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ESTIMATION OF LIPID PROFILE IN PATIENT WITH GALLSTONES

Samah Sajad Kadhim[1]*, Fatima Hakim Obaid[2] and Kazem Mehdi Kazem[3]

[1]*Department of Physiology and Medical Physics, Hammurabi College of Medicine, University of Babylon, Hilla 51002, Iraq.
[2]Department of Medical Microbiology, Hammurabi College of Medicine, University of Babylon, Hilla 51002, Iraq.
[3]Department of Medical Microbiology, Hammurabi College of Medicine, University of Babylon, Hilla 51002, Iraq.

***Corresponding Author: Samah Sajad Kadhim**
Department of Physiology and Medical Physics, Hammurabi College of Medicine, University of Babylon, Hilla 51002, Iraq.

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ABSTRACT

Background and objective: The gallbladder is a small, pear-shaped organ. It is part of the biliary system, which includes ducts inside and outside the liver. It contributes to fat digestion by storing and concentrating bile. Gallbladder dysfunction leads to gallstone formation and may require surgical intervention. This study aimed to evaluate changes in lipid profiles (cholesterol, triglycerides, HDL, LDL, and VLDL) in patients with gallstones compared to healthy controls. **Methods:** A prospective, case-control study was conducted from February 10, 2022, to March 1, 2023. 50 randomly selected, clinically diagnosed, participants (healthy and patients with gallstones) aged 25–65 were included. Patients were divided into three age groups, with emphasis on the 40–60 age group due to its high incidence (50%). **Results & conclusion:** The results showed that HDL levels were higher in the control group, while total cholesterol, triglycerides, LDL, and VLDL levels were statistically significantly higher in gallstone patients ($P \leq 0.01$). The results indicate a relationship between dyslipidemia and gallstone formation, supporting the importance of regular lipid monitoring in at-risk groups.

KEYWORDS: Dyslipidemia, Gallstone, lipid profile, Biliary system.

INTRODUCTION

Gallbladder and Bile System: Common anatomical language for the channel via which the liver secretes bile that then travels to the first section of the small intestine, often known as the duodenum, is the biliary tract. All of the ducts, structures, and organs engaged in the generation, storage, and secretion of bile are referred to as the biliary tract. Common to most members of the mammal family, this structure—which starts with numerous minor branches ending in the common bile duct—sometimes known as the trunk of the biliary tree—is shared by most people. The portal triad centers on the duct, the branches of the hepatic artery, and the portal vein. The bile runs in the opposite direction from the blood present in the other two channels.[1]

Gallbladder Purpose

Comprising a hollow, muscular organ, the gallbladder stores and discharges bile into the small intestine's duodenum. Under normal conditions the liver generates bile, a yellowish-green liquid mostly consisting of cholesterol, bile salt, lecithin, and water that emulsifies dietary fat in the small intestine and releases it into the duodenum when fat is present. Produced but not yet needed in the intestine, bile is stored and concentrated in the gallbladder. The biliary system—liver, gallbladder, and related ducts—is kept in normal functioning to guarantee the best bile output into the gastrointestinal tract. But a disturbance in the physiology of this system can cause inadequate bile release, extreme discomfort, inflammation, and infection.[2]

The gallbladder mostly serves to concentrate and store hepatic bile during the fasting state and to send bile into the duodenum in response to a meal.[3]

Secretion and Composition of Bile

Formed in the hepatic lobules, bile is released into a complex network of canaliculi, tiny bile ductules, and larger bile ducts running with lymphatic and portal vein branches in portal tracts between hepatic lobules. Larger septal bile ducts resulting from the coalescence of these interlobular bile ducts connect to produce the right and left hepatic ducts, which in turn merge to form the common hepatic duct. Often following the main pancreatic duct, the cystic duct of the gallbladder joins the common hepatic duct to form the common bile duct, which passes via the ampulla of Vetter into the duodenum.[4] Figure 1: 1-1.

Graph (1-1): Human gallbladder in terms of Kalloo et al., 2001.

