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CHANGES IN THE GALLBLADDER WALL AT DIFFERENT DEGREES OF OBESITY

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Abstract

Background: Obesity is a growing global health concern with wide-ranging systemic effects, including changes in biliary tract structure and function. While gallstone formation has been well-documented in obese individuals, less attention has been paid to early histological changes in the gallbladder wall associated with varying degrees of obesity.

Objective: To evaluate the histopathological changes of the gallbladder wall in patients with different body mass index (BMI) categories and determine the relationship between obesity severity and structural alterations.

Methods:A total of 90 patients undergoing elective cholecystectomy were categorized into three groups based on BMI: normal weight (BMI 18.5–24.9), overweight (BMI 25.0–29.9), and obese (BMI ≥30.0). Gallbladder wall specimens were evaluated for thickness, mucosal hyperplasia, smooth muscle hypertrophy, subepithelial fibrosis, and inflammatory infiltration. Statistical analysis assessed correlations between BMI and histological changes.

Results: Gallbladder wall thickness and severity of histopathological changes increased significantly with higher BMI ($p < 0.001$). Mucosal hyperplasia, muscular hypertrophy, and fibrosis were significantly more prevalent in obese patients. Strong positive correlations were found between BMI and both wall thickness and fibrosis scores.

Conclusion: Obesity is associated with progressive structural changes in the gallbladder wall that may precede clinical symptoms or gallstone formation. Early identification of such alterations may aid in preventing future biliary complications in at-risk populations.

Keywords: Gallbladder wall; Obesity; Body mass index; Histopathology; Inflammation; Fibrosis; Mucosal hyperplasia; Cholecystectomy.

Introduction

Obesity has become one of the most pressing global public health challenges of the 21st century. According to the World Health Organization, the prevalence of overweight and obesity has significantly increased over the past few decades, affecting individuals of all ages and socioeconomic backgrounds [1]. Obesity is known to be associated with a wide range of metabolic disorders, including type 2 diabetes, cardiovascular diseases, non-alcoholic fatty liver disease, and various gastrointestinal pathologies [2,3].

Among the organs affected by obesity, the gallbladder plays a crucial role in digestive physiology. It stores and concentrates bile produced by the liver and releases it into the duodenum in response to food intake, particularly fats. The structural integrity and contractile function of the gallbladder wall are essential for normal bile flow and efficient digestion. Alterations in gallbladder wall

morphology can disrupt bile dynamics and contribute to the development of biliary disorders, such as gallstones and chronic cholecystitis [4].

Several studies have reported a higher incidence of gallbladder diseases among individuals with obesity. It has been suggested that increased cholesterol saturation in bile, altered gallbladder motility, and chronic low-grade inflammation may contribute to gallbladder dysfunction in obese individuals [5,6]. However, limited data are available on how the degree of obesity influences specific morphological changes in the gallbladder wall. In particular, histological and structural changes such as wall thickening, mucosal hyperplasia, smooth muscle hypertrophy, and fibrosis have not been comprehensively studied in relation to obesity severity [7].

Given the increasing rates of obesity worldwide and the associated burden of gallbladder diseases, it is essential to investigate the morphological changes in the gallbladder wall across different degrees of obesity. This study aims to analyze and compare the structural alterations of the gallbladder wall in patients with varying body mass index (BMI) categories, thereby providing insights into the potential pathophysiological mechanisms linking obesity with biliary tract pathology.

Materials and Methods (Expanded with Citations)

This study was designed as a prospective, cross-sectional observational analysis aiming to evaluate the structural changes in the gallbladder wall among patients with varying degrees of obesity. All procedures were carried out in accordance with the Declaration of Helsinki and were approved by the Ethics Committee of [Institution Name]. Written informed consent was obtained from all participants prior to their inclusion in the study.

Study Population

A total of 90 adult patients (aged 18–65 years) undergoing elective laparoscopic cholecystectomy at [Hospital Name] between January 2022 and December 2024 were included in the study. The inclusion criteria comprised patients diagnosed with chronic cholecystitis or symptomatic cholelithiasis confirmed by clinical and ultrasonographic findings. Exclusion criteria included the presence of diabetes mellitus, metabolic syndrome, acute cholecystitis, history of hepatic cirrhosis, malignancies, prior biliary tract surgery, or any systemic inflammatory or autoimmune disease, as these conditions may independently affect gallbladder wall morphology [1,2].

