Gallbladder Diseases, Disorders, Complications and Management, Including Treatment

STATE INSTITUTE OF HEALTH AND FAMILY WELFARE
UTTAR PRADESH

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FOREWARD

Shri Partha Sarthi Sen Sharma

Principal Secretary
Department of
Medical Health and Family Welfare
Government of Uttar Pradesh

Continuing Medical Education (CME) module serves as a mechanism through which medical professionals can remain updated on the rapidly evolving practices in the field of medicine and surgery. Given the post COVID era, it has become increasingly important for medical officers to keep pace with the emerging modes of diagnosis and management, which are developed in response to feedback from the medical community.

Medical officers at the primary level encounter numerous challenges in managing and diagnosis of Gallbladder diseases and its complications. Continuous knowledge and skill enhancement are required to effectively address these challenges. However, due to their responsibilities in managing healthcare centers and implementing government policies, medical officers have limited time to dedicate to learning.

To address and rectify this situation, the State Institute of Health & Family Welfare (SIHFW) in Uttar Pradesh has developed a CME module specifically focused on Gallbladder Diseases, diagnosis and management for Medical Officers in the Provincial Health & Medical Services. This CME module has been created in collaboration with subject matter experts.

The module provides a comprehensive overview of recent developments in the modern diagnosis and management of Gallbladder Diseases. Its primary goal is to enhance the skills and knowledge of Medical Officers, ultimately leading to improved healthcare services for the general population.

I would like to extend my congratulations to SIHFW and the other subject matter experts involved in the development of this comprehensive module. I hope that this CME module will shed light on Gallbladder Diseases, Disorders, Complications and Management Including Treatment contribute to better healthcare outcomes.

(Partha Sarthi Sen Sharma)


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MESSAGE

Dr. Brijesh Rathor

Director General
Medical and Health Services
Uttar Pradesh

The effective diagnosis and management of Gallbladder Diseases plays a critical role in preserving lives and preventing severe health complications. Timely access to healthcare facilities equipped to handle health emergencies is instrumental in saving lives and minimizing physical impairments.

To address the needs of Medical Officers in the Provincial Health & Medical Services of Uttar Pradesh, the State Institute of Health & Family Welfare (SIHFW) has developed a comprehensive Continuing Medical Education (CME) program focused on Gallbladder Diseases, diagnosis and management. This program incorporates the latest advancements in the field and provides detailed guidance on essential diagnosis strategies for these conditions at the primary level. The aim is to educate the Medical Officers about the standards for Gallbladder Diseases that have changed greatly over the last few decades to facilitate optimal treatment of such patients.

It is expected that Medical Officers in Uttar Pradesh, after completing this CME program, will be able to enhance their service delivery by effectively diagnosis and managing such patients and also treating emergencies timely. As a result, communities will benefit from improved access to healthcare services, increased patient satisfaction, and enhanced population health. This CME program not only enhances clinical and technical expertise but also strengthens the provision of healthcare services and bridges the gap between theory and practice in healthcare management.

With the development of this CME module on Gallbladder Diseases, Diagnosis and Management for Medical Officers in Provincial Health and Medical Services in Uttar Pradesh, SIHFW will improve health services at PHC and CHC level. I extend my best wishes to the Director, Faculties and Research team at SIHFW and hope to see the publication of many more tailored CME modules in the future.

(Dr. Brijesh Rathor)


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MESSAGE

Dr. Shailesh Kumar Srivastava

Director General Family Welfare,
Directorate of Family Welfare
Uttar Pradesh

Gallbladder diseases, disorders, complications and management including treatment is very important in saving lives and serious health conditions. The reaching of an effected person to a center which has facilities for management of health-related emergencies helps in saving lives and physical impairment.

This module on Continuing Medical Education (CME) on Gallbladder diseases, disorders, diagnosis and management for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh provides a coherent and research-based insight to Gallbladder Diseases, disorder, management including treatment. It has been designed and written for Medical Officers and healthcare professionals and takes government perspective in consideration, drawing upon and comparing ideas and developments from national and international healthcare practices.

I hope that after this CME, Medical Officers in Uttar Pradesh will be able to scale up the services delivery in provide optimal treatment in their health facilities, thus benefitting communities. In addition to improving clinical and technical area of expertise, this CME will lead to providing improved access to health services and enhancing patient satisfaction and population health.

The director and team at State Institute of Health & Family Welfare, Uttar Pradesh and the team of experts of the field has done a commendable job by publishing this module on CME on Gallbladder Diseases, Disorders, Complications and Management Including Treatment for Medical Officers in Provincial Health & Medical Services in Uttar Pradesh. I hope the participants coming to attend their upcoming CME will take advantage of this initiative and make the most in their field with this handy module.

(Dr. Shailesh Kumar Srivastava)


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MESSAGE

Dr. Narendra Agarwal

Director General (Training)
Medical Health and Family Welfare
Uttar Pradesh

Continuing Medical Education (CME) modules serve as a means for healthcare professionals to stay updated on the rapidly evolving practices in the field of medicine and surgery. In the context of evolving Gallbladder Diseases, Disorders, Complications and Management Including Treatment, it has become increasingly crucial for medical officers to keep pace with the primary treatment and management.

Medical officers at the primary healthcare level face several difficulties in effectively diagnosis and treatment of Gallbladder Diseases conditions such as Gallstones and Cholecystitis, biliary dyskinesia, Gangrene, Cancer of the gallbladder, Cholangiopathy. Continuous acquisition of knowledge and skills is necessary to address these challenges. However, due to their responsibilities in running healthcare places and implementing government policies, medical officers have limited time available for pursuing additional education and skill development.

To address and resolve this issue, the State Institute of Health & Family Welfare (SIHFW), Uttar Pradesh, has developed a CME module focused on the Gallbladder diseases, disorders, diagnosis and management for Medical Officers in the Provincial Health & Medical Services in Uttar Pradesh. This CME module integrates the modern treatment of Gallbladder diseases, disorders, complications. Its primary goal is to enhance the skills and knowledge of Medical Officers, leading to improved healthcare services for the general population.

I would like to extend my congratulations to SIHFW and the subject matter experts involved in the development of this comprehensive module. I am optimistic that this CME module will illuminate on the effective treatment of Gallbladder diseases, disorders, complications and management.

(Dr. Narendra Agarwal)


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ACKNOWLEDGEMENT

Dr. Rajaganapathy R.
Director
State institute of
Health & Family Welfare
Government of Uttar Pradesh

The primary objective of Continuing Medical Education (CME) is to ensure the perpetual learning and advancement of Medical Officers in order to deliver optimal medical care to their patients. The purpose of CME is to aid Medical Officers in augmenting their performance in terms of patient care and satisfaction.

Gallbladder disease refers to any condition that affects the health of a human's gallbladder. The gall bladder is a small organ in the human digestive system. It stores some of the bile made by the human liver and sends it to the small intestine to help break down food. It does this through a series of pipes (bile ducts). Gallbladder disease may begin in the gall bladder itself or in the bile ducts associated with it. Any infection or blockage in these ducts can back up into the gallbladder. Because the bile ducts connect the gallbladder to other organs in the digestive system, gallbladder disease can affect these other organs as well.

Within the healthcare domain, there has been a noteworthy endeavor to emphasize the significance of effectively managing the treatment of gallbladder disease among Medical Officers in Provincial Health & Medical Services. It has been observed that gallbladder disease, diagnosis and management demonstrated superiority in short-term results. Hence, there is a requirement for a tailored CME program aimed at equipping Medical Officers in Uttar Pradesh with exposure to the latest advancements in the field of gallbladder disease diagnose.

In order to accomplish this objective and enhance knowledge, the training faculties and research team of the State Institute of Health and Family Welfare (SIHFW), Uttar Pradesh, in collaboration with the help of Head of Surgery (General) Prof. Abhinav Arun Sonkar and his team, King George's Medical University (KGMU), Lucknow have helped in the formulation of this CME module. It is anticipated that this CME module will be widely disseminated, and feedback on its efficacy will be received in the upcoming months.

(Dr. Rajaganapathy. R)


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CHAPTER - 1

ANATOMY OF GALL BLADDER AND BILIARY TREE

EMBRYOLOGY

By the end of the fourth week of embryogenesis, the hepatic diverticulum out pouches from the developing duodenum. The hepatic diverticulum goes on to become the biliary tree, while a second outpouching, known as the cystic diverticulum, immediately below develops into the gallbladder.

