

Correlation between Complexity of Coronary Lesions and Significant Delta High-Sensitivity Troponin I Levels in Patients with Non-ST Elevation Myocardial Infarction

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Abstract

Introduction: The role of high-sensitive cardiac troponin (hs-cTn) assays has higher analytical precision at lower concentrations to detect myocardial injury. The changes in troponin concentration between two assays conducted within a specified time interval refers to “Delta troponin”. This study aimed to assess the correlation between the complexity of coronary lesions and significant delta high-sensitivity troponin I levels in patients with non-ST elevation myocardial infarction. **Methods:** This cross-sectional study was conducted in the Department of Cardiology, Ibrahim Cardiac Hospital & Research Institute, Dhaka, Bangladesh from July 2022 to June 2023. A total of 70 patients with significant delta hs-cTnI were included and divided into two groups: Group-A (n = 36) with a delta hs-cTnI rise between >20% to 49%, and Group-B (n = 34) with a delta hs-cTnI rise \geq 50%. Coronary angiography was performed and the SYNTAX Score was calculated for both groups. Data were collected using SPSS version 25.0. **Result:** Patients with a high-rise delta cTnI (\geq 50%) showed a significantly higher proportion of lesions in major coronary arteries LCx and LAD compared to those with a low-rise of cTnI (20% - 49%) ($p = 0.007$ and 0.004 , respectively). The presence of triple vessel diseases was higher in the former group than in the latter ($p < 0.001$). The SYNTAX score was significantly higher in the high-rise delta hs-cTnI group ($p < 0.001$). Furthermore, 32.4% of patients with a high-rise delta cTnI had a SYNTAX score >

22, compared to none in the low-rise group ($p < 0.001$). There was a moderately significant linear correlation between delta hs-cTnI and SYNTAX score ($p = 0.001$). **Conclusion:** A high rise in delta hs-cTnI is linked to higher SYNTAX scores, signifying complex coronary lesions in NSTEMI patients, with a significant linear correlation between them. Patients with a high rise in delta cTnI may exhibit more significant coronary artery lesions and triple vessel diseases compared to those with a low rise in cTnI.

Keywords

Complexity of Coronary Lesions, High-Sensitivity Troponin I, Non-ST Elevation, Myocardial Infarction

1. Introduction

Cardiovascular diseases (CVD) are the leading cause of mortality worldwide, with coronary artery disease (CAD) responsible for around 32% of global deaths [1]. Acute coronary syndrome (ACS) includes conditions like ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina. Each year, over 20 million patients present with symptoms indicative of myocardial infarction (MI) in emergency departments across Europe and North America [2]. Diagnosing ACS relies on detecting changes in cardiac enzymes alongside clinical symptoms and ECG findings. High-sensitivity troponin (hsTn) assays, essential for early MI diagnosis, offer improved sensitivity and precision compared to conventional markers, enabling earlier exclusion of acute myocardial infarction (AMI) [3] [4]. These assays, however, may lead to false-positive results if used indiscriminately [4].

Classified as “sensitive” or “high-sensitivity” based on their detection thresholds, hsTn assays can accurately quantify low troponin concentrations, identifying minor changes over short intervals and correlating with higher MI likelihood [2] [5] [6]. “Delta troponin,” referring to the change in hsTn levels over time, helps gauge myocardial injury severity and assess MI risk, with guidelines recommending a $\geq 20\%$ increase in troponin levels within 3 - 6 hours for early MI diagnosis [6]. Coronary angiography (CAG) is commonly performed for ACS management, including NSTEMI, though up to 13% of NSTEMI cases show normal angiograms [7]. Elevated troponin I levels correlate significantly with severe CAD and complex lesions on coronary angiography [8] [9].

The SYNTAX score is used to quantify the anatomical complexity of coronary lesions in NSTEMI patients [10], though limited studies specifically examine its association with hsTn levels [9]. Prior research suggests that elevated troponin I exceeding ten times the normal range is associated with severe coronary lesions in NSTEMI [11]. This study aims to assess the relationship between coronary lesion complexity and significant delta high-sensitivity troponin I levels in patients with NSTEMI.

