BG Pulse Journal USA
ISSN:1947-2536 http://www.bgpulseusa.com/index.php/bg

# THE ROLE OF CARDIOSPECIFIC BIOMARKERS (TROPONIN I AND T) IN THE DIAGNOSIS OF ACUTE MYOCARDIAL INFARCTION

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**Keywords:** cardiac troponin I; cardiac troponin T; myocardial infarction

**Relevance**

Acute myocardial infarction (AMI) remains one of the leading causes of morbidity and mortality worldwide. Early and accurate diagnosis is essential for effective treatment and improved patient outcomes. Among various diagnostic tools, cardio-specific biomarkers play a crucial role. Cardiac troponins I and T are considered the gold standard biomarkers for myocardial injury due to their high sensitivity and specificity. This article reviews the biochemical characteristics of troponin I and T, their pathophysiological release mechanisms, diagnostic value, and clinical significance in the diagnosis of acute myocardial infarction.

Acute myocardial infarction is a life-threatening condition resulting from prolonged ischemia and necrosis of myocardial tissue, most commonly caused by coronary artery occlusion. Clinical symptoms, electrocardiography (ECG), and laboratory findings form the basis of AMI diagnosis. However, clinical presentation may be atypical, and ECG changes are not always definitive. Therefore, biochemical markers of myocardial injury have become essential components of modern cardiology. Among them, cardiac troponins I (cTnI) and T (cTnT) are the most reliable and widely used biomarkers.

Diagnosing cardiac emergencies is a critical responsibility in the emergency department. Clinicians must rapidly and accurately reduce the broad differential diagnosis of chest pain to ensure the timely delivery of life-saving interventions. Beyond medical history and physical examination, several essential diagnostic tools are used to differentiate the causes of chest discomfort. Among these, measurement of cardiac troponins has become a cornerstone of cardiac evaluation and diagnosis.

Troponins are regulatory proteins found in muscle cells, present both as a small free cytosolic pool and as structural components of the myofibrils. Three molecular forms of troponin exist, each corresponding to specific isotypes found in cardiac muscle, slow-twitch skeletal muscle, and fast-twitch skeletal muscle. The cardiac isoform differs significantly from skeletal isoforms, sharing only about 60% sequence similarity and containing an additional 31 amino acids at the amino-terminal region.

Cardiac troponin T (cTnT) and cardiac troponin I (cTnI) are essential proteins involved in muscle contraction and play key roles in regulating cardiac and skeletal muscle function. Despite their related roles, they differ in tissue specificity and function. cTnI is exclusively found in myocardial tissue, making it highly specific to cardiac muscle. In contrast, cTnT is minimally expressed in skeletal muscle; however, modern cTnT assays are designed to avoid detection of

skeletal muscle troponins [7]. Functionally, cTnI binds to actin within thin filaments, stabilizing the troponin–tropomyosin complex and inhibiting actomyosin ATPase activity. cTnT, on the other hand, binds to tropomyosin, anchoring the troponin complex to thin filaments and regulating its interaction during muscle contraction.

Because cardiac troponin is not normally present in the bloodstream, individuals without myocardial injury have extremely low circulating levels of this protein. Consequently, clinical attention is focused solely on elevated troponin concentrations, in contrast to many other laboratory markers that may be clinically relevant at both low and high values.

The diagnostic value of cardiac troponin I (cTnI) and cardiac troponin T (cTnT) lies in their high sensitivity and specificity for myocardial injury, particularly in the setting of myocardial infarction and other cardiac disorders. These heart-specific proteins are typically confined within cardiomyocytes; however, when myocardial damage occurs, such as during ischemia or infarction, they are released into the circulation. Thus, increased blood concentrations of cTnI or cTnT reflect the presence and severity of cardiac muscle injury. Moreover, troponin levels can provide an estimate of infarct size, as greater myocardial damage results in higher circulating concentrations. A large multicenter study involving 2,226 patients demonstrated that both troponins exhibit high diagnostic accuracy for acute myocardial infarction, despite differences in their biochemical properties and release kinetics.

Cardiac troponins enter the bloodstream following acute cardiac injury associated with transient local ischemia. Patients commonly experience dull chest or arm discomfort within the initial hours after the event. Serum troponin concentrations typically begin to rise approximately two to three hours after symptom onset and continue to increase until reaching a peak, usually between 12 and 48 hours, although peak levels may occur as early as 6 hours or as late as 3 days depending on the type of myocardial infarction. In acute myocardial infarction, cTnI levels often increase to a greater extent than cTnT, whereas cTnT concentrations may be relatively higher in stable conditions such as atrial fibrillation. When measured 6–12 hours after the onset of chest pain, troponin assays demonstrate near-100% sensitivity and significantly greater specificity for myocardial injury compared with earlier cardiac biomarkers.

