Study of Some Physiological and Biochemical Aspects in The Serum of Myocardial Infarction Patients

Mohammed Qais Al-Ani Ali M. Sameen Ahmed S. Farhan Collage of Science – University of Al-Anbar

Accepted:2011/6/19, Received:22/11/2010

<u>Abstract</u>

Blood samples were collected from myocardial infarction patients and control groups in Ramady city / Anbar governorate – Iraq. Serum was used to know myocardial infarction effect on the lipid profile, three enzymes activity, proteins and urea . The results were showed the following: (a) Significant increment in cholesterol, triglyceride , LDL and VLDL, while we were observed significant decrement in HLD and urea. (b) Change in the electrophoretic bands of proteins by using polyacrylamide gel electrophoresis. (c) Significant increment in enzymes activity of CK , LDH , GOT and protein content in the myocardial infarction patients. (d) C-Reactive Protein was showed that 84% of MI patients gave positive results .

Introduction

Heart diseases will continue to be a very important cause of mortality, morbidity, and rising costs far into this new century (Horton,1999). Patients at increased risk of developing myocardial infarction (MI) include those with multiple coronary risk factors. Acute myocardial infarction (AMI) is considered the leading cause of human death in the world (Ismail *et al.*,2009). A dramatic increase in the incidence of AMI has been observed in many countries forming major cause of mortality in the middle aged and elderly.

Myocardial ischaemia results from the reduction of coronary flow to such an extent that supply of oxygen to the myocardium does not meet the oxygen demand of myocardial tissue. When this ischaemia is prolonged and irreversible then myocardial cell death and necrosis occurs which is defined as myocardial infarction (MI). The diagnosis of MI is based on clinical symptoms, electrocardiographic (ECG) changes and characteristic pattern of changes in some serum enzymes such as creatine (LDH) dehydrogenase kinase (CK), lactate and Aspartate aminotransferase (AST/GOT) (Alpert et al., 2000). Elevated plasma CRP levels detected in the first days of myocardial infarction may reflect an increased systemic inflammatory response, induced by myocardial necrosis (Anzai et al., 1997). Biochemical markers for disease should be

found in high concentrations in the target tissue only and should be limited in, or absent from, other locations. The marker should be released from the tissue rapidly after injury, ideally in direct proportion to the extent of tissue damage, and persist in the plasma long enough to provide a diagnostic interval for detection. The interval, however, should not be so long as to prevent detection of recurrent injury (Adams *et al.*, 1993).

Coronary artery disease remains the most common cause of death despite significant advancements in its prevention and treatment. Serum markers that are used for cholesterol risk assessment and management are total cholesterol, low density lipoprotein (LDL) and high-density lipoprotein (HDL) (Sacks *et al.*, 1996; Castelli, 1984).

The aim of the present study is to investigate the changes in serum levels of triglycerides, total cholesterol, HDL, LDL and of some enzymes activity as CK, LDH and GOT AMI patients. Moreover, we aimed at investigating the effects of AMI on total protein, protein patterns when using polyacrylamide gel electrophoresis, C-reactive protein and urea.

Methods

The study was conducted in CCU of Ramady general hospital during the period between November 2008 till April 2009. forty persons recruited into this study. Careful history was obtained and proper clinical examination was done. five milliliters of venous blood were collected in white tube, samples were centrifuged and stored at 4 C° for a period before estimation or frozen before assaying them.

Determination of lipid profile was done according to (Tietz, 1995), cholesterol according to(Zaltis *et al.*, 1953) Triglyceride according to (Bucolo & David, 1973). Assay of protein content was carried out according to Biuret method, GOT according to Reitman and Frankel, LDH and CK (Tietz, 1995). Urea done according to Nesslers method (Al-Aumary, 1990). Electrophoresis was done in Hsi slab gel electrophoresis unit connected to LKB50 power supply. The slab of polyacrylamide gels (7.5%) were prepared in the laboratory (Hames & Rickwood, 1984). The concentration of CRP is determined using latex agglutination test and it is quantitative estimation was done by a series of doubling dilution technique.

