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 cTnl 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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References
cannabinoids, endogenous cannabinoids, and synthetic cannabinoids. The most well-known example of natural cannabinoids is marijuana. “Bonzai” is the one of the commercial names of synthetic cannabinoids. “Bonzai” is a herbal blend containing multiple synthetic cannabinoids (1). The exact composition of this drug is unknown and may be variable. The compound most frequently found in the herbal mixture is JWH-018. Cannabinoid inhalation has been linked to a higher rate of acute myocardial infarction and mortality after myocardial infarction (2). Most case reports describe relatively young patients in their second or third decades with normal coronary arteries or minimal atherosclerosis, suggesting that marijuana and cannabinoids do not lead to the development or acceleration of atherosclerotic damage in healthy adults (3). However, our case presented with thrombus formation and occlusion of the coronary artery. As a big problem in the world, physicians have to be aware of the cardiac effects of cannabinoids.

References


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