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RESEARCH ARTICLE

ASSOCIATION OF BIOCHEMICAL TESTS CARDIAC ENZYME WITH MYOCARDIAL INFARCTION IN EMERGENCY HOSPITAL ERBIL- IRAQ

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ABSTRACT

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Key words:

Cardiac enzyme, Myocardial infarction. The relationship between biochemical tests and myocardial infarction has been studied. The study performed in 75 patients (45 males, 30 females) that had chest pain. The myocardial infarction tissue (MI) occurred in the locale of the hypoperfused myocardium. The whole blood taken from patients (3ml) was suffering from chest pain. Furthermore, the blood was estimated by Nano-Check in cardiovascular marker cTnl, CK-MB, and myoglobin. The patients revealed that had normal tests in spite of having chest pain. These results were taken in emergency hospital indicated that eleven patients hadpositive results, but other had negative results.

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INTRODUCTION

At the point when a myocardial infarction happens in the hypoperfused district of the myocardium, oxygen can never again be provided to the cells in the locale. Cell passing is inevitable if oxygen isn't re-established in 10-15 minutes after the arrival of specific proteins from the inside cytoplasm into the bloodstream. Several proteins are restricted to and prevalent in the heart muscle cells; they can work as cardiovascular markers and be distinguished in the blood examples of AMI patients by particular immunoassays (M. Panteghini et al, 1999, FS. Apple 1992). Regrettably, none of the cardiac specificity, and a big lifetime in circulation. This situation has led to a panel method for the utilization of markers in patients with AMI. The ingredients of this cardiac panel must consist of a marker that unexpectedly increases after cardiac damage and exceptionally cardiac tissues specific. The mixture of nTnl, CK-MB and Myoglobin extensively used in panel assays meant for the dedication of AMI in the chest pain sufferers (H.B. Alan et al, 1999). Troponin is a contractile regularity protein complex determined in the skeletal and cardiac muscle. The Troponin complex consists of three different polypeptide components, troponin-I (Tnl), troponin T (TnT), and troponin C (TnC) and plays a crucial position in the transmission of the intracellular calcium sign actinmyosinm, which interaction (JP. Mehegan and LS. Tobacman 1991).

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TnC of cardiac tissues is same as that in skeletal tissues, but Tnl ad TnT of cardiac isoforms are special to these of skeletal isoform which allow the improvement of cardiac unique antibodies (GS. Bodor et al, 1992). Moreover, the cTnl level will become expanded by the blood as an end result of myocardial infarction (JE. Adam et al, 1994). Studies on the release kinetics point out that cTnl is now not an early marker of myocardial necrosis. It seems in serum 3-6 hours after symptom onset, similar to the release of CK-MB, However, cTnl remains elevated for 4-9 days post- AMI (J. Mair et al, 1995, J. Mair et al, 1996). In addition to its utility in diagnosis, multiplied cTnl levels convey prognostic data and has been shown two to perceive patients having an increased risk of death (EM. Antman et al, 1996). Troponin is a contractile consistency protein complex decided in the skeletal and cardiovascular muscle. The Troponin complex comprises of three distinctive polypeptide parts, troponin-I (Tnl), troponin T (dynamite), and troponin C (TnC) and plays a vital position in the transmission of the intracellular calcium sign actinmyosinm, which communication (JP. Mehegan and LS. Tobacman 1991). TnC of heart tissues is same as that in skeletal tissues, yet Tnl advertisement dynamite of cardiovascular isoforms are extraordinary to these of skeletal isoform, which permit the change of heart remarkable antibodies. In addition, the cTnl level will wind up extended in the blood as a final product of myocardial infarction (JE. Adam et al, 1993, JE. Adams et al, 1994). A Concentrates on the discharge energy call attention to that cTnl is presently not an early marker of myocardial putrefaction. It appears in serum 3-6 hours after indication beginning, like the arrival of CK-MB, In any case, cTnl stays hoisted for 4-9 days post-AMI (J. Mair et al, 1995, J. Mair et al, 1996). Notwithstanding its

