

## 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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