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Review of high sensitive cardiac troponin T levels in patients with acute coronary syndrome in a tertiary hospital in Benin City, Nigeria

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Acute coronary syndrome is a spectrum of thrombotic coronary diseases which includes unstable angina, non ST segment elevation and ST segment elevation myocardial infarction (STEMI). Incidence is on the increase worldwide and in Nigeria. Early diagnosis and prompt intervention is of utmost importance. Cardiac troponin T is an important biomarker of myocardial ischaemia, and levels become elevated in myocardial infarction. The aim of this work is to evaluate serum high sensitive troponin T levels in patients diagnosed with acute coronary syndrome and determine its usefulness in diagnosis of STEMI and NSTEMI, and its prognostic significance. This is a retrospective study. Data of 30 patients diagnosed with acute coronary syndrome and seen in the Emergency unit of the University of Benin Teaching Hospital were analyzed. 15 (14 males and 1 female) were diagnosed with STEMI and 15 (10 males and 5 females) were diagnosed with NSTEMI. Diagnosis was made based on chest pain history, ECG and Echocardiography findings and elevated High sensitive troponin T levels. High sensitive troponin T assay was done with Elecys (Roche) 4th generation. Abnormal values were established at ≥ 0.014ng/ml. Mean age of the patients was (50.04 ± 12.0 years) and (53.3 ± 19 years) in subjects with STEMI and NSTEMI, respectively. Elevated high sensitive troponin T levels ≥ 0.014 ng/ml was seen in 93.3% of subjects with ST Segment elevation myocardial infarction and 66.7% of subjects with non ST Segment elevation myocardial infarction. Mean high sensitive troponin T levels were higher in subjects with ST Segment elevation myocardial infarction (0.43±0.56 ng/ml) than those with non-ST Segment elevation myocardial infarction (0.018±0.01 ng/ml). Difference was statistically significant (p<0.05). High sensitive troponin T levels are useful in the diagnosis of acute coronary syndrome, both in STEMI and NSTEMI and may have prognostic significance.

Key words: ST segment myocardial infarction (STEMI), non ST segment myocardial infarction (NSTEMI), high sensitive cardiac troponin T (hs-cTnT), acute coronary syndrome (ACS).

INTRODUCTION

Acute coronary syndrome encompasses a range of thrombotic coronary artery diseases, including unstable

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angina and ST-segment elevation and non-ST segment elevation myocardial infarcation (STEMI and NSTEMI) (Achar et al., 2005). It is becoming an increasingly important problem in developing countries (Montalescot et al., 2007). To reduce mortality and morbidity, rapid clinical determination of the likelihood of obstructive coronary artery diseases with subsequent management is necessary for all patients with chest discomfort or other symptoms suggestive of an acute coronary syndrome (Liu and Huang, 2011; Kushner et al., 2009).

In 2000, the joint European Society of Cardiology/ American College of Cardiology (ESC/ACC) Committee for the redefinition of myocardial infarction suggested that any amount of necrosis resulting from ischaemia should be diagnosed as a myocardial infarction (Montalescot et al., 2007; Liu and Huang, 2011). As a result of this new definition, patients who were previously considered to have unstable angina are being diagnosed with myocardial infarcation (Montalescot et al., 2007; Liu and Huang, 2011). Since then higher serum levels of troponin have also been associated with more severe prognosis (Montalescot et al., 2007). The main tools used to determine likelihood of acute coronary syndrome in the emergency unit are the chest pain history, electro-cardiogram findings and Blood markers of myocardial injury such as troponins (Goodman et al., 2006). In acute coronary syndrome, common electrocardio graphic abnormalities include T wave tenting or inversion, ST-segment elevation or depression (including J point elevation in multiple leads) and pathologic Qwaves (Achar et al., 2005). Patients exhibiting clinical symptoms of ischaemia, but with no evidence of myocardial necrosis based on serum biomarkers are considered to have unstable angina (Goodman et al., 2006; Mokhtari et al., 2015), whereas those patients who have positive cardiac biomarkers and demonstrate ischaemic symptoms, with or without electrocardiographic ST segment depression or T wave inversion, are experiencing NSTEMI (Goodman et al., 2006). Further along the acute coronary syndrome are patients with new ST segment elevation on the electrocardiogram (ECG) which is diagnostic of acute STEMI (Goodman et al., 2006; Mokhtari et al., 2015).