2. Methodology

This cross-sectional study was conducted in the Department of Cardiology, Ibrahim Cardiac Hospital & Research Institute, Dhaka, Bangladesh, from July 2022 to June 2023. Seventy consecutive NSTEMI patients were enrolled using consecutive sampling. Written informed consent was obtained from each patient, and relevant demographic and clinical data were collected.

Blood samples for high-sensitivity troponin I (hs-cTnI) levels were obtained at admission and, if elevated, repeated three hours later to calculate delta troponin. Patients without significant delta troponin elevation were excluded. Participants were categorized into two groups based on delta hs-cTnI changes: a rise of >20% - 49% and a rise of \geq 50% from baseline. All patients underwent coronary angiography within one to four days, and the SYNTAX score was calculated to assess lesion complexity. A score \leq 22 was considered low, while a score >22 indicated intermediate to high risk.

Data were analyzed using SPSS version 25, applying standard tests such as the Chi-square test for categorical data and t-tests for continuous data, with significance set at $p < 0.05$.

3. Result

The study compared two groups (Group A: delta hs-cTnI rise > 20% - 49%, Group B: delta hs-cTnI rise \geq 50%) to evaluate the association of delta hs-cTnI levels with coronary lesion complexity. Baseline demographics and risk factors (**Table 1**) were largely comparable between groups, except for a higher prevalence of hypertension in Group B (82.4% vs. 61.1%, $p = 0.049$) and a trend toward more dyslipidemia (79.4% vs. 58.3%, $p = 0.058$). Investigation findings (**Table 2**) showed significantly higher delta hs-cTnI levels in Group B (101.5 ± 49.3 ng/L) compared to Group A (34.5 ± 1.3 ng/L, $p < 0.001$), with no notable differences in other biochemical or echocardiographic parameters. Angiographic analysis (**Table 3**) revealed a significantly higher prevalence of lesions in the left circumflex (LCx, 73.5% vs. 41.7%, $p = 0.007$) and left anterior descending (LAD, 82.4% vs. 50.0%, $p = 0.004$) coronary arteries in Group B. Treatment modalities (**Table 4**) showed that coronary artery bypass grafting (CABG) was more frequently performed in Group B (32.4%) compared to Group A (0%, $p < 0.001$), reflecting greater disease severity. Lastly, the association between delta hs-cTnI levels and SYNTAX scores (**Table 5**) indicated that none of Group A patients had a SYNTAX score > 22, whereas 32.4% of Group B patients exceeded this threshold ($p < 0.001$), highlighting a significant correlation between higher delta hs-cTnI levels and more complex coronary lesions (**Figure 1**).

4. Discussion

In this study, we observed a significant positive correlation between delta hs-cTnI levels and the complexity of coronary lesions, as quantified by the SYNTAX score ($r = 0.4$, $p = 0.001$, **Figure 1**). These results align with findings from Altun *et al.*

[10], who also reported a correlation between high-sensitivity troponin (hsTn) levels and lesion complexity, albeit with different study designs and statistical power.

Our findings highlight the potential clinical utility of monitoring delta hs-cTnI levels in NSTEMI patients. Patients in Group B, who exhibited a delta hs-cTnI rise $\geq 50\%$, had significantly higher SYNTAX scores compared to those in Group A ($p < 0.001$, **Table 5**). Notably, none of the patients in Group A had a SYNTAX score > 22 , whereas 32.4% of Group B patients exceeded this threshold, indicating more complex coronary lesions. The presence of triple vessel disease (TVD) was also significantly higher in Group B ($p < 0.001$, **Figure 2**), supporting the association between high delta hs-cTnI levels and severe coronary artery disease (CAD).

