Proceeding S.Z.P.G.M.I vol: 10(3-4) 1996, pp. 94-95.

Role of Cardiac Troponin I (cTn I) in The Management of Chest Pain, Peri-operative Myocardial Damage and Thrombolytic Treatment

S. Shahjahan
Department of Biochemistry, Shaikh Zayed Medical Complex, Lahore

ABSTRACT

A new marker for myocardial Infarction Cardiac Troponin I (cTn I) has recently been reported. cTn I shows earliest rise after 90-180 minutes and level remains high upto 6-9 days. cTn I estimation has shown great promise in diagnosis of MI in cases of chest pain, perioperative myocardial damage, monitoring of thrombolytic treatment for reperfusion. cTn I has greater specificity for M I than CK/MB and Myoglobin estimations.

INTRODUCTION

Myocardial injury causes release of many substances, like any other tissue injury. As the methods became available, the estimation of cardiac enzymes, CPK, AST and LDH became standard ancillary investigations for suspected myocardial infarction (MI) and its follow up. As all these enzymes could also be contributed by other tissues CK/MB fraction of CPK was found to be more specific for myocardial injury. However specificity of CK-MB fraction was found to be only 85.6% as it could be contributed by muscles and renal tissue as well; Myoglobin, a muscle protein was found to be even less specific (61.4%) for MI[1]. Increased levels of CK/MB have been reported in cases without myocardial infarction having muscle injury and disease (21.86%), in marathon runners (4.7%) and in 74% of patients who were on renal dialysis[2].

Role of cardiac troponin I in diagnosis of MI

Troponin I is a contractile muscle protein comprising of three forms. Two are related to skeletal muscle and one to cardiac fibres, disignated as cardiac Troponin I (cTn I). Another cardiac Troponin fraction, cardiac Troponin T (cTn T) was also found in cardiac fibres[3]. 784 monoclonal antibodies were investigated by immunoenzymatic assay for an optimal pain for measuring cTn I[3]. These investigators were able to detect. 0.2-20 ug/L of cTn I in the serum of acute myocardial infarction (AMI) patients. cTn I was not found in cases of muscle injury, rhabdomyosis and marathon runners[4]. cTn I was detected relatively earlier (2.4-6.4 hrs) after on-set of chest pain and detection levels remained between 5 to 9 days after the chest pain[3]. Specificity of cTn I was also verified by other workers[5,6]. A greater sensitivity of cTn I over other markers 90 minutes after onset of chest pain, has also been reported[2].

Perioperative M.I. in non-cardiac surgery patients

Peri-operative myocardial infarction has been reported to be the most common cause of mortality in non-cardiac surgery[7]. Diagnosis of peri-operative MI has remained a challenging task. Clinical symptoms and ECG changes had inherent limitations. CK-MB estimation has enhanced detection of peri-operative MI[8], but skeletal muscle damage during surgery limits its specificity[1,2]. In one study, out of 108 patients undergoing vascular surgery 8 patients developed AMI, all patients were monitored before and after operation, with six hourly investigations which included CK-MB while all the eight patients had elevated serum cTn I level. False positive CK-MB was seen in 19% of these patients while only 1% showed slight elevation of

cTn I[4]. Similar findings have also been reported by a number of other workers[8-11].

MI in critically ill patients in medical and respiratory ICU

Critically ill patients in the medical ward and respiratory ICU were studied for any myocardial damage. Out of 209 patients studied 32 cases (15%) showed myocardial damage on basis of raised cTn I levels. Out of these 32 cases only 12 were recognised by ICU staff as acute MI while in other 20 young black patient myocardial damage was not suspected and showed increased morbidity and mortality[12].

Monitoring of thrombolytic therapy

No biochemical test was available for evaluation of the successful reperfusion after thrombolytic therapy. Now a number of recent reports are available showing a significantly increased level of cTn I as compared to pre-therapy level after 90 minutes of thrombolytic therapy, in all cases where reperfusion has successfully occured[2,13].

