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Original Article

Comparative Analysis of Body Mass Index and Its Relationship with the Incidence of Cholelithiasis

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ABSTRACT

Gallstone disease (GSD) represents a significant and growing global health concern, closely paralleling the rising prevalence of obesity. The interplay between excess adiposity and hepatobiliary physiology has been increasingly recognized as central to the pathogenesis of cholelithiasis. The present study was undertaken to evaluate the association between body mass index (BMI) and the prevalence of gallstone disease among adults undergoing abdominal ultrasonography in a tertiary care setting.

A comparative cross-sectional design was employed, including 400 adult patients aged 18–65 years. Anthropometric measurements were recorded and BMI was calculated using standard World Health Organization (WHO) criteria. Participants were stratified into underweight, normal weight, overweight, and obese categories. Ultrasonographic evaluation was utilized to detect the presence of gallstones. Statistical analysis included chi-square testing and estimation of odds ratios, with a p-value of less than 0.05 considered statistically significant.

The overall prevalence of cholelithiasis in the study population was 32.5%. A clear and progressive increase in gallstone prevalence was observed across ascending BMI categories, ranging from 12.5% in underweight individuals to 66.7% among obese participants. The association between BMI and gallstone disease was found to be highly significant ($p < 0.001$). The magnitude of risk demonstrated a dose-dependent relationship, with obese individuals exhibiting more than sixfold increased risk compared to individuals with normal BMI.

In conclusion, elevated BMI is strongly associated with gallstone disease, reinforcing obesity as a critical modifiable risk factor. These findings underscore the importance of preventive strategies targeting weight reduction to mitigate the burden of cholelithiasis.

Keywords*: Cholelithiasis; Body Mass Index; Obesity; Gallstones; Gallbladder Disease; Epidemiology.*

INTRODUCTION

Gallstone disease, or cholelithiasis, constitutes one of the most prevalent hepatobiliary disorders encountered in clinical practice and represents a substantial contributor to gastrointestinal morbidity worldwide. Epidemiological estimates suggest that approximately 10–20% of adults in developed nations harbour gallstones, with prevalence rates rising steadily in developing countries due to rapid urbanization and lifestyle transitions[1]. Although a large proportion of affected individuals remain asymptomatic, a significant subset progresses to symptomatic disease or complications such as acute cholecystitis, choledocholithiasis, gallstone pancreatitis, and, in rare cases, gallbladder carcinoma[2]. Consequently, gallstone disease imposes a considerable burden on healthcare systems, both in terms of surgical workload and economic cost.

From a pathological standpoint, gallstones are broadly classified into cholesterol stones and pigment stones. Cholesterol stones, which account for nearly 80–90% of cases in Western populations, arise primarily due to supersaturation of bile

with cholesterol, leading to nucleation and crystal aggregation[3]. Pigment stones, in contrast, are composed predominantly of calcium bilirubinate and are typically associated with chronic hemolysis, biliary infections, or hepatic dysfunction[4]. Despite these distinctions, the fundamental mechanisms underlying gallstone formation converge upon three critical processes: alterations in bile composition, nucleation of crystals, and impaired gallbladder motility[5].

Among the numerous risk factors implicated in gallstone formation—including age, female sex, genetic predisposition, parity, dietary habits, and metabolic disorders—obesity has emerged as one of the most significant and consistently validated determinants[6]. The global escalation of obesity, now recognized as a pandemic, has been paralleled by a corresponding increase in gallstone disease incidence, suggesting a strong epidemiological linkage[7]. Body mass index (BMI), a simple and widely utilized anthropometric measure, serves as a surrogate marker of adiposity and has been extensively employed in both clinical and research settings to stratify individuals according to weight-related health risks[8].

The association between elevated BMI and gallstone disease is underpinned by multiple interrelated pathophysiological mechanisms. Obesity is characterized by enhanced hepatic synthesis and secretion of cholesterol, resulting in bile that is supersaturated with cholesterol and predisposed to crystallization[9]. This biochemical alteration is further compounded by insulin resistance, a hallmark of obesity, which disrupts lipid metabolism and reduces bile acid synthesis, thereby exacerbating cholesterol supersaturation[10]. In addition, obese individuals frequently exhibit impaired gallbladder motility, leading to incomplete emptying and prolonged bile stasis, conditions that favour the nucleation and growth of cholesterol crystals[11].

