

RESEARCH ARTICLE

LEVELS OF TROPONIN AND CREATINE KINASE MB IN MYOCARDIAL INFARCTION PATIENTS

Tariq Elfatih Elmisbah¹, Mohammed Aiderous²

¹Department of Medical Laboratory Sciences, College of Applied Medical Sciences, Taif University, Kingdom of Saudi Arabia

²Elgad International College for Medical Sciences.

Received: 21 May, 2018/ Revision: 4 July, 2018/ Accepted: 10 November, 2018

ABSTRACT: Background. Myocardial infarction (MI) commonly known as a heart attack occurs when blood flow stops to part of the heart causing damage to the heart muscle. Troponin, or troponin complex, is a complex of three regulatory proteins (troponin C, troponin I, and troponin T), that is integral to cardiac muscles. An increased level of the cardiac protein isoform of troponin circulating in the blood has been shown to be a biomarker of heart disorders, the most important of which is myocardial infarction. Creatine kinase –MB is an enzyme found in the heart and rises when heart muscle is damaged. **Study design.** This study was designed as a case control study in a certain state, Saudi Arabia to determine the frequency of determine the levels of troponin and creatine MB in myocardial infarction patients. **Materials and methods.** One hundred fifty Saudi myocardial infarction patients admitted to Khamis Mushiat central hospital in KSA during the period from March 2016-April 2017 were recruited to participate in this study as well as fifty apparently healthy volunteers were enrolled as a control group. A venous blood 2.7ml sample was collected in a plastic tube containing sodium citrate anticoagulant for immunological analysis. Non-probability sampling method was used. A sandwich electrochemiluminescence immunoassay which employs 2 monoclonal antibodies was used. **Results.** Results showed that the mean of troponin among study sample was increased in compared to control (P-value is significant), and also the study showed that the mean of creatine kinase MB levels was increased in compared with control (P-value is significant). **Conclusion.** This study proved that the levels of troponin and creatine kinase were increased in myocardial infarction patients.

KEYWORDS: Myocardial infarction, troponin, creatine kinase MB.

INTRODUCTION:

Myocardial-infarction (MI) commonly known as a **heart attack** occurs when blood flow stops to part of the heart causing damage

to the heart muscle. The most common symptom is chest pain or discomfort which may travel into the shoulder, arm, back, neck,

Corresponding Author:

Dr Tariq Elfatih Elmisbah,

Department of Medical Laboratory Sciences, College of Applied Medical Sciences, Taif University, Kingdom of Saudi Arabia.



or jaw. Often it is in the center or left side of the chest and lasts for more than a few minutes. The discomfort may occasionally feel like heartburn. Other symptoms may include shortness of breath, nausea, feeling faint, a cold sweat, or feeling tired.^[16] About 30% of people have atypical symptoms,^[17] with women more likely than men to present atypically.^[18] Among those over 75 years old, about 5% have had an MI with little or no history of symptoms.^[19] An MI may cause heart failure, an irregular heartbeat, or cardiac arrest.^{[4][5]}

Most MIs occur due to coronary artery disease.^[20] Risk factors include high blood pressure, smoking, diabetes, lack of exercise, obesity, high, poor diet, and excessive alcohol, among others.^{[21][22]} The mechanism of an MI often involves the rupture of an atherosclerotic leading to complete blockage of a coronary artery.^[23] MIs are less commonly caused by spasms which may be due to cocaine, significant emotional stress, and extreme cold, among others.^{[24][25]} A number of tests are useful to help with diagnosis including electrocardiograms (ECGs), blood tests, and coronary angiography.^[26] An ECG may confirm an ST elevation MI if ST elevation is present.^[27] Commonly used blood tests include troponin and less often creatine kinase MB.^[28] Troponin, or troponin complex, is a complex of three regulatory proteins (troponin C, troponin I, and troponin T), that is integral to cardiac muscles. Troponin is attached to the protein tropomyosin and lies with the groove between actin filaments in muscle tissue. In relaxed muscles, tropomyosin blocks the attachment site for the myosin crossbridge, thus preventing contraction.^[17]