Gallstones

Gallstones, or cholelithiasis, are the most often occurring disorder connected to a change in the gallbladder physiology.[5] Gallstone disease is the disorder wherein gallstones either lie in the gallbladder or the common bile duct. From the Greek: chol- (bile) + ang- (vessel) + itis- (inflammation), a major infection of the bile ducts, choledocholithiasis is usually linked with blockage of the biliary tree, which in turn can lead to acute ascending cholangitis. Gallstones in the ampulla of Vater can block the exocrine system of the pancreas, therefore causing pancreatitis.[5] Gallstones in the gallbladder are the sign of cholelithiasis. Generally speaking, gallstones are hard, calcified stones composed of cholesterol, bilirubin, and calcium that develop when the bile component ratio is reversed, therefore hardening the bile into tiny stones. Gallstones come primarily in two varieties: pigment stones and cholesterol stones.[6]

Pathogenesis

Comprising mostly of cholesterol crystals, cholesterol gallstones are the product of cholesterol metabolism gone wrong. Three kinds of aberrations have been thought to be involved in the production of cholesterol gallstones. Bile supersaturation in cholesterol is the first and most crucial need. The three main biliary lipids—cholesterol, bile acids, and phospholipids—determine the percent saturation of the cholesterol in bile by their molar ratio.

Excessive cholesterol production (increasing 3-hydroxy-3-methylglutaryl (HMG) coenzyme) may cause cholesterol super-saturation, the necessary condition for cholesterol gallstone development.[7]

Gallstone Disease Symptoms

Usually, gallstones cause symptoms via the irritation or blockage they cause when they migrate into the cystic duct. Biliary colic is the most particular and defining sign of gallstone disease. The resulting visceral pain is typically a severe, steady ache or pressure in the epigastrium or right upper quadrant of the abdomen with frequent radiation in the interscapular area, right scapula, or shoulder when a stone obstructs the cystic duct and increases intraluminal pressure and distension of the viscus that cannot be relieved by repeated biliary contractions.

Etiology and Risk Variables

Gallstones have been linked to important risk factors that are enumerated here.

Ethnicity

The type of stone that develops as well as the frequency of gallstone disease depend on ethnicity; brown pigment stones in the bile ducts are more common in Asia, whereas cholesterol gallstones are rather widespread in the developed Western countries.

Family Background & Genetics

Gallstone development is mostly dependent on genetic predisposition; family studies find a roughly 5 times higher risk in gallstone patients compared to others. In monozygotic twins at 12% and dizygotic twins at 6%, these rates are much higher.[8]

Age

Gallstone detection rates rise with age; so, one can include gallstone disease as one of the risk factors, as it is infrequent in newborns and young children. Once assumed to be solely connected to pigment stones emerging in the context of hemolysis, cholesterol stones are becoming ever more prevalent in youngsters.[9]

Gender

Female gender is among the most critical risk factors. Gallstones affect women two to three times more than they affect males. Women predominate, especially while young [20–30 years of age], with a range of female-to-male ratio of 10:1 among Pima Indians to 2–3:1 in Europeans; this ratio diminishes beyond the fifth decade.[10]

Diabetes Mellitus/Dyslipidemia with Obesity/Metabolic Syndrome/Rapid Weight Loss

Because GB stone illness connects with metabolic problems like diabetes, dyslipidemia, obesity, and hyperinsulinemia, gallstone (GS) stone development is a manifestation of metabolic syndrome. Common elements tying cholesterol GB stones, diabetes, and obesity include hyperinsulinemia and insulin resistance.[11]

Pregnancy, Parity, and Female Sex Hormones

Two major risk factors for cholesterol gallstone development are pregnancy and parity. The clear basis for this gender difference is female sex hormones. Therefore, it is not unexpected to learn that parity is a risk factor; gallstones form in 1-3%, whereas biliary sludge—which consists of cholesterol crystals, calcium bilirubinate, and mucin—develops in up to 30%.[12]

Diet and Total Parenteral Nutrition (TPN)

Apart from acute cholecystitis in severely unwell individuals, total parental nutrition (TPN) is a well-known risk factor for gallstone disease development. One theory explaining this might be lack of the enteric stimulation of the gallbladder in the absence of food, which causes gallbladder stasis.[13]

