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Case Report

Acute Coronary Syndrome in Well Trained Young Adult: A Rare Cases

A. A. Indah[1*], P. V. Ramadhan[2], V. Pratama[3], Haikal[3], and Irnizarifka[4]
[1]*General Practitioner, Department of Cardiology and Vascular Medicine, Indonesia Army Central Hospital Gatot Soebroto, Jakarta, Indonesia.*
[2]*General Practitioner, Emergency Department, Tengku Rafi'an Hospital, Riau, Indonesia.*
[3]*Cardiologist, Department of Cardiology and Vascular Medicine, Indonesia Army Central Hospital Gatot Soebroto, Jakarta, Indonesia.*
[4]*Cardiologist, Department of Cardiology and Vascular Medicine, Sebelas Maret University, Solo, Indonesia.*

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**Corresponding author:*
aprilia.aqmarina@gmail.com

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ABSTRACT

Acute Coronary Syndrome (ACS) has been observed in the older population (>40 years old), sedentary lifestyle and unhealthy behavior. Recently, there is an increase of ACS reported in the younger population even without a sedentary lifestyle. We report a case of a well-trained 25-years old man that came with late presentation of ST elevation myocardial infarction (STEMI) occurred 10 minutes after vigorous exercise. His traditional risk factor was a smoker. Electrocardiogram (ECG) showed a pathological Q wave in anteroseptal lead and T wave inversion in anterior lead with elevated cardiac biomarker. Primary percutaneous coronary intervention (PPCI) was performed due to prolonged chest pain and it showed subtotal occlusion in mid left anterior descending artery (LAD) with high thrombus burden. Drug eluting stent (DES) was implanted at mid LAD and GPIIb/IIIa inhibitor and low molecule weight heparin was given with good clinical result.

Introduction

Acute Coronary Syndrome (ACS) is well known as the most likely to occur after 45 years of age especially in patients with high cardiovascular risk [1]. The incidence of ACS in young adults is increasing recently especially in people with cardiovascular risk such as smoking, dyslipidemia, diabetes, and family history of ACS.[2]

However, risk of acute coronary event in young adult, can increase in healthy people with vigorous physical activity even without cardiovascular risk.[3]

Case Presentation

A 25-years old man and well-trained was referred from District Hospital to Gatot Soebroto Army Central Hospital with typical chest pain, diaphoresis, nausea and vomiting that lasted for 18 hours before hospitalization. These symptoms occurred 10 minutes after doing vigorous exercise for one hour in the afternoon such as running, push up and pull up. One month earlier, he had the same symptoms after exercise which relieved by rest. His traditional risk factor was smoking. He routinely exercises such as running, sit up and push up 4-5 times a week for 1 hour for the last 8 years**.** He also performed weight lifting exercise at gym as daily activities. There was no history of alcohol consumption nor family history who had a sudden cardiac death and acute coronary syndrome.

Physical examination within normal limit with no sign of acute heart failure, his body mass index (BMI) was within the normal limit. The chest X-Ray also showed cardiomegaly. Electrocardiogram (ECG) showed sinus bradycardia 50bpm, Q wave in lead V1-V3 with T wave inversion in lead V1-V6 (Figure 1).

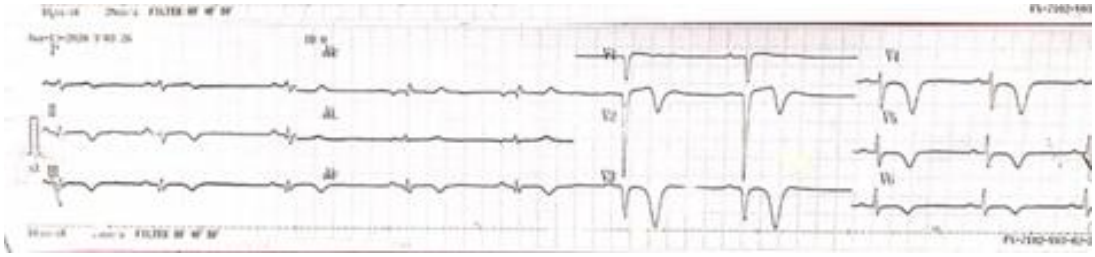

Figure 1. The ECG at Emergency Room

Patient was then diagnosed as acute ST-elevation Myocardial Infarction (STEMI). He was given a standard protocols medication such as loading dose of asetylsalisilatacid (ASA) 160 mg, loading dose of clopidogrel 300 mg and sublingual nitrate. Cardiac biomarker was increased (CKMB 15 mg/dl, CPK 112 mg/dl, Troponin I 0.13 ng/ml).

