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.

Adem İlkay Diken
Department of Cardiovascular Surgery, Faculty of Medicine, Hitit University; Çorum-Turkey

References

Address for Correspondence: Dr. Adem İlkay Diken, Türkiye Yüksek İhtisas Hastanesi, Kalp ve Damar Cerrahisi 06100, Şehhiye, Ankara-Türkiye
Phone: +90 530 687 33 15
E-mail: ademilikay@gmail.com

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 phenomenon. Res Cardiovasc Med 2013; 2: 77-8. [CrossRef]

Address for Correspondence: Anita Sadeghpour, MD, FASE, FACC, Associated Professor of Cardiology, Fellowship of Echocardiography, Rajaie Cardiovascular Medical and Research Center, Valiasr Street, Tehran-Iran
Phone: +982123922145
E-mail: alizadehasl@gmail.com
©Copyright 2015 by Turkish Society of Cardiology - Available online at www.anakarder.com
DOI: 10.5152/akd.2015.5871

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-
caturalization 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.

Kaan Okyay, Aylin Yıldırır
Department of Cardiology, Faculty of Medicine, Başkent University; Ankara-Turkey

References
4. Fleming SM, O’Byrne L, Finn J, Grimes H, Daly KM. False-positive cardiac troponin I in a routine clinical population. Am J Cardiol 2002; 89: 1212-5. [CrossRef]