COMPARISON OF TROPONIN T AND ENZYME LEVELS IN ACUTE MYOCARDIAL INFARCTION AND SKELETAL MUSCLE INJURY

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Background: The objective of this study was to compare the levels of troponin T and enzymes levels in myocardial infarction and skeletal muscle injury. **Methods:** This study was carried out at Basic Medical Sciences Institute, JPMC Karachi, Pakistan. Ninety subjects were selected. Thirty controls, thirty patients suffering from myocardial infarction and thirty suffering from skeletal muscle injury were selected from National Institute of Cardiovascular Diseases. Creatine kinase, aspartate amino-transferase, lactate dehydrogenase and Troponin T were determined by kit methods. **Results:** Troponin T level rises significantly ($p<0.01$) in patients suffering from myocardial infarction. Creatine kinase (CK), CKMB, aspartate aminotransferase and lactate dehydrogenase levels rises significantly ($p<0.01$) in both groups compared with controls. **Conclusion:** Troponin T is an early indicator of myocardial infarction and is superior to CKMB in diagnosis of myocardial injury. There is no increase in troponin T levels in skeletal muscle injury.

Key words: Troponin T, Acute, Myocardial infarction, Skeletal, muscle, injuries

INTRODUCTION

Acute myocardial infarction (AMI) is a focus of necrosis resulting from inadequate perfusion of tissues. Clinical syndrome resulting from such ischemia is manifested by sudden cardiac death. Typical signs and symptoms of infarction such as crushing chest pain, diaphoresis, malignant vascular arrythmia, congestive cardiac failure (CCF) or shock or atypical presentation that can be clinically silent may occur[1]. Low arterial flow, low oxygen retention and presence of anaerobic glycolysis characterize myocardial ischemia. Two forms of silent myocardial ischemia are recognized. Type I silent ischemia occurs in patients with obstructive coronary artery disease, which do not experience angina at any time in any of its recognized form. The second and much more frequent form designated type II silent ischemia, occur in usual form of chronic stable angina, unstable angina and Prinzmetal angina[2]. Recently, popularized ambulatory ECG in studies to detect ischemia has revealed much higher incidence of silent ischemia. It has documented that ST segment shift either increased or decreased correlate well with other markers of myocardial ischemia.

The impressive performance of CK, LDH and their respective isozymes did not stop investigators for searching better markers for myocardial damage. During the past decade a number of new analysis have been studied and their respective performance have been documented which include myoglobin, CK isoform, myosin light chain, troponin T and troponin I. Sensitivity is not a major concern but specificity is a problem because the standard markers are not restricted to heart. CKMB is present in low concentrations in skeletal muscles and rhabdomyolysis can elevate CKMB into abnormal range. An increase in LDH isoenzyme-1 is characteristic of myocardial damage but hemolysis and cortical necrosis also can generate this result[3].

The objective of the study was to compare the levels of Troponin T and enzymes in patients with acute myocardial infarction and skeletal muscle injury.

MATERIAL AND METHODS

The study included 90 subjects. Among them 30 were control subjects, 30 suffering from myocardial infarction and 30 were patients suffering from skeletal muscle injury. The subjects were matched for age, sex, height and weight. Blood samples were collected from NICVD and JPMC, Karachi

Those subjects were included in myocardial infarction group who fulfilled following criteria:

Onset of typical retrosternal pain or discomfort of recent onset lasting for 20–30 minutes.

ECG findings of ST segment, appearance of Q waves of more than 0.045 second duration or >4 mm depth.

Increased activity of standard cardiac enzymes i.e. LDH, AST, CKMB

Diagnosis of acute skeletal muscle trauma (rhabdomyolysis) was made by obvious physical acute skeletal muscle injury diagnosed by attending casualty medical officer along with attending physician.

Creatine kinase (CK), aspartate amino-transferase (AST), lactate dehydrogenase (LDH) were determined by kit methods supplied by Boehringer Mannheim. Semi-automated analyses were done on photometer 5010. Troponin T was also estimated by kit method supplied by Boehringer Mannheium. Automated analysis of Troponin T was done on ELISA (ES-300).

Mean and standard error of mean was calculated. Student's t-test was applied and probability was determined.

RESULTS

Table 1 shows blood pressure and pulse rate in all the three groups. Systolic, diastolic blood pressure and pulse rate was significantly increased (p<0.01) in subjects suffering from myocardial infarction and skeletal muscle injury compared with controls.

Table 1: Comparison of Systolic and Diastolic Blood Pressure and Pulse Rate among Normal Healthy Controls, Acute Myocardial Infarction and Skeletal Muscle Injury Groups

(The values are expressed as mean and number of subjects is given in parenthesis)

Groups	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Pulse Rate Per Minute
Normal Healthy Controls (30)	129.16 ± 1.52	82.90 ± 0.86	82.40 ± 1.14
Acute Myocardial Infarction (30)	139.00* ± 4.79	90.66* ± 3.11	85.80 ± 2.17
Skeletal muscle Injury (30)	135.66* ± 2.96	81.83 ± 1.48	87.10* ± 0.80

* p< 0.01 significant compared with healthy controls

LDH, AST and CK levels increased significantly in patients suffering from myocardial infarction as well as skeletal muscle injury compared with controls. CKMB and Troponin T levels increased significantly in sufferers of myocardial infarction compared with controls. Both these levels remained non significant in patients suffering from skeletal muscle injury compared with controls (Table-2).