Group Classification

Participants were stratified into three groups based on their body mass index (BMI), in accordance with World Health Organization (WHO) classification [3]:

- **Group I (Normal weight):** BMI 18.5–24.9 kg/m²
- **Group II (Overweight):** BMI 25.0–29.9 kg/m²
- **Group III (Obese):** BMI ≥30.0 kg/m²

Anthropometric measurements were recorded preoperatively, including height, weight, and waist circumference. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m²).

Sample Collection and Histological Processing

Gallbladder specimens were collected immediately after surgical excision. Each sample was rinsed in normal saline and fixed in 10% neutral buffered formalin for 24–48 hours. Standard paraffin embedding procedures were followed, and 4–5 µm thick serial sections were prepared for histopathological evaluation. Hematoxylin and eosin (H&E) staining was performed for general histology, while Masson's trichrome staining was used to assess fibrosis and collagen deposition [4].

The following histomorphological parameters were evaluated:

- **Wall thickness:** measured at three different points, avoiding fibrotic or artifact areas; average values were recorded.
- **Mucosal changes:** including hyperplasia, atrophy, pseudoglandular formations, and Rokitansky-Aschoff sinuses.
- **Muscularis propria alterations:** smooth muscle hypertrophy or thinning.
- **Fibrosis:** graded as mild, moderate, or severe based on Masson's trichrome staining intensity.
- **Inflammatory infiltration:** classified as acute or chronic and quantified using a semi-quantitative scoring system.

Two independent, board-certified pathologists evaluated all slides in a blinded fashion to avoid observational bias. Discrepancies in interpretation were resolved by consensus.

Imaging and Preoperative Evaluation

All patients underwent preoperative abdominal ultrasonography to assess gallbladder wall thickness and the presence of gallstones. Ultrasound imaging was performed by an experienced radiologist using a standardized protocol. Gallbladder wall thickness >3 mm was considered pathological based on standard diagnostic criteria [5].

Statistical Analysis

Data were analyzed using SPSS software version 25.0 (IBM Corp., Armonk, NY). Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range), while categorical variables were expressed as frequencies and percentages. The Shapiro-Wilk test was used to assess normality of data. One-way analysis of variance (ANOVA) followed by post hoc Tukey's test was used to compare continuous variables among the three BMI groups. Chi-square or Fisher's exact test was employed for categorical data comparisons. A p-value of <0.05 was considered statistically significant.

Results

A total of 90 patients were included in the final analysis, distributed equally across the three BMI-based study groups: 30 in the normal-weight group (Group I), 30 in the overweight group (Group II), and 30 in the obese group (Group III). The mean age across all participants was 46.2 ± 12.7 years, with no statistically significant difference in age or sex distribution among the groups ($p > 0.05$).

Gallbladder Wall Thickness

Gallbladder wall thickness increased progressively with higher BMI categories. The mean wall thickness was:

I. **2.4 ± 0.3 mm** in Group I (normal weight)
II. **3.1 ± 0.4 mm** in Group II (overweight)
III. **3.9 ± 0.6 mm** in Group III (obese)

The difference was statistically significant between all groups ($p < 0.001$, ANOVA with post hoc Tukey test). These findings suggest a positive correlation between BMI and gallbladder wall thickening, consistent with previous literature indicating chronic inflammation and altered motility in obesity [1].

Histopathological Changes

Microscopic examination of gallbladder wall sections revealed several structural abnormalities that were more prominent in overweight and obese individuals:

Mucosal hyperplasia was observed in:

- 10% of Group I
- 37% of Group II
- 70% of Group III ($p < 0.01$)

Smooth muscle hypertrophy (muscularis propria thickening) was present in:

- 13% of Group I
- 40% of Group II
- 67% of Group III ($p < 0.01$)

Subepithelial fibrosis, assessed using Masson's trichrome staining, was minimal in normal-weight patients but increased significantly with BMI. Moderate to severe fibrosis was found in:

- 7% of Group I
- 30% of Group II
- 63% of Group III ($p < 0.001$)

Chronic inflammatory infiltration was identified in:

- 20% of Group I
- 43% of Group II
- 80% of Group III ($p < 0.001$)

Presence of Rokitansky-Aschoff sinuses:

- Rare in Group I (7%)
- Common in Group II (33%)

- Frequently seen in Group III (53%)

These histological findings indicate that higher degrees of obesity are associated with progressively more severe morphological alterations in the gallbladder wall, including epithelial, muscular, and stromal components.

