Original Article

IAHS Medical Journal
Volume 08 Issue 02 December 2025; 49-54

Linking Fatty Liver, Liver Function Abnormalities and Calculus Cholecystitis: A Hospital-Based Study

A.M.O. Ibrahim Shamsi[1] Sheikh Firoj Kabir[2] Syeda Tashfia Jahan[3]
Md. Toufiqul Islam[4] Mohammad Nurunnabi[5*]

ABSTRACT

Background: *Calculus cholecystitis is a prevalent hepatobiliary condition treated by cholecystectomy. Evidence suggests an association with fatty liver disease and their co-existence may worsen outcomes and increase healthcare burdens. This study aimed to assess the association between calculus cholecystitis, abnormal liver function tests and fatty liver disease.*

Material and methods: *A hospital-based, observational cross-sectional study was conducted in the Department of Surgery, Medical College for Women and Hospital, Dhaka. A total of 50 patients diagnosed with calculus cholecystitis were enrolled after obtaining informed written consent. Data on socio-demographics, clinical features and laboratory findings were collected using a structured questionnaire. Hepatobiliary ultrasonography was performed to detect gallstones and fatty liver.*

Results: *The mean age of participants was 39±14.2 years, with the majority (56%) aged between 30 and 50 years. A female predominance was observed (54%). Liver function tests revealed mean values for total bilirubin, AST, ALT and ALP as 0.74±0.56 mg/dL, 33.1±14.3 U/L, 34.9±14.3 U/L and 115.3±91 U/L, respectively. Lipid profile assessment showed mean triglyceride and total cholesterol levels of 144.7±99.7 mg/dL and 187.2±44.1 mg/dL, respectively. Fatty liver was detected in 19 (38%) of the 50 patients with calculus cholecystitis. Those with fatty liver exhibited significantly higher AST (40.7±5.41 vs. 36.3±6.7 U/L) and ALT (41.7±4.37 vs. 37.1±6.54 U/L) levels compared to those without fatty liver ($p < 0.05$).*

Conclusion: *The study found a substantial relation between calculus cholecystitis and fatty liver, marked by elevated cholesterol and liver enzymes. This suggests fatty liver may worsen liver function in gallstone patients, highlighting the need for further research and integrated management.*

Key words: Calculus cholecystitis; Fatty liver; LFT.

Introduction

Gallstone Disease (GSD) or calculus cholecystitis is a prevalent hepatobiliary disorder with significant public health implications. It is one of the most common causes of abdominal pain requiring hospitalization and frequently necessitates cholecystectomy as a definitive treatment.[1] Gallstones form due to the precipitation of calcium, bilirubin, cholesterol, mucous and proteins within the hepatic bile duct, common bile duct or gallbladder. While many patients remain asymptomatic, gallstone disease can lead to severe complications such as pancreatitis and necrosis, contributing to its clinical and economic burden.[2,3] The global prevalence of GSD is estimated at 18.5% of the world's population, with a yearly incidence of 3-22%.[4,5] The prevalence rates in Asia range from 3% to 15%.[6,7] In Bangladesh, asymptomatic GSD prevalence is reported at 6%, while neighboring Nepal reports higher rates (11.76%) reflecting regional variability influenced by genetic, dietary and socio-economic factors.[8,9]

Several risk factors contribute to gallstone formation, including age, sex, genetics, obesity and dietary habits.[8] Globally, women face twice the risk of men due to estrogen's role in cholesterol secretion.[10] A regional study from Bangladesh reported that 77% of gallstone patients were female, with a higher prevalence among housewives and individuals under 40.[8] Similarly, a Nepalese study found a male-to-female ratio of 1:1.98, peaking in ages 45–60.[9] Emerging Asian data reveal unique regional risk factors for gallstone disease. In Bangladesh, rates are rising among the middle class,

1. Registrar of Surgery
Medical College for Women and Hospital, Dhaka.
2. Professor of Surgery
Medical College for Women and Hospital, Dhaka.
3. Post Graduate of Reproductive Endocrinology & Infertility, Bangladesh Medical University, Dhaka.
4. Registrar of Hepatobiliary & Pancreatic Surgery
BRB Hospital, Dhaka.
5. Assistant Professor of Community Medicine and Public Health
Sylhet Women's Medical College, Sylhet.

