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COMPARISON OF HIGH-SENSITIVITY TROPONIN I AND T IN PREDICTING EARLY CARDIAC RISK IN ACUTE CORONARY SYNDROME

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Abstract

Background: Acute Coronary Syndrome (ACS) is a major cause of morbidity and mortality in the world and risk stratification needs to be done quickly and accurately. High-sensitivity cardiac troponins, hs-cTnI and hs-cTnT, are important in the early diagnosis and prognostication but the relative effectiveness of the two in predicting early cardiac risk is not well understood especially in the local Pakistani population. ***Objective:*** The predictive validity of high-sensitivity Troponin I (hs-cTnI) versus Troponin T (hs-cTnT) in early cardiac risk stratification in patients presenting with ACS. ***Methodology:*** A potential observational study was carried out at Omer hospital and cardiac center Lahore, on 138 patients who came with ACS. The measurement of hs-cTnI and hs-cTnT in serial was performed at 0, 1, and 3 hours. Early cardiac risk was determined by in-hospital complications. ROC curve analysis, independent t-tests, and logistic regression that used SPSS-v27 were considered as statistical methods, and $p < 0.05$ was treated as significant. ***Results & Findings:*** No significant correlation was present between baseline clinical and demographic variables and early cardiac risk ($p > 0.05$), other than an association of prior myocardial infarction ($p = 0.013$). The dynamic changes in both biomarkers (03 hours hs-cTnI and 03 hours hs-cTnT) were significant in patients with early cardiac risk ($p = 0.01$ and 0.03, respectively). The predictor performance of Δ hs-cTnI (AUC = 0.63) was slightly better than that of Δ hs-cTnT (AUC = 0.60). In multivariate analysis, both of these biomarkers were predictors of early cardiac risk. ***Conclusion:*** High-sensitivity troponins dynamically change have been shown to be better predictors of early cardiac risk than baseline values, although hs-cTnI has a slight advantage over hs-cTnT, and should be used in combination with

clinical and ECG data. This research offers important local information to assist better early risk stratification measures in ACS patients.

Introduction

Acute coronary syndrome (ACS) encompasses a spectrum of conditions triggered by an abrupt limitation of coronary blood flow, resulting in myocardial ischemia and, without timely intervention, myocardial infarction or sudden cardiac death. Rapid and accurate diagnosis is essential, as any delay substantially increases morbidity and mortality. Conventional diagnostic tools electrocardiography and clinical assessment frequently fail to provide definitive information in the first hours after symptom onset, particularly in patients with atypical presentations such as women, the elderly, and individuals with diabetes mellitus [1]. High-sensitivity cardiac troponin (hs-cTn) assays have therefore become indispensable for early detection and risk stratification in suspected ACS. The isoforms hs-cTnI and hs-cTnT are highly sensitive and specific markers of myocardial injury; they are released into the circulation even after minimal ischemic damage, enabling earlier identification of ACS than conventional troponin tests [2]. Despite their widespread use, differences in release kinetics and diagnostic performance between hs-cTnI and hs-cTnT are well documented. hs-cTnI tends to rise slightly earlier after myocardial injury, potentially facilitating a more rapid diagnosis, whereas hs-cTnT levels are more frequently elevated in the presence of renal dysfunction and systemic inflammation, which can confound interpretation [3]. These disparities underscore the need to directly compare both biomarkers in the early phase of ACS to determine which assay is more reliable in specific patient subgroups. The introduction of high-sensitivity troponin assays has transformed early ACS management worldwide, allowing clinicians to detect myocardial injury within a few hours of symptom onset and promptly initiate antiplatelet therapy, reperfusion strategies, and intensive monitoring [4]. The choice of assay can influence clinical decision-making, as different troponin results may reclassify patients into rule-out, observation, or rule-in pathways, ultimately affecting treatment and outcomes. However, data from South Asian populations, including Pakistan, remain scarce. Ethnicity, comorbid burden, healthcare infrastructure, and laboratory capacity can all affect troponin kinetics and assay reliability [5]. Most Pakistani hospitals rely on international reference values and protocols that may not fully reflect the characteristics of the local population, and comparative data on hs-cTnI versus hs-cTnT performance in Pakistani emergency departments (EDs) are lacking [6]. A head-to-head evaluation in this setting would provide essential local evidence to guide early diagnostic algorithms, improve patient triage, and optimize resource allocation. Beyond diagnosis, high-sensitivity troponins carry important prognostic information. Early risk stratification helps clinicians identify patients at high risk of in-hospital complications including acute heart failure, malignant arrhythmias, recurrent ischemia, cardiogenic shock, and death and tailor management accordingly [7]. Serial changes in troponin concentration over 6–12 hours enhance both diagnostic accuracy and prognostic assessment; dynamic patterns of myocardial injury predict adverse cardiac outcomes more effectively than single measurements [8]. Elucidating the early rise patterns of hs-cTnI and hs-cTnT in a local cohort can offer practical guidance on which assay supports more reliable immediate clinical decisions, especially in resource-constrained EDs. Patient-related factors further complicate troponin interpretation. Age-related myocardial remodeling, chronic metabolic conditions such as diabetes and hypertension, and renal dysfunction can chronically elevate troponin levels in the absence of acute ischemia. Notably, hs-cTnT is disproportionately affected by impaired renal clearance and chronic myocardial stress, conditions highly prevalent in the Pakistani population [9]. Differentiating true ACS-related elevations from non-specific rises is therefore critical. By comparing hs-cTnI and hs-cTnT in a real-world ED setting, it is possible to identify which assay retains greater specificity

