

EDİTÖRE MEKTUP / EDİTÖRE MEKTUP

Takotsubo syndrome in the postpartum period

Postpartum dönemde gelişen Takotsubo sendromu

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Dear Editor,

The 30-year-old female patient presented to the emergency department with the complaint of palpitation that had been present for two days. From her history, it was found out that her baby she gave birth to two days ago by normal delivery was in the newborn intensive care unit due to jaundice and that our patient was accompanying her baby. Her vital findings at the emergency department were measured as arterial blood pressure (BP): 110/60 mmHg, pulse: 120/ beat/min, body temperature: 36.4 °C, peripheral oxygen saturation (SPO2): 92%, respiratory rate: 18/min and the finger-tip blood glucose level: 140 mg/dl. On physical examination, she was tachycardic and tachypneic, and the abdomen appeared distended. The examinations of the other systems were normal. The biochemical values at presentation were normal. In the blood count, only the Hb value (10.8 g/dl) was identified as low. At presentation, the Troponin I and CK-MB values were normal, and sinus tachycardia was observed on electrocardiography (ECG). After the patient was monitorized and 25 mg of Diltizem (IV) (Mustafa Nevzat, Istanbul, Turkey) was delivered the normal sinus rhythm returned, the patient's complaints regressed, and she was discharged following the recommendation of the cardiology polyclinic. However, the patient reapplied to the emergency department with the same complaint approximately four hours later. The second ECG revealed sinus tachycardia and increased CK-MB (22.08 ng/ml) (0-4, 9) and Troponin I (8.9 ng/ml) (0-0,16) levels. The patient was admitted to intensive care with the preliminary diagnosis of ACS. The echocardiography (ECHO) performed revealed

hypertrophy in the left heart chambers, MR (1/4), slight global hypokinesia, left ventricular apical ballooning-akinesia and the ejection fraction (EF) was 50%. The patient's medical treatment regimen was prepared. Coraspin 1x300 mg (oral) (Bayer, Istanbul, Turkey) and Clexane 1x0.6(subcutaneous) (Sanovi-Aventis, Istanbul, Turkey) were delivered. The patient whose TS diagnosis became definite had a stable course during the four days of monitoring, the cardiac enzymes regressed and the control EF (55%) was higher. The patient did not give consent for coronary angiography and was discharged on the fourth day following the recommendations of the cardiology polyclinic.

We present the young postpartum patient that was initially regarded as acute coronary syndrome (ACS) in the emergency department but was later definitively diagnosed with Takotsubo Syndrome (TS) who signed the informed consent form of her free will.

Takotsubo Syndrome (TS) is a cardiac syndrome recently described, it mimics ACS and is characterized by reversible left ventricular (LV) failure1. It was first defined by Hikaru Sato et al.2 in Japan in 1990, and it was given its name because the echocardiographic appearance of the left ventricles of cases resembled the shape of the container with a narrow neck used by Japanese fishermen to hunt octopuses. The syndrome is also called the Takotsubo cardiomyopathy, ampulla cardiomyopathy, human stress cardiomyopathy or the broken heart syndrome3.

In these cases in which no coronary artery problems are detected, coronary spasm due to emotional

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stresses is held responsible. While the mechanism of the TS is not entirely known, it is thought to be caused by the excessive response of the organism to catecholamines that enter circulation due to a variety of reasons. It has been stated that 1-2% of the cases considered to be ACS are TS and that it is seen in postmenopausal women⁴. TS is characterized by left ventricular (LV) apical dysfunction and runs its course with acute cardiomyopathy. However, in this illness, the LV dysfunction, wall motion disorders, and dyskinesia are reversible. The increase in the serum catecholamine levels is essential in this syndrome that initially mimics acute myocardial infarction (AMI)⁵. It is reported that various stress factors such as anesthesia or surgery and mainly emotional reasons induce subendocardial ischemia in the apex of the LV independent of coronary pathologies and that it may range between a course that mimics AMI and complications such as severe cardiac failure⁶. The case had no history of any diseases and the stress she was exposed to after delivery was held responsible for the cardiomyopathy that developed. The fact that our case was young and in the reproductive period despite the fact that studies 7 report TS is usually more common among older and postmenopausal women is important for us.

The most common symptoms are substernal chest pain, dyspnea, syncope, and palpitation. On ECG, ST segment elevation occurs in 34-56% of the patients and often in anterior precordial derivations. Conduction disorders such as severe ventricular arrhythmias, left or right branch blocks, tachyarrhythmia, and bradyarrhythmia may also develop8. In nearly all of the patients, a moderate increase is observed in the troponin T levels that peak at 24 hours³. Our case presented with palpitation, the ECG recorded showed sinus tachycardia and the control Troponin I and CK-MB levels were measured high.

On ECHO, LV wall motion disorders, apical ballooning, LV anteroapical akinesia or dyskinesia are characteristic for TS. In addition, systolic dysfunction was present, and the EF was measured between 20-49%. Studies conducted have shown motion disorders in the LV together with the right ventricle. In consistence with the literature, the observation of LV hypertrophy together with apical ballooning and measuring low EF levels have made the diagnosis of TS definite. Within a few weeks, the LV dysfunction of patients regresses and the EF

returns to normal values. Some series have reported that full recovery occurs within 4 to 8 weeks¹⁰. In our patient, the control ECHO performed by the same cardiology specialist four days later showed that the EF value had risen to 55% and that the cardiac enzyme levels had regressed.

Currently, there is no definitive treatment for the treatment of patients with TS. In the beginning, AMI therapy is applied until the diagnosis of TS is established. After diagnosis is accurate, treatment includes monitoring and supportive treatment. ACE inhibitors, B blockers, aspirin and diuretics may also be added as a medical treatment. In our case, AMI treatment was given at the beginning and after TS diagnosis was finalized, follow-up decision was taken.

The pathophysiology of TS remains unclear; it is a condition that mimics ACS and is rarely life-threatening. This syndrome should particularly be remembered in patients that present at the emergency department after stress with cardiac complaints.

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Cukurova Medical Journal

Ertekin et al.

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