Comparative Analysis of Abnormal Troponin and ST Deviations in Diagnosis of Myocardial Infarction

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Abstract

Introduction: Troponin levels are vital for diagnosing myocardial infarction (MI) and assessing cardiac chest pain. While traditionally used for MI, elevated troponin is also observed in various cardiovascular and non-cardiovascular conditions. Recent research across Nigeria indicates an increasing trend in MI cases and coronary artery disease risk factors. This study aimed at examining the connection between troponin levels, ECG abnormalities, and causes of heightened troponin and abnormal ECG.

Method: The study included 47 patients with elevated troponin levels from 1266 who underwent troponin testing at GoodHeart Medical Consultants Hospital between January 2018 and December 2022. ECG data were assessed, considering ST elevation or depression as present if the ST-segment sloped upward or downward for at least 0.08s in one or more of the 12 leads, excluding aVR.

Results: All patients in the study had 100% compliance with ECG recordings and troponin measurements. The mean age was 61 ± 16.58 years, with 53% males and 47% females. Hypertensive heart disease affected 74.47% of the population. Over five years, abnormal troponin prevalence was 3.71% (n = 47/1266), with myocardial infarction prevalence at 46.81%. Among those with myocardial infarction, 72.73% showed ST elevation, 22.73% had ST depression, and 4.54% showed neither.

Conclusion: The study demonstrated the complementary roles of cardiac troponin (cTn) status and quantitative ST deviation in assessing Acute Coronary Syndrome (ACS) patient risk, usable dependently or independently for prognosis. While both are valuable risk indicators, cTn proves more helpful than ST elevation in diagnosing non-ST-segment elevation myocardial infarction (NSTEMI).

Keywords: Myocardial infarction; Elevated troponin; Electrocardiogram; ST Elevation

1. Introduction

Cardiac Troponin (cTn) represents a pivotal protein complex involved in the contraction of the myocardium. Detectable levels of cTn in the bloodstream serve as a critical marker for assessing damage to the heart muscle. The measurement and interpretation of troponin levels play a significant role in diagnosing acute myocardial infarction (AMI) and evaluating cases of potential cardiac chest pain (1).
It’s important to note that while the presence of troponin in the bloodstream is not exclusive to myocardial infarction, it demonstrates an exceptional level of sensitivity in the diagnosis of AMI. The high-sensitive troponin tests have demonstrated their significant utility in accelerating the diagnosis of AMI and forecasting the occurrence of cardiovascular disease (CVD) in individuals who may lack symptoms or any prior record of CVD (2). These assessments hold the promise of improving the speed at which AMI can be diagnosed and offer valuable information about the risk of developing cardiovascular disease, often before any symptoms become apparent.

Measurable levels using modern or high-sensitive troponin tests serve as a clear indicator of cardiac injury in acute coronary syndrome (ACS) and various other pathological conditions, encompassing heart failure (HF), pulmonary embolism, and arrhythmias (3).

Three different clinical presentations—unstable angina, non ST-segment elevation MI (NSTEMI), and ST-segment elevation MI (STEMI)—are indicative of acute coronary syndrome (4). The existence of myocardial infarction symptoms, abnormalities in the electrocardiogram (ECG), and cardiac biomarkers, especially cardiac troponins, are used to differentiate between these subtypes (5). When deciding whether patients need higher-level monitoring—such as telemetry or the cardiac care unit—troponin can be a helpful metric.

Troponin testing (assay) has always been used to diagnose MI but the presence of elevated troponin levels does not really mean one has MI, so this study is to examine or find out if everyone who did troponin testing with elevated troponin level result at GoodHeart Medical Hospital was diagnosed of MI or other heart conditions and also to compare with their ECG to know if troponin alone can be used to diagnose MI.

Recent research in the majority of Nigeria’s states has revealed a progressive deterioration of MI cases and risk factors for coronary artery disease (CAD). In a five-year evaluation of CAD in Kano, Nigeria, 22 patients were treated (6). Similarly, four acute MI cases that were reported in 2003 in Zaria, Northern Nigeria, raised concerns about its prevalence (7). A tertiary hospital in Ilorin, North-Central Nigeria has reported a rise in the number of MI cases among patients hospitalized to our medical units since 2006 (8).

