Clinical Study Of Some Physiological Parameters in Patients With Acute Coronary Syndrome (ACS) in Thi-Qar Governorate / Iraq

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Abstract

Objective: The acute coronary syndromes [unstable angina (UA) and acute myocardial infarction (AMI)] are more dangerous than others ischemic heart diseases (IHD) due to acute morphological changes in atherosclerotic plaques which cause (acute ischemia) severe imbalance between myocardium demand and oxygen supply. The present study was designed to determine and compare the levels of (GOT, GPT, Total protein , Albumin ,Globulin and Uric acid) in patients with (ACS) and healthy individuals. **Material and Methods:** serum glutamate oxaloacetate transaminase (S.GOT) , serum glutamate pyruvate transaminase (S.GPT) , serum total protein , serum albumin , serum globulin and serum uric acid levels were measured in 75 subjects which were divided into three groups : (25) patients of UA and (25) patients of AMI , were compared with (25) healthy subjects (control**).Results:**The levels of serum glutamate oxaloactate transaminase(S.GOT) , serum glutamate pyruvate transaminase(S.GPT), serum albumin and serum uric acid were significant increase in patients of (UA) and (AMI) when compared with control. Whereas the levels of serum albumin showed a significant decrease in patients of (UA) in comparison with control group , whereas its levels was not affected in blood serum of (AMI) patients in comparison with control. While the levels of serum globulin showed significant increase in patients of AMI in comparison with control group, whereas its levels was not affected in blood serum of (UA) patients in comparison with control ($p \leq 0.01$).**Conclusion:** High levels of GOT , GPT , globulin and low levels of total protein and albumin could be causative for ACS.

Key word : Acute coronary syndrome , ischemic heart diseases , GOT , GPT , total protein , albumin , uric acid , coronary heart diseases .

Introduction

Acute Coronary Syndromes (ACS) is a term used to describe a group of conditions resulting from acute myocardial ischemia (insufficient blood flow to heart muscle) and ranging from unstable angina (increasing, unpredictable chest pain) to non–STelevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI). The conditions are related to varying degrees of narrowing or blockage of single or multiple coronary arteries that provide blood, oxygen and nutrients to the heart. This life-threatening disorder is a major cause of emergency medical care and hospitalization.[1] Coronary heart disease (CHD) is the leading cause of death in the whole world .[2] Patients with ACS are divided into two groups: Patients with and without ST segment elevation. ACS without ST segment elevation (NSTEACS) also includes unstable angina pectoris (UA) and non-ST elevation myocardial infarction (NSTEMI). It is important to note that UA is defined as ischemic chest pain at rest without a rise in serum cardiac biomarkers, while the establishment of NSTEMI diagnosis requires a rise in serum cardiac biomarkers. ACS with ST segment elevation (STEMI) includes both ST segment elevation and a rise in serum cardiac biomarkers .[3] Connection of UA, NSTEMI and STEMI is based on the fact that these are closely connected conditions with similar pathogenesis and clinical presentation, but they do differ by the level of severity .[4] The World Health Organization estimates that air pollution is responsible for 800,000 premature death worldwide each year .[5]

Numerous nonspecific manifestations may be recognized in patients with acute MI. Although they are not generally employed in establishing the diagnosis, awareness of their coexistence with infarction is important to avoid misinterpretation or erroneous diagnosis of other disorders .[6] Aspartate aminotransferase (AST, also sometimes termed S.GOT) and alanine aminotransferase (ALT, also sometimes termed S.GPT) are widely distributed in cells throughout the body. AST is found primarily in heart, liver, skeletal muscle, and kidney, while ALT is found primarily in liver and kidney, with lesser amounts in heart and skeletal muscle. AST and ALT activity in liver are about 7,000 and 3,000 times serum activities, respectively.[7] ALT is exclusively cytoplasmic; both mitochondrial and cytoplasmic forms of AST are found in all cells .[8] The half-life of total AST is 17 ± 5 hours, while that of ALT is 47 ± 10 hours .[9] The half-life of mitochondrial AST averages 87 hours .[10] In adults,

