

## Some physiological changes in men and women after an acute myocardial infarction

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### الخلاصة

تضمنت هذه الدراسة معرفة التغييرات التي تحدث لبعض المعايير الدموية والكيميوحيوية لدى المرضى المصابين بمرض احتشاء القلب الحاد وذلك لأهميتها في مساعدة الفريق الطبي لمعرفة كيفية استجابة الجسم للعلاج المعطى مؤدياً إلى علاج أفضل. شملت الدراسة ثمانية وأربعون شخصاً مصاباً بمرض احتشاء القلب الحاد و من غير المصابين (السيطرة) من كلا الجنسين ، ست وعشرون (أربعة عشر ذكر و اثنتي عشر أنثى) مريضاً و اثني وعشرون (اثنتا عشر ذكر و عشرة أنثى) شخصاً سليم . تراوحت أعمارهم ما بين 40 – 60 سنة. تمت معاينة المرضى من قبل أطباء اختصاص بواسطة الفحص الايجابي لتحليل التروبونين I ، الم الصدر النموذجي و التغييرات في التخطيط . أنجز هذا البحث في ردهة الإنعاش في مستشفى مرجان التعليمي خلال الفترة من آذار إلى آب 2010. تميز الأشخاص المصابين بمرض احتشاء القلب الحاد بارتفاع معنوي (0.001) في عدد كريات الدم البيض وسرعة ترسيب كريات الدم الحمر لدى المرضى مقارنة بالأصحاء. أما بخصوص المتغيرات الكيميوحيوية أظهرت نتائج مستوى الكوليسترول الكلي، الشحوم الثلاثية، البروتينات الدهنية قليلة الكثافة زيادة معنوية (0.001) و البروتينات الدهنية قليلة الكثافة جداً (0.01) بينما مستوى البروتينات الدهنية عالية الكثافة أظهرت تناقصاً معنوياً (0.001) لدى المرضى مقارنة بالأصحاء. سجلت نتائج مستويات المغنيسيوم و الزنك انخفاضاً معنوياً (( 0.05 و 0.01 في الذكور، إما في الإناث كانت 0.01 ؛ 0.01 ، على التوالي) في حين أظهرت تراكيز كل من الكالسيوم ، ليوريا والكرياتينين لدى المرضى المصابين باحتشاء القلب الحاد ارتفاعاً معنوياً (0.001) (عدى في الإناث سجل الكالسيوم 0.01) مقارنة بالأصحاء.

### Abstract

This study was designed to assess some hematological and biochemical changes related to the acute myocardial infarction patients ,since the levels of these constituents are very important for the health care team to know how the body is responding to the different therapies that being provided and this will help the medical staff for proper management with less morbidity and mortality .

The study proceeded from March /2010 to August / 2010. There are 48 patients with acute myocardial infarction (AMI) and healthy subjects of both sexes.

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The patients were diagnosed by specialist physicians by using positive troponin I tests, typical chest pain and changes in ECG . A total number of 26 patients ; 14 males and 12 females and 22 of healthy control subjects ; 12 males and 10 females. The ages of subjects ranged between 40 to 60 years. Those patients were admitted to the cardiac care unit (CCU) in Marjan Teaching hospital in Hilla city. A history and physical examination were obtained and laboratory tests were performed in all subjects during first 3 days of attack .

Concerning the hematological parameters , it was found that white blood cells(WBCs) count and erythrocyte sedimentation rate (ESR) of male and female AMI patients showed significant increase( $P<0.001$ ) in comparison with healthy controls.

Regarding the biochemical parameters ,it was found significant increase( $P<0.001$ ) in total serum triglycerides, total cholesterol, LDL and VLDL ( $P<0.01$ ) in male and female AMI patients in comparison with healthy controls . While HDL significantly decrease( $P<0.001$ ) in both male and female patients in comparison with that of control. Serum magnesium and zinc concentration of both male and female AMI patients showed significant decrease(in male ,  $P<0.05$  ;  $P<0.01$  and in female ,  $P<0.01$ ;  $P<0.01$  , respectively) in comparison with control. While ,serum calcium, blood urea and serum creatinine in patients with AMI of both males and females are significantly increased ( $P<0.001$ ) (exception in calcium, female at  $P<0.01$  ) in comparison with the controls groups.