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utility in determination, duplicated cTnl levels pass on prognostic information and have been indicated two to see patients having an expanded danger of death (EM. Antman et al, 1996). CK (Creatine Kinase) is current in most tissues and is in specific involved with ATP regeneration. The enzyme is dimeric and exists as three isozymes, MM muscle, MB hybrid, and BB talent (D. Neumeier 1981). The MB isozyme has its best cognizance in the coronary heart muscle, subsequently, its stage in the serum has diagnostic value. The CK-MB level in day-to-day serum is less than 5 ng/ml. In cases of simple AMI, CK-MB level will emerge as accelerated internal 4-8 hours after the onset of chest pain, engaging in a peak between 12-24 hours and then drops down to ordinary with the resource of forty-eight hours. The pinnacle stage of CK-MB is 21 ng/ml or greater (JA. Lott 1984, B. Guibis et al, 1990). CK-MB has been viewed the gold accepted for the prognosis of AMI due to the fact of its cardio-specificity. However, CK-MB is now not an ideal marker to use on my personal due to the truth its stage dose now not amplify early enough to make a quick evaluation and might additionally also be elevated in one-of-a-kind condition. Although CK-MB greater focused in the myocardium (approximately 15% of the whole CK), it is moreover in skeletal muscle. False-positive elevations take place in an of clinical settings, trauma, heavy exertion, myopathies(M. Ruppert and H. R. Van 2001, CM. Schneider et al, 1995).

Myoglobin, a gas (oxygen) binding protoheme supermolecule exist within the muscle tissue, together with internal organ, skeletal and sleek muscle, has attracted tidy interest as an early marker of MI (AH. Wu 1997, C. Montague 1995). The injury to any of those muscles, hemoprotein seems within the blood faster than a different marker (H.B. Alan 1999). Levels could also be increased as early in concert hour following the onset of hurting once CK-MB levels square measure still within the vary of rang (normal)(AH. Wu 1997, P. Carraro et al, 1994, A. Clerico et al, 1993). This speedy look is due to the placement of hemoprotein within the cell and its low relative molecular mass. hemoprotein usually elevates two-four hours when the onset of an infraction, peaks at six-twelve hours and returns to normal inside twenty-four - thirty-six hours. Usually, the extent of myoglobin in liquid body substance is thirty-eighty ng/ml. In patients with MI, the extent may increase or so ten times on top of the limit of normal. Hemoprotein exhibits a high clinical sensitivity for AMI however poor specificity (M. Panteghini 1999, FS. Apple et al. 1990). Several studies recommend that hemoprotein could also be an honest screening assay in emergency rooms for the first identification of AMI. However, elevated hemoprotein values ought to be cautiously understood if the patient has excretory organ pathology of musculus injury as a result of detection of hemoprotein in a very patient suspected of AMI might have to be supplemented by the presence of a lot of definitive internal organ marker. However, a negative lead to a patient admitted inside two- nine hours when the onset of hurting might facilitate in ruling out AMI.

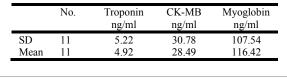
METHOD AND MATERIALS

The Nano-CheckTM AMI 3 in 1 test contain all the reagents necessary for the detection of cTnl, CK-MB and myoglobin in human whole blood, serum, and plasma. The strip coated with monoclonal mouse anti-CK-MB, anti-Myoglobin, and streptavidin on the test line, and dye pad infused with biotinylated monoclonal mouse anti-cTnl antibody and gold colloidal particles coupled with anti-CK-MM, anti-cTnl, and anti-myoglobin antibodies. Stabilizer containing 0.05% sodium azide and BSA protein is deposited in the dye pad in dried form. The nano - CheckTM test is containing a membrane strip in a sealed pouch with desiccant, instruction for use, a suitable pipette.

RESULTS

The samples of whole blood were taken around (3ml) transfer into anticoagulant tubes. Seventy-five tubes (45 males, 30 females) when the investigation of the patients suffering from chest pain. Measure the sample by apparatus of Nano-Checker 710 reader. When the whole blood was transferred by dropper into the strip after 15 minutes check a result. Eleven of the patients have the positive result of cTnl, CK-KB, Myoglobin but other patients have the negative result. The normal value of tests must be in range cTnl< 0.5 ng/ml, CK-MB< 5.0 ng/ml, myoglobin < 80 ng/ml. Table.1 Showed the standard deviation and mean values for patients had abnormal values. Table.1 showed the standard deviation and mean of 11 patients. Figure.1 showed the distribution of 11 patients, which had positive results (above the normal range) troponin, CK-MB, and Myoglobin. Figure 2 to 4 Showed the distribution of eleven patients with troponin, CK-MB and Myoglobin values which have positive results.