AST and ALT activities are significantly higher in males than in females, and reference intervals vary with age. Examination of the proteins in plasma can provide information reflecting disease states in many different organ systems. Although the total protein determination gives some information about the patient's general status regarding nutrition, or severs organ disease, further fractionations yield far more clinically useful information .(11) The calculated difference between the total protein and albumin represents the value of all globulins, a composite of the other fractions that individually may raise several folds in severe disorders .(12)

Albumin is the major protein of human plasma, usually consisting up to two thirds of the total plasma protein .(13) About 40% of the albumin is present in the plasma, and the other 60% is present in the extracellular space .(14) However, the concentration of albumin in the smaller intravascular compartment is much higher because of the relative impermeability of the blood vessel wall. This concentration gradient across the capillary membrane is important in maintaining plasma volume . (15)Uric acid is a naturally occurring product of purine metabolism(by xanthine oxidase from xanthine and hypoxanthine), which plays different roles in human body.(16) Uric acid is present in plasma in relatively high concentrations: in men 302±60 µmol/L; in women, 234±52µmol/L. (16)Humans have no enzyme to further oxidase uric acid, so an access of uric acid is excreted by kidney.(17)Uric acid is a weak acid (pKa 5.8) and distributed throughout the extracellular fluid compartment as sodium urate. (18)We aiming to measure some physiological parameters including (GOT, GPT, total protein , albumin , globulin and uric acid in serum of ACS patients.

Materials and Methods

This study was conducted at AL-Hussein Teaching Hospital in Thi-Qar/Iraq, especially coronary care unit (CCU). It included (75) subjects, control(25) and patients(50) diagnosed with(Acute Myocardial Infarction and unstable Angina/ Non STEMI).

About(5mL)of blood samples of the patients with unstable angina (UA) NSTEMI, acute myocardial infarction(AMI) patients and controls were taken and allowed to clot at room temperature in empty disposable tubes centrifuge to separate it in the centrifuge at 3000 rotor per minute (rpm)for 10min,the serum samples were separated and stored at (-20ºC) until analyzed for serum glutamate oxaloacetate transaminase

(S.GOT) , glutamate pyruvate transaminase(S.GPT),serum total protein ,serum albumin(Alb) , globulin , serum uric acid . Kits of glutamate oxaloacetate transaminase(GOT) and glutamate pyruvate transaminase(GPT), total protein ,Albumin(Alb) , uric acid were purchased from Biolabo (France)

Statistical Analysis : Data were statistically analyzed using Package Social Sciences (SPSS) for Windows version 12.0 software. All experimental data were expressed as mean ± standard deviation(SD) .

Results

In this study we measured the level of glutamate oxaloacetate transaminase (GOT) , glutamate pyruvate transaminase (GPT),total protein , albumin(Alb),globulin ,uric acid among patients with different ACS (UA/Non STEMI , AMI and healthy individuals.

Table (1) shows a significant increase in concentrations of serum glutamate oxaloacetate transaminase (GOT) in group AMI in comparison with group UA/NSTEMI ($p<0.01$). Also there is a significant increase in concentrations of serum glutamate oxaloacetate transaminase (GOT) in all patients groups in comparison with CTR group ($p<0.01$).

Also there is a significant increase in concentrations of serum glutamate pyruvate transaminase (GPT) in all patients groups in comparison with CTR group ($p<0.01$).

Table(2) shows a significant decrease ($p<0.01$) in concentrations of serum total protein in all patients groups in comparison with control group CTR , and there is no a significant differences in concentration of serum total protein between AMI and UA group can be observed. While there is a significant decrease($p<0.01$) in concentrations of serum albumin in group UA in comparison with CTR group and there is no a significant differences in concentrations of serum albumin in group AMI in comparison with groups (CTR and UA).Whereas a significant increase ($p<0.01$) in concentration of serum globulin in group AMI in comparison with group CTR, and there is no a significant differences in concentrations of serum globulin in group UA in comparison with groups (CTR and AMI).While, there is a significant increase($p<0.01$)in concentrations of serum uric acid in all patients groups in

comparison with control group CTR , and there is no a significant differences in concentrations of serum uric acid between AMI and UA groups can be observed .

Table (1) Serum GOT and GPT concentrations of CTR , UA and AMI groups.