Results and Discussion

The results of the experiment on the total cholesterol, triglyceride concentration, HDL, LDL and VLDL in the patients and control groups are showed in fig.1. Significant increment in total cholesterol of MI patients compartment with control group. The patients groups mean total cholesterol was 197mg/dl, while the mean total cholesterol in control

group was 168mg/dl. Significant increment in triglyceride concentration of MI patients compartment with control group. The patients groups mean triglyceride concentration was 156mg/dl, while the mean triglyceride concentration in control group was 87mg/dl. Significant decrement in HDL of MI patients compartment with control group. The patients groups mean HDL was 39.1mg/dl, while the mean HDL in control group was 56mg/dl. Significant increment in LDL of MI patients compartment with control group. The patients groups mean LDL was 123.7mg/dl, while the mean LDL in control group was 95.4mg/dl. Significant increment in VLDL of MI patients compartment with control group. The patients groups mean VLDL was 33mg/dl, while the mean VLDL in control group was 17.3mg/dl. These results were in agreement with (Metwalli et al., 1998; Al-shamary, 2008) when they were confirmed that the cholesterol, triglyceride, LDL and VLDL increasing while HDL decreasing. Abnormal lipid profile conceders seriousness factor to ischemic heart disease (IHD) and atherosclerosis (Al-Aumary, 2007). Partially increment of cholesterol in MI patients may be genetically (Goldstein et al., 1973), or may because of they have great quantity of fat which leads to increasing cholesterol in the serum (Gracia et al., 1980). Increasing triglyceride level have hereditary foundation (Goldstein et al., 1973). Feed usage are no healthy increasing triglyceride level as world production from unsaturated fat (Kaplan et al., 2003). Increasing triglyceride level in MI patients because of increasing fatty acids and weaken in subtract VLDL from the plasma (Fredrickson. 1969). HDL decreasing ascribes to many mechanisms. It may because of decreasing in its production and secretion from the liver and intestine, or because of subtract it blood stream reasons to increased extravasations (Johansson et al., 1972). Increasing LDL level may be reasons to low subtract it or excess in the production it (Grundy et al., 1985; Crawfored et al., 2004) referred that increasing triglyceride lead to increase VLDL.

Fig.2: marks significant increment in CK activity of MI patients compartment with control group. The patients groups mean CK activity was 973 I.U/L, while the mean CK activity in control group was 66 I.U/L. Fig.3: marks significant increment in LDH activity of MI patients compartment with control group. The patients groups mean LDH activity was 984 I.U/L, while the mean LDH activity in control group was 102 I.U/L . Fig.4: marks significant increment in GOT activity of MI patients compartment with control group . The patients groups mean GOT activity was 86.2 I.U/L, while the mean GOT activity in control group was 10.75 I.U/L . These results were in agreement with (Al-Shamary, 2008; Al-Mahdawi, 2008 and Al-Aumary, 2007). The myocardium contains these enzymes greatly. Increasing these enzymes activity because of myocardium death and necrosis reasons the myocardial infarction. Therefore, releases great concentration of these enzymes to the blood (Crawfored, 2004).

Fig.5: indicates significant increment in total protein of MI patients compartment with control group. The patients groups mean total protein was 7.96g/dl, while the mean total protein in control group was 7.23g/dl. Fig.6: demonstrates the electrophoresis of protein, we observed prominent differences in the electrophoretic bands of proteins between MI patients and control groups. However, losing some protein bands and showing other newly in MI patients compartment with control groups and increase in the intensity other. These results disagreement with Olusi et al. (Olusi *et al.*, 1997; Al-Aumary 2007). They were found that total protein decreasing reasons albumin decreasing. Heat shock proteins (HSP) synthesis in the myocardium cells quickly through exposure the heart to damage as high temperature, also its synthesis with MI symptoms (Cleeg *et al.*, 1998).

Fig.7: shows no significant increment in urea concentration of MI patients compartment with control group. The patients groups mean urea concentration was 39.2mg/dl, while the mean urea concentration in control group was 32.1mg/dl. These results in agreement with Ismail et al. (Ismail *et al.*, 2009) that they were found no change in urea concentration in the rats with myocardial infarction.

Table 1– demonstrates distribution of CRP in the MI patients and control group. Twenty five MI patients were surrendered to this test, twenty one patients (84%) were given to positive results and four patients (16%) were given to negative results. All control group (15 persons) were given negative results. This result in agreement with (Al-Aumary, 2007and Shaya *et al.*, 2002). Increasing CRP in this study may be result to damage of myocardium or plaque rupture (Hon-Kan *et al.*, 2004) or inflammatory response after myocardium necrosis (Lagrand *et al.*, 1997).