Table 1. Showed the number of patients, standard deviation and mean for troponin, CK-MB, Myoglobin



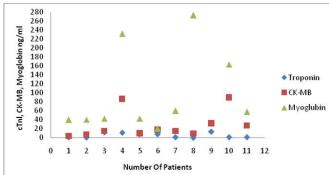


Figure1. Showed the distribution of eleven patients,which have positive results in troponin, CK-MB, and Myoglobin

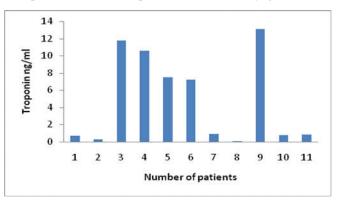


Figure 2. Showed the distribution of eleven patients with troponin values which have positive results

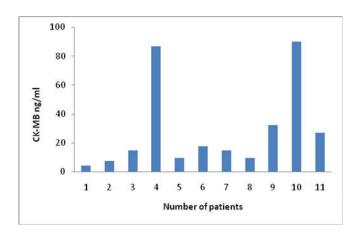


Figure 3. Showed the distribution of eleven patients with CK-MB values which have positive results

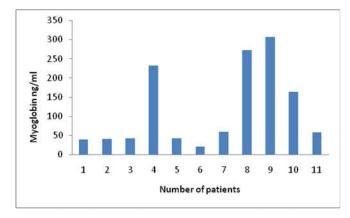


Figure 4. Showed the distribution of eleven patients with Myoglobin values which have positive results

DISCUSSION

The cardiac enzyme is a significant enzyme in the muscle of the heart. The Nano- Checker is testing the samples troponin, cardiac enzyme, and myoglobin. In this study was taken static analysis, among patients who suffering from chest pain in the province of Erbil- Iraq who is visiting the emergency hospital. The patients had a biochemical investigation of cardiac enzyme, the negative result indicates that had no problem with the heart muscle only may be stress caused a heart attack, but the positive result indicates that had myocardial infarction. In this study, we found that the patients had myocardial infarction, the elevation of creatine kinase within four-eight hours after that reaching to the normal range during twentyfour, thirty-six hours. In figure (2) showed that the troponin in the patients, which had positive results. Figure (3) and figure (4) showed positive values of CK-MB and myoglobin. This result indicated that patients may be suffering from heart failure or myocardial infarction. The sixty four patients had negative results only had chest pain. The negative result may be coming from stress. The standard deviation and mean were calculated. The results provided as the chest pain not indicated that patients suffering from myocardial infarction. The biochemical test of cardiac enzyme is a good monitor to check the heart muscle and prevent the patients from the risk of heart failure.

Conclusion

The study of biochemical investigations is crucial in medicine to diagnose the diseases. Specially cardiac enzyme is playing an important role to detect whether the heart muscle damaged or not. The men or women avoid myocardial infarction by doing physical activity. Also eating healthy food which lowering cholesterol and LDL. The aim of this study to show the patients who had myocardial infarction visiting the emergency hospital in Erbil- Iraq.

