Case Reports

Anatomical Variations of the Human Gallbladder and Their Proteomic and Clinical Implications: A Cadaveric Case Study

Kirubhanand Chandrasekaran[1] | Bharat Ramrao Sontakke[1] | Vishwajit R Deshmukh[1] | Shital Maske[1] | Snehal A. Deulkar[1] | Mythili Krishnan Rathinasabapathy[2] |

1. Department of Anatomy, All India Institute of Medical Sciences (AIIMS), Nagpur, Maharashtra, India – 441108
2. Department of Anatomy, Palakkad institute of medical sciences, Walayar, Palakkad, Kerala - 678624

Abstract

The gallbladder is an important component of the hepatobiliary system and exhibits several anatomical variations that may have significant clinical and surgical implications. Variations such as Hartmann's pouch, Phrygian cap deformity, septate gallbladder, and diverticula are often asymptomatic but may predispose individuals to bile stasis, gallstone formation, inflammation, and biliary obstruction. The present study analyses morphological variations of the human gallbladder observed in cadaveric specimens and correlates these anatomical findings with biochemical markers and potential protein involvement in gallbladder disease progression. Emphasis is placed on proteins involved in bile metabolism, inflammation, extracellular matrix remodeling, and cholesterol transport, which may contribute to gallstone formation and gallbladder structural changes. The study further proposes a hypothesis linking mucin proteins, inflammatory cytokines, and fibrosis-related proteins to the development of Hartmann's pouch, bile stasis, and gallstone formation. Additionally, a medical informatics framework is proposed for integrating anatomical, biochemical, proteomic, and imaging data to develop predictive models for gallbladder disease and surgical risk assessment. Understanding the relationship between anatomical variations and protein expression patterns may help in early diagnosis, personalized treatment strategies, and improved surgical planning. This study highlights the importance of integrating anatomy, biochemistry, proteomics, and medical informatics to better understand gallbladder diseases and their complications.

ISSN: 3108-2696 (Online)

1. Introduction

The gallbladder develops from the hepatic diverticulum of the foregut during the fourth week of embryological development and functions primarily as a reservoir for bile produced by the liver. Bile plays an essential role in fat digestion and cholesterol metabolism. The gallbladder concentrates bile by absorbing water and electrolytes and releases bile into the duodenum during digestion (Çerçi *et al.* 2009). Any anatomical variation or structural abnormality of the gallbladder may affect bile storage, concentration, and flow, leading to bile stasis and gallstone formation (Takahashi *et al.* 2026). Gallbladder anatomical variations are relatively common and include Hartmann's pouch, Phrygian cap deformity, septate gallbladder, bilobed gallbladder, hourglass gallbladder, and diverticula. These variations may be congenital or acquired (Yason *et al.* 2024). Although many of these anatomical variations are asymptomatic, they are clinically significant because they may predispose individuals to gallstones, cholecystitis, biliary obstruction, Mirizzi syndrome, mucocele, empyema, and fistula formation. Furthermore, these variations are important in laparoscopic cholecystectomy because they may increase the risk of bile duct injury and surgical complications (Jesani *et al.* 2022). Hartmann's pouch is a sacculation or diverticulum located at the neck of the gallbladder and is frequently associated with gallstone impaction and obstruction of the cystic duct or common hepatic duct. Gallstones impacted in Hartmann's pouch may cause external compression of the common hepatic duct, resulting in Mirizzi syndrome and obstructive jaundice. Chronic inflammation due to gallstone impaction may lead to fibrosis and structural changes in the gallbladder wall. Phrygian cap deformity is a congenital variation characterized by folding of the gallbladder fundus over the body of the gallbladder (Khan *et al.* 2020). This condition is usually asymptomatic but may cause bile stasis if the communication between the folded compartments is narrow. Bile stasis is one of the most important factors in gallstone formation. Recent studies suggest that gallstone formation and gallbladder diseases are not solely due to bile composition and anatomical variations but are also associated with protein expression changes, inflammatory pathways, cholesterol metabolism proteins, and extracellular matrix remodelling proteins (Sun *et al.* 2022). Mucin proteins secreted by the gallbladder epithelium play a major role in cholesterol crystal nucleation and gallstone formation. Inflammatory cytokines contribute to gallbladder inflammation and fibrosis, while extracellular matrix proteins contribute to structural changes in the gallbladder wall (Yoo *et al.* 2016). Advances in medical informatics, bioinformatics, and artificial intelligence allow integration of anatomical, biochemical, imaging, and proteomic data to develop predictive models for gallbladder disease and surgical risk assessment. Such integrative approaches may help in early diagnosis, personalized treatment, and improved surgical planning (Wang *et al.* 2025). The present study aims to analyse morphological variations of the gallbladder, correlate them with biochemical and protein-related mechanisms, and propose a protein-based hypothesis explaining the relationship between anatomical variations, gallstone formation, inflammation, and gallbladder structural changes (Yason *et al.* 2024). The study also proposes a medical informatics framework for integrating multidimensional data for predictive modeling of gallbladder diseases.