The ESC and the ACC established troponin as the biomarker of choice in the diagnosis of myocardial inferction (Daubert and Jeremias, 2010). Troponin complex has 3 sub-units, which are C, T and I. Troponin T and 1 are expressed by cardiac muscles only. The 2007 guidelines of ACC and American Heart Association (AHA) stated that one of the criteria for diagnosis of acute myocardial infarction is the detection of rising and falling cardiac biomarker levels (preferably troponin) with at least one value above the 99th percentile of the upper reference limit (Anderson et al., 2007). This has also been promoted by the ESC/ACCT/AHA/WHF Taskforce (Alpert et al., 2000; Thygesen, 2015).

Some studies have reported elevated troponin T levels in acute coronary syndrome, in both STEMI and NSTEMI (Okoye and Anyabola, 2016; Riedlinger et al., 2018). In this study, a retrospective study conducted to evaluate high sensitivity troponin T levels in patients seen in the Emergency department of our hospital, with diagnosis of acute coronary syndrome, with the aim of determining its usefulness in diagnosis of STEMI and NSTEMI and its prognostic significance.

MATERIALS AND METHODS

This was a retrospective study carried out on 30 subjects (15 STEMI and 15 NSTEMI) seen in the emergency unit of the university of Benin teaching hospital within the period of February 2017 to March 2018. Data of patients whose blood samples were sent for high sensitive cardiac troponin T assay during this period were retrospectively analyzed. Relevant clinical data including demographic characteristics along with Electrocardiographic and Echocardiographic findings were retrieved from the medical records of these patients. Diagnosis of myocardial infarcation was based on typical history of chest pain, Electrocardiographic findings and in some cases Echocardiographic findings, and cardiac troponin T levels. The subjects were categorized into STEMI and NSTEMI based on Electrocardiographic findings. hs – Troponin T assay was done using Elecys (Roche) 4th Generation 99th percentile cut-off value for this assay method ≥ 0.01 ng/ml. Ethical committee protocol was observed

Statistical analysis

This was done using SPSS (Statistical Package for the Social Sciences) version 21. Simple descriptive statistical analysis was used, and difference between means of variables was established using student “t” test. Correlation between variables was established using pearson’s correlation. Level of significance was established at P≤0.05.

RESULTS

Data of 30 subjects diagnosed with acute coronary syndrome were analyzed. 15 (14 males and 1 female) are subjects with STEMI and 15 (10 males, 5 females) are subjects with NSTEMI. Mean age of subjects with STEMI was 50.4 + 12.0 years and NSTEMI, 53.3±19 years. No statistically significant difference, p = 0.324 (Table 1) 93.3% of subjects with STEMI had hs – troponin T levels ≥ 0.01 n g/ml, while those with NSTEMI was 66.7% (Table 1). Mean serum troponin levels were 0.43±0.056 ng/ml in STEMI and 0.018±0.01 ng/ml in patients with NSTEMI. Difference was statistically significant P =0.01 (Table 1). Figure 1 shows the mean troponin T levels in STEMI and NSTEMI. ECG changes in subjects with STEMI showed 46.7% with ST segment elevation, 6.7% with Q waves and 13.3% with bundle branch block (Table 2). While ECG changes in subjects with NSTEMI showed 6.7% with ST segment depression, 6.7% with T wave abnormality, 6.7% with Q waves, 13.3% with atrial fibrillation, 6.7% with hemi-bundle branch block and 60% other findings such as ventricular Arrhythmias, sinus ventricular tachycardia (Table 3).

Table 1. Demographic characteristics of subjects with STEMI and NSTEMI.

Parameter	STEMI (n = 15)	NSTEMI (n = 15)	P value
	Mean ± SD	Mean ± SD	
Age (years)	50.4+12.0	53.3±19.0	0.324
Gender			
Male	14 (93.3)	10 (66.7)	0.204
Female	1 (6.7)		
Chest pain history	15 (100)	15 (100)	
Troponin (ng/ml)	0.43±0.56	0.018±0.01	0.010*
Troponin (ng/ml)			
<0.014	14 (6.7)	5 (33.3)	0.072
≥0.014	15 (93.3)	10 (66.7)	

*Statistically significant difference in Troponin T between subjects with STEMI and NSTEMI, P<0.05.

