Copeptin Adds to High-Sensitivity Troponin T in Rapid Rule Out of Acute Myocardial Infarction

To the Editor:

Karakas and colleagues (1) reported on copeptin and high-sensitivity troponin T (hsTnT) (2) in a sub-study of ROMICAT (Rule Out Myocardial Infarction by Computed Tomography) (2) that evaluated coronary computed tomography for ruling out myocardial infarction in low- to intermediate-risk patients with chest pain who were eligible for coronary computed tomography and subsequent coronary angiography. The authors concluded that copeptin does not add diagnostic information to hsTnT in this patient cohort. Only 8 patients in this study were classified as having myocardial infarction, and another 27 patients were classified as having unstable angina pectoris (UAP) (see the authors’ Table 1). Copeptin is a marker of severe hemodynamic stress and increases immediately with the index event. Therefore, copeptin concentrations are increased at admission, particularly early after the onset of symptoms, and decline over time (3, 4). Karakas et al. drew their samples at the time of the computed tomography angiography evaluation, which occurred at a median of 4.5 h after patient admission to the emergency department. The comparison of copeptin and hsTnT at this late point in time is not relevant for this patient group, because an early rule out of myocardial infarction at admission is what clinically matters and what other groups have found (3, 4). For this rule out, blood samples must be drawn as early after the onset of symptoms as possible, that is, at the time of a patient’s arrival at the emergency department. Blood samples drawn several hours after admission and (in most cases) after the initiation of treatment cannot be used for this purpose.

The second limitation of the study is the very low number of patients with acute coronary syndrome. Although 366 patients with complete data sets were enrolled, acute coronary syndrome occurred in only 35 of these patients, of whom 27 were classified as having UAP, leaving 8 patients with acute myocardial infarction. To draw a conclusion on the validity of a marker in a defined patient group with a very low number of actual patients is disputable.

Additionally, this number is probably incorrect, because the authors state that UAP was defined according to “standard criteria.” These criteria must include at least 2 serial negative results (<0.03 µg/L) in a fourth-generation TnT test. According to Table 1 in the report of Karakas et al. (1), the median hsTnT concentration in the UAP group 4.5 h after admission was 16.2 pg/mL (interquartile range, 44.7 pg/mL). This result means that even when applying the conventional troponin cutoff, many patients with non–ST-segment elevation myocardial infarction were misclassified as UAP, which has a particular impact considering the low number of acute coronary syndrome patients in the entire study cohort. The authors failed to report routine serial troponin results, which would have led to a reclassification of some of their UAP patients.

Copeptin is not a marker of coronary artery disease, angina pectoris, or the extent of the disease, but is rather an early rule-out marker of acute myocardial infarction. This question was not addressed in the actual report, and therefore the general conclusions are not warranted.

Author Contributions: All authors confirmed they have contributed to the intellectual content of this paper and have met the following 3 requirements: (a) significant contributions to the conception and design, acquisition of data, or analysis and interpretation of data; (b) drafting or revising the article for intellectual content; and (c) final approval of the published article.

References


Martin Möckel*

* Address correspondence to the author at: University Hospital Charité Campus Virchow-Klinikum Augustenburger Platz 1 Berlin 13353, Germany Fax +49-30-4507553203 E-mail martin.moeckel@charite.de

Previously published online at DOI: 10.1373/clinchem.2011.171058