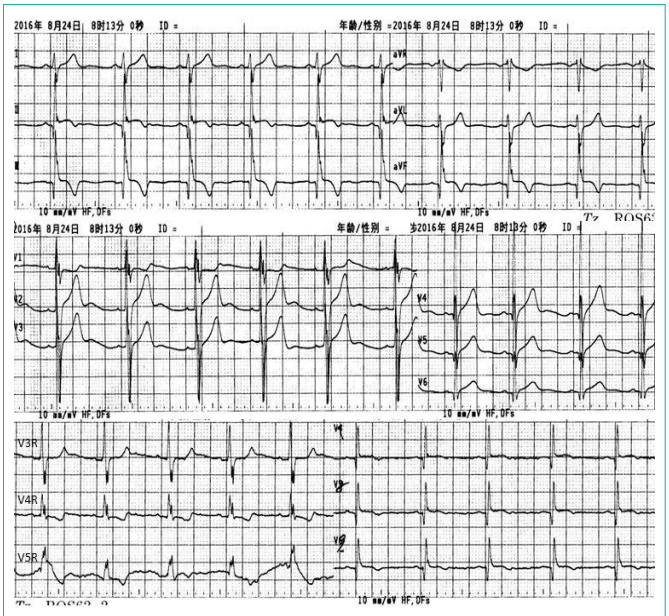

Figure 4: ECG 12 days after last episode chest pain.

admission, echocardiography showed normal findings and the LVEDD and LVEF had improved to 47 mm and 69%, respectively. Conclusion: normal cardiac structure. Clinical diagnosis: coronary heart disease, with acute inferior and posterior wall ST segment elevation myocardial infarction, Killip grade I. In hospital treatment with anti platelet, anticoagulation and others prevented the recurrence of chest pain with overall improvement in patient condition. Eleven days after admission, patient was discharged from hospital (Figure 1).

Discussion

Atherosclerosis is the main etiological factor in the development of coronary heart disease, which often starts around middle age and progresses rapidly to cause clinical symptoms and adverse event. While scientific research suggests that the process of atherosclerosis may start as early as the fetal period, infancy and childhood, juvenile cases develop very rarely because the slow rate of the process often means that it manifests later in adult life as a result of increasing lipid deposits that form part of the atheromatous and fibrous plaque, which ultimately causes vascular stenosis and angina. Furthermore, if the cap of the fibrous plaque is ruptured, released lipids and cytokines can further worsen the problem by causing AMI [2].

In recent years, advanced healthcare means cases of AMI are easily diagnosed and hence the increasing cases that are reported among younger persons. Primary and secondary prevention on a country-wide scale are often aimed at effective disease prevention mostly among the older age group. Among juveniles, the risk factors of coronary heart disease are not as common and as such little emphasis is given to this group. From the analysis of the current patient, the common risk factors of coronary heart disease were not found when he developed AMI, suggesting that closer attention ought to be given to other factors that may predispose adolescents to AMI. The patient also showed some signs that were characteristic of AMI in the young, which contrasts with the presentation in middle and old age. Younger patients often do not have obvious atherosclerosis, and most cases are related to coronary spasm. The current patient's episodic and spontaneously-resolving chest pain at night when in his sleep supports spasm as the cause of the chest pain. The patient did not have any thrombus formation. Moreover, juvenile cases of AMI usually do not present with obvious thrombosis, which has higher chances of spontaneous dissolution compared to older patients. Interestingly, most quinquagenarian AMI patients will have ventricular wall movement abnormalities, enlarged ventricular and reduced LVEF, which this patient also had. Although this patient did not have any serious complications, if his episodic chest pain was left for longer periods, his risks of malignant arrhythmia, heart failure and even sudden death would naturally increase. After relieving his immediate symptoms, CAG showed blood vessels were unobstructed with normal blood flow, although ECG still showed ST segment elevation, which did not completely revert to the equipotential line, and T wave evolution was slow (Figures 2,3 and 4). These features are different from the middle age AMI in which ST segment reverts back to normal with faster T wave evolution after vascular reperfusion.

There have been reports of juvenile AMI [3-5], which have been closely associated with several lifestyle factors including smoking, binge drinking, drugs, obesity, spontaneous or traumatic dissection, malformation of coronary artery, hypertrophic cardiomyopathy, left atrial myxoma , eosinophilia, sickle cell anemia, primary thrombocythemia, systemic lupus erythematosus, Antiphospholipid antibody syndrome, chronic graft versus host disease, Kounis syndrome, pregnancy and surgery. On the contrary, this patient was very young, and did not have any history of flu or related symptoms one month prior to his presentation at the hospital. At the hospital, checks on his immunoglobulins, complement system and antinuclear antibody spectrum showed no abnormal findings. Thus, it is likely that the previous history of viral myocarditis may have predisposed him to AMI.

Upon review of the patient's history, it was evident that he did not visit the hospital after the first episode of chest pain, which reflects the common lack of recognition of AMI signs, especially the juvenile type, among individuals or perhaps the ignorance that it could even occur at such an age. And although chest pains in juvenile patients without tests results like ECG seldom elicit the suspicion of AMI among clinicians, this case is a reminder of the need for complete, immediate and thorough examination and evaluation of patients with chest pain in order to avert such missed diagnosis. The absence of classical risk factors attributable to AMI in this patient also calls for vigilance among

clinicians and the need for implementation of prevention strategies. Thus, higher sense of duty, deeper understanding of cardiovascular diseases, and standardized operating procedures on handling acute cardiovascular events are needed to reduce the occurrence of adverse cardiovascular events.

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