## Introduction

Acute myocardial infarction (AMI) ,commonly known as a heart attack is the interruption of blood supply to a part of the heart , causing heart cells to die. This is most commonly due to occlusion of a coronary artery following the rupture of a vulnerable atherosclerotic plaque , which is an unstable collection of lipids (fatty acid ) and white blood cells (especially macrophages) in the wall of an artery .This resulting ischemia and oxygen shortage<sup>(1)</sup>. AMI is one of the major causes of mortality and morbidity in the world <sup>(2)</sup> . Several risk factors for coronary heart disease have been well documented , including hypertension , hyperlipidemia , diabetes , a positive family story , smoking , obesity and inactivity<sup>(3)</sup>. There is evidence that antioxidants

can protect against free radical production, which is responsible for reperfusion-induced damage and lipid peroxidation, and may thereby inhibit thrombosis, myocardial damage and arrhythmias during AMI<sup>(4)</sup>. AMI is associated with an increased susceptibility of serum lipids to oxidation in vitro. This propensity for oxidation may reflect enhanced in vivo formation of free radicals and / or reduced efficiency of defense mechanisms. Both these possibilities may carry detrimental effects on the course, complications, and prognosis of the patients after acute myocardial infarction.<sup>(5)</sup> In ST-elevation myocardial infarction, a larger release of cardiac necrosis markers soon after reperfusion therapy relates to abnormal perfusion. Troponin appears as the most reliable necrosis marker for an early detection of cardiovascular magnetic resonance-derived abnormal microvascular reperfusion<sup>(6)</sup>. The diagnostic performance of sensitive cardiac troponin assays is excellent and these assays can substantially improve the early diagnosis of AMI, particularly in patients with a recent onset of chest pain<sup>(7; 8)</sup>. As well as early diagnosis of AMI in patients with chest pain is necessary to initiate appropriate treatment. Elevation of ST-segment in ECG is the only marker that cardiologists depend on in diagnosis<sup>(9)</sup>. Marginal intake of dietary zinc can be associated with increased risk of cardiovascular disease. These studies suggest that zinc nutrition can markedly modulate mechanisms of the pathology of inflammatory diseases such as atherosclerosis<sup>(10)</sup>. Studies in human and animals suggest that zinc has the potential to affect lipoprotein metabolism and hence impact cardiovascular disease risk<sup>(11)</sup>. Contractility of the heart and systemic vascular resistance are compromised at low ionized calcium levels. Combining beneficial cardiovascular and coagulation effects, the level for ionized calcium concentration should therefore be maintained above 0.9 millimol /Liter<sup>(12)</sup>. Blood urea nitrogen (BUN) is a simple clinical variable that provided useful prognostic information in patients admitted for decompensated heart failure. Elevated BUN levels probably reflect the cumulative effects of hemodynamic alterations that result in renal hypoperfusion<sup>(13; 14)</sup>. Renal insufficiency is common and is associated with higher risks for death in patients with a broad range of acute coronary syndromes<sup>(15)</sup>. 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<sup>(16)</sup>. Elevated white blood cell count play important role in the vascular injury and atherogenesis, the development of an atherosclerotic plaque rupture, and thrombosis<sup>(17)</sup>. Smoking is associated with lowered antioxidant status in MI<sup>(18)</sup>. 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 by high density lipoprotein (HDL) associated enzymes<sup>(5)</sup>. High prevalence of hypertriglyceridemia and low HDL in Pakistani AMI patients is suggestive that these two lipid abnormalities could be playing a major development of atherosclerosis in Pakistani population<sup>(19)</sup>. Active hospital –based programs to ensure routine LDL-C measurements in patients for acute myocardial infarction increased the use of appropriate lipid –lowering therapy in these individuals and could contribute to reducing the incidence of recurrent coronary artery diseases<sup>(20)</sup>.

### **Aim of the study**

This study aimed to estimate some hematological and biochemical changes in acute myocardial infarction patients in order to assist the medical staff for proper management with less morbidity and mortality. So this study was designed to determine the changes in the following :-  
(1):-To detect the some hematological changes (ESR and WBC count).  
(2):-Biochemical changes (lipid profile, serum magnesium, serum zinc, calcium, blood urea and serum creatinine).

