



Diagnostic Value of Cardiac Troponin I in Acute Myocardial Infarction: A Clinical Study

Kholud Musbah Mohamed Ahmed *

Department of Laboratories, Faculty of Medical Technology, University of Bani Waleed,
Libya

القيمة التشخيصية لتروبونين القلب (I) في احتشاء عضلة القلب الحاد: دراسة سريرية

خلود مصباح محمد أحمد *

قسم المختبرات، كلية التقنية الطبية، جامعة بني وليد، ليبيا

*Corresponding author: kholoodahmed@bwu.edu.ly

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Abstract:

This study aims to clarify the diagnostic value and clinical importance of troponin I in identifying heart diseases, particularly in emergency settings. The study comprised 116 adults, aged 35 to 80 years, who presented with symptoms indicative of heart disease. Participants with chronic kidney disease or recent trauma were excluded to prevent confounding results. Venous blood samples were analyzed for troponin I levels using an electrochemiluminescence immunoassay. The findings revealed that 82.8% of participants had normal troponin levels, while 17.2% exhibited elevated levels. The highest frequency of elevated troponin was observed in the 40–59 age group. Furthermore, males accounted for 55% of the elevated cases compared to 45% for females. The results confirm that cardiac troponin I serves as a highly specific and sensitive biomarker for detecting myocardial injury. Its integration into clinical protocols enables timely diagnosis and improved patient management in cases of acute myocardial infarction.

Keywords: Troponin I, Heart disease, Acute myocardial infarction.

المخلص

أجريت هذه الدراسة لتوضيح القيمة التشخيصية والأهمية السريرية لتروبونين (I) في تحديد أمراض القلب، خاصة في حالات الطوارئ. شملت الدراسة 116 شخصاً بالغاً، تتراوح أعمارهم بين 35 و80 عاماً، ممن ظهرت عليهم أعراض تشير إلى الإصابة بأمراض القلب. تم استبعاد المرضى الذين يعانون من أمراض الكلى المزمنة أو الإصابات الحديثة لتجنب تداخل النتائج. تم تحليل عينات الدم الوريدي لقياس مستويات التروبونين (I) باستخدام المقاييس المناعية للتألق الكهرومغناطيسي. كشفت النتائج أن 82.8% من المشاركين لديهم مستويات طبيعية من التروبونين، بينما أظهر 17.2% مستويات مرتفعة. لوحظ أن الفئة العمرية (40-59) هي الأكثر تسجيلاً لارتفاع مستويات التروبونين. وعلاوة على ذلك، بلغت نسبة الذكور 55% من الحالات المرتفعة مقارنة بـ 45% للإناث. تؤكد النتائج أن تروبونين القلب (I) يعمل

كعلامة حيوية ذات حساسية وخصوصية عالية للكشف عن إصابات عضلة القلب. إن دمج هذا الفحص في البروتوكولات السريرية يساهم في التشخيص المبكر وتحسين إدارة المرضى في حالات احتشاء عضلة القلب الحاد.

الكلمات المفتاحية: التروبونين (I)، أمراض القلب، احتشاء عضلة القلب الحاد.

1. Introduction

According to the World Health Organization (WHO), heart disease and its various complications claim the lives of over 17.9 million people annually, establishing cardiovascular diseases (CVD) as the leading cause of global mortality. Among these, heart attacks and strokes are responsible for more than 80% of CVD-related deaths (Ahsan et al., 2021). Heart disease (HD) typically presents with debilitating symptoms such as acute shortness of breath, generalized physical weakness, and peripheral edema, particularly swollen feet. Given the progressive nature of these conditions, implementing effective diagnostic tools and timely therapeutic interventions is paramount to reducing mortality rates and improving patient quality of life (Durairaj & Ramasamy, 2016).