Table 3 further emphasizes these findings by demonstrating that significant lesions in major coronary arteries (LAD and LCx) were more prevalent in Group B compared to Group A ($p = 0.004$ and $p = 0.007$, respectively). This suggests that elevated delta hs-cTnI levels are not only linked to overall lesion complexity but also to the anatomical distribution of significant coronary lesions.

Table 1. Demographic and risk factors variables.

Variables	Group		p-value
	A (n = 36)	B (n = 34)	
Mean \pm SD age	54.2 \pm 11.3 (31 - 77 years)	55.7 \pm 8.9 (38 - 73 years)	0.524
Sex			
Male	25 (69.4%)	22 (64.7%)	0.673
Female	11 (30.6%)	12 (35.3%)	
Comorbidities			
DM	28 (77.8%)	28 (82.4%)	0.632
Hypertension	22 (61.1%)	28 (82.4%)	0.049
Dyslipidemia	21 (58.3%)	27 (79.4%)	0.058
Smoking	10 (27.8%)	7 (20.6%)	0.714
Hypertension	22 (61.1%)	28 (82.4%)	0.049
TCP	27 (75.0)	24 (70.6)	0.678
FH of CAD	13 (36.1%)	12 (35.3%)	0.943
BMI (kg/m ²)			
18.5 - 24.9	12 (33.3%)	7 (20.6%)	0.277
<18.5	1 (2.8%)	0 (0.0%)	
25 - 29.9	20 (55.6%)	20 (58.8%)	
30 - 39.9	3 (8.3%)	7 (20.6%)	

DM: Diabetes mellitus, TCP: Typical chest pain, FH: Family history. The data were analyzed using an Unpaired t-test and between the mean \pm SD values. The chi-square (χ^2) test was used for categorical data, with percentages presented in parentheses.

Table 2. Investigation findings between groups.

Investigation	Group		p-value
	A (n = 36)	B (n = 34)	
Hb (%)	12.1 ± 2.0	11.9 ± 1.7	0.775
RBS (mg/dl)	9.4 ± 6.3	8.8 ± 2.8	0.857
HbA1c (%)	8.1 ± 1.8	8.3 ± 2.3	0.681
STC (mg/dl)	184.9 ± 44.2	192.5 ± 59.7	0.543
S. LD (mg/dl)	113.1 ± 35.5	109.5 ± 51.8	0.728
S. HDL (mg/dl)	33.9 ± 9.0	39.9 ± 18.1	0.087
S. TG (mg/dl)	223.1 ± 132.6	200.4 ± 91.9	0.411
SC (mg/dl)	1.1 ± 0.4	1.3 ± 1.1	0.384
eGFR (ml/1.73m ²)	78.6 ± 29.8	77.6 ± 33.9	0.896
D. hs-cTnI (ng/L)	34.5 ± 1.3	101.5 ± 49.3	<0.001
Echocardiographic findings			
RWMA	15 (41.7)	14 (41.2)	0.967
Ejection Fraction (%)	56.7 ± 7.0	53.8 ± 8.9	0.138

STC: Serum total cholesterol. The data were analyzed using an Unpaired t-test and between the mean ± SD values. The chi-square (χ^2) test was used for categorical data, with percentages presented in parentheses.

Table 3. Significant lesions in vessels.

Lesions	Group		p-value
	A (n = 36)	B (n = 34)	
LM (>50%)	2 (5.6%)	2 (5.9%)	0.671
RCA (>50%)	16 (44.4%)	19 (55.9%)	0.339
LCx (>50%)	15 (41.7%)	25 (73.5%)	0.007
LAD (>50%)	18 (50.0%)	28 (82.4%)	0.004

LM: Left Main (Coronary artery), RCA: Right Coronary Artery, LCx: Left Circumflex (Coronary artery), LAD: Left Anterior Descending (Coronary artery). Data were analyzed by using a Fisher's Exact Test; figures in the parenthesis denote corresponding percentages.

Table 4. Comparison of treatment received by the two study groups.