Lower Physical Activity

Physical exercise has an inverse link, according to several research studies, between risk of gastrointestinal-associated disorders, including cholelithiasis.[14]

Type of Gallstone: Cholesterol Stones More than 70% of the stone dry weight[15] are cholesterol stones, stones

containing cholesterol. Cholesterol crystallizing from gallbladder bile results in the development of cholesterol stones. Three elements determine this: cholesterol hyper saturation of bile, crystallization encouraging elements within bile, and gallbladder motility.[16]

Pigmented Stones

Pigment stones have their etiology totally unrelated to cholesterol gallstones. Pigment gallstones generally fall into two types: black and brown. Calcium bilirubinate and a network of mucin glycoproteins interlace with salts such as calcium carbonate and/or calcium phosphate to form black pigment gallstones. These stones exhibit a glass-like cross-sectional surface on breaking and range in color from deep black to extremely dark brown.[17]

Mixed stone

Two layers make up the mixed stone, which also commonly consists of cholesterol and varies in calcium and bile pigment content. In micelles in support of phospholipids (mostly lecithin), the bile cholesterol is maintained in solution and bile salts Its solubility is determined by the ratio of phospholipid plus pile salt concentration to that of cholesterol.[18]

Gallstone Disease Factor Related to It

Cholesterol

An absolutely vital steroid component of plasma membrane and lipoprotein is cholesterol. It is an essential element in the body during the first day of life. As the precursor of steroid hormones, bile acids and vitamin D3.[19]

Channel of cholesterol biosynthesis. Synthesis starts with acetyl-CoA from the mitochondrion moving to the cytosol. Experts advise a cholesterol level below 200 mg/dl for excellent health.[20] The rate-limiting step is at the 3-hydroxy-3-methylglutaryl-CoA (HMG-CoA) reductase, HMGR-catalyzed phase.

Triglycerides (TG)

Triglycerides, which have three long chains of fatty acids coupled to glycerol, make up most of dietary fat. Hydrolysis by pancreatic lipase breaks two of the fatty acid chains from the sites of the glycerol molecule, departing mono glycerol first in fat digestion. The second stage relies on the creation of micelles by the bile salts carrying the monoacylglycerol and long-chain fatty acids into the aqueous phase and consequently to the lipophilic surface of the enterocyte.[21]

Low-density lipoprotein (LDL)

With a high concentration of cholesterol and cholesterol esters, LDL particles serve primarily to provide cholesterol to the peripheral tissues (or send it back to the liver). Their triacylglycerol content is far lower than that of their VLDL ancestors. They bind to the cell–surface membrane to do this. Macrophage scavenger receptor Macrophages scavenge high levels of scavenger receptor activity chemically altered LDL.[22]

HDL, or high-density lipoprotein, These are the densest of the lipoprotein particles and the smallest ones. Though relatively little triglyceride, HDLs constitute a varied family of lipoproteins. They have a high protein content and almost equal levels of cholesterol and phospholipids. Direct secretion of HDL particles from the liver and gut into the circulation comes from HDL particles absorbing unesterified cholesterol, acting as a circulating reservoir of APO (the apolipoprotein transmitted to VLDL and chylomicron remnants).

MATERIALS AND METHODS

Subjects

Outside of hospitals, in labs, the study was often carried out. Research for this project began on February 10, 2022, and ended on March 1, 2023. Half of the subjects were patients and half were controls. Members of the research group's ages varied from 25 to 65. Both the experimental group and the control group were categorized.

Subjects

The dates of February 10, 2022, and March 1, 2023, were used for this prospective investigation, which took place in labs outside of hospitals. This randomized, controlled trial included 50 participants; all patients, including those with gallstones and those without the condition, were investigated. Their ages varied from twenty-five to sixty-five years old. Medical professionals made diagnoses for these patients.