At the molecular level, troponins are regulatory proteins expressed by specific genes within skeletal and cardiac muscle tissues (De Backer et al., 2003). In the event of acute ischemia, the lack of oxygen leads to cardiomyocyte necrosis. This process results in the destruction of cell membranes and intracellular organelles, which subsequently releases cytoplasmic proteins into the systemic circulation. Consequently, the concentration of cardiac troponin (cTn) in the blood rises significantly following myocardial injury, typically reaching peak levels within 10 to 20 hours after the onset of symptoms (Bertinchant et al., 1996; Laugaudin et al., 2016).

Traditionally, the diagnosis of heart disease relied heavily on clinical history, physical examination, and the physician's subjective analysis of presenting symptoms. However, relying solely on these traditional methods often lacks the necessary precision to accurately identify patients at high risk for HD. Furthermore, advanced imaging and traditional diagnostic procedures can be prohibitively expensive, time-consuming, and computationally complex to interpret in acute care settings (Thygesen et al., 2018).

The introduction of cardiac troponin I (cTnI) and troponin T (cTnT) as biomarkers has revolutionized clinical cardiology. These proteins are now utilized as highly specific and sensitive indicators of myocardial injury, particularly in the diagnosis of acute myocardial infarction (AMI). As these proteins are essential to the contractile function of cardiac muscle, their presence in the bloodstream is a definitive sign of myocardial cell damage (Wang et al., 2020). The measurement of troponin levels has fundamentally altered the clinical approach to patients presenting with acute chest pain or suspected acute coronary syndrome (ACS). Modern clinical guidelines emphasize that elevated levels of cTnI or cTnT, characterized by a dynamic rise and/or fall over time, are primary criteria for confirming AMI. Beyond diagnosis, increased troponin levels serve as powerful prognostic indicators, as they are strongly associated with adverse cardiovascular events and increased mortality risk (Hartikainen et al., 2020).

Despite the widespread use of these markers, there is a continuous need to evaluate their diagnostic performance across different demographic groups and clinical environments. Therefore, this study aimed to further elucidate the critical role of Troponin I as a sensitive and specific biomarker in the diagnosis of heart diseases, providing evidence-based insights into its clinical application.

2. Materials and Methods

This cross-sectional study was conducted to evaluate the diagnostic efficacy of cardiac Troponin I (cTnI) in patients suspected of having myocardial injury. The study cohort consisted of 116 adult participants, ranging in age from 35 to 80 years, who presented to the clinical

setting with acute chest pain and other clinical manifestations indicative of cardiovascular disease. To ensure the accuracy of the troponin measurements and to maintain high diagnostic specificity, strict exclusion criteria were applied. Patients with a documented history of chronic kidney disease (CKD) or those who had experienced recent physical trauma were excluded from the study, as these conditions are known to cause non-cardiac elevations in troponin levels, which could lead to confounding results (Thygesen et al., 2018; Jarolim, 2015).

2.1. Blood Sampling and Preparation

Venous blood samples (4 ml) were meticulously collected from each participant via venipuncture using sterile techniques. The samples were immediately transferred into tubes containing Ethylenediaminetetraacetic acid (EDTA) as an anticoagulant. To preserve the integrity of the cardiac biomarkers, the samples were processed within 20 minutes of collection. The blood was centrifuged at a standardized speed to achieve efficient separation of the plasma. Following centrifugation, the supernatant (plasma) was carefully harvested, aliquoted, and stored at appropriate temperatures until the biochemical analyses were performed.

2.2. Biomarker Assays (ELISA Protocol)

The quantitative determination of plasma Troponin I levels was performed using a high-sensitivity electrochemiluminescence immunoassay based on the Enzyme-Linked Immunosorbent Assay (ELISA) technique. The assay followed a rigorous multi-step protocol:

