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SELÇUK TIP DERGİSİ

Psikososyal Travma Tarafından Tetiklenen Koroner Arter Spazmının Yol Açtığı Akut Miyokard Enfarktüsü

Acute Myocardial Infarction due to Coronary Artery Spasm Triggered by Psychosocial Trauma

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Özet

Koroner arter spazmı, epizotlar sırasında geçici ST segment elevasyonunun eşlik ettiği spontan göğüs ağrısı epizotları ile karakterize olan iskemik kalp hastalığının özel bir tipidir. Bu sendrom, ani ölüme ek olarak akut miyokard enfarktüsü, ventriküler taşikardi veya fibrilasyon ile ilişkili olabilir. Fizyolojik stres de akut miyokard enfarktüsü için artmış bir risk ile ilişkilidir. Ağır iş temposundan kaynaklanan psikososyal travmadan hemen sonra başlayan, devam eden göğüs ağrısı için acil servise başvuran 23 yaşında bir erkek hastayı sunduk. Başvuru sırasında ne EKG'sinde ne de kardiyak enziminde değişme olmadı, fakat takip edilirken akut ST elevasyonlu MI gelişti. Anjiyografide koroner arterlerde stenoz yoktu. Tipik semptomları göz önüne alındığında ergonovin testi gerekli görülmedi. Ca-kanal blokeri ve nitratlar ile semptomları düzeldi. Hasta bir Ca-kanal blokeri reçete edilerek taburcu edildi ve ileride göğüs ağrısı olmadı. Sonuç olarak, ağır iş yükünden kaynaklanan psikososyal stresden ve sempatik hiperaktiviteden dolayı gelişebilen vazospazmın, bu genç hasta için akut miyokard enfarktüsünün sebebi olduğunu düşünüyoruz.

Anahtar kelimeler: Psikososyal travma, vazospazm, akut miyokard enfarktüsü

Abstract

Coronary artery spasm is a special type of ischemic heart disease characterized by spontaneous episodes of chest pain accompanied by transitory ST segment elevations during the episodes. This syndrome may be associated with acute myocardial infarction, ventricular tachycardia or fibrillation, as well as with sudden death. Psychosocial stresses are also associated with an increased risk for acute myocardial infarction. We reported a 23 years old, male patient who applied to the emergency department for ongoing chest pain which had begun just after psychosocial trauma arising from heavy business tempo. He had neither ECG, nor cardiac enzym changes during admission, however acute MI with ST elevation occurred while following up period. There was no stenosis in coronary arteries on angiography. Given the typical symptoms, an ergonovine test was considered unnecessary. Administration of Ca-channel blocker and nitrates ameliorated his symptoms. The patient was prescribed a calcium channel blocker at discharge and has had no further chest pain. Finally, we thought that the cause of acute myocardial infarction for this young patient that occurred after vasospasm might be due to sympathetic hyperactivity and psychosocial stresses resulting from heavy work load conditions.

Key words: Psychosocial trauma, vasospasm, acute myocardial infarction

INTRODUCTION

Psychosocial stresses are associated with an increased risk of acute myocardial infarction (AMI) (1). Angiographically normal coronary arteries are found in 1% to 12% of patients with myocardial infarction (2). In the presence of normal coronary arteries, young individuals are more likely to have myocardial infarction than older ones. Acute extraordinary stress may trigger AMI. Increased risk for AMI associated with high levels of stress is still significant after adjustment of other cardiovascular risk factors (3). Clinicians may ignore the fact that acute stress may cause AMI especially in young patients without any risk factor for coronary artery disease. We hereby report a young patient with normal coronary arteries who developed AMI after sustaining acute physical and psychological trauma due to heavy work load.

CASE

A 23-year-old man who had experienced heavy psychological trauma at work admitted to the emergency department with excruciating substernal chest pain continuing for about 6 hours and radiating to the left arm. His medical history included no use of over-the-counter drugs, tobacco, or alcohol. His father has had the history of myocardial infarction previously. The results of physical examination were within normal limits. ECG, cardiac enzyme panel and also echocardiographic examination at admission were normal. Laborator tests revealed normal hematocrit, normal electrolytes. Ten hours later, he experienced severe chest pain again in the early morning. The ECG demonstrated 2-3 mm ST segment elevations at high lateral derivations (Figure 1a). CPK-MB was 100.9 ng/mL (normal values = 0.3–4.0 ng/mL) with troponin-I of 14.05 ng/mL (normal values = 0.0–0.04 ng/mL). Intravenous nitroglycerin was administered, ST segment elevation returned to isoelectric line quickly,

