Editorial

For Patients with Acute Coronary Syndrome, is Initial Measurement of Troponin Alone the Best we Have to Offer?

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For decades it has been known that there is good correlation between the levels of certain enzymes and the size of myocardial infarction evaluated by necropsy.

As it might be expected, several publications have found highly significant correlations between the levels of these markers and the incidence of events, mainly deaths - the higher the value, the higher the mortality.

On the other hand, with the emergence of new markers, basically troponins and CKMB mass ("biomarkers of myocardial necrosis"), new evidence began to emerge. For example, many patients who were categorized as having unstable angina have received a diagnosis of acute myocardial infarction without ST segment elevation, because these biomarkers are more sensitive than the enzymes previously used. On the other hand, it was also found that approximately 30% of patients with increased troponin levels have normal CKMB activity, and that these patients have a worse prognosis than those with normal troponin.

This relevant study by Santos et al[1] contributes to a better understanding of the topic, to analyze the role of troponin and CKMB mass in an unselected population of patients with clinical symptoms compatible with acute coronary syndrome (somewhat with surprisingly 2/3 of patients with unstable angina, and only 1/3 with AMI without ST elevation).

Taking into account the measurements taken during the first 24 hours of onset, and treating myocardial necrosis markers as dichotomous variables, the authors conclude (correctly) that, by measuring troponin, the additional measurement of CKMB mass would have been unnecessary. However, some questions remain unanswered, which could cast doubt on a so blatant conclusion:

1. If the biomarkers studied were treated as continuous variables rather than categorical variables, would the results be the same?
2. Apparently, the ROC curves are similar when including troponin or CKMB mass alone, which could suggest that the CKMB mass would be as effective as troponin as a predictor of clinical events.
3. Instead of an initial measurement, if we built CKMB mass curves and analyzed the prognostic value of this marker, taking into account its peak (which usually occurs after the first 24 hours of onset), or even the area under the curve, which is a methodology used by the vast majority of studies on this topic, would not we have different results?
4. A study of necropsy shows that the peak of CKMB mass correlates better with AMI size than the troponin obtained in the first hours of admission[2]. In the same direction, analyzing the incidence of necrosis after angioplasty by magnetic resonance imaging, Lim et al[3] conclude that CKMB is superior to troponin in the diagnosis of this type of infarction.

In conclusion, it seems somewhat premature to propose the eradication of CKMB mass curve from our routines. However, if the option is to perform only measurements of biomarkers in the first 24 hours of onset, the results reported here (and the consequent conclusion) must be certainly taken into consideration when implementing institutional routines. Finally, it is worth remembering that the new ultrasensible biomarkers, which start coming to the markets, perhaps would substantially alter the understanding we have today on this subject.

Keywords

Myocardial infarction; coronary disease; troponin; biological markers/blood.

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