

Twist of Fate of a Modern Day Pheidippides - Young Marathon Runner with Acute Right Coronary Artery Thrombosis

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Abstract

The occurrence of coronary artery disease is rare in young and physically fit individual. Regular exercise reduced incidences of coronary atherosclerotic disease. However, there are reported cases of myocardial infarction and sudden death after heavy and prolonged exercise. We describe an apparent healthy young male marathon runner presented with acute myocardial infarction shortly after completion of the run. Coronary angiography revealed occlusive thrombus in the distal right coronary artery and thromboaspiration was performed. Repeated angiography showed complete resolution of thrombus. The relationship between physical activity and acute coronary thrombosis remain elusive.

Keywords: Marathon runner; Coronary thrombosis; Young; Myocardial infarction

Introduction

The occurrence of coronary artery disease is rare in young and physically active individual. Marathon running is an increasingly popular sport. Sudden cardiac death among marathon runners is 1 event per 100,000 participants [1]. There were reported cases of myocardial infarction after prolonged exercise with coronary artery thrombosis [2,3].

Case Report

A previously fit and healthy 30-year-old male presented to local emergency department, ED of a non-cardiac center seventeen hours after local 10K run with typical chest pain associated with vomiting, breathlessness and numbness over the left upper limb. He had no recent fever or flu like symptoms.

He was working as a pediatric medical officer with no significant past medical history. He did not smoke and denied usage of recreational drugs. He has no family history of coronary artery disease. He was active physically as he ran 10 kilometers (km) for 3 to 4 times a week and participated in half marathon (21 km) every month for the past 2 years. He had body mass index of 25 kg/m².

Upon arrival at ED, he was assessed by emergency physician and revealed stable hemodynamic. Electrocardiogram and serum troponin I at 1 hour and 5 hour were normal. He was discharged home after 6 hours of observation. He experienced persistent chest pain and he presented 36 hours later to a private cardiologist. He had stable vital signs. His serial electrocardiograms (ECG) revealed sinus bradycardia with ventricular rate of 53 beats per minute. There was ECG changes compatible with early repolarization at precordial leads. There were non-pathological Q waves at lead II and aVF and significant Q wave at lead III. Serum troponin T was elevated 859 ng/L (normal range, NR <50). He was referred to national cardiac center for coronary angiogram.

On examination, he had blood pressure of 118/70 mmHg, heart rate of 60 beats per minutes and oxygen saturation of 97% under room air. His cardiovascular and respiratory system examination were normal. Transthoracic echocardiogram displayed preserved left ventricular systolic function of 50% to 55% with hypokinesia at mid and basal inferior region. He had good right ventricular function, intact interatrial septum and no other structural cardiac abnormality.

His ECG prior to coronary angiogram showed sinus bradycardia with ventricular rate of 58 beats per minute, pathological Q in lead III and T inversion and flattened T wave in lead aVF. Serum high sensitive troponin level was 843 pg/ml (NR<14). He was diagnosed with Non ST- Elevation Myocardial Infarction (NSTEMI) with GRACE score of 103.

Coronary angiogram was performed and revealed total occlusion of distal right coronary artery (RCA) caused by thrombus (Figure 1). His left coronary arteries; left anterior descending and circumflex arteries were normal. Multiple thromboaspiration via thrombus aspiration

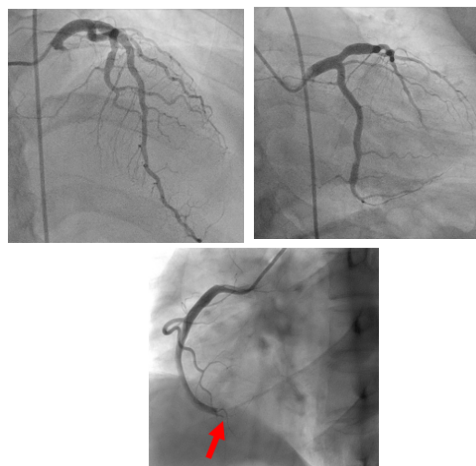


Figure 1: Coronary angiogram upon presentation showing normal left anterior descending artery and left circumflex artery with occluded distal right coronary artery with thrombus.

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catheter; Thrombuster II were performed. Red thrombi were aspirated. Despite coronary flow distal to thrombus was established (Figure 2A), thrombus burden remained high and intracoronary abxiximab 0.25 mg/kg bolus was given. He was transferred to coronary care unit (CCU) for continuation of treatment; Aspirin 100 mg daily, Clopidogrel 75 mg daily, subcutaneous Fondaparinux 2.5 mg daily and Atorvastatin 80 mg nocte.

Laboratory data showed hemoglobin level was 16.1 g/dl; platelet was $300 \times 10^9/L$, total cholesterol 4.7, triglycerides 0.7, LDL-C 3.0, HDL-C 1.4 and fasting blood glucose 3.3 mmol/L. Serial troponin levels were elevated with peak level of 1293 pg/ml on day 2 admission.

On day 6 admission, coronary angiogram was repeated and revealed persistent thrombus at distal RCA with distal coronary TIMI 3 flow (Figures 2B and 2C). Thrombus aspiration was done using Medtronic Export Advance Aspiration Catheter. Intracoronary Abxiximab 0.25 mg/kg bolus was given in view of large thrombus.