### **Materials and Methods**

#### **The Subjects**

This study lasted from March /2010 to August / 2010. A total number used was 48 patients and healthy subjects of both sexes, males and females (clinically assessed by specialist doctor). The total number of patients was 26; 14 males and 12 females while the number of control subjects was 22; 12 males and 10 females. The ages of all subjects ranged between 40 to 60 years. Those patients were admitted to the cardiac care unit (CCU) in Marjan Teaching hospital in Hilla city. A history and physical examination were obtained and laboratory tests

were performed in all subjects during first 3 days of attack. The patients were diagnosed as acute myocardial infarction by positive rapid troponin I test with typical ischemic chest pain and with electrocardiograph evidence of ST segment elevation (clinically assessed by specialist doctor). The patients had history of diabetes mellitus, hypertension, previous attack, family history and smoking.

## **Methods**

### **(1):-Blood collection**

The collection of blood was performed in Marjan Teaching hospital in Hilla city during first 3 days of attack. The vein on the front of the elbow is almost employed. Five milliliter (ml) of venous blood was taken from all subjects before breakfast at 7 o'clock. The sera were separated from blood samples and frozen at  $-20^{\circ}\text{C}$  until used for biochemical analysis (lipid profile, serum zinc, serum magnesium, serum calcium, blood urea and serum creatinine). As well as, White blood cell count (WBC), and erythrocyte sedimentation rate (ESR) was analyzed<sup>(21)</sup>.

### **(2):-Hematological studies**

#### **(a):- Total white blood cells (WBCs) count**

Blood was diluted with Turk's solution. Blood is drawn in a clean and dry WBC pipette up to the mark 0.5 and outside of the pipette is wiped off with gauze. Then diluting fluid (Turk's solution) is drawn up to mark 11 (dilution 1:20) the contents are mixed for three minutes; the counting chamber a (Neubaur hemocytometer) was filled. It is waited for three minutes to let the cells for setting down and then the chamber is examined under 40X objective lens of the microscope to count WBCs in the four corners secondary squares<sup>(22)</sup>.

#### **(b):- Measurement of erythrocyte sedimentation rate(ESR)**

A Westergren tube: length- 300mm (open at both ends), diameter 2.5mm were used. one part of anticoagulant (3.8% trisodium citrate solution) was added to 4 parts of blood (0.5 ml of anticoagulant is added to 2 ml of blood). The mixture was drawn into a Westergren tube up to the zero mark and the tube was set upright in a stand with a spring clip

on the top and rubber at the bottom. The level of the top of the red cell column was read at the end of 1 hour<sup>(22)</sup>.

## **(2):- Biochemical studies**

### **(1):-Determination of lipid Profiles**

Total serum cholesterol, serum triglyceride, and serum HDL by direct method<sup>(23)</sup>. The VLDL and LDL were calculated by use friedewald formula:  $VLDL = \text{triglyceride}/5$ . Total cholesterol = HDL +LDL +VLDL<sup>(23)</sup>.

### **(2):-Determination of serum magnesium**

The colored calmagite – magnesium complex is formed immediately on mixing calmagite reagent with 50 micro liter( $\mu\text{L}$ ) of serum and is stable for over 30 minute . Serum magnesium is calculated by comparing the spectral absorbance of the sample at 520 nanometer(nm) with magnesium iodate calibrators( according by calmagite photometric method)<sup>(24)</sup>.

### **(3):- Determination of serum calcium**

Calcium in the sample, reacts with O-cresolphthaleine at alkaline pH, yields a red colored complex, which intensity is proportional to the calcium concentration. The intensity of the color was measured photometrically by using spectrophotometer at 570 nm wave length (according to procedure recommended by the Human company ,Germany)<sup>(25 ; 24)</sup>.

### **(4):- Determination of serum zinc**

Serum zinc react with chromogen present in the reagent forming a colored compound , which color intensity that is proportional to the zinc concentration present in the sample by using spectrophotometer at 578nm and the color is stable for 20-30 minutes (Zinc kit L-T-A Italy )<sup>(26)</sup>.

### **(5):- Determination of blood urea**

In the first step of preparation of urea :1ml of reagent 1 urease was added to 0.01 ml of serum and let for 5 minutes to complete reaction, the second step added 0.2 ml of reagent phosphate buffer sodium salicylate, sodium nitroprusside and EDTA to the mixture. In an alkaline median ,the ammonium ions react with salicylate and hypochlorite to form a green colored indophenols (2,2-dicarboxylindophenol) and the reaction is catalyzed by sodium nitroprussid. Then put in incubator or water path

for 10 minute at 37 C°, and read at 580 nm by using spectrophotometer , according to the procedure recommended by the urea kit from the Biomerieux company , France <sup>(27)</sup>.

