Wasit Journal for Pure Science

Journal Homepage: https://wjps.uowasit.edu.iq/index.php/wjps/index

e-ISSN: 2790-5241 p-ISSN: 2790-5233

Evaluating of Some Heart and Liver Enzymes in Patients with Myocardial Infarction in Wasit

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DOI: https://doi.org/ 10.31185/wjps.253

Received 20 October 2023; Accepted 25 November 2023; Available online 30 December 2023

ABSTRACT:

The aim of the study was to evaluate some heart and liver enzymes in patients with myocardial infarction in Wasit Province. Myocardial infarction (MI) is a major cause of death and disability worldwide a heart attack, occurs when blood flow to a section of the heart is cut off, resulting in the death of heart cells. This usually happens when a coronary artery becomes blocked due to the rupture of a vulnerable plaque made up of cholesterol, fatty acids, and white blood cells.

The study took place in the cardiac resuscitation unit at Al-Zahra Teaching Hospital and Al-Karama Teaching Hospital in Wasit Governorate from November 2022 to January 2023. Its objective was to examine the significance of physiological, immunological indicators, and inflammatory biomarkers for accurately diagnosing, assessing risk, and predicting the future outcomes of patients with acute myocardial infarction.

The study included totally 150 samples, 100 patients and 50 as control and they were divided into three age groups. The first group is 30-45 years old; the second group is 46-60 years old, and the third group is 61-75 years old. Taking into account the risk factors for acute myocardial infarction, which include Sex, age, high blood pressure, diabetes, smoking, and dyslexia. Lipid lipids through a questionnaire.

The results of our study with regard to the diagnostic signs of myocardial infarction revealed a significant increase in troponin, myoglobin, and Heart- Fatty Acid Binding Protein, and a significant decrease in Creatine kinase. This study also indicated, regarding the results of liver enzymes, a significant increase in both Aspartate aminotransferase and Alanine transaminase at $p \leq 0.5$.

Conclusions: In patients with Acute myocardial infarction, The plasma level of significant increase in troponin, myoglobin, and Heart- Fatty Acid Binding Protein, and a significant decrease in Creatine kinase AST and ALT at $p \leq 0.5$.

Key Words: Acute myocardial infarction, troponin, myoglobin, Creatine kinase.

1. INTRODUCTION

Cardiovascular disease is the primary reason for global deaths, with approximately 16.7 million fatalities occurring annually (1). Myocardial infarction, commonly known as a heart attack, is a significant contributor to these deaths and disabilities on a global scale. It happens when the blood flow to a section of the heart is disrupted, causing the death of heart cells. This disruption is usually caused by a blockage in a coronary artery, resulting from the rupture of a vulnerable plaque made up of unstable accumulations of lipids (such as cholesterol and fatty acids) and white blood cells (specifically macrophages) present in the artery wall. (2).

The accumulation of plaque is a process and also can produce chest pain symptom known as angina pectoris. A myocardial infarction occurs when a plaque rupture suddenly and it causes a rapid accumulation of clotting factors at the rupture site which leads a sudden obstruction of blood flow in the coronary artery. Sudden obstruction prevents blood reaching the heart muscle. The heart muscles start to die if there is no vital supply of oxygen-rich blood. The longer the obstruction persists, the greater the amount of heart muscle dies (3) .

Aim of the study of some biochemical and immunological parameters and their relationship to myocardial infarction.

2. Material and Methods:

2.1 Study design:

The present work included a case control study for a group of (150) samples: (100) patient samples, (50) control samples. The study was conducted from care unit at AL-Zahraa teaching hospital and AL-Karama Teaching Hospital from" November 2022 - February 2023", with age ranged between (30 to 75 Year). The study included 100 patients with acute myocardial infarction, including 71 men aged 30-70 years, and 29 women aged 33-75 years. And 50 samples from the control, 28 samples from men and 22 samples from women, and they were divided into three age groups.