He had prolonged chest pain, and we performed primary percutaneous coronary intervention (PPCI). The result showed subtotal occlusion in mid left anterior descending (LAD) with organized thrombus from mid-distal. TIMI flow was 0-1, other coronary artery was normal. Drug eluting stent (DES) was implanted at the mid part of LAD. We use GPIIb/IIIa inhibitor with Eptifibatide Intra Vein (IV) were given due to the high thrombus burden and it showed TIMI III flow (Figure 2).

Echocardiography showed decreased contractility left ventricle (LV) function with ejection fraction (EF) 43 % (Simpson), concentric left ventricular hypertrophy (LVH), with akinetic in mid septal and apex, hypokinetic at mid anterior and anteroseptal, normal valves. There was thrombus in LV apex (Figure 3).

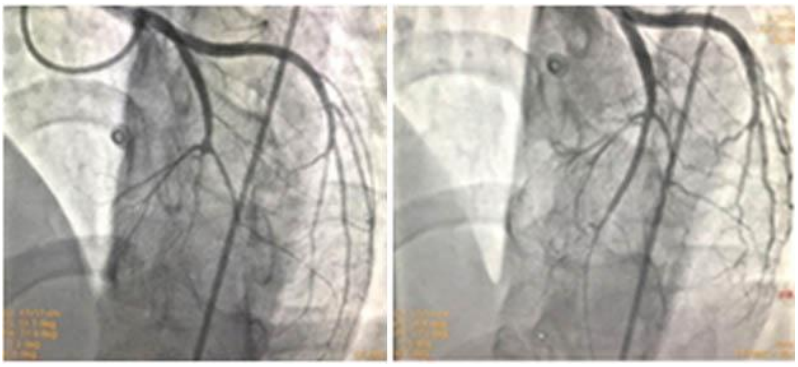

Figure 2. Primary Percutaneous Coronary Intervention

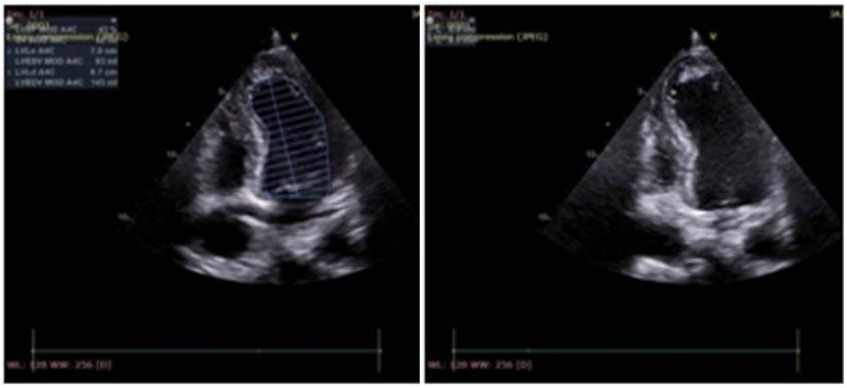

Figure 3. Echocardiography

Then, patient was clinically stable, he had no chest pain nor dyspnoea and treated by Enoxaparin 0.6ml/12 hours subcutaneous (SC), Ticagelor 90 mg/12 hours, ASA 80 mg/24 hours, Atorvastatin 40 mg/24 hours, warfarin 2 mg/24 hours, Laxative 10 ml/24 hours as daily medicine. ECG showed no evolution. Laboratory result showed normal lipid profile (Triglyceride 89mg/dl, Cholesterol 165 mg/dl, HDL 40 mg/dl and LDL 107 mg/dl), and normal fasting glucose result (Glucose 89mg/dl).