for ischemic myocardial injury amid the competing influences of prevalent comorbidities and non-cardiac conditions such as sepsis, pulmonary embolism, severe anemia, and hypertensive emergencies that also cause troponin release [10, 11]. This differentiation is particularly relevant for South Asian patients, who exhibit higher rates of diabetes, hypertension, and chronic kidney disease, all of which can confound biomarker performance. A previous investigation demonstrated significant discordance between hs-cTnI and hs-cTnT in patients with suspected ACS, particularly in the low-to-intermediate troponin range. While informative, that study was conducted in a selected trial population and does not fully reflect the dynamics of EDs in low- and middle-income countries [7]. This gap reinforces the need for comparative research in Pakistani tertiary-care hospitals to establish evidence-based, context-specific guidelines.

In Pakistan, healthcare delivery is often limited by shortages of beds, catheterization suites, and critical care units. Accurate early risk stratification is therefore imperative to prioritize high-risk ACS patients for urgent intervention while safely managing low-risk individuals through observation or early discharge. Selecting the most valid troponin assay for early risk detection could enhance the efficient use of scarce resources and alleviate overcrowding in emergency departments [8]. Although international studies have examined the prognostic value of hs-cTnI and hs-cTnT, region-specific evidence from Pakistan remains minimal, and much of the existing literature focuses on diagnostic accuracy or long-term outcomes rather than early in-hospital complications [9]. Differences in laboratory platforms, assay calibration, and reference cut-off values further limit the applicability of international findings to local practice, leaving clinicians without clear guidance on optimal biomarker selection during routine emergency care.

The present study was designed to address this gap by directly comparing the predictive performance of hs-cTnI and hs-cTnT for early cardiac risk in patients presenting with ACS in a Pakistani ED. The objectives were: (1) to compare the predictive power of hs-cTnI and hs-cTnT for early cardiac risk; (2) to determine the correlation of each biomarker with in-hospital complications; and (3) to identify confounding variables including age, sex, comorbidities, and electrocardiographic changes. We tested the null hypothesis that no significant difference exists between hs-cTnI and hs-cTnT in predicting early cardiac risk, against the alternative hypothesis that a significant difference does exist.

MATERIALS AND METHODS

This prospective observational study was conducted over a period of four months at the Cardiology and Emergency Department of Omer Hospital and Cardiac Center, Lahore. Ethical approval was obtained from the Institutional Review Board of Superior University, Lahore, and all procedures adhered to the principles of the Declaration of Helsinki. Written informed consent was secured from every participant after a thorough explanation of the study objectives, procedures, absence of anticipated risks, and the right to withdraw at any stage without affecting clinical care. Confidentiality was maintained by anonymizing all records, storing hard-copy data in locked cabinets, and protecting electronic files with password access. The sample size was calculated using the standard z-formula for proportions based on anticipated sensitivity and specificity of the troponin assays, and consecutive sampling was employed to enroll patients who met the eligibility criteria during the study period. Adults presenting with chest pain or other symptoms suggestive of acute coronary syndrome and an electrocardiogram indicating myocardial ischemia were included, provided they were able to give informed consent. Patients with known non-ischemic causes of troponin elevation (e.g., acute pulmonary embolism, myocarditis, or severe sepsis without acute coronary plaque rupture), those with end-stage renal disease requiring dialysis, and individuals who declined participation were excluded.

At the time of enrollment, demographic and clinical data were recorded on a structured case report form. Variables included age (completed years), gender, presenting symptoms (chest pain, dyspnea, diaphoresis), and the final type of acute coronary syndrome ST-elevation myocardial infarction, non-ST-elevation myocardial infarction, or unstable angina as determined by the attending cardiologist using standard criteria. The presence of traditional cardiovascular risk factors was documented: hypertension (documented history or current antihypertensive medication), diabetes mellitus (documented history or glucose-lowering therapy), and smoking status (current, former, or never). Venous blood samples were drawn immediately upon presentation for measurement of high-sensitivity troponin I and troponin T. High-sensitivity troponin I (hs-cTnI) was quantified using a high-sensitivity immunoassay on an Abbott/Siemens analyzer, while high-sensitivity troponin T (hs-cTnT) was determined on a Roche immunoassay platform. Both assays were performed according to the manufacturers' instructions, and concentrations exceeding the 99th percentile upper reference limit were considered indicative of myocardial injury. In addition to laboratory data, standard 12-lead electrocardiography was recorded using a dedicated ECG machine, and vital parameters, including oxygen saturation and blood pressure, were obtained with a pulse oximeter and sphygmomanometer. For the purposes of this investigation, early cardiac risk was defined as the occurrence of one or more in-hospital cardiac complications such as acute heart failure, malignant arrhythmias, recurrent ischemia, cardiogenic shock, urgent revascularization, or death within the initial 6 to 12 hours following symptom onset. This definition was operationalized as a binary outcome (yes/no) and served as the primary endpoint against which the predictive performance of hs-cTnI and hs-cTnT was evaluated.