Groups	n	GOT(U/L)	GPT(U/L)
CTR	25	29.54±6.57^{c}	21.33±5.43^{b}
UA	25	77.60±6.98^{b}	48.80±7.17^{a}
AMI	25	94.80±7.35^{a}	52.66±11.00^{a}

* Each value represents mean ± SD values with non-identical superscript (a , b or c …etc.) were considered significantly differences ($P \leq 0.01$).

CTR : Control group .

UA: unstable angina group .

AMI: Acute myocardial infarction group.

Table (2) Serum Tatal protein , Albumin , Globulin and Uric acid of CTR , UA and AMI groups.

Groups	n	Total protein(mg/dL)	Albumin(mg/dL)	Globulin(mg/dL)	Uric acid(µmol/L)
CTR	25	6.59±0.49 ^{a}	5.32±0.05 ^{a}	1.27±0.23 ^{a}	210.30±65.90 ^{b}
UA	25	5.31±0.58 ^{b}	3.79±0.64 ^{b}	1.51±0.46 ^{ab}	308.65±96.37 ^{a}
AMI	25	5.90±0.47 ^{b}	4.07±0.49 ^{ab}	1.82±0.48 ^{b}	290.50±70.80 ^{a}

Legend as in table (1).

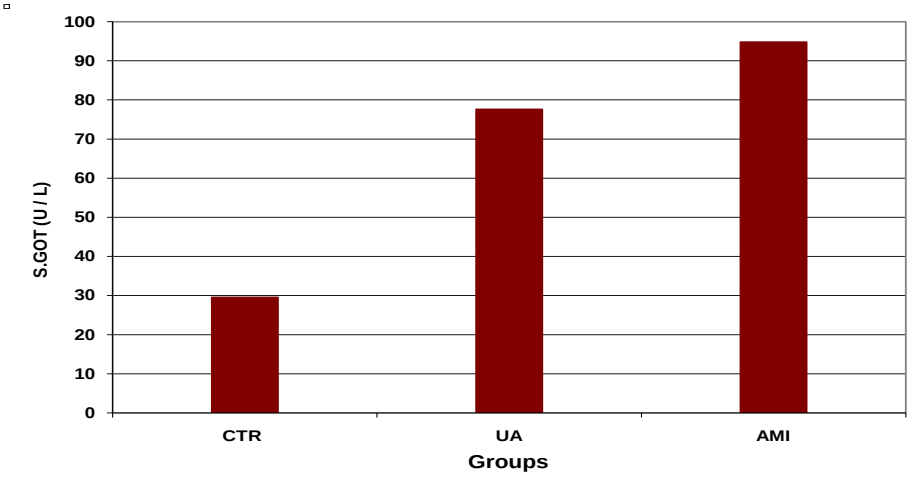

Figure (1) : Serum GOT levels of (CTR),(UA/NSTEMI) and (AMI) groups**.**

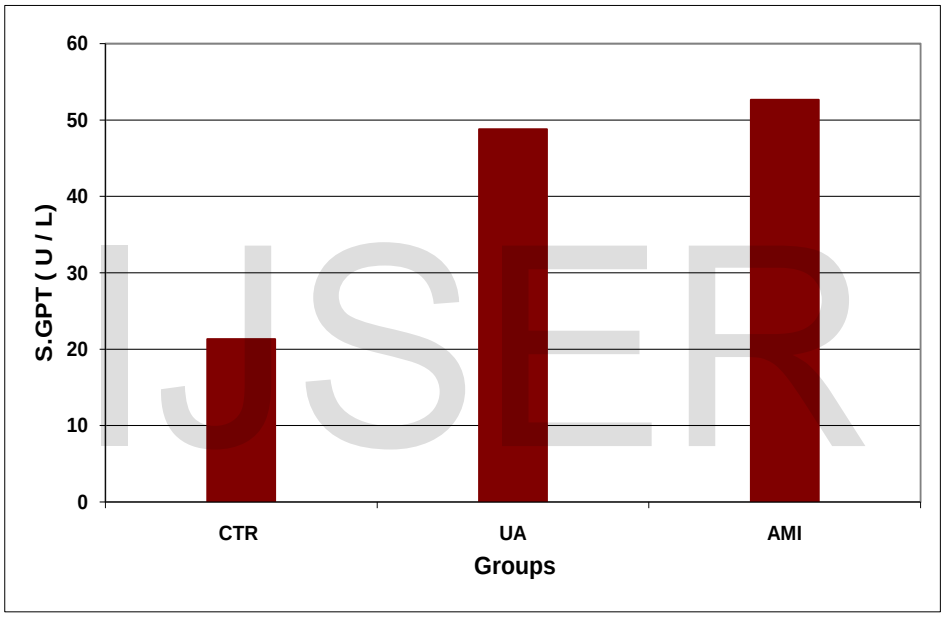

Figure (2) : Serum GPT levels of (CTR),(UA/NSTEMI) and (AMI) groups**.**

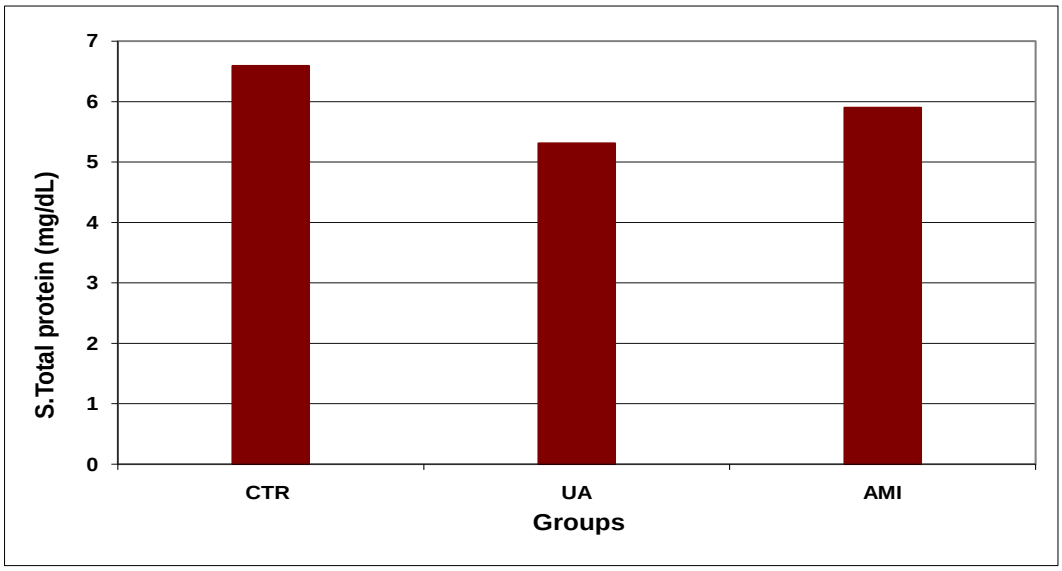

Figure (3) : Serum Total protein levels of (CTR),(UA/NSTEMI) and (AMI) groups.