1. **Incubation and Binding:** To initiate the assay, 100 μL of standardized Troponin I solutions (for the calibration curve) and diluted patient samples were dispensed into the pre-coated designated wells of the ELISA plate. The plate was sealed and incubated at 37°C for 60 minutes, facilitating the specific binding of the Troponin I antigen to the immobilized antibodies.
2. **Washing Phase:** Post-incubation, the wells were subjected to a rigorous washing cycle (3–5 times) using a specialized wash buffer to eliminate any unbound proteins or detached compounds that might interfere with the signal.
3. **Antibody Application:** 100 μL of biotin-labeled anti-Troponin I antibody was added to each well. The plate was then incubated again at 37°C for 60 minutes to form the "antibody-antigen-antibody" sandwich complex.
4. **Enzyme Conjugation:** After another washing step to remove excess detection antibodies, 100 μL of streptavidin-HRP (Horseradish Peroxidase) solution was introduced. The high affinity of streptavidin for biotin ensured the stable attachment of the enzyme conjugate. The plate was incubated for an additional 30 minutes at 37°C.
5. **Colorimetric Development:** After a final wash, 100 μL of Tetramethylbenzidine (TMB) substrate was added. The plate was then incubated in a dark environment at room temperature for 10–15 minutes, during which a blue color developed in proportion to the concentration of Troponin I.
6. **Detection:** The enzymatic reaction was terminated by injecting 100 μL of an acidic stop solution into each well, which shifted the hue from blue to yellow. The optical density (absorbance) was measured within 10 minutes using a professional ELISA microplate reader at a wavelength of 450 nm.

2.3. Statistical Analysis

The collected data were systematically organized and analyzed using descriptive statistical techniques. Categorical variables, including gender and troponin status (normal vs. elevated), were summarized using frequencies, proportions, and percentages. The mean and age distribution were also calculated to describe the study population's demographics. All statistical computations and data management were performed using the IBM SPSS Statistics software, version 20.0 (IBM Corp., Armonk, NY, USA). This approach ensured a robust interpretation of the prevalence and distribution of elevated troponin levels across the different study subgroups.

3. Results

The average age of the 116 participants was 35 years. The results showed that 82.8% of the samples had normal troponin I levels (n=96), whereas 17.2% (n=20) had elevated levels (Table 1). The highest troponin I levels were found in the 40–59 age group, as evidenced in Figure 1. Regarding gender distribution among those with elevated levels, 55% were males and 45% were females (Figure 2).

Table 1: Comparison between normal and high troponin I rates

Status	Frequency	Percent (%)	Valid Percent (%)	Cumulative Percent (%)
Normal	96	82.8	82.8	82.8
High	20	17.2	17.2	100.0
Total	116	100.0	100.0	-

(Note: Data analyzed using IBM SPSS Statistics v20.0)

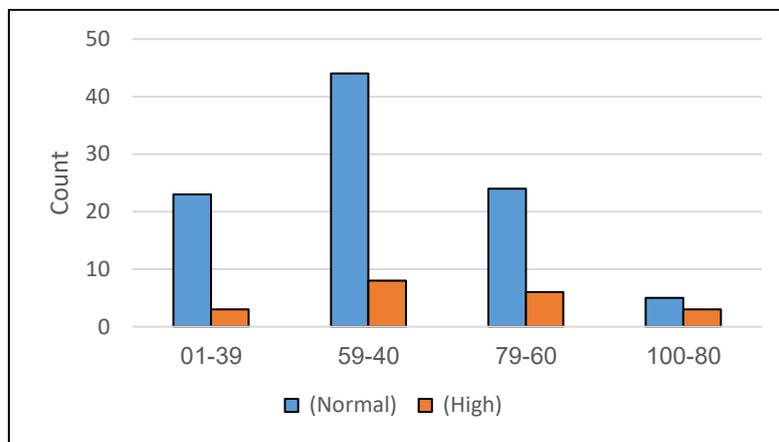


Figure 1: Normal and high troponin I levels in each age group.