All statistical analyses were performed using SPSS version 27.0. Continuous variables, including age and troponin concentrations, were summarized as mean ± standard deviation or median with interquartile range depending on distribution normality. Categorical variables gender, type of acute coronary syndrome, risk factors, and early cardiac risk status were expressed as frequencies and percentages. Differences in troponin levels between patients who experienced early cardiac events and those who did not were compared using the independent-samples t-test or the Mann–Whitney U test, as appropriate. The association between each biomarker category and early cardiac risk was examined with the chi-square test. Receiver operating characteristic curves were constructed for hs-cTnI and hs-cTnT to assess their discriminatory capacity, and the areas under the curves were compared to determine relative predictive power. Pearson or Spearman correlation coefficients were calculated to quantify the relationship between troponin levels and in-hospital complications. A two-sided p-value of less than 0.05 was considered statistically significant throughout.

RESULTS & FINDINGS

A total of 138 patients were enrolled in the study. The mean age was 60.1 ± 14.5 years. The average systolic and diastolic blood pressures were 134.8 ± 20.0 mmHg and 78.3 ± 11.3 mmHg, respectively. The mean heart rate was 84.7 ± 15.8 bpm, and the average oxygen saturation (SpO_2) was 96.1 ± 2.4% as shown in table 4.1

Table 4.1: Baseline Clinical Characteristics of 138 Patients Enrolled in the Study

Variable	Mean ± SD
Age (years)	60.10 ± 14.49
Systolic BP (mmHg)	134.76 ± 20.02

Diastolic BP (mmHg)	78.31 ± 11.27
Heart Rate (bpm)	84.73 ± 15.81
SpO_2 (%)	96.11 ± 2.39

Majority were male patients in this cohort as shown in figure 4.1

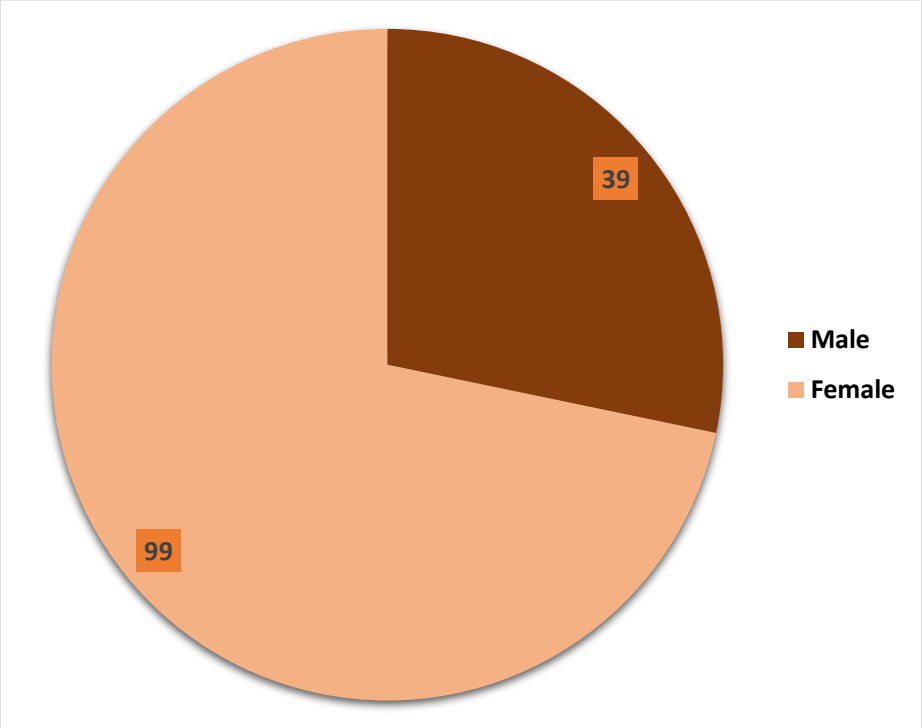

Figure:4.1 Gender distribution(n=138)

Most patients (42.0%) presented 1–3 hours after symptom onset. Regarding chest pain, radiating pain was the most common (42.8%), followed by pressure-type pain (34.8%).(Table 4.2)

Table 4.2: Time to Presentation and Chest Pain Characteristics (n = 138)

Variable	Categories	n(%)
Time at Presentation	<1 hour	38 (27.5)
	1–3 hours	58 (42.0)
	3–6 hours	42 (30.4)
Chest Pain Character	Radiating	59 (42.8)
	Pressure	48 (34.8)
	Tightness	31 (22.5)

Figure 4.2 shows the most common associated symptoms were diaphoresis and nausea/vomiting (26.1% each), followed by palpitations (25.4%) and combined diaphoresis with nausea (22.5%).