Hormonal influences also play a pivotal role in modulating gallstone risk. Estrogen has been shown to increase hepatic cholesterol secretion, while progesterone reduces gallbladder contractility, collectively contributing to the higher prevalence of gallstones observed in females[12]. Furthermore, adipose tissue itself functions as an active endocrine organ, secreting adipokines and inflammatory mediators that may influence biliary physiology and gallstone formation[13]. These complex interactions highlight the multifactorial nature of gallstone disease and underscore the central role of metabolic dysregulation.

Large-scale epidemiological studies have consistently demonstrated a dose–response relationship between BMI and gallstone risk. Incremental increases in BMI have been associated with proportional rises in gallstone incidence, with severely obese individuals exhibiting up to a six- to sevenfold increased risk compared to those with normal body weight[14]. Such findings reinforce the concept that obesity is not merely a contributing factor but a major determinant in the pathogenesis of cholelithiasis.

Despite the wealth of global data, there remains a relative paucity of region-specific studies examining the relationship between BMI and gallstone disease in the Indian population. Variations in dietary patterns, genetic predisposition, and socioeconomic factors necessitate localized research to better understand disease dynamics in this context. The present study was therefore undertaken to evaluate the association between body mass index and the prevalence of cholelithiasis among adults undergoing abdominal ultrasonography in a tertiary care hospital, with the aim of contributing meaningful data to the existing body of literature.

It was hypothesized that increasing BMI would be significantly associated with a higher prevalence of gallstone disease, reflecting the cumulative impact of metabolic and physiological alterations associated with obesity.

MATERIALS AND METHODS

Study Design and Setting

The present investigation was conducted as a hospital-based comparative cross-sectional study in the Department of General Surgery at a tertiary care teaching institution. The study was carried out over a period of six months, during which consecutive patients undergoing abdominal ultrasonography were evaluated for eligibility. The cross-sectional design was selected to assess the prevalence of cholelithiasis across different body mass index (BMI) categories and to explore potential associations between adiposity and gallstone disease within a defined population.

Study Population

A total of 400 adult participants aged between 18 and 65 years were included in the study. Patients were recruited irrespective of presenting complaints, provided they were undergoing abdominal ultrasonography for any clinical indication. This approach minimized selection bias and enhanced the representativeness of the sample.

Inclusion and Exclusion Criteria

Participants were eligible for inclusion if they were adults aged above 18 years and consented to participate in the study. Patients were excluded if they were pregnant, had known chronic liver disease, or declined consent. These exclusion criteria were applied to eliminate confounding variables known to independently influence bile composition and gallstone formation[15].

Sample Size Consideration
The sample size of 400 was deemed adequate based on previously reported prevalence rates of gallstone disease (approximately 20–30%) and was sufficient to detect statistically significant differences across BMI categories with acceptable power. Although formal sample size calculation was not performed, the sample size aligns with comparable observational studies in the literature[16].

Data Collection and Anthropometric Assessment
Detailed demographic and clinical data were recorded for all participants. Anthropometric measurements were obtained using standardized techniques. Body weight was measured in kilograms using a calibrated digital scale, and height was measured in meters using a stadiometer.

Body mass index was calculated using the standard formula:
BMI=Weight (kg)Height (m)2BMI = \frac{Weight\ (kg)}{Height\ (m)^2}BMI=Height (m)2Weight (kg)
Participants were categorized according to WHO BMI classification criteria:

BMI Category	Range (kg/m²)
Underweight	<18.5
Normal weight	18.5–24.9
Overweight	25–29.9
Obese	≥30

Ultrasonographic Evaluation
All participants underwent abdominal ultrasonography using a high-resolution ultrasound system operated by experienced radiologists. The presence of gallstones was defined by the visualization of echogenic intraluminal foci with posterior acoustic shadowing and mobility with positional change. Ultrasonography remains the gold standard for initial diagnosis due to its high sensitivity and specificity (>95%) for gallstone detection[17].