2. Materials and method

The present study was conducted on formalin-fixed human cadaveric gallbladder specimens obtained during routine anatomical dissection in the Department of Anatomy. The study was conducted over several years, and gallbladder specimens were examined for morphological variations, including size, shape, position, external morphology, and internal structural variations. This study is a descriptive cadaveric observation. No experimental biochemical or proteomic analysis was performed.

Received on	:	2026-01-04
Revised on	:	2026-03-01
Accepted on	:	2026-03-10
Published Online	:	2026-04-28
Review Model	:	Single-Blind Review
No. of Reviewers	:	Two
Edited by	:	Dr Shibojit Talukhdar
Vol and Issue	:	02 (02)
Page No	:	01-05
Plagiarism Level	:	12% and 18% (AI)
Correspondence	:	Dr. C. Kirubhanand
Contact Email	:	

Key Words:
Gallbladder anatomy
Hartmann's pouch
Phrygian cap deformity
Cadaveric study
Gallstone disease
Healthcare
Good Health and Well-Being

DOI: 10.64659/jomi/220427

The abdominal cavity was opened by making a vertical incision along the linea alba and rectus sheath. The peritoneal cavity was exposed, and the liver was lifted superiorly to visualize the gallbladder and extrahepatic biliary apparatus. The gallbladder was carefully dissected from the gallbladder fossa, preserving the cystic duct and surrounding structures **(Nagral 2005)**. Each gallbladder specimen was examined macroscopically for the presence of Hartmann's pouch, Phrygian cap deformity, septa, diverticula, mucosal folds, and gallstones. The gallbladder was opened longitudinally to examine the lumen, mucosal surface, and internal septations. The neck, body, and fundus were examined for structural abnormalities and gallstone impaction **(Khan *et al.* 2020)**. Biochemical markers associated with gallbladder disease were analysed from literature data and included bilirubin levels, alkaline phosphatase, liver enzymes (AST and ALT), cholesterol levels, bile salt composition, C-reactive protein, leukocyte count, ferritin levels, amylase, and lipase levels. These biochemical markers are associated with gallbladder obstruction, inflammation, gallstone formation, and biliary disease **(Cheng *et al.* 2025)**. Protein association analysis was performed based on literature evidence to identify proteins involved in mucin secretion, inflammation, cholesterol transport, bile salt transport, and extracellular matrix remodelling. These proteins were correlated with gallbladder anatomical variations and gallstone formation mechanisms. A conceptual medical informatics model was developed to integrate anatomical data, biochemical markers, proteomic data, imaging findings, and clinical parameters for predictive modeling of gallbladder disease progression and surgical risk assessment **(Liu *et al.* 2018)**.

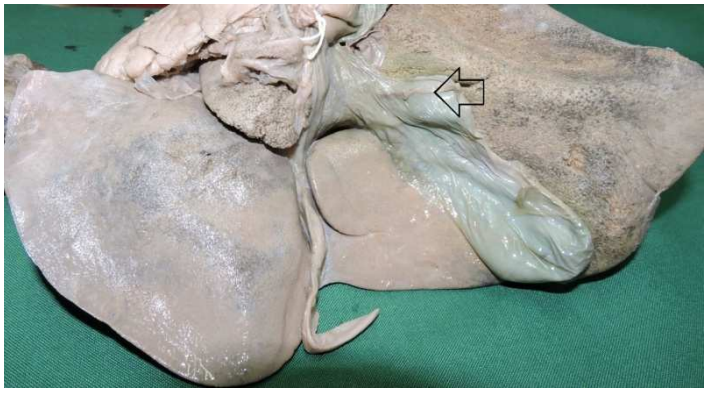

Figure 1. Cadaveric specimen showing Hartmann's pouch (arrow) at the neck of the gallbladder.

