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Relationship between Severity of Coronary Artery Disease and Cardiac Troponin Levels in the Setting of Myocardial Infarction: A Prospective Observational Study

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ABSTRACT
Background & Aim: The measurement of cardiac troponin (cTn) is established as a crucial tool for the diagnosis of myocardial infarction (MI). However, there is a great deal of debate surrounding the relationship between the severity of coronary artery disease (CAD) and cTn levels. Therefore, we designed a prospective, observational study to compare the severity of CAD among MI patients who had achievedcTn positive (cTn+) or negative (cTn-) levels. Our secondary focus was to estimate the prevalence of elevated levels of cTn in the setting of MI.
Methods: A total of 70 consecutive patients aged 18 to 80 years who were diagnosed with MI during the period from November 2021 until April 2022, were included. Prospectively, data as to demography, thorough medical history, clinical examinations, cTn test, haematological test, and coronary angiography were collected. Subjects were categorized into cTn+ orcTn-groups based on the findings of cTn testing.(cTn+:>0.1-100ng/ml and cTn-:<0.04ng/ml).
Results: Of the 70 study patients, there were 25 (35.7%) patients with cTn+ and 45 (64.3%) patientswith cTn- levels. In cTn+group, patients were significantly less likely to develop single-vessel disease (18.5% vs. 44.7%; p = 0.023) and more likely to develop triple-vessel disease (40.7% vs. 17.0%; p = 0.025) in comparison with cTn-group.
Conclusion: Authors concluded that the positivity of cTn (>0.1-100ng/ml) can act as an integral part of diagnostic workup to assess the severity of CAD in the setting of MI.
Keywords: Coronary artery disease, myocardial infarction,prevalence,severity,single-vessel disease,triple-vessel disease,troponin

I. INTRODUCTION

Cardiac troponin (cTn) is the biomarker of choice in the diagnosis and prognosis for myocardial infarction (MI) in a clinical setting(1). It is a myocardial protein whose serum levels increase within 3 to 4 hours after the onset of cardiac symptoms in patients withMI(2). Due to its high sensitivity and specificity, elevated levels of cTnimplies the presence, not the mechanism, of myocardial damage. The introduction of cTn testing into routine laboratory practice hasdramatically revolutionized the diagnosis of MI in the past few decades. Consequently, based upon raised serum concentrations of cTn, the definition of MIhas been revised by the joint European Society of Cardiology (ESC)/American College of Cardiology (ACC) in 2000(3). The elevated levels of cTn werelinked to a higher incidence of multivessel coronary artery disease (CAD), as few publications had demonstrated(4, 5).However, the finding of elevated cTn in patients who were later found to have non-critical epicardial CAD at angiography has perplexed clinicians and raised concern about "false-positive" results with this biomarker (6). Moreover, there is a lack of published literature that has explicitlyaddressed the relation betweenthe severity of CAD and cTn levels. Hence, the present study aimed to bridge this gap by comparing the severity of CAD among MI patients who had achievedcTn positive (cTn+)or negative (cTn-) levels. An additional objective was to evaluate the prevalence of elevated levels of cTn in this subset.

II. METHODS

Study design

This was a prospectiveobservational studyconsisting of 70 consecutive MI patients. The

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study was undertaken at a tertiary health care centre during the periodfrom November 2021 until April 2022. Patients aged 18 to 80 years who were diagnosed with MI were included. The exclusion criteria were as follows:renal, muscle and liver impairment; cancer; patients who were not willing to give consent; pregnant and lactating women; or incomplete patients' medical data. The study protocol was approved by Institutional Ethical Committee (IEC) and the study was conducted according to the Declaration of Helsinki. All patients gave written informed consent.

Data collection

All patients were subjected toproviding demographic details and thorough history, clinical examination, cTn test, haematological test, and coronary angiography. All data were collected in a prospective, standardized manner. Based on the results of cTn testing, subjects were stratified intocTn+orcTn-groups.

Troponin measurements

Blood samples for cTn measurements were drawn into plain red colour tubes with no additives, and plasma for sample analysis was obtained after allowing the sample to form clot . The cTn was estimated using a cardiac troponin I fast test kit which works on the principle of antigen-antibody complex. In this there is a comparision between cTnI monoclonal antibody with conjugated fluorescence latex and humn-cTnI monoclonal anti-body coated on test line after test sample application)(MISPA REVO immunofluorescence quantitative analyser). The analytical sensitivity (lower detection limit) of this assay was 0.01 ng/mL.