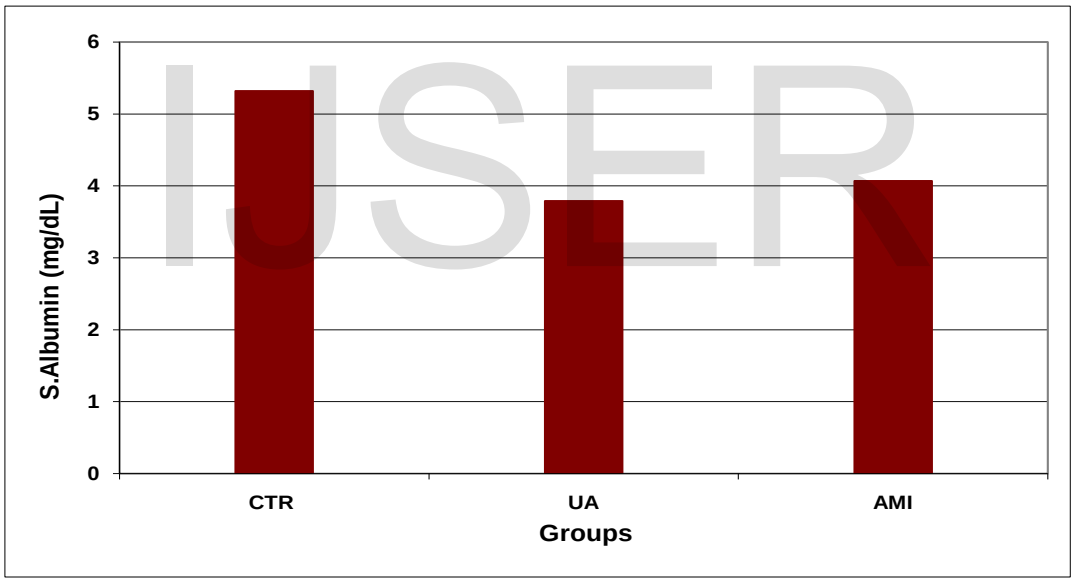

Figure (4) : Serum Albumin levels of (CTR),(UA/NSTEMI) and (AMI) groups.