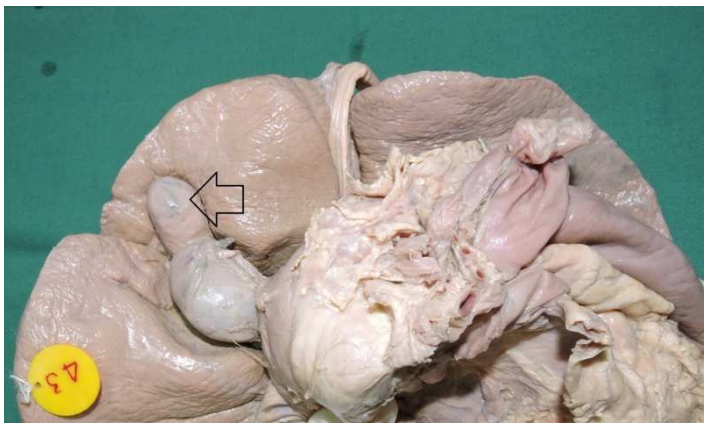

Figure 2. Cadaveric specimen showing Phrygian cap deformity (arrow), with folding of the fundus over the body.

3. Results

The morphological examination of the gallbladder specimens revealed several anatomical variations, including Hartmann's pouch, Phrygian cap deformity, and internal septations. These anatomical variations were observed during routine cadaveric dissection and were analysed for their clinical and biochemical significance. The variations identified are known to influence bile flow dynamics, gallstone formation, and biliary obstruction. Hartmann's pouch was observed as a sacculation arising from the neck region of the gallbladder. This diverticulum-like outpouching was located near the cystic duct junction and is considered a morphologic variation rather than a distinct anatomical structure. The presence of Hartmann's pouch is clinically significant because it is a common site for gallstone impaction. **Figure 1** shows a specimen demonstrating Hartmann's pouch located at the neck region of the gallbladder, appearing as a prominent sacculation projecting from the gallbladder wall. Another important anatomical variation observed was the Phrygian cap deformity, characterized by folding of the gallbladder fundus over the body of the gallbladder. This folding creates a mucosal fold that partially divides the gallbladder lumen into two communicating compartments. **Figure 2** shows a specimen demonstrating the Phrygian cap deformity, where the fundus of the gallbladder is folded over the body, creating a partial division of the gallbladder lumen.

4. Hypothesis of the study

The morphological variations of the gallbladder such as Hartmann's pouch, Phrygian cap deformity, septate gallbladder, and diverticulum formation have traditionally been considered congenital anatomical anomalies or consequences of mechanical obstruction caused by gallstones. However, recent advances in molecular biology, proteomics, and structural biology suggest that these anatomical variations may also be associated with protein expression changes, extracellular matrix remodelling, inflammatory signalling pathways, and bile metabolism proteins **(Takahashi *et al.* 2026)**. Therefore, gallbladder morphological variations may not be purely anatomical abnormalities but may represent structural and molecular adaptations mediated by protein-level changes in response to bile stasis, inflammation, and metabolic alterations **(Ding *et al.* 2023)**. One of the most important protein groups associated with gallbladder disease and gallstone formation is mucin proteins **(Hahm *et al.* 1992)**. The gallbladder epithelium secretes mucin glycoproteins such as MUC1, MUC2, MUC5AC, and MUC5B. These mucin proteins are large gel-forming glycoproteins that play a protective role in the gallbladder mucosal lining. Structurally, mucin proteins contain heavily glycosylated tandem repeat domains that allow them to form gel-like networks through disulfide bonds and intermolecular interactions **(Finzi *et al.* 2006)**. When bile stasis occurs due to anatomical variations such as Hartmann's pouch or septate gallbladder, mucin secretion increases as a protective response to epithelial irritation **(Zhao *et al.* 2024)**. The mucin gel matrix acts as a nucleation site for cholesterol crystals, leading to gallstone formation. From a structural biology perspective, mucin proteins create a three-dimensional polymeric network that traps cholesterol crystals, calcium bilirubinate, and bile salts, forming gallstones. Therefore, mucin protein overexpression may be a molecular link between bile stasis and gallstone formation. Another important group of proteins involved in gallbladder disease includes inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor alpha (TNF-α), transforming growth factor beta (TGF-β), and C-reactive protein (CRP) **(Nigam *et al.* 2023)**. When gallstones obstruct the cystic duct or Hartmann's pouch, inflammation occurs in the gallbladder wall. Inflammatory cytokines activate signalling pathways such as the NF-κB pathway, which leads to increased expression of extracellular matrix proteins and fibrosis-related proteins. From a structural biology perspective, cytokines bind to specific receptors and activate intracellular signalling cascades that regulate gene expression, protein synthesis, and extracellular matrix remodelling **(Adhikari *et al.* 2024)**. Chronic inflammation may therefore lead to thickening of the gallbladder wall, fibrosis, and diverticulum formation, which may explain the development of Hartmann's pouch in some cases as an acquired structural modification