Treatment	Group		p-value
	A (n = 36)	B (n = 34)	
PCI	21 (58.3%)	17 (50.0%)	0.484
CABG	0 (0.0%)	11 (32.4%)	<0.001
MM	11 (30.6%)	6 (17.6%)	0.208

MM: Medical management. Data were analyzed using a Chi-square (χ^2) Test.

Table 5. Association between significant delta hs-cTnI and SYNTAX score.

Score	Group		p-value
	A (n = 36)	B (n = 34)	
>22	0 (0.0%)	11 (32.4%)	<0.001
≤22	36 (100.0%)	23 (67.6%)	

Data were analyzed using a Chi-square (χ^2) Test.

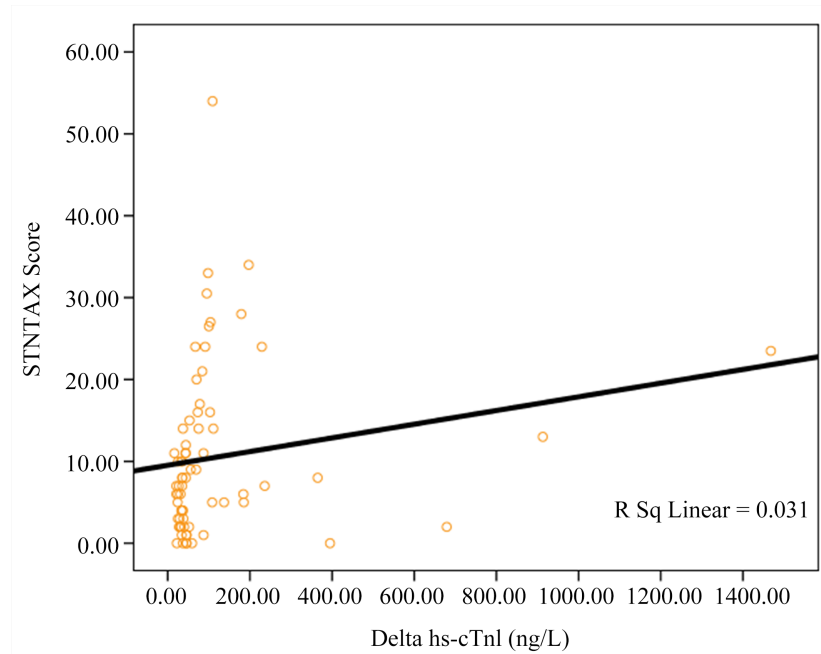


Figure 1. Correlation between delta hs-cTnI and SYNTAX score.

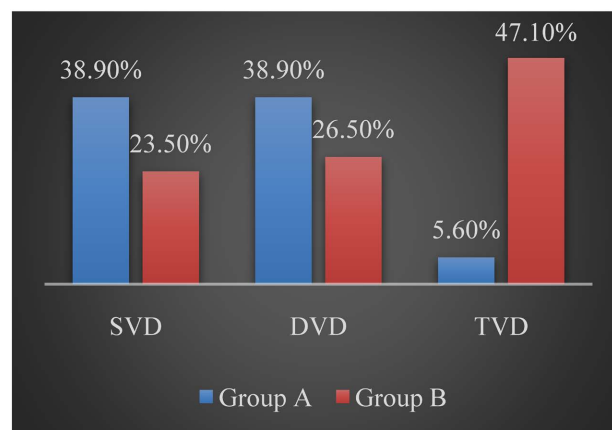


Figure 2. Severity of CAD.

Delta hs-cTnI could serve as an effective non-invasive marker for identifying patients at higher risk for severe CAD, enabling targeted clinical decisions. For instance, monitoring delta hs-cTnI levels may aid in early identification of NSTEMI patients who are more likely to have complex coronary lesions. Elevated delta hs-cTnI levels could signal the need for urgent intervention and potentially justify earlier coronary angiography to assess lesion complexity. This is further supported by treatment data in **Table 4**, which shows that patients in Group B were more likely to undergo coronary artery bypass grafting (CABG) (32.4% vs. 0%, $p < 0.001$), reflecting the greater severity of their conditions. Conversely, a higher proportion of patients in Group A were managed with medical therapy, suggesting a less complex CAD profile.