Patients group

The participants in this study ranged in age from 25 to 65 and comprised both those who already had gallstones and 50 healthy individuals; the majority of the healthy individuals in this group reported no symptoms of gallstones. Nevertheless, while these are the most typical symptoms, everyone could have them in their own unique way. Possible symptoms include: intense upper abdominal discomfort that builds up quickly and last for 30 minutes to several hours, Shoulder blade to shoulder blade back pain, Symptoms of right shoulder pain, Feeling queasy, throwing up, running a temperature, shivers, jaundice (a skin or eye yellowing), gas in the abdomen, Intestinal gas, bloating, belching, and dietary intolerances are all possibilities.[23] Experts in the medical field conduct clinical evaluations and send patients with any of these symptoms to the lab for further testing.

Control group

The control group consists of people who were collected 50 from relatives who were free from signs and symptoms of gallstone.

Instruments and tools used in this study.
As shown in the following table (2-1)

Table (2-1): The instruments and tools used in this study.

Instrument	Company
BT 125	BİLİMSEL TIBBİ ÜRÜN (Turkey)
spectrophotometer	(England)
Plane tube	Afco(Jordan)
Gel tube	Hiprove(india)
EDTA	Afco(Jordan)
Syringe (5 ml)	ELVER (TURKEY)

Clinical assessment

Laboratory tests were performed for all patients using the best laboratory devices, after blood was drawn from the patient and sent to the laboratory for the necessary tests, including cholesterol, TG and lipid profile using the BT 125 device. All patients and the control group were evaluated clinically and hematology as follows.

History

A complete history was taken from gallstone patient regarding personal data such as name, age, are you married, number of birth is your menstrual cycle regular, do you have chronic diseases such as pressure, diabetes, etc, at any stage of disease, and any symptoms Others include vomiting, abdominal pain, etc.[23]

Blood Collection

A-Serum samples

Venous blood samples were taken for hyperthyroidism before any treatment from each patient, 5 ml of blood were drawn in tubes and CHO, TG and lipid profile with BT 125 instrument.

Blood Collection and Preparation

Using a sterile (21 g) plastic syringe, blood was drawn from the anterior vein. According to[24], prior to piercing, the skin was cleansed and sterilised using 70% ethyl alcohol. It was then let to dry. The lipid profile, TG, and CHO blood tests required five milliliters of blood. To make the serum, blood was placed in regular tubes that were treated with an anticoagulant so that it could not clot.[25] According to Lewis et al. (2006), every sample was assigned a unique serial number and labeled with the patient's name.

Biochemical assessment

Measurement of Cholesterol level (Test principle)

Cholesterol determination upon oxidation and enzymatic hydrolysis.[3,4] Quinoneimine is the colorimetric indicator; it is produced from 4-aminoantipyrine and phenol via a catalytic peroxidise process involving hydrogen peroxide.[3]

A cholesterol ester and water react to form cholesterol and fatty acid.

Cholesterol + O_2 …..CHE …> This is the chemical formula for cholesterol-3-one: 2 hydroxide ions + phenol + 4 aminoantipyrine...POD...> quinine + 4 H20.

The content of cholesterol in the sample is directly proportional to the intensity of the pinkish-red hue.

Measurement of TG level (Procedure's principle)

Hydrolysis of triglycerides by lipase enzyme yields glycerol and free fatty acids. Glycerol kinase (GK) and adenosine triphosphate (ATP) phosphorylate glycerol to form glycerol-3-phosphate and adenosine diphosphate (ADP). Hydrogen peroxide (H_2O_2) is produced when glycerol phosphate oxidase (GPO) oxidizes glycerol-3-phosphate to dihydroxyacetone phosphate (DAP). The peroxidase-catalyzed color reaction involves the interaction of H_2O_2 with 4-aminoantipyrine (4-AAP) and 4-chlorophenol (4-CP) to yield a red dye. The amount of triglyceride in the sample is directly related to the dye's absorption. Fossati et al.[4] and McGowan et al.[5] laid forth the reaction sequence that this analytical approach is built upon. Instead of 2-hydroxy-3,5-dichlorobenzenesulfonate, which was utilized in the research conducted by Fossati and McGowan, this reagent makes use of 4-chlorophenol.