*Correspondence : **Dr. Mohammad Nurunnabi**
Cell : +88 01717 87 05 59
Email : nur.somch@gmail.com

Date of Submission : 21st July 2025
Date of Acceptance : 10th November 2025

IAHS Medical Journal
Volume 08 Issue 02 December 2025; 49-54

while in Nepal, non-vegetarian diets and obesity account for 51% of cases.[8,10] Korean studies note shifting trends: gallbladder stones now dominate over bile duct stones, correlating with rising socio-economic status.[11] Other risk factors for gallstone disease include multiple pregnancies, oral contraceptive use, smoking and alcohol consumption.[12] Gallstones are classified as cholesterol, pigment (Brown/black) or mixed stones. Metabolic syndrome factors obesity, dyslipidemia and insulin resistance play key roles in gallstone disease, with cholesterol supersaturation from obesity and high-calorie diets driving stone formation.[13,14] Gallstone formation, particularly cholesterol stones, occurs when bile becomes supersaturated with cholesterol or has low bile acid levels, causing unstable phospholipid vesicles and cholesterol crystals to form factors worsened by obesity, high-calorie diets, and medications like oral contraceptives.[15]

Non-Alcoholic Fatty Liver Disease (NAFLD) is a key metabolic syndrome-related liver condition with rising global prevalence. It ranges from simple steatosis to cirrhosis and shares risk factors with gallstone disease, such as obesity, diabetes and insulin resistance. Insulin resistance in NAFLD disrupts cholesterol metabolism and bile acid synthesis, promoting gallstone formation, while chronic inflammation may impair gallbladder motility.[16] Conversely, cholecystectomy often performed for GSD may exacerbate hepatic steatosis by disrupting enterohepatic bile acid circulation. The hepatic manifestation of metabolic syndrome, NAFLD, has been implicated in various extra-hepatic complications, including cardiovascular diseases and type 2 diabetes mellitus.[17] The diagnosis of NAFLD is typically based on abnormal liver function tests, imaging studies, and, in some cases, liver biopsy.[18] Abnormal LFTs, particularly elevated Gamma-Glutamyl Transferase (GGT) and Alkaline Phosphatase (ALP) are common in both GSD and NAFLD. Cholestasis from gallstone obstruction elevates ALP, while NAFLD-driven hepatocellular injury raises Alanine Transaminase (ALT).[8,9] Among the imaging modalities, ultrasonography remains the most widely used technique for screening fatty liver disease in the general population.

Despite the overlapping risk factors of gallstone disease and NAFLD, evidence remains conflicting.[2,3,19,20] Some studies report NAFLD doubling GSD risk, while others find no independent association after adjusting for metabolic confounders.[2,3,19,21,22,23] This inconsistency underscores the need for population-specific analyses, particularly in regions like South Asia, where genetic and environmental factors diverge from Western cohorts.Given the high prevalence of both conditions, their potential co-existence could have significant implications for patient outcomes and healthcare resource utilization. Understanding the correlation between fatty liver, abnormal liver function and calculus cholecystitis could provide insights into their pathophysiology, aid in early detection and inform targeted interventions for high-risk populations, ultimately reducing disease burden and improving patient care in resource-limited settings in Bangladesh.

Materials and methods

This hospital-based, observational cross-sectional study was conducted over twelve months in the Department of Surgery at the Medical College for Women and Hospital, Dhaka. Patients aged 20–60 years, of any sex, diagnosed with calculus cholecystitis and classified as ASA physical status I or II were invited to participate. Those with a history of ERCP, bile duct disorders or other systemic comorbidities were excluded. Simple random sampling was used. Based on an assumed 50% prevalence of abnormal liver function tests and fatty liver, a 95% confidence level and a 5% margin of error, the calculated sample size was 384. However, due to COVID-19 restrictions and limited time, only 50 patients were enrolled.

Eligible participants were briefed on the study's objectives, procedures and anticipated outcomes by the research team. After providing verbal agreement, each participant signed informed consent before any data were collected. Data collection comprised a structured, face-to-face interview using a semi-structured questionnaire covering socio-demographic information (Age, sex, residence, socioeconomic status, occupation) and clinical parameters (BMI, blood pressure, liver function tests, comorbidities and hepatobiliary ultrasound findings). Calculus cholecystitis was diagnosed ultrasonographically by identifying mobile, echogenic gallstones with acoustic shadows, while fatty liver was defined by increased hepatic brightness and diffuse parenchymal echogenicity. All demographic, sonographic, and laboratory data were recorded systematically.

