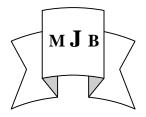
Some Hematological and Biochemical Changes in A Cute Myocardial Infarction (MI) in Babylon Government

Chafil Saihood Hassan Al-Shujiari Dept. of Physiology, College of Medicine, University of Babylon, Hilla, Iraq.



Abstract

This study was carried on thirty five subjects (18 patients, and 17 healthy subjects as controls). Their ages ranged from 40 - 60 years. The patients were diagnosed by specialist physicians by positive troponin I tests, typical chest pain and changes in ECG. This work was performed in coronary care unite (CCU) ward of Marjan Teaching Hospital, during period from March to September 2010. The Age, weight, height, WBC count, differential leukocyte count, ESR, Lipid profile laboratory analysis, Serum total protein, serum albumin, serum globulin, Serum copper, serum zinc and serum potassium ion level were measured in all subjects.

The results of present study revealed that WBC count, ESR, the differential leukocyte count (e.g. neutroplil cells) in patients increased significantly (P < 0.01) (P < 0.05) (P < 0.01) respectively comparison to controls. While, the differential leukocyte count of lymphocyte revealed to decrease significantly (P < 0.05) in patients. Serum triglyceride, serum total cholesterol, serum VLDL and LDL increased significantly (P < 0.05) (P < 0.05) (P < 0.05) (P < 0.05) respectively, while, serum HDL decreased significantly (P < 0.01) in patients comparison to controls. serum globulin, copper and potassium ion showed significant increase (P < 0. 0.01) (P < 0.05) (P < 0.01) respectively in patients comparison to controls. While, serum albumin, and serum zinc revealed significant decrease

(P < 0.05) (P < 0.01) respectively in patients comparison to controls.

ألخلاصه

شملت الدراسة خمسه وثلاثون شخصا (ثمانية عشر مريضا و سبعه عشر شخصا سليما) أعمارهم تراوحت ما بين 40 – 60 سنه. المرضى شخصوا من قبل أطباء اختصاص بواسطة الفحص الايجابي لتحليل التروبونين I ، الم الصدر النمونجي و التغيرات في التخطيط الكهربائي للقلب أنجز هذا البحث في ردهة الإنعاش في مستشفى مرجان التعليمي خلال الفترة من آذار إلى أيلول 2010. العمر، الطول ،عدد الكريات البيضاء ونسبها المئوية، سرعة ترسب الخلايا الدموية ، التحليل ألمختبري للصور الدهنية ، البروتينات في مصل الدم ، مستوى الزنك و النحاس و البوتاسبوم في مصل الدم قيست لجميع الأشخاص نتائج البحث أشارت إلى زيادة معنوية مقدارها P < 0.01 / P < 0.05 / P < 0.01 ليوالى في عدد الكريات البيضاء وسرعة ترسب الخلايا

الحمراء والخلايا البيضاء العدلية على التوالي لدى المرضى مقارنة بالأصحاء. بينما أشارت إلى نقص معنوي مقداره P < 0.05 في عدد الكريات البيضاء اللمفية لدى المرضى مقارنة بالأشخاص السليمين. مستوى الكولسترول الكلي، الشحوم الثلاثية، البروتينات الدهنية قليلة الكثافة جدا، و البرو تبنات الدهنية قليلة الكثافة از دادت بشكل معنوي مقدار ه 0.05 P < P < 0.05 P / P < 0.05 P / P حلي النو الي لدي المرضي مقارنة بالأشخاص السليمين. بينما مستوى البروتينات الدهنية عالية الكثافة أظهرت تناقصا معنويا مقدارة CP < 0.01). مستوى الكلوبولين ، النحاس و البوتاسيوم اظهر زيادة معنوية مقدارها O.O.1 / P < 0.0 / P < 0.0 / P = 0 التوالي لدى المرضى مقارنة بالأشخاص السليمين. بينما مستوى الالبومين و الزنك في مصل الدم اظهر تناقصا هاما مقداره P < 0.05 / P < 0.01 على التوالي لدى المرضي مقارنية بالأشيخاص الأصحاء

Introduction

yocardial infarction (MI) is almost always due to the formation of occlusive thrombus at the site of rupture or erosion of an athermanous plaque in a coronary artery. The leucocytosis is usual, reaching peak on the first day. The erythrocyte sedimentation rate (ESR) become raised and may remain so far several days. Echocardiography is a very useful technique for assessing left and right ventricular function and for detecting complication such as thrombus, cardiac ventricular septal defect, mitral regurgitation and pericardial effusion [1].