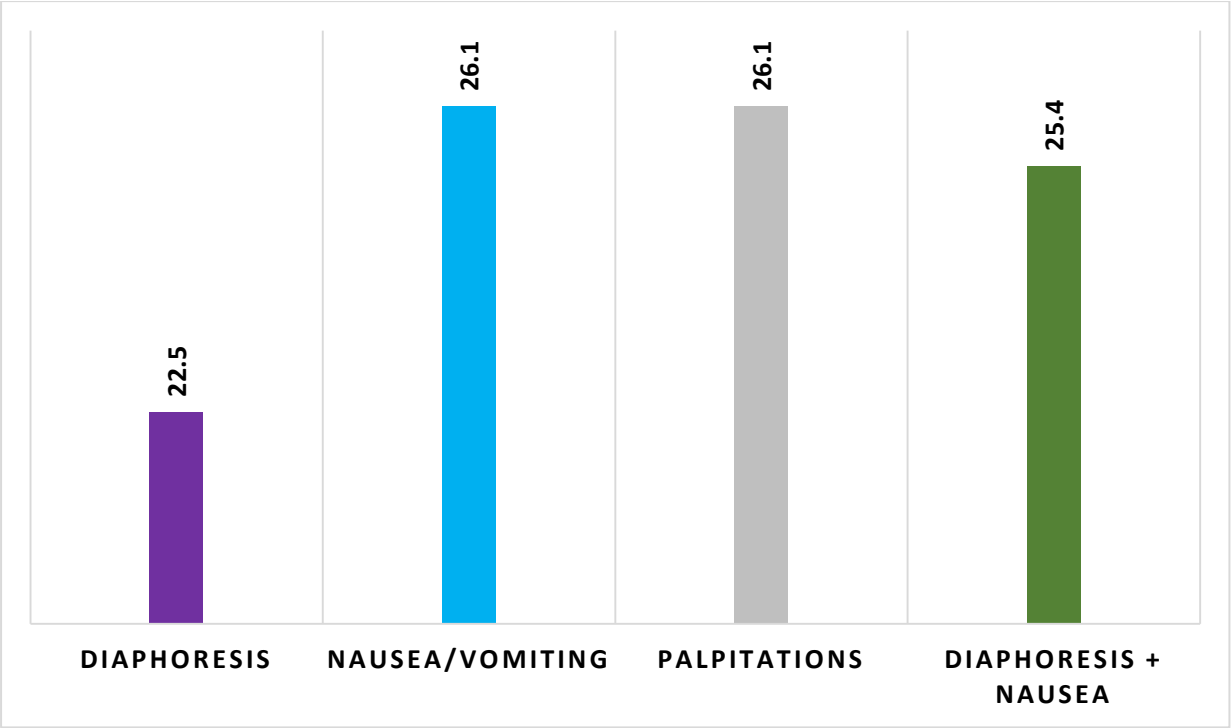

Figure 4.2: Frequency of Associated Symptoms in the Study Population (n = 138)

Among the 138 patients, hypertension was most common (64.5%), followed by diabetes (34.8%), smoking (34.1%), previous MI (18.8%), and chronic kidney disease (7.2%). (Figure 4.3)

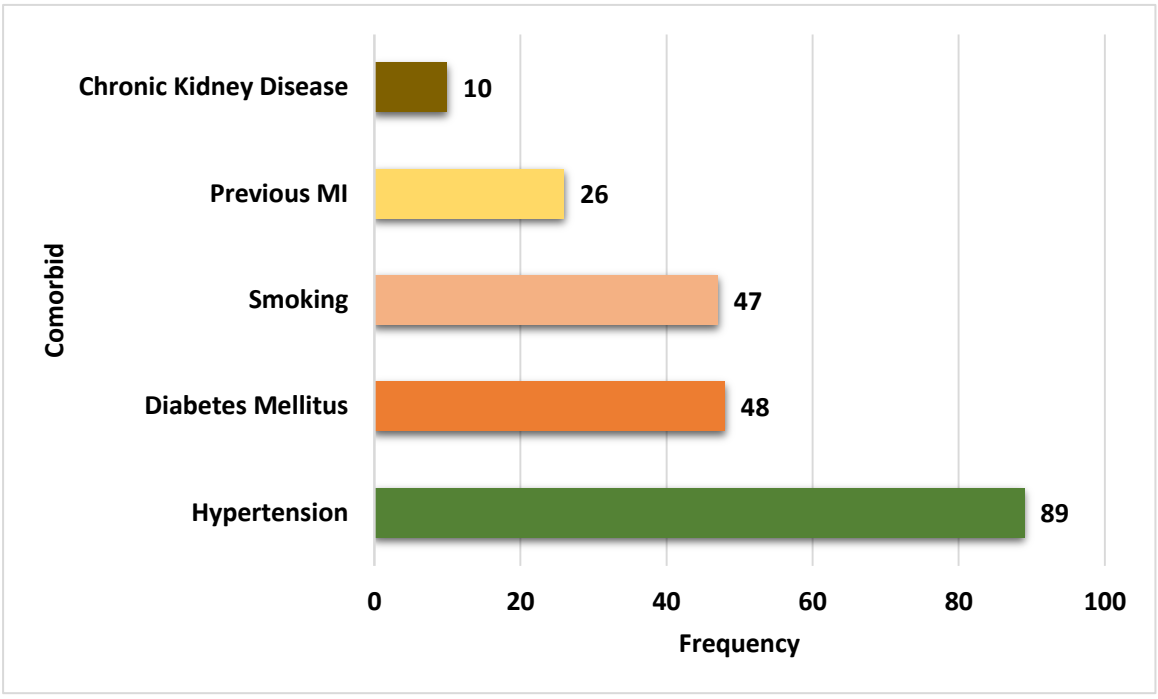

Figure 4.3: Prevalence of Comorbidities (n = 138)

The most prevalent ECG study observation was ST elevation at 37.0per cent which was seen in many patients, followed by T-wave inversion (27.5per cent) and ST depression (22.5per cent). Normal ECGs and non-specific changes were also less common as in 10.1% and 2.9% of patients respectively as indicated in table 4.3.