The gallbladder is a small, 7.5–12 cm long, with a normal capacity of about 25–30 mL, pear-shaped organ that is located inferior to the margin of the right lobe of the liver. It is divided into distinct segments: the fundus, body, infundibulum, and neck. The fundus is the expanded end of the gallbladder that projects away from the margin of the liver. It is anatomically associated with the anterior abdominal wall and hepatic flexure of the colon. The body of the gallbladder is attached to the liver by loose connective tissue superiorly. Inferiorly, the free margin abuts the duodenum and transverse colon. Superiorly, it is anatomically associated with segments IV and V of the liver. The neck of the gallbladder extends from the infundibulum to the cystic duct.

Its relationship to the liver varies from being embedded within the liver substance to being suspended by a mesentery. The gallbladder wall is composed of several layers. The innermost layer is made up of columnar epithelium arranged in a microvillous formation, somewhat like the lining of the intestine. The other layers are the lamina propria, smooth muscularis, and serosa. Rotitansky-Aschoff sinuses are deep inclusions from the mucosal layer extending into the muscularis layer.

The muscle fibres in the wall of the gallbladder are arranged in a criss-cross manner, being particularly well developed in its neck. The mucous membrane contains indentations (crypts of Luschka) that sink into the muscle coat.

The cystic duct is about 3 cm in length, but this is variable. Its lumen is 1–3 mm in diameter; its mucosa is arranged in spiral folds (valves of Heister); and the wall is surrounded by the sphincter of Lütkens. The cystic duct joins the common bile duct along its course from the liver to the duodenum. The level of the juncture and course of the cystic duct can vary.

The common hepatic duct is usually less than 2.5 cm long and is formed by the union of the right and left hepatic ducts. The common bile duct (CBD) is about 7.5 cm long and is formed by the junction of the cystic and common hepatic ducts. It is divided into four parts:

1. supraduodenal portion, about 2.5 cm long, runs in the free edge of the lesser omentum;
2. retroduodenal portion;
3. infraduodenal portion, lies in a groove, at times in a tunnel, on the posterior surface of the pancreas;
4. intraduodenal portion, passes obliquely through the wall of the second part of the duodenum, where it is surrounded by the sphincter of Oddi and terminates by opening on the summit of the ampulla of Vater.

Blood Supply

The gallbladder is supplied by the cystic artery, which is a branch of the right hepatic artery. The cystic artery courses along the cystic duct and divides at the neck of the gallbladder into superficial and deep branches, which supply the anterior/inferior and posterior/superior aspects of the gallbladder, respectively. The cystic artery or an accessory cystic artery can arise from the right hepatic, left hepatic, or common hepatic artery. (Fig 3) The venous drainage of the gallbladder generally parallels the course of the arteries draining into the

cystic vein, which drains into the gallbladder fossa directly into the middle hepatic vein (ie, it does not accompany the cystic artery).

NERVE SUPPLY

The sympathetic nerves that supply the gallbladder are derived from the ninth thoracic segment and from the celiac plexus. The gallbladder and cystic duct receive innervation from the following three nerves –

1) the right phrenic nerve conveys sensory information,

2) the hepatic branch of the right vagus nerve provides parasympathetic innervation, and

3) the celiac plexus provides sympathetic information

The primary function of the gallbladder is the storage of bile, a fluid produced by the liver that aids with the digestion of fat. Bile is released into the duodenum in response to cholecystokinin (CCK), the major hormone responsible for gallbladder contraction and pancreatic enzyme secretion. CCK is produced in discrete endocrine cells that line the mucosa of the small intestine.

LYMPHATIC DRAINAGE

The lymphatic drainage of the gallbladder is via several pathways and does not always follow a predictable drainage pattern . In some cases, lymph nodes associated with gallbladder cancer can first be seen posterior to the pancreas or portal vein.

The cholecysto-retropancreatic pathway is the principal pathway and drains along the cystic duct (cystic nodes), common bile duct (pericholedochal nodes, station 12b), portal vein (portocaval node, station 12p), and posterior pancreas (superior retropancreatic node, station 13) to the paraaortic region (paraaortic nodes, station 16).

The cholecysto-celiac pathway, the next most common pathway, drains along the cystic duct (cystic nodes) and medially along the hepatoduodenal ligament (station 12a) superior to the head of the pancreas to the portal vein (station 12), hepatic artery (station 8), and celiac axis (station 9). Among lymph nodes along this route, one study found that the posterior common hepatic node was most frequently stained (45 percent).

The cholecysto-mesenteric pathway drains to the left in front of the portal vein (station 12) connecting with the nodes at the root of the mesentery (superior mesenteric nodes, station 14).

SURGICAL CONSIDERATION:

The triangle of Calot (Fig1) is of surgical importance and has the following boundaries: 1) cystic duct on the right, 2) common hepatic duct on the left, and 3) the undersurface of the liver superiorly. The original definition of this triangle from 1891 listed the cystic duct, common hepatic duct and the cystic artery as boundaries. This has since been modified to allow better identification of landmarks for locating the cystic artery. Of surgical importance is that within the modern definition of the triangle of Calot runs the cystic artery and lymph node. Sometimes, the gallbladder neck is connected to the first part of the duodenum with loose fibrous bands known as the peritoneal cholecystoduodenal fold.

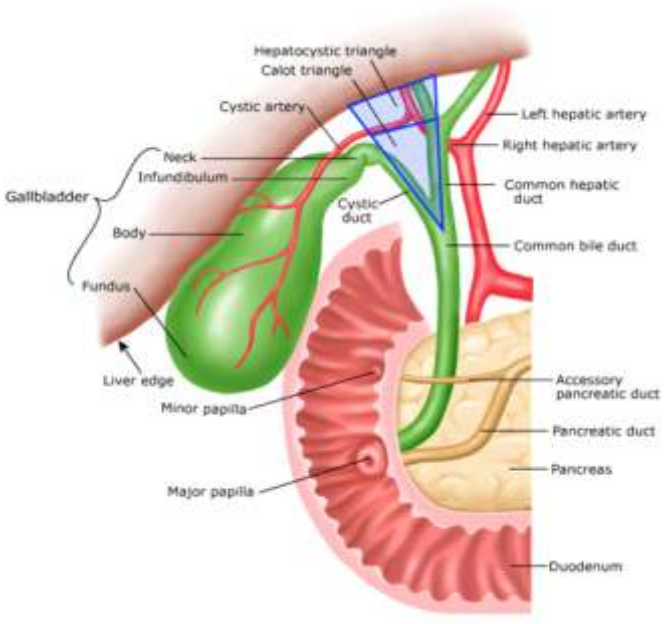

Fig. 1

***Image courtesy*: Surgical management of gall bladder, 2024 UpToDate, Inc. and/or its affiliates**

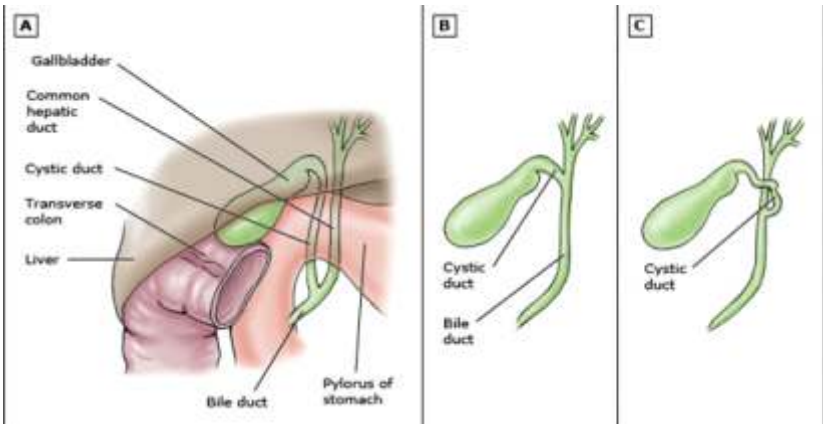

Fig. 2

Image courtesy: Moore KL, Dalley AF. Clinical Oriented Anatomy, 4th ed, Lippincott Williams & Wilkins, Baltimore 1999. Copyright © 1999 Lippincott Williams & Wilkins.

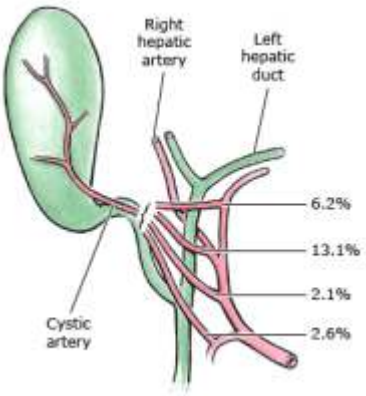

Fig. 3

Image courtesy: Moore KL, Dalley AF. Clinically Oriented Anatomy, 4th ed, Lippincott Williams & Wilkins, Baltimore 1999. Copyright © 1999 Lippincott Williams & Wilkins.