Discussion

Acute Coronary Syndrome (ACS) is known globally as a major cause of disability and death. Based on international Survey of Acute Coronary Syndrome in Transitional Countries, incidence of ACS in young age was 6% which is associated with a sedentary lifestyle [4]. Regular physical activity is proven to delay the atherosclerosis process and reduce coronary heart disease incidence.[5] But vigorous physical activity could also lead to acute Myocardial Infarction (MI) and Sudden Cardiac Death (SCD) in susceptible populations [6].

In general physical activity can be categorized according to metabolic equivalent (METs). METs is a useful, convenient and standardized way to describe the absolute intensity of a variety of physical activities. Intensity of physical activity could be divided according to the METs category into light intensity (2.0-2.9), moderate (3.0 - 5.9), and vigorous (≥ 6.0) [7]. Depending on the various previous experience and the fitness level of each individuals, the intensity of different forms of physical activity might also vary.

Vigorous exercise is defined as an absolute exercise work rate for at least 6 metabolic equivalent (METs) (Table 1) [8]. Vigorous exercise could increase the risk of cardiovascular events during or immediately after exercise and it happens frequently in the afternoon or in early evening [9]. Vigorous-intensity-exercise or high-intensity-exercise is a physical activity which requires a large amount of effort resulting in a higher heart rate, and rapid breathing, such as jogging or running [10]. In this case, the patient he routinely do the vigorous exercise as a daily exercise and as a daily work, and it was defined as METs >6.

The American Collage of Sport Medicine – American Heart Association (ACSM-AHA) Primary Physical Activity Recommend that all healthy adult age 18 to 65 years old should participate in vigorous intensity aerobic activity for a minimum of 20 minutes on 3 days a week or moderate intensity aerobic Physical Activity for a minimum of 30 min on 5 day a week [11].

Some studies estimated that an individual's risk of AMI onset was transiently increased 5-fold within 2 hours of an episode of vigorous exercise (METs ≥ 6) and 1.6-fold within 2 hours of moderate exercise compared lower levels of exercise [12]. Interestingly, compare with indoors activity, strenuous outdoor activity was more associated with an increase of AMI symptom onset [12]. Vigorous intensity exercise has a small but measurable acute risk of cardiovascular complication. Therefore, it is important to mitigate this risk in susceptible individuals.

Table 1. METs by the Physical Activity

Physical Activity	MET
Light Intensity Activities	<3
Sleeping	0.9
Watching television	1.0
Writing, desk work, typing	1.8
Walking 1.7 mph (2.7km/h), level ground, strolling, very slow	2.3
Walking 2.5 mph (4km/h)	2.9
Moderate intensity activities	3 to 6
Bicycling, sttaionary 50 watts, very light effort	3.0
Waling 3.0 mph (4.8km/h)	3.3
Calisthenics, home exercise, light or moderate effort, general	3.5
Walking 3.4 mph (5.5km/h)	3.6
Bicycling, <10mph (16km/h), leisure, to work for pleasure	4.0
Bicycling, stationary, 100 watts, light effort	5.5
Vigorous intense activities	>6
Jogging, general	7.0
Calisthenics (push-up, sit-up, pull-up, jumping back), heacy vigorous effort	8.0
Running, jogging, in place	8.0
Rope jumping	10.0

The exact mechanism in which vigorous activity led to AMI was poorly understood. But it is thought to be a triggering mechanism including increased wall stress, coronary spasm, the thrombotic occlusion. The increase of wall stress is causing an increase of the heart rate and blood pressure [3]. Vigorous exercise could also trigger the coronary spasm of the diseased artery segment and increase the flexing of atherosclerotic epicardial coronary arteries which lead disruption of plaque and thrombotic occlusion [13]. The other mechanism is thrombosis by deepening existing coronary fissure, augmenting catecholamine-induced platelet aggregation.[14]

Mildly fissure coronary plaque requires some exacerbating event to induce coronary thrombosis [14] not despite an increases of myocardial oxygen demand, Vigorous exercise can also induce simultaneously shortening diastole and coronary perfusion time resulting in myocardial ischemia.[15]

