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ORIGINAL ARTICLE

Analysis of serum lipid profiles in patients with and without gallstones

[1]Naval Khurana, [2]Mukesh Kakkar

[1]Associate Professor, [2]Assistant Professor, Department of General Surgery, Rajshree Medical Research Institute, Bareilly, India

ABSTRACT:

Background: To study and analyse serum lipid profiles in patients with and without gallstones. **Materials & Methods:** 30 patients who met the inclusion criteria were included in the study. Additionally, a control group of 30 in patients with no personal or family history of gallstones was recruited for comparison purposes. Statistical analysis was performed using SPSS software. **Results:** A total of 60 subjects were included. The HDL level in both the groups was 30.5 and 42.06 and the p- value was 0.001. The mean triglyceride in case and control group was 202.42 and 174.84 with p- value 0.01. **Conclusion:** The serum triglyceride levels and serum HDL levels showed statistically significant differences in the gallstone patients when compared to the control group.

Keywords: Gall stones, Serum LDL, HDL.

Corresponding author: Mukesh Kakkar, Assistant Professor, Department of General Surgery, Rajshree Medical Research Institute, Bareilly, India

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INTRODUCTION

Gallstone disease is one of the most prevalent gastrointestinal diseases with worldwide distribution and with a substantial burden to health-care delivery system. [1,2] Gallstone disease is a chronic recurrent hepatobiliary disease, which may result from impaired metabolism of cholesterol, bilirubin and bile acid (BA), and is characterized by the formation of the gallstone in hepatic bile duct, common bile duct or gallbladder. [3] Many studies have shown an association between gallstones and abnormal lipids. Gallstones (GS) are formed in the gall bladder and biliary tract and are of two types: namely cholesterol and pigment stones.[4] GS is one of the main causes for number of upper gastrointestinal surgical casualties. [5] Despite the wider exploration into the aetiopathogenesis of GS, knowledge on exact pathogenesis of GS is yet incomplete. Complex interaction of multiple environmental risk factors with the genetic risk factor is the most possible explanation given for this incomplete understanding. Biliary cholesterol supersaturation is identified as a main prerequisite for the formation of cholesterol GS and elevated unconjugated bilirubin in bile is considered as the primary cause for the pigment GS.[6,7] Thus GS are formed as a result of impaired metabolic regulation of human body. Biliary cholesterol hypersecretion is the main cause for biliary cholesterol supersaturation and bile stasis also plays an additional role.[8] Impaired lipid homeostasis can give rise to cholesterol hypersecretion from biliary canaliculi. Therefore high incidence of cholesterol GS compared to pigment GS can be expected in patients with impaired lipid homeostasis. Cholesterol is insoluble in water, it is secreted from the canalicular membrane in unilamellar phospholipid vesicle. Cholesterol solubility in the bile requires sufficient bile salts and phospholipids, predominantly phosphatidyl choline. If there is an excess of cholesterol or reduced phospholipids and/or bile acid, multi lamellar vesicles are formed causing nucleation of the cholesterol crystals which leads to the stone formation. The secretion of cholesterol supersaturated lithogenic bile, decreased concentration of phospholipids, gallbladder dysmotility, delayed large bowel transit times (favoring reabsorption of deoxy-cholic acid), and the resection of ileum (depleting the acid pool) have all been implicated in the gallstone formation. [9] Hyperlipidemia is a condition that incorporates various genetic and acquired disorders that describe elevated lipid levels within the human body. Hyperlipidemia is extremely common, especially in the Western hemisphere, but also throughout the world. Alternatively, a more objective definition describes hyperlipidemia as low-density lipoprotein (LDL), total cholesterol, triglyceride levels, or lipoprotein levels greater than the 90th percentile in comparison to the general population, or an HDL level less than the 10th percentile when compared to the general population. [10] Lipids typically include cholesterol levels, lipoproteins, chylomicrons, VLDL, LDL, apolipoproteins, and HDL. [11] Hence, this study was conducted to analyse serum lipid profiles in patients with and without gallstones.

MATERIALS & METHODS

The present study was conducted to analyse serum lipid profiles in patients with and without gallstones.30 patients who met the inclusion criteria were included in the study. Additionally, a control group of 30 inpatients with no personal or family

history of gallstones was recruited for comparison purposes.This study included male and female patients with gallstone disease, aged between 25 to 65 years. Gallstones were diagnosed using ultrasound, where they were identified as mobile echoes in the gallbladder with acoustic shadows.The data were presented as mean, and a Student's t-test was employed to compare the results between the patient and control groups ($p < 0.05$ was considered statistically significant). Statistical analysis was performed using SPSS software.