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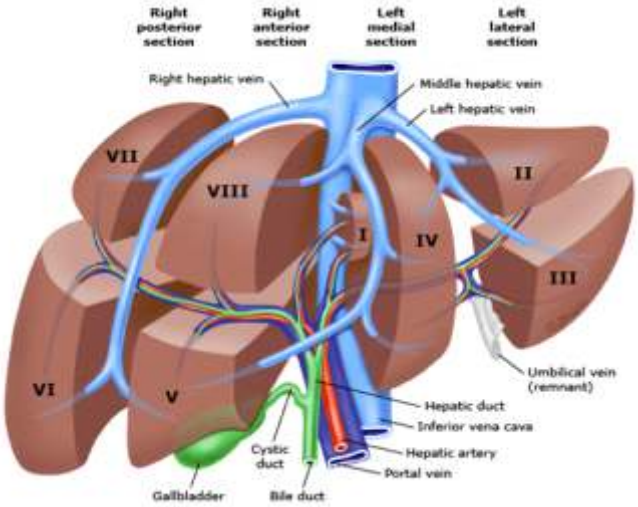

Fig. 4

Drawing depicting the functional segments of the liver (Couinaud segments). Segments II to IV make up the left lobe, and segments V to VIII constitute the right lobe.
Image courtesy: Surgical management of gall bladder, 2024 UpToDate, Inc. and/or its affiliates

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CHAPTER - 2

BENIGN GALL BLADDER DISEASES

Gallstone Disease (Cholelithiasis):

Gallstone disease, also known as cholelithiasis, is a prevalent condition affecting the biliary system. It involves the formation of hard, crystallized deposits called gallstones within the gallbladder. Cholelithiasis is the most common disease of gallbladder and biliary tree, affecting 10% to 15% of the population.

Types of Gallstones:

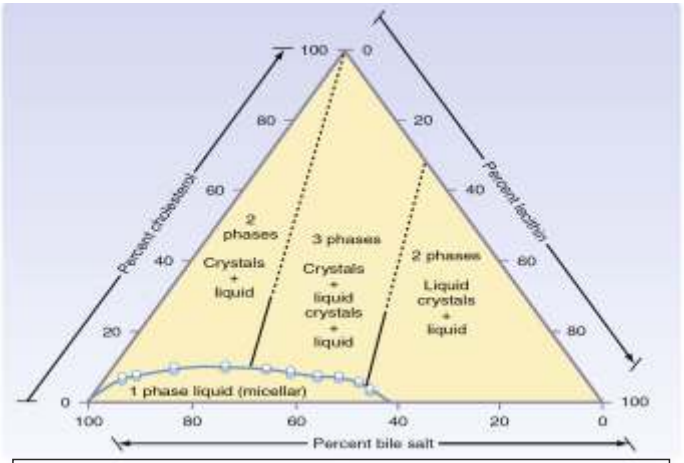
There are two main types of gallstones:

- **Cholesterol Gallstones:** These are the most common type in Western societies, typically containing a mix of cholesterol, calcium salts, bile acids, and other substances. Formation is linked to high cholesterol levels, high-calorie diets, obesity, and certain medications.
- **Pigment Gallstones:** These are less common in the West but more prevalent in Asia. They contain less cholesterol and are further categorized into:
    - **Black Pigment Stones:** Associated with conditions like hereditary spherocytosis and sickle cell disease that cause increased hemolysis causing concentration of bilirubin.
    - **Brown Pigment Stones:** Occur within the biliary tree and suggest a disorder of biliary motility and associated bacterial infection.

Pathophysiology of Gallstone Formation:

Four key factors in gallstone formation:

1. **Supersaturation of Bile with Cholesterol:** When cholesterol levels in bile exceed its solubility limit, it precipitates as crystals. High cholesterol secretion or decreased bile salt concentration can contribute to this supersaturation.
2. **Impaired Bile Flow (Gallbladder dysmotility):** Stagnant bile creates more time for cholesterol crystals to aggregate and form stones. Conditions like prolonged fasting, vagotomy, or somatostatin therapy can contribute to stasis.
3. **Concentration of bile in gallbladder:** Once in the gallbladder, bile is concentrated further through the absorption of water and sodium, increasing the concentrations of the bile solutes and calcium. Bile salts act to solubilize cholesterol. With respect to cholesterol stones cholesterol precipitates out into crystals when the concentration in the gallbladder vesicles exceeds the solubility of cholesterol.



Triangle of solubility. With the three major components of bile that determine cholesterol solubility and stability, each can be quantified by molar percentage to show a a relative ratio to the other two.
(From Admirand WH, Small DM. The physicochemical basis of cholesterol gallstone formation in man. J Clin Invest. 1968;47:1043-1052.)

4. **Nucleation:** Crystal formation is further accelerated by pro- nucleating agents, including glycoproteins and immunoglobulins.

Clinical presentation:

Gallstones are mostly asymptomatic (no symptoms) in most individuals. However, when a gallstone blocks a bile duct, it can cause various symptoms, including:

- **Pain:** The most common symptom is sudden and severe pain in the upper right abdomen (biliary colic). This pain may last for minutes to hours and often worsens after eating fatty foods.
- **Nausea and Vomiting:** These frequently accompany abdominal pain.
- **Fever:** This is a sign of inflammation, often pointing towards complication like acute calculous cholecystitis.
- **Dyspepsia:** Symptoms like bloating, indigestion, and food intolerance, particularly to fatty foods, can occur.

Complications:

Untreated gallstones can lead to serious complications, including:

- **Acute Cholecystitis:** Inflammation of the gallbladder due to a blocked bile duct. This causes intense pain, fever, nausea, and vomiting and requires immediate medical attention.
- **Empyema of the Gallbladder:** A pus collection within the inflamed gallbladder, a complication of acute cholecystitis.
- **Gallstone Ileus:** A rare complication where a large gallstone migrates to the small intestine and causes blockage.
- **Cholangitis:** Inflammation of the bile ducts due to a gallstone blockage. This can cause severe pain, fever, chills, and jaundice.
- **Pancreatitis**

Diagnosis:

1. Accurate medical history and examination
2. **USG:** The investigation of choice for gall stones, USG is highly specific and sensitive. The gall stones show up as bright echoes and can be seen moving with postural change.
3. **MRI/ MRCP:** Heavily T2 weighted images can be used to diagnose gall stones and also give a clearer picture of the hepatobiliary tract as well as provide information about hepticolithiasis and choledocholithiasis. However, its high cost make it an adjunctive investigation and not a primary one.
4. **CT scan:** Although not as sensitive as a USG for diagnosing gall stones, CT scan can often provide useful information regarding the hepatobiliary anatomy and can also assist in making a diagnosis associated with the complications of gall stones.

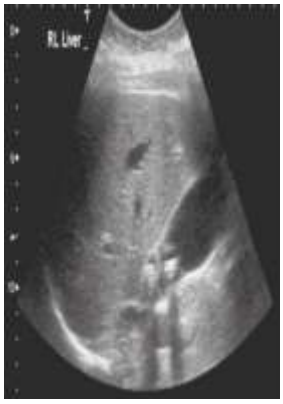
Treatment:

1. Medical treatment of gallstones is generally unsuccessful and includes oral bile salt therapy, contact dissolution that requires cannulation of the gallbladder and infusion of organic solvent,and extracorporeal shock wave lithotripsy. With the dissolutionstrategies, unacceptable recurrence rates of up to 50% limit their application to the most select group of patients. Extracorporeal shock wave lithotripsy has a lower recurrence rate, approximately 20%, and can be used in patients with single stones 0.5 to 2 cm in size.
2. Surgical treatment includes laparoscopic/open cholecystectomy. Due to ease, safety and relatively

early discharge, laparoscopic cholecystectomy has become the standard of care for symptomatic gallstones.

Only 20% to 30% of patients with asymptomatic stones will develop symptoms within 20 years, and because approximately 1% of patients with asymptomatic stones develop complications of their stones before onset of symptoms, prophylactic cholecystectomy is not warranted in asymptomatic patients.

Gallbladder with characteristic yellow cholesterol stones.



Ultrasound examination. Gallstones in neck of gallbladder with acoustic shadowing.

(Thoracic key, chap 55, biliary system, Patrick G. Jackson, Steven R.T. Evans, Fig 55-20)

Cholecystitis:

2.1 Acute Calculous Cholecystitis:

Cause:

- A gallstone blocking the cystic duct.