rather than a purely congenital anomaly. Extracellular matrix proteins such as collagen type I, collagen type III, fibronectin, laminin, and elastin also play a significant role in gallbladder structural changes. Matrix metalloproteinases (MMP-2 and MMP-9) regulate extracellular matrix remodelling by degrading collagen and other matrix proteins. If extracellular matrix remodelling becomes dysregulated due to chronic inflammation, structural weakening of the gallbladder wall may occur, leading to diverticulum formation or outpouching such as Hartmann's pouch. Structural biology studies have shown that collagen fibers form triple helix structures that provide tensile strength to tissues **(Reghunath *et al.* 2020)**. Alterations in collagen cross-linking and extracellular matrix composition may change the mechanical properties of the gallbladder wall, making it susceptible to diverticulum formation and structural deformation. Cholesterol metabolism proteins also play a major role in gallstone formation. Proteins such as ABCG5, ABCG8, NPC1L1, apolipoprotein A1, and apolipoprotein B regulate cholesterol transport and secretion into bile. ABCG5 and ABCG8 are ATP-binding cassette transporters that transport cholesterol from hepatocytes into bile. Overexpression or increased activity of these transporters may lead to cholesterol supersaturation in bile, which is a major cause of cholesterol gallstone formation. From a structural biology perspective, ABC transporters contain transmembrane domains and nucleotide-binding domains that undergo conformational changes during ATP hydrolysis to transport cholesterol molecules across the membrane. Structural changes or mutations in these proteins may alter cholesterol transport efficiency and contribute to gallstone formation **(Kidambi and Patel 2008)**. Bile salt transport proteins such as the bile salt export pump (BSEP/ABCB11), multidrug resistance protein 3 (MDR3/ABCB4), and cystic fibrosis transmembrane conductance regulator (CFTR) also play an important role in bile composition and bile flow. Bile salts help keep cholesterol soluble in bile. If bile salt transport proteins are dysfunctional, bile salt concentration decreases, leading to cholesterol precipitation and gallstone formation. Structural biology studies have shown that these transport proteins function through conformational changes that allow bile salts and phospholipids to move across the membrane. Protein misfolding or altered protein expression may therefore contribute to bile composition imbalance and gallstone formation **(Sohail *et al.* 2021)**. Smooth muscle contraction proteins in the gallbladder wall also play a role in gallbladder emptying. Proteins such as actin, myosin, calmodulin, and myosin light chain kinase regulate gallbladder contraction. If gallbladder motility proteins are dysfunctional, gallbladder emptying may be impaired, leading to bile stasis. Bile stasis is one of the most important factors in gallstone formation and gallbladder inflammation. Structural biology studies show that actin and myosin interact through ATP-dependent conformational changes to produce muscle contraction. Alterations in these proteins may therefore affect gallbladder motility and contribute to bile stasis **(Portincasa *et al.* 2004)**. From a systems biology perspective, gallbladder anatomical variations, bile stasis, gallstone formation, inflammation, fibrosis, and diverticulum formation may be interconnected through protein-mediated pathways. The sequence of events may be described as follows: anatomical variation leads to bile stasis; bile stasis increases mucin secretion; mucin promotes cholesterol crystal nucleation and gallstone formation; gallstones cause obstruction and inflammation; inflammation activates cytokines and extracellular matrix remodeling proteins; extracellular matrix remodeling leads to fibrosis and structural changes; structural changes lead to diverticulum formation and further bile stasis. This creates a positive feedback loop that promotes gallbladder disease progression **(Cullen and Stalker 2016)**. From a structural biology perspective, this process involves multiple protein structural interactions including mucin polymer formation, cytokine-receptor binding interactions, collagen triple helix formation, matrix metalloproteinase catalytic activity, ABC transporter conformational changes, and actin-myosin interaction during gallbladder contraction. Therefore, gallbladder disease progression can be viewed as a protein structure–function relationship problem involving protein folding, protein interactions, membrane transport proteins, extracellular matrix proteins, and signalling proteins. In the context of medical informatics and bioinformatics, proteomic data, gene expression data, imaging data, and biochemical markers can be integrated to develop predictive models for gallbladder disease progression. Machine learning models could predict the risk of gallstone formation, Mirizzi syndrome, gallbladder inflammation, and surgical complications based on protein biomarkers, biochemical markers, and anatomical variations. Therefore, the proposed hypothesis suggests that gallbladder anatomical variations such as Hartmann's pouch and Phrygian cap are not purely anatomical anomalies but may be associated with protein expression changes, extracellular matrix remodelling, bile metabolism proteins, inflammatory cytokines, and smooth muscle proteins that collectively influence gallbladder structure, bile flow, gallstone formation, and gallbladder disease progression **(Manka *et al.* 2019)**. Understanding gallbladder diseases at the protein and structural biology level may provide new insights into disease mechanisms and may lead to the development of protein biomarkers and targeted therapies for gallbladder diseases.

