Acute myocardial infarction triggered by acute intense stress in a patient with panic disorder

Panik bozukluğu olan bir hastada ani yoğun stresin tetiklediği akut miyokard infarktüsü

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Psychosocial stresses are associated with an increased risk for acute myocardial infarction (AMI). We report a 35-year-old male patient who developed AMI after acute psychological trauma. He presented with chest pain that began after being involved in a fight. He was extremely agitated. He did not have any risk factors for coronary disease except for panic disorder which was diagnosed six years before. Cardiac enzymes were found elevated. His electrocardiogram (ECG) showed ST-segment elevation in leads V2-6. After thrombolytic therapy with tissue plasminogen activator, his chest pain relieved and ST elevations on ECG regressed. Coronary angiography showed normal coronary arteries. Acute extraordinary stress may be responsible for AMI in this young patient as a result of sympathetic hyperactivity and coronary vasospasm.

Key words: Coronary angiography; myocardial infarction/etiology; stress, psychological/complications.

Psikososyal stres miyokard infarktüsü gelişme riskini artırmaktadır. Bu yazıda, akut psikolojik travma sonrasında akut miyokard infarktüsü geçiren 35 yaşında erkek hasta sunuldu. Hasta, bir kavga sonrasında başlayan göğüs ağrısı yakınmasıyla ve aşırı derecede huzursuz ve endişeli bir şekilde başvurdu. Altı yıl önce tanı konan panik bozukluk dışında koroner hastalık için herhangi bir risk faktörü yoktu. Kardiyak enzimleri yükselmiş bulundu. Elektrokardiyogramda V2-6 derivasyonlarında ST-segment yükselmesi izlendi. Doku plazminojen aktivatörü ile trombolitik tedaviden sonra hastanın göğüs ağrısı düzeldi ve elektrokardiyografide ST yükselmesi gerileme gösterdi. Koroner anjiyografide koroner arterler normal bulundu. Olağandışı yoğun stresin tetiklediği sempatik hiperaktivite ve koroner vazospazm bu genç hastada akut miyokard infarktüsünden sorumlu olabilir.

Anahtar sözcükler: Koroner anjiyografi; miyokard infarktüsü/etyoloji; stres, psikolojik/komplikasyon.

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. The etiology of myocardial infarction in individuals with normal coronary arteries has not been elucidated, despite many factors implicated including coronary vasospasm and concomitant thrombosis, endothelial dysfunction, platelet dysfunction, infectious and noninfectious coronary arteritis, genetic abnormalities, coagulation disorders, collagen tissue disorders, embolization, oral contraceptive use, and vasospastic syndromes.[3-5] Acute extraordinary stress may trigger AMI and stressful life events such as earthquakes, war, threat of attack, and fear of death may precipitate AMI. Increased risk for AMI associated with high levels of stress is still significant after adjustment of other cardiovascular risk factors.[6] 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 on the basis of panic disorder.

CASE REPORT

A 35-year-old male patient presented with a complaint of chest pain that began after getting into a fight with his opponents. He was extremely agitated, diaphoretic,

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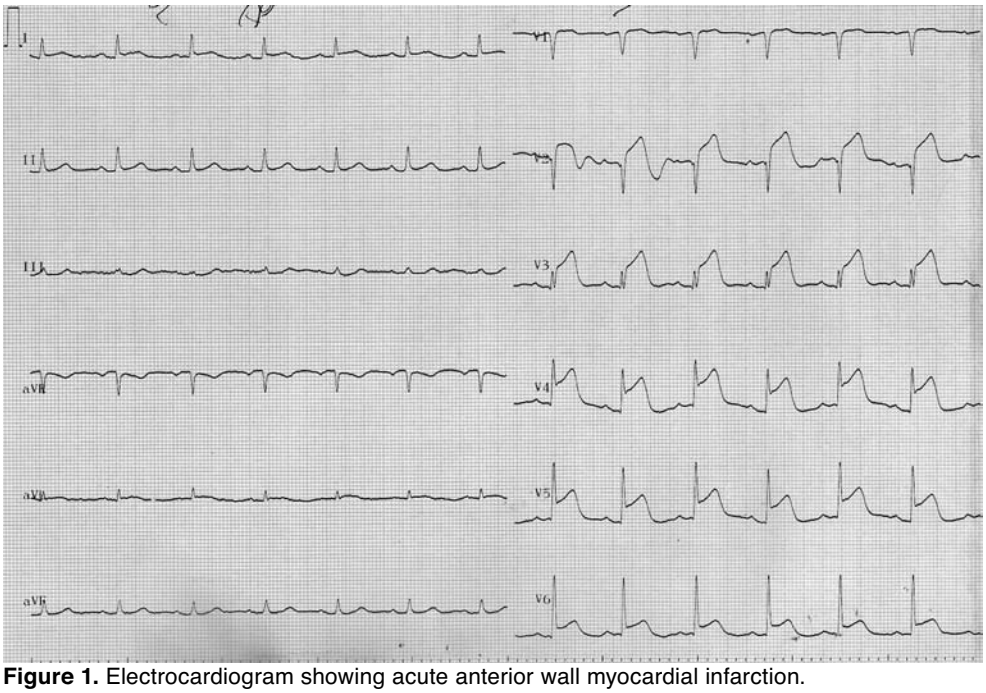

Figure 1. Electrocardiogram showing acute anterior wall myocardial infarction.

and in fear of being killed. He had no history of smoking, use of medications, substance abuse such as cocaine or alcohol, nor a family history of premature coronary artery disease. He was diagnosed as having panic disorder six years before and treated medically, but he gave up taking his medicines after four years of treatment.