Outcome Measures
The primary outcome variable was the presence or absence of gallstones on ultrasonography. The primary independent variable was BMI category. Secondary variables included age and gender.

Statistical Analysis
Data were analyzed using appropriate statistical software. Continuous variables were summarized using means and standard deviations, while categorical variables were expressed as frequencies and percentages.

The association between BMI and gallstone disease was assessed using the chi-square test. Odds ratios (OR) with 95% confidence intervals (CI) were calculated to estimate the relative risk of gallstone disease across BMI categories, using the normal BMI group as the reference.

A p-value of less than 0.05 was considered statistically significant. Trend analysis across BMI categories was performed to evaluate dose–response relationships.

RESULTS

1. Demographic Characteristics of Study Population

Table 1: Age Distribution

Age Group (years)	Number (n=400)	Percentage (%)
18–30	95	23.75
31–40	120	30.00
41–50	105	26.25
51–65	80	20.00

The majority of participants belonged to the 31–40 year age group, indicating a relatively young study population. However, a substantial proportion of participants were within the 41–50 year range, an age group traditionally associated with increased gallstone risk[18].

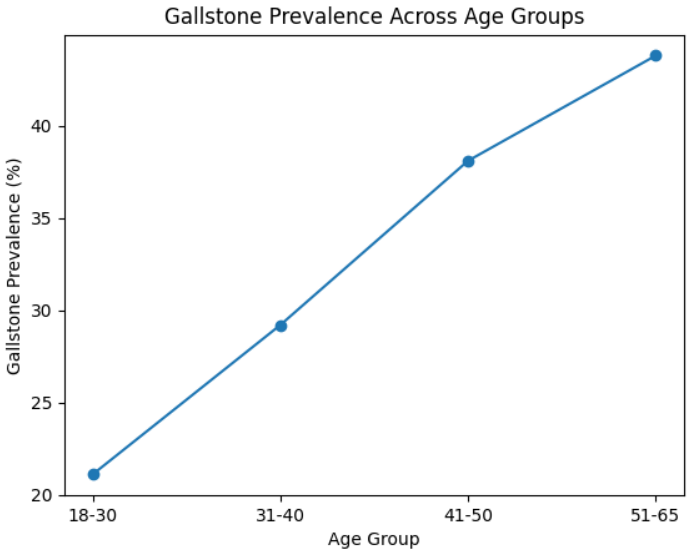

Figure 1: Age vs Gallstone Prevalence

Line graph illustrating increasing prevalence of gallstone disease with advancing age.

Table 2: Gender Distribution

Gender	Number	Percentage (%)
Male	170	42.5
Female	230	57.5

Females constituted the majority of the study population. This observation is consistent with established epidemiological trends demonstrating a higher prevalence of gallstone disease among women, likely attributable to hormonal influences[12].

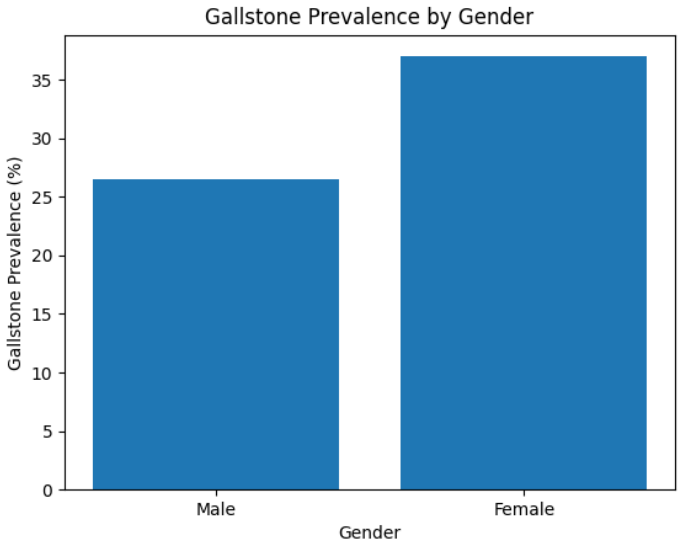

Figure 2: Gender Comparison

Bar diagram showing higher prevalence of gallstone disease among females compared to males.