Data were analyzed using SPSS version 24. Continuous variables were presented as mean ± standard deviation, and categorical variables as frequencies and percentages. The Student's t-test assessed continuous data, while the chi-square test compared categorical data. A p-value <0.05 was deemed statistically significant.

Ethical approval was obtained from the Ethical Review Committee (ERC) of the Medical College for Women and Hospital, Dhaka. Data confidentiality was strictly maintained. Written informed consent was taken after explaining the study. No financial incentives were provided.

IAHS Medical Journal
Volume 08 Issue 02 December 2025; 49-54

Results

Table I Demographic and clinical profile of the participants (n=50)

Variables	n(%)
Age (Years)	
Mean±SD	39±14.2
Age group (Years)	
<30	13 (26)
30-50	28 (56)
>50	9 (18)
Gender	
Male	23 (46)
Female	27 (54)
Residence	
Rural	32 (64)
Urban	18 (36)
Education	
Illiterate	4 (8)
Below SSC	5 (10)
SSC	19 (38)
HSC	14 (28)
Graduate and above	8 (16)
Monthly income (BDT)	
<10000	16 (32)
10000-15000	19 (38)
15001-20000	13 (26)
>20000	2 (4)
BMI (kg/m^2)	
Mean±SD	24±4.54
Normal	17 (34)
Overweight	23 (46)
Obese	10 (20)
Comorbidities	
Diabetes	28 (56)
Hypertension	30 (60)
Dyslipidemia	34 (68)

In the Table I, this study included 50 patients with calculus cholecystitis, with a mean age of 39 ± 14.2 years. Most were aged 30–50 years (56%) and 54% were female. Rural residents comprised 64%. Education levels varied, with 38% completing SSC. Monthly income was mostly between 10,000–15,000 BDT. The mean BMI was 24 ± 4.54 kg/m²; 46% were overweight and 20% obese. Comorbidities were common, with 56% of participants diagnosed with diabetes mellitus, 60% with hypertension and 68% with dyslipidemia.

Table II Laboratory findings of Liver Function tests (n=50)

Laboratory parameters	
Total bilirubin (mg/dl)	0.74±0.56
AST (U/L)	33.1±14.3
ALT (U/L)	34.9±14.3
ALP (U/L)	115.3±91.2
Triglycerides (mg/dl)	144.7±99.7
Total cholesterol (mg/dl)	187.2±44.1
HDL (mg/dl)	41.9±9.31
LDL (mg/dl)	114±35.6

Table II shows findings of the liver function tests total bilirubin of 0.74 ± 0.56 mg/dl, AST of 33.1 ± 14.3 U/L, ALT of 34.9 ± 14.3 U/L, and ALP of 115.3 ± 91.2 U/L. Lipid profiles revealed mean triglycerides at 144.7 ± 99.7 mg/dl, total cholesterol at 187.2 ± 44.1 mg/dl, HDL at 41.9 ± 9.31 mg/dl and LDL at 114 ± 35.6 mg/dl.

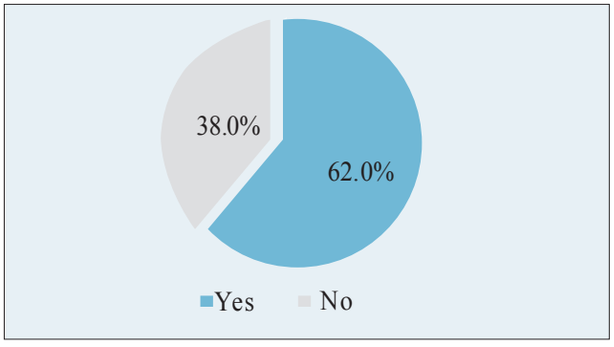

Figure 1 Presence of fatty liver (n=50)

Fatty liver was detected in 62% of patients, indicating a high prevalence among those with calculus cholecystitis and highlighting the need for further investigation into their association (Figure 1).