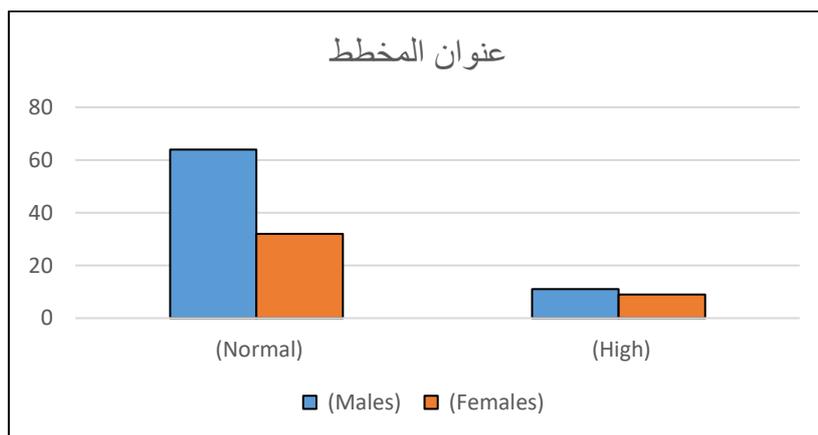


Figure 2: Normal and high levels seen in both genders, with males exhibiting the highest frequency of elevated levels.

4. DISCUSSION

Cardiac troponin I and T (cTnI and cTnT) assays are now universally recognized as the "gold standard" for the biochemical detection of myocardial injury. According to the most recent universal definition of myocardial infarction, the detection of a rise and/or fall of cardiac

troponin (cTn) values is an essential requirement for diagnosing acute myocardial infarction (AMI) (Thygesen et al., 2018). In the present study, significant elevations in troponin I levels were identified in 17.2% of the symptomatic participants. The findings highlighted that the 40–59 age group exhibited the highest frequency of elevated troponin levels, with a notable predominance in males (55%). This demographic trend is consistent with the findings of Clerico et al. (2023), who suggested that middle-aged males often present with a higher burden of cardiovascular risk factors, leading to a higher incidence of acute coronary syndromes. The clinical significance of Troponin I extends beyond simple diagnosis. Research indicates that patients with even minor elevations in cTnI levels are considerably more likely to experience adverse cardiac outcomes, including heart failure and in-hospital mortality (Smith et al., 2020). The high sensitivity of modern assays allows for the detection of minimal myocardial damage that was previously undetectable. Furthermore, in patients with chronic comorbidities such as diabetes mellitus and chronic renal failure, elevated cTnI levels have been strongly correlated with an increased risk of long-term cardiovascular death (Lee et al., 2019). This suggests that troponin serves not only as a diagnostic tool for acute events but also as a marker of chronic myocardial strain and ongoing subclinical injury.

Aside from its primary role in AMI, cardiac troponin has emerged as a critical prognostic indicator across a spectrum of both cardiac and non-cardiac diseases. Elevated levels, even in the absence of classic ischemic symptoms or EKG changes, are associated with a heightened risk of severe cardiovascular events, including life-threatening arrhythmias and sudden cardiac death (Giannitsis et al., 2010). The specificity of Troponin I to cardiac tissue makes it a superior biomarker compared to older markers like CK-MB, as it minimizes the risk of false positives from skeletal muscle injury, thereby enhancing the clinician's ability to provide an accurate and rapid diagnosis in emergency settings (Jarolim, 2015).

5. CONCLUSION

In conclusion, this study reaffirms that Cardiac Troponin I has transformed the landscape of cardiovascular diagnostics by providing unrivaled specificity and sensitivity for the detection of myocardial injury. The results demonstrate that cTnI is an indispensable tool in clinical decision-making, particularly for identifying high-risk patients in the 40–59 age group. Integrating these biomarkers into standardized clinical protocols facilitates timely intervention, optimizes patient management, and significantly improves survival outcomes in cases of acute myocardial infarction. As assay technology continues to advance, the move toward high-sensitivity troponins promises even earlier detection of cardiac events, further empowering clinicians to intervene effectively and reduce the global burden of cardiovascular mortality.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that they have no conflict of interest.

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