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EVALUATION OF SOME BIOCHEMICAL INDICATORS IN PATIENTS WITH GALLSTONES

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ABSTRACT

Gall stones are the primary cause of acute cholecystitis, this study was designed to evaluate the levels of blood biochemical markers in patients with gallbladder inflammation, such as superoxide dismutase and liver function enzymes, as well as total protein and bilirubin. One hundred twenty individuals participated in this study, divided into two groups: Forty healthy control individuals versus eighty cholecystitis patients, who attended private laboratories and hospitals in Kirkuk, northern Iraq, from February to April of 2023. Serum examinations of patients and control groups were performed to test liver function, total serum bilirubin, and superoxide dismutase. Results showed an increase in hepatic enzymes activities with high total serum bilirubin in patient group versus control individuals. This study concluded that it is possible to rely on liver enzymes in diagnosing inflammation of the gallbladder and bile ducts.

KEYWORDS: Blood markers, Cholecystitis, Serum examinations.

INTRODUCTION

Gallstone is the most common cause of acute cholecystitis, or inflammation of the gallbladder.[1,2] It is one of the most common gastrointestinal diseases, affecting more than 10% of people in Western countries.[3] Most of people with gallstones do not show any symptoms, and a fair percentage of individuals with symptoms of gallstones develop acute cholecystitis.[4] The inflammatory process begins when the cystic duct becomes blocked. If the inflammation is not treated, the gallbladder may rupture or develop gangrene.[5,6] Clinical indicators are used to diagnose acute cholecystitis, and ultrasound results are used to confirming it.[7] Surgery is the primary method of treatment, while there is disagreement about when surgery should be performed.[8,9] Severe trauma, low postoperative cardiac output, severe burns, prolonged prolongation, and injections are risk factors.[10] Sepsis, gallbladder perforation, and death can occur as a result of acute cholecystitis that goes untreated.[11] Some chemicals that may lead to gallstones are bilirubin and cholesterol.[12] These compounds raise risk degree of cholecystitis and cholelithiasis in some conditions as sickle cell disease, where the destruction of erythrocytes leads to increased bilirubin and the development of pigmented stones.[13]

Otherwise, many people suffering from excessive calcium high levels, as those with hyperparathyroidism, may induce calcium stones. Cholesterol stones can form in people with high cholesterol levels.[14] In addition, stagnant bile flow caused by tumors or strictures that obstruct bile duct may lead to the formation of gallstones.[15] A number of risk variables, such as gender (female), pregnancy, age, obesity, and low physical activity, are associated with the development of gallstones.[16,17]

PATIENTS AND METHODS

One hundred and twenty individuals participated in the study, forty of whom were in good health as controls, and eighty others with cholecystitis who presented to private laboratories and hospitals in Kirkuk, northern Iraq, from February to April 2023. Informed consent was taken from each individual to participate in this study. The ages of the participants ranged between 25 and 65 years of both genders. Serum examinations of patients and controls were performed to test liver function, total serum bilirubin and superoxide dismutase. About 10 ml of venous blood was collected from each one by disposable syringe after an 8–12 h fast, and the blood was then allowed to clot in a plain tube at room temperature. The serum was centrifuged for 15 minutes at 3000 rpm before aspiration, divided into aliquots, and kept at (−20°C) until estimation. Statistical significances were determined using Graph pad prism 8, and the data were presented as (means ± standard deviation). The

variance between the two groups were determined by (t-test) and a P<0.05 were required for significances.

