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Raised level of cardiac troponin I (50 folds upper limit of normal) is a sensitive and specific marker for extent of coronary artery disease in first attack of non-ST segment elevation myocardial infarction in Bangladeshi population

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Background: Coronary Heart Disease (CHD) is the most common cause of heart disease and is the single most important cause of premature death in developed world. Recognizing a patient with ACS is important because the diagnosis triggers both triage and management. Cardiac troponin-I (cTnI) is 100% tissue-specific for the myocardium and it has been shown to be a very sensitive and specific marker for acute myocardial infarction (AMI).

Objective: The study aimed to determine the correlation of cardiac troponin I (cTnI) 50 folds Upper Limit of Normal (ULN) and extent of Coronary Artery Disease (CAD) in Non-ST-Elevation Myocardial Infarction (NSTEMI).

Methods: This cross-sectional analytical study was conducted in the Department of Cardiology in Mymensingh Medical College Hospital, United Hospital Limited and Enam Medical College Hospital from December 2015 to November 2016. Total 230 first attack of NSTEMI patients were included considering inclusion and exclusion criteria. cTnI was measured using immune-metric assay method. All patients underwent coronary angiography in the united hospital and Enam Medical College Hospital. Stenosis ≥70% in any of the three major epicardial vessels was considered significant CAD. Extent of CAD was defined as significant single, two or three vessel CAD. The sample population was divided into two groups: Group-1: Patients with first attack of NSTEMI with troponin-I level ≤50 folds upper limit of normal (Trop-I: ≤6.6 ng/ml) and Group-2: Patients with first attack of NSTEMI with Troponin-I level >50 folds upper limit of normal (Trop-I: ≥6.6 ng/ml). Chi-square test was applied to test the association between cTnI levels and CAD extent.

Results: Out of 230 patients, in 111 patients with cTnI levels ≤50 folds upper limit of normal, 25 (22.52%) had single vessel, 40 (36%) had two vessel and 34 (30.6%) had three vessel significant CAD, whereas in 119 patients with cTnI levels > 50 folds ULN, 23 (19.3%) had single vessel, 37 (31.1%) had two vessel and 55 (46.2%) had three vessel significant CAD. The results suggest that there was an insignificant association between the cTnI levels and single vessel, two vessel and the overall CAD extent (p=0.35, p=0.21 and p=0.13, respectively), however there was a statistically significant association between the cTnI levels and three vessel CAD (p=0.04). Our study discovered that troponin-I level ≥6.6 ng/ml is a very sensitive and specific marker for extent of CAD.

Conclusion: The study enabled us to conclude that, higher cTnI levels are associated with an increased proportion of severe three vessel CAD involvement. Prompt identification and referral of this patient subset to early revascularization strategies would improve clinical outcomes.

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