The chest pain is the most common symptom of acute myocardial infarction and is often described as a sensation of tightness, pressure, or squeezing. Pain radiate most often to the left arm, lower jaw, neck, right arm, back and epigastrium, where it may mimic heart burn. [2] Clinically, a myocardial infarction can be further subclassified into a ST elevation MI (STEMI) versus a non-ST elevation MI (non-STMI) based on ECG changes.[3]

Electrocardiography (ECG) may show acute changes with elevation in the ST segment and T wave inversion. Within 1 or 2 days of infarction deeping of Q wave occurs, ST and T wave change will disappear over time. The Q wave changes remain and can be used to detect a past infarction. Systemic signs of inflammation occur; including fever, increasing leukocytes and increasing erythrocyte sedimentation rate (ESR) begin about 24 hours after infarction and continue for up to 2 weeks [4]. The American College of cardiology and the European Society of Cardiology have redefined MI as a typical rise in cardiac troponin I with at least one of the following: development ischemic symptoms. of pathological Q waves on the ECG, ischemic ECG changes (ST depression or elevation) [1].

The important risk factors are previous cardiovascular disease, older age, tobacco smoking, high blood levels of certain lipids (triglycerides, low-density lipoprotein) and low level of high density lipoprotein (HDL), diabetes, high blood pressure, obesity, chronic kidney disease, heart failure, excessive alcohol consumption, and chronic high stress level. [5][6] Among the diagnostic tests available to detect heart muscle damage are an electrocardiogram (ECG), chest x-ray, and various blood tests. The most often used marker is the troponin levels. [7] Elevated white blood cell count play important role in the vascular injury and atherogenesis, the development of an atherosclerotic plaque rupture, and thrombosis. [8]

The zinc was recognized 46 years ago. Zinc deficiency resulting in growth retardation, hypogonadism, and immune dysfunction.[9] Zinc is typically the second most abundant after iron and it is the only metal which appears in all enzyme classes.[10] There is 2 - 4 gm of zinc distributed through out the human body.[11] Anxiety, stress, anger and other personality factors are implicated in the relationship of psychosocial issues to cardiovascular disease.[12] Smoking is associated with lowered antioxidant status in MI.[13] Low density lipoprotein (LDL) oxidation in the arterial intima plays a pivotal role in an atherogenesis. Under physiological conditions. several mechanisms protect LDL against oxidation, including hydrolysis of oxidation products high density lipoprotein (HDL) by associated enzymes. Some of these protective mechanisms are less effective under acute phase conditions. [14]

Aim of study

To detect the some hematological changes (ESR, WBC count, differential count,) and biochemical changes (lipid profile, serum protein, serum zinc, copper and potassium).

Materials and Methods

Subjects

The present study was conducted at cardiac care unit in Marjan Teaching Hospital / Hilla in cooperation with department of physiology/ Babylon College of Medicine during period from March 2010 to September 2010. The subjects were recruited from inpatient CCU ward. A history and physical examination were and laboratory obtained tests were performed in all subjects. The patients were diagnosed as acute myocardial infarction by positive rapid troponin I test with typical ischemic chest and with pain electrocardiograph evidence of ST segment elevation. The study was conducted on thirty five subjects (24 men and 11 women). They were divided into two groups. The group 1 included 18 patients, their mean ages were 53 ± 7.3 years (12 men and 6 women). The group 2 includes 17 control subjects (normal individuals) (12 men and 5 women), their mean ages 50 ± 5.6 years. They had no history of obesity, hypertension (HT), diabetes mellitus (DM), cardiac diseases, smoking or alcohol drinking.