Table III Comparative analysis between groups (n=50)

Variables	Fatty liver	Non-fatty liver	p-value
Laboratory parameters			
Total bilirubin (mg/dl)	1.2±0.23	1.2±0.27	0.435
AST (U/L)	40.7±5.41	36.3±6.7	0.025
ALT (U/L)	41.7±4.37	37.1±6.54	0.001
ALP (U/L)	83.7±8.81	82.8±8.43	0.679
Triglycerides (mg/dl)	184.5±6.59	159.5±7.93	<0.001
Total cholesterol (mg/dl)	202.4±8.61	195.4±6.90	0.042
HDL (mg/dl)	38.5±3	41.8±3.28	0.024
LDL (mg/dl)	113.7±8.03	113.1±8.68	0.534
Comorbidities			
DM	15(78.9)	7(22.6)	<0.001
HTN	13(68.4)	7(22.6)	0.001
Dyslipidemia	12(63.2)	4(12.9)	<0.001
Obesity	6(31.6)	2(6.5)	0.012

Chi-square and Student t-test done, p<0.05 considered as statistically significant value

IAHS Medical Journal
Volume 08 Issue 02 December 2025; 49-54

Table III interprets that a comparative analysis was conducted between patients with and without fatty liver to evaluate differences in laboratory parameters and comorbidities. The mean total bilirubin levels were similar between the fatty liver group (1.2 ± 0.23 mg/dl) and the non-fatty liver group (1.2 ± 0.27 mg/dl), with no significant difference ($p = 0.435$). However, liver enzymes were significantly higher in the fatty liver group, with mean AST at 40.7 ± 5.41 U/L versus 36.3 ± 6.7 U/L ($p = 0.025$) and mean ALT at 41.7 ± 4.37 U/L versus 37.1 ± 6.54 U/L ($p = 0.001$). ALP levels showed no significant difference between groups ($p = 0.679$).

Lipid profile analysis showed significantly higher triglyceride levels in patients with fatty liver (184.5 ± 6.59 mg/dl) compared to those without fatty liver (159.5 ± 7.93 mg/dl) ($p < 0.001$). Total cholesterol was also elevated in the fatty liver group (202.4 ± 8.61 mg/dl) versus the non-fatty liver group (195.4 ± 6.90 mg/dl) ($p = 0.042$). In contrast, HDL levels were significantly lower in the fatty liver group (38.5 ± 3 mg/dl) compared to those without fatty liver (41.8 ± 3.28 mg/dl) ($p = 0.024$). LDL levels did not differ significantly between groups ($p = 0.534$).

Patients with fatty liver had significantly higher rates of metabolic comorbidities: diabetes mellitus occurred in 78.9% versus 22.6% of those without fatty liver ($p < 0.001$) hypertension in 68.4% versus 22.6% ($p = 0.001$) dyslipidemia in 63.2% versus 12.9% ($p < 0.001$) and obesity in 31.6% versus 6.5% ($p = 0.012$).

Discussion

Calculus cholecystitis is a frequently encountered gastrointestinal disorder in clinical practice, often associated with metabolic disturbances, including fatty liver disease and abnormal liver function. Several studies have highlighted the relationship between GD, fatty liver and impaired hepatic function, which may result from compromised gallbladder motility and increased bile lithogenicity.[2,19,24] This study demonstrates a significant association between calculus cholecystitis, fatty liver disease and metabolic dysregulation, consistent with global trends. In this study, 38% of patients diagnosed with calculus cholecystitis had concurrent fatty liver disease, suggesting a strong association between the two conditions. This relationship can be attributed to shared risk factors such as obesity, dyslipidemia, insulin resistance, and type 2 diabetes mellitus, all of which are components of metabolic syndrome. Previous studies have demonstrated that insulin resistance, elevated blood pressure, diabetes and obesity are linked to both fatty liver disease and gallstone formation.[19,25-27] This research finding aligns with U.S. data showing a 3.3-fold higher NAFLD risk in gallstone patients (OR = 6.32, $p < 0.001$).[28] Therefore, clinicians should be vigilant in assessing the presence of one condition when diagnosing the other to facilitate timely management and prevention of complications.