References

- Adams, J. Abendschein, D. and Jaffe, A. (1993): Biochemical markers of myocardial injury: is MB creatine kinase the choice for the 1990s? *Circulation*. vol.88, no.2, pp.755–63.
- Al-Aumary, M. R. (1990): practical Clinical chemistry. The house of books for printing and Distribution . Al-Mosul university.
- Al-Aumary, S. F. H. (2007): Evaluation of the oxidation stress in myocardial infarction patients. PhD. University of Baghdad.
- Al-Mahdawi, Z. M. (2008): Study of levels of some enzymes in patients with cardiac diseases in Tikrit city and its Suburbs . *Tikrit J. of Pure Sci.* vol.13, pp.1-4.
- Alpert, J. Thygeson, K. and Antman, E. (2000): Myocardial infarction redefined a consensus document of the Joint European Society of Cardiology/American College of Cardiology Committee for redefinition of myocardial infarction. *J Am. Coll. Cardiol.* Vol.36, pp.959-69.
- Al-Shamary, O. S. (2008): Study of osmotic fraction to RBC and its relationship with numerous biochemical changes for patients with heart diseases. Thesis . Science collage. Tikrit university .
- Anzai, T. Yoshikawa, T. and shiraki, H. (1997): C-reactive protein as predictor of infarct expansion and cardiac rupture after a first Q wave acute myocardial infarction. *Circulation*. vol.96, pp.778-784.
- Bucolo, G. and David, H. (1973): Quantitative determination of serum triglycerides by the use of enzyme. *Clin. Chem.* Vol.19, pp.476-482.
- Castelli, W. (1984): Epidemiology of coronary heart disease: the Framingham Study. *Am. J. Med.* Vol.27, pp.4–12.
- Cleeg, J. S. Uhlinger, K. R. Jackson, S. A. Rifkin, Cherr, G. N. and Friedman, C. S. (1998): Induced thermotolerance and the heat shock protein-70 family in the pacific oyster *Crassoteria gigas . Mol. Mar. Biol. Biotechnol.* vol.7, pp.21-30.
- Crawfored, M. DiMarco, J. P. and Paulus, W. J. (2004): Cardiology. (2nd ed.). Mosby. Limited. Spain.
- Fredrickson, D. (1969): The role of lipids in acute myocardial infarction. *Circulation*. vol.39, pp.99-107.
- Goldstein, J. Hazzard , W. and Schrott , H. (1973): Hyperlipidemia in coronary heart disease. *J. Clin. Invest.* Vol.52, pp.1533.

• Gracia, R. Sorlie, P. and Costas, R. (1980): Relationship of dietary intake to subsequent coronary heart disease incidence. *Am. J. of Clin. Nutrition*. Vol.33, pp.1818.

• Grundy, S. Vega, G. and Kesaniemi, Y. (1985): Abnormalities in metabolism of LDL associated with coronary heart disease. *Acta. Medica. Scand.* Vol.701, 23p.

• Hames, B and Rickwood, D. (1984): Gel electrophoresis of proteins, apractical approach. (3rd ed.). IRL press limited. England.

• Hon-Kan, V. Chiung-Jen, W. U. Hsueh-Wen, C. Cheng-Hsu, Y. Kuo-Ho, Y. Sarah, C. and Morgan, F. V. (2004): Levels and values of serum high-sensitivity c-reactive protein within 6 hours after the onset of acute myocardial infarction. *Chest.* Vol.126, pp.1417-1422.

• Horton, R. (1999): Future of European cardiology: continentally isolated or globally integrated? *Lancet*. Vol.354, pp.791–3.

- Ismail, Z. B. Abu Abeeleh, M. Alzaben, K. R. Abu-Halaweh, S. A. Aloweidi, A. S. Al-Ammouri, I. A. Al-Essa, M. K. Jabaiti, S. K. Al-Smady, M. M. Al-Majali, A. (2009): Effect of experimental acute myocardial infarction on blood cell counts and plasma biochemical values in a nude rat model (Cri:NIH-Fox1^{RNU}). *Com. Clin. Pathol.*, vol. 6, pp.1-5.
- Ismail, Z. B. Abu Abeeleh, M. Alzaben, K. R. Abu-Halaweh, S. A. ALoweidi, A. S. AL-Ammouri, I. A. AL-Essa, M. K. Jabaiti, S. K. ALsmady, M. M. AL-Majali, A. (2009): Effect of experimental acute myocardial infarction on blood cell counts and plasma biochemical values in a nude rat model (Cri:NIH-Fox1^{RNU}). *Com. Clin. Pathol.* Vol.6, pp.1-5.
- Johansson, B. Kindmark, C. and Trell, E. (1972): Sequential changes of plasma proteins after myocardial infarction. *J. Clin. Lab. Invest.* Vol.29, 117p.