Pathogenesis:

1. **Obstruction:** The gallstone blocks the cystic duct, preventing bile from leaving the gallbladder.
2. **Inflammation:** Bile accumulates, causing irritation and inflammation of the gallbladder wall.
3. **Ischemia and Necrosis:** If the blockage persists, blood flow is compromised, potentially leading to tissue death (necrosis) in severe cases.
4. **Secondary Infection:** Stagnant bile creates a breeding ground for bacteria, leading to potential life-threatening infection (e.g. acute mphysematous cholecystitis).

Clinical Presentation:

Symptoms:

- **Sudden and severe pain:** Often in the upper right abdomen.
- **Nausea and vomiting**
- **Fever:** Suggests potential infection
- **Loss of appetite**
- **Jaundice**

Examination:

- Tenderness in right hypochondrium
- Guarding in right hypochondrium
- Voluntary cessation of respiration when the examiner exerts constant pressure under the right costal margin, known as a Murphy sign, suggests inflammation of the visceral and parietal peritoneal surfaces.

Demonstration of Murphy's sign;
From EPONYMOUS SIGNS OF ACUTE CHOLECYSTITIS -
A REVIEW by Sajad Ahmad Salati
Qassim University

Classification:

Grade III (severe) acute cholecystitis	
Associated with dysfunction of any one of the following organs/ systems:	
1 Cardiovascular dysfunction	Hypotension requiring treatment with dopamine ≥5 μg/kg/min, or any dose of epinephrine
2 Neurological dysfunction	Decreased level of consciousness
3 Respiratory dysfunction	PaO_2/F_iO_2 ratio <300
4 Renal dysfunction	Oliguria; creatinine >2.0 mg/dL
5 Hepatic dysfunction	Prothrombin time (PT-INR) >1.5
6 Haematological dysfunction	Platelet count <100 000/mm^3
Grade II (moderate) acute cholecystitis	
Associated with any one of the following conditions:	
1 Elevated white cell count (>18 000/mm^3)	
2 Palpable tender mass in the right upper abdominal quadrant	
3 Duration of complaint >72 hours	
4 Marked local inflammation (gangrenous cholecystitis, pericholecystic abscess, hepatic abscess, biliary peritonitis, emphysematous cholecystitis)	
Grade I (mild) acute cholecystitis	
Does not meet the criteria of grade II or grade III acute cholecystitis. Grade I can also be defined as acute cholecystitis in a healthy person with no organ dysfunction and mild inflammatory changes in the gallbladder, making cholecystectomy a safe and low-risk operative procedure	

PaO_2/F_iO_2 ratio is the ratio of arterial oxygen partial pressure (PaO_2 in mmHg) to fractional inspired oxygen (F_iO_2) expressed as a fraction (not a percentage) at sea level, the normal PaO_2/F_iO_2 ratio is ~400–500 mmHg (~55–65 kPa); PT-INR, prothrombin time–international normalised ratio.

Tokyo classification of acute calculous cholecystitis; from Yokoe M et al. Tokyo Guidelines 2018: diagnostic criteria and severity grading of acute cholecystitis (with videos). J Hepatobiliary Pancreat Sci 2018; ***25****(1) 41-54*

Diagnosis:

- **Ultrasound:** Transabdominal ultrasonography is a sensitive, inexpensive, and reliable tool for the diagnosis of acute cholecystitis, with a sensitivity of 85% and specificity of 95%.

  USG findings:

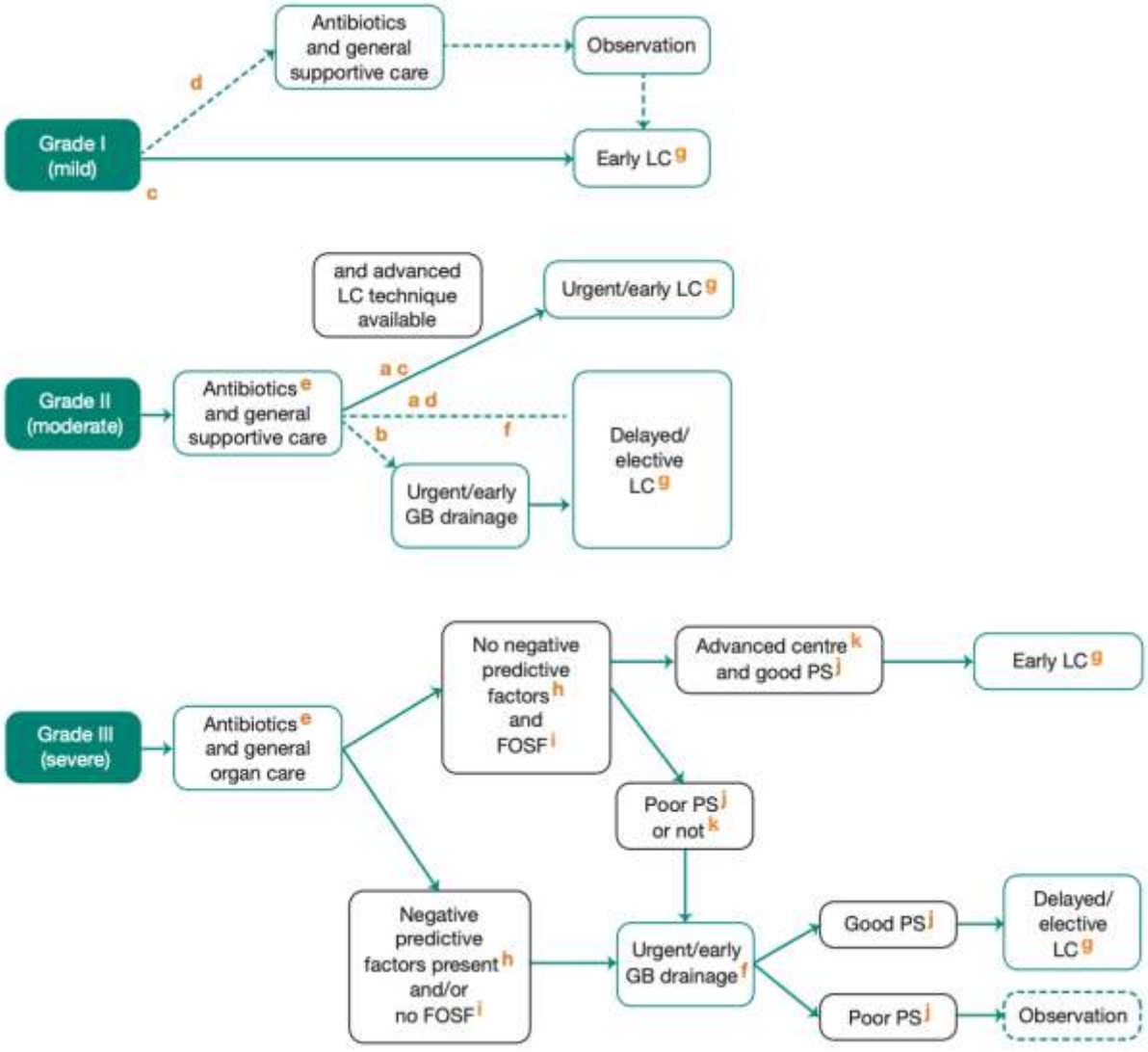
  i. Pericholecystic fluid

  ii. Gallbladder wall thickening

  iii. Sonographic Murphy's sign (probe tenderness)

- **HIDA Scan (Gold Standard):** Non visualisation of gall bladder on HIDA scan is diagnostic of acute cholecystitis. Filling of the gallbladder during a HIDA scan essentially eliminates the diagnosis of cholecystitis.
- **CT Scan:** Provides detailed abdominal images, used in complex cases or unclear ultrasound findings.

Treatment:

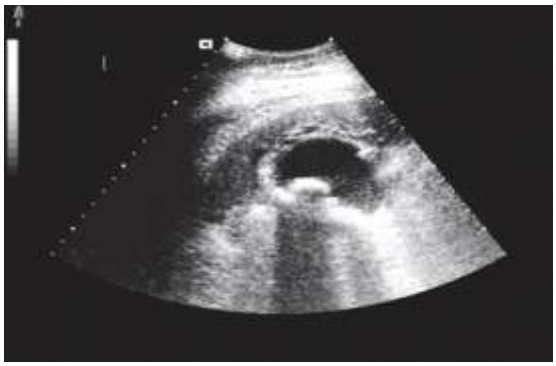


Tokyo Guidelines for the management of acute cholecystitis

Yokoe M et al. Tokyo Guidelines 2018: diagnostic criteria and severity grading of acute cholecystitis (with videos). J Hepatobiliary Pancreat Sci 2018; 25(1) 41-54

Acute Acalculous Cholecystitis:

Acute acalculous cholecystitis (AAC) is an inflammatory condition of the gallbladder that occurs in the absence of gallstones. It is a relatively uncommon but potentially life-threatening complication often encountered in critically ill patients.