#### **(6):- Determination of serum creatinine**

The serum creatinine is prepared by adding 0.5 ml of trichloro acetic acid to 0.5 ml serum and centrifugation for 5 minutes at 3000 rpm , take 0.5 ml of the precipitant that forms after centrifugation and adding to 0.25 of NaOH and 0.25 ml of picric acid and let for heating to about 20 minutes , at 25 C° . After reading at 520 nm by using spectrophotometer and the results multiply by the factor (4.9) ,according to the procedure recommended by the company Randox, united kingdom <sup>(28 ; 24)</sup>.

#### **Statistical Analysis**

SPSS program was used in this study. All values were expressed as mean ± standard error (SE). Independent t-test was used to estimate differences between groups. The differences were considered significant when the probability (P) was less than 0.05 (P<0.05) and highly significant when the probability (P) was less than 0.001 (P<0.001) <sup>(29)</sup>.

### **Results**

#### **Hematological studies**

##### **(1):-Total white blood cells (WBCs) count**

The results of WBCs count in male and female patients with acute myocardial infarction (AMI) and control are presented in table 1 .There were significantly (P<0.001) increase in AMI patients in comparison with healthy control in both male and female.

##### **(2):-Erythrocyte sedimentation rate(ESR):-**

The values of ESR in male and female patients with acute myocardial infarction are illustrated in table 1 . The mean and stander error of ESR for males and females of AMI patients were significantly (P<0.001) increase in comparison with healthy control in both male and female.

**Table 1 :-The changes in white blood cell count (WBC) and erythrocyte sedimentation rate (ESR) of males and females for controls and patients after acute myocardial infarction .**

Parameter	Male		Female	
	Control	Patients	Control	Patients
WBC × 10 <sup>9</sup> / L	5.025 ± 0.536	9.888 ± 0.696 ***	5.466 ± 0.325	11.542 ± 0.771 ***
ESR mm / hr	9.925 ± 0.704	25.857 ± 3.097 ***	10.312 ± 0.922	29.833 ± 2.414 ***

-Values represent mean ± SE .

- \*\*\* are significant at P > 0.001 relative to control group.

### Biochemical studies

#### (1):-Lipid Profiles

It include serum triglyceride (TG), cholesterol , LDL, HDL and VLDL in male and female patients with AMI in comparison with control as shown in table 2 .

The mean and stander error of TG, cholesterol , LDL for males and females of AMI patients were significantly (P<0.001) increase (VLDL increase at P<0.01) in comparison with healthy control in both male and female . While , the values of HDL in male and female patients recorded significant (P<0.001) decrease relative to the male and female control.

**Table 2 :-The changes in serum Triglyceride (TG) , Cholesterol , Low density lipoprotein (LDL),High density lipoprotein(HDL) and Very Low density lipoprotein(VLDL) of males and females for controls and patients after acute myocardial infarction .**

Parameter	Male		Female	
	Control	Patients	Control	Patients
Triglyceride mg/dl	146.166 ± 3.644	205.454 ± 16.039 ***	150.90 ± 3.981	203.454 ± 6.689 ***
Cholesterol mg/dL	171.541 ± 5.338	201.254 ± 3.491 ***	173.740 ± 6.479	205.90 ± 4.097 ***
LDL mg/dL	97.444 ± 5.230	124.554 ± 2.985 ***	98.511 ± 5.107	125.250 ± 1.780 ***
HDL mg/dL	49.240 ± 1.550	36.550 ± 1.523 ***	49.20 ± 1.871	34.70 ± 1.970 ***
VLDL mg/dL	29.250 ± 0.833	40.054 ± 3.037 **	30.150 ± 1.093	43.622 ± 3.668 **