III. DATA ANALYSIS

Data were analysed using the Statistical Package for Social Sciences (SPSS) version 15.0 (Chicago, IL, USA). Continuous variables were described as mean ± SD or categorical variables were reported as numbers and percentages. Variables between the two subgroups were compared using the chi-square test for categorical variables and the independent T-test for the continuous variables.

IV. RESULTS

A total of 70study patients were assessed, of which, there were 25 (35.7%) patients with cTn+ and 45 (64.3%) patientswith cTn- levels.Their demographic and clinical characteristics are compared in **Table 1,**which revealed that the two groups were comparable regarding age, sex, comorbidities (hypertension, diabetes, CAD, cerebrovascular accident, thyroid, stroke, and anterior wall MI withmoderate left ventricular function), chief complaints (shortness of breath, chest tightness, sweating, and palpitation), clinical examinations (>100 beats/min pulse rate, ≥140 mm Hg systolic blood pressure,≥90 mm Hg diastolic blood pressure), and laboratory data (>11,000cells/mm^{3}white blood count, haemoglobin) with insignificant p-values. Moreover, chest pain was the most commonpresentation in the cTn+group (p-value = 0.045).

As depicted in **Table 2**, the incidence of single-vessel disease (SVD) was significantly lower in thecTn+group as opposed to the cTn-group (18.5% vs. 44.7%; p = 0.023), while the incidence of triple-vessel disease (TVD) was significantly greater incTn+group than cTn-group (40.7% vs. 17.0%;p = 0.025).

V. DISCUSSION

The cTn, in particular, has been verified to bea highly sensitive and specific biomarker for myocardial injury in comparison with other traditional biomarkers. Pioneering work on this matter had been carried out earlier byAbd Elrahman et al.(7), wherein theyexamined the clinical values of serum total creatine kinase (CK) (Total-CK), CK myocardial band (CK-MB) isoenzyme, and total lactatedehydrogenase (LDH) in comparison with cTn-I in patients with acute coronary syndromes (ACS). The obtained findings confirmed cTn-I as the most sensitive biomarker as compared to the other traditional biomarkers (Total-CK, CK-MB, and LDH) in establishing the diagnosis of ACS during the first 48 hours after admission to the hospital. Several landmark studies had demonstrated that cTn was a more sensitive and specific marker of cardiomyocyte injury than CK, its MB isoenzyme (CK-MB), and myoglobin(8-10).

Until recently, the prognostic and diagnostic avails of cTn among MI patients withcTn+and cTn- levelsremain controversial. Here, we purported to compare the severity of CAD across these two groups.As envisioned, the present study showed a higher incidence of TVD and a lower incidence of SVDin thecTn+group, indicating the complexity of CAD with the elevated levels of cTn.This finding was backed up by the evidence of one prospective study published by deFilippi et al.(4)which showed more prevalence of multivessel disease, greater coronary narrowing, and frequently complex lesion morphology incTn+ CAD patients presented with chest pain without ischemic electrocardiographic changes.

Additionally, Heeschen et al.(5)presented thatpositivity of cTnwas mainly associated with complex and severely obstructive plaques in patients with refractory unstable angina.In the present study, majority of the caseshad TVD (40.7%) followed by DVD (33.3%), SVD (18.5%), andLMCAdisease (7.4%) in thecTn+group at troponin cut-off range of(>0.1-100ng/ml).Wong et al.(11)postulated thatthe incidences of SVD, DVD, and TVD were 39.2%, 31.4%, and 18%, respectively at troponin cut-off range of >0.01 ng/L, and these values were 42.9%, 36.4%, 20.7% at troponin cut-off range of >0.1 ng/L. Moreover, the incidence of SVD, DVD, TVD, and LMCA in the cTn+group were 26%, 40%, and 23%, 1%, respectively at troponin cut-off range of >0.1 ng/ml inpatients presented with CAD within chest painwithout ischemic electrocardiographic changes(4).