Transverse view of the gallbladder on ultrasound in a patient with calculous cholecystitis, revealing gallstones and gallbladder wall thickening.

(Cholecystitis, Kaitlyn Kelly, Sharon Weber, Fig 31.1)

Pathophysiology:

The exact mechanisms underlying AAC remain unclear. However, several factors are believed to contribute:

- **Bile Stasis:** Impaired gallbladder emptying (dyskinesia) due to critical illness, medications, or prolonged fasting can lead to bile stasis and promote inflammation.
- **Ischemia:** Reduced blood flow to the gallbladder caused by shock or vasoconstrictive states can compromise tissue viability and trigger an inflammatory response.
- **Inflammatory Mediators:** Systemic inflammatory processes associated with critical illness may affect the gallbladder through circulating cytokines and other inflammatory mediators.
- **Bacterial Infection:** In some cases, bacterial translocation from the gut or biliary tract can contribute to AAC.

Risk Factors:

- **Age:** Older adults are more susceptible.
- **Critical Illness:** Sepsis, major surgery, trauma, and burns can significantly increase the risk.
- **Prolonged TPN (Total Parenteral Nutrition):** This can lead to bile stasis.
- **Immunosuppression:** Patients with weakened immune systems are more prone to infections that might contribute to AAC.
- **Diabetes:** Diabetics may have impaired gallbladder emptying.

Clinical Presentation:

- AAC often presents with similar symptoms to acute calculous cholecystitis, including:
    - Right upper quadrant pain (may be absent or atypical in critically ill patients)
    - Nausea and vomiting
    - Fever (may be absent in critically ill patients)
    - Right upper quadrant tenderness (may be unreliable in critically ill patients)
- The atypical presentation in critically ill patients can make diagnosis challenging. A high index of suspicion is crucial, particularly for unexplained right upper quadrant pain or fever.

Diagnosis:

- **History and Physical Examination:** A detailed medical history focusing on risk factors, medications, and the presence of critical illness is vital. Physical examination findings may be unreliable in critically ill patients.

- **Imaging Studies:**
    - **Ultrasound:** The most useful investigation for visualizing gallbladder wall thickening, pericholecystic fluid collection, and absence of gallstones.
    - **HIDA Scan (Hepatobiliary Iminodiacetic Acid Scan):** Non-visualization of the gallbladder on HIDA can be suggestive of AAC, but interpretation requires caution due to potential limitations in critically ill patients (e.g., impaired hepatic function).
    - **CT Scan:** Provides detailed abdominal images but may not be the first-line option due to radiation exposure concerns.
    - **MRCP (Magnetic Resonance Cholangiopancreatography):** Can be helpful in excluding choledocholithiasis (gallstones in the bile duct).

Complications:

- AAC can lead to severe complications like:
    - Gangrene and perforation of the gallbladder
    - Emphysematous cholecystitis (gas in the gallbladder wall)
    - Sepsis
    - Multi-organ dysfunction syndrome (MODS)

Treatment:

- **Early Intervention:** Prompt diagnosis and treatment are critical in AAC due to its potential for rapid deterioration.
- **Conservative Management:** This may involve:
    - Intravenous fluids for hydration
    - Electrolyte correction
    - Broad-spectrum antibiotics to address potential bacterial infection
    - Pain management
    - Nutritional support
- **Cholecystectomy:** Laparoscopic cholecystectomy is the preferred approach when the patient's clinical condition permits surgery. This offers definitive treatment by removing the inflamed gallbladder.
- **Percutaneous Cholecystostomy:** In high-risk surgical candidates, drainage of the gallbladder through a catheter (percutaneous cholecystostomy) may be considered as a bridge to definitive surgery. This less invasive procedure can provide immediate relief and allow for patient stabilization before definitive surgery.

Chronic cholecystitis:

Chronic cholecystitis refers to long-term inflammation of the gallbladder.

Pathophysiology:

- Recurrent episodes of **biliary colic**, caused by temporary blockage of the cystic duct by gallstones, are a major contributor to chronic cholecystitis.
- These repeated episodes lead to inflammation and scarring of the gallbladder neck and cystic duct, ultimately resulting in fibrosis (thickening and hardening) of the gallbladder wall.

Clinical Presentation:

- Chronic cholecystitis often presents similarly to **symptomatic cholelithiasis** (gallstones causing symptoms).
- Key presenting features include:
    - Pain:
        - Location: Right upper quadrant or epigastrium (upper middle abdomen)
        - Timing: Often occurs after fatty meals, but the association may not always be present.
        - Character: Aching, constant pain rather than sharp, cramping ("colicy") pain.
        - Duration: Typically lasts a few hours. Pain exceeding 24 hours or accompanied by fever suggests acute cholecystitis.
    - Other gastrointestinal symptoms: Nausea, vomiting, and bloating may accompany the pain.

Diagnosis:

- A detailed medical history focusing on symptoms suggestive of biliary tract disease is crucial for diagnosis.
- **Transabdominal ultrasound** is the investigation of choice for chronic cholecystitis. It can effectively identify:
    - **Gallstones:** Their presence is a major indicator of chronic cholecystitis.
    - **Gallbladder wall thickness:** Thickening of the gallbladder wall can support the diagnosis.
    - **Other gallbladder abnormalities:** Ultrasound can detect potential complications like CBD dilation, gallbladder polyps, or cholesterolosis (cholesterol accumulation in the gallbladder).
- In some cases, even without visible gallstones, the presence of **gallbladder sludge** on ultrasound, along with appropriate symptoms, can be suggestive of biliary colic and possible chronic cholecystitis.

Treatment:

- A balanced approach to treatment based on symptom severity and risk of complications is adviced.
- **Elective laparoscopic cholecystectomy:**
    - This is the definitive treatment for chronic cholecystitis with moderate to severe symptoms.
- **Observation:**
    - For patients with mild symptoms and a low complication rate from gallstones (less than 3% per year), observation might be considered initially.
    - This approach often involves dietary and lifestyle modifications to manage symptoms, such as avoiding fatty foods that can trigger biliary colic.

Decision-Making:

- The decision to pursue cholecystectomy or observation depends on a careful evaluation of:
    - Symptom severity and frequency
    - Potential risks associated with surgery
    - Patient's overall health and preferences
- For patients with severe or recurrent symptoms and a higher complication rate from gallstones (more than 7% per year), elective laparoscopic cholecystectomy is generally recommended to prevent future complications and improve quality of life.

Xanthogranulomatous Cholecystitis:

Xanthogranulomatous cholecystitis (XGC) is a rare inflammatory disorder of the gallbladder characterized by a destructive inflammatory process with the accumulation of lipid-laden macrophages (xanthoma cells) and fibrosis. This condition can be challenging to diagnose due to its non-specific presentation and its ability to mimic gallbladder cancer on imaging studies.

Epidemiology:

- XGC is an uncommon variant of chronic cholecystitis.
- It is more prevalent in females and individuals over 60 years old.
- A higher prevalence has been reported in India and Japan.

Pathophysiology:

The exact cause of XGC remains unclear, but several factors are believed to contribute:

- **Bile extravasation:** Leakage of bile into the gallbladder wall from ruptured Rokitansky-Aschoff sinuses or mucosal ulceration is thought to initiate the inflammatory process.
- **Lipid accumulation:** Extravasated bile and cholesterol crystals can lead to the formation of xanthoma cells.
- **Chronic inflammation:** A sustained inflammatory response contributes to tissue destruction and fibrosis.

Clinical Presentation:

- XGC often presents with symptoms similar to other types of cholecystitis, including:
    - Right upper quadrant pain
    - Nausea and vomiting
    - Fever
    - Right upper quadrant tenderness
- Due to the non-specific nature of these symptoms, preoperative diagnosis of XGC can be challenging.

Diagnosis:

- **Ultrasound:** May show thickening of the gallbladder wall and sometimes gallstones. However, these findings are not diagnostic for XGC.
- **CT Scan:** Can reveal intramural nodules within the gallbladder wall, but differentiation from other conditions like cancer can be difficult.
- **MRI Scan:** May be helpful in some cases, but limitations include cost and accessibility.
- **HIDA Scan (Hepatobiliary Iminodiacetic Acid Scan):** Non-visualization of the gallbladder can be suggestive of XGC, but interpretation requires caution due to potential limitations in some patients.
- **Gallbladder Biopsy:** This is the most definitive diagnostic tool; however, it is rarely performed before surgery due to the risk of complications.