The first group is 30-45 years old; the second group is 46-60 years old, and the third group is 61-75 years old. Patients presented with chest pain, Electrocardiograph and Echo findings of myocardial ischemia. Risk factor for MI were evaluated (Age, Sex, Hypertension, Diabetes Mellitus, Smoking and hyperlipidemia). Biomarker of myocardial ischemia Troponin, Creatine Kinase and Myoglobin and Aspartate aminotransferase and Alanine transaminase.

2.2 Blood sample:

6 ml of venous blood samples were withdrawn in a tube containing gel and clot activator for check the level of Ast ,Alt, Total Protein , Albumin , troponin, CK , Myoglobin

Determination liver function (AST, ALT):

These parameters are measured by Abbott Diagnostic device in the clinical chemistry laboratory unit at Al-Zahraa Teaching Hospital

Determination of troponin, Creatine kinase, Myoglobin H- FABP ELISA Kit

The Sandwich-ELISA principle is utilized in this ELISA kit.

Statistical Analysis The data were analyzed statistically using SPSS vet.25, and the averages were compared using Chi-square under a probability level of 0.05.

3. RESULTS AND DISCUSSION

Table 1: Explains the Effect of Gender and age Group on the liver Enzyme Troponin

Sex	Case	Age	Troponin	p-value
Male	**Patient**	**30-45**	0.042	0.03
		46-60	0.038	
		61-75	0.034	
	Control	**30-45**	0.019	
		46-60	0.021	
		61-75	0.024	
Female	**Patient**	**30-45**	0.035	0.03
		46-60	0.040	
		61-75	0.033	
	Control	**30-45**	0.018	
		46-60	0.022	
		61-75	0.018	

The results of the statistical analysis in Table 1 showed that there were significant differences between the age groups for males, where the highest rate of Troponin was recorded for affected people in the age group (30-45), where it was recorded (0.042), compared to control people, where it was recorded (0.019) . The age group (46-60) also recorded the second highest rate, reaching (0.038), compared to control people, where it reached (0.021) . While the lowest rate was observed in the age group (61-75) for infected males, where it reached (0.034), compared to control people, where it reached (0.024).

The results of the statistical analysis in Table 3.1 also showed that there were significant differences between the age groups for females, where the highest rate of Troponin was recorded for affected people in the age group (46-60), where it was recorded (0.040), compared to control people, where it was recorded (0.022) The age group (30-45) also recorded the second highest rate, reaching (0.035), compared to control people, where it reached. (0.018) While the lowest rate was observed in the age group (61-75) for infected females, where it reached (0.033), compared to control people, where it reached (0.018).

Table 2 : Explains the Effect of Gender and age Group on the CK

Sex	Case	Age	CK	p-value
Male	**Patient**	**30-45**	5.95	0.07
		46-60	5.62	
		61-75	5.34	
	Control	**30-45**	4.67	
		46-60	5.12	
		61-75	5.73	
Female	**Patient**	**30-45**	5.80	0.08
		46-60	4.88	
		61-75	4.90	
	Control	**30-45**	4.20	
		46-60	4.31	
		61-75	4.80	

The results of the statistical analysis in Table 2 showed that there were no significant differences between male age groups and between infected and control people, where the average CK for infected people in the age group (30-45) was recorded (5.95) compared to control people, where it was recorded (4.67) The age group (46-60) also recorded (5.62) compared to control people, which reached (5.12) . While the lowest rate was observed in the age group (61-75) for infected males, where it reached (5.34), compared to control people, where it reached (5.73).

The results of the statistical analysis in Table 3.2 also showed that there were no significant differences between the age groups for females, as the rate of CK for infected people was recorded in the age group (30-45), where it was recorded (5.80), compared to control people, where it was recorded (4.20) The age group (46-60) also recorded (4.88) compared

to control people (4.31) While the lowest rate was observed in the age group (61-75) for infected females, where it reached (4.90), compared to control people, where it reached (4.80).