Measurement of lipid profile level (Test Principle)

To assess total cholesterol, HDL cholesterol, and triglycerides, the SD LipidoCare System uses a combination of enzymatic methods and solid-phase technology. A complete blood sample taken from a capillary is placed onto an SD LipidoCare test strip. The next step is to insert the test strip into the SD LipidoCare Analyzer. The test strip has a special mechanism that separates the blood cells from the plasma. The response pads for total cholesterol, triglycerides, and HDL cholesterol receive a part of the sample that flows to the bottom layer of the test strip. Through an enzymatic process that results in a color shift in the test region of the strip, the LipidoCare analyzer analyzes TC, TG, and HDL. As the concentration of lipids in the sample increases, the color changes accordingly. The formula LDL=TC-HDL-(TG/5), which takes into account TC, HDL, and TG levels, is used to get the LDL value. Triglyceride levels of 400 mg/dL or less are required for the reporting of calculated LDL-cholesterol values; levels of 400 mg/dL or more do not warrant the reporting of calculated LDL-cholesterol values. By detecting a little electrical current that is the product of a chemical interaction between glucose in the blood and glucose oxidase on the glucose test strip, the SD LipidoCare Blood Glucose Test Systems are able to quantitatively quantify glucose in a capillary whole blood sample. The blood glucose level determines the quantity of current generated. A blood glucose number is displayed to the user from the resultant current.

Figure (2-1): Show the instrument BT 125 BİLİMSEL TIBBİ ÜRÜN (Turkey).

RESULTS AND DISCUSSION

Statistical Analysis

All values were expressed as means ± SD. The data were analyzed by using of computer SPSS program and taking p value < 0.05 as the lowest limit of significant and p value < 0.01 as highest limit of significant. The one-way ANOVA was used to examine the differences between different groups (Daniel, 1999).

Demographical data

Number of data

The number of pregnant patient in this study with signs of gallstone are (50) and control group without any signs of gallstone are (50) as shown in figure (4.1). N=50.

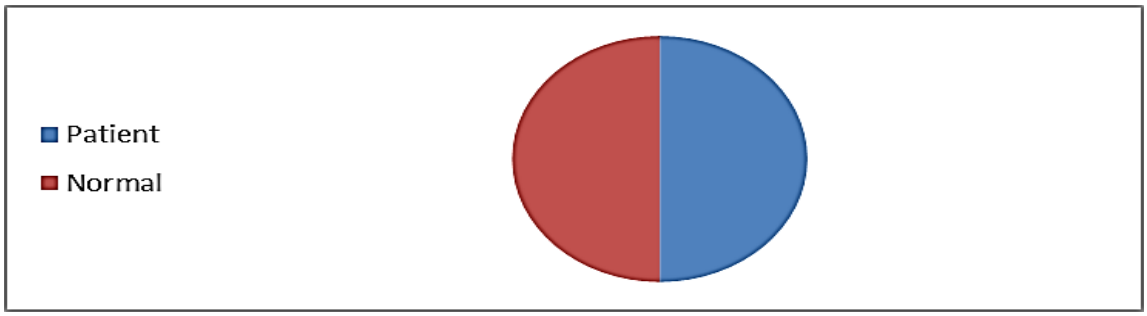


Figure (3.1): The number of patients group with gallstone and control group without any signs of gallstone.