Differential Diagnosis:

- Gallbladder cancer: The biggest challenge in diagnosing XGC is its close resemblance to gallbladder cancer on imaging studies.
- Acute acalculous cholecystitis

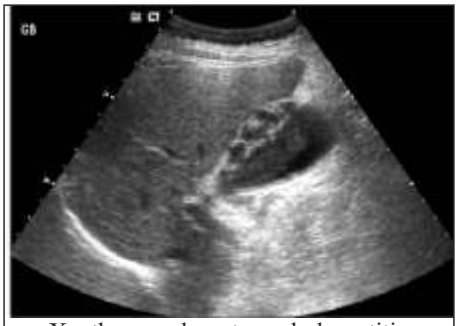
- Chronic cholecystitis with complications

Treatment:

- **Cholecystectomy (surgical removal of the gallbladder):** This is the definitive treatment for XGC. Laparoscopic cholecystectomy is the preferred approach whenever possible.

Challenges and Considerations:

- **Preoperative diagnosis:** The difficulty in differentiating XGC from gallbladder cancer preoperatively necessitates a high index of suspicion, especially in elderly patients with atypical presentations.
- **Frozen-section examination:** During surgery, frozen-section examination of a gallbladder tissue sample can help distinguish XGC from cancer. Coexistence of XGC and gallbladder cancer has been reported in a small percentage of cases.
- **Open cholecystectomy:** If a high suspicion of XGC exists preoperatively due to diagnostic difficulties, open cholecystectomy might be considered to allow for better visualization and tissue sampling



Xanthogranulomatous cholecystitis (Adapted from Semantic Scholar, xanthogranulomatous cholecystitis-appearance on USG)

3. Cholecystoses:

Cholecystoses encompass a spectrum of benign gallbladder conditions characterized by abnormal growth of gallbladder tissues without significant inflammation. These conditions can affect the mucosal lining, muscular wall, or both, and often present a challenge in differentiating them from more serious pathologies.

Types of Cholecystoses:

- **Cholesterolosis (strawberry gallbladder):** This is the most frequent type of cholecystosis. It involves the accumulation of cholesterol and other lipids within the gallbladder mucosa. Manifestations include:
    - **Focal or diffuse thickening of the gallbladder wall** on imaging studies.
    - **Multiple small, non-shadowing polyps (usually <10 mm) on ultrasound.**
    - **Characteristic "strawberry gallbladder" appearance:** This refers to yellow deposits on a background of inflamed mucosa, visible during surgery or on pathology.
    - **Possible association with cholesterol gallstones.**
- **Cholesterol polyps:** These are benign protrusions of the gallbladder wall containing cholesterol deposits. They are usually small (1-10 mm) and carry no malignant potential.
- **Adenomyomatosis:** This condition involves overgrowth of the gallbladder mucosa and thickening of the muscular wall. It can manifest as:
    - **Cyst-like structures within the gallbladder wall.**
    - **Polypoid projections from the mucosa.**
    - **Intramural diverticula (Rokitansky-Aschoff sinuses) that can trap bile and cholesterol.** These can be identified with a characteristic "comet tail" artifact on ultrasound.
    - **Adenomyomatosis is often associated with chronic inflammation and gallstones.**

Clinical Presentation:

Cholecystoses are frequently asymptomatic and discovered incidentally during investigations for other abdominal conditions. However, some patients might experience symptoms similar to biliary colic, including:

- Right upper quadrant pain
- Nausea and vomiting
- Dyspepsia (indigestion)

Diagnosis:

- **Ultrasound:** Ultrasound is the preferred modality for initial evaluation due to its non-invasive nature, affordability, and accuracy in detecting:
    - **Cholesterolosis:** Focal or diffuse wall thickening with possible hyperechoic striations.
    - **Cholesterol polyps:** Multiple small, non-shadowing lesions without an acoustic shadow.
    - **Adenomyomatosis:** Focal wall thickening with a "lamellated" appearance or Rokitansky-Aschoff sinuses with a characteristic "comet tail" artifact.
- **HIDA Scan (Hepatobiliary Iminodiacetic Acid Scan):** May show normal or mildly delayed gallbladder emptying, but findings are not diagnostic for specific types of cholecystoses.
- **CT Scan or MRI Scan:** May be used in some cases for further evaluation, but are not typically the first-line options due to cost and radiation exposure concerns.
- **Gallbladder biopsy:** Rarely performed due to the low risk of malignancy and the availability of accurate non-invasive diagnostic tools.

Differential Diagnosis:

- Cholecystitis (acute or chronic)
- Gallbladder cancer
- Choledocholithiasis (gallstones in the bile duct)

Treatment:

Cholecystoses are often managed conservatively, particularly if asymptomatic. Regular follow-up with ultrasound might be recommended to monitor for changes. Treatment is only considered for patients experiencing bothersome symptoms. In such cases, cholecystectomy is the definitive treatment. Laparoscopic cholecystectomy is the preferred surgical approach whenever possible.

4. Gallbladder Polyps:

Definition:

Gallbladder polyps are localized protrusions arising from the inner lining (mucosa) of the gallbladder. These lesions can be benign or malignant, and their identification often occurs incidentally during abdominal imaging studies performed for other reasons.

Incidence and Types:

- **Prevalence:** Gallbladder polyps are detected in approximately 3-7% of abdominal ultrasound examinations and 2-12% of cholecystectomy specimens.
- **Types:**
    - **Benign (majority):**

- § **Cholesterol polyps (most common):** Accumulations of cholesterol deposits within the mucosa, typically presenting as small, multiple polyps (<10 mm).
- § **Adenomyomas:** Localized areas of mucosal and muscular overgrowth that can manifest as single or multiple polyps.
- § **Inflammatory polyps:** Focal areas of inflammation that can appear as single or multiple polyps.
- § **Miscellaneous benign types:** Less common benign polyps may also exist. [Bailey]

o **Malignant (minority):**

- § **Adenocarcinomas (most common):** Cancers arising from the glandular epithelium of the gallbladder.
- § **Squamous cell carcinoma:** A rare type of gallbladder cancer.
- § **Cystadenomas:** Mucin-producing tumors that can be benign or malignant. [Bailey]

Clinical Presentation:

Gallbladder polyps are frequently asymptomatic and discovered incidentally. However, in some cases, patients might experience symptoms similar to biliary colic, including:

- Epigastric or right upper quadrant abdominal pain
- Nausea and vomiting
- Biliary colic or pancreatitis

Diagnosis:

- **Imaging Studies:** Ultrasound is the initial imaging modality of choice due to its non-invasive nature, affordability, and ability to differentiate between polyp types:
  - o **Cholesterol polyps:** Typically appear brightly echogenic (white) on ultrasound without an acoustic shadow (dark line behind the polyp). [Blumgart]
  - o **Adenomas:** May appear as smooth intraluminal masses, sometimes lobulated. Importantly, for benign adenomas, the adjacent gallbladder wall thickness should be normal (<3 mm). [Blumgart]
- **Additional Imaging Studies (if indicated):**
  - o **CT Scan or MRI Scan:** May be used in specific cases for a more detailed evaluation, but are not typically first-line options due to cost and radiation exposure concerns.
- **Gallbladder biopsy:** Rarely performed due to the low yield (difficulty obtaining accurate tissue) and potential complications associated with the procedure. It is typically reserved for large, suspicious polyps.

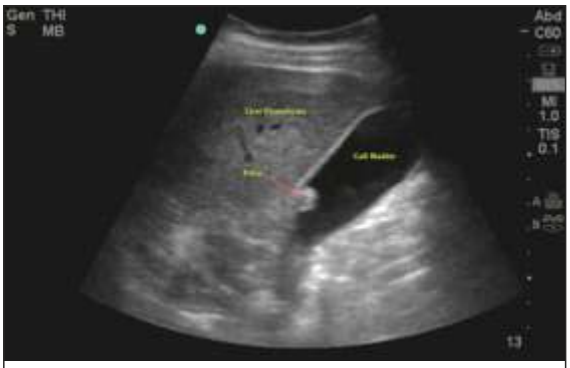
Management:

The management approach for gallbladder polyps depends on several factors, including:

- **Size:**
  - o **< 5 mm:** Observation with serial ultrasound is often recommended due to the low risk of malignancy in small polyps.
  - o **5-10 mm:** May be observed or surgically removed depending on other factors. Close monitoring with ultrasound is crucial for this size range.
  - o **> 10 mm:** Generally warrant cholecystectomy (surgical removal of the gallbladder) due to a

higher risk of malignancy in larger polyps.