Cardiac troponin T and unstable angina

Another marker Cardiac Troponin T (cTn T) has also been studied, but due to its biphasic response it was not found to be as useful as cTn I during early stages of MI. However cTn T lasts longer than cTn I. Thus many cases labelled as unstable angina were reclassified as having micro-infarctions on basis of raised cTn T level[13]. Thus, there may be need for redefining unstable angina. Due to its longer life cTn T may be a better marker for detection of remote cardiac damage when CK-MB, SGOT and LDH have come down to normal levels[13].

Methods for cTn I

Both quantitative and qualitative methods are available for assay and detection of significant level of cTn I. Quantitative Enzymes Immunoassays are time consuming and difficult to run as stat samples[3]. However qualitative latex based detection has proved to equally accurate in the diagnosis of MI within 90 minutes to 2 hours of chest pain. Test takes only about 10 minutes reporting time.

CONCLUSION

Cardiac Troponin I levels have shown great promise in the diagnosis of MI in cases of chest pain peri-operative myocardial damage and monitoring of thrombolytic treatment, showing greater specifity as compared to cardiac enzymes, including CK-MB and Myglobin estimation.

REFERENCES

1. Adam JE, 3rd; Bodor GS, Davila Roman VG, Delmez JA, Apple FS, Ladenson JH, Jafee AS. Cardiac Troponin I. A marker with high specificity for cardiac injury. Circulation 1993; 88: 101-6.
2. Apple FS, Sharkey SW, Henry TD. Early serum cardiac troponin I and T concentration after successful thrombolysis for acute myocardial infarction. Clin Chem 1995: 41 (8pt 1): 1197-8.
3. Larue C, Calzolari C, Bertinchant JP, Leclercq F, Grolleau R and Pan B. Cardiac Specific immunoenzymometrio assay of Troponin I in the early phase of acute myocardial infarction. Clin Chem 1993; 39: 972-9.
4. Adams JE 3rd, Schechtman KB, Landt Y, Jaffe AS. Comparable detection of acute myocardial infarction by creatine kinase MB Isoenzyme and cardiac troponin I. Clin Chem 1994; 40 (7 pts 1): 1291-5.
5. Sela BA. Troponins T and I new markers for cardiac damage. Harefuah 1994; 127: 321-4.
6. Suetomi K, Takahama K. A sandwich enzyme immunoassay for cardiac troponin I. Nippon Hoigaku Zasshi 1995; 49: 26-32.
7. Adams JE 3rd, Sicard GA, Allen BT, Bridwell KH, Lenke LG, Davila, Roman VG, Bodor GS, Landenson JH, Jafee AS. Diagnosis of Perioperative Myocardial infarction with measurement of cardiac troponin-I. N Engl J Med 1994; 330: 670-4.
8. Mangano DT. Beyond CK-MB. Biochemical markers for perioperative myocardial infarction. Anaesthesiology 1994; 81: 1317-20.
9. Drown JD. Cardiac Troponin I: a marker for cardiac injury occuring peri-operatively. Prog Cardiovasc 1994; 9: 42.
10. MAIR J, Larue C, Mair P, Balogh D, Calzolari C, Puschendorf B. Use of cardiac troponin I to diagnose perioperative myocridial infarction in coronary artery bypass grafting. Clin Chem 1994; 40 (11 Pt1): 2066-70.
11. Ordonez-Lianos J, Guido-Soldevila J, Ferrer-Garcia, D. The usefulness of Biochemical markers in detecting myocardial damage associated with a peri-operative infarct and heart transplant rejection. Rev Esp Cardial 1995; 48(2): 77-84.
12. Guest TM, Ramanathan AV, Tuteur PG, Schechtman KB, Landerson JH, Jaffe AS. Myocardial injury in critically ill patients. A frequently unrecognized complication. JAMA 1995; 173: 1945-9.
13. Scheffold T, Zehelein J, Muller Bardorff M, Katus HA. Monitoring ischemia with new marker. Z Kardial 1994; 83 Supp 6: 75-82.

The Author

S. Shahjahan
Professor
Department of Biochemistry,
Shaikh Zayed Medical Complex,
Lahore

Address for Correspondence

S. Shahjahan
Professor
Department of Biochemistry,
Shaikh Zayed Medical
Complex, Lahore