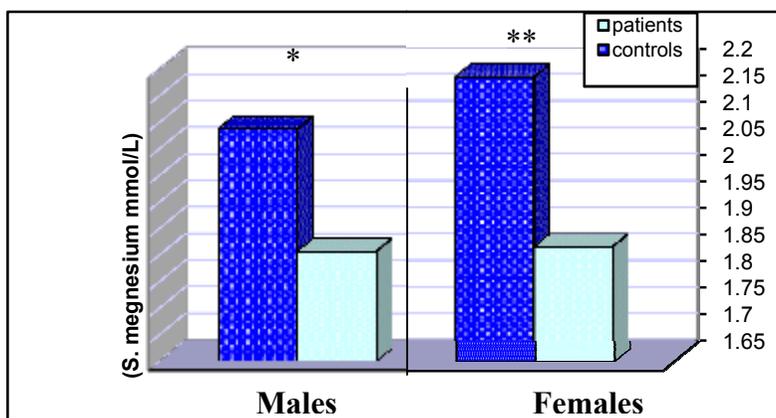
-Values represent mean ± SE .

- \*\* are insignificant at P < 0.01 relative to control group.

- \*\*\* are significant at P < 0.001 relative to control group.

## (2):-Serum magnesium

The values of serum magnesium of acute myocardial infarction patients and control are illustrated in figure 1 .The mean and stander error of serum magnesium for males and females of patients with AMI were :  $1.857 \pm 0.029$  and  $1.866 \pm 0.033$  millimole / Litter (mmol / L), respectively) and were significantly ( $P < 0.05$ ) lower than control males ( $2.0875 \pm 0.076$  mmol / L )and significantly lower at ( $P < 0.01$ ) than control females ( $2.185 \pm 0.079$  mmol / L).



**Figure 1:-Changes in serum magnesium concentration of males and females for controls and patients after acute myocardial infarction.**

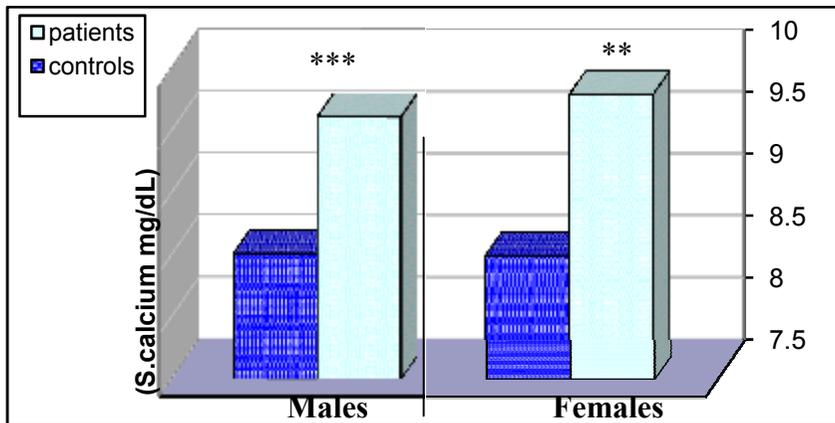
-Values represent mean  $\pm$  SE .

- \* are significant at  $P < 0.05$  relative to control group.

- \*\* are insignificant at  $P < 0.01$  relative to control group.

## (3):-Serum calcium

Results of serum calcium of both males and females affected with AMI are illustrated in figure 2. Values of serum calcium in males and females patients were ( $9.611 \pm 0.211$  and  $9.800 \pm 0.427$  milligram / deciliter (mg/dL), respectively) and were significantly ( $P < 0.001$ ) higher than males and at ( $P < 0.01$ ) higher than females controls ( $8.514 \pm 0.229$  and  $8.500 \pm 0.057$  mg/dL, respectively).



**Figure 2:-Changes in serum calcium concentration of males and females for controls and patients after acute myocardial infarction.**