Mutations in the cardiac troponin subunits can result in cardiac myopathies.^[12] Creatine kinase (CK) is an enzyme found in body muscles. The level of the CK enzymes rises when there is a damage to body muscles.^[17] The three types of CK are called isoenzymes, they are CK-MM, CK-MB, and CK-BB. CK-MB found in the heart and rises when heart muscle is damaged. CK-MB generally rises after heart attack.^[22] Normal level for CK-MB is 0%.^[12]

MATERIALS AND METHODS

In this study two hundred samples were collected, one hundred fifty samples from myocardial

patients in Abha general hospital-Abha-KSA used as test, and fifty samples were collected from healthy individuals used as control. The levels of troponin and creatine kinase MB were -Anti thrombin III in myocardial infarction will be detected using Enzyme-linked Immunosorbent Assay (ELISA).

Measurement of CK-MB activity

Principle

A sandwich electrochemiluminescence immunoassay which employs 2 monoclonal antibodies. Creatine kinase MB (CKMB) in the specimen reacts with both a biotinylated monoclonal CKMB-specific antibody and a monoclonal CKMB-specific antibody labeled with a ruthenium complex to form a sandwich complex. Streptavidin-coated microparticles are added and the mixture is aspirated into the measuring cell where the microparticles are magnetically captured onto the surface of the electrode. Application of voltage to the electrode induces the chemiluminescent emission, which is then measured.

Procedure

CK-MB activity was measured by Full automated cs200 fluorometric analyzer

Measurement of Troponin

Principle

Troponin T method employs 2 monoclonal antibodies specifically directed against human cardiac Troponin T. A biotinylated monoclonal antibody and a second monoclonal antibody labeled with a ruthenium complex react with Troponin T to form a sandwich complex. After the addition of streptavidin-coated microparticles, the complex becomes bound to the solid phase via interaction of biotin and streptavidin. The reaction mixture is aspirated into the measuring cell where the microparticles are magnetically captured onto the surface of the electrode. Application of a

voltage to the electrode then induces chemiluminescent emission, which is measured by a photomultiplier.

Procedure

Troponin was measured by Full automated stratus cs200florometric analyzer

Data Collection

Data was collected using data collection sheet.

Data Analysis

The data were analyzed using the SPSS computer program version 20. Qualitative data was presented as mean \pm SD qualitative data as frequency and present Chi-Square and regression analysis was done to determine the association and to estimate the risk independent 2-5 sample test was used to compare means of two quantitative variables. The level of significance was set at less than 0.05.

RESULTS

Demographic data

A total of 150 patients with MI and 50 apparently healthy volunteers were involved in this study, 101 (67.3%) of patients and 39 (78%) of control group were males and 49 (32.7%) of patients and 11 (22%) of control were female Table .1.

Table. 1: Distribution of sex among the study population

Gender	Patients		Control	
	Frequency	%	Frequency	%
Females	49	32.7	11	22
Males	101	67.3	39	78
Total	150	100%	50	100%

MI was most frequently within the age group 51-60 years followed by the age group

61-70 years, 41-50 years, >70 years, 31-40 years, 21-30 years, <10 years and 10-20 years consequently Table (2).

Table. 2: Distribution of Age groups among the study population

Age group (years)	Patients		Control	
	Frequency	%	Frequency	%
<10	4	2.7	3	6
10-20	3	2.0	3	6
21-30	10	6.7	12	24
31-40	14	9.3	5	10
41-50	27	18.3	9	18
51-60	36	24.0	8	16
61-70	30	20.0	3	6
>70	26	17.3	7	14
Total	150	100%	50	100%
Mean \pmSD	53.90 \pm11.13		44.49 \pm24.93	

The majority 133(88.7%) of patients their level greater than 25 mcg/L and 17(11.3%) of them their CK MB level was \leq 25 mcg/L, While all 50(100%) of control subjects their Creatine Kinase MB level was equal 25 mcg/L or less (Table 3).

