

# Acute myocardial infarction in an adolescent

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## ABSTRACT

يعد احتشاء عضلة القلب من الأمراض الشائعة لدى البالغين، غير أننا نستعرض في هذا المقال حالة نادرة لمريض يبلغ من العمر 15 عاماً فقط. لقد أتى هذا المريض إلى المستشفى بعد إصابته باحتشاء في عضلة القلب وذلك على إثر وجود تاريخ أسري بالإصابة بفرط الدهون ومرض كاواساكي في الشهور السابقة. ولم تفلح محاولات العلاج أو المتابعة في إنقاذ المريض إذا وافته المنية بعد مرور عامين. وبالرغم من ندرة هذه الحالة فإنها تظهر أهمية أخذ تشخيص هذا المرض بعين الاعتبار عند معاينة المراهقين الذين يشكون من ألم معتاد في الصدر وذلك للكشف المبكر عن المرض ومعالجته قبل فوات الأوان.

Myocardial infarction (MI) is known to be common in adults. Interestingly, we report a case of a 15-year-old boy who presented with typical chest pain secondary to myocardial infarct attributable to a combination of familial hyperlipidemia and possible episode of Kawasaki disease in the past. The patient failed treatment and follow-up care, and died 2 years later. Although rare, this case demonstrates that MI should be considered as a diagnosis in adolescents presenting with typical chest pain as early detection, and management is vital for survival.

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Myocardial infarction (MI) is a rare occurrence among adolescents. The occurrence is higher among those aged between 16-18 years compared to those aged between 13-15 years.<sup>1</sup> Myocardial infarction among the young may occur due to hypercoagulable state, coronary artery spasm or accelerated atherosclerosis secondary to familial hypercholesterolemia. Familial hypercholesterolemia is an autosomal dominant disorder resulting in high serum cholesterol, xanthoma and premature atherosclerosis.<sup>2</sup> Rarely, MI may occur due to Kawasaki disease which is an acute vasculitis of unknown etiology affecting infants and children. The typical or classical Kawasaki disease diagnosed as the presence of 5 clinical features which are fever, bilateral conjunctival injection, erythema of the lips and oral mucosa, rash, and cervical lymphadenopathy. The term incomplete or atypical Kawasaki disease is used in the absence of at least 4 of these features and is more frequently associated with coronary artery aneurysms.<sup>3</sup>

**Case Report.** We report a 15-year-old boy who presented 3 hours after sudden onset of severe central chest pain while riding a bicycle. This was the first episode and was associated with sweating. He was a non-smoker, denied substance abuse, and had no significant health problems, such as diabetes or hypertension. However, his mother had a positive history of raised cholesterol. There was a strong family history of premature cardiovascular event, whereby 2 of his maternal uncles had undergone coronary artery bypass grafting (CABG) at the age of 40 years. Parents could not recall any episode of illness suggestive of Kawasaki's disease in the past. Clinically, he was in pain but remained alert, conscious with blood pressure of 125/70 mm Hg, and pulse of 70 beats per minute. Heart sounds and peripheral pulses

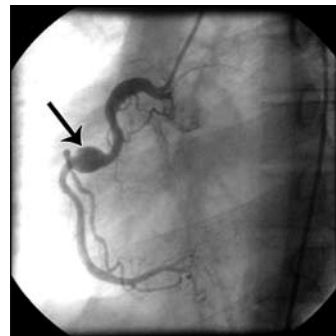
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were normal. Tendon xanthoma and xanthalesma were absent. His body mass index for age was below 85th percentile (22.5). A 12 lead electrocardiogram showed sinus rhythm with ST elevation of 2-3 mm in leads V2, V3, V4, V5 with deep Q in V2 and V3 (Figure 1). Troponin T was positive (0.5 ng/ml) and subsequent serial cardiac enzymes were elevated with peak serum creatinine kinase of 3911 IU/L. Lipid profile on admission showed raised total cholesterol of 9.6 mmol/L, low-density lipoprotein (LDL) of 7.7 mmol/L and normal triglycerides (TG) (1.0 mmol/L) and high-density lipoprotein (HDL) (1.41 mmol/L). Serum homocystine was 12 umol/L (5-15 umol). The venereal disease research laboratory test (VDRL) and anti nuclear factor (ANF) was negative suggesting of the absence of connective tissue disorder as a cause for thrombus formation. Screening blood tests for thrombophilia was negative. Patient was diagnosed as acute ST elevation myocardial infarction (STEMI) with familial hyperlipidemia and managed at the coronary care unit (CCU). He was treated with morphine, aspirin, clopidogrel, beta blocker, statins and streptokinase. Streptokinase was given in a dose

