Medical Research (2022), **V4N3**, 1–7
doi:https://doi.org/10.6913/mrhk.040301

Medical Research

ARTICLE

A Case of Exercise-Induced Coronary Thrombosis in a Young Adult

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(Received 25 May 2022; revised 29 May 2022; accepted 25 June 2022; first published online 30 September 2022)

Abstract

This case suggests that attention should be paid to the effect of physical exercise on the time of occurrence of cardiovascular events. Exercise is a double-edged sword. On the one hand, acute exercise may be the direct cause of thrombotic events; on the other hand, exercise training can reduce the potential risk of cardiovascular events. This case suggests that appropriate studies are needed to determine the effects of long-term and short-term exercise, exercise time and exercise intensity on platelet aggregation and activity, thereby reducing the occurrence of cardiovascular events.

Keywords: Thrombosis,Physical exercise,Coronary thrombosis,Coronary angiography

1. Introduction

A 31-year-old man was admitted to the Department of Cardiovascular Medicine in our hospital on July 6, 2021 due to "dull pain in the middle and lower parts of chest for 17 hours". The patient began to experience an obtuse pain in the middle and lower sternum 17 hours ago (from 23:00 on July 5, 2021). The tolerable condition lasted for about 5 hours and then was gradually relieved without radiation to other parts, accompanied by nausea, no vomiting, no palpitations, shortness of breath, dyspnea, dizziness, no blackout and syncope, and tearing pain. The patient used to be healthy and denied any history of hypertension, diabetes, coronary heart disease or dyslipidemia. He also reported no history of smoking or alcohol intake and no family history of premature coronary heart disease. The patient works as a programmer. He ran 5 km in four hours prior to the onset of illness.

1.1 Physical examination

Temperature: 36.8 °C, pulse: 87 beats/min, respiration: 20 times/min, blood pressure: 126/82mmHg (1mmHg = 0.133kpa). Jugular veins were not observed to be distended. No dry or moist rales were heard in both lungs, and no pleural friction rub was heard. No precordial prominence and no abnormal pulsation were detected. His apical pulse was palpated to be located in the left fifth intercostal space, 1 cm inside the midclavicular line, with strong power, no elevating sensation, no friction sensation and fremitus. The relative cardiac dullness was normal in size. The heart sounds were normal with a heart rate of 87 beats/min, and the cardiac rhythm was regular; no murmurs were heard in any of the valve areas, and no pericardial friction rub was detected. There were no alternating pulses, water-hammer pulses and Durozxiez signs. His abdomen was soft without tenderness or throbbing pains, and his liver and spleen underneath the ribs were not palpable. Bowel sounds were 4 times/min. No edema of lower limbs was observed, and the dorsal arteries of feet were detected to pulse well.

1.2 Auxiliary examination

ECG (performed during the outpatient visit at 07:49 on July 6, 2021)

1. Sinus rhythm 2. Roughly normal ECG

Combined examination of acute myocardial infarction (at 10:39 on July 6, 2021): Troponin I (cTn-I) 2.0805 ng/mL, (<0.032), creatine kinase-MB (CK-MB) 20.40 ng/mL (<5.10), myoglobin (Mb) 144.40 ng/mL(<154.90).

Echocardiography (at 11:34 on July 6, 2021): No obvious abnormalities observed in the intracardiac structure. Mild mitral and aortic regurgitation. Normal range of left ventricular systolic and diastolic function.

2. Clinical diagnosis:Acute non-ST segment elevation myocardial infarction? Killip class I sinus rhythm

After admission, the patient was treated with Ticagrelor and aspirin for antiplatelet therapy, Clexane for anticoagulation and Rosuvastatin for lipid regulation and plaque stabilization. He received the coronary angiography at 11:00 on July 7, 2021. The results showed no significant stenosis of right coronary artery in the whole course and distal TIMI flow grade 3. No significant stenotic lesions were observed in the left main coronary artery and its ostium, a thrombus shadow was identified on the distal segment of left anterior descending coronary artery (Figure 1), and distal thrombolysis in myocardial infarction (TIMI) flow grade was detected at 2-3; no significant stenotic lesions were observed in the left main circumflex artery and its branch with distal TIMI flow grade 3. 2000U of heparin was given additionally, the guiding tube was replaced to the left coronary ostium, and Tirofiban 0.6 mg was given by intracoronary injection, that is, it was maintained by intravenous pumping at 0.25 mg/h. The reexamination by angiography suggested that TIMI flow in the distal anterior descending coronary artery was restored to grade 3. After standardized anticoagulant and antiplatelet therapy, the patient was reexamined by coronary angiography on July 15, 2021. The whole vascular intima of the right coronary artery was observed to be smooth without significant stenosis, and distal TIMI flow grade was detected at 3. The left

coronary artery showed no significant abnormalities, the vascular intima of the left anterior descending coronary artery was smooth, no thrombus shadow was observed on the distal segment (Figure 2), and distal TIMI flow grade was detected at 3.