Table 4.3: Distribution of ECG Findings in the Study Population (n = 138)

ECG Finding	Frequency (%)
ST Elevation	51 (37.0)
T-wave Inversion	38 (27.5)
ST Depression	31 (22.5)
Non-specific	14 (10.1)
Normal	4 (2.9)

Among the patients, arrhythmias were the most common complication, occurring in 28.3%, followed by heart failure in 19.6%. Cardiogenic shock was less frequent, seen in 8.7% of the study population**. (**Figure 4.3)

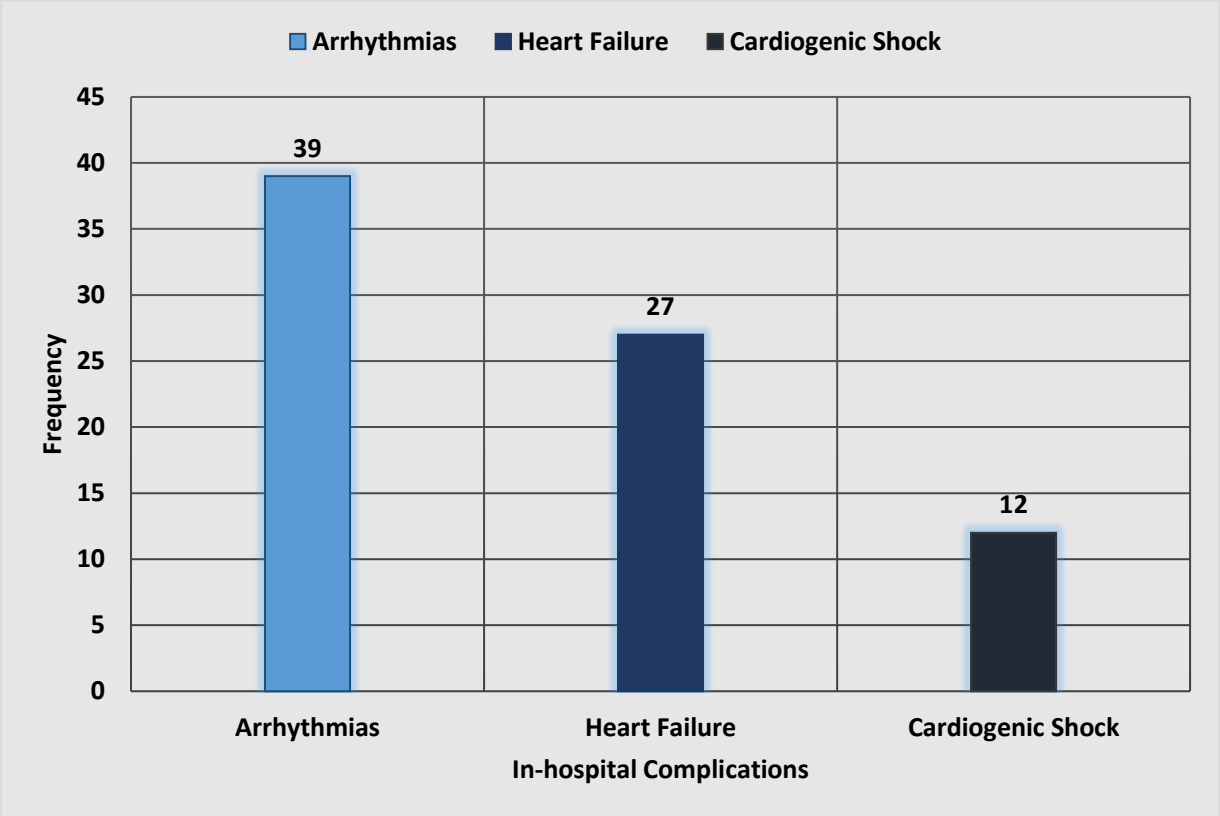

Figure 4.3: In-Hospital Complications Among the Study Population (n = 138)

INFERENTIAL ANALYSIS

There were no significant changes in most of the clinical and demographic factors such as gender, comorbidities (HTN, DM, CKD), smoking status, and ECG findings which were significantly linked to early cardiac risk ($p > 0.05$). It was only associated with previous MI whereby the patients who had a prior history of MI were found to be more likely to be classified as being at risk ($p = 0.04$) as categorized in table 4.4.