The demographic analysis of the study population revealed that more than half of the patients with calculus cholecystitis were above 30 years of age, with a mean age of 39 ± 14.2 years. The increasing prevalence of cholecystitis with age may be attributed to the rising burden of metabolic syndrome, sedentary lifestyles and prolonged exposure to risk factors such as poor dietary habits and physical inactivity.[29,30] Additionally, the study found a higher prevalence of calculus cholecystitis among females (54%) which aligns with findings from previous research. Xu Li et al. reported a similar trend in a study conducted among a Chinese population, where 55.7% of female participants were diagnosed with gallstones.[19] The higher predisposition in females is believed to be influenced by hormonal factors, particularly estrogen, which enhances cholesterol secretion and progesterone, which reduces bile acid secretion, leading to the supersaturation of bile with cholesterol and promoting gallstone formation.[31] Moreover, long-term use of oral contraceptive steroids and estrogen therapy has been associated with an increased risk of cholesterol gallstones.[10] However, the stronger NAFLD-gallstone association in women reported by NIH studies (OR = 6.05 vs. 6.67 in men) was not fully replicated here, possibly due to women smaller sample.[28]

In terms of liver function, this study observed that patients with calculus cholecystitis had mean levels of total bilirubin, AST, ALT and ALP at 0.74 ± 0.56 mg/dl, 33.1 ± 14.3 U/L, 34.9 ± 14.3 U/L and 115.3 ± 91.2 U/L, respectively. While these values remained within normal reference ranges, AST and ALT levels were in the upper tier, indicating a potential subclinical hepatic insult in these patients. In this study, patients with fatty liver had significantly higher AST and ALT levels compared to those without fatty liver, with the exception of ALP, which did not show a statistically significant difference. Similar findings were reported in a large cross-sectional study among 950 patients, where AST and ALT levels were significantly elevated in those with fatty liver.[32] Studies by Novakovic et al. and Zakeri et al. also support the association of ALT elevation with fatty liver disease and dyslipidemia, suggesting that altered hepatic function may play a role in disease progression.[33,34] The lipid profile analysis demonstrated that patients with calculus cholecystitis had elevated total cholesterol levels (187.2 ± 44.1 mg/dl) which supports the hypothesis that altered lipid

IAHS Medical Journal
Volume 08 Issue 02 December 2025; 49-54

metabolism plays a role in gallstone formation. Increased cholesterol levels contribute to decreased bile acid production, reducing the solubility of cholesterol in bile ultimately leading to gallstone precipitation.[35,36] This finding highlights the importance of lifestyle modifications, including increased physical activity and dietary adjustments, to mitigate gallstone formation and its metabolic consequences.

The 56% diabetes and 68% dyslipidemia rates in our cohort mirror U.S. data where metabolic syndrome mediates the NAFLD-gallstone link.[28] Elevated triglycerides (184.5 mg/dL) and low HDL (38.5 mg/dL) in fatty liver patients reflect insulin resistance-driven cholesterol hypersecretion, a key lithogenic mechanism. This parallels findings from Santhoshakumari et al. where dyslipidemia strongly predicted dual NAFLD-cholecystitis diagnoses.[35] The lipid profile analysis further demonstrated that patients with fatty liver had significantly higher triglyceride and total cholesterol levels, along with lower HDL levels, compared to those without fatty liver. These findings are consistent with previous studies, including research by Santhoshakumari et al. which reported elevated total cholesterol, LDL and triglycerides and reduced HDL in patients with fatty liver.[35] Similarly, Mahaling et al. found a significant association between increased fatty liver severity and elevated total cholesterol and reduced HDL levels, reinforcing the metabolic link between dyslipidemia and hepatic steatosis.[36]

Limitation

This study has several limitations. It was single-center with a small sample size (n=50) limiting generalizability and statistical power. The cross-sectional design prevents causal inference. Liver biopsy was not used, so fatty liver diagnosis relied on ultrasonography, which may reduce accuracy. Long-term outcomes and confounders like diet, activity and genetics were not assessed.

Conclusion

This study reveals a strong association between calculus cholecystitis and fatty liver disease, driven by shared metabolic risks like obesity and dyslipidemia. Elevated liver enzymes and abnormal lipids were common, suggesting the need for routine hepatic screening and lifestyle interventions in gallstone patients.

Recommendation

Early laparoscopic cholecystectomy is recommended, with conservative management for high-risk cases. Larger, multi-center studies are needed to clarify causal relationships and guide targeted interventions.