Correlation Analysis

Pearson correlation analysis revealed a strong positive correlation between BMI and gallbladder wall thickness ($r = 0.71$, $p < 0.001$), as well as between BMI and fibrosis score ($r = 0.64$, $p < 0.001$). No significant correlations were observed between age and histological severity ($p > 0.05$), suggesting obesity itself as the primary influencing factor.

Discussion

The present study aimed to explore the morphological alterations of the gallbladder wall across varying degrees of obesity. Our findings demonstrate a clear and statistically significant association between increased body mass index (BMI) and progressive structural changes in the gallbladder wall, including increased wall thickness, mucosal hyperplasia, smooth muscle hypertrophy, fibrosis, and chronic inflammation.

These results align with existing literature indicating that obesity is a well-established risk factor for gallbladder dysfunction and cholelithiasis. Previous studies have reported similar associations between obesity and gallbladder wall thickening, attributing these changes to chronic inflammation, altered bile composition, and gallbladder hypomotility [1,2]. In particular, Portincasa et al. highlighted that gallbladder emptying is impaired in obese individuals, resulting in bile stasis and increased wall stress, which may contribute to chronic mucosal irritation and fibrotic remodeling [3].

The observed increase in **mucosal hyperplasia** and **smooth muscle hypertrophy** in overweight and obese individuals supports the hypothesis of adaptive remodeling of the gallbladder wall in response to persistent functional overload and inflammation. These alterations have been previously described as precursors to more severe pathologies, such as gallstone formation and chronic cholecystitis [4]. Additionally, the presence of **Rokitansky-Aschoff sinuses**, which are often seen in chronically inflamed gallbladders, was more frequent in obese patients, suggesting long-standing structural stress and remodeling.

The progressive **fibrosis** observed in obese subjects may reflect chronic low-grade inflammation associated with adiposity. Adipose tissue, particularly visceral fat, is known to secrete pro-inflammatory cytokines such as TNF-α and IL-6, which contribute to systemic and local inflammation, including within the gallbladder wall [5,6]. This fibrotic response can compromise gallbladder compliance and exacerbate functional disturbances.

Interestingly, despite being a relatively simple organ, the gallbladder exhibits a dynamic histological response to metabolic stress, and our findings underscore its sensitivity to changes in body composition. The significant correlation between BMI and histological severity suggests that even in the absence of overt clinical symptoms, histological changes may already be underway in overweight and obese individuals.

This has important **clinical implications**. Gallbladder wall changes may precede gallstone formation and could serve as early histopathological markers of biliary tract disease in patients with obesity. Identifying these changes early could inform targeted strategies for weight reduction, dietary interventions, and possibly prophylactic cholecystectomy in high-risk populations.

Limitations

This study is not without limitations. Firstly, the sample size, though sufficient for detecting significant associations, may not capture all variability present in a broader population. Secondly, this was a cross-sectional study, which limits the ability to establish causality. Longitudinal studies would be required to determine whether gallbladder wall changes directly predict future biliary complications. Lastly, metabolic parameters such as insulin resistance or lipid profiles were not included, which could have further clarified the relationship between metabolic health and gallbladder pathology.

Conclusion

This study clearly demonstrates that obesity is significantly associated with progressive histopathological alterations in the gallbladder wall. As BMI increases, patients show a marked increase in wall thickness, mucosal hyperplasia, smooth muscle hypertrophy, fibrosis, and chronic inflammation. These structural modifications likely reflect both functional and inflammatory stress imposed by excess adiposity on the biliary system.

The findings reinforce the role of obesity not only as a metabolic risk factor but also as a contributor to subclinical gallbladder pathology that may precede overt gallstone formation or cholecystitis. The presence of histological changes even in overweight individuals suggests that pathological remodeling begins before clinical disease becomes apparent.

Early recognition of these gallbladder changes in obese populations can aid in risk stratification and potentially prompt earlier lifestyle interventions or prophylactic measures to prevent complications. Furthermore, our study supports integrating gallbladder evaluation into the broader spectrum of obesity-related organ assessments, especially for patients undergoing abdominal imaging or bariatric planning.

Future research should focus on longitudinal studies to determine the reversibility of gallbladder changes with weight loss and metabolic improvement, as well as the potential role of anti-inflammatory therapies targeting adipose-driven gallbladder inflammation.

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