Table. 3: Creatine kinase MB Levels among the study population

mcg/L	Patients		Control	
	Frequency	%	Frequency	%
\leq 25	17	11.3	50	100
>25	133	88.7	0	0
Total	150	100%	50	100%
Range	18-81		0-23	
Mean \pmSD	45.41 \pm19.55		12.02 \pm7.84	

Table. 4: Troponin Levels among the study population

mcg/L	Patients		Control	
	Frequency	%	Frequency	%
\leq 0.06	8	5.3	50	100
>0.06	142	94.7	0	0
Total	150	100%	50	100%
Range	0.05-3.01		0-0.05	
Mean \pmSD	0.698 \pm0.73		0.0169 \pm0.02	

Results of Troponin level of control subjects and patients were presented in Table (4), 50 out of 50 (100%) of control subjects their level was ≤ 0.06 mcg/L; whereas (5.3%) and (94.7%) of patients their levels are ≤ 0.06 and > 0.06 mcg/L, respectively.

DISCUSSION:

Myocardial infarction (MI) or acute myocardial infarction (AMI), commonly known as a heart attack occurs when blood flow stops to part of the heart causing damage to the heart muscle (McCarthy et al., 2016). Many biochemical factors play great role in regulation of coagulation process and lead to prevention of clot formation. Some of these factors are Antithrombin III, Protein C and Protein S. The etiology of MI is remains undetermined in a significant number of cases. The recognition of the naturally occurring factors, such as ATIII, PC and PS, and the fibrinolytic system, has increased our knowledge in relation to hemostatic abnormalities that may promote thrombosis and thereby contribute to MI. The objective of this study was to evaluate the plasma levels of naturally occurring inhibitors of coagulation in Saudi patients with MI and explore the role of inhibitors deficiencies as a risk factor for MI. The results of the current study revealed that, most patients with MI were found to have high CK-MB (88.7%) and Troponin (94.7%) levels; while both were normal in all subjects of the control group subjects. The diagnosis of MI is established in patients with chest pain and equivocal electrocardiogram changes by demonstrating a rise in blood levels of creatine kinase MB (CK-MB) and/or an increase in cardiac troponin I (cTnI) or cardiac troponin T (cTnT). The European Society of Cardiology (ESC) and American College of Cardiology (ACC) state that any elevation, however small, of a troponin or the creatine kinase MB iso-enzyme is evidence of myocardial necrosis and that the patient should be classified as having myocardial infarction (Antman *et al.*, 2004).

CONCLUSION:

Initial use of the sensitive troponin and creatine kinase substantially improved the early diagnosis of myocardial infarction.

REFERENCES:

1. Bjork, I; Olson, JE (1997). Antithrombin, A bloody important serpin (in Chemistry and Biology of Serpins). Plenum Press. pp. 17–33. ISBN 0-306-45698-2.
2. Collen D, Schetz J, de Cock F, Holmer E, Verstraete M (1977). "Metabolism of antithrombin III (heparin cofactor) in man: Effects of venous thrombosis of heparin administration". Eur. J. Clin. Invest 7 (1): 27–35. doi:10.1111/j.1365-2362.1977.tb01566.x. PMID 65284.
3. Conard J, Brosstad F, Lie Larsen M, Samama M, Abildgaard U (1983). "Molar antithrombin concentration in normal human plasma". Haemostasis 13 (6): 363–368. doi:10.1159/000214823. PMID 6667903.
4. Jordan RE (1983). "Antithrombin in vertebrate species: Conservation of the heparin-dependent anticoagulant mechanism". Arch. Biochem. Biophys 227 (2): 587–595. doi:10.1016/0003-9861(83)90488-5. PMID 6607710
5. Olson ST, Björk I (1994). "Regulation of thrombin activity by antithrombin and heparin". Sem. Thromb. Hemost. 20 (4): 373–409. doi:10.1055/s-2007-1001928. PMID 7899869.
6. Brennan SO, George PM, Jordan RE (1987). "Physiological variant of antithrombin-III lacks carbohydrate side-chain at Asn 135". FEBS Lett 219 (2): 431–436. doi:10.1016/0014-5793(87)80266-1. PMID 3609301.
7. https://en.wikipedia.org/wiki/Antithrombin#cite_ref-8.
8. Persson E, Bak H, Olsen OH (2001). "Substitution of valine for leucine 305 in factor VIIa increases the intrinsic enzymatic activity". J. Biol. Chem. 276 (31): 29195–29199. doi:10.1074/jbc.M102187200. PMID 11389142.
9. Stephens AW, Siddiqui A, Hirs CH (1987). "Expression of functionally active human antithrombin III". Proceedings of the National Academy of Sciences of the United States of America 84 (11): 3886–3890. doi:10.1073/pnas.84.11.3886. PMC 304981. PMID 3473488
10. Zettlmeissl G, Conradt HS, Nimt M, Karges HE (1989). "Characterization of recombinant human antithrombin III synthesized in Chinese

- hamster ovary cells". J. Biol. Chem. 264 (35): 21153–21159. PMID 2592368.
11. Gillespie LS, Hillesland KK, Knauer DJ (1991). "Expression of biologically active human antithrombin III by recombinant baculovirus in *Spodoptera frugiperda* cells". J. Biol. Chem. 266 (6): 3995–4001. PMID 1995647.
 12. Ersdal-Badju E, Lu A, Peng X, Picard V, Zendeirouh P, Turk B, Björk I, Olson ST, Bock SC (1995). "Elimination of glycosylation heterogeneity affecting heparin affinity of recombinant human antithrombin III by expression of a beta-like variant in baculovirus-infected insect cells". Biochem. J. 310 (Pt 1): 323–330. PMC 1135891. PMID 7646463.
 13. Ogston D, Murray J, Crawford GP (1976). "Inhibition of the activated C1s subunit of the first component of complement by antithrombin III in the presence of heparin". Thromb. Res. 9 (3): 217–222. doi:10.1016/0049-3848(76)90210-3. PMID 982345.
 14. Danielsson A, Björk I (1980). "Slow, spontaneous dissociation of the antithrombin-thrombin complex produces a proteolytically modified form of the inhibitor". FEBS Lett 119 (2): 241–244. doi:10.1016/0014-5793(80)80262-6. PMID 7428936.
 15. Chang WS, Wardell MR, Lomas DA, Carrell RW (1996). "Probing serpin reactive-loop conformations by proteolytic cleavage". Biochem. J. 314 (2): 647–653. PMC 1217096. PMID 8670081.
 16. "What Are the Signs and Symptoms of Coronary Heart Disease?". <http://www.nhlbi.nih.gov/>. September 29, 2014. Retrieved 23 February 2015.
 17. Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology, (ESC); Steg, PG; James, SK; Atar, D; Badano, LP;

CONFLICT OF INTEREST: Authors declared no conflict of interest

SOURCE OF FINANCIAL SUPPORT: Nil

- ✓ International Journal of Medical Laboratory Research (IJMLR) - Open Access Policy
- ✓ Authors/Contributors are responsible for originality of contents, true references, and ethical issues.
- ✓ IJMLR publishes all articles under Creative Commons Attribution- Non-Commercial 4.0 International License (CC BY-NC). <https://creativecommons.org/licenses/by-nc/4.0/legalcode>

Cite of article: [Elmisbah TE, Aiderous M; Levels of Troponin and Creatine Kinase MB in Myocardial Infarction Patients. Int. J. Med. Lab. Res. 2018, 3\(3\): 18-22](#)