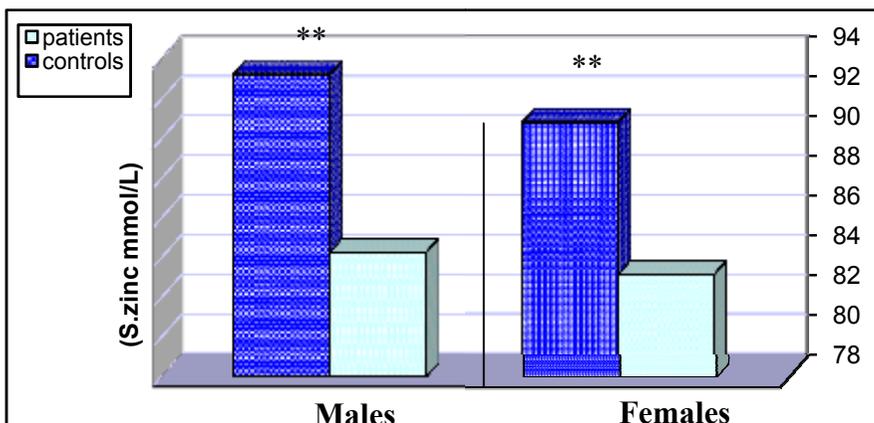
-Values represent mean ± SE

- \*\* are significant at P < 0.01 relative to control group.

- \*\*\* are insignificant at P < 0.001 relative to control group.

**(4):-Serum zinc**

As shown in the figure 3 The mean and stander error of serum zinc for male and female patients with AMI are : 84.285 ± 0.746 and 83.166 ± 0.542 millimole / Litter (mmol / L) respectively. Both of their means shows significant ( P< 0.01 ) decrease in comparison with both male and female healthy control( 93.250 ± 2.833 and 90.833 ± 1.661 mmol / L respectively ).



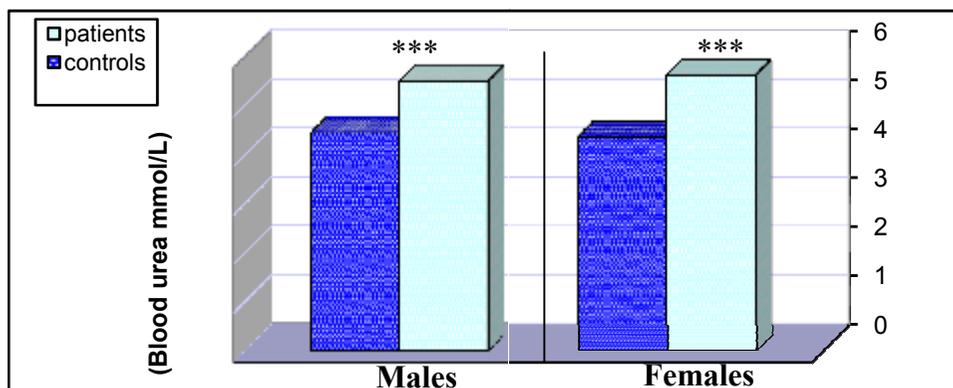
**Figure 3:-Changes in serum zinc concentration of males and females forcontrols and patients after acute myocardial infarction.**

-Values represent mean ± SE .

- \*\* are significant at P < 0.01 relative to control group.

### (5):-Blood urea

There was significant increase ( $P < 0.001$ ) in blood urea concentration in male and female patients with AMI ( $5.507 \pm 0.198$  and  $5.630 \pm 0.244$  mmol /L respectively) relative to the male and female control ( $4.454 \pm 0.132$  and  $4.371 \pm 0.108$  mmol /L respectively)( Figure 4).



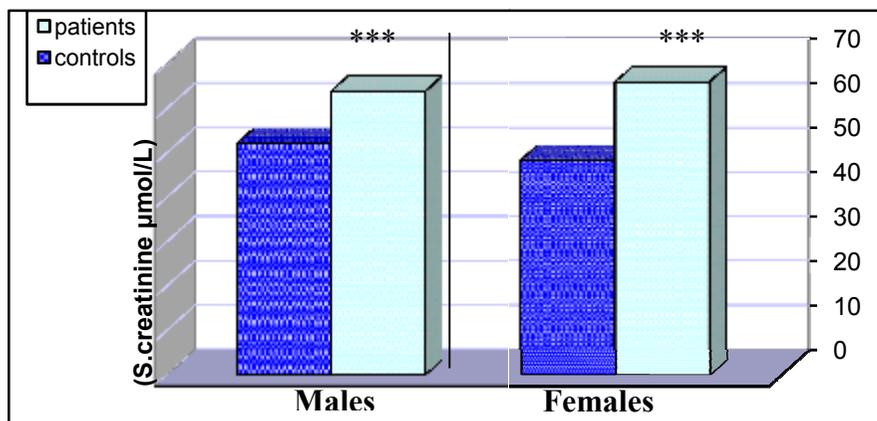
**Figure 4:-Changes in blood urea concentration of males and females for controls and patients after acute myocardial infarction.**

-Values represent mean  $\pm$  SE .