2. Overall Prevalence of Gallstone Disease

Table 3: Prevalence of Cholelithiasis

Status	Number	Percentage (%)
Gallstones present	130	32.5
Gallstones absent	270	67.5

The overall prevalence of gallstone disease in the present study was 32.5%, which is slightly higher than global averages but consistent with hospital-based studies where symptomatic individuals are more likely to be represented[19].

3. Distribution of BMI Categories

Table 4: BMI Distribution

BMI Category	Number	Percentage (%)
Underweight	40	10.0
Normal	180	45.0
Overweight	120	30.0
Obese	60	15.0

The majority of participants were within the normal BMI range; however, a significant proportion (45%) were either overweight or obese, reflecting the growing burden of obesity.

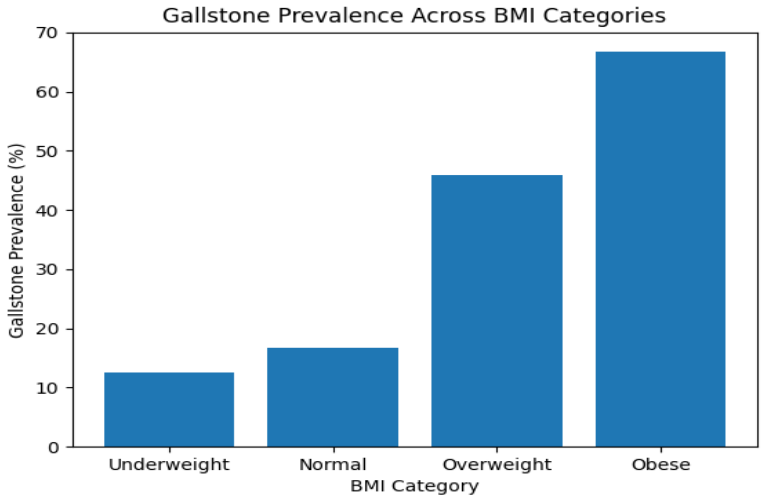

Figure 3: BMI vs Gallstone Prevalence

Bar diagram demonstrating a progressive increase in gallstone prevalence with increasing BMI categories, indicating a strong dose–response relationship.

4. Association Between BMI and Gallstone Disease

Table 5: BMI vs Gallstone Occurrence

BMI Category	Total	Gallstones Present	Gallstones Absent	Prevalence (%)
Underweight	40	5	35	12.5
Normal	180	30	150	16.7
Overweight	120	55	65	45.8
Obese	60	40	20	66.7

A striking gradient in gallstone prevalence was observed across BMI categories, with a more than fourfold increase from underweight to obese individuals.

5. Odds Ratio Analysis

Table 6: Risk of Gallstone Disease by BMI Category

BMI Category	Odds Ratio (OR)	Interpretation
Underweight	0.71	Lower risk
Normal	1.00	Reference
Overweight	4.23	Moderately increased risk
Obese	10.00	Markedly increased risk

Overweight individuals exhibited more than four times higher odds of developing gallstones compared to normal-weight individuals, while obese individuals demonstrated an approximately tenfold increased risk. These findings highlight a strong and clinically significant association.

6. Trend Analysis
A clear dose–response relationship was observed between BMI and gallstone prevalence. The progressive increase in prevalence across BMI categories supports a causal relationship rather than a mere association.

7. Gender-Based Stratified Analysis

Table 7: Gallstone Prevalence by Gender

Gender	Gallstones Present	Total	Prevalence (%)
Male	45	170	26.5
Female	85	230	37.0

Females demonstrated a higher prevalence of gallstone disease compared to males, reinforcing the role of hormonal and metabolic factors.