Methods

Complete history (history of diabetes mellitus, family hypertension, history, smoking and previous attack of disease), symptoms and signs of disease, site of patients and ECG, echo, and x-ray were obtained. Five milliliter of venous blood was taken from all subjects after 12 - 14 hours for lipid profile, serum proteins, serum zinc, serum copper, serum potassium, WBC count, differential count, and erythrocyte sedimentation rate. The tests were done during first 3 days of attack. Body mass index (BMI) was calculated from weight and height of patients and controls in kilogram/meter2 [15].

WBC count and differential leucocyte count was mentioned in Dacie and Lewis practical hematology [16].

The method of lipid Profile laboratory analysis: After fasting 12 - 14 hours, a

venous fresh blood sample of 3 ml obtained, blood was centrifuged and collected serum was investigated for serum cholesterol, serum triglyceride, and serum HDL by direct method [17]. The VLDL and LDL were calculated by use friedewald formula: VLDL = triglyceride/5. Total cholesterol = HDL +LDL +VLDL. [17] Serum total protein and serum albumin were done according method mentioned by Berne and Lovy. [18] Serum copper was done according method mentioned by Ciuti. [19] Serum zinc was done according method mentioned by Tetsuo. [20] Serum potassium was done according method mentioned by Inouve. [21]

Statistical analysis

All data were expressed as mean \pm standard error (SE). The differences were assessed by paired student's test. A value of p < 0.05 was considered to be statistically significant [22].

Results

Effect of gender: There was no significant difference in data between male and female patients with comparisons. Therefore, the data of both sexes regarding the acute myocardial infarction were polled together and were considered as one group.

A- The history and physical examination:-

| Site | Number of Patients | Percentage |
|-------|--------------------|------------|
| Urban | 13 | 70 % |
| Rural | 5 | 30 % |
| Total | 18 | 100% |

| Table 1 The site of p | patients. |
|------------------------------|-----------|
|------------------------------|-----------|

<u>Table 2</u> The history of patients.

| Type of history | Diabetes mellitus | Hypertension | Previous attack | Family history | Smoking |
|-----------------|----------------------|--------------|-----------------|-------------------|---------|
| Number | 6 | 5 | 1 | 3 | 12 |

<u>**Table 3**</u> The signs and symptoms of patients.

| Symptoms | and | Pale | Sweating | Fever | Nausea | Vomiting | Syncope |
|----------|-----|------|----------|-------|--------|----------|---------|
| signs | | | | | | | |
| Number | | 18 | 13 | 12 | 15 | 11 | 8 |

| Type of parameter | Normal | Abnormal |
|-------------------|--------|----------|
| X-ray | 15 | 3 |
| Echo | 9 | 9 |

<u>Table 4</u> The type of parameters of x- ray and echo in patients

<u>**Table 5**</u> The site of MI by ECG changes in patients.

| Type of MI | Inferior MI | Anterolateral MI |
|------------|-------------|------------------|
| Number | 11 | 7 |

There is no significant difference (P > 0.05) between age of patients (53 \pm 7.3 years, n = 18) and age of controls (50 \pm 5.6 years, n = 17). Also there is no significant difference

(P > 0.05) between BMI of patients $(26 \pm 3 \text{ kg/m}^2, n = 18)$ and BMI of controls $(26.6 \pm 2.2 \text{ kg/m}^2, n = 17)$. See figure (6).

Table 6 The parameters of age and BMI.

| Parameter | Patient subjects | Control subjects | Significant |
|--------------------------------------|------------------|-------------------------|-------------|
| Age (years) | 53 ± 7.3 | 50 ± 5.6 | P > 0.05 |
| Body mass index (Kg/m ²) | 26 ± 3 | 26.6 ± 2.2 | P > 0.05 |

B- The hematological and biological changes:-

The ESR showed significant increase (P < 0.05) in patients group comparison to the controls, and serum copper (P < 0.05) as

well as serum potassium (P < 0.01) in patients when compare with healthy controls (table 7). While serum zinc showed decrease significantly (P < 0.01) in patients comparison to healthy controls.

