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EVALUATION OF CARDIAC TROPONIN I AND HS-CRP IN SUDANESE PATIENTS WITH ACUTE CORONARY SYNDROME

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ABSTRACT

Cardiac biomarkers should be measured in all patients who present with chest discomfort consistent with acute coronary syndrome (ACS). Elevations of cardiac enzyme levels should be interpreted in the context of clinical and ECG findings. Cardiac troponins T and I are the preferred markers for myocardial injury as they have the highest sensitivities and specificities for the diagnosis of acute myocardial infarction. The objective of this study wasto estimate levels of cardiac troponin I and hs-CRP in patients with acute coronary syndrome. This study was cross section study, was conducted in Alshaab Hospital and Sudan cardiac Centre, Khartoum state. During September to November 2014. A total of 70 patients categorized according to ECG results to non-ST elevation myocardial infarction (NSTEMI) or ST myocardial infarction (STEMI) patients or unstable angina (UA). Serum troponin I was estimated using immune assay method, while serum HSCRP was estimated using immunofluorescence method. The (mean±SD)of serum hs-CRP and cTnI in patients respectively were (10.5±5.7,6.5±8.2). 35.7% of patients with UA show elevated hs-CRP (> 5 mg/L) while 6.7% of patients with STEMI and 50% show elevated levels of hs-CRP in patients with NSTEMI. cTnI seen elevated (>1.5 ng/ml) as follows: 3.6% in patients with UA, 96.6% in patients with STEMI, and 50% in patients with NSTEMI. This study concluded that, no available biomarker offers ideal diagnostic properties for ACS, such as early detection, high sensitivity and specificity, easy availability, and cost effectiveness. But cTnI and hs-CRP with ECG were with sufficient sensitivity and specificity in diagnosis of STEMI patients.

INTRODUCTION

The diagnosis and management of patients with acute coronary syndrome (ACS) have evolved dramatically over the past decade. The term acute coronary syndrome (ACS) refers to any group of clinical symptoms compatible with acute myocardial ischemia and covers the spectrum of clinical Conditions ranging from unstable angina (UA) to non–ST - segment elevation myocardial Infarction (NSTEMI) to ST-segment elevation myocardial infarction (STEMI) [1, 2, 3].Cardiac biomarkers should be measured in all patients who present with chest discomfort consistent with acute coronary syndrome (ACS). Elevations of cardiac enzyme levels should be interpreted in the context of clinical and ECG findings. Cardiac troponins T and I are the preferred markers for myocardial injury as they have the highest sensitivities and specificities for the diagnosis of acute myocardial infarction [4]. Troponin is a protein released from myocytes when irreversible myocardial damage occurs. It is highly specific to cardiac tissue and accurately diagnoses myocardial infarction with a history of ischemic pain or ECG changes reflecting ischemia. Cardiac troponin level is dependent on infarct size, thus

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providing an indicator for the prognosis following an infarction [5].

New high-sensitivity cardiac troponin assays have been developed that can measure troponin values at much lower levels. With the use of these high-sensitivity assays, more patients with unstable angina will be classified as having non-ST-elevation myocardial infarction. These assays may therefore define a high-risk patient population and may lead to more appropriate therapy and improved outcomes in these patients [6]. Cardiac troponins T and I are highly sensitive and specific for cardiac damage. Troponin I and T are of equal clinical value. Serum levels increase within 3-12 hours from the onset of chest pain, peak at 24-48 hours, and return to baseline over 5-14 days. The risk of death from an ACS is directly related to troponin level and patients with no detectable troponins have a good short-term prognosis [7].

Inflammatory markers such as high-sensitivity C-reactive protein (HSCRP) may provide a novel method for detecting individuals at high risk of plaque rupture. Several large-scale prospective studies demonstrate that HSCRP is a strongly independent predictor of future myocardial infarction and stroke among apparently healthy men and women [8]. Elevated CRP levels detected by a high-sensitivity CRP test relate to an increased risk of mortality. C-reactive protein levels allowed a differentiation between high-risk and low-risk groups among patients with normal troponin levels, for whom the overall 14-day mortality rate was only 1.5% when these patients had an elevated CRP level [9].

MATERIALS AND METHODS

Study population

This study was cross sectional study. Was conducted in Alshaab Hospital and Sudan cardiac Centre, Khartoum state. During September to November 2014. A total of 70 patients categorized according to ECG results to non-ST elevation myocardial infarction (NSTEMI) or ST myocardial infarction (STEMI) patients or unstable angina (UA). The age ranged between 40-90 years.

Blood sample and data collection

The data was collected using questionnaire. Blood Samples were collected and separated to obtain serum to be analyzed for troponin I and hs-CRP, centrifuged at 4000 rpm for 3 minutes, and then serum samples were stored at -20 0c until analysis. serum troponin I was estimated using immune assay method, while serum HSCRP was estimated using immunofluorescence method

Inclusion criteria

Patients should be with Acute Coronary Syndrome.

