Assessment of serum hepcidin levels in patients with non-ST elevation myocardial infarction

To the Editor,

We read the article, entitled "Assessment of serum hepcidin levels in patients with non-ST elevation myocardial infarction," by Altun et al. (1) published in Anatolian J Cardiol 2014; 14: 515-8. Serum hepcidin levels were comparable among NSTEMI patients and control subjects. Also, its concentration did not change 6 hours after admission. The authors concluded that hepcidin could not be used as a marker of myocardial necrosis in NSTEMI patients. We thank the authors for drawing attention to a very important and challenging field of cardiology: markers in acute coronary syndromes. However, in their study, we think that there are some important questions that need to be answered.

The peptide hormone hepcidin is the main conductor of systemic iron hemostasis (2). The expression of the hepcidin gene has been shown to be regulated by hypoxia and inflammation (3). According to this finding, Suzuki et al. (4) argued that the human heart might also react to ischemia, and they measured serum hepcidin levels in patients with acute myocardial infarction. They found an elevated serum hepcidin level within 4 hours after the heart attack and showed that hepcidin levels decreased to normal levels in 7 to 14 days. In the present study of Altun et al. (1) the time interval between the onset of the symptoms and blood sampling was not mentioned. Additionally, the authors retested serum samples of the NSTEMI patients only 6 hours later. However, hepcidin levels are detectable after several days following myocardial injury (4). The racial and genetic differences between the study population of Suzuki et al. (4) and Altun et al. (1) can explain the negative result of the latter study. The authors did not mention anything regarding coronary artery lesions of the study population; control subjects were aged between 50 and 70 years, and they can also have coronary atherosclerosis. Hence, it is an important limitation of this study if hepcidin might reflect destabilization of the coronary plaques, as expected from an inflammatory biomarker. The authors provided that CRP levels were increased in NSTEMI patients. In this point, performing a correlation analysis between CRP and hepcidin levels is very essential. In the case of showing this relationship, it could be argued that hepcidin might be a surrogate marker of inflammation, although plasma kinetics were not identified properly. Moreover, since this biopeptide is not a structural element of the myocardial cell like cardiac troponin I, it naturally might not be elevated at the same time. Finally, serum levels of hepcidin in patients and in control subjects were unevenly distributed: 24.55±32.13 and 23.67±33.62 ng/mL. It can be concluded that there are many extreme cases in the laboratory results, which can affect all analyses and interpretations in this small-sized study.

Therefore, we think that although the study conducted by Altun et al. (1) draws attention to a very important and interesting subject, there are several points in the study design and data evaluation that need to be discussed, and the study results should be interpreted with caution.

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