Acknowledgments

The authors sincerely acknowledge the cooperation of all participants and the hospital authorities for their support throughout the study.

Disclosure

All the authors declared no competing interest.

References

1. Sepehrimanesh M, Niknam R, Ejtehadi F, Fattahi MR, Safarpour A. Association Between Non-Alcoholic Fatty Liver Disease and Metabolic Syndrome with Gallstone Disease, South Iran : A Population-Based Study. Diabetes Metab Syndr Obes. 2020;13:1449–1458.

2. Ahmed F, Baloch Q, Memon ZA, Ali I. An observational study on the association of non-alcoholic fatty liver disease and metabolic syndrome with gall stone disease requiring cholecystectomy. Ann Med Surg. 2017;17:7–13.

3. Kim YK, Kwon OS, Her KH. The grade of non-alcoholic fatty liver disease is an independent risk factor for gallstone disease: An observational Study. Medicine (Baltimore). 2019;98(27):e16018.

4. Gomes CA, Junior CS, Di Saverio S, Sartelli M, Kelly MD, Gomes CC et al. Acute calculous cholecystitis: Review of current best practices. World J Gastrointest Surg. 2017;9(5):118–126.

5. Sajjan DSC, Javali DS, BV DM, B DR. Clinical profile of patients with cholelithiasis at a tertiary care hospital. Int J Surg Sci. 2021;5(2):244–246.

6. Everhart JE, Khare M, Hill M, Maurer KR. Prevalence and ethnic differences in gallbladder disease in the United States. Gastroenterology. 1999;117(3):632–639.

7. Stinton LM, Shaffer EA. Epidemiology of gallbladder disease: Cholelithiasis and cancer. Gut Liver. 2012;6(2):172–187.

8. Saha M, Nahar K, Hosen MA, Khan MH, Kumar Saha S, Shil BC, et al. Prevalence and Risk Factors of Asymptomatic Gallstone Disease in North-East Part of Bangladesh. Euroasian J hepato-gastroenterology. 2015;5(1):1–3.

9. Pahari S, Basukala S, Piya U, Khand Y, Thapa B, Thapa O et al. Gallstone among Patients Presenting to the Department of Surgery in a Tertiary Care Center: A Descriptive Cross-sectional Study. JNMA J Nepal Med Assoc. 2023;61(260):315–319.

10. Wang HH, Liu M, Clegg DJ, Portincasa P, Wang DQH. New insights into the molecular mechanisms underlying effects of estrogen on cholesterol gallstone formation. Biochim Biophys Acta. 2009;1791(11):1037–1047.

IAHS Medical Journal
Volume 08 Issue 02 December 2025; 49-54

11. Chang YR, Jang JY, Kwon W, Park JW, Kang MJ, Ryu JK et al. Changes in demographic features of gallstone disease: 30 years of surgically treated patients. Gut Liver. 2013 Nov;7(6):719–724.

12. Pak M, Lindseth G. Risk Factors for Cholelithiasis. Gastroenterol Nurs Off J Soc Gastroenterol Nurses Assoc. 2016;39(4):297–309.

13. Méndez-Sánchez N, Chavez-Tapia NC, Motola-Kuba D, Sanchez-Lara K, Ponciano-Rodríguez G, Baptista H, et al. Metabolic syndrome as a risk factor for gallstone disease. World J Gastroenterol. 2005;11(11):1653–1657.

14. Tsunoda K, Shirai Y, Hatakeyama K. Prevalence of cholesterol gallstones positively correlates with per capita daily calorie intake. Hepatogastroenterology. 2004;51(59):1271–1274.

15. O'Connell P R, McCaskie A W & Sayers RD. The gallbladder and bile ducts. In: Bailey & Love's Short Practice of Surgery. 28th ed. 2023.

16. Muzurovi E, Mikhailidis DP, Mantzoros C. Non-alcoholic fatty liver disease, insulin resistance, metabolic syndrome and their association with vascular risk. Metabolism. 2021;119:154770.

17. Golabi P, Otgonsuren M, de Avila L, Sayiner M, Rafiq N, Younossi ZM. Components of metabolic syndrome increase the risk of mortality in non-alcoholic fatty liver disease (NAFLD). Medicine (Baltimore). 2018;97(13):e0214.