Table 3 : Explains the Effect of Gender and age Group on the Myoglobin

Sex	Case	Age	Myoglobin	p-value
Male	**Patient**	**30-45**	93.15	0.04
		46-60	87.07	
		61-75	84.05	
	Control	**30-45**	54.53	
		46-60	64.57	
		61-75	61.13	
Female	**Patient**	**30-45**	76.34	0.04
		46-60	71.39	
		61-75	67.33	
	Control	**30-45**	45.91	
		46-60	54.56	
		61-75	50.15	

The results of the statistical analysis in Table 3 showed that there were significant differences between the age groups for males, where the highest rate of Myoglobin was recorded for infected people in the age group (30-45), where it was recorded (93.15), compared to control people, where it was recorded (54.53).

The age group (46-60) also recorded the second highest rate, reaching (87.07), compared to control people, reaching (64.57) While the lowest rate was observed in the age group (61-75) for infected males, where it reached (84.05), compared to control people, where it reached (61.13) . The results of the statistical analysis in Table 3.3 showed that there were significant differences between the age groups for females, where the highest rate of Myoglobin was recorded for infected people in the age group (30- 45), where it was recorded (76.34), compared to control people, where it was recorded (45.91) The age group (46-60) also recorded the second highest rate, reaching (71.39), compared to control people, reaching (54.56) While the lowest rate was observed in the age group (61-75) for infected females, where it reached (67.33), compared to control people, where it reached (50.15).

Table 4 : Explains the Effect of Gender and age Group on the H-FABP

Sex	Case	Age	H-FABP	p-value
Male	**Patient**	**30-45**	10.19	0.04
		46-60	9.89	
		61-75	8.92	
	Control	**30-45**	6.73	
		46-60	7.41	
		61-75	7.46	
Female	**Patient**	**30-45**	9.37	0.05
		46-60	8.97	
		61-75	9.32	
	Control	**30-45**	6.09	
		46-60	6.44	
		61-75	7.10	

The results of the statistical analysis in Table 4 showed that there were significant differences between the age groups for males, where the highest rate of H-FABP was recorded for affected people in the age group (30-45), where it was recorded (10.19), compared to control people, where it was recorded (6.73) The age group (46-60) also recorded the second highest rate, reaching (9.89), compared to control people, where it reached (7.41) While the lowest rate was observed in the age group (61-75) for infected males, where it reached (8.92), compared to control people, where it reached (7.46).
The results of the statistical analysis in Table 3.4 showed that there were significant differences between the age groups for females, where the highest rate of H-FABP was recorded for affected people in the age group (30-45), where it was recorded (9.37), compared to control people, where it was recorded (6.09) The age group (61-75) also recorded the second highest rate, reaching (9.32), compared to control people, where it reached (7.10) While the lowest rate was observed in the age group (46-60) for infected females, where it reached (8.97), compared to control people, where it reached (6.44).

Table 5: Explains the Effect of Gender and age Group on the AST

Sex	Case	Age	AST	p-value
Male	**Patient**	**30-45**	239.11	0.01
		46-60	144.35	
		61-75	180.65	
	Control	**30-45**	32.23	
		46-60	32.33	
		61-75	83.66	
Female	**Patient**	**30-45**	299.85	0.02
		46-60	197.10	
		61-75	128.08	
	Control	**30-45**	31.09	
		46-60	31.33	
		61-75	50.50	

The results of the statistical analysis in Table 5 showed that there were significant differences between the age groups for males, where the highest rate of AST was recorded for infected people in the age group (30-45), where it was recorded (239.11), compared to control people, where it was recorded (32.23)The age group (61-75) also recorded the second highest rate, reaching (180.65), compared to control people, where it reached (83.66) While the lowest rate was observed in the age group (46-60) for infected males, where it reached (144.35), compared to control people, where it reached (32.33).
The results of the statistical analysis in Table 3.11 also showed that there were significant differences between the age groups for females, where the highest rate of AST was recorded for infected people in the age group (30-45), where it was recorded (299.85), compared to control people, where it was recorded (31.09) . The age group (46-60) also recorded the second highest rate, reaching (197.10), compared to control people, where it reached (31.33)While the lowest rate was observed in the age group (61-75) for infected females, where it reached (83.66), compared to control people, where it reached (50.50).