On physical examination, his blood pressure and pulse rate were 140/80 mmHg and 105/min, respectively. There were several skin bruises on the face and extremities, and no sign of chest trauma. Cardiac auscultation showed mild tachycardia and a gallop rhythm with a third heart sound. There were no signs of pulmonary or peripheral congestion. His electrocardiogram (ECG) showed sinus rhythm and ST-segment elevation of 5 to 10 mm in leads V2-6 (Fig. 1). Echocardiography revealed hypokinesia of the anterior and apical wall segments of the left ventricle and no sign of pericardial effusion and traumatic injury to cardiac structures. A diagnosis of acute coronary syndrome was made based on the presence of typical chest pain, significant ECG changes, echocardiographic signs, and elevated levels of cardiac enzymes including MB fraction of creatine phosphokinase (CK) and troponins. Medical therapy was instituted with intravenous administration of 20 to 40 μg/min nitroglycerine for 20 minutes, oral administration of 300 mg aspirin and 10 mg diazepam, but there was no regression of ECG changes and the severity of chest pain. Therefore, thrombolytic therapy with tissue plasminogen activator was administered, which resulted in relief of chest pain and regression of ST elevations on ECG. Laboratory findings showed a typical rise and fall in CK and its MB fraction (CK-MB) consistent with AMI. After one week, coronary arteriography and ventriculography were performed. Coronary arteriography showed normal coronary arteries without any sign of atherosclerosis or coronary dissection (Fig. 2). Ventriculography showed akinesia of the apical segments of the left ventricle. Medical therapy including beta-blocker and salicylate was continued with addition of sertraline hydrochloride 50 mg/day and diazepam 5 mg twice daily for the treatment of panic disorder. Other laboratory findings such as lipid profile, lipoprotein (a), apoprotein B, fibrinogen, homocysteine levels, protein C, protein S, antithrombin III activities, and thyroid functions were within normal limits except for high levels of apoprotein A-I (1.80 g/l; N=0.80-1.60 g/l) and CRP (0.71 g/l).

Serological tests for *Chlamydia pneumonia* and *Helicobacter pylori* infections were negative. Test for factor V Leiden mutation was negative. Anticardiolipin antibodies (IgM and IgG) were also negative. Use of cardiotoxic agents such as cocaine, ergot derivatives, alcohol, tobacco, or amphetamines were excluded by the patient's medical history. Based on these findings, it was thought that prolonged coronary vasospasm induced by acute extraordinary and intense psychological stress might have caused Q wave myocardial infarction with concomitant thrombosis.

Thirty days after myocardial infarction, echocardiography revealed an akinetic apical aneurysm, ante-