- **Growth Rate:** Rapidly growing polyps raise suspicion for malignancy and might necessitate surgery regardless of size.
- **Symptoms:** Symptomatic polyps, regardless of size, are often treated with cholecystectomy to alleviate symptoms and potentially remove a precancerous lesion.
- **Patient Characteristics:** Age, overall health, and presence of other risk factors for gallbladder cancer (e.g., primary sclerosing cholangitis, history of gallstones) influence the decision for surgery.



Gall Bladder Polyp on USG
(Adapted from Emory School of Medicine, Emory)

Treatment:

- **Observation:** For small, stable polyps with low malignant risk, serial ultrasound monitoring is recommended to track for any changes in size or characteristics.
- **Cholecystectomy:** This is the definitive treatment for symptomatic polyps, large polyps (>10 mm), rapidly growing polyps, or polyps with concerning features on imaging studies. Laparoscopic cholecystectomy is the preferred method whenever possible.

Cholecystectomy:

Preoperative Preparation

Prior to cholecystectomy, a thorough patient evaluation is essential to ensure suitability for surgery. This includes:

- **History taking and physical examination:** To assess underlying medical conditions, previous surgeries, and any risk factors for complications.
- **Laboratory investigations:** Including a full blood count, coagulation profile, liver function tests, and possibly abdominal imaging (ultrasound or CT scan) to confirm the presence of gallstones and evaluate biliary anatomy.
- **Informed consent:** The patient must understand the procedure, alternative treatment options, potential risks and complications, and the expected postoperative course.
- **Prophylactic antibiotics:** A single dose of a second-generation cephalosporin is typically administered intravenously at induction of anaesthesia to reduce the risk of surgical site infection.
- **Thromboprophylaxis:** Subcutaneous heparin injections and anti-embolic stockings may be used to prevent blood clots in the legs, especially in high-risk patients.

Laparoscopic Cholecystectomy

Laparoscopic cholecystectomy is the preferred minimally invasive approach for most patients with symptomatic gallstones.

Steps:

1) Room set-up and patient positioning: Standard supine position with the surgeon standing at the patient's left and monitors at the head of the bed on both sides.
2) Following induction and maintenance of general anaesthesia, the abdomen is prepared in a standard fashion.

3) Abdominal access: Supraumblical 10mm incision given and pneumoperitoneum created using verres needle and CO_2 insufflation. This is followed by insertion of 10mm port which will function as the camera port

4) Additional operating ports are inserted in the subxiphoid area, in the right subcostal area and in the right anterior axillary line at the level of the umbilicus.

5) The patient is then placed in reverse trendelburg position slightly rotated to the left which exposes the fundus.

6) Fundus is then retracted towards the diaphragm and the neck of gallbladder is retracted towards the right iliac fossa exposing the calot's triangle.

7) The surgeon then carefully dissects Calot's triangle, a critical area containing the cystic duct, cystic artery, and common bile duct.

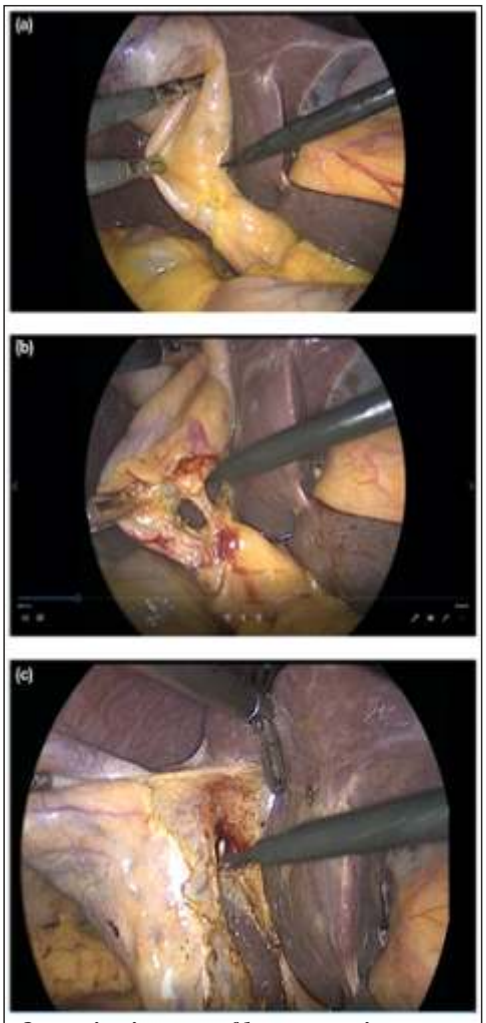
8) The cystic duct and artery are meticulously dissected, clipped, and divided to isolate the gallbladder.

9) Cholangiography (optional): In some cases, an intraoperative cholangiogram may be performed to confirm the anatomy of the bile ducts, especially if there is any ambiguity.

10) Gallbladder dissection: The gallbladder is dissected from the liver bed using

electrocautery or sharp dissection.

11) Gallbladder removal: The gallbladder is retrieved from the abdomen through one of the laparoscopic ports using a retrieval bag.



Operative images of laparoscopic cholecystectomy. (a) Retraction; (b) Achieving CVS [anterior doublet view]; (c) Exposure of the cystic plate. (Pic courtesy of Dr Sameer Rege, Mumbai, India).

Open Cholecystectomy:

Open cholecystectomy is an alternative surgical approach used when laparoscopy is contraindicated or when conversion from a laparoscopic attempt becomes necessary due to unforeseen complications.

Steps:

1) Patient positioning: Standard supine position with the surgeon standing at the patient's right.

2) Following induction and maintenance of general anaesthesia, the abdomen is prepared in a standard fashion.

3) Incision: An upper midline, right upper transverse, or short subcostal (Kocher) incision is made, providing direct access to the gallbladder.

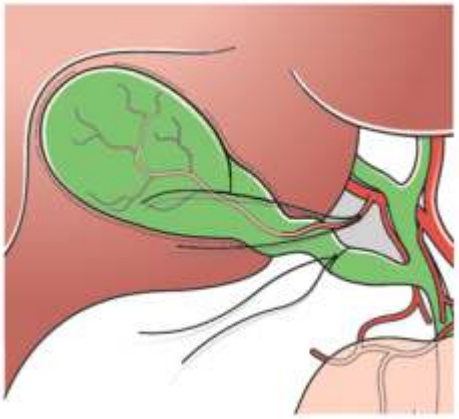
4) The surgeon meticulously exposes the gallbladder and surrounding structures by retracting the abdominal contents with packs.

5) Calot's triangle dissection: Similar to the laparoscopic approach, Calot's triangle is identified and carefully dissected to isolate the cystic duct and artery.

6) Cystic duct and artery ligation: The cystic duct and artery are tied off with sutures or clips to prevent

bleeding.

7) Gallbladder removal: The gallbladder is dissected from the liver bed and removed through the abdominal incision.



Ligatures are passed and tied around the cystic artery and cystic duct. The grey shaded area represents calot's triangle. (Adapted from Bailey & Love's Short Practice of Surgery 28th edition)

Tenets of safe cholecystectomy:

1. Use the Critical View of Safety (CVS) approach to identify the cystic duct and cystic artery during laparoscopic cholecystectomy.

- Three criteria are required to achieve the CVS:

  i. The hepatocystic triangle is cleared of fat and fibrous tissue. The hepatocystic triangle is formed by the cystic duct, the common hepatic duct, and the liver's inferior border. The common bile duct and common hepatic duct are not required to be exposed.

  ii. The lower one-third of the gallbladder is detached from the liver, revealing the cystic plate. The cystic plate, commonly known as the liver bed of the gallbladder, is located in the gallbladder fossa.

  iii. There should only be two structures entering the gallbladder.

2. Understand the potential for aberrant anatomy in all cases.

- Examples of abnormal anatomy include a short cystic duct, abnormal hepatic ducts, or a abnormal right hepatic artery that crosses the common bile duct anteriorly. These are some examples of the more prevalent varieties.

3. Make extensive use of cholangiography or other technologies to scan the biliary tree intraoperatively.

- Cholangiography may be especially essential in challenging situations or uncertain anatomy.
- Several studies have found that cholangiography lessens the incidence and extent of bile duct injury yet dispute persists on this subject.

4. Consider an Intra-operative Momentary Pause during laparoscopic cholecystectomy before clipping, severing or transecting any ductal structures.