Table 2: Comparison of AST, LDH, CK, CKMB and Troponin T among Normal Healthy Controls, Acute Myocardial Infarction and Skeletal Muscle Injury Groups

Groups	AST (U/L)	LDH (U/L)	CK (U/L)	CK-MB Troponin T (U/L) (ηg/ml)	Troponin T (U/L) (ηg/ml)
Normal Healthy Controls (30)	24.76 ± 1.95	266.63 ± 12.14	87.00 ± 9.13	9.56 ± 0.020	0.020 ± 0.003
Acute Myocardial Infarction (30)	218.13* ± 26.3	1108.16* ± 132.14	809.83* ± 94.69	51.96* ± 3.01	3.01* ± 0.69
Skeletal Muscle Injury (30)	32.66* ± 3.18	323.90* ± 15.2	594.9* ± 57.4	13.83* ± 0.032	0.032 ± 0.005

(The values are expressed as mean and number of subjects is given in parenthesis)

* p < 0.01 significant compared with healthy controls

DISCUSSION

The myocardial infarction is the common medical emergency in developed as well as developing countries. However 50 to 70% of patients admitted in hospitals with diagnosis of unstable angina or acute myocardial infarction are subsequently not found to have acute ischemic syndrome. After acute myocardial infarction, cardiac enzymes and proteins are released into the plasma and are used as biochemical markers of cardiac muscle injury. In this study cardiac troponin T was measured with all cardiac enzymes in myocardial injury as well as skeletal muscle injury patients.

Serum LDH, CK, CKMB, AST levels increase significantly in myocardial infarction as well as skeletal muscle injury. Lott and Stang[4] reported increased AST and CK levels in myocardial ischemic injury because it is present in high concentration in myocardial cells and is released from irreversibly damaged cells. Vessels *et al*[5] indicated the importance of raised levels of LDH in acute myocardial infarction. Serum LDH and AST levels increase significantly in skeletal muscle injury groups compared with controls but remained within normal limits. CK level rises significantly in skeletal muscle injury compared with controls. Since CK is present in abundance in skeletal muscle and brain, elevated levels of Ck can be procured in number of disorders like trauma, surgery, electric shock, rhabdomyolysis, polymyositis and muscle dystrophy.

In our study CKMB level increased significantly in acute myocardial infarction group. This is in accordance with studies by Collinson *et al*[6] and Wu *et al*[7].

Cardiac Troponin T levels also increased significantly in patients suffering from acute myocardial infarction. This is in accordance with studies by Wu *et al*[7], Wu and Lane[8], Collinson,[6], Brown and Bertolet,[9], Mulner *et a.*,[10], Apple,[11] and Badroff *et al*[12].

CKMB and Troponin T levels in skeletal muscle injury group was non significant compared with controls. This is in accordance with study by Collinson *et al*[13]. None of the patient showed any sort of cross reactivity of skeletal

muscle troponin with cardiac troponin. In our study population there was normal level of troponin T in skeletal muscle, having no cardiac symptoms or other evidence of cardiac disease. Troponin T is used to successfully differentiate the rise in CK after skeletal muscle injury as non-cardiac source. This might explain higher sensitivity and specificity for troponin T to diagnose AMI and is investigation of choice to exclude myocardial damage in patients with increase CK.

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REFERENCES

1. Sobel BE. Acute myocardial infarction. In Cecil Textbook of Medicine W.B. Saunders Company London. 20th edition (1), 1996; pp. 301.
2. Braunwald E. Diseases of the heart, aorta and pulmonary vascular bed. In: Heart Disease. W.B. Saunders Company Philadelphia, 1997; pp. 877-1642.
3. Schreiber WE. Laboratory assessment of myocardial damage. Am J Clin Path 1997; 107(4): 383-4.
4. Lott JA, Stang JM. Serum enzyme and isoenzyme in the diagnosis and differential diagnosis of myocardial ischemia and necrosis. Clin Chem 1980; 26: 1241-50.
5. Vessel ES, Bean AG. Localization of lactic acid dehydrogenase activity in serum fractions. 1957, Proceedings of the Society of Exp Biol Med; 94: 96-99.
6. Collinson PO, Mosley D, Stubbs PJ, Carter GD. Troponin T for differential diagnosis of ischemic myocardial damage. Am Clin Biochem 1993;30: 11-16
7. Wu AHB, Valdes JR, Apple FS, Gornet T, Stone MA, Stokes MS, et al. Cardiac Troponin T immunoassay for diagnosis of acute myocardial infarction. Clin Chem 1994;40 (6): 900-907
8. Wu AHB, Lane PL. Meta analysis in clinical chemistry: Validation of cardiac Troponin T as a marker for ischemic heart diseases. Clin Chem 1995; 41/8(b):1288-1233.
9. Brown CS, Bertolet BD. Cardiac troponin. Chest 1997;111/1:2-4
10. Mulner M, Hirschl MM, Herkner H, Sertz F, Leitha T, Exner M et al. Creatine Kinase MB fraction and cardiac troponin T to diagnose acute myocardial infarction after cardiopulmonary resuscitation. JACC 1996;28(5):1220-5
11. Apple FS. Acute myocardial infarction and coronary reperfusion. AJCP 1992; 97(2): 217-226.
12. Badroff MM, Hallermayor KI, Schroder A, Ebert C, Borgya A, Gerhardt W. Improved troponin T ELISA specific for cardiac troponin T isoform assay development and analytical clinical validation. Clin Chem 1997;43(3): 458-66.
13. Collinson PO, Chandler HA, Stubbs PJ, Moseley DS, Levis D and Suman MD. Measurement of serum troponin T, creatine kinase MB isoenzyme and total creatine kinase following ardous physical training. Ann Clin Biochem 1995; 32:445-50.

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