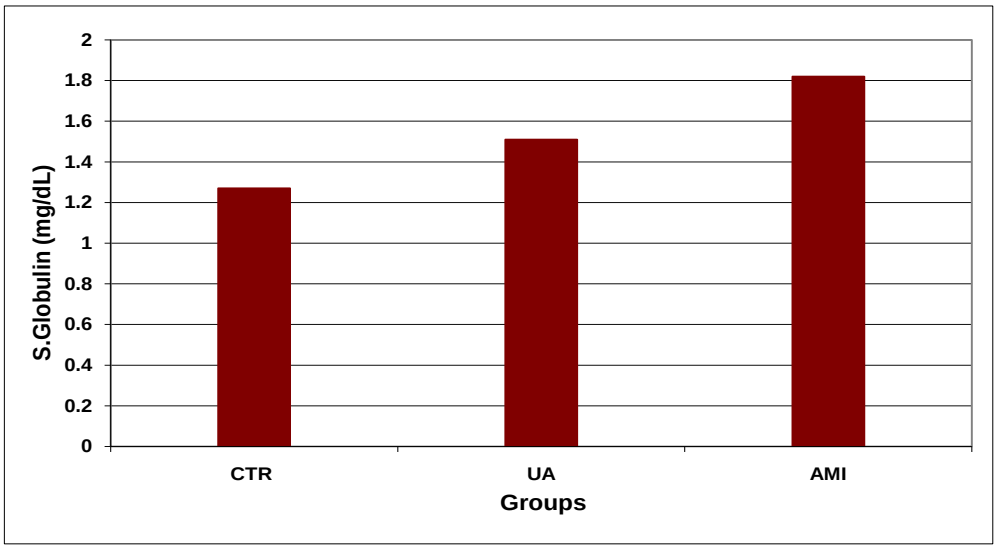

Figure (5) : Serum Globulin levels of (CTR),(UA/NSTEMI) and (AMI) groups**.**

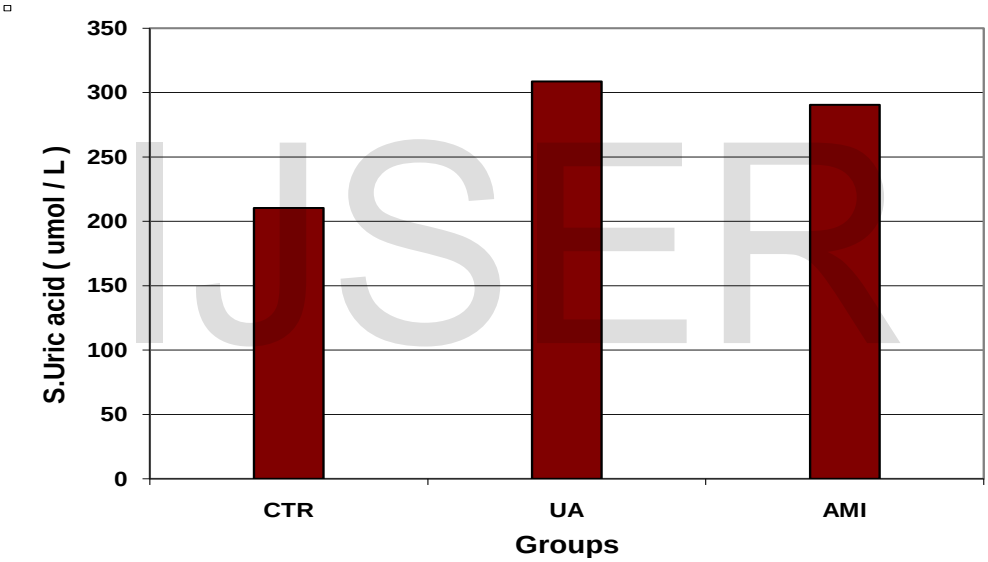

Figure (6) : Serum Uric acid levels of (CTR),(UA/NSTEMI) and (AMI) groups**.**

Discussion

Ischemic heart disease (IHD) is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium; it typically occurs when there is an imbalance between myocardial oxygen supply and demand. The most common cause of myocardial ischemia is atherosclerotic disease of an coronary artery (or arteries) sufficient to cause a regional reduction in myocardial blood flow and inadequate perfusion of the myocardium supplied by the involved coronary artery[(19)]and hence IHD are often termed coronary heart disease or coronary artery

diseases (CAD) .[20]Patients with ischemic heart disease fall into two large groups: patients with chronic CAD who most commonly present with stable angina and patients with acute coronary syndrome (ACS). The latter group, in turn, is composed of patients with acute myocardial infarction (AMI) with ST-segment elevation on their presenting electrocardiogram (STEMI) and those with unstable angina (UA) and non-ST-segment elevation MI (UA/NSTEMI).[19]

In the present study , markers of myocardial injury including GOT , GPT were significantly higher in AMI and UA groups compared with control group . These results were agreed with those stated by Bergovec *et al*.(2009)[3] who observed that markers of myocardial injury can be detected in the blood stream after the onset of ischemic chest pain, which then allows the differentiation between UA(i.e., no biomarkers in circulation ; usually transient, if any) and NSTEMI (i.e., elevated biomarkers). The decrement in total serum proteins concentration in patients with UA and AMI can be explained by the fact that myocardial infarction is one of the condition that initiates the acute phase response, and that can affect the levels of specific hepatic proteins (e.g: acute phase protein).[11] The depression, which happened in serum albumin levels in case of UA, is according to some causes:

(1) Increasing of the albumin excretion by kidney.[21]

(2) Diffusing of the albumin into damaged tissues by means of increased permeability of blood vessels.[22]

(3) Inflammation is considered the principle cause of a decrease in the serum albumin, however, IL-6 directly decreases the expression of albumin messenger RNA and finally. [23]

(4) Its antioxidant function.

According to the points (3) and (4): (stopping for the albumin production and consumption of the albumin to scavenge free radicals with continuously. On the other hand, an elevation in serum globulins that is observed in serum of AMI patients can be explained by the fact that tissue damage triggers the sequence of biochemical and cellular events associated with inflammation, which include stimulation of synthesis of the acute phase proteins, with a rise in the α1- and α2- globulin fractions .[15] The changes of globulins inA MI patients can be confirmed by the significant differences in the PAGE pattrens of serum patients when compared with control group . The different or additional bands that appear in glycoprotein profile of the AMI patients, particularly, in the globulins region, can be attributed to the fact, that many proteins of

cellular membrans are glycoproteins, therefore when the myocyte cells became necrotic or dead (cell damage), these proteins are released into the blood and cause altration in serum glycoprotein profile .[24,25]This difference between the mean level of serum uric acid for patients in ACS group and control subjects can be related to adenosine synthesis and release which are up regulated under conditions of hypoxia and tissue ischemia .[26,27]

Ischemia also promotes the conversion of xanthine dehydrogenase (XDH) to xanthineoxidase (XO), as the likely result of increased intracellular calcium, and activation of proteases.Whereas XDHactivity does not produce active oxygen species, the XOreaction is a major source of free radicals during ischemia/reperfusion injury. So raised serum uric acid concentrations are a powerful predictor of cardiovascular risk and poor outcome . [28]

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