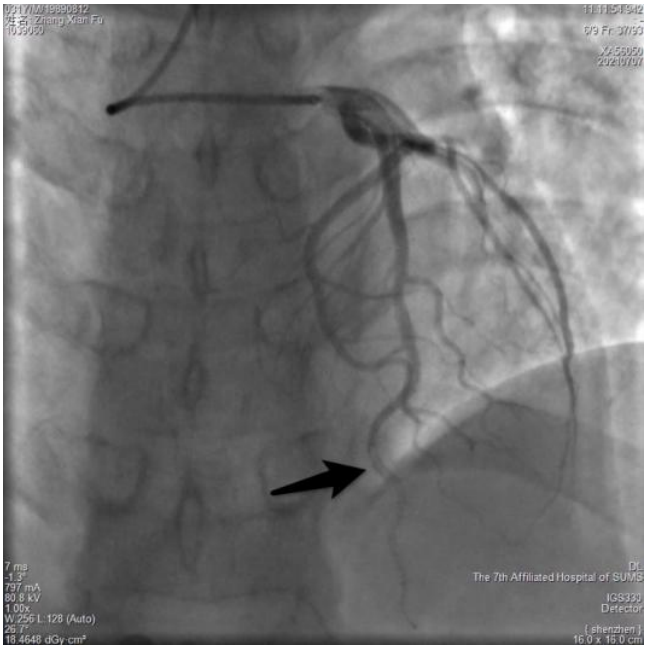

Figure 1

3. Discussion

There are data in the literature suggesting that physical exercise can help to reduce the risk of cardiovascular events [1]; however, physical exercise is considered to be a double-edged sword as it can induce a thrombotic environment[2]. This patient is a young man without common high risk factors of coronary heart disease. He took strenuous physical exercise before the onset of illness and felt retrosternal pain after 4 hours of exercise. The ECG after admission reported no abnormality and dynamic changes in troponin T (Table 1), which was considered to be non-ST segment elevation myocardial infarction. The coronary angiography showed a thrombus shadow on the distal segment of the left anterior descending coronary artery (Figure 1), so the diagnosis was established. Most coronary thrombi occur secondary to plaque rupture. This case was reexamined by coronary angiography after standardized anticoagulant therapy and observed to have smooth coronary walls (Figure 2). In order to exclude other underlying diseases leading to coronary thromboembolism, such as patent foramen ovale, antiphospholipid antibody syndrome, malignant tumors, liver disease, myeloproliferative diseases and paroxysmal nocturnal hemoglobinuria, we also improved the corresponding examinations and tracked the abnormal results (Table 2). Combining the patient's clinical manifestations to exclude various secondary factors, intracoronary thrombosis the patient's medical history, we considered strenuous exercise before the illness to be the predisposing factor.