Our study contributes to an expanding body of evidence supporting hs-cTn as

a predictor of CAD severity. Studies by Ndrepepa *et al.* [12] and Tahhan *et al.* [13] similarly found that elevated hsTnI levels were predictive of both CAD complexity and adverse outcomes, including cardiovascular mortality. These studies suggest that hsTnI could be incorporated into routine risk assessment for NSTEMI patients, alongside other risk markers. Despite these promising findings, our data in **Table 1** show that key comorbidities such as hypertension and dyslipidemia were more prevalent in Group B ($p = 0.049$ and $p = 0.058$, respectively), which may contribute to lesion complexity and warrant further exploration.

Future studies should address several areas: conducting larger multicenter studies to improve the generalizability of delta hs-cTnI as a marker across diverse populations; investigating its long-term predictive value for adverse outcomes, such as recurrent MI or cardiovascular mortality; and assessing the economic feasibility of routine delta hs-cTnI monitoring in NSTEMI management to inform healthcare policy. By identifying patients who need immediate intervention, delta hs-cTnI monitoring may ultimately reduce healthcare costs associated with delayed or missed diagnoses of complex CAD.

This study's single-center design and limited sample size constrain its applicability to wider populations. Moreover, factors such as ethnic and socioeconomic variations were not fully addressed, which may impact delta hs-cTnI levels. Addressing these limitations through future research would strengthen the case for delta hs-cTnI as a standard risk marker in clinical practice. In summary, elevated delta hs-cTnI levels are associated with complex coronary lesions in NSTEMI patients. By incorporating delta hs-cTnI monitoring into clinical protocols, healthcare providers can potentially improve early diagnosis and optimize intervention strategies, enhancing care outcomes for patients at risk of severe CAD.

5. Limitation of the Study

While the researcher took utmost care throughout the study, limitations persist. The single-center design may limit the generalizability to the wider community. With a small sample size, the prospective cohort study lacked sufficient power for clinical outcomes. Furthermore, the short-term follow-up precluded assessment of disease progression.

6. Conclusion

Patients exhibiting a substantial increase in delta cTnI levels may likely have more severe coronary artery lesions compared to those with a minimal rise. Elevated delta hs-cTnI levels correlate with higher SYNTAX scores, indicating greater anatomical complexity in NSTEMI patients. Additionally, a moderate linear correlation exists between delta hs-cTnI levels and SYNTAX scores. Furthermore, patients with significant delta hs-cTnI elevation tend to have more triple vessel diseases (TVDs). These findings suggest that non-invasive delta hs-cTnI testing could identify high-risk patients with complex anatomies, facilitating timely referral for advanced cardiac care and early revascularization.

7. Recommendations

Due to the small sample size of the present study, further research with a larger sample is recommended to validate its findings. A larger sample size would enhance the statistical power and robustness of the results, providing more confidence in the conclusions drawn from the study.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