RESULTS

Table (1) and Figure (1) show the levels of serum indicators in both control and patient groups. The results showed an increase in the ALT level at a probability level (0.0431) in patients (32.04±7.950 U/l) compared to the control group (16.13±6.084 U/l). The findings revealed elevate in AST level with a probability level (0.0001) in patients (46.56±11.31 U/I) compared with control group (15.47±4.747 U/I). It was also proven that ALP level increased with a probability (0.0157) in patients (65.87±16.88 U/I) compared to the control group (119.15±15.36 U/I). Also an increase in GGT level with P < 0.0001 was observed in patients (111.3±60.61 U/I) compared with control group (40.43±15.29 U/I). The results showed an increase in TSB concentration with P<0.0001 in patients compared to control group. Oxidative stress indicators are important prognostic variables for many disorders, including cholelithiasis. As indicated patients with gallstones (148.6±11.62 U/I) outperformed control group (148.6±11.62 U/I) with P<0.0474.

Table 1: Comparing hepatic indicators levels in patient with control groups.

Indicators (U/l)	Control (n=40)	Patient (n=80)	P-Value
ALT	16.13±6.084	32.04±7.950*	0.0431
AST	15.47±4.747	46.56±11.31**	<0.0001
ALP	65.87±16.88	119.15±15.36*	0.0157
GGT	40.43±15.29	111.3±60.61**	<0.0001
TSB	0.466±0.212	6.723±2.381**	<0.0001
SOD	192.6±14.07	148.6±11.62*	0.0474

* Significant difference (≤0.05), ** High Significant difference (≤0.01)

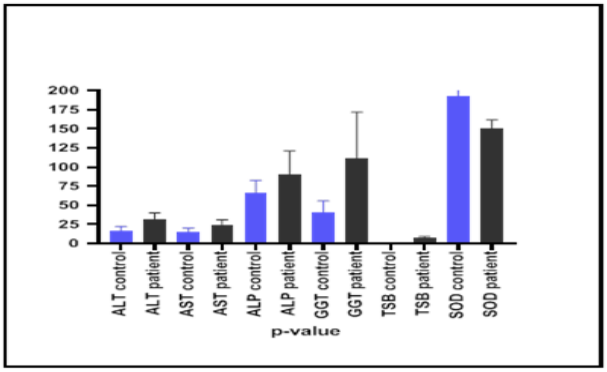

Figure 1: Compared hepatic indicators levels between Patients and Control groups.

Results of patient in table (2) were confirmed the presence of significant positive highly correlation between AST and ALP (0.173), AST and total protein (0.165), AST and GGT (0.148), ALT and ALP (0.137). A significant moderate positive correlation was between total protein and SOD (0.097), AST and TSB (0.081), ALP and GGT (0.079), ALT and SOD (0.074). While a weak positive correlation was between AST and SOD (0.054), ALT and TSB (0.05), ALP and total protein (0.025).

Results of control group were showed the presence of significant positive highly correlation between AST and ALP (0.345), ALT and total protein (0.229), total protein and SOD (0.180), ALT and SOD (0.176). A significant moderate positive correlation was between TSB and SOD (0.132), ALT and AST (0.081), AST and total protein (0.067). While a weak positive correlation was between GGT and TSB (0.032), GGT and SOD (0.025), GGT and ALP (0.019).

There was significant positive correlation appeared in patients and control group between AST and ALP, AST and TSB, TSB and SOD, ALP and GGT, ALT and SOD

Table 2: Correlation between studied serological indicators.

	Patient group					
	ALT	**AST**	**ALP**	**GGT**	**TSB**	**SOD**
ALT	1					
AST	-0.136	1				
ALP	0.137	0.173	1			
GGT	-0.299	0.148	0.079	1		

TSB	0.050	0.081	-0.232	-0.025	1	
SOD	0.0742	0.054	-0.092	-0.056	-0.138	1
Control group						
	ALT	**AST**	**ALP**	**GGT**	**TSB**	**SOD**
ALT	1					
AST	0.081	1				
ALP	-0.101	0.345	1			
GGT	-0.331	-0.272	0.019	1		
TSB	-0.164	-0.182	-0.157	0.032	1	
SOD	0.176	-0.115	-0.298	0.025	0.132	1