Age

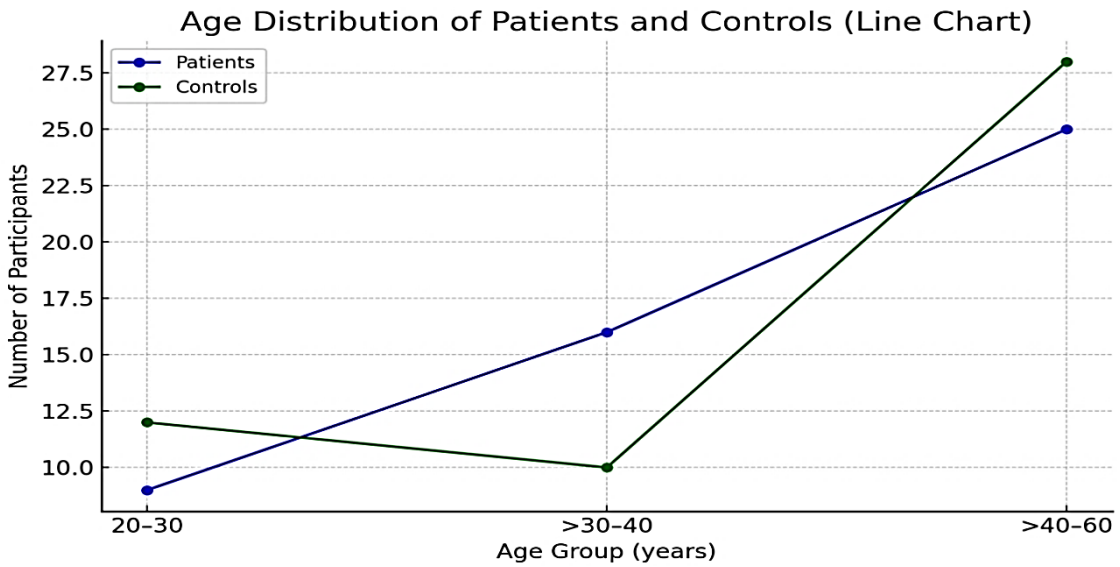
Table (3-1) and fig(3-1)shows the age group in the (patient and control group). These group recorded are highly percentage in the age (60 < - 40) other than other group as shown.

Table (3-1): Distribution of (patient and control) group with gallstone according to age. N =50

Age (year)	Case N =50	Number N =50	Percentage %
20 -30	Patient	9	(18%)
	Control	12	(24%)
> 30 -40	Patient	16	(32%)
	Control	10	(20%)
40 - < 60	Patient	25	(50%)
	Control	28	(56%)
P value	0.001**		

**highly significant difference at P<0.01

Table (3-1) Based on their age, the research categorizes hyperthyroidism patients into three groups. In the age bracket of 60–40, gallstones occur at a rate of 50%. The difference between the control group and the patient group is statistically significant (P<0.01). Other investigations have found similar findings.[26]



Fig(3-1): The age distribution shows most participants in the 40-60 group, with 50% of patients and 56% of controls. Fewer participants were in the 20-30 group, especially among patients (18% vs 24% controls). The difference is statically significant (p= 0.001) indicating age may influence group assignment.

The amount of cholesterol released into the bile by your body increases with age. You are more likely to develop gallstones if you have excess cholesterol. By the age of 60, gallstones can affect as many as 20% of women and 10% of men, making aging a major risk factor for this condition.

To keep yourself healthy as you become older, it's vital to get checkups regularly and pay attention to any pain or discomfort you may be experiencing. Age is inversely proportional to the pigment-to-cholesterol gallstone ratio. After the age of fifty, the bilirubin, phosphate, and carbonate content of stones increases slightly, but the cholesterol level declines progressively. Based on these findings, calcium carbonate, phosphate, or bilirubinate salts may dissolve cholesterol and substitute it as we age.[27]

Gender

Table (3-2) shows the gender group in the (patient and control group). These group recorded are highly percentage in the female other than male as shown.

Table 3-2: Distribution of (patient and control) group with gallstone according to gender.

Gender	Number N =50	Percentage %
Female	37	(74%)
Male	13	(26%)

Table (3-2) Display the gender-based classification of the gallstone patients included in the study. Among females, gallstones are more common (74%). This finding is consistent with previous research.[28]

Gallstones seem to manifest more frequently in women. Women have gallstones at a rate that is two to three times higher than men. Because estrogen raises biliary cholesterol levels, gallstones can form in the bile when estrogen levels are high. Cholesterol oversaturation in the gallbladder can occur when biliary cholesterol levels are high. In addition, being pregnant, taking hormonal birth control, or using hormone replacement therapy all raise the risk of gallstones, which can be a problem for women with high estrogen levels. Estrogen, a feminine hormone, can raise cholesterol levels in the bile, putting women at a higher risk of gallstone development. Another risk factor that increases the likelihood of gallstone development is obesity, which is particularly prevalent in women due to the impact of adipose tissue on estrogen production.[29]

Biochemical study

Cholesterol CHO

Table (3-3) shows the mean ± SD. Of CHO in the (patient and control group). These values of CHO recorded are highly significant p value (0.001) other than control group as shown.