This study is aimed at determining the relationship between levels of abnormal troponin and ECG abnormalities (ST deviation) in a newly established private tertiary hospital. It also assesses the prevalence of abnormal troponin and myocardial infarction (MI), and assess if there are patient with abnormal troponin with any deviation in the ST segment of their ECG result.

2. Materials and methods

2.1. Study design/population

The medical records of forty-seven patients with abnormal troponin T and I level over a five-year period (January 2018 to December 2022) at Goodheart Medical Consultants Hospital laboratory information system were retrieved for analysis.

The information extracted included factors such as age, gender, height, blood pressure, pulse, TnT, TnI, S-T Elevation, S-T depression, Rhythm, ECG indication and mortality.

2.2. Troponin measurement

All troponin quantitative measurement done in the GoodHeart Medical Consultants were done using Point of Care Test (POCT) machine. The ‘Cobas’ machine measured troponin T (TnT) while the ‘Ichromax’ machine measured for troponin I (TnI). The normal range for TnI <0.10 ng/mL and that of TnT is <50 ng/mL.

2.3. ECG parameters

All ECG data were evaluated from the electronic database at the ECG room of GoodHeart Medical Consultants and also from the ECG printouts in the patients’ folders.

The ECGs were recorded in 12-lead format at a paper speed of 25 mm/s. The ST elevation and depression was judged to be present if there was an upward or downward sloping ST-segment for at least 0.08s respectively in one or more of the 12 leads except aVR.
2.4. Statistical Analysis

A descriptive analysis of the included studies was performed. MI prevalence for the study was calculated by dividing the number of patients diagnosed with MI by the total number of patients evaluated in the study. Statistical analysis was undertaken using Excel 2016 and SPSS V15. Descriptive analysis and cross tabulation were used for the results in forms of tables and Bar chart.

3. Results

3.1. Patients

A total of 47 patients with elevated troponin levels were screened in GoodHeart Medical Consultants Hospital during the study period. The frequency of ECG recordings and troponin measurements for each patient, was at 100% for all included in the study.

The prevalence of abnormal/elevated troponin levels within the five - year period calculated by

\[
\text{Prevalence} = \frac{\text{Abnormal troponin levels}}{\text{Total number of patients}} \times 100
\]

The prevalence of abnormal troponin is 3.71%

Fig 1 shows the year distribution. This study analyzed abnormal troponin level between year 2018 and 2022. The highest number of abnormal troponin was recorded in year 2020. The second highest was in year 2019, then 2018. The lowest number of abnormal troponin was recorded in year 2021.

Patient characteristics according to ST segment deviation (Elevation and Depression) are summarized in Table 1. The mean ± standard deviation age of the patients was 61 ± 16.58 years and 25 (53.19%) were males while 22 (46.81%) were females.

Patients with ST depression were older, more of female as compared to male, and less likely to have undergone prior percutaneous coronary intervention. These patients had higher heart rates and diastolic blood pressure (BP). Patients with ST elevation most likely had hypertensive heart disease (HHDX) and myocardial infarction.

![YEAR DISTRIBUTION](image)

**Figure 1** The distribution of number of patients with abnormal troponin across the years
### Table 1  Patient clinical characteristics

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Total</th>
<th>ST elevation</th>
<th>ST depression</th>
<th>No ST deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years; mean ±SD)</td>
<td>61 ± 16.58</td>
<td>60.15 ± 15.13</td>
<td>68.23 ±17.12</td>
<td>54.44 ± 18.47</td>
<td></td>
</tr>
<tr>
<td>Male (n; %)</td>
<td>25 (53.19)</td>
<td>18 (72)</td>
<td>6 (24)</td>
<td>1 (4)</td>
<td></td>
</tr>
<tr>
<td>Female (n; %)</td>
<td>22 (46.81)</td>
<td>8 (36.36)</td>
<td>7 (31.82)</td>
<td>7 (31.82)</td>
<td></td>
</tr>
<tr>
<td>Heart Rate (bpm; mean ±SD)</td>
<td>86.55 ± 21.62</td>
<td>87 ± 20.04</td>
<td>89.4 ±25.78</td>
<td>78.0 ±15.60</td>
<td></td>
</tr>
<tr>
<td>Systolic (mmHg; mean ±SD)</td>
<td>168 ±63.49</td>
<td>175.33 ± 67.37</td>
<td>152.92 ±58.44</td>
<td>173.89 ±60.47</td>
<td></td>
</tr>
<tr>
<td>Diastolic (mmHg; mean ±SD)</td>
<td>74.15 ±23.00</td>
<td>75.96 ±23.92</td>
<td>74.77 ±25.59</td>
<td>69.33 ±13.19</td>
<td></td>
</tr>
<tr>
<td>Diagnosis (n; %)</td>
<td>HHDX</td>
<td>35 (74.47)</td>
<td>20 (42.55)</td>
<td>11 (23.40)</td>
<td>5 (10.64)</td>
</tr>
<tr>
<td></td>
<td>Myocardial infarction</td>
<td>22 (46.81)</td>
<td>16 (72.73)</td>
<td>5 (22.73)</td>
<td>1 (4.54)</td>
</tr>
</tbody>
</table>