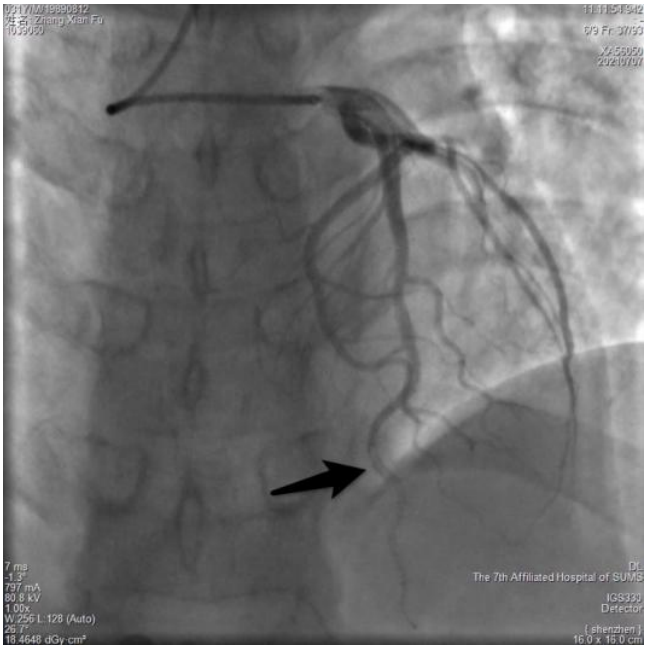

Figure 2

Table 1 Dynamic reexamination of markers for myocardial infarction

Examination indicators (Reference values)	7/6/2021	7/7/2021	7/9/2021	7/14/2021	8/14/2021
Myoglobin(<154.9ng/ml)	36.5	42.3	36.5	27.6	43.9
Creatine kinase-MB © (<5.10ng/ml)	2	13.5	2	0.4	0.4
Troponin I(<0.0320ng/ml)	1.8286↑	4.0889↑	1.8286↑	0.0406↑	0.0022

Regular exercise can reduce the risk of major cardiovascular thrombotic events[3], but an intense physical activity may transiently increase the risk of acute myocardial infarction or stroke during exercise[4]. It is reported that this increased risk would last until 2 hours after exercise[5]. According to the literature review analysis by Line Nørregaard Olsen et al[6], smoking, unhealthy diet and lack of physical activities are the risk factors for arterial thrombosis, and the risk of myocardial infarction increases significantly for sedentary people after strenuous exercise. Another important point is that for sedentary individuals, the risk of myocardial infarction increases by 50–100 times after strenuous exercise, while that for people who exercise regularly is much lower[7]. This suggests that the intensity and frequency of exercise may influence the susceptibility to thrombosis. According to the

researches by Buckley et al[8], the increased risk of myocardial infarction is associated with high physical exertion, and the risk of developing myocardial infarction after strenuous exercise increases by more than 5 times. This may be related to exercise-induced activation of the body 's coagulation system.

Table 2 Dynamic serological reexamination results of the patient

Examination indicators (Reference values)	7/6/2021	8/17/2021	1/12/2022
Thrombophilic indicators			
Antithrombin III (75-125%)	69.4↓	83.7	87.2
Protein C activity (70-140%)	91	99.4	113.1
Protein S activity (75-130%)	85.3	182.0↑	88.3
Primary screening test of lupus anticoagulant (31-44 seconds)	30.7↓	103.6↑	33
Determination test of lupus anticoagulant (30-38 seconds)	30.8	59.7↑	31.1
Primary screening of lupus/Confirmation of lupus (0.8-1.2))	1	1.74↑	1.06
Anticardiolipin antibody assay			
Anti-β2-glycoprotein 1 antibody (0-20 U/ml)	33.48↑	24.20↑	< 2.00
Anticardiolipin antibody lgA assay (negative)	negative	negative	negative
Anticardiolipin antibody lgG assay (negative)	negative	negative	negative
Anticardiolipin antibody lgM assay (negative)	negative	negative	negative

Regular exercise can reduce the risk of major cardiovascular thrombotic events[3], but an intense physical activity may transiently increase the risk of acute myocardial

According to the markers on exercise and platelet/platelet activation researched by Petidis et al[13], fibrinogen increases after strenuous exercise. Kestin et al[14] observed that platelet activation and reactivity increased in sedentary subjects after standard treadmill exercise, which was significantly different between the two groups compared with the physical exercise group. Vascular blood flow and blood velocity increase during the strenuous exercise, resulting in increased vascular shear stress [9, 10]. High shear stress levels can damage endothelium and promote intravascular thrombosis[11]. Sympathetic stimulation during the strenuous exercise leads to significantly increased release of catecholamines. Excessive catecholamines can cause coronary vasospasm, activate platelets and promote intravascular thrombosis[12].

4. Conclusion

This case suggests that attention should be paid to the effect of physical exercise on the time of occurrence of cardiovascular events. Exercise is a double-edged sword. On the one hand, acute exercise may be the direct cause of thrombotic events; on the other hand, exercise training can reduce the potential risk of cardiovascular events. This case suggests that appropriate studies are needed to determine the effects of long-term and short-term exercise, exercise time and exercise intensity on platelet aggregation and activity, thereby reducing the occurrence of cardiovascular events. For people with a high cardiovascular risk or those who exercise infrequently, they are advised to start exercise at low to moderate intensity to reduce the risk of thrombosis.

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