DISCUSSION

In general, when the gallbladder is disturbed, it causes damage to liver cells, thus liver-related enzymes such as ALT, AST, ALP, and TSB escape into the blood, leading to an increase in their levels in the serum.[18] Therefore, high levels of activity of these liver enzymes can be taken as a sign of the risk of developing gallstones. Also, a high level of GGT enzyme activity can be considered a diagnostic marker, as it remains higher for a longer period than other liver enzymes.[19] In the present study, when comparing gallstones patients with control, the mean level of ALT was higher than the control; this result was in agreement with previous study.[20] Undoubtedly, if the liver is damaged, the levels of this enzyme in the blood will be higher than normal. Alternately, hepatic or bile duct blockage may be the cause of increased enzyme levels.[21,22] The fact that AST activity is higher suggests cholecystitis (Acute and Chronic) or bile duct obstruction.[23] An elevated level of this enzyme activity in acute phases may be due to enhanced release from the cytoplasm of affected hepatocytes and a change in cell membrane permeability. Thus, it is transferred to blood, and the increase is proportional to the degree of damage, that is, it increases with the number of injured cells.[24,25] In other words, because the third area of the hepatic acinus contains the most AST enzyme, any injury to this region increases the concentration of AST enzyme in the serum, which is carried from the liver by sinusoidal cells.[26] In this study, when comparing patients with gallstones and controls, the mean level of AST was higher than the control, this result was in agreement with other study findings.[27] A higher-than-normal alkaline phosphatase activity level implies acute cholecystitis and bile duct blockage, as well as hepatic inflammation.[28,29] It is in the first plasma membrane of hepatocytes as well as the epithelial cells of the bile duct, through which a substantial amount of bile is excreted. The main function of this enzyme is to transport substances across cell membranes. As a result, injury to these tissues causes alkaline phosphatase to be released into the circulation.[30,31] The activity of gamma-glutamyl transpeptidase may be used as a strong indicator and a reliable marker suggesting risk of gallstones or cholecystitis. This enzyme was shown to have a higher degree of activity and survive for a longer amount of time than other liver enzymes in instances of liver and bile duct illnesses. It is present in the biliary system's cell membranes, makes it extremely susceptible to biliary system illnesses, whether they originate inside or outside of the liver.[32,33] A rise in bilirubin levels in cholecystitis patients' serum implies a blockage in the bile ducts caused by gallstones. High bilirubin levels in the blood can be caused by liver disorders due to a reduction in unconjugated bilirubin absorption, and inability of liver cells to conjugate bilirubin that is not linked to glucuronic acid and so is not deposited in the bile sac, which is frequently accompanied with elevated bilirubin levels in the blood and significant impairment of liver function.[34] In addition to a high concentration of direct bilirubin in the blood caused by biliary blockage of the normal path of direct bilirubin in the liver, which prevents it from being discharged to the biliary tree, resulting in an increase in its concentration in the blood.[35]

Oxidative stress indicators are important prognostic variables for many disorders, including cholelithiasis.[36,37] Gallstones can cause inflammation in the gallbladder wall and modify the composition of bile, and might enhance biliary free radical generation while also altering bilirubin metabolism, which is a powerful antioxidant through radical scavenging and lowering actions.[38] Inflamed gallbladders showed increased levels of inducible nitric oxide production activity, which has an influence on oxidative stress and fluid transport. Activated phagocytes can generate reactive oxygen metabolites, resulting in oxidative stress. However, free radicals and other peroxide derivatives are created naturally in the body and amplified in many pathological circumstances. As a result, is shown to be considerably lower in cholelithiasis patients than in controls.[39,40]

CONCLUSION

It has been found that cholecystitis patients have higher ALT, AST, ALP, GGT, TSB, and SOD activities than healthy individuals. The study came out with recommendation including the possibility of relying on liver enzymes to diagnose inflammation of the gallbladder and bile ducts compared to other markers such as C-reactive protein.

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