

used above the wrist in our study because of inadequate calibration of veins and arteries; thus, we did not compare in "very" different regions. Among the complications, infections or other severe complications were not observed in both groups. This issue was described in detail in the study.

The other question of the authors is about the patency that is in close relationship with the localization. PTFEs were used only between the brachial artery and high brachial vein. The reason for this selection was the diameter of the graft. Because thinner PTFEs are more likely to be thrombosed, the selected grafts were at least in 6 mm in diameter. The main finding of our study is the limited patency of the PTFE compared with saphenous veins, although they were used in larger calibers and anastomosed between larger vessels.

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## Cardiac enzyme (troponin levels) elevation in cardiac myxomas: Is it real?

To the Editor,

Constituting almost half of the cases of primary cardiac tumors (1), myxomas are frequently detected in adult female patients; moreover, familial patterns have also been identified for these tumors. The left atrium, right atrium, and ventricles are affected in 85%, 10%, and 5% of the cases, respectively. Furthermore, the fossa ovalis of the septum and the posterior atrial wall are common sites for the attachment of atrial myxomas (2). Interestingly, more than one myxoma or a polycentric myxoma can be detected in some patients (1, 2).

Atrial myxomas might be related to varied clinical presentations such as obstructive, constitutional, or embolic scenarios. Because of the blockage of the atrioventricular valves, the obstruction pattern mimics mitral disease or, rarely, tricuspid valvular disease and can cause dyspnea or left heart failure; in such cases, it is sometimes difficult to differentially diagnosis myxomas from mitral or tricuspid valve stenosis (1, 3).

Although myxomas cause systemic embolism in about one-third of the patients, the incidence of coronary artery embolization has been reported to be 0.06-0.1% (3, 4). Although rare, the condition could be fatal. In a case series by Panos et al. (4), inferior, anterior, and posterior myocardial infarctions were diagnosed by electrocardiogram (ECG) in 63.6%, 22.7%, and 9.1% of cases, respectively. Two possible explanations have been suggested for the low incidence rate of coronary artery embolization by myxomas: the vertical position of the coronary ostia to the aortic blood flow and the coverage of the coronary ostia by the

opening aortic valve leaflets during cardiac systole. Elevation of cardiac troponin levels has also been reported in atrial myxomas, all of which were secondary to the coronary artery embolization (4, 5).

Interestingly, however, we examined 10 patients (age: 49±13 years; six females) with atrial myxoma and normal coronary arteries by angiography and normal ECG but with elevation of cardiac enzymes. Cardiac troponin and CK-MB levels were measured on admission; these markers were elevated in six patients (four females; normal value of cardiac troponin: I=0.4 ng/mL; increased values in our six patients: 0.70, 1.10, 2.35, 0.86, 1.67, and 1.45 ng/mL, respectively), all of whom had normal coronary arteries, based on angiography findings and normal ECG findings, and had no accompanying chest pain. Patients were further investigated for exclusion of other reasons for elevated cardiac troponin levels, including renal failure, sepsis, pulmonary emboli, tachy, or bradyarrhythmias. These findings suggest that atrial myxoma increases cardiac markers without involvement of coronary arteries. Actually, we think such constitutional symptoms (fever, weight loss, or symptoms resembling connective tissue disease) are due to cytokine (interleukin-6) secretion; cardiac markers could be secreted in cardiac myxomas as well. Moreover, cardiac myxomas could be considered as the differential diagnosis for the diseases with elevated cardiac enzymes. However, further studies are required to reveal this association.

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## The preanalytical and analytical factors responsible for false-positive cardiac troponins

To the Editor,

Cardiac troponins (cTn) are the cornerstone of the diagnosis, risk assessment, prognosis, and determination of antithrombotic and revas-

cularization therapies in acute coronary syndrome (ACS). Cardiac troponins are still evolving via the introduction of the high-sensitive new generation assays. There are adequate data focused on the causes of troponin elevation other than ACS. The well-known conditions are chronic renal failure, advanced heart failure, myo/pericarditis, cerebrovascular accident, pulmonary embolism, sepsis, strenuous exercise, trauma etc. (1). Beyond these clinical factors, some drawbacks can be experienced with cTn assays.