Troponin T is found in a 3-unit complex of the contractile apparatus in myofibrils, and there is also a cytosolic pool of unbound troponin that is released acutely, mimicking the appearance kinetics of other cytosolic proteins. This pool represents about 6% of cTnT, which explains the late but not early release of troponin. The increased overall amount of troponin in the heart is not why troponin is more sensitive than other proteins. The increased early sensitivity likely reflects the fact that the percentage of troponin released that reaches the blood after cardiac injury is greater for troponin than for other proteins. The unique release kinetics and the presence of a cytosolic pool of unbound cTnT may contribute to the observed pattern of cTnT concentration, characterized by an initial drop, a plateau, and then another drop.

Both cTnI and cTnT have a short half-life in plasma, lasting around two hours. The apparent half-life is 24 h, with cTnT being slightly longer, due to the ongoing release of troponin from the necrotic myocardium.

Troponins have been measured using a variety of methods over time, each with unique benefits in terms of sensitivity and specificity. The clinical situation and the ideal balance between sensitivity and specificity—both necessary for a correct diagnosis—often influence the assay selection. Antibodies used in immunoassays can detect very low troponin concentrations with great sensitivity and specificity. Chemiluminescent detection is the most common choice, and together with automated analysis provides quick results. Point-of-care testing (POCT), which offers rapid

diagnostic results close to patient treatment sites, can have variable sensitivity and specificity and is less frequently used.

The most recent international guidelines suggest that high-sensitivity techniques should be used for measuring cardiac cTnI and cTnT in order to detect myocardial damage and differentiate between acute coronary syndromes. Acute myocardial infarction diagnosis requires the presence of myocardial damage, which is a separate condition. The 2018 Fourth Universal Definition of Myocardial Infarction states that myocardial injury is detected when at least one value above the 99th percentile upper reference limit is measured in a patient with high-sensitivity methods for cTnI or cTnT.

However, the primary justification for utilizing the 99th percentile upper reference limit is the current deficiency in standardization and harmony among the many cardiac troponin assays. This means that a single numerical cut-off cannot be used as a physiological threshold for myocardial injury.

With the development of troponin assays, the 99th percentiles of the assays became ever lower, and the sensitivity became ever higher. In addition to providing a CV ≤ 10% at the 99th percentile, the most recent generation of high-sensitivity assays can now measure troponin concentrations above the level of detection in more than 50% of the healthy population. Additionally, gender-specific 99th percentiles have been set, with the value for females usually lower than for males.

The levels of cTnI and cTnT can provide valuable insights into different types of myocardial infarction. ST-elevation myocardial infarction (STEMI) causes a larger blockage of one or more coronary arteries, leading to a complete deprivation of blood to the heart. On the other hand, non-ST-elevation myocardial infarction (NSTEMI) causes only partial or incomplete blockage of a coronary artery. STEMI is considered a more serious event than NSTEMI due to the larger blockage and the resulting impact on the heart's blood supply. This results in more long-term heart health problems and increases the risk of death in the short term. The severity of myocardial injury in STEMI is typically more extensive due to the complete blockage of a coronary artery.

In STEMI, both cTnI and cTnT levels are typically elevated at presentation and are associated with a worse prognosis in terms of both short-term and long-term mortality. Higher troponin values generally correlate with a larger infarct, providing an estimate of the infarct size and the severity of myocardial damage. In NSTEMI, elevated levels of cTnI and cTnT at presentation are also associated with a worse prognosis in terms of both short-term and long-term mortality. Peak troponin values can provide an estimate of the infarct size and the severity of myocardial damage, with higher values generally correlating with a larger infarct.

In Type 2 myocardial infarction, an elevated troponin in the first blood sample is expected in patients with renal failure. A rising concentration of troponin indicates acute injury and may be due to an acute myocardial infarction. It is crucial to classify the etiology of a troponin elevation and treat patients accordingly.

With the widespread use of high-sensitivity troponin assays, an expanding range of clinical conditions has been recognized as causes of myocardial injury through mechanisms other than ischemia. Although elevated troponin concentrations indicate myocardial damage, they are not specific for myocardial infarction. Several non-ischemic conditions, including congestive heart failure, tachyarrhythmias, and renal dysfunction, may also lead to increased troponin levels and potential diagnostic confusion.