- \*\*\* are significant at  $P < 0.001$  relative to control group.

### (6):-Serum creatinine

A comparison between mean and stander error of serum creatinine concentration in both male and female patients with AMI ( $63.909 \pm 2.121$  and  $66.0 \pm 2.679$  micromole / Litter ( $\mu\text{mol} / \text{L}$ ) respectively) with the male and female control( $52.181 \pm 1.901$  and  $48.357 \pm 3.371$   $\mu\text{mol} / \text{L}$  respectively ) showed that there was significant increase( $P < 0.001$ ) in both male and female patients as compared with both sex control respectively(Figure 5 ).



**Figure 5:-Changes in serum creatinine concentration of males and females for controls and patients after acute myocardial infarction.**

-Values represent mean ± SE .

- \*\*\* are significant at P < 0.001 relative to control group.

### Discussion

This study is aimed has been undertaken, since the levels of the studied substances are very important in letting the health care team to know how the body is responding to the different therapies that being provided .

Concerning total white blood cells (WBCs) count and erythrocyte sedimentation rate(ESR) in acute myocardial infarction(AMI) in both males and females patients which are illustrated in table(1), they showed significant increase in AMI males and females when compared with control groups. The leucocytosis is usual, reaching peak on the first day. The erythrocyte sedimentation rate (ESR) become raised and may remain so far several days<sup>(30)</sup> . Elevated white blood cell count play important role in the vascular injury and atherogenesis, the development of an atherosclerotic plaque rupture, and thrombosis<sup>(17)</sup>. Patients with elevated WBCs count have been shown to have a higher risk of developing an MI and to be at higher risk for adverse events during the acute setting<sup>(31)</sup>. Leukocytosis , which are consistent with a significant role of inflammation in acute phase of MI<sup>(32)</sup>. As well as elevated leukocytes and neutrophil counts after primary percutaneous coronary intervention in patients with ST-segment elevation MI are directly related to myocardial infarct size and the left ventricular ejection

fraction<sup>(33;34)</sup>. This results of leukocytosis was in agreement with other studies<sup>(35;36)</sup>.

The present study showed that ESR was increase significantly may be due to mediate inflammatory response to acute myocardial injury by some biochemical mechanisms<sup>(37)</sup>. Also, increased ESR in patients with AMI occurs via significantly increased RBCs aggregation compared to control<sup>(38)</sup>. This results are consistent with Timmer *et al.*,<sup>(39)</sup>.

The values of serum triglycerides, total cholesterol, LDL and VLDL of male and female AMI patients show significant increase in comparison with healthy controls. While HDL significantly decrease in both male and female patients incomparison than control (table2). These results agree with the results of other studies<sup>(40;41;42;43;44;45)</sup> in that total cholesterol(TC), triglyceride (TG) low –density lipoprotein (LDL),very low –density lipoprotein (VLDL) levels were found significantly high but HDL recorded significantly decreased in patients with AMI as compared to healthy subjects. Changes in the concentration of plasma lipids including cholesterol are complications frequently observed in patients with MI and certainly contribute to the development of vascular disease. However, there is a difference in opinion on the changes that occur in serum lipids and lipoproteins following MI. Couple of recent studies reported contrast finding, one with no significant change in serum total lipid and total cholesterol levels<sup>(46)</sup>. Cholesterol has been singled out as the primary factor in the development of atherosclerosis. While, HDL is regarded as one of the most important protective factors against arteriosclerosis. Since HDL's protective function has been attributed to its active participation in the reverse transport of cholesterol. Numerous cohort studies and clinical trials have confirmed the association between a low HDL and an increased risk of coronary heart disease<sup>(47;48)</sup>. As well as hyperlipidemia has been proven to be an important modifiable risk factor for AMI, but many MI occur in patients with normal lipid values, who otherwise appear to be at low clinical risk<sup>(49)</sup>. Wiesbauer *et al.*,<sup>(50)</sup> found that the familial-combined hyperlipidaemia increases the risk for MI in individuals below 60 years. Karthikeyan *et al.*,<sup>(48)</sup> pointed out that the risk of AMI associated with increases in LDL and decrease in HDL.