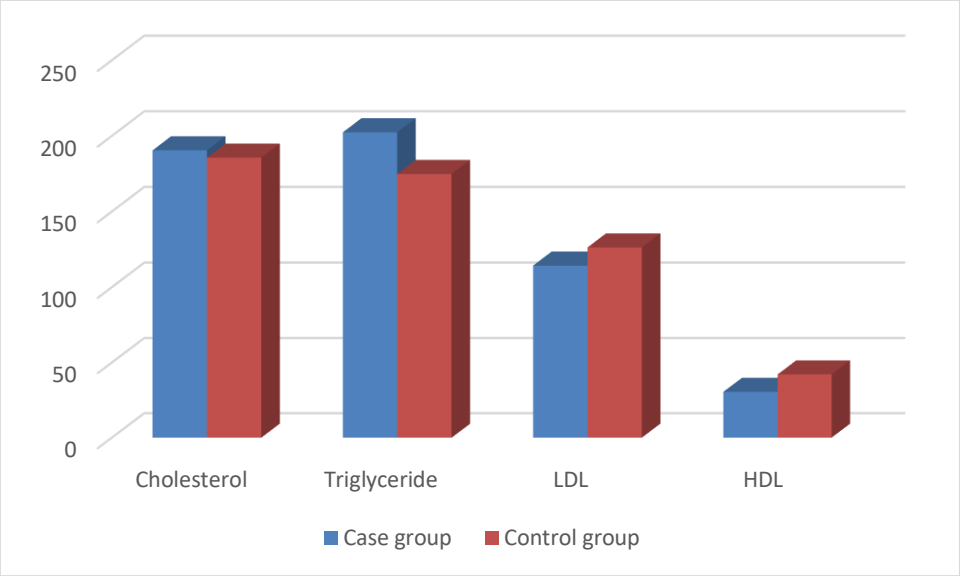
RESULTS

A total of 60 subjects were included. They were divided into two groups as case and control group. The case group depicted the mean cholesterol level as 190.5 and control group as 185.64. The HDL level in both the groups was 30.5 and 42.06 and the p- value was 0.001. The mean triglyceride in case and control group was 202.42 and 174.84 with p- value 0.01.

Table 1: Means of lipid profile in disease and controls.

Profile	Case group	Control group	P –value
Cholesterol	190.5	185.64	0.62
Triglyceride	202.42	174.84	0.010 (Significant)
LDL	114.26	126.27	0.74
HDL	30.5	42.06	0.001 (Significant)

Graph 1: Means of lipid profile in disease and controls.



DISCUSSION

Cholesterol is a lipid. Its major synthesis site is the liver, and its only excretion site is the biliary system. [12] The pathogenesis of cholesterol gallstones is multifactorial and complex. The known factors associated with cholesterol gallstones include cholesterol hypersecretion and supersaturation, bile salt and phospholipid concentrations, crystal nucleation, gallbladder dysmotility and gallbladder absorption and secretion functions. [12,13] Low-density lipoproteins (LDLs) and high-density lipoproteins (HDLs) are plasma lipids, and their main function is to transport cholesterol. LDL transports cholesterol from the liver to the peripheral tissues, and HDL transports cholesterol from the peripheral tissues to the liver.[12]Hence, this study was conducted to analyse serum lipid profiles in patients with and without gallstones. In the present study, a total of 60 subjects were included. They were divided into two groups as case and control group. The case group depicted the mean cholesterol level as 190.5 and control group as 185.64. A study by Hayat S et al, The mean age of the patients was 40.90 years and that of controls was 34.74 years. 46 patients were females and 44 controls were females. The serum cholesterol levels were high in the patients as compared to the controls but the comparison was not statistically significant. Serum triglycerides levels were high in the patients as compared to the controls and the analysis was statistically significant. Furthermore, the serum HDL levels were low in the patients as compared to the controls with a statistically significant p-value. However, the serum LDL levels were low in the patients as compared to the control group. Serum triglyceride levels and serum HDL levels were statistically significant in gallstone patients and there was a positive correlation between these parameters and gallstone disease.[14] In the present study, the HDL level in both the groups was 30.5 and 42.06 and the p-value was 0.001. The mean triglyceride in case and control group was 202.42 and 174.84 with p- value 0.01. Another study by Gill GS et al, the study was

conducted on 50 patients with gallstones and 30 healthy volunteers for comparison of lipid levels. Subsequently, cholecystectomy was conducted on patients with gallstones and pre- and post-operative lipid levels were compared. There was a significant decrease in total cholesterol, and triglycerides levels and increase in high-density lipoprotein levels after 1 month of surgery, while low-density lipoprotein levels and very low-density lipoprotein were not statistically changed.[15]The prevalence of GS depends on the gender where females have a higher incidence.[16] Multiple reasons have been evaluated in the identification of the cause for this gender difference. However a possible effect of serum lipids causing high incidence of GS among females was not identified in this study. Interestingly female patients in the study group had high serum HDL-C than that of male patients. The favorable effects of female reproductive hormones on serum lipids can be considered as a possible reason for such observation, as mean age of females in the study group was below the menopausal age which is considered as 51 years for Sinhalese.[17] However, a high risk of GS among patients with low HDL-C has been observed. [18]The mean serum cholesterol levels were high in the patient as compared to the control group however, the correlation of high cholesterol levels and gallstones was not statistically significant. Similarly, Channa NA et al. [19] and described that the serum cholesterol levels were not statistically significant in the gallstones patients as compared to the control group. Similar results were also demonstrated by Öner C et al. [20] in their study. In contrast to the above results, Al-Saadi N et al. [21] found that the serum cholesterol levels were significantly elevated in the gallstones patients as compared to the control group. Although the saturation of bile with cholesterol has definite role in pathogenesis of gallstones but association of gallstones and high level of serum cholesterol levels in patients is controversial in literature and can be explained by multiple factors like genetics, geographical, social and dietary habits in pathogenesis of different type of gallstones.