Table (3-3): Comparison of CHO in patients with gallstone and control group.

Parameter	Group	mean ± SD.	P value
CHO (mmol/L)	Patients	6.397 ± 0.539	0.001**
	Control	3.87 ± 0.742	

**highly significant difference at P<0.01

The current medical consensus holds that gallstones form when the gallbladder's bile chemical composition becomes unbalanced. The majority of the time, stones are the result of excessive cholesterol in the bile. Cholesterol is a lipid that your body uses to make hormones, vitamins, and healthy cells; elevated amounts of this lipid are responsible for the formation of gallstones in around 80% of cases. Cholesterol crystals can aggregate to become gallstones when cholesterol levels are excessive or supersaturated. In addition to inflaming and infecting the gallbladder, gallstones can impede lipid digestion.

Triglyceride TG

Table (3-4) shows the mean ± SD. Of deficiency in TG in the (patient and control group). These values of TG recorded are highly significant p value (0.001) other than control group as shown.

Table (3-4): Comparison of TG level in patients with gallstone and control group.

Parameter	Group	mean ± SD.	P value
TG (mmol/L)	Patients	3.595 ± 0.082	0.001**
	Control	2.042 ± 0.197	

**highly significant difference at P<0.01

Cholesterol and triglycerides are crucial components of human metabolism. Muscles and adipose tissue are the primary locations for triglyceride storage and use. Cholesterol has an important role in the production of bile acids, vitamin D, and steroid hormones and in maintaining the fluidity of the plasma membrane. The blood carries triglycerides and cholesterol in the form of water-soluble lipoproteins. An increased risk of gallstone disease may be associated with elevated triglyceride levels, which may reduce cholecystokinin sensitivity, raise biliary cholesterol saturation and bile viscosity via higher mucin formation, and so on. However, there is a self-perpetuating cycle at work here: being overweight may cause the liver to secrete more cholesterol, which in turn may cause bile supersaturation and an increase in the amount of cholesterol secreted into the bloodstream. Obesity is associated with an increase in biliary cholesterol saturation, bile acid synthesis, turnover rates, and pool sizes; meanwhile, cholesterol production is linearly related to body fat.[30]

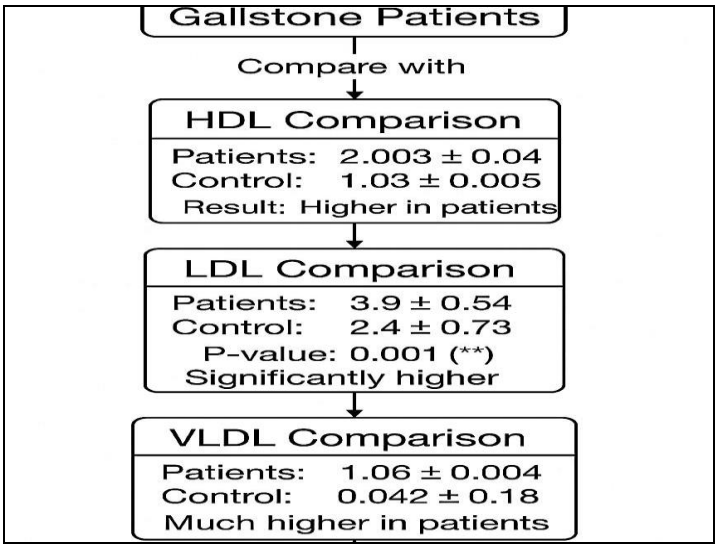
Lipid profile (HDL, LDL and VLDL)

Table (3-5),Fig (3-2) shows the mean ± SD. Of HDL, LDL and VLDL in the (patient and control group). These values of HDL, LDL and VLDL recorded are highly significant p value (0.001) other than control group as shown.

