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"COMPARATIVE ANALYSIS OF SERUM LIPID PROFILES IN PATIENTS WITH CHOLELITHIASIS AND NON-CHOLELITHIASIS CONTROLS."

General Surgery

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ABSTRACT

Background: Cholelithiasis is a common hepatobiliary disorder influenced by multiple metabolic factors, including lipid abnormalities. Dyslipidemia contributes to bile cholesterol supersaturation, promoting gallstone formation. This study aimed to compare serum lipid profiles between patients with cholelithiasis and non-cholelithiasis controls to evaluate their association. **Methods:** A hospital-based cross-sectional case-control study was conducted at RNT Medical College and Associated Hospitals, Udaipur. A total of 80 subjects were included-40 patients with ultrasonographically confirmed cholelithiasis and 40 age- and sex-matched controls without gallstones. Fasting serum samples were analyzed for total cholesterol, triglycerides, HDL, LDL, and VLDL using enzymatic methods. Data were analyzed using SPSS v25.0, and $p < 0.05$ was considered significant. **Results:** Patients with cholelithiasis exhibited significantly higher mean serum cholesterol (162.59 ± 47.06 mg/dL, $p = 0.014$) and LDL (96.52 ± 37.42 mg/dL, $p = 0.008$), and significantly lower HDL (35.68 ± 15.16 mg/dL, $p = 0.022$) compared to controls. Triglycerides and VLDL levels were higher among cases but not statistically significant. The LDL/HDL ratio was markedly elevated in cholelithiasis patients ($p = 0.012$), indicating a more atherogenic lipid profile. **Conclusions:** The study demonstrates a strong association between dyslipidemia and cholelithiasis, suggesting that altered lipid metabolism-particularly high LDL and low HDL-plays a significant role in gallstone pathogenesis. Routine lipid profiling in at-risk individuals may facilitate early detection and prevention.These findings align with recent studies highlighting the metabolic link between dyslipidemia and gallstone disease

KEYWORDS

Cholelithiasis; Gallstone disease; Dyslipidemia; Serum lipid profile; Total cholesterol; Low-density lipoprotein (LDL); High-density lipoprotein (HDL)

INTRODUCTION

Cholelithiasis, or gallstone disease, is a prevalent gastrointestinal disorder characterized by the formation of calculi within the gallbladder or biliary tract. It affects approximately 10–15% of the global adult population and represents a major cause of morbidity, particularly among women and older adults.(1) Gallstone formation is a multifactorial process involving genetic, metabolic, and environmental influences that disrupt the delicate balance of bile composition. Among these factors, disturbances in lipid metabolism have been increasingly recognized as central to gallstone pathogenesis. Since cholesterol constitutes the primary component of most gallstones, evaluating serum lipid profiles can provide critical insight into the metabolic derangements associated with the disease.(2)

The pathophysiology of cholelithiasis involves supersaturation of bile with cholesterol, nucleation of cholesterol monohydrate crystals, and impaired gallbladder motility.(3) Abnormalities in serum lipid levels-particularly elevated low-density lipoprotein (LDL), total cholesterol, and triglycerides, coupled with decreased high-density lipoprotein (HDL) may contribute to this supersaturation. Several studies have demonstrated a strong relationship between dyslipidemia and the risk of gallstone formation.(4) For instance, a multi-center study by Zhang et al. (2022) revealed significantly higher serum total cholesterol, LDL, and triglycerides, and lower HDL levels in gallstone disease patients compared to controls.(5) Similarly, Nagappa and Bhandarkar (2025) reported a significant association between gallstone disease and hyperlipidemia (particularly elevated total cholesterol), emphasizing the potential of lipid profile alterations as risk factors and predictive markers for disease prevention.(6)