CONCLUSION

The serum triglyceride levels and serum HDL levels showed a positive correlation between these parameters and the occurrence of gallstone disease.

REFERENCES

1. Sandler RS, Everhart JE, Donowitz M, Adams E, Cronin K, Goodman C, et al. The burden of selected digestive diseases in the United States. Gastroenterology. 2002;122:1500–11.
2. Aerts R, Penninckx F. The burden of gallstone disease in Europe. Aliment PharmacolTher. 2003;18(Suppl 3):49–53.
3. Belousov Yu V. Up-to-date guide. Moscow: Exma; 2006. Pediatric gastroenterology; p. 112
4. Trotman BW, Soloway RD. Pigment gallstone disease: summary of the national institutes of health-international workshop. Hepatology. 1982;2:879–884.
5. Ahmed A, Cheung RC, Keeffe EB. Management of gallstones and their complications. Am Fam Physician. 2000;61:1673–1680.
6. Wang DQ, Carey MC. Complete mapping of crystallization pathways during cholesterol precipitation from model bile: influence of physical-chemical variables of pathophysiologic relevance and identification of a stable liquid crystalline state in cold, dilute and hydrophilic bile salt-containing systems. J Lipid Res. 1996;37:606–630.
7. Vítek L, Carey MC. Enterohepatic cycling of bilirubin as a cause of 'black' pigment gallstones in adult life. Eur J Clin Invest. 2003;33:799–810.
8. Wang DQ, Cohen DE, Carey MC. Biliary lipids and cholesterol gallstone disease. J Lipid Res. 2009;50(Suppl):406–411.
9. Einarsson K., Hellström K., Kallner M. Gallbladder disease in hyperlipoproteinaemia. Lancet. 1975;305(7905):484–487.
10. Fredrickson DS. An international classification of hyperlipidemias and hyperlipoproteinemias. Ann Intern Med. 1971 Sep;75(3):471-2.
11. Ballantyne CM, Grundy SM, Oberman A, Kreisberg RA, Havel RJ, Frost PH, Haffner SM. Hyperlipidemia: diagnostic and therapeutic perspectives. J Clin Endocrinol Metab. 2000 Jun;85(6):2089-112.
12. Atamanalp RS. Is there a relationship between the serum cholesterol level and the biochemical structure of gallstone in patients with cholelithiasis. IB Extended Essay. 2012:1–20.
13. Van Erpecum KJ. Pathogenesis of cholesterol and pigment gallstones: An update. Clin Res Hepatol Gastroenterol. 2011;35(4):281–287.
14. Hayat S, Hassan Z, Changazi SH, Zahra A, Noman M, Zain UlAbdin M, Javed H, Ans AH. Comparative analysis of serum lipid profiles in patients with and without gallstones: A prospective cross-sectional study. Ann Med Surg (Lond). 2019 Apr 24;42:11-13
15. Gill GS, Gupta K. Pre- and Post-operative Comparative Analysis of Serum Lipid Profile in Patients with Cholelithiasis. Int J Appl Basic Med Res. 2017 Jul-Sep;7(3):186-188.
16. Shaffer EA. Epidemiology and risk factors for gallstone disease: has the paradigm changed in the 21st century? Curr Gastroenterol Rep. 2005;7:132–140.
17. Jayasekara R. Factors influencing the age at natural menopause in Sinhalese women. Ceylon Med J. 1994;37:23–27
18. Thijs C, Knipschild P, Brombacher P. Serum lipids and gallstones: a case–control study. Gastroenterology. 1990;99:843–849.
19. Channa N.A., Ghanghro A.B., Soomro A.M. Quantitative analysis of serum lipid profile in gallstone patients and controls. Pakistan J. Anal. Environ. Chem. 2010;11(1):59–65.
20. Öner C., Güneri M.C. Lipid profiles of patients with gallstones. Turk. J. Family Pract. 2012;22:123–126.
21. Al-Saadi N., Al-Ardhi S. Biochemical and demographical study of lipid profile in sera of patients with gallstone. Iraqi J. Sci. 2012;53:2.