Table 4.4: Association of Demographic, Clinical, and ECG Variables with Early Cardiac Risk

Variable	Category	No Risk n (%)	Risk n (%)	p-value
Gender	Female	19 (25.7)	20 (31.3)	0.35
	Male	55 (74.3)	44 (68.8)	
HTN	No	25 (33.8)	24 (37.5)	0.61
	Yes	49 (66.2)	40 (62.5)	
DM	No	47 (63.5)	43 (67.2)	0.62
	Yes	27 (36.5)	21 (32.8)	
Smoking Status	No	46 (62.2)	45 (70.3)	0.34
	Yes	28 (37.8)	19 (29.7)	
Previous MI	No	65 (87.8)	47 (73.4)	0.04*
	Yes	9 (12.2)	17 (26.6)	
CKD	No	68 (91.9)	60 (93.8)	0.69
	Yes	6 (8.1)	4 (6.3)	
ECG Interpretation	Non-specific	10 (13.5)	4 (6.3)	0.23
	Normal	1 (1.4)	3 (4.7)	
	ST Depression	15 (20.3)	16 (25.0)	
	ST Elevation	31 (41.9)	20 (31.3)	
	T-wave Inversion	17 (23.0)	21 (32.8)	

HTN=hypertension: DM=Diabetes Mellitus; CKD=chronic kidney disease; ECG=Electrocardiography

The mean values of age, blood pressure, heart rate, SpO_2, and individual hs-Troponin I and T levels at 0, 1, and 3 hours were similar between patients with and without early cardiac risk ($p > 0.05$). However, the 0–3 hour change (Δ) in hs-Troponin I and T was significantly higher in patients at risk ($p = 0.01$ and 0.03, respectively), suggesting that these dynamic troponin changes are better predictors of early cardiac risk than baseline levels. (Table 4.5)

Table 4.5: Clinical Variables and hs-Troponin Changes Between Patients With and Without Early Cardiac Risk (n = 138)

Variable	No Risk (Mean ± SD)	Risk (Mean ± SD)	p-value
Age (years)	61.15 ± 14.56	58.89 ± 14.44	0.28
Systolic BP (mmHg)	133.76 ± 18.86	135.92 ± 21.37	0.52
Diastolic BP (mmHg)	77.31 ± 11.14	79.47 ± 11.39	0.28
Heart Rate (bpm)	83.30 ± 15.74	86.39 ± 15.85	0.29
SpO_2 (%)	96.00 ± 2.38	96.23 ± 2.43	0.64
hs-Troponin I 0 hr	74.85 ± 39.72	80.51 ± 41.95	0.38
hs-Troponin I 1 hr	140.18 ± 52.19	141.53 ± 60.22	0.87
hs-Troponin I 3 hr	236.90 ± 66.02	234.23 ± 78.51	0.78
Δ Troponin I (0–3 hr)	126.31 ± 60.97	156.82 ± 61.50	0.01*
hs-Troponin T 0 hr	63.30 ± 33.27	71.01 ± 38.04	0.18
hs-Troponin T 1 hr	116.24 ± 44.37	122.81 ± 36.05	0.42
hs-Troponin T 3 hr	190.50 ± 65.98	199.02 ± 55.93	0.43
Δ Troponin T (0–3 hr)	111.66 ± 64.34	135.50 ± 48.69	0.03*

BP= Blood Pressure; bpm= Beats per Minutes; SpO_2= Peripheral Oxygen Saturation; hs-Troponin I (hs-cTnI) = High-Sensitivity Cardiac Troponin I; hs-Troponin T (hs-cTnT) =High-Sensitivity Cardiac Troponin T; Δ Troponin I / Δ Troponin T – Change in hs=Troponin I / T over 0–3 hours

The 0–3-hour change in hs-Troponin I (ΔI) demonstrated better diagnostic performance for early cardiac risk than hs-Troponin T (ΔT), with an AUC of 0.63 versus 0.60. Both markers showed statistically significant predictive ability, although sensitivity and specificity were moderate as mentioned in Table 4.6 and Figure 4.4.

Table 4.6: Diagnostic Accuracy of Δ hs-Troponin I and T in Predicting Early Cardiac Risk

Variable	Cut-off	Sensitivity (%)	Specificity (%)	AUC (95% CI)	p-value
Δ hs-cTnI (0–3 hr)	**144**	**64**	**60**	**0.63 (0.53–0.72)**	**0.008***
Δ hs-cTnT	**136**	**53**	**50**	**0.60 (0.50–0.69)**	**0.043***

(0–3 hr)					