Exclusion criteria

Patients with inflammatory disease or recent infectious were excluded from the study.

Statistical analysis

The data was analyzed by the computer software program Statistical Package for Social Sciences (SPSS version 10, Chicago). Results were expressed in mean ± standard deviation (M±SD).

RESULTS

This study shows that 6.7% of total 30 STEMI patients had significantly higher Hs-CRP compared to NSTEMI patients who show 50 % of total 12 NSTEMI patients had elevated Hs-CRP at baseline samples in the other ACS 35.7% of total 28 OF UA shows slightly elevated Hs-CRP and 64.3% had normal Hs-CRP regarding too Troponin I, the shows 96.6% of total 30 STEMI patients had significantly higher cTn compared to NSTEMI patients which shows 50% of total 12 NSTEMI patient also had significantly higher cTn while in the other side of UA the results shows only 3.6% of 28 patient with UA had high cTn There was a significant difference regarding peak of Troponin I and Hs-CRP levels between the 3 groups, STEMI patients had significantly higher peak Troponin I and Hs-CRP levels compared to NSTEMI patients and UA.

Table 1. Frequency of patients according to ECG results

Diagnosis	Patients Frequency	Percentage%
STEMI	30	42.9%
NSTEMI	12	17%
UA	28	40%

Table 2. Mean of age, hs-CRP, and Troponin I in study population

Variable	Mean ± SD
Age	65.0 ±10.4
Troponin I ng/ml	6.5 ± 8.2
Hs-CRP mg/ml	10.5 ± 5.7

Table 3. Descriptive analysis of cardiac markers according to final diagnosis

Cardiac Markers	Final diagnosis		
	UA	STEMI	NSTEMI
Hs-CRP			
Positive (>5mg/L)	35.7%	6.7%	50%
Negative (<5 mg/L	64.3%	93.3%	50%
Troponin I			
Positive (>1.5 ng/mL)	3.6%	96.6%	50%
Negative (<0.6 ng/mL)	96.4%	3.33%	50%

DISCUSSION

Several well-designed studies have shown that cardiac troponin (cTnI and cTnT) to be the most diagnostically sensitive and specific biomarker of myocardial injury [10-12].

Moreover, on the basis of improved sensitivity and superior tissue specificity compared to other biomarkers of necrosis, cTn is recommended for the diagnosis of AMI by the National Academy of Clinical Biochemistry (NACB) Laboratory Medicine Practice Guidelines[10] and the International Committee `of Experts in Epidemiology, Pathology, Clinical, and Laboratory Medicine [13].The kinetics of cTnI and cTnT are similar; cTnI and cTnT are detectable in the serum within 4 to 12 hours after the onset of AMI, and depending on the duration of ischaemia and reperfusion status, peak values occur 12 to 48 hours from symptoms onset [14]. Therefore, serial sampling, including a baseline sample and follow-up examination 8 to 12 hours after symptom onset is recommended [15]. Because of the tissue specificity of cTn, any reliably-detected concentration of cTn in the peripheral circulation as abnormal and indicative of myocardial injury [16]. Moreover, several studies showed that patients presenting with an increased cTn concentration had a poor prognosis compared to those without detectable cTn[17]. Although cTnI is cleared more quickly from the circulation than cTnT, both isoforms remain elevated in the serum for several days after injury, allowing for diagnostic confirmation, even in patients presenting with delayed symptoms. Because of the long half-lives, one of the disadvantages of using cTn is that neither cTnI nor cTnT assays can be used for detection of re-infarction after an index event. The other disadvantage is that cTnT is present in small amounts in skeletal muscle and is re-expressed in diseases that involve skeletal muscle degeneration (e.g., Duchenne muscular dystrophy)[18].

Moreover, myocardial injuries, including cardiac trauma, congestive heart failure, and an elevated cTn without clinical evidence of ACS should prompt a search for other possible hypertension, as an elevated cTn does not indicate its mechanism [11].

When the cTn is not available, the best alternative marker is the High-Sensitivity C-Reactive protein(hsCRP).

C-reactive protein (CRP) is increased after ACS, particularly in association with myocardial necrosis, ref lecting the level of inflammation in the myocardium.

Elevated serum CRP obtained within 12 to 24 hours of symptom onset in a study of 448 patients with acute MI (ST-elevation MI, 76%) was associated with larger echocardiographic infarct size, higher 30- day mortality rate, and development of heart failure [19].In contrast, CRP measured within 6 hours of symptom onset in 483 patients with acute ST-elevation MI was not associated with 30-day mortality rate or development of heart failure [20].

H-s CRP is relatively sensitive, but the specificity is in doubt as H-s CRPis elevated in any inflammatory disease.