He remained well and asymptomatic after repeat coronary angiogram and discharged home. Coronary angiogram was scheduled

in 2 months which shown resolution of thrombus in RCA with TIMI 3 flow (Figure 3). Stent was not implanted in view of no stenosis of the said artery. He was subsequently reviewed in outpatient cardiology clinic and he has no angina with functional class I. Thrombophilia screening result was normal and he was enrolled into cardiac rehabilitation program.

Discussion

Regular physical exercise has long been advocated in therapeutic lifestyle changes for optimal cardiovascular health. Physical activity exerts many physiological benefits in the prevention and management of many chronic diseases including coronary heart disease [1]. However, there are contradicting evidences to suggest heavy physical exertion may potentially trigger the onset of myocardial infarction and sudden cardiac death [4].

Sudden cardiac death is rare among marathon runners with 1 event per 100,000 participants [1]. The cardiac death rate for the London marathon is 1 in 80,000 finishers [5]. It is suggested that prolonged strenuous exercise such as marathon running may transiently increases the absolute and relative risk of sudden cardiac death especially in previously sedentary individuals. A haemostatic imbalance with prothrombotic effects include *in vivo* platelet activation and elevated inflammatory biomarkers during the race may lead to acute cardiac exertional event due to atherothrombosis and shear stress plaque rupture [6].

Acute coronary thrombosis has been described in several marathon runners immediately after completing the 2011 Boston Marathon [2]. Our case illustrates a fit, young seasonal marathon runner presented with acute occlusive coronary thrombosis which was successfully treated with thromboaspiration without balloon angioplasty and stenting. He had no cardiovascular risk factor and structural heart disease. Among previously asymptomatic adult, evidence of acute coronary plaque disruption; including plaque rupture or erosion with acute thrombotic occlusion is common [7]. Vigorous exercise also could provoke acute coronary thrombosis by deepening existing coronary fissure, augmenting catecholamine induced platelet aggregation or both [7]. Intravascular ultrasound (IVUS) would be useful in our case to demonstrate coronary plaque or fissure.

The relationship between physical activity and acute coronary thrombosis remain elusive and derives hypothesis for future research in the field of sports cardiology and translational science.

Conclusion

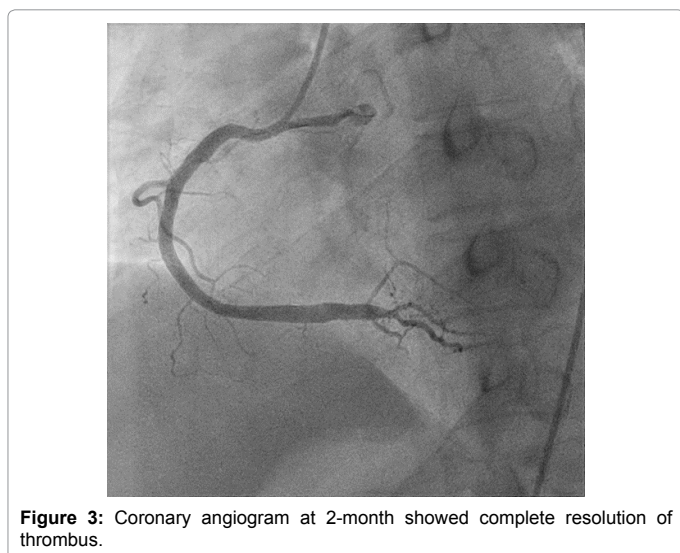
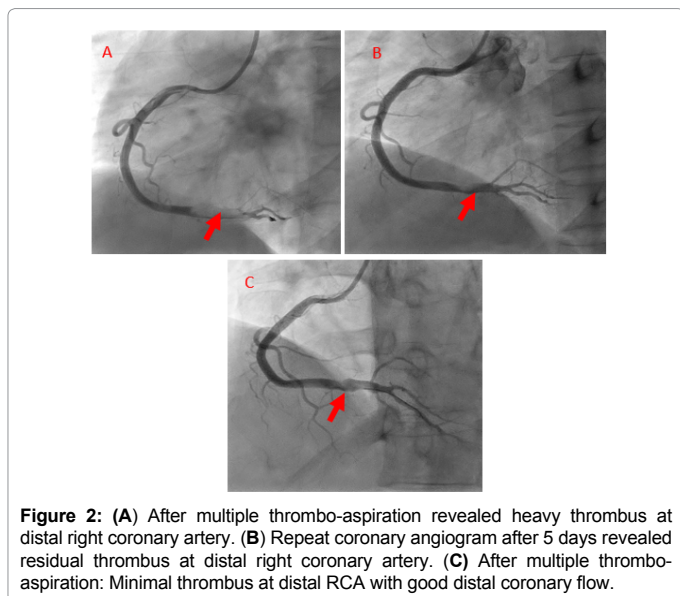
Marathon running is a prevalent sport indulge not only by athletes but “weekend warriors”. Myocardial infarction secondary to acute coronary thrombosis may result in sudden cardiac death. It is therefore important to educate the runner about symptoms of coronary heart disease and to seek urgent medical attention. It is also a call to outline screening and preventative strategies for these apparent healthy and fit individuals and “weekend warriors” embarking on physical activities.

Conflict of Interest

Nil.

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