Troponin elevations are additionally observed in acute coronary syndromes, myocarditis, and infiltrative disorders such as amyloidosis. Other contributing factors include exposure to certain medications or toxins, transplant vasculopathy, and critical illness. In acute or chronic systolic and

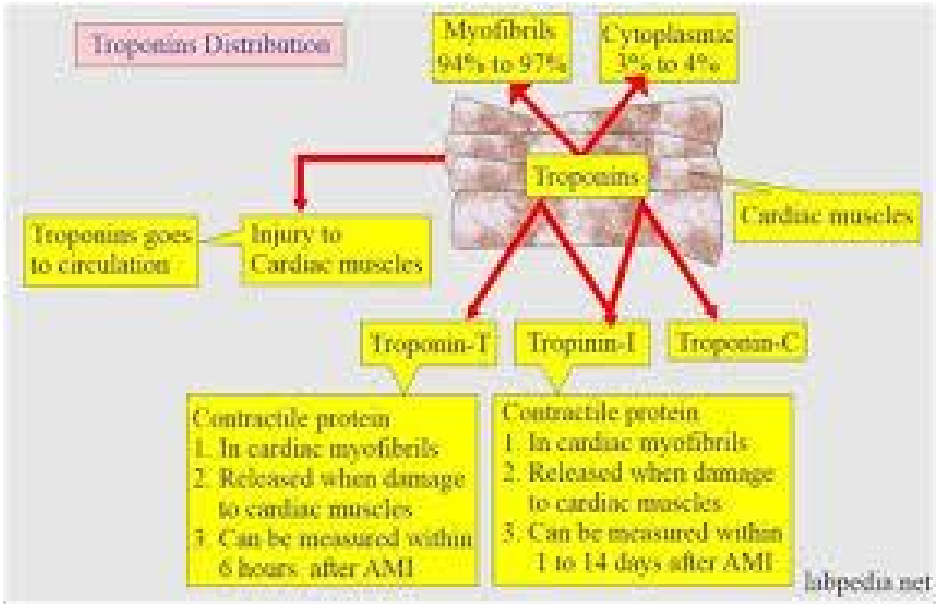
diastolic heart failure, myocardial injury is commonly related to ventricular wall stretch or strain, resulting in slower troponin release patterns with relatively modest elevations. Inflammatory processes directly damage the myocardium in conditions such as myocarditis and pericarditis.

Cardiac injury may also occur following cardiopulmonary resuscitation, either due to mechanical chest compressions or electrical defibrillation. Furthermore, stress-related surges in catecholamines and neurohormonal activation can cause transient myocardial injury and ventricular dilation, a phenomenon known as stress-induced cardiomyopathy. Acute pulmonary embolism leads to troponin elevation through sudden right ventricular strain rather than primary myocardial ischemia. Finally, chronic kidney disease or severe acute renal failure is associated with elevated troponin levels, at least in part due to impaired renal clearance.

Both cTnI and cTnT were associated with adverse classical cardiovascular disease (CVD) risk factors. The Spearman correlation coefficient between cTnT and cTnI was found to be 0.443 ($p < 0.001$). Previous cohort studies have examined either cTnI or cTnT, but rarely both, most likely because assays are assumed to be interchangeable in the diagnosis of an acute cardiac event. This has resulted in an evidence base that does not directly show which biomarker is actually better. A meta-analysis, which included 13 studies that measured cTnI and 7 studies that measured cTnT—none of which measured both—reflects this. The risk of future CVD events was greater for cTnT than cTnI in the top versus bottom third of the distribution (hazard ratio = 1.60 versus 1.36; $p = 0.171$), and cTnT was more significantly related to fatal CVD ($p = 0.027$).

The troponin complex is a regulatory protein system located on the thin filaments of striated muscle. It consists of three subunits: troponin C, troponin I, and troponin T. Cardiac troponin I and T are structurally distinct from their skeletal muscle counterparts, making them highly specific for cardiac muscle tissue.

Troponin I inhibits actin-myosin interaction, while troponin T binds the troponin complex to tropomyosin. In healthy individuals, circulating levels of cardiac troponins are extremely low or undetectable. Myocardial cell injury leads to disruption of cell membranes and release of troponins into the bloodstream.




**Pathophysiology of Troponin Release in Acute Myocardial Infarction**

During an acute myocardial infarction, prolonged ischemia results in irreversible myocardial cell damage and necrosis. This process causes the release of intracellular cardiac proteins, including troponins, into the circulation. Troponin levels begin to rise within 3–6 hours after the onset of myocardial injury, peak at 12–24 hours, and may remain elevated for 7–14 days, depending on the extent of myocardial damage.