<u>Table 7</u> The measured parameters of the groups under study; serum ESR, serum potassium, serum zinc and serum copper.

| Parameter | Patient subjects | Control subjects | Significant |
|--------------------------|------------------|-------------------------|-------------|
| ESR (mm/hr) | 31 ± 12.7 | 21.9 ± 5.3 | P < 0.05 |
| Serum potassium (mmol/L) | 4.1 ± 0.3 | 2.7 ± 0.37 | P < 0.01 |
| Serum zinc (mmol/L) | 86.6 ± 4.8 | 96 ± 9.6 | P < 0.01 |
| Serum copper (mmol/L) | 103 ± 8.6 | 97 ± 5 | P < 0.05 |

The total serum protein recorded no significant difference (P > 0.05) between patients and controls (table 8). While there is significant decrease (P < 0.05) between

serums albumin of patients in comparison with controls. Where as, the serum globulin showed significant increase (P < 0.01) in patients as compared with control (table 8).

Table 8 The measured parameters of serum proteins of groups study.

| Parameter | Patient subjects | Control subjects | Significant |
|-----------------------------|------------------|------------------|-------------|
| Serum Total protein (gm/dl) | 6.34 ± 0.4 | 6.4 ± 0.3 | P > 0.05 |
| Serum albumin (gm/dl) | 3.3 ± 0.4 | 3.7 ± 0.3 | P < 0.05 |
| Serum globulin (gm/dl) | 3.3 ± 0.5 | 2.6 ± 0.2 | P < 0.01 |

The serum of cholesterol, serum triglyceride, VLDL and LDL significant increase (P < 0.05) in patients in comparison

to controls. While serum HDL recorded significant decreases (P < 0.01) in patients in comparison to controls (table 9).

| Parameter | Patient subjects | Control subjects | Significant |
|----------------------------|------------------|------------------|-------------|
| Serum cholesterol (mg/dl) | 199 ± 16.6 | 177.5 ± 30 | 0.05 P < |
| Serum triglyceride (mg/dl) | 198 ± 89 | 141.6 ± 20 | P < 0.05 |
| Serum HDL (mg/dl) | 37 ± 7.7 | 51.7 ± 5.7 | P < 0.01 |
| Serum VLDL (mg/dl) | 40 ± 17.7 | 28.5 ± 5.8 | P < 0.05 |
| Serum LDL (mg/dl) | 124 ± 21.3 | ± 29 98.3 | P < 0.05 |

Table 9 The measured parameters of lipid profile of groups study.

There is significant increase (P < 0.01) in serum of WBC count and neutrophil (P < 0.01) in patients comparison to controls (table 10). Where as the lymphocytes showed significant decrease (P < 0.05) in patients when compared with controls (table 10).

Table 10 The measured parameters of WBC count and differential count in present study.

| Parameter | Patient subjects | Control subjects | Significant |
|-------------------------------------|------------------|-------------------------|-------------|
| WBC count 10 ³ cells/cmm | 9.5 ± 3.1 | 6.4 ± 0.3 | P < 0.01 |
| Neurophile % | $68 \% \pm 6\%$ | $60\% \pm 1\%$ | P < 0.01 |
| Lymphocyte % | 27% ± 5% | 32% ± 1% | P < 0.05 |

Discussion

In this study about 70% of patients lived in urban (table 1) due to life style and chronic stress. The smoking was the most risk factor in history of patients (67%) (table 2). Smoking increases the risk of mortality rate from the effect of coronary heart disease 1.4 to 2.4 fold (even light smoking) and in heavy smoker up to 3.5 fold. [23]. It is not clear how smoking promoter by nicotine, displacement of O2 in the Hb molecule by carbon monoxide, increased platelet endothelial adhesion. and raised permeability, induced by constituents in smoke.[23]

Patients were pale (100%) in table 3 due to stimulation of sympathetic nervous system, 82% of patients complained nausea due to cardiac pain. About 65% of patients had normal x-ray, 35% abnormal, while, half of them had normal echo. About 11 patients had inferior MI, while, seven had anterolateral MI.