Table 6 : Explains the Effect of Gender and age Group on the ALT

Sex	Case	Age	ALT	p-value
Male	**Patient**	**30-45**	67.57	0.04
		46-60	70.64	
		61-75	89.85	
	Control	**30-45**	36.61	
		46-60	37.00	
		61-75	81.08	
Female	**Patient**	**30-45**	134.42	0.03
		46-60	69.30	
		61-75	47.83	

	Control	**30-45**	35.18	
		46-60	38.55	
		61-75	49.00	

The results of the statistical analysis in Table 6 showed that there were significant differences between the age groups for males, where the highest rate of ALT was recorded for affected people in the age group (61-75), where it was recorded (89.85), compared to control people, where it was recorded (81.08) The age group (46-60) also recorded the second highest rate, reaching (70.64), compared to control people, where it reached (37.00).While the lowest rate was observed in the age group (30-45) for infected males, where it reached (67.57), compared to control people, where it reached (36.61).

The results of the statistical analysis in Table 3.12 also showed that there were significant differences between the age groups for females, where the highest rate of ALT was recorded for affected people in the age group (30-45), where it was recorded (134.42), compared to control people, where it was recorded (35.18). The age group (46-60) also recorded the second highest rate, reaching (69.30), compared to control people, where it reached (38.55) While the lowest rate was observed in the age group (61-75) for infected females, where it reached (47.83), compared to control people, where it reached (49.00).

4. DISCUSSION

Troponin

The significant increase recorded in the results of this study regarding the troponin enzyme may be attributed to the continued collapse of the myofibrillar complex in damaged cardiac muscle cells and to a prolonged rise in the concentration of troponin in the blood (4,5) This increase in the concentration of the troponin enzyme can also be attributed to the diseases underlying the development of ACS resulting from the erosion and rupture of the fibrous cap that contains the atherosclerotic plaque rich in lipids, which accelerates the formation of a thrombus within the coronary artery (6).

The results of the troponin enzyme concentration were consistent with what the researchers indicated (7.8.9,10,11).

Creatine kinase

As for the results of this particular study regarding the Creatine kinase enzyme, a non-significant difference was recorded, meaning that there was a decrease in the concentration of the Creatine kinase enzyme that may be due to the early release pattern that limits its use in diagnosing late myocardial infarction (12).

The results of the concentration of the enzyme Creatine kinase were consistent with what the researchers indicated (13), as this study showed a decrease in the concentration of Creatine kinase and a decrease in the specificity and sensitivity of the heart. Another study also showed that troponin is a more sensitive indicator of heart muscle cell injury than creatine kinase (14).

Is also reported that the weak sensitivity and specificity for of Creatine kinase in this regard and its accuracy is still controversial, especially after resuscitation (15). However, it seems that it can be a useful marker along with other diagnostic approaches for MI detection.

There are other studies that contradict the current study on the enzyme Creatine kinase, as they indicate that there is a significant increase in the concentration of the enzyme Creatine kinase in patients with myocardial infarction as a result of damage to heart muscle cells, which leads to the secretion of large amounts of Creatine kinase (16). There is also a study that contradicts our study, as it shows that there is a significant increase in the concentration of the enzyme Creatine kinase (8).

H-FABP, Myoglobin

As for the study on the enzymes H-FABP and myoglobin, a significant increase in the concentrations of these two enzymes was recorded in patients suffering from myocardial infarction. The reason for this increase may be explained by damage to the heart muscle or ischemic cause of H-FABP elevation.

These results of the H-FABP study were identical to what researchers reached in previous studies (17,18), as well as another study (19,20), which revealed that the protein associated with Heart-type fatty acid binding (H-FABP) is a new biomarker that appears to be released from injured myocardium and is detected in the blood within one hour after the onset of ischemia. (21) Several studies have shown that it is a sensitive early marker of myocardium. Injury.