La Gerce et al. (2012) found that youngster who had a greater prior exposure to vigorous exercise will develop a fibrosis and scarring in the interventricular septum. Cardiac-MRI can show the patchy scanning and myocardial fibrosis. Myocardial fibrosis is a result of the extreme training with more than five-years of continuous intense training.[16]

In some studies, vigorous training could also lead to cardiac hypertrophy. The physical training defines into dynamic and static. Both of them lead into two different kinds of chronic cardiac pressure that can induces morphological changes of the heart, such as eccentric and concentric physiological cardiac hypertrophy.[17]

A dynamic or endurance training such as swimming, running, cycling (or any other aerobic exercise) changes the hemodynamic by increasing heart rate and stroke volume. Furthermore, endurance exercises increase the skeletal muscle pump and venous return to the heart resulting eccentric left ventricular hypertrophy due to overload of volume. It is characterized by an increase of cardiac cell length and mass with increased chamber volume [18]. Based on Mc-Ardle, et al. (2006), aerobic exercise leads an increase of the average cardiac output from 5 litres per minute to a maximum of 35 litres per minute. This change put a strain on the heart causing the dilatation of right ventricle (RV) and right atrium (RA).[19]

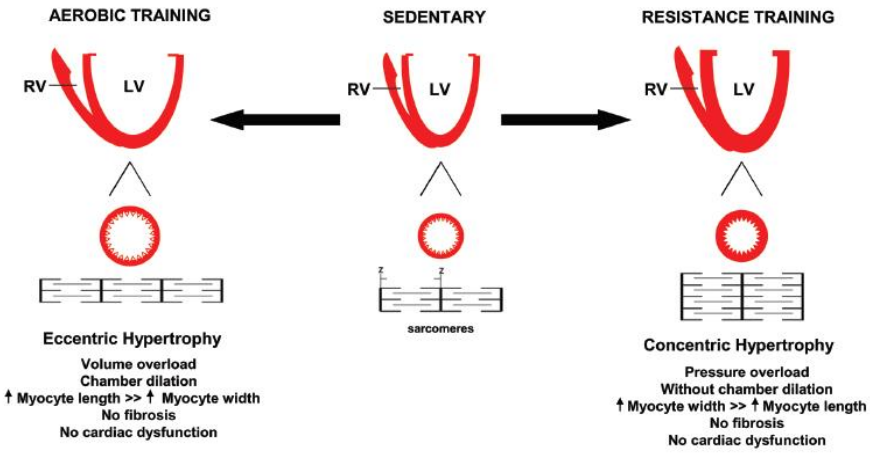

Figure 4. Cardiac Remodelling due to particular training [17]

On the other hand, the static training (e.g., Bodybuilding, wrestling, hammer throwing, and weight lifting) with no or less movement can develop the strength. This type of activities is also called resistance training which is purposed to strengthen power and muscle. Both cardiac and skeletal muscle has a response to this type of training. This type of training causes an elevation of blood pressure leading to pressure overload in the heart. This training also increase the thickness of left ventricular without reducing the internal cavity's diameter in diastole (concentric hypertrophy).[17]

This type of hypertrophy can be seen in this patient due to his weight lifting activity.

Interestingly, not all people who exercise vigorously lead to myocardial infarction.

It is presumed that cardiovascular risk factors play a part in this matter [6, 19]. The most important risk factor observed and reported in the young adult with MI was cigarette smoking [20]. It is proven that smokers are more susceptible to myocardial infarction compared to non-smokers [21]. It was concluded based on the fact that cigarette smoking has a role in the development of atherosclerosis and vascular spasm by increasing the number of mLDL (which is the key to atherosclerosis formation), platelet aggregation, endothelial dysfunction, inflammation and decreasing number of Nitric Oxide (NO) [22, 23].

Conclusion

Exercise is one of the recommended activities to prevent coronary heart disease. But, exercise vigorously or too much could also lead to myocardial infarction that could happen even in healthy young adults. It is also important to address that a cardiovascular risk factor such as cigarette smoking has a role as catalyst in the development of acute myocardial infarction. We conclude that vigorous exercise combined with cigarette smoking might be the cause of acute myocardial infarction in this young and healthy man.

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