
The keypoints in this article can be found in the “Abstract” and the “Discussion” sections of the article. Also, the “Introduction” does a good job of explaining when and how cTnI and CK-MB are released.

Keypoints:

- **Important Note:** In literature published prior to the year 2000 (including this article), the term “acute myocardial infarction (AMI)” was defined using the WHO classification which required: chest discomfort; total CK and CK-MB mass rise; and ECG changes, including ST-deviations of 0.1 mV or more in 2 leads, new T-wave inversions of 0.1 mV or more, or Q-waves. Any patient that did not meet the above definition but had signs and symptoms of ischemic heart disease were said to have “unstable angina.” The term “non-ST-segment elevation myocardial infarction (NSTEMI)” had not been coined before 2000. With the arrival of cardiac troponins on the scene as the preferred marker after 2000, the old “unstable angina” definition was divided into “NSTEMI” patients in which heart damage has occurred as demonstrated by cTnI elevation and new “unstable angina (UA)” patients in which no heart damage is evident because there is no cTnI elevation observed.

- **When discussing the cTnI cartridge with clinicians, try to get a sense for whether he/she is using the “old AMI” definition or the current “MI” definitions. Based on the cTnI cutoffs that we have seen so far in our performance verification evaluations, it appears that most hospitals are still using the “old AMI” definition to set their cut-offs for “MI.”

- **In this paper, the following points are important:**
  - 39 consecutive AMI (old definition) patients were studied
  - On average, the patients presented 4.5 hours after the onset of chest pain (range = 0.7 to 12.1 hrs)
  - Compared CK-MB and cTnI serum measurements on the Dade Stratus II central lab instrument, in serial samples over 36 hrs
  - Findings –
    - There was a significant correlation between CK-MB and cTnI over 36 hrs.
    - The kinetics for CK-MB and cTnI were not significantly different between 0 to 8 hrs following the onset of chest pain.
    - cTnI was increased significantly different over CK-MB from 9 to 36 hrs following the onset of chest pain.
    - The larger the infarct, the longer the marker remained elevated. Early release of cTnI is from the minor (<5% of total myocardial cTnI cell content) but readily releasable cytoplasmic cTnI pool.
    - Left ventricular ejection fraction (LVEF) was significantly correlated with both peak CK-MB mass and peak cTnI.
    - Peak CK-MB and peak cTnI were significantly correlated.

- **Conclusions:** Based on these findings, cTnI shows excellent promise as a useful marker of infarct size, for the assessment of left ventricular function, and may potentially replace CK-MB as the cardiac-specific marker for detection of myocardial injury, including AMI.