HHDX, Hypertensive Heart Disease; SD, Standard deviation

### Table 2  Troponin level and ST elevation categories

<table>
<thead>
<tr>
<th>Troponin Ranges (ng/mL)</th>
<th>1 mm ST Elevation</th>
<th>2mm ST Elevation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponin T</td>
<td></td>
<td></td>
</tr>
<tr>
<td>100 - 300</td>
<td>1</td>
<td>13</td>
</tr>
<tr>
<td>301 - 600</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>701 - 1000</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>&gt;1000</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Troponin I</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.11 – 1.0</td>
<td>1</td>
<td>14</td>
</tr>
<tr>
<td>1.01 – 2.0</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>2.01 – 3.0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>&gt;3.0</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

Total number of patient that did troponin T = 33
Total number of patient that did troponin I = 33

Majority (13) of the patients who had ST elevation had troponin T levels between 100 and 300 ng/mL. Also, majority (14) of the patient who had ST elevation had troponin I levels between 0.11 and 1.0 ng/mL.

### Table 3  Troponin and ST depression categories

<table>
<thead>
<tr>
<th>Troponin Ranges (ng/mL)</th>
<th>1 mm ST Depression</th>
<th>2mm ST Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponin T</td>
<td></td>
<td></td>
</tr>
<tr>
<td>100 - 300</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>301 - 600</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>701 - 1000</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>&gt;1000</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Troponin I</td>
<td>0.11 – 1.0</td>
<td>5</td>
</tr>
<tr>
<td>-----------</td>
<td>------------</td>
<td>---</td>
</tr>
<tr>
<td></td>
<td>1.01 – 2.0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>2.01 – 3.0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>&gt;3.0</td>
<td>1</td>
</tr>
</tbody>
</table>

In terms of ST segment depression majority (5) of the patient whose ECG shows depression had troponin T levels between 100 and 300ng/mL and also elevated troponin I levels between 0.11-1.0ng/mL.

4. Discussion

The prevalence of abnormal (elevated) troponin within the 5 years in study, was 3.71% (n = 47/1266) this value was lesser than prevalence of 12.4% (n=114/918) found among patients presenting to the Emergency Department of a hospital in Scotland (9). The prevalence of myocardial infarction (MI) among the study population was 46.81%, this was similar to the prevalence of 47.8%(6) among forty six patients who were diagnosed to have ischemic heart disease in Aminu Kano Teaching Hospital, Kano, but much different from the prevalence which was found to range from 0.1 to 10.4% among a total of 92,378 participants from highly heterogeneous study populations in five different countries in sub-Saharan Africa (10) and much higher than the prevalence of 0.21% of a total 6647 admissions in a 3-year period in a tertiary hospital in Ilorin (8). This difference could be as result of size of the study population.