The prolonged elevation of troponin levels allows for late diagnosis of AMI, even when patients present several days after symptom onset.

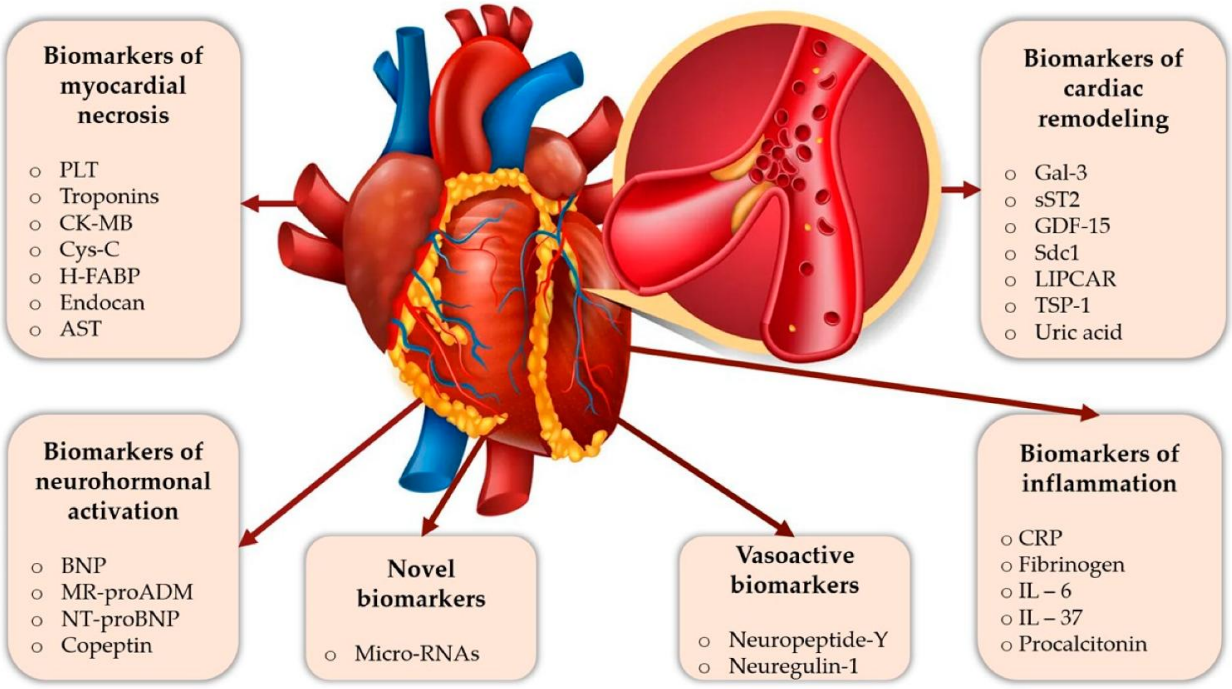
**Diagnostic Value of Troponin I and T**

Cardiac troponins I and T are the most sensitive and specific biomarkers for diagnosing acute myocardial infarction. According to current international guidelines, the diagnosis of AMI requires evidence of myocardial injury with a rise and/or fall of cardiac troponin values, along with at least one clinical criterion such as ischemic symptoms, ECG changes, or imaging evidence of myocardial damage.

High-sensitivity troponin assays have further improved early detection of myocardial injury, allowing diagnosis within the first hours after symptom onset. These assays can detect very low troponin concentrations, enabling faster clinical decision-making and risk stratification.

**Clinical Significance and Limitations**

Beyond diagnosis, troponin levels provide important prognostic information. Higher troponin concentrations are associated with larger infarct size, increased risk of complications, and poorer outcomes. Troponin measurement is also useful in differentiating myocardial infarction from unstable angina.




However, elevated troponin levels are not exclusively specific to AMI. They may also be increased in other conditions such as myocarditis, heart failure, pulmonary embolism, sepsis, and chronic kidney disease. Therefore, troponin results must always be interpreted in the clinical context.

**Conclusion**

Cardiac troponins I and T play a central role in the diagnosis of acute myocardial infarction due to their high sensitivity, specificity, and prognostic value. They have become the cornerstone of modern diagnostic algorithms for myocardial infarction. Despite certain limitations, when used in combination with clinical assessment and ECG findings, troponin measurement significantly improves diagnostic accuracy and patient management in acute coronary syndromes.

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