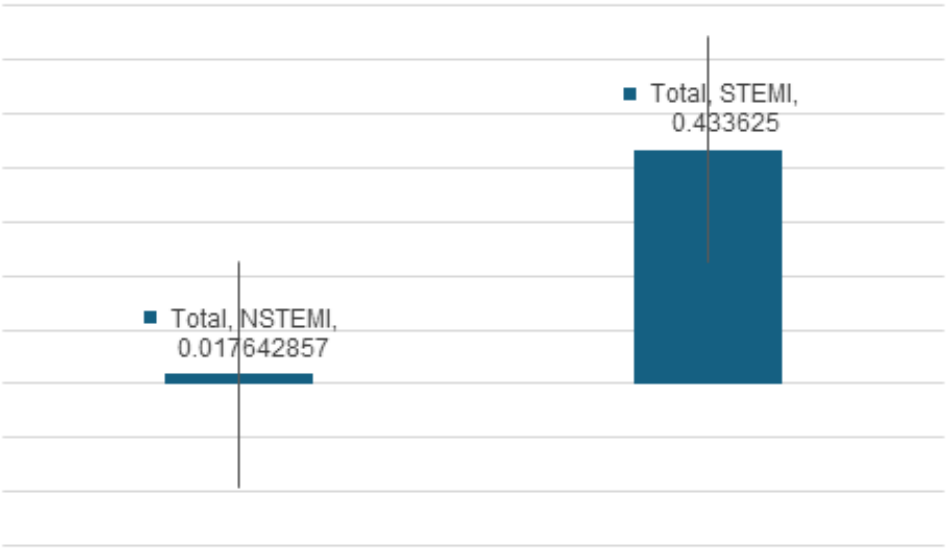

Figure 1. Mean Troponin levels in NSTEMI and STEMI.

Table 2. ECG changes in STEMI and NSTEMI.

Parameter	STEMI (%)	NSTEMI (%)
ST segment elevation, %	7 (46.7)	-
ST segment Depression, %	1 (6.7)	1 (6.7)
Other ECG Changes	-	-
-T wave abnormalities	3 (20)	1 (6.7)
-Poor R wave progression	1 (6.7)	1 (6.7)
-Q waves	1 (6.7)	1 (6.7)
-Atrial fibrillation with rapid ventricular response	-	2 (13.3)
Bundle branch block	2 (13.3)	1 (6.7)
Other changes	-	8 (53.3)

Table 3. Short term prognosis (one month post infarction).

Parameter	STEMI (%)	NSTEMI (%)
Death	2 (13.3)	-
Heart failure	-	2 (13.3)
On follow up in clinic	6 (40)	13 (87.7)
Defaulted from clinic	7 (47.7)	-

Short term prognosis review showed mortality in 13.3% of the subjects with STEMI, while 40% are being followed up in the clinic, and 13.3% defaulted from clinic (Table 3). For subjects with NSTEMI, 13.3% developed heart failure, 86.6% are on follow up in the clinic and 13.3% defaulted from clinic.

DISCUSSION

Incidence of acute coronary syndrome is on the increase both in developed and developing countries. The need for early diagnosis and intervention will reduce mortality and morbidity drastically.

In our study, mean age of subjects with STEMI was lower than mean age of those with NSTEMI, though the difference was not statistically significant. Mean age recorded in both groups of patients is similar to that recorded by Anjorin et al. (2005) in their study "myocardial infarction at the University of Maiduguri Teaching Hospital, North eastern Nigeria. Male preponderance in incidence of myocardial infarction was also reported in their study. While male/female ratio in their study was 8:1, another study (Akpa et al., 2012) reported ratio of 2:1. Male preponderance may be due to cardiovascular risk factors such as cigarette smoking and alcohol intake. High sensitive serum troponin T level was ≥ 0.014 ng/ml in 93.3% of subjects with STEMI and 66.7% in subjects with NSTEMI. This shows that cardiac troponin estimation is of diagnostic importance in our area of study and should be one of the immediate assays to be done in a patient presenting with clinical signs and symptoms suggestive of myocardial ischaemia. Avilles et al. (2002), in the study on troponin T levels in patients with acute coronary syndrome with or without renal dysfunction, reported 64% of the patients had elevated levels of troponin T levels > 0.03 ng/ml and 52% with levels > 0.1 ng/ml. These findings support the usefulness of cardiac troponin T as a diagnostic marker of myocardial infarction in acute coronary syndrome.