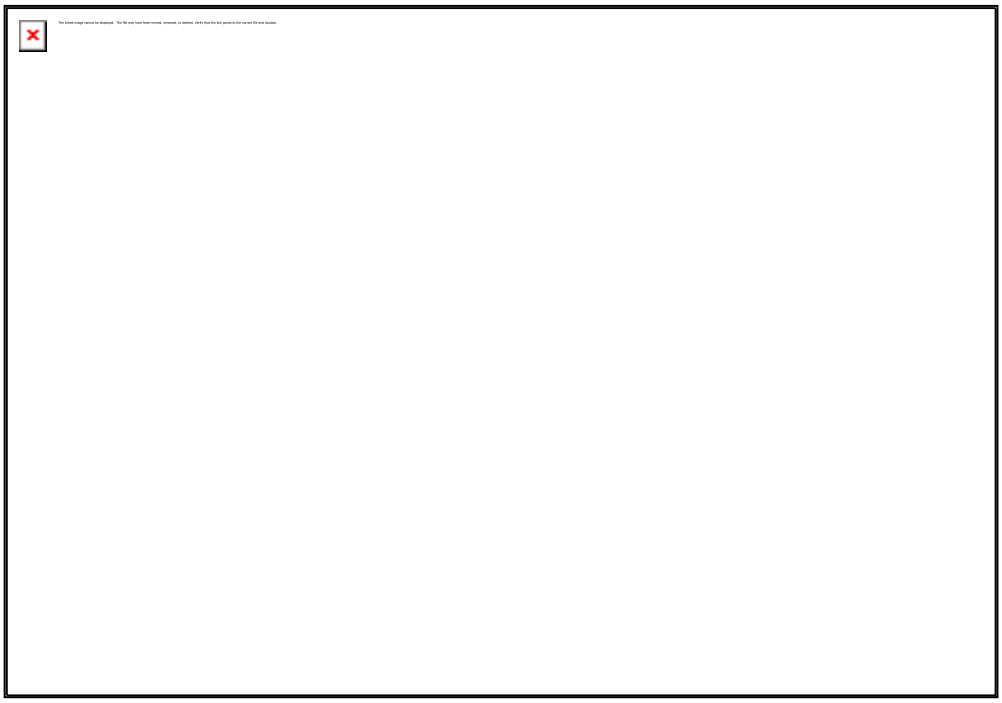

Figure 4.4: Receiver Operating Characteristic (ROC) Curve for Δ hs-Troponin I and Δ hs-Troponin T in Predicting Early Cardiac Risk

The previous MI in the multivariate logistic regression analysis had a significant relation with early cardiac risk with odds ratio being 0.273 (95% CI: 0.098-0.759, p = 0.013) that is, patients with prior MI were less likely to be considered high risk in this cohort. Neither 0 -hs -troponin I (0 -3 h) nor 0 -hs -troponin T (0 -3 h) remained insignificant in predicting the outcome. Additional predictors that were not statistically significant in this model were age, gender, high blood pressure, diabetes, smoking and chronic kidney disease (p > 0.05) as was observed in table 4.7.

Table 4.7: Logistic Regression Analysis of Predictors of Early Cardiac Risk (n = 138)

Variable	Odds Ratio	95% CI for OR	p-value
Age	0.989	0.963 – 1.015	0.396
Gender	0.837	0.334 – 2.100	0.705
Hypertension	0.890	0.380 – 2.080	0.787
Diabetes Mellitus	1.431	0.646 – 3.173	0.377
Smoking	1.787	0.709 – 4.504	0.218
Previous Myocardial Infarction	0.273	0.098 – 0.759	0.013*
Chronic Kidney Disease	2.247	0.514 – 9.820	0.282
Δ hs-Troponin I (0–3 hr)	1.007	1.001 – 1.013	**0.030***
Δ hs-Troponin T (0–3 hr)	1.007	1.000 – 1.014	**0.044***

DISCUSSION

Through this 138-patient study of suspected patients of acute coronary syndrome (ACS), clinical and demographic data including age, sex, hypertension, diabetes, smoking status, and initial ECG results showed little difference between patients with early cardiac risk and those without. Nevertheless, the extent of dynamic changes in the high sensitivity levels of cardiac troponin I and T within the first 3 hours (= hs cTnI and = hs cTnT) were markedly more elevated in at risk patients (p =.01 and.03, respectively) and predictors of early cardiac risk independently in the multivariate analysis (p =.05 and.05, respectively). Also, the risk status was linked to a history of prior myocardial infarction (p =.013) in the regression-modeling. This was accompanied by moderate diagnostic performance that was observed with Δ hs cTnI (AUC =.63), and with Δ hs cTnT (AUC =.60), which were found to have an incremental value compared to single baseline measurement. These results are in line with the increased number of evidence slated on the significance of serial hs troponin measurements and delta values in risk stratification of ACS. Previous researches have identified that serial high sensitivity troponin measurements are better at increasing prognostic accuracy than single measures and can be improved when included in fast triage algorithms. [10]. Both short term and long term adverse cardiovascular events such as myocardial infarction and death have been associated with serial changes in hs troponin and incremental predictive value is added even when baseline outcome is nondiagnostic [11]. The historic literature has shown that both cardiac troponin I and T increments have been proven to be antecedent to a high risk of adverse cardiac event within ACS groups, pooled odds ratios of 30 days averaging in the 4-5 range of risks are observed at elevation above normative cutoffs [12]. These papers show that cardiac troponins have broad applicability in risk stratification but did not particularly test delta changes. Additional data that lend more weight to the usefulness of high sensitivity troponin assay in selecting higher risk groups after ACS is more more recent real world data indicating that persistent elevations of high sensitivity troponin are associated with adverse outcome regardless of traditional risk factors and echocardiographic abnormalities [13]. These observations are in line with the finding of the present study that shows serial changes as opposed to non-comparable levels provided more information against early risk assessment.