The results of the Myoglobin study were also consistent with the findings of researchers in previous studies (22,23), as well as another study (24). Patients who suffer from acute infarctions have higher levels of myoglobin in the blood by about Ten times higher on average than that found in normal individuals.

This research has shown that out of all the biomarkers being investigated, H-FABP has the potential to be used in the early diagnosis of AMI. H-FABP has been of interest as a biochemical marker of heart muscle injury since 1988, when it was discovered to be released from damaged heart tissue (25). The release pattern of H-FABP after an AMI indicates that its levels can be detected as early as 1 hour after symptoms appear, reach a peak between 2 to 4 hours, and return to normal within 16 to 24 hours due to rapid clearance by the kidneys. Several studies that highlighted the usefulness of H-FABP as an early marker for AMI were conducted before cardiac troponins became widely used. The current findings suggest that when it comes to detecting AMI, serum H-FABP has a higher level of diagnostic sensitivity and specificity compared to cTnI and CK-MB, similar to myoglobin, especially in the early stages (within 1-2 hours and at 3 hours) after symptoms appear(26).

The diagnostic sensitivity of H-FABP has been proposed to be high, surpassing that of myoglobin in patients who seek medical attention within 6 hours of experiencing chest pain. This advantage may be due to the fact that H-FABP levels increase more quickly than myoglobin levels. Following thrombolysis treatment, levels of H-FABP reach their highest point about 4 hours after the onset of chest pain and return to normal within 24 hours. Due to its speedy return to baseline, H-FABP can serve as an early biological indicator of post-thrombolysis recovery. (27,25). Furthermore, the concentration of H-FABP in the heart muscle is higher than in the skeletal muscle when compared to myoglobin. Additionally, the baseline concentration of H-FABP is much lower than that of myoglobin. These factors make H-FABP a potentially better cardiac marker than myoglobin. (28).

In contrast, Alansari and Croal Xiano et al. suggested that when evaluating patients with chest pain, measuring HFABP and myoglobin at the time of presentation (3-12 hours) has limited clinical significance compared to measuring cTnI. (29),(30).

AST-ALT

As for the results of the study on liver enzymes, a significant increase in the concentrations of these enzymes was recorded. This may explain the significant increase in low blood pressure associated with AMI, as it will directly affect hepatic blood flow and thus increase ALT and AST. Levels of hepatic transaminases may also be elevated due to hepatic congestion caused by severe right ventricular dysfunction. Current research suggests that venous congestion, decreased ability of hepatocytes to extract oxygen, and reperfusion injury are of particular importance (31), although the source of elevated blood transaminases may be ischemic myocardial tissue. However, increased levels of these enzymes often reflect HLI secondary to both impaired anterior perfusion and passive posterior congestion, which are prevalent during myocardial infarction (32).

There is another reason why these enzymes are elevated. Incomplete peripheral lipolysis of very low-density lipoprotein (VLDL) particles rich in triglycerides is known to increase the number of small dense LDL (sdLDL) particles, which carry a disproportionately large amount of LDL. (33,34) ALT levels are directly related to the desmostrol:TC ratio, a marker of cholesterol synthetic activity, suggesting that ALT elevations are associated with increased hepatic triglycerides(35).

This study is consistent with previous studies referred to by researchers (36). AST and ALT are often elevated, especially in patients with myocardial infarction. Lofthus and his colleagues also confirmed in their study the elevation of AST and ALT in AMI patients (38). Some recent studies have reported that elevated serum transaminases were independently associated with poor clinical outcomes in patients with AMI. The extent of liver injury, especially in patients with pre-existing metabolic syndrome, may have a direct impact on cardiac outcomes (38).

Conclusions: In patients with AMI, The plasma level of significant increase in troponin, myoglobin, and H-FABP, AST and ALT at $p \leq 0.5$. and a significant decrease in Creatine kinase at $p \leq 0.5$. We conclude from these results that H-FABP , Troponin and Myoglobin are important markers in the diagnosis of myocardial infarction, in contrast to Creatine Kinase, which showed a decrease in the diagnosis of the disease.

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