The results of serum magnesium of both male and female AMI patients show significant decrease in comparison with control (Figure 1). This study also consistent with Ahmed *et al.*,<sup>(51)</sup> who pointed that at the time of admission, serum magnesium levels in patients with AMI were highly significantly lower as compared with control. As well as, in patients with AMI in both men and women the plasma magnesium and plasma zinc increased significantly between day one and day 12<sup>(52)</sup>. As well as these results agree with the results of other studies<sup>(53; 54; 55;56)</sup> in that serum magnesium were significant lower in patients with AMI than healthy control.

The means of serum calcium for males and females of AMI patients show significant increase in comparison with control groups (Figure 2). This result agrees with Ahmed and Nasser<sup>(53)</sup> who showed significant elevation in serum calcium of AMI patients within 72 hours in comparison than control. While other study recorded significant decrease in level of serum calcium in patients with MI when compared with control group<sup>(57)</sup>.

The means of serum zinc in patients with AMI of both males and females are significantly decreased in comparison with the controls groups (Figure 3). This results is in agreement with Gomeze *et al.*,<sup>(58)</sup> who pointed out that serum zinc decreased significantly with respect to control from the first day on, with the lowest values being found on the third day after the acute myocardial infarction. Low zinc concentration can be associated with an increased risk of cardiovascular diseases. Since, mice fed the zinc –deficient diet had significantly increased concentrations of cholesterol and triglycerides, zinc supplementation decreased these lipid variables compared with control mice<sup>(59)</sup>. Which mice have a lipoprotein profile similar to that of humans<sup>(60)</sup>. Also, low zinc concentration is probably that zinc might not have a role in pathogenesis in coronary artery disease. However, low serum zinc level has been related to excess release of steroids due to release factor called leukocyte endogenous mediator by polymorphonuclear cells. Stimuli that cause release of this factor include infections as well as tissue injury such as myocardial infarction, when this factor is released, amino acid flow to the liver increases, the amount of zinc in the serum decrease<sup>(61)</sup>. As well as, this results of serum zinc is consistent with other studies<sup>(62)</sup>;

<sup>63 ; 54</sup>). While, These results are in disagreement with Zainal et al ., <sup>(57)</sup> who found significantly increase in levels of zinc in patients with MI when compared with control group.

The results of blood urea and serum creatinine of both male and female AMI patients are significantly increase in comparison with control (Figure 4 and Figure 5 respectively ). This study agrees with Aroson *et al.*, <sup>(64)</sup> , who pointed out that the significant increased of blood urea (BUN) and BUN / creatinine ratio on admission are independent predictors of long –term mortality in patients with ST-elevation myocardial infarction (STEM). Since, Renal dysfunction is associated with increased mortality in acute coronary syndromes and other cardiovascular disease .The prognostic value of kidney dysfunction has been investigated using creatinine-based measures of renal function. Few data are available on the prognostic significance of blood BUN , a sensitive marker of hemodynamic alteration and renal perfusion. As well as Saygitov *et al.* , <sup>(65)</sup> stated that the results of both blood urea and serum creatinine significantly increase in AMI patients and an increased level of BUN is a more significant risk factor for acute coronary syndromes(AMI) outcomes than that of creatinine .The result of serum creatinine is in agreement with the result of Zhao *et al.* , <sup>(66)</sup> they found that there were significant increase in serum creatinine MI patients than in the normal group. And this elevated admission serum creatinine levels are associated with impaired myocardial flow and poor prognosis in STEMI patients .The elevated blood urea nitrogen would be associated with adverse outcomes independent of serum creatinine based estimates of kidney function in patients with acute coronary syndromes<sup>(67)</sup>.The results of other study indicate that a rise in plasma urea level is common if not universal after myocardial infarction and is caused by either a fall in the glomerular filtration rate or an increased urea production rather than a mixture of the two<sup>(68)</sup> .

## Conclusions

1-Acute myocardial infarction is associated with an increased lipid profile (Serum cholesterol, triglyceride, LDL, VLDL and decrease in HDL).

2- WBC count and ESR are significantly increased in patients which may be associated with inflammation that occurs in acute myocardial infarction.

3- Serum zinc, and serum magnesium showed significant decrease and increased in serum calcium, blood urea and serum creatinine that related with an increased risk of cardiovascular diseases. As well as. Renal dysfunction is associated with increased mortality in acute coronary syndromes

## References

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