Mean serum troponin T levels was significantly higher in subjects with STEMI than those with NSTEMI in our study. This could be due to the extent of myocardial ischaemia, and necrosis, since in NSTEMI there is subendocardial infarction, and in STEMI, there is transmural infarction. Some studies have actually reported troponin T levels correlating with infarct size. Higher values generally correlate with a large infarct. Arruda-Olson et al. (2011), in their study, reported cardiac troponin T levels as an independent predictor of infarct size on Days 1, 2, 3 and peak troponin levels.

Correlation of troponin T levels with infarct size seems to be stronger in STEMI than NSTEMI. Findings by Giannitisis et al. (2008) and Hallén (2012) suggest that correlations between cardiac troponin and infarct size are significantly weaker in NSTEMI than STEMI. This could be due firstly to the fact that NSTEMIS are on the average substantially smaller than STEMIs. Thus, imaging quantification of the extent of infarcted tissue may be somewhat less precise; also timing of the ischaemia onset is less well defined in NSTEMI (Giannitisis et al., 2008). Hassan et al. (2009), in their study, "usefulness of peak troponin to predict infarct size and long-term outcome in patients with first acute myocardial infarction after primary percutaneous coronary intervention", concluded that peak cardiac troponin T after primary percutaneous coronary intervention for STEMI offers a good estimation of infarct size and is a prognostic indicator in patients with first acute myocardial infarction. In all, cardiac troponin T level is not only useful in detecting and diagnosing acute myocardial infarction, it also has prognostic value.

100% of the subjects diagnosed with STEMI showed the typical ECG findings consistent with STEMI, ranging from ST segment elevation, ST segment depression, Q waves, Poor R wave progression, and bundle branch block. Most of them with NSTEMI showed ECG changes ranging from verticular arrythimias, T wave abnormality, ST segment depression and other abnormalities. This finding also shows the role Electro cardiography plays, not only in diagnosing myocardial infarction, but also in categorizing it into STEMI and NSTEMI.

Short term prognosis review showed that those with STEMI have a tendency for a poorer outcome compared with those with NSTEMI. While 13.3% of those with STEMI died within a period of few days to a month, none of the patients with NSTEMI died during that one month post infarction, though some subjects were lost to follow up.

In this study, 93.3% of patients with STEMI and over 66.7% of patients with NSTEMI had elevated high sensitive troponin T levels; there was a statistically significant difference between mean troponin T levels in subject with STEMI, compared with NSTEMI; subjects

with STEMI had higher levels of troponin T. Further studies still need to be carried out especially in establishing a correlation between troponin T levels and infarct size.

Conclusion

High sensitive troponin T assay is useful in the diagnosis of acute myocardial infarction, both in STEMI and NSTEMI and may be of prognostic significance.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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REFERENCES

Achar SA, Kundu S, Norcross WA (2005). Diagnosis of Acute Coronary Syndrome. American Family Physician 72(01):119-126.

Akpa MR, Agada Z, Odia J (2012). Myocardial infarction in Nigeria: Trends from a tertiary institution in Portharcourt, Southern Nigeria. Gazette of Medicine 2(2).

Alpert JS, Thygesen K, Antman E, Bassand JD (2000). Myocardial infarction Redefined – A common document of Joint European society of cardiology/American College of cardiology committee for the Redefinition of myocardial infarction. Journal of the American College of Cardiology 36(3):959-969.

Anderson JL, Adams CD, Antman EM, Bridges CR, Califf RM, Casey DE, Chavey WE, Fesmire FM, Hochman JS, Levin TN, Lincoff AM (2007). ACC/AHA 2007 guidelines for the management of patients with unstable angina/non–ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction) developed in collaboration with the American College of Emergency Physicians, the Society for Cardiovascular Angiography and Interventions, and Journal of the American College of Cardiology 50(7):e1-57.