There have been conflicting outcomes of comparative studies comparing the use of hs cTnI vs hs cTnT relying on the context of clinical use. A massive prospective study has shown that hs cTnI and hs cTnT had similar predictive capacity of major coronary lesions in non ST elevation ACS, with no apparent advantage of either of the assays over the other [14]. However, other studies with patient populations, including symptomatic atrial fibrillation, have reported hs cTnT had better predictive ability than hs cTnI of old myocardial infarction that necessitates coronary artery revascularization [15]. These inconclusive results demonstrate that comparative performance of the two troponin tests can be conditional on the precise clinical outcome and patient group, and the two tests are both useful, based on the objective of the diagnosis or clinical pattern. In this research, Δ hs cTnI and Δ hs cTnT both were predictors of early cardiac risk, although neither of the two markers had high discrimination (AUCs 0.60-0.63), which indicates that troponin dynamics cannot be used independently but should be considered together with other clinical and ECG variables. Although both sensitivity and specificity in our ROC analysis would be moderately high, the significance of 2 hs cTnI and 2 hs cTnT indicates that troponin kinetics can make a significant contribution to early risk, according to the analysis. This is also in agreement with modern guidelines that put importance on delta values in diagnostic and prognostic pathways in suspected ACS and acknowledge that dynamic changes provide better discrimination in comparison to single measurements [16]. Some of the possible confounders that we analyzed in our analysis included age,

gender, comorbidity burden, and ECG changes. The relative non-predictive power of these variables strong is consistent with some previous literature that did determine that traditional risk factors, although imperative to long term prognosis, did not actually reliably distinguish early high-risk presentations in the acute environment unless accompanied by available corroborative biomarkers [17]. On the same note, although ECG abnormalities form an essential diagnostic data, the relative value rates their prognostic utility after dynamic changes in troponin in the early risk stratification. This is favorable to the modern practice recommendations that encourage integrative assessment, a combination of serial troponin kinetics, along with clinical scores and ECG interpretation to enhance greater accuracy in early risk prediction. In this research or work the dynamic changes in high-sensitivity troponins (Δ hs cTnI and Δ hs cTnT) during the first 3 hours proved to be the important independent predictors of early cardiac risk, whereas clinical and demographic variables as well as ECG factors were not of much predictive use. Prior history of myocardial infarction was also another risk strident factor [18]. The ability of ROC analysis to diagnose both Δ hs cTnI and Δ hs cTnT showed moderate performances of 0.60 and 0.63 AU, respectively, indicating their added value to the performance of either of the single measurements. These results are consistent with the existing evidence on the prognostic significance of serial troponin measurements and delta values in ACS to use them as early markers of a high-risk group and to render clinical decisions, as well as the findings confirm that conventional risk determinants and ECG alterations are useful, but not defining.

FUTURE RECOMMENDATION

According to the results of the given research, serial high-level monitoring of troponin I and T may be included as a part of clinical procedures with patients presenting with both possible and actual cardiac risk cases in the first instance, since dynamic changes during the first three hours of the patient's course are highly predictive of cardiac toxicity manifestation. This troponin kinetics are to be used in combination with clinical evaluation and ECG results and not independently so as to have a comprehensive approach to stratification of risks and prompt management. Since Δ hs cTnI and Δ hs cTnT have moderate discriminative power, then future studies should concentrate on creating complete early risk predictive models which integrates the dynamics of troponin and other biomarkers with other imaging outcomes and previous clinical risk scores to increase the predictive power of early prognostic tests. Particular consideration is to be given to patients with history of past myocardial infarction since they were reported to be at high risk of early cardiac events meaning that they should be closely monitored and perhaps subjected to therapies sooner. Also bigger multicenter and longitudinal investigations are suggested to ascertain these conclusions and determine the long-term results and benefit in the creation of uniform guidelines. Healthcare providers should also receive education to help them optimize the detection and treatment of high-risk patients by educating them on the timing, interpretation, and clinical importance of serial troponin measurements. Lastly, this data confirms the role of delta troponin in institutional and national practice guidelines on ACS management to enhance the initial risk stratification and patient outcomes.

LIMITATIONS OF THE STUDY

This research project has a number of limitations. It was also single centered study, and this could limit the application of the results to a wide population. The sample was small, which may have limited the possibility of finding smaller associations of some variables. Early troponin changes were only measured in the first 3hours with no long-term outcomes or results of troponin kinetics. Moreover, there was a lack of adequacy in controlling possible confounding factors, that is, drugs, previous

interventions, and lifestyle factors. Lastly, despite the significant predictive ability of Δ hs cTnI and Δ hs cTnT on predicting early cardiac risk, their moderate prediction abilities would suggest that these factors should not be applied independently of other clinical and ECG variables.

CONCLUSION

High-sensitivity troponin I and T changes in the first 3 hours (Δ hs cTnI and Δ hs cTnT) were found to be significant independent predictors of the risk of early cardiac events, with nonsignificant predictive value exhibited by baseline demographics, comorbidities, and ECG outcomes. There was also the history of past myocardial infarction which corresponds to a greater risk in the early stage. ROC analysis showed that Δ hs cTnI and Δ hs cTnT had moderate levels of discriminatory ability (0.60-0.63 AUC), which indicate that dynamic troponin measurements are incremental to single baseline values. These results suggest that, serial troponin measurements are a useful tool that should be used along with clinical evaluation and ECG interpretation as a way of enhancing early risk stratification and facilitating prompt management of patients with suspected acute coronary syndrome.

Conflict of interest

The authors declared no conflict of interest.

Author Contribution

All authors reviewed the results and approved the final version of the manuscript. They are also accountable for the study's integrity.

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