5. Conclusion

Gallbladder anatomical variations such as Hartmann's pouch and Phrygian cap deformity are clinically significant because they may lead to bile stasis, gallstone formation, inflammation, and biliary obstruction. These conditions are associated with biochemical changes and protein expression changes related to bile metabolism, inflammation, cholesterol transport, and extracellular matrix remodelling. Understanding the relationship between anatomical variations, biochemical changes, and protein expression may help in early diagnosis, prevention, and treatment of gallbladder diseases. Medical informatics and artificial intelligence provide new opportunities for integrating anatomical, biochemical, proteomic, and imaging data to develop predictive models for gallbladder disease progression and surgical risk assessment. Future research should focus on proteomic analysis of gallbladder tissue, gene expression studies, and development of predictive models using machine learning and medical informatics approaches.

6. Disclosure Statements

6.1. Author Contribution

KC: Conceptualization, study design, data collection, manuscript drafting, and supervision. **BRS:** Data collection, anatomical analysis, and manuscript preparation. **VRD:** Data curation, literature review, and manuscript drafting. **SM:** Morphological analysis, data interpretation, and manuscript editing. **SAD:** Literature review, data validation, and manuscript preparation. **MKR:** Conceptual support, supervision, and critical revision of the manuscript. All authors have read and approved the final version of the manuscript.

6.2. Declaration of Generative AI

The authors used generative artificial intelligence tools solely for language editing and grammatical correction. The AI tools did not contribute to the study design, data collection, analysis, interpretation, or generation of scientific content. All content has been reviewed and approved by the authors, who take full responsibility for the integrity and accuracy of the work.

6.3. Ethics approval (for clinical/animal studies)

This cadaveric study utilized specimens obtained from routine anatomical dissections conducted under institutional guidelines for the ethical use of human donated bodies. No living subjects or identifiable personal data

were involved. Therefore, formal ethical approval was waived/not required as per institutional policy.

6.4. Informed Consent Statement

Informed consent was not required as the study utilized cadaveric specimens obtained through authorized institutional protocols for body donation. All procedures complied with ethical guidelines, and no identifiable personal information was used.

6.5. Data Availability Statement

The data supporting the findings of this study are derived from observations of cadaveric specimens obtained during routine anatomical dissection. All relevant data are included within the article. Additional details may be made available by the corresponding author upon reasonable request, subject to institutional and ethical guidelines.

6.6. Acknowledgment

The authors gratefully acknowledge the Karuna Medical College, Chittur, Palakkad, Kerala, India, for providing the cadaveric specimens and necessary facilities to carry out this study. The authors also express their sincere respect and gratitude to the individuals who donated their bodies for medical education and research, without whom this study would not have been possible.

6.7. Funding Statement

This research received no external funding. All work was conducted using institutional resources without dedicated grant support.

6.8. Conflicts of Interest

The authors declare that they have no known financial, personal, academic, or other relationships that could inappropriately influence, or be perceived to influence, the work reported in this manuscript. All authors confirm that there are no competing interests to declare.

6.9. Corresponding Author Contact Information

The corresponding author **Dr. Kirubhanand Chandrasekaran** can be contacted via email kirubhanand[at]aiimsnagpur.edu.in.

6.10. ORcID Information

Chandrasekaran	0000-0002-3525-2342
Sontakke	0000-0001-7935-0940
Deshmukh	0000-0002-6970-6699
Maske	0000-0002-0350-1471
Deulkar	0009-0004-5201-6466
Rathinasabapathy	0009-0007-8859-7794

6.11. Supplementary Information

No supplementary material is available for this article.

6.12. Handling Editor Information

This manuscript was handled by **Dr. Shibojit Talukdar**, Assistant Professor of Surgery, Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh, India.

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How to Cite: Chandrasekaran, K., Sontakke, B. R., Deshmukh, V. R., Maske, S., Deulkar, S. A., and Rathinasabapathy, M. K. (2026). Anatomical Variations of the Human Gallbladder and Their Proteomic and Clinical Implications: A Cadaveric Case Study. *Journal of Medico Informatics*, 02(02), 01–05. **doi:** https://doi.org/10.64659/jomi/220427