- [1] Twerenbold, R., Boeddinghaus, J. and Mueller, C. (2018) Update on High-Sensitivity Cardiac Troponin in Patients with Suspected Myocardial Infarction. *European Heart Journal Supplements*, **20**, G2-G10. <https://doi.org/10.1093/eurheartj/suy020>
- [2] Thygesen, K., Alpert, J.S., Jaffe, A.S., Simoons, M.L., Chaitman, B.R., White, H.D., et al. (2012) Third Universal Definition of Myocardial Infarction. *Journal of the American College of Cardiology*, **60**, 1581-1598. <https://doi.org/10.1016/j.jacc.2012.08.001>
- [3] Piegas, L., Timerman, A., Feitosa, G., Nicolau, J., Mattos, L., Andrade, M., et al. (2015) V Diretriz Da Sociedade Brasileira De Cardiologia Sobre Tratamento Do Infarto Agudo Do Miocárdio Com Supradesnível Do Segmento ST. *Arquivos Brasileiros de Cardiologia*, **105**, 1-105. <https://doi.org/10.5935/abc.20150107>
- [4] Maznyczka, A., Kaier, T. and Marber, M. (2015) Troponins and Other Biomarkers in the Early Diagnosis of Acute Myocardial Infarction. *Postgraduate Medical Journal*, **91**, 322-330. <https://doi.org/10.1136/postgradmedj-2014-133129>
- [5] Reichlin, T., Irfan, A., Twerenbold, R., Reiter, M., Hochholzer, W., Burkhalter, H., et al. (2011) Utility of Absolute and Relative Changes in Cardiac Troponin Concentrations in the Early Diagnosis of Acute Myocardial Infarction. *Circulation*, **124**, 136-145. <https://doi.org/10.1161/circulationaha.111.023937>
- [6] Storrow, A.B., Nowak, R.M., Diercks, D.B., Singer, A.J., Wu, A.H.B., Kulstad, E., et al. (2015) Absolute and Relative Changes (Delta) in Troponin I for Early Diagnosis of Myocardial Infarction: Results of a Prospective Multicenter Trial. *Clinical Biochemistry*, **48**, 260-267. <https://doi.org/10.1016/j.clinbiochem.2014.09.012>
- [7] Qadir, F., Farooq, S., Khan, M., Hanif, B. and Lakhani, M.S. (2010) Correlation of Cardiac Troponin I Levels (10 Folds Upper Limit of Normal) and Extent of Coronary Artery Disease in Non-ST Elevation Myocardial Infarction. *Journal of the Pakistan Medical Association*, **60**, 423-428.
- [8] López-Fernández, S., Cequier, Á., Iràculis, E., Gómez-Hospital, J.A., Teruel, L., Valero, J., et al. (2004) Las elevaciones importantes de troponina I en el síndrome coronario agudo sin elevación del segmento ST se asocian a estenosis coronarias más complejas. *Revista Española de Cardiología*, **57**, 291-298. [https://doi.org/10.1016/s0300-8932\(04\)77106-7](https://doi.org/10.1016/s0300-8932(04)77106-7)
- [9] Sianos, G., Morel, M.A., Kappetein, A.P., Morice, M.C., Colombo, A., Dawkins, K., et al. (2005) The SYNTAX Score: An Angiographic Tool Grading the Complexity of Coronary Artery Disease. *EuroIntervention*, **1**, 219-227.
- [10] Altun, B., Turkon, H., Tasolar, H., Beggı, H., Altun, M., Temiz, A., et al. (2013) The Relationship between High-Sensitive Troponin T, Neutrophil Lymphocyte Ratio and SYNTAX Score. *Scandinavian Journal of Clinical and Laboratory Investigation*, **74**, 108-115. <https://doi.org/10.3109/00365513.2013.860619>

- [11] Sajid, S. and Abbas, K. (2018) Severity of Coronary Artery Disease in Non-ST Elevation Myocardial Infarction (NSTEMI) Patients with High Troponin-I Level. *Pakistan Heart Journal*, **51**, 114-118.
- [12] Ndrepepa, G., Braun, S., Schulz, S., Mehilli, J., Schömig, A. and Kastrati, A. (2011) High-sensitivity Troponin T Level and Angiographic Severity of Coronary Artery Disease. *The American Journal of Cardiology*, **108**, 639-643.
<https://doi.org/10.1016/j.amjcard.2011.04.012>
- [13] Samman Tahhan, A., Sandesara, P., Hayek, S.S., Hammadah, M., Alkhoder, A., Kelli, H.M., *et al.* (2018) High-Sensitivity Troponin I Levels and Coronary Artery Disease Severity, Progression, and Long-Term Outcomes. *Journal of the American Heart Association*, **7**, e007914. <https://doi.org/10.1161/jaha.117.007914>