Addition finding was that the prevalence of elevated cTn in our study cohort was 35.7%.Through his prospective cohort study, Lim et al.(12)cited 26% prevalence of elevated levels of cTnin MI patients. Another study published by the same corresponding author quoted 19.9% prevalence of elevated levels of cTn(13).We found a high prevalence (35.7%) of elevated levels of cTnas compared to what had been found previously. Both the previous studies were conducted in England population whereasour findings were based on the Indian population.Altogether, the reported prevalence of elevated levels of cTn in MI patients has been widely varied.The observed discrepancy in frequency rates, attributed to the troponin threshold at which measurement considered positive and ethnic differences.

Several limitations of the present study merit discussion. First, the study was designed with a relatively small sample size of patients; and second, the study was an observational type of study, hence, many confounding factors might influence final outcomes. Last but not least, the study was conducted in the small localized Indian population; hence findings cannot be generalised to the entire population as cTn levelsshow significant interindividual variations. We suggest future multicentre studies with a large sample size in the Indian population. Moreover, a comparison of elevated cTnlevels across the different ethnicity will provide additional insights into this interesting matter.

VI. CONCLUSION

From this study, one can conclude that the positivity of cTn was associated with a lower incidence of SVD and a higher incidence of TVD. Besides, the prevalence of elevated troponin levels was considerably higher in the Indian population.

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Table legends
Table 1: Comparisons of demographic and clinical characteristics acrosscardiac troponinpositive andnegativegroups
Table 2: Comparison ofseverity of coronary artery disease acrosscardiac troponinpositive andnegativegroups

Table 1: Comparisons of demographic and clinical characteristics across cardiac troponinpositive andnegative groups

Variables	**cTn+(>0.1-100ng/ml)(n=25)**	**cTn-(n=45)**	**p-value**
Age (years)			
<60	19 (76%)	28 (62.2%)	0.240
≥60	6 (24%)	17 (37.8%)	
Sex			
Male	12 (48%)	27 (60.0%)	0.333
Female	13 (52%)	18 (40.0%)	
Comorbidities			
Hypertension	10 (40%)	19 (42.2%)	0.856
Diabetes	19 (76%)	35 (77.8%)	0.865
CAD	0	1 (2.2%)	0.453
CVA	1 (4%)	0	0.177
Thyroid	2 (8%)	1 (2.2%)	0.253
Stroke	0	1 (2.2%)	0.453
AWMI with moderate LV function	0	1 (2.2%)	0.453
Chief complaints			
Chest pain	24 (96%)	35 (77.8%)	0.045**
Shortness of breath	10 (40%)	15 (33.3%)	0.577

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Chest tightness	0 (0.0%)	1 (2.2%)	0.453
Sweating	25 (100%)	44 (97.8%)	0.453
Palpitation	4 (16%)	8 (17.8%)	0.850
Clinical examination			
Pulse rate (> 100 beats/min)	3 (12%)	4 (8.9%)	0.678
Systolic blood pressure (≥140 mm Hg)	5 (20%)	18 (40.0%)	0.088
Diastolic blood pressure (≥90 mm Hg)	10 (40%)	15 (33.3%)	0.577
Laboratory data			
White blood count (>11, 0000 cells/mm^3)	14 (56%)	25 (55.6%)	0.971
Haemoglobin (g/dL)	10.09 ± 2.15	10.58 ± 2.35	0.390

§ The resultsare expressed as mean ± SD or n (%).
Data were compared using independent T-test or chi-square test. * p <0.05. Abbreviations:
cTn, Cardiac troponin, CAD, Coronary artery disease; CVA, Cerebral vascular accident;
LV, Left ventricular;AWMI, Anterior wall myocardial infarction

Table 2: Comparison ofseverity of coronary artery disease across cardiac troponinpositive andnegativegroups

Angiographic findings	**cTn+(>0.1-100ng/ml)) (n=27)**	**cTn- (n=47)**	**p-value**
Single-vessel disease	5 (18.5%)	21 (44.7%)	0.023*
Double-vessel disease	9 (33.3%)	16 (34.0%)	0.950
Triple-vessel disease	11 (40.7%)	8 (17.0%)	0.025*
Left main coronary artery disease	2 (7.4%)	2 (4.3%)	0.564

§ The results are expressed as n (%).
Data were compared using chi-square test.*p <0.05. Abbreviations:cTn, Cardiac troponin.