Present study showed a significant increase in serum potassium. A little data

exist on the relationship between potassium level and infarct size after ST-segment in acute myocardial infarction. In patients with ST elevation MI, higher potassium levels are associated with large infarct size. [24] Serum copper showed a significant increase, while serum zinc decreased significantly. Copper increased significantly after the fifth days after the acute myocardial infarction. While zinc decrease significantly to control group from first day with the lowest value being found on the third day after attack. Copper correlate with concentration of both serum protein (albumin and globulin), while zinc bound to albumin only. [25]

This study also showed increase significantly in serum cholesterol, triglyceride, VLDL and LDL in patients comparison to controls, while, HDL decreased significantly. This result was in agreement with other previous studies. [26] [27]

The present study showed that ESR was increase significantly and revealed a significant increase in globulin and decrease significantly in albumin due to mediate inflammatory response to acute myocardial injury by numerous biochemical mechanisms.[28]

The data of the present study demonstrate that, WBC count and neutrophil increased significantly in patients comparison to controls, while lymphocytes decreased significantly. This study was in agreement with other studies. [29, 30, 31]

Conclusion

1- Smoking is most risk factor; pale and sweating are more common in MI.

2- WBC count, serum k ions, and serum globulin are significantly increased in patients comparison to controls.

3- Serum zinc, and serum HDL, are decrease significantly in patients comparison to controls.

References

1- Boon N.A, Colledge N.R, Walker B.R, and Hunter JA, A (2006). Davidson's principle and practice of medicine, 20th edition, volume 1 by Elsevier Company, page 591 – 594.

2- Marcus GM, Cohen J, Varosy PD, et al. (2007). The utility of gestures in patients with chest discomfort. Am. J. Med.; 120 (1): 83 – 89.

3- Moe KT, Wong P, (2010). Current trends in diagnostic biomarker of acute coronary syndrome. Ann. Acad. Med. Singap ; 39 (3): 210 - 215.

4- Corwin, Elizabeth J, (2008). Handbook of pathophysiology, 3rd edition, chapter 13, by Lippincett Williams and Wilkins, page 441.

5- Bax L, Algra A, Mali WP, Edinger M, Beutler JJ, Vander Graaf (2008). Renal function as a risk indicator for cardiovascular events in 3216 patients with manifest arterial disease. Atherosclerosis; 200 (1): 184.

6- Pearte CA, Furberg CD, O'Meara ES, et al. (2006). Characteristics and baseline clinical predictors of future fatal versus non fatal heart disease event in older adult: the cardiovascular health study. Circulation; 113 (18): 2177 – 2185. 7- Erhardt L, Herlitz J, Bossaert L, et al. (2002). Task force on the management of chest pain. Eur. Heart J.; 23 (15): 1133 – 1176.

8- Grzybowski M, Welch RD, Parsons L, Ndumele CE, ChenE, Zalenski R, Barron HV,(2004). The association between white blood cell count and acute myocardial infarction in hospital mortality: Findings from national registry of myocardial infarction. Academic Emergency Medicine; 11: 1049 – 1060.
9- Prasad AS, (2009). Impact of the discovery of human zinc deficiency on health. J Am Col Nutr; 28 (3): 257 – 265.

10- Broadly MR, White PJ, Hammond JP,Zelko I, and Lox A, (2007). Zinc in plants.Newphysiologist;173:677.