Table (3-5): Comparison of HDL, LDL and VLDL in patients with gallstone and control group. N= 50

Parameter	Group	mean ± SD.	P value
HDL (mmol/L)	Patients	2.003 ± 0.04	0.001**
	Control	1.03 ± 0.005	
LDL (mmol/L)	Patients	3.9 ± 0.54	
	Control	2.4 ± 0.73	
VLDL (mmol/L)	Patients	1.06 ± 0.004	
	Control	0.042 ± 0.18	

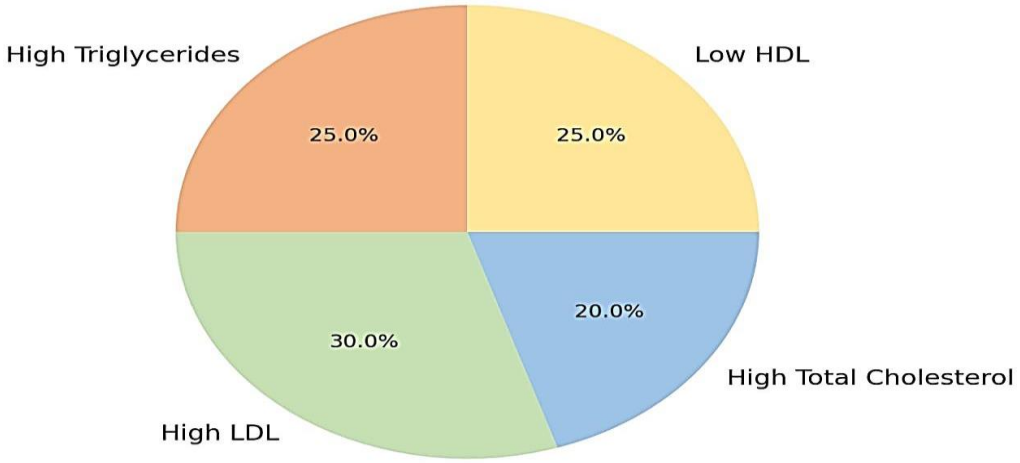
**highly significant difference at P<0.01



Fig(3-2): indicate relation between patients analysis results vs. control results as described in table above.

The hallmarks of hyperlipidemia include abnormally high concentrations of triglycerides, low-density lipoproteins (LDL), and total cholesterol in the bloodstream and abnormally low concentrations of high-density lipoprotein (HDL). Whether or whether hyperlipidemias are linked to gallstones is a matter of debate. Research has linked gallstones to hyperlipidemias, particularly hypertriglyceridemia and elevated LDL levels. Compared to the control group, patients in this research had substantially lower mean blood HDL values. These findings are consistent with those of the study by Batajoo H et al. (2013). However, there is conflicting evidence linking low HDL levels to gallstones. Studies have demonstrated a favorable link between low HDL cholesterol and gallstones; this is because HDL cholesterol is the primary source of biliary cholesterol. However, not all research has shown a link between low HDL and the development of gallstones.[31,32]

Although patients' mean blood LDL levels were lower than those of the control groups, this difference was not statistically significant. There was no statistically significant change in LDL concentration when compared to the control group, according to research by Al-Saadi N et al. 2012. These findings went against the grain of previous research, which had shown no link between gallstones and blood LDL levels or found a positive correlation. While some research has shown a link between high blood LDL levels and gallstones, other investigations have failed to find any such link. Nonetheless, compared to the control group, patients in this research had decreased blood LDL levels.[33]



Fig(3-3): Relative contribution of lipid factors to gallstone risk.

CONCLUSION

1. Increase level of cholesterol and triglyceride was the most indicator of patient with gallstones.
2. Decrease level of HDL, increase level of LDL and VLDL which is the major modulator of gallstone function in patient with gallstones.
3. In the age and gender group is increase level of lipid profile in older patient and occur in female other than male.

RECOMMENDATION

There is no clear way to prevent gallstone but you can reduce your risk by doing the following:

1. Eat three well-balanced meals daily
2. Maintain a normal weight, and get regular exercise (at least 30 minutes a day most days of the week).
3. Eating a high-fat diet
4. Eating a high-cholesterol diet
5. Eating a low-fiber diet
6. Having a family history of gallstones

CONFLICT OF INTEREST

Not applicable.

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