The main preanalytic factors for false-positive cardiac troponins include hemolysis and fibrin compounds in the sample. Fibrin molecules can adhere to the well of the plate, resulting in false-positive results (1). Hemolysis is a challenging problem, because it may increase cTnI values for some assays; interestingly, it may also decrease cTnT values with another assay provided by a different manufacturer. Moreover, these problems may become more crucial with high-sensitive assays (2). The other preanalytical factors are erroneous calibration, analyzer malfunction, reagent deterioration, instrumental carry-over, and inappropriate sample dilution (1, 2), all of which concern laboratory of biochemistry but also directly affect the clinician. Beyond paying attention in drawing and storing blood samples, dealing with these problems requires a close and compatible contact between the laboratory and cardiologists.

The most challenging analytical factor is the presence of heterophilic antibodies (HA) in the serum of the test sample. Troponin assays are performed on the principle of the two-site ELISA. Heterophilic antibodies bind nonspecifically to the Fc portion of the assay antibodies, leading to deceptive elevations in troponins (3). In autoimmune diseases, rheumatoid factor was shown to cross-react with troponin assays. On the other hand, HA emerge may be facilitated by frequent contact with animals, vaccinations, immunotherapies, blood transfusion, and diagnostic and therapeutic use of animal monoclonal antibodies as well as even dietary antigens (1, 3). The incidence of HA was found as much as 50%; fortunately, the prevalence of false-positive troponin was declared in about 3% of the general population (4). To prevent interference, dilution of the sample and precipitation with polyethylene glycol can be performed. However, the best way to overcome HA is to use heterophile blocking tubes (3), which takes additional cost. However, these tubes should be kept available in centers evaluating high number of ACS patients. In fact, detection of a rise and/or fall in troponin levels is crucial for the diagnosis of myocardial cell damage (5). On the other hand, a sustained increase in troponin levels, which indicates no change in plasma kinetics over time, and troponin increase not supported by either chest pain with ECG changes or increase in other cardiac markers such as CK-MB makes an observation of false-positive troponin more reasonable.

Finally, because the evaluation of acute chest pain is one of the most challenging issues in cardiology, clinicians should be aware of the problems that result from false-positive troponin elevations. In this manner, preanalytical and analytical factors related to this dilemma and improvements in assay methods should be considered carefully.

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## Acute anterior myocardial infarction after “Bonzai” use

To the Editor,

Illicit drug use is one of the major public problems in Turkey. “Bonzai” is a mixture of synthetic cannabinoids, and its use is a growing health problem. Because of its easy access and cheaper price, there is a higher tendency of its abuse. Cardiovascular effects of this drug should be well known by the physicians.

A 29-year-old previously healthy man without cardiovascular risk factors such as hypertension, diabetes, and hyperlipidemia was admitted to our emergency department by paramedics via ambulance. He smoked half a packet of cigarettes per day for 10 years. His family history was uneventful for cardiovascular diseases, and he had no first-degree relatives with diabetes. According to the information gathered from the paramedics, he had lost his consciousness about 30 min before finding him, and on finding him, his cardiac rhythm was ventricular fibrillation. After electrical cardioversion, a hemodynamic response was obtained, but he was intubated because of loss of consciousness. His friend stated that he had for the first time tried to use “Bonzai” three times in the last 3 h. On presentation to the emergency department, his vital signs were as follows: heart rate, 135 bpm and blood pressure, 95/60 mm Hg; his electrocardiography showed acute anterior myocardial infarction. He immediately underwent coronary angiography, and his left anterior descending coronary artery was completely occluded proximally by a thrombus. Other coronary segments did not have any atherosclerotic plaque and free of coronary artery disease. After thrombus aspiration, a 4.5×22-mm bare metal stent was implanted at 14-atm pressure, and using a 5.0×12-mm balloon, the proximal part of stent was dilated successfully. The angiographic result obtained at the end of procedure was good. He was extubated 2 days after coronary angiography and discharged from the hospital after 5 days with good health status.

Cannabinoids are a diverse group of substances acting on cannabinoid receptors; they are classified mainly into three groups: natural