11- Rink L, Gabriel P, (2000). Zinc and the immune system. Pro Nutr Soc; 59: 541. 12- Cameron OG, (2007). Delirium, depression, other psycho-social and neurobehavioral issues in cardiovascular disease. Crit Care Clin; 23 (4): 881 – 900. 13- Kharb S, Singh GP, (2000). Effect of smoking on lipid profile, lipid peroxidation and antioxidant status in normal subjects and in patients during and after acute myocardial infarction. Clin Chim Acta; 302 (1 -2): 213 – 219.

14 Fainaru O, Fainaru M, Assal AR, Pinchuk I, and Lichtenbarg D, (2002). Acute myocardial infarction is associated with increased susceptibility of serum lipids to copper-induced peroxidation in vitro. Clin Cardio; 25 (2): 63 - 68.

15- Halls SB, (2008). Body mass index (BMI) calculator and weight comparison.). http://www.halls.md/body-mass-

index/av.htm

16- Lewis SM, Bain BJ, and Bates I, (2006). Dacie and Lewis practical haematology, tenth edition, Churchill Livingstone Elsevier Company.

17- Friedewald WT, Levy R, Fredrickso DS, (1972). Estimation of the concentration of low-density lipoprotein cholesterol in plasma without use of the preparative

ultracentrifuge: Clin CHem; 18: 499 – 502 (cited in: Cli. Chem. (1990); 36: 15 – 19).

18- Berne RM, Lovy M, (1983). Physiology .St. Louis; Cv. Mosby: 407 – 408.

19- Ciuti R, Galli A, Giorn IT, (1987). Determination of serum copper. Chim Clin; 12 (2): 101 – 111.

20- Tetsuo M. (1991). Determination of serum Zinc. Chimica Clinica Acta; 197: 209 - 220.

21- K. Inouye I., Ueno S. Yokoyama, and T. Sakaki J., (2002). Biochem; 131: 97 - 105.

22- Daniel WW, (2005). Biostatistics, eight edition, Wiley Company.

23- S. Silbernagl, F. Lang, (2006). Color Atlas of pathophysiology, international edition, page 220 – 238.

24- Roos M, Ndrepepa G, Baumann M, Pan CR, Heemann V, Lutz J, Keta D, SchutzS, Byme RA, Mehilli J, Schoming A, Kastrati A, (2009). Serum potassium on admission and infract size in patients with acute myocardial infarction. Clin Chim Acta; 409 (1-2): 46 – 51.

25- Gomez E, Del-Diego C, Orden I, Elosegui LM. Borque L, Escanero JF, (2000). Longitudinal study of serum copper and zinc level and their distribution in blood proteins after acute myocardial infarction. J Trace Elem Med Biol; 14 (2): 65 – 70.

26- Karthikeyan G and *et al*, (2009). Lipid profile and risk of a first myocardial

infarction among Asians analysis from the interheart study. J Am Coll Cardiol; 53 (3): 244 - 253.

27- Kumar A, Nagtilak S, Sivakanesan R, Gunasel Cera S, (2009). Cardiovascular risk factors in elderly normolipidemic acute myocardial infarct patients. Southeast Asia J Trop Med Public health; 40 (3): 581 – 592.

28- Tamhance UU, Aneja S, Montgomery D, Eva-Kline Rogers, Kim A, Eagle, (2008). Association between admission neutrophil to lymphocyte ratio and outcomes in patients with acute coronary syndrome. Am J Cardiol; 102: 653 – 657.

29- Grzybowski M, Welch RD, Parsons R, Ndumele CE, Chen E, Zalenski R, Barron HV, (2004). The association between white blood cell count and acute myocardial infarction in-hospital mortality: Findings mergency Medicine; 11: 1049 – 1060.

30- Stephen P. Thomson, Raymond J Gibbons, Peter A. Smars, Vera J. Suman and *et al*, (1995). Incremental value of the leukocyte differential and the rapid creatine kinase-MB isoenzyme for early diagnosis of myocardial. infarction. Annals of internal medicine; 122; 5: 335 - 341.

31- Al-Fatlawi M A-K, (2010). Leucocytes count and neutrophil/lymphocytes ratio in predicting in-hospital outcome after acute myocardial infarction. A thesis in medicine.