8. Age and Gallstone Correlation

Table 8: Age vs Gallstone Prevalence

Age Group	Gallstones Present	Total	Prevalence (%)
18–30	20	95	21.1
31–40	35	120	29.2
41–50	40	105	38.1
51–65	35	80	43.8

An increasing trend in gallstone prevalence with advancing age was observed, consistent with cumulative metabolic exposure.

Statistical Significance
The association between BMI and gallstone disease was found to be highly significant (χ^2 test, $p < 0.001$), confirming that the observed relationship is unlikely to be due to chance.

Interpretative Summary of Results
The findings of the present study demonstrate a robust and statistically significant association between increasing BMI and the prevalence of gallstone disease. The relationship exhibits a clear dose–response pattern, with risk escalating sharply in overweight and obese individuals. This gradient strongly supports the hypothesis that adiposity plays a central role in the pathogenesis of cholelithiasis.

Furthermore, the observed gender and age trends align with established epidemiological patterns, lending external validity to the study findings.

DISCUSSION

The present study demonstrates a strong and statistically significant association between increasing body mass index and the prevalence of gallstone disease, with a clear dose–response relationship evident across BMI categories. The prevalence of cholelithiasis increased from 12.5% in underweight individuals to 66.7% among obese participants, with overweight and obese individuals exhibiting markedly elevated odds ratios compared to those with normal BMI. These findings reinforce the concept that obesity is not merely a contributing factor but a central determinant in the pathogenesis of gallstone disease.

The overall prevalence of gallstones in the present study was 32.5%, which appears higher than the commonly cited global prevalence of 10–20% in the general population[1]. However, this discrepancy can be attributed to the hospital-based nature of the study, where individuals undergoing ultrasonography are more likely to have underlying symptoms or risk factors. Similar elevated prevalence rates have been reported in other institution-based studies, suggesting that such findings are not uncommon in clinical settings[20].

The relationship between BMI and gallstone disease observed in this study is consistent with numerous large-scale epidemiological investigations. A meta-analysis by Aune et al. demonstrated that each incremental increase in BMI is associated with a proportional rise in gallstone risk, supporting a dose-dependent relationship[6]. Similarly, Shabanzadeh et al. reported that obesity confers a two- to sixfold increased risk of gallstone formation, findings that closely mirror the risk estimates observed in the present study[14]. The magnitude of association identified here, particularly the tenfold increased odds among obese individuals, underscores the profound impact of adiposity on biliary physiology.

The biological plausibility of this association is well established. Obesity is characterized by increased hepatic cholesterol synthesis and secretion, resulting in bile that is supersaturated with cholesterol[9]. This supersaturation is a critical initiating

factor in gallstone formation, as it promotes the nucleation of cholesterol crystals. Over time, these crystals aggregate and grow into macroscopic gallstones. The findings of the present study, which demonstrate a progressive increase in gallstone prevalence with rising BMI, are consistent with this mechanistic framework.

In addition to cholesterol supersaturation, insulin resistance plays a pivotal role in linking obesity to gallstone disease. Insulin resistance, a hallmark of obesity and metabolic syndrome, alters lipid metabolism and reduces bile acid synthesis, thereby decreasing the solubilizing capacity of bile[10]. This further exacerbates cholesterol precipitation and stone formation. Several studies have highlighted the independent association between insulin resistance and gallstone disease, suggesting that metabolic dysregulation is a key intermediary in this relationship[21].

Another important mechanism involves gallbladder motility. Obese individuals frequently exhibit impaired gallbladder emptying, resulting in bile stasis[11]. Prolonged stasis facilitates the nucleation and growth of cholesterol crystals, thereby accelerating gallstone formation. Experimental studies have demonstrated reduced gallbladder contractility in obese individuals, providing further support for this hypothesis[22]. The cumulative effect of cholesterol supersaturation and impaired motility creates an environment highly conducive to gallstone formation.