Anjorin CO, Buba E, Ene AC (2005). Myocardial infarction at the University of Maiduguri Teaching Hospital, North Eastern Nigeria. Long Term Review. Journal of Medical Sciences pp. 328-362.

Arruda-Olson AM, Roger VL, Miller TD (2011). Troponin T levels and infarct size by SPECT myocardial perfusion imaging. Jacc cardiovascular 4(5):523-533.

Avilles RJ, Askari AT, Lindali B, Wallentin L, Jua G (2002), Troponin T levels in patients with acute coronary syndrome with or without renal dysfunction. The New England Journal of Medicine 346(26):2047-2052.

Daubert MA, Jeremias A (2010). The utility of troponin measurement to detect myocardial infarction. A review of current findings. Vascular health and risk management 6:691.

Giannitisis E, Steen H, Kurz K, Ivandic B, Simon AC, Futterer S, Schild C, Isfort P, Jaffe AS, Katus HA (2008). Cardiac magnetic resonance imaging study for quantification of infarct size comparing directly serial versus single time-point measurements of cardiac troponin T. Journal of the American College of Cardiology 51(3):307-314.

Goodman SG, Steg PG, Eagle KA, Fox KA, López-Sendón J, Montalescot G, Budaj A, Kennelly BM, Gore JM, Allegrone J, Granger CB, Gurfinkel EP, GRACE Investigators (2006). The diagnostic and prognostic impact of the redefinition of acute myocardial infarction. Lessons from the Global Registry of Acute Coronary Events (GRACE). American Heart Journal 151(3):654-660.

Hallén J (2012). Troponin for the estimation of infarct size: what have we learned?. Cardiology 121(3):204-212.

Hassan AK, Bergheanu SC, Hasan-Ali H, San Liem S, van der Laarse A, Wolterbeek R, Atsma DE, Schalij MJ, Jukema JW (2009). Usefulness of peak troponin-T to predict infarct size and long-term outcome in patients with first acute myocardial infarction after primary percutaneous coronary intervention. The American Journal of Cardiology 103(6):779-784.

Kushner FG, Hand M, Smith SC Jr, King SB 3rd, Anderson JL, Antman EM, Bailey SR, Bates ER, Blankenship JC, Casey DE Jr, Green LA, Hochman JS, Jacobs AK, Krumholz HM, Morrison DA, Ornato JP, Pearle DL, Peterson ED, Sloan MA, Whitlow PL, Williams DO. (2009). SCJR 2009 focused updates. ACC/AHA guidelines for the management of patients with ST elevation myocardial infarction (updating the 2004 guidelines and 2007 focused updates) and ACC/AHA/SCA guidelines on percutaneous coronary intervention (updating the 2005 guideline and 2007 focused update). Circulation. a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Journal of the American College of Cardiology 54(23):2205-2241.

Liu CH, Huang YC (2011). Comparison of STEMI and NSTEMI patients in the emergency Department. Journal of Acute Medicine pp. 1-4.

Mokhtari A, Dryver E, Soderhelhom M, Ekelhund U (2015). Diagnostic values of chest pain history, ECG, troponin and clinical gestalt in patients with chest pain and potential acute coronary syndrome assessed in the emergency department. Springerplus 4(1):219.

Montalescot G, Dallongeville J, Van Belle E, Rouanet S, Baulac C, Degrandsart A, Vicaut E (2007). STEMI and NSTEMI: are they so different? 1 year outcomes in acute myocardial infarction as defined by the ESC/ACC definition (the OPERA registry). European Heart Journal 28(12):1409-1417.

Okoye IC, Anyabola EN (2016). Acute myocardial infarction in a resource poor setting: A case report in Awka, Nigeria. International Journal of Research in Medical Sciences 4(12):5488-5490 doi: 10.18203/2320-6012.ijrms20164236.

Riedlinger D, Möckel M, Müller C, Holert F, Searle J, Von Recum J, Slagman A (2018). High-sensitivity cardiac troponin T for diagnosis of NSTEMI in the elderly emergency department patient: A clinical cohort study. Biomarkers 23(6):551-557.

Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR (2015). ESC/ACCF/AHA/WHF. Expert Consensus Document Third universal definition of myocardial infarction. Circulation Circulation 126(16):2020-2035.