



Update on Cardiac Markers—Diagnosing AMI Using Troponin

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Since 1954, the search for a defining cardiac biomarker began as the breakdown products of myocardial ischemia were discovered¹. The ideal marker would be effective and accurate would indicate how severe and when an acute myocardial infarction (AMI) has occurred. Thus far, no single marker can fulfill all criteria, but troponins can be very useful in making an important clinical decision to admit or discharge a patient in the ER or to treat a patient on the inpatient floor. Accurately deciding when an AMI has occurred can save both lives and resources.

Cardiac troponins are proteins that control the calcium-mediated interaction between actin and myosin, allowing contraction at the sarcomere level. Cardiac troponin is found almost exclusively in the heart, being composed of three subunits: I, T, and C. The clinically useful I and T subunits bind to tropomyosin and inhibit coupling of actin and myosin, respectively. These molecules have proven advantageous in clinical practice by providing a high specificity for cardiac damage in the presence of multiple injuries. Troponin levels peak at 18–24 hours and remain in circulation 7–14 days after an AMI (Table 1). Since troponin degrades slowly, clinicians have a large window for diagnosing a recent AMI. Newer high precision assays can now detect troponin elevations as early as two hours after the onset of ischemic symptoms, giving it an important role as an early marker.² Troponin is now the standard of care in most centers and is the preferred marker in defining an AMI.³ At least three serial measurements are recommended spaced at least three hours apart for detection of a cardiac event.⁴ However, troponin is not the marker of choice to detect re-infarction, as it will be masked by the first event.¹

According to the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction, in addition to clinical symptoms of ischemia, detection of cardiac biomarkers (preferably serial troponin I or T) above the 99th percentile of the upper reference limit (URL) is required for diagnosis of AMI, although the WHO criteria differs by requiring above the 95th percentile.⁴ Lower cutoff values for troponin may identify a population at risk for cardiac events, providing valuable risk stratification information rather than simple "infarction or not" categories. A barrier to achieving these lower cutoff limits for troponin is the lack of standardization among the troponin I assays, due to: 1) different antibody configurations recognizing different epitopes, 2) assays with varying stabilities, and 3) different coefficients of variability. With this lack of standardization in assays, relaxing cutoff values could result in an increase of false-positives. The International Federation of Clinical Chemistry and Laboratory Medicine has a listing of troponin assays and their performances at lower concentrations that may be useful as a guide in AMI diagnosis.⁵

In the rapidly changing climate of medicine, cardiac biomarkers have evolved from curious laboratory findings into standards of care. Troponins now have the diagnostic power to detect subtle myocardial changes in a timely enough fashion to impact patient care and quality of life. A new set of challenges lies ahead in predicting the prognosis of AMI patients and screening those at risk for future cardiac events, thus making the advent of a new generation of markers an imperative.

Table 1. Characteristics of Cardiac Biomarkers after AMI in Hours¹

Biomarker	Onset	Peak	Duration
Myoglobin	1–4	6–7	18–24 hours
Total CK	3–12	18–24	36–48 hours
CK-MB	3–12	18–24	24–36 hours
CKMB2/CKMB1	2–6	6–9	Unknown
Troponin I	2–12	18–24	7–10 days
Troponin T	2–12	18–24	10–14 days

References

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