Epidemiological research has also highlighted the interplay between gender and lipid metabolism in gallstone formation. Females, particularly those exposed to estrogenic influences such as pregnancy or oral contraceptive use, exhibit increased cholesterol saturation of bile and are therefore more predisposed to gallstone formation. (7) Age, obesity, sedentary lifestyle, and high-fat diets are additional contributory factors that modulate serum lipid concentrations and gallstone risk. A recent Mendelian randomization study further established a causal relationship between elevated low-density lipoprotein cholesterol (LDL-C), apolipoprotein B, and cholelithiasis, underscoring the metabolic underpinnings of this disease.(8)

Despite these findings, inconsistencies remain in the literature. Some studies, such as Florettira et al(2022) have reported normal lipid profiles among gallstone patients, suggesting that local dietary patterns, genetic predispositions, and sample variability may influence results.(9) Thus, regional investigations are necessary to elucidate population-specific lipid abnormalities and their role in gallstone disease. Given this context, the present study aims to perform a comparative analysis of serum lipid profiles-including total cholesterol, triglycerides, HDL, LDL, and VLDL-in patients with cholelithiasis and non-cholelithiasis controls. By identifying statistically significant lipid alterations associated with gallstone disease, this study seeks to enhance understanding of the metabolic mechanisms contributing to cholelithiasis and support the role of lipid monitoring in preventive and therapeutic strategies.

METHODS

This was a hospital-based cross-sectional case-control study conducted in the Department of General Surgery, RNT Medical College and Associated Group of Hospitals, Udaipur, Rajasthan.

Study Population

A total of 80 subjects were included, comprising 40 patients with cholelithiasis confirmed by ultrasonography and 40 age- and sex-matched controls without gallstones.

Inclusion Criteria

- Patients aged 18–75 years with ultrasonographically proven cholelithiasis.
- Both male and female patients willing to give informed consent.

Exclusion Criteria

- Patients with diabetes mellitus, nephrotic syndrome, hypothyroidism, liver diseases, or pancreatitis.
- Those on lipid-lowering therapy or with chronic infections, inflammatory disorders, or pregnancy.

Data Collection

Detailed demographic data, clinical history, and relevant investigations were recorded using a standardized proforma. Fasting

venous blood samples (after 10–12 hours of overnight fasting) were collected for biochemical analysis.

Biochemical Analysis

Serum lipid profile parameters-Total Cholesterol, Triglycerides, HDL, LDL, and VLDL-were analyzed in the Clinical Biochemistry Laboratory, RNT Medical College, Udaipur using automated analyzers and enzymatic methods. LDL was calculated using Friedewald's formula.

Statistical Analysis

Data were analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± SD and compared using the independent t-test. Categorical variables were compared using the Chi-square test. A p-value < 0.05 was considered statistically significant.

RESULTS

Table 1. Comparative Analysis Of Demographic Characteristics Between Cholelithiasis And Non-cholelithiasis Groups

Parameters	Cholelithiasis (n = 40)	Non-cholelithiasis (n = 40)	p-value
Age (years)	53.23 ± 15.65	50.28 ± 15.66	0.402
Gender			
Male	21 (52.5%)	11 (27.5%)	0.022*
Female	19 (47.5%)	29 (72.5%)	

The mean age difference between the two groups was not statistically significant (p = 0.402). However, a significantly higher proportion of males was observed among the cholelithiasis patients compared to the control group (p = 0.022).

Table 2. Comparison Of Lipid Profile Parameters Between Cholelithiasis And Control Groups

Lipid Parameters	Cholelithiasis (Mean ± SD)	Controls (Mean ± SD)	p-value
Serum Cholesterol (mg/dL)	162.59 ± 47.06	135.72 ± 48.41	0.014*
Triglycerides (mg/dL)	148.69 ± 75.63	126.84 ± 74.44	0.197
HDL (mg/dL)	35.68 ± 15.16	45.73 ± 22.67	0.022*
LDL (mg/dL)	96.52 ± 37.42	75.87 ± 29.81	0.008*
VLDL (mg/dL)	29.85 ± 15.17	28.89 ± 13.32	0.766
Cholesterol/HDL Ratio	5.31 ± 3.04	4.17 ± 3.83	0.143
Triglycerides/HDL Ratio	5.50 ± 5.37	4.74 ± 6.24	0.561
LDL/HDL Ratio	3.02 ± 1.43	2.18 ± 1.51	0.012*

Significant at p < 0.05

DISCUSSION

In the present study, the serum lipid profile of patients with cholelithiasis was compared to that of age- and sex-matched controls without gallstones. The findings demonstrated significant alterations in lipid metabolism among gallstone patients, indicating a clear association between dyslipidemia and cholelithiasis.