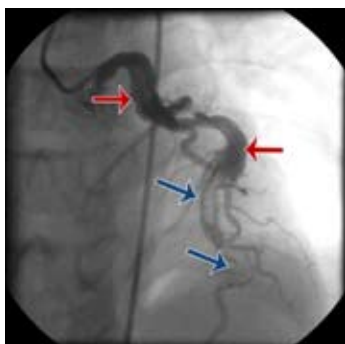
of 1.5 million units over one hour (adult dose). A 2 dimensional echocardiogram revealed hypokinesia of the middle and apical part of the inter-ventricular septum with an ejection fraction of 45%. Thrombus was absent. Coronary angiogram revealed a tortuous, ectatic left anterior descending (LAD) artery with areas of aneurysmal dilatation (9 mm) with luminal irregularities. Thrombus was noted in the proximal and middle segments (Figure 2). Aneurysmal dilatation of 9 mm was also observed in the right coronary artery (RCA) (Figure 3). After angiogram, the patient was anticoagulated with low molecular weight heparin and warfarin. Statin, aspirin, clopidogrel, warfarin and ACE-I were also prescribed. Family screening for lipid profile confirmed familial hyperlipidemia whereby his mother's total cholesterol level was 9 mmol/L while his elder brother's was 9.3 mmol/L. The patient gradually recovered over a week and was discharged. Improvement in lipid levels was observed after 2 months of treatment (total cholesterol 4.5 mmol/L, HDL 1.02 mmol/L, LDL 3.3 mmol/L, TG 0.4 mmol/L). Repeat angiography was carried out, 4 months later revealed a resolution



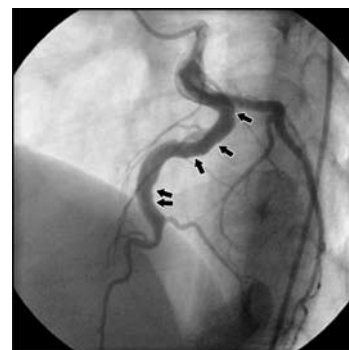
**Figure 1** - The 12 lead electrocardiogram showing sinus rhythm with ST elevation of 2 to 3 mm in leads V2, V3, V4, V5 (red arrows) and deep Q in V2 & V3 (blue arrows).



**Figure 3** - Angiogram showing right coronary artery (left anterior oblique view) with aneurysm (arrow).



**Figure 2** - Angiogram (left cranial view) showing aneurysm (red arrows) and thrombus (blue arrows) seen as filling defect in the proximal and middle segment of left anterior descending artery.



**Figure 4** - Post-treatment angiogram showing left anterior descending artery (left anterior-oblique view) with thrombus clearance and multiple mild stenosis seen as indentations (arrows).

of thrombus and multiple mild stenosis was noted. However, focal areas of significant stenosis were absent (Figure 4). Echocardiogram revealed improvement of ejection fraction from 45-50%. He was continued on treatment with aspirin, clopidogrel, ACE-I and statin. Warfarin was discontinued at family's request. Subsequently, he discontinued medication and follow up care, despite being counseled for lifelong treatment. He eventually collapsed and died while playing football 2 years later.

**Discussion.** Myocardial infarction is a rare entity among adolescents with an incidence of 6.6% events per 1 million patient-year with approximately 80% events occurring in males.<sup>1</sup> Myocardial infarction is mainly a disease of the adult population secondary to atherosclerosis (plaque deposition), rupture of plaque and thrombus formation. However, in children and adolescents, the common underlying cause for MI is secondary to congenital anomalies of the coronary arteries. Other known causes include inflammation, thrombophilia, hypercoagulable states, medications such as amphetamine and substance abuse.<sup>1,4</sup> Adolescents presenting with MI should be inquired into substance abuse and smoking. Cocaine is known to produce vasoconstriction while smoking is associated with endothelial dysfunction.<sup>1</sup> Among the inflammatory causes, Kawasaki's disease, Takayasu's disease, systemic lupus erythematosus (SLE) and Behcet's disease have been implicated.<sup>5</sup> Kawasaki is a vasculitic disease which commonly occurs among children less than 2 years of age. Cardiac complications include coronary artery aneurysm, pericardial effusion, myocarditis and myocardial infarct. Coronary artery aneurysm is a common complication occurring in about 25% of untreated children.<sup>6</sup> However, even among those treated with intravenous immunoglobulin, approximately 10-15% are at risk of developing coronary aneurysms.<sup>7,8</sup> Children less than one year of age with atypical Kawasaki have higher risk of developing coronary artery aneurysm.<sup>9</sup>