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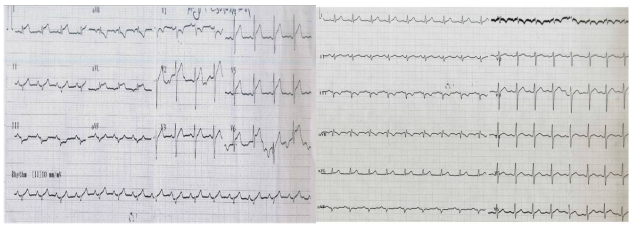

Figure 1 a. Electrocardiogram demonstrating 2 to 3 mm ST segment elevation in DI, aVL,V4-V6 **b.** The same patient's electrocardiogram showing normalized ST segments in the DI, aVL, V5-V6 leads after IV nitroglycerin administration.

and his pain resolved. (Figure 1b). Coronary angiography in the cardiac catheterization laboratory revealed normal cardiac anatomy and no evidence of occlusive disease (Figure 2). Given the typical symptoms, an ergonovine test was considered unnecessary. The presumptive diagnosis of coronary artery spasm was made. The patient remained asymptomatic for the rest of his hospitalization period and was discharged with 240 mg of diltiazem per day. He described a symptom-free period throughout 1-mounth follow-up duration and his cardiac status was stable within 1 month after the myocardial infarction.

DISCUSSION

Severe emotional stress may be responsible for the development of myocardial infarction (4). Emotional stress may trigger AMI and sudden cardiac death in vulnerable patients. The relative risk for acute cardiovascular events during acute stressful situations ranges from 1.82 to 3 (5). Mortality rate ranges from 22% to 34%, with cardiovascular mortality accounting for 92% of deaths associated with acute stress (6). The main physiologic responses to acute psychological stress include increased sympathetic activity, and elevations in heart rate, systolic and diastolic blood pressure, cardiac output, and plasma norepinephrine levels which may cause to excessive vasospasm (7). Systemic vascular resistance also increases during acute mental stress, whereas it decreases during physical exercise. Increased heart rate and blood pressure may augment myocardial oxygen demand and mental stress may also reduce myocardial oxygen supply (8). Several studies have been designed to investigate cardiovascular effects of stress. Intense mental stress enhances platelet aggregation secondary to sympathetic hyperactivity. Endothelial dysfunction caused by acute mental stress may decrease fibrinolytic response, which may further contribute to prothrombotic imbalance in favor of thrombosis (3). Blood flow abnormalities have also been reported. It has been demonstrated that 43% of patients with mental stress exhibit decreased left ventricular ejection fraction and increased peripheral vascular resistance. This decrease in ejection fraction may be related to peripheral vasoconstriction caused by acute stress (9). By positron emission tomography, Arrighi et al (10). demonstrated a blunted augmentation of myocardial blood flow during mental stress. This response was also noted in nonatherosclerotic areas, suggesting an important role of microvascular dysfunction (10). These changes induced by acute intense mental stress may be related to hypersecretion of norepinephrine in the plasma which may cause prolonged coronary vasospasm and subsequent thrombosis. Therefore, acute and extraordinarily intense mental stress causing long-lasting coronary vasospasm may be responsible for this detrimental event in this young patient with normal coronary arteries (11). AMI case reports in young age caused by coronary vasospasm published until now were generally inferior MI ones. Anterior Mİ due to coronary vasospasm is rare. We reported a young patient presented with anterior MI resulting from coronary vasospasm after experiencing heavy psychological stress. Therefore, this possibility should be kept in mind in the evaluation of chest pain in individuals experienced psychosocial stresses.

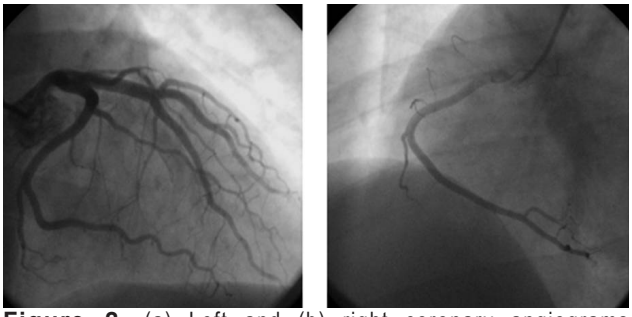

Figure 2. (a) Left and (b) right coronary angiograms demonstrating normal coronary arteries

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