By comparing with those studies , this study shows that 6.7% of total 30 STEMI patients had significantly higher Hs-CRP compared to NSTEMI patients which show 50 % of total 12 NSTEMI patients had elevated Hs-CRP at baseline samples in the other ACS 35.7 % of total 28 OF UA shows slightly elevated Hs-CRP and 64.3% had normal Hs-CRP regarding too Troponin I, the shows 96.6% of total 30 STEMI patients had significantly higher cTn compared to NSTEMI patients which shows 50% of total 12 NSTEMI patient also had significantly higher cTn while in the other side of UA the results shows only 3.6% of 28 patient with UA had high cTn There was a significant difference regarding peak of Troponin I and Hs-CRP levels between the 3 groups, STEMI patients had significantly higher peak Troponin I and Hs-CRP levels compared to NSTEMI patients and UA. So we can relies that the cTn appears to be the most sensitive and specific biomarker among all other diagnostic biomarkers for ACS.

CONCLUSION

Troponin have significantly higher peak compared to Hs-CRP in all 3 groups of ACS STEMI, NSTEMI AMI and UA patient. These data suggest that the lack of the specificity of Hs-CRP will play an independent role in replacing Hs-CRP testing by cTn as the gold standard, serial testing of cTn and Hs-CRP has been suggested to increase the sensitivity and specificity in detecting myocardial injury.

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REFERENCES

1. Fuster V, Badimon L, Cohen M, Ambrose JA, Badimon JJ, Chesebro J. (1988). Insights into the pathogenesis of acute ischemic syndromes. *Circulation*, 77(6), 1213-1220.
2. Fuster V, Badimon L, Badimon JJ, Chesebro JH. (1992). The pathogenesis of coronary artery disease and the acute coronary syndromes (2). *N Engl J Med*, 326(5), 310-318.
3. Libby P. (2001). Current concepts of the pathogenesis of the acute coronary syndromes. *Circulation*, 104(3), 365-372.
4. Chest pain of recent onset, NICE Clinical Guideline (March 2010).
5. Chan D, Ng LL. (2010). Biomarkers in acute myocardial infarction. *BMC Med*, 7, 8, 34.
6. McCord J. (2013). Will high-sensitivity troponin assays lead to improved outcomes in patients with acute coronary syndrome? *Coron Artery Dis*, 24(8), 713-5.
7. Paul M, Ridker, MD, MPH. (2001). High-Sensitivity C Reactive Protein Potential Adjunct for Global Risk Assessment in the Primary Prevention of Cardiovascular Disease. *Circulation*, 103, 1813-1818.
8. Morrow DA, Rifai N, Antman EM et al. (1998). C-reactive protein is a potenn predictor of mortality independently of and in combination with troponinT in acute coronary syndromes, a TIMI 11 A substudy. *J Am Coll Cardiol*, 31(7), 1460-146.
9. Morrow DA, Cannon CP, Jesse RL, Newby LK, Ravkilde J, Storrow AB et al. (2007). National Academy of Clinical Biochemistry Laboratory Medicine Practice Guidelines, clinical characteristics and utilization of biochemical markers in acute coronary syndromes. *Clin Chem*, 53, 552-74.
10. Panteghini M. (2002). Acute coronary syndrome, biochemical strategies in the troponin era. *Chest*, 122, 1428-35.
11. Gibler WB, Lewis LM, Erb RE, Makens PK, Kaplan BC, Vaughn RH et al. (1990). Early detection of acute myocardial infarction in patients presenting with chest pain and nondiagnostic ECGs, serial CK-MB sampling in the emergency department. *Ann Emerg Med*, 19, 1359-66.
12. Alpert JS, Thygesen K, Antman E, Bassand JP. (2000). Myocardial infarction redefined—a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. *J Am Coll Cardiol*, 36, 959-69.
13. Boden WE, Shah PK, Gupta V, Ohman EM. (2008). Contemporary approach to the diagnosis and management of non-ST-segment elevation acute coronary syndromes. *Prog Cardiovasc Dis*, 50, 311-51.
14. No authors listed. (1997). Evaluation of a bedside whole-blood rapid troponin T assay in the emergency department. Rapid Evaluation by Assay of Cardiac Troponin T (REACTT) Investigators Study Group. Acad Emerg Med, 4, 1018-24.
15. Jaffe AS, Ravkilde J, Roberts R, Naslund U, Apple FS, Galvani M et al. (2000). It's time for a change to a troponin standard. *Circulation*, 102, 1216-20.
16. Bonaca MP, Morrow DA. (2008). Defining a role for novel biomarkers in acute coronary syndromes. *Clin Chem*, 54, 1424-31.
17. Bodor GS, Survant L, Voss EM, Smith S, Porterfi eld D, Apple FS. (1997). Cardiac troponin T composition in normal and regenerating human skeletal muscle. *Clin Chem*, 43, 476-84.
18. Suleiman M, Aronson D, Reisner SA et al. (2003). Admission C-reactive protein levels and 30-day mortality in patients with acute myocardial infarction. *Am J Med*, 115, 695-701.
19. Mega JL, Morrow DA, De Lemos JA et al. (2004). B-type natriuretic peptide at presentation and prognosis in patients with ST-segment elevation myocardial infarction, an ENTIRE-TIMI-23 substudy. *J Am Coll Cardiol*, 44, 335-339