This study revealed that patients with cholelithiasis had significantly elevated serum cholesterol and LDL levels with decreased HDL levels compared to non-cholelithiasis controls, indicating that dyslipidemia contributes to gallstone formation by promoting cholesterol supersaturation of bile. Similar findings have been widely reported across multiple studies, confirming that abnormalities in lipid metabolism are closely linked with the pathogenesis of gallstone disease.(10)

In our study, total cholesterol and LDL were significantly higher among cholelithiasis patients, while HDL was markedly reduced. This pattern agrees with the work of Sethulakshmi et al. (2021), who reported that 51.2% of gallstone patients exhibited dyslipidemia, with high LDL and triglycerides being the most prevalent abnormalities.(11)

Similarly, Jabeen et al. (2022) found a significant difference in HDL and LDL levels between gallstone and control groups, supporting the conclusion that lipid disturbances predispose individuals to cholesterol gallstone formation.(12)

Our results are therefore consistent with international evidence showing that hypercholesterolemia and high LDL are major biochemical risk factors for gallstone disease. Our study also observed lower HDL among patients, implying impaired reverse cholesterol transport. This finding is reinforced by Alam et al. (2021), who reported that reduced HDL levels significantly increase the risk of gallstone formation, particularly in women.(13) Similarly, a study by Hendarto et al. (2023) found that 90.9% of gallstone patients had high LDL and 18.2% had low HDL, demonstrating the same atherogenic lipid pattern as our findings.(10) This means that the reduction in HDL observed in our study aligns well with the global evidence indicating that HDL plays a protective role by maintaining cholesterol homeostasis in bile. Our study also noted that the LDL/HDL ratio was significantly higher among cholelithiasis patients, suggesting a pro-atherogenic and lithogenic state. One more study conducted by Malik et al. (2011) showed that lipid abnormalities, particularly elevated LDL and cholesterol levels, normalize following cholecystectomy, which supports the idea that gallstone disease is metabolically linked with dyslipidemia.(14)

Together, these findings suggest that the lipid changes seen in gallstone patients are not merely coincidental but represent part of a systemic metabolic imbalance that contributes to gallstone formation.

The strengths of our study include a well-defined case-control design, matched controls, and standardized laboratory procedures, which minimized analytical bias. However, the relatively small sample size and cross-sectional nature limit the ability to establish causation. Dietary patterns, BMI, and hormonal influences were not analyzed, which could have provided deeper metabolic insight. An unexpected finding was the male predominance in our cholelithiasis group, differing from prior reports that found higher incidence in females. This discrepancy may reflect regional dietary factors or lifestyle differences in the RNT Medical College population.

This study was conducted to test the hypothesis that dyslipidemia contributes to gallstone formation by altering bile composition through changes in serum cholesterol and lipoprotein levels. The purpose was to compare the lipid profile between cholelithiasis patients and controls and identify lipid abnormalities that predispose to gallstone formation. Our findings have clinical significance as they highlight the potential of serum lipid profiling as a preventive screening tool for gallstone disease. Routine lipid evaluation in patients at risk could facilitate early intervention. Future research should include longitudinal studies assessing the effect of lipid-lowering therapy on gallstone prevention, as suggested by recent evidence linking dyslipidemia control with reduced gallstone recurrence.

CONCLUSION

This study concludes that patients with cholelithiasis exhibit significantly higher serum cholesterol and LDL levels and lower HDL levels compared to non-cholelithiasis individuals. The altered lipid profile suggests a metabolic predisposition to gallstone formation. Monitoring and management of dyslipidemia could play an important role in the prevention and overall management of gallstone disease.

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