18. Mansour-Ghanaei R, Mansour-Ghanaei F, Naghipour M, Joukar F. Biochemical markers and lipid profile in non-alcoholic fatty liver disease patients in the PERSIAN Guilan cohort study (PGCS), Iran. J Fam Med Prim care. 2019;8(3):923–928.

19. Li X, Gao P. Fatty liver increases gallstone disease risk in younger Chinese patients. Medicine (Baltimore). 2019;98(22):e15940.

20. Sude K, Mm R, Ar K, Ma A, Parveen Z. Evaluation of Aetiological Factors and Outcome of Cholelithiasis in Children : Experience in Tertiary Level Hospitals. 2019;15(2):140–143.

21. Lee YC, Wu JS, Yang YC, Chang CS, Lu FH, Chang CJ. Moderate to severe, but not mild, non-alcoholic fatty liver disease associated with increased risk of gallstone disease. Scand J Gastroenterol. 2014;49(8):1001–1006.

22. Chang Y, Noh YH, Suh BS, Kim Y, Sung E, Jung HS, et al. Bidirectional Association between Non-alcoholic Fatty Liver Disease and Gallstone Disease: A Cohort Study. J Clin Med. 2018;7(11).

23. Chen YC, Chiou C, Lin MN, Lin CL. The prevalence and risk factors for gallstone disease in taiwanese vegetarians. PLoS One. 2014;9(12):e115145.

24. Padda MS, Singh S, Tang SJ, Rockey DC. Liver test patterns in patients with acute calculous cholecystitis and/or choledocholithiasis. Aliment Pharmacol Ther. 2009;29(9):1011–1018.

25. Ahmed MH, Ali A. Non-alcoholic fatty liver disease and cholesterol gallstones: Which comes first? Scand J Gastroenterol. 2014;49(5):521–527.

26. Smelt AHM. Triglycerides and gallstone formation. Clin Chim Acta. 2010;411(21–22):1625–1631.

27. Chen LY, Qiao QH, Zhang SC, Chen YH, Chao GQ, Fang LZ. Metabolic syndrome and gallstone disease. World J Gastroenterol. 2012;18(31):4215–4220.

28. Kichloo A, Solanki S, Haq KF, Dahiya D, Bailey B, Solanki D et al. Association of non-alcoholic fatty liver disease with gallstone disease in the United States hospitalized patient population. World J Gastrointest Pathophysiol. 2021;12(2):14–24.

29. Völzke H, Baumeister SE, Alte D, Hoffmann W, Schwahn C, Simon P et al. Independent risk factors for gallstone formation in a region with high cholelithiasis prevalence. Digestion. 2005;71(2):97–105.

30. Kriska AM, Brach JS, Jarvis BJ, Everhart JE, Fabio A, Richardson CR et al. Physical activity and gallbladder disease determined by ultrasonography. Med Sci Sports Exerc. 2007;39(11):1927–1932.

31. Tierney S, Nakeeb A, Wong O, Lipsett PA, Sostre S, Pitt HA et al. Progesterone alters biliary flow dynamics. Ann Surg. 1999;229(2):205–209.

32. Li YY. Genetic and epigenetic variants influencing the development of non-alcoholic fatty liver disease. World J Gastroenterol. 2012;18(45):6546–6551.

33. Novakovic T, Mekic M, Smilic L, Smilic T, Ini - Kostic B, Jovicevic L et al. Anthropometric and biochemical characteristics of patients with non-alcoholic fatty liver diagnosed by non-invasive diagnostic methods. Med Arch (Sarajevo, Bosnia Herzegovina). 2014;68(1):22–26.

34. Zakeri A, Karamat-Panah S. Prevalence of non-alcoholic fatty liver disease and its risk factors in patients referred to Ardabil city hospital during 2015-2016. Int J Community Med Public Heal. 2018;5(3):917.

35. Santhoshakumari TMJ. A Study of Anthropometric And Lipid Profile Parameters in Non-Alcoholic Fatty Liver Disease Patients Attending A Tertiary Care Hospitalat Puducherry. IOSR J Dent Med Sci. 2017;16(01):33–37.

36. Mahaling DU, Basavaraj MM, Bika AJ. Comparison of lipid profile in different grades of non-alcoholic fatty liver disease diagnosed on ultrasound. Asian Pac J Trop Biomed. 2013;3(11):907–912.