In the intensive care unit (ICU), elevated C-TN levels are frequently seen, and they can also arise from illnesses other than acute coronary syndromes (9). The Fourth Universal Definition of Myocardial Infarction states that acute myocardial injury identified by aberrant cardiac biomarkers along with acute myocardial ischaemia are the clinical criteria for the diagnosis of MI (11). In this single centre study of patients with abnormal troponin levels in a 5-year period, 46.81% of the total population had myocardial infarction, and of this population 72.73% had ST elevation while 22.73% had ST depression. These individuals with ST elevation met the diagnostic criteria for MI based on a typical rise or fall in elevated troponin measurements and ischemic changes on a 12-lead ECG, with ECGs performed as clinically indicated (12). Those who had MI with no diagnostic ST-elevation ECG abnormalities fell in the category of non-ST elevation MI (NSTEMI). It was also reported that the patients may have presented with typical chest pain which led to investigation for NSTEMI with subtle abnormalities on ECG, including ST-depressions and T wave changes (13).
74.47% of the study population had hypertensive heart disease (HHDX), with 42.55% having ST elevation on their ECG and 22.73% having ST depression. ST elevation is an indication of myocardial infarction while ST depression indicates myocardial ischemia (ischemic heart disease). This means that high blood pressure (BP) is a well-known risk factor for cardiovascular (CV) diseases, and interventions that lower BP have generally reduced CV events. Individuals with elevated levels of cTnT (high-sensitivity troponin-T) face a significantly higher risk of experiencing new cardiovascular (CV) events even when they fall within specific narrow categories of systolic blood pressure (SBP) (14). The risk is most pronounced in individuals with the highest cTnT levels within each SBP category, and this association is particularly robust in the context of heart failure (HF) (15,16). While previous studies, including the ARIC study, have examined the relationship between cTnT and various CV events, the significance of measuring high-sensitivity troponin-T as an indicator of the impact of blood pressure on the development of new CV outcomes had not been previously reported (17). The magnitude and rate of change of troponin can help differentiate type 1 from type 2 myocardial infarction and acute or chronic myocardial injury.

High-sensitivity troponin was independently linked to cardiovascular mortality and the development of CHF, but not MI, in a sizable observational cohort of patients with known coronary artery disease (CAD) who did not have acute coronary syndrome or congestive heart failure (CHF) (PEACE [Prevention of Events with Angiotensin Converting Enzyme Inhibition] trial population) (18). The patients were followed for a median of 5.2 years. The composite outcome of cardiovascular death and future MI over a mean follow-up of 4.5 years was found to be independently correlated with high-sensitivity troponin levels in comparable research of patients at high risk for CAD events (HOPE [Heart Outcomes Prevention Evaluation] study population (19).

Majority (n=13) of the patients who had ST elevation had troponin T levels between 100 and 300 ng/mL. Also, majority (n=14) of the patient who had ST elevation had troponin T levels between 0.11 and 1.0 ng/mL. In terms of ST segment depression majority (n=5) of the patient whose ECG shows depression had troponin T levels between 100 and 300ng/mL and also elevated troponin I levels between 0.11-1.0ng/mL. As part of the Thrombin Inhibition in Myocardial Ischemia (TRIM) sub study, investigation was carried out to ascertain the combined utility of ECG and biochemical testing for very early risk classification among patients with unstable coronary artery disease. 64 (14%) of the 470 patients whose ECGs could be read showed a ST depression of at least 1 mm in any lead. Both ST depression and cTnT 0.1 ng/ml were highly predictive of mortality or recurrent MI within 30 days in univariate analysis (20).

In a 2021 study, ST-segment depression (STD) was present in triage ECG of some of the study population. Some of the leads exhibited mild elevation of the ST-segment along with reciprocal STD in aVL. Angiography was delayed for some hours after presentation since none of their ECGs fit the criteria for ST-segment elevation myocardial infarction. Upon completion, a complete (TIMI [Thrombolysis in Myocardial Infarction]) left circumflex artery blockage was discovered and stented. These patients also had elevated cardiac troponin T levels (21).

5. Conclusion

This study showed that cardiac troponin (cTn) status and quantitative ST deviation (elevation and depression) were complementary in evaluating risk among patients with ACS, and both should be used to establish prognosis and support medical decision-making. Although cTn and ST deviation are both useful risk indicators, cTn seems to be more helpful than ST elevation in diagnosing NSTE MI. Furthermore, risk-assessment analysis should take into account values of cTnT less than 0.1 ng/ml, as they contain significant prognostic information. Further research is needed to understand how the different values of abnormal cTn can be compared to ST deviation on ECG within different time range.

Compliance with ethical standards

Acknowledgment

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Disclosure of conflict of interest

No conflict of interest to be disclosed.
Statement of ethical approval

Ethical approval for this study was obtained from the University of Port Harcourt Teaching Hospital Ethical Committee.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

References