Coronary aneurysms are localized dilatation which exceeds 1.5 times the diameter of the adjacent normal segment of artery. Aneurysms secondary to Kawasaki disease commonly occur in the proximal coronary arteries and myocardial infarction occurs most commonly after one year of initial illness. Mortality due to MI secondary to Kawasaki is approximately 20%. The presence of coronary aneurysms in the present case suggests possible undiagnosed Kawasaki's disease during childhood. Positive remodeling secondary to atherosclerosis also results in aneurysm formation;

hence, it cannot be dismissed as a possible cause in this patient. Although atherosclerosis is more prevalent in adult population, accelerated atherosclerosis, which is an entity described for atheroma among the young has been reported in 2% of males aged between 15-19 years. The process of atherosclerosis in the coronary vessels is found to begin early and affects approximately one in 6 adolescents.<sup>10,11</sup> A strong family history of hyperlipidemia, premature cardiovascular events and a personal history of hyperlipidemia in this patient explains the coronary endothelial damage evidenced by multiple mild stenosis on coronary angiography.

Management of these cases with initial percutaneous coronary intervention (PCI) in the presence of large thrombus should be avoided as there is high risk of no reflow or acute closure and stent thrombosis. Complications related to coronary aneurysm include thrombus formation and risk of rupture. In this case, medical treatment with anticoagulants and antiplatelet cleared the thrombus.

In conclusion, the present case illustrates the rare combination of familial hyperlipidemia and coronary artery aneurysm due to atherosclerosis or possible Kawasaki's disease in the past resulting in myocardial infarction. Although myocardial infarct in this age group is most commonly associated with congenital coronary artery anomalies, combination of risk factors such as hyperlipidemia, atherosclerosis and coronary aneurysms are other contributory factors and hence need to be ruled out. Although rare, MI should not be missed in children and adolescents presenting with typical chest pain.

## References

1. Mahle WT, Campbell RM, Favaloro-Sabatier J. Myocardial infarction in adolescents. *J Pediatr* 2007; 151: 150-154.
2. Osula S, Bell GM, Hornung RS. Acute myocardial infarction in young adults: causes and management. *Post Graduate Med J* 2002 ; 78: 27-30.
3. Freeman AE, Shulman ST. Kawasaki disease: summary of the American Heart Association guidelines. *Am Fam Physician* 2006; 74: 1141-1148.
4. Koestenberger M, Nagel B, Gamillscheg A, Temmel W, Cvirn G, Beitzke A. Myocardial infarction in an adolescent: anomalous origin of the left main coronary artery from the right coronary sinus in association with combined prothrombotic defects. *Pediatrics* 2007; 120: 424-427.
5. Safi M, Taherkhani M, Badalabadi RM, Eslami V, Movahed MR. Coronary aneurysm and silent myocardial infarction in an adolescent secondary to undiagnosed childhood Kawasaki disease. *Exp Clin Cardiol* 2010; 15: 18-19.
6. Newburger JW, Takahashi M, Burns JC, Beiser AS, Chung KJ, Duffy CE, et al. The treatment of Kawasaki syndrome with intravenous gammaglobulin. *N Engl J Med* 1986; 315: 341-347.

7. Sundel RP, Burns JC, Baker A, Beiser AS, Newburger JW. Gamma globulin re-treatment in Kawasaki disease. *J Pediatr* 1993; 123: 657-659.
8. Burns JC, Capparelli EV, Brown JA, Newburger JW, Glode MP. Intravenous gamma-globulin treatment and retreatment in Kawasaki disease. *Pediatr Infect Dis J* 1998; 17: 1144-1148.
9. Rosenfeld EA, Corydon KE, Shulman ST. Kawasaki disease in infants less than one year of age. *J Pediatr* 1995; 126: 524-529.
10. McGill HC, McMahan CA, Zieske AW, Tracy RE, Malcom GT, Herderick EE, Strong JP. Association of Coronary Heart Disease Risk Factors with microscopic qualities of coronary atherosclerosis in youth. *Circulation* 2000; 102 : 374-379.
11. Tuzcu EM, Kapadia SR, Tutar E, Ziada KM, Hobbs RE, McCarthy PM, Young JB, Nissen SE. High Prevalence of Coronary Atherosclerosis in Asymptomatic Teenagers and Young Adults Evidence from Intravascular Ultrasound. *Circulation* 2001; 103 : 2705-2710.

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Ma Y, Luo M. Research of glycosylated granulocyte colony-stimulating factor on mobilizing cell regeneration of bone marrow stem cells and repairing injured myocardium. *Saudi Med J* 2010; 31: 634-639.

Panduranga P, Sulaiman KJ, Al-Zakwani IS, Al-Lawati JA. Characteristics, management, and in-hospital outcomes of diabetic acute coronary syndrome patients in Oman. *Saudi Med J* 2010; 31: 520-524.

Ding HS, Yang J. High mobility group box-1 and cardiovascular diseases. *Saudi Med J* 2010; 31: 486-489.