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REFERENCES

- Adam JE 3rd, Bordor GS, Davila-Roman VG, Delmez JA, Apple FS, Ladenson JH, Jaffe AS 1993 Cardiac troponin I. A marker with high specificity for cardiac injury. Circulation. 88(1): 101-106.
- Adams JE 3rd1, Sicard, G. A., Allen, B. T., Bridwell, K. H., Lenke, L. G., Dávila-Román, V. G., Bodor, G. S., ... Jaffe, A.Diagnosis of perioperative myocardial infarction with measurement of cardiac troponin I. S. (1994 March 10) New England Journal of Medicine, 330(10), 670–674.
- Alan, H. B., Wu, A. H. B., Apple, F. S., Gibler, W. B., Jesse, R. L., and Warshaw, M.1999. The National Academic of Clinical Biochemistry Medicine practice: Recommendation for the use of cardiac markers in coronary artery diseases.
- Antman, E. M., Tanasijevic, M. J., Thompson, B., Schactman, M., McCabe, C. H., Cannon, C. P., Fischer, G. A., Fung, A. Y., Thompson, C., Wybenga, D., Braunwald, E., ... Braunwald, E. 1996. Cardiac-Specific troponin I Levels to Predict the Risk of Mortality in Patients with Acute Coronary Syndromes. *New England Journal of Medicine*, 335(18), 1342–1349.
- Apple, F. S. 1992. Acute myocardial infraction and coronary reperfusion.Serum cardiac markers for the 1990s. *American Journal of Clinical Pathology*, 97(2), 217–226.
- Bodor, G. S., Porter, S., Landt, Y. and Ladenson, J. H. 1992. Development of monoclonal antibodies for an assay of cardiac troponin-I and preliminary results in suspected cases of myocardial infarction. *Clinical Chemistry*, 38(11), 2203–2214.
- Carole, M. 1995. SchneiderPhD*Carolyn Dennehy PhD*Susan JRodearme IMS*J.Reid Hayward MS. In Effects of Physical Activity on Creatine Phosphokinase and the Isoenzyme Creatine kinase–MB, 25(Issue 4), April (pp. 520–524).
- Carraro, P., Plebani, M., Varagnolo, M. C., Zaninotto, M., Rossetti, M., and Burlina, A. 1994. A new immunoassay for the measurement of myoglobin in serum. *Journal of Clinical Laboratory Analysis*, 8(Issue 2) 70–75.
- Clerica A, Emdin M,Del Chicca MG, Carpeggiani C, Zuccchelli GC, Boni C, DI Pasquale G, Pinelli G, 1993. Immunoradiometric assay of serum myosin as a marker of myocardial cell damage: methodological and clinical evaluation, Mar;37(1):33-37.
- Gulbis, B., Unger, P., Lenaers, A., Desmet, J. M., and Ooms, H. A. (1990 October). Mass concentration of creatine kinase MB isoenzyme and lactate dehydrogenase isoenzyme 1 in the diagnosis of perioperative myocardial infarction after coronary bypass surgery. *Clinical Chemistry*; 36(10), 1784–1788.
- J Mair, D Morandell, N Genser, P Lechleitner, F Dienstl, B Puschendorf. Equivalent early sensitivities of myoglobin,

creatine kinase MB mass, creatine **kinase** isoform ratios, and cardiac troponins I and T for acute myocardial infarction. Vol. 41, Issue 9 September 1995.

- Lott, J. A., and Landesman, P. W. 1984. Serum enzyme determinations in the diagnosis of acute myocardial infarction, 15(Issue 8), 706–716.
- Mair, J., Genser, N., Morandell, D., Maier, J., Mair, P., Lechleitner, P. Puschendorf, B. (1996 February 9). Cardiac troponin I in the diagnosis of myocardial injury and infarction. Clinica Chimica Acta; *International Journal of Clinical Chemistry*, 245(1), 19–38.
- Mehegan, J. P., and Tobacman, L. S. 1991 Jan. 15. Cooperative interactions between troponin molecules bound to the cardiac thin filament. *Journal of Biological Chemistry*, 266(2), 966–972.
- Montague, C., and Kircher, T. (October 1 1995). Myoglobin in the early evaluation of acute chest pain. *American Journal of Clinical Pathology*, 104(issue 4), 472–476.

- Neumeier D., Jockers-Wretou E. 1981. Tissue Specific and Subcellular Distribution of Creatine Kinase Isoenzymes. In: Lang H. (eds) Creatine Kinase Isoenzymes. Springer, Berlin, Heidelberg. pp 85-131.
- Panteghini, M., Pagani, F., and Bonetti, G. 1999. The sensitivity of cardiac markers an evidence based approach. *Clinical Chemistry and Laboratory Medicine*, 37(11–12), 11-12:1097-106.
- Ruppert, M., and Van Hee, R. (2001 September). The Creatinine-Kinase-MB determination in non-cardiac trauma: Its difference with cardiac infarction and its restricted use in trauma situations. *European Journal of Emergency Medicine: Official Journal of the European Society for Emergency Medicine*, 8(3), 177–179.
- Wu, A. H. 1997. Use of cardiac markers as assessed by outcomes analysis. Clinical Biochemistry Clinical Biochemistry., 30 (4), 339–350.