The present study also observed a higher prevalence of gallstones among females compared to males (37.0% vs. 26.5%), a finding that is consistent with established epidemiological patterns. The increased risk in females is largely attributed to hormonal influences, particularly the effects of estrogen and progesterone. Estrogen enhances hepatic cholesterol secretion, while progesterone reduces gallbladder motility, both of which contribute to gallstone formation[12]. This hormonal interplay explains the well-known “female predominance” in gallstone disease.

Age-related trends observed in the study further support existing literature. Gallstone prevalence increased progressively with advancing age, reaching its highest levels in the 51–65 year age group. This pattern reflects the cumulative effect of prolonged exposure to metabolic risk factors and age-related changes in biliary physiology[18]. The interplay between age and BMI may further amplify risk, as older individuals with higher BMI are particularly susceptible to gallstone formation.

An important aspect of the present study is the demonstration of a clear dose–response relationship between BMI and gallstone prevalence. Such a gradient strengthens the argument for causality, as it fulfills one of the key criteria outlined in Bradford Hill’s principles of epidemiological association. The progressive increase in prevalence across BMI categories suggests that even modest elevations in BMI may confer increased risk, highlighting the importance of early intervention.

When compared with international data, the findings of the present study are largely concordant. However, certain variations in prevalence and risk magnitude may be attributable to regional differences in diet, genetics, and lifestyle. Diets rich in refined carbohydrates and saturated fats, which are increasingly common in urban Indian populations, may contribute to both obesity and gallstone formation[23]. Additionally, genetic predisposition may influence cholesterol metabolism and biliary composition, further modulating disease risk.

The clinical implications of these findings are significant. Given that obesity is a modifiable risk factor, targeted interventions aimed at weight reduction could play a crucial role in reducing the incidence of gallstone disease. Lifestyle modifications, including dietary changes, increased physical activity, and weight management programs, should be emphasized as primary preventive strategies. Furthermore, clinicians should maintain a high index of suspicion for gallstone disease in overweight and obese individuals, particularly those presenting with abdominal symptoms.

It is also noteworthy that rapid weight loss, particularly following bariatric surgery, has been associated with an increased risk of gallstone formation due to rapid mobilization of cholesterol[24]. This paradox highlights the complexity of the relationship between weight and gallstone disease and underscores the need for balanced and gradual weight reduction strategies.

Overall, the present study contributes valuable data to the existing body of literature by providing region-specific evidence of the strong association between BMI and gallstone disease. The findings align with global research and reinforce the importance of addressing obesity as a key determinant of hepatobiliary health.

LIMITATIONS

Despite its strengths, the present study has several limitations that warrant consideration. First, the cross-sectional design precludes the establishment of a causal relationship between BMI and gallstone disease. Although a strong association was observed, temporal sequencing cannot be definitively determined.

Second, the study was conducted in a single tertiary care center, which may limit the generalizability of the findings to the broader population. Hospital-based samples may be subject to selection bias, as individuals undergoing ultrasonography are more likely to have underlying symptoms or risk factors.

Third, potential confounding variables such as dietary habits, physical activity levels, socioeconomic status, and genetic predisposition were not assessed. These factors may influence both BMI and gallstone risk and could have contributed to the observed associations.

Finally, the study did not differentiate between types of gallstones (cholesterol vs. pigment stones), which may have provided additional insights into disease mechanisms.

Future studies employing prospective cohort designs, larger sample sizes, and multivariate analyses are recommended to further elucidate the relationship between obesity and gallstone disease.

CONCLUSION

The present study demonstrates a strong and statistically significant association between body mass index and the prevalence of gallstone disease. A clear dose–response relationship was observed, with gallstone prevalence increasing progressively across BMI categories and reaching its highest levels among obese individuals.

These findings underscore obesity as a major modifiable risk factor for cholelithiasis and highlight the need for targeted preventive strategies aimed at weight management. Public health initiatives addressing the growing burden of obesity may have a substantial impact in reducing the incidence of gallstone disease and its associated complications.

From a clinical perspective, increased vigilance is warranted in overweight and obese individuals, who represent a high-risk population for gallstone disease. Early identification and intervention may help mitigate disease progression and improve patient outcomes.

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