• Kaplan, L. Amudeo, J. P. and Steven, C. K. (2003): Clinical chemistry. (4th ed.). Mosby. USA.

- Lagrand, W. K. Niessen, H. W. Wolbink, J. Jaspars, L. H. Visser, C. A. and Verheugt, F. W. (1997): C-reactive Protein coloca lize with complement in human hearts during acute myocardial infarction. *Circulation*. Vol.95, pp.97-103.
- Metwalli, O. Al-Okbi, S. Motawi, T. El-Ahmady, O. Abdoul-Hafeez, S. and El-Said, E. (1998): Study of serum metals and lipids

profile in patients with acute myocardial infarction. J. of Islamic Aca. of Sci. vol.11, pp.5-12.

- Olusi, S. O. Parbha, K. and Sugathan, T. N. (1997): Annals of Saudi Medicine. vol.17, pp.124 125.
- Sacks, F. Pfeffer, M. and Moye, L. (1996): The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels: Cholesterol and Recurrent Events Trial investigators. *N. Engl. J. Med.* Vol.335, pp.1001–1009.
- Shaya, G. H. AL-Rawi, K. A. Dalli Ali, S. N. and Selman, J. A. (2002): The diagnostic and prognostic value of C-reactive Protein in patients with acute myocardial infarction in the CCU in a Saddam general hospital in Al-Ramadi city. *AL-Anbar Med. J.* vol.4, pp.59-63.
- Tietz, N.W. (1995): Clinical guide to laboratory Tests. W. B. Saunders Co. Philadelphia. pp.271-285.
- Zaltis, A. Zak, B. and Boyle, A., (1953): A new method for the direct determination of serum cholesterol . *J. Lab. Clin. Med.* Vol.41, pp.486-492.



Appendences













Fig. (4): GOT activity in MI patients and control groups



Fig. (5): serum total protein in MI patients and control groups



Co.1 Co.2 Co.3 MI1 MI2 MI3 MI4 MI5

Fig. (6): electrophoresis to serum protein in MI patients and control



Fig. (7): urea concentration in MI patients and control

	r	Total		Positive		Negative	
	No.	%	No.	%	No.	%	
MI	25	62.5	21	84	4	16	
Control	15	37.5	0	0	15	100	
Total	40	100	21	84	19	116	

Table (1): Distribution CRP in MI patients and control groups

دراسة بعض الجوانب الفسلجية الكيموحيوية في مصل مرضى احتشاء العضلة القلبية

محمد قيس العاني علي محمد سمين احمد سامي فرحان كلية العلوم جامعة الانبار تاريخ الاستلام:٢٠١٠/١١/٢٢ ، تاريخ القبول:٢٠١١/٦/١٩

الخلاصة

جمعت عينات الدم من مرضى احتشاء العضلة القلبية في مدينة الرمادي من محافظة الانبار – العراق. استخدم مصل الدم لمعرفة أثر احتشاء العضلة القلبية في البروتينات وفعالية ثلاث أنزيمات وكمية الدهون واليوريا. أظهرت نتائج الدراسة مايأتي: (أ) زيادة معنوية في فعالية أنزيمات XCD و GOT والمحتوى البروتيني لدى مرضى احتشاء العضلة القلبية. (ب) عند أجراء الترحيل الكهربائي على هلام متعدد الاكريل أمايد طرأت عدة تغيرات في الحزم الالكتروفوريتية للبروتينات. (ج) زيادة معنوية في تركيز كل من الكوليسترول والدهون الثلاثية والبروتينات الدهنية واطئة الكثافة والبروتينات الدهنية واطئة الكثافة جداً، في حين وجد أن هناك انخفاض معنوي في تركيز البروتينات الدهنية عالية الكثافة وتركيز اليوريا. (د) اظهر اختبار بروتين سي الفعال أن 84 % من المرضى أعطوا نتيجة ايجابية تجاه هذا الاختبار .