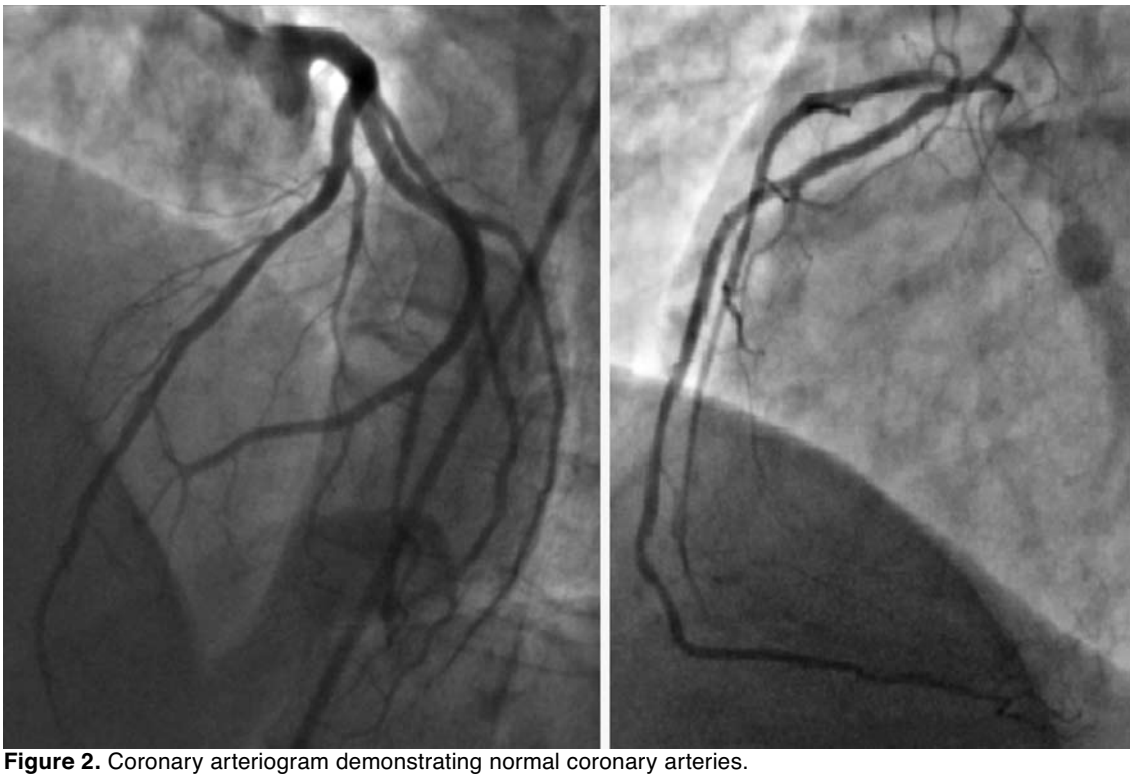

Figure 2. Coronary arteriogram demonstrating normal coronary arteries.

rolateral hypokinesia of the left ventricular wall, and decreased ejection fraction (45%). In ECG, there was persistent loss of R waves in leads V1-6 (Fig. 3).

DISCUSSION

Severe emotional stress may be responsible for the development of myocardial infarction during periods of natural disasters such as earthquakes and war threats, and women seem to be more sensitive to such frightening and stressful situations.[7] 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%,

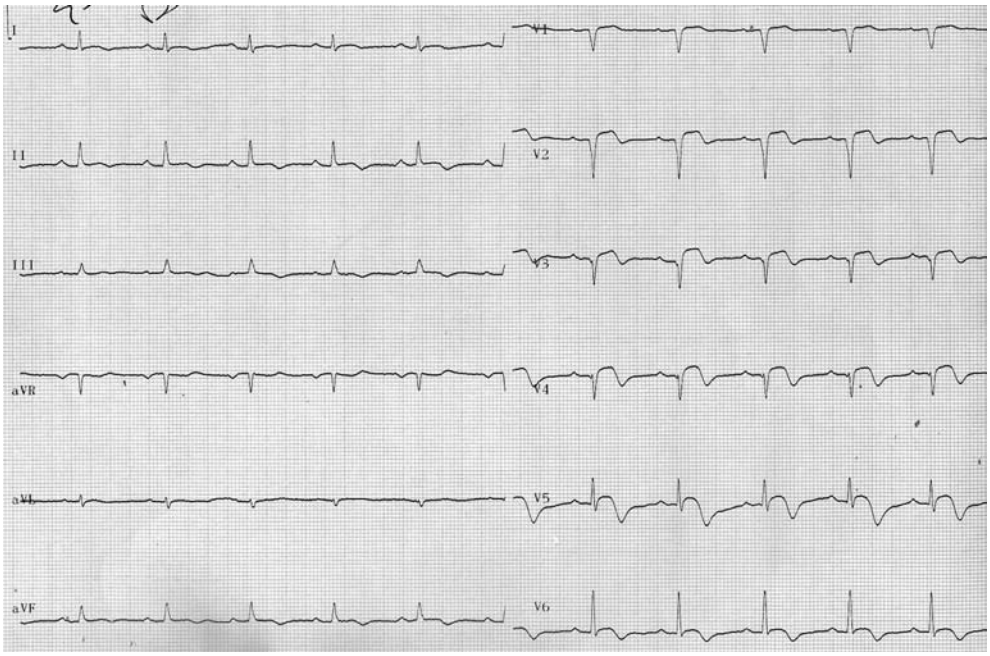

Figure 3. Electrocardiogram obtained 30 days after acute myocardial infarction.

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.[8] 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.[9] Several studies have been designed to investigate cardiovascular effects of stress. It has been demonstrated that patients with high stress have increased levels of hematocrit, fibrinogen, D-dimer, von Willebrand factor, and plasmin inhibitor complex. As seen in this young patient, these hemodynamic and hematologic alterations may change the balance between thrombosis and fibrinolysis in favor of the former, resulting in AMI even in patients with normal coronary arteries. These changes are transient and return to normal by 4 to 6 months after stressful period.[10] Intense mental stress also 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.[6] 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.[11] By positron emission tomography, Arrighi et al.[12] demonstrated a blunted augmentation of myocardial blood flow during mental stress. This response was also noted in non-atherosclerotic areas, suggesting an important role of microvascular dysfunction.[12] 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. In our case, stress-induced transient apical ballooning of the left ventricle mimicking anterior wall myocardial infarction, also called Takotsubo cardiomyopathy,[13] was considered in the differential diagnosis, but echocardiographic detection of an akinetic apical aneurysm and anterolateral hypokinesia of the left ventricle 30 days after myocardial infarction ruled out the possibility of this phenomenon. Therefore, acute and extraordinarily intense mental stress causing long-lasting coronary vasospasm in the setting of panic disorder may be responsible for this detrimental event in this young patient with normal coronary arteries.[14] This possibility should be kept in mind in the evaluation of chest pain in patients with panic disorder.

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