- Use the intra-operative Momentary pause to validate CVS accomplished with the Doublet View.

5. Recognize when the dissection is reaching a zone of high danger and halt The dissection before entering the zone. If conditions around the gallbladder are too risky to perform a cholecystectomy, complete the operation using a safer method.

- Severe inflammation in the porta hepatis and neck of the gallbladder can make CVS harder to obtain. The fact that establishing a CVS appears difficult is a significant advantage of the procedure since it warns the surgeon of possibility of injury.
- The surgical judgment that a zone of significant risk is being approached can be made when there is failure to obtain adequate exposure of the anatomy of the hepatocystic triangle or when the dissection is not progressing due to bleeding, inflammation or fibrosis.
- Consider laparoscopic subtotal cholecystectomy or cholecystostomy tube insertion, and/or conversion to an open operation based on attending surgeon's judgement.

2. Seek assistance from another surgeon if the dissection or circumstances are tough.

    - Seeking input from a second surgeon might be beneficial in situations where the dissection is blocked, the anatomy is unclear, or the surgeon deems the situation "difficult".

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CHAPTER - 3

MANAGEMENT OF BILIARY INJURY

INTRODUCTION

Bile duct injury is a serious complication that can occur during cholecystectomy, which is the surgical removal of the gallbladder. The incidence of BDI ranges from 0.4 to 1.5% of cholecystectomies. The two most frequent scenarios are bile leak and bile duct obstruction. BDIs can be recognized during the surgery or in the immediate postoperative period, although sometimes they are discovered later, leading to delayed or inappropriate treatments. The most common reason for injuring the bile duct is due to the misidentification of normal biliary anatomy [1]. Once the bile duct is injured, early recognition is crucial to facilitate appropriate treatment.

ETIOLOGY

With an incidence of 0.3 to 0.7%—historically three times higher than in open cholecystectomy—iatrogenic biliary damage is most usually caused by misidentifying the common bile duct for the cystic duct during laparoscopic cholecystectomy [2][3] One of the elements contributing to the injury's cause is the varied biliary anatomy. With a 0.1% trauma hospital admission rate, injury to the biliary tree is a rare consequence of penetrating or severe abdominal trauma. The therapy options include cholecystectomy, drainage, reconstruction to reestablish the bile flow into the intestine, or hepatic resection, depending on the site and timing of the diagnosis.[4]

EPIDEMIOLOGY

The incidence of bile duct injury increased with increasing adoption of the laparoscopic technique for cholecystectomy and treatment of other biliary pathologies.

Anatomical variations, patient status, gallbladder pathology, and surgeon-related factors are risk factors for bile duct damage. It is possible to misidentify the cystic duct if it is short or if it runs parallel to the common bile duct. Additional conditions that can lead to damage or leakage include variations in the cystic duct and common hepatic duct junction, a cystic duct that attaches to the right hepatic duct, an auxiliary cystic duct, or the existence of Luschka ducts. Although 80% of injuries happen in the absence of any risk factors, patients with significant obesity, previous hepatobiliary surgery, or underlying liver disease might reduce vision and increase the chance of injury. Acute cholecystitis increased the rate of bile duct injuries due to the associated inflammation, adhesions, gallbladder wall thickening, and increased bleeding [5]. Intra-operatively, these injuries are due to misidentification of the CBD (classical Davidoff injury) [6] while delayed leaks are usually due to thermal or vascular injury during dissection. [7] Multiple strategies like the critical view of safety have been suggested to minimize bile duct injuries and associated morbidity from bile leaks and strictures.

CLASSIFICATION OF BILE DUCT INJURY

STRASBERG CLASSIFICATION-

The Strasberg classification, which is a variant of the bismuth classification, makes it possible to distinguish between minor injuries sustained during laparoscopic cholecystectomy (type A to D) and major injuries (bile leaking from the cystic duct or abnormal right sectoral branch). The Bismuth classification is analogous to Type E of the Strasberg classification [8] The Strasberg categorization is quite straightforward and easily applicable to bile duct injuries, as illustrated in Fig. 1. The Strasberg classification's primary drawback is that it provides no description of extra vascular involvement. Because of this, the Strasberg classification was unable to show a meaningful correlation between the removal of liver tissues and the identification of

particular injury patterns.

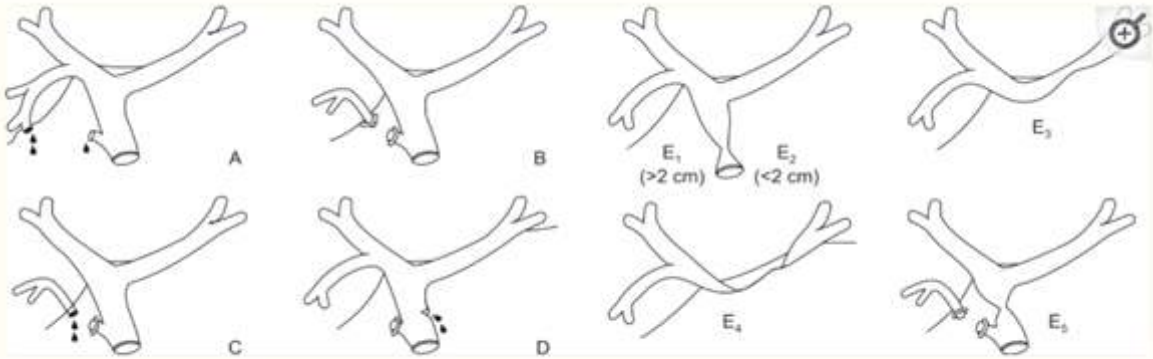


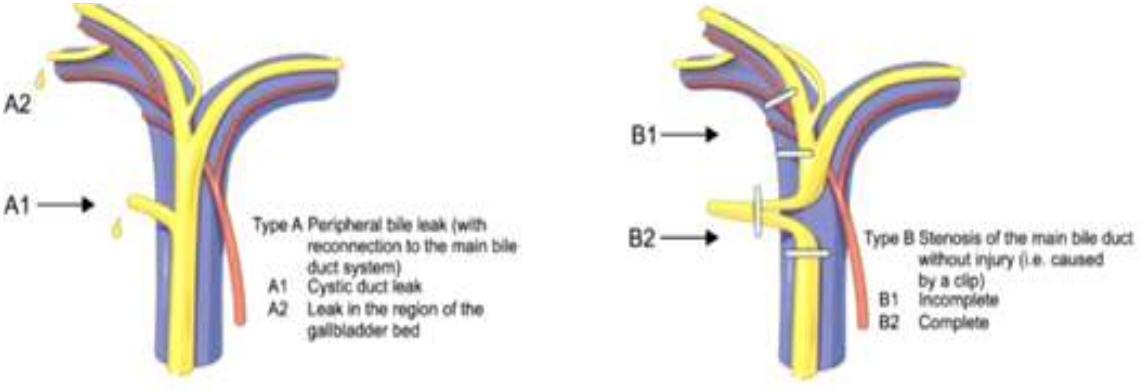
Fig. 1

Strasberg classification.[3,22] (A) Bile leak from cystic duct stump or minor biliary radical in gallbladder fossa. (B) Occluded right posterior sectoral duct. (C) Bile leak from divided right posterior sectoral duct. (D) Bile leak from main bile duct without major tissue loss. (E_1) Transected main bile duct with a stricture more than 2 cm from the hilus. (E_2) Transected main bile duct with a stricture less than 2 cm from the hilus. (E_3) Stricture of the hilus with right and left ducts in communication. (E_4) Stricture of the hilus with separation of right and left ducts. (E_5) Stricture of the main bile duct and the right posterior sectoral duct.

Fig 1

HANNOVER CLASSIFICATION

Following a comparison of the bile duct injury classification for 72 consecutive iatrogenic bile injuries following laparoscopic cholecystectomy, Bektas et al. proposed a novel classification scheme called the Hannover classification. Bile duct injuries in the Hannover were categorized into five kinds, ranging from A to E [9]. Peripheral biliary leakage is type A. Type B is a CHD or CBD stricture that is injury-free. Lateral CHD, or CBD injury, is type C. Type D refers to complete transection of CHD. In the postoperative state, type E bile duct stricture of the main bile duct is defined as the absence of bile leakage. Type C and Type D contain vascular damage (Fig.2). In a limited number of cases, the Hannover classification identified 21 distinct damage types in all. A significant shortcoming of other classification systems is that the Hannover classification offers discriminators for the localization of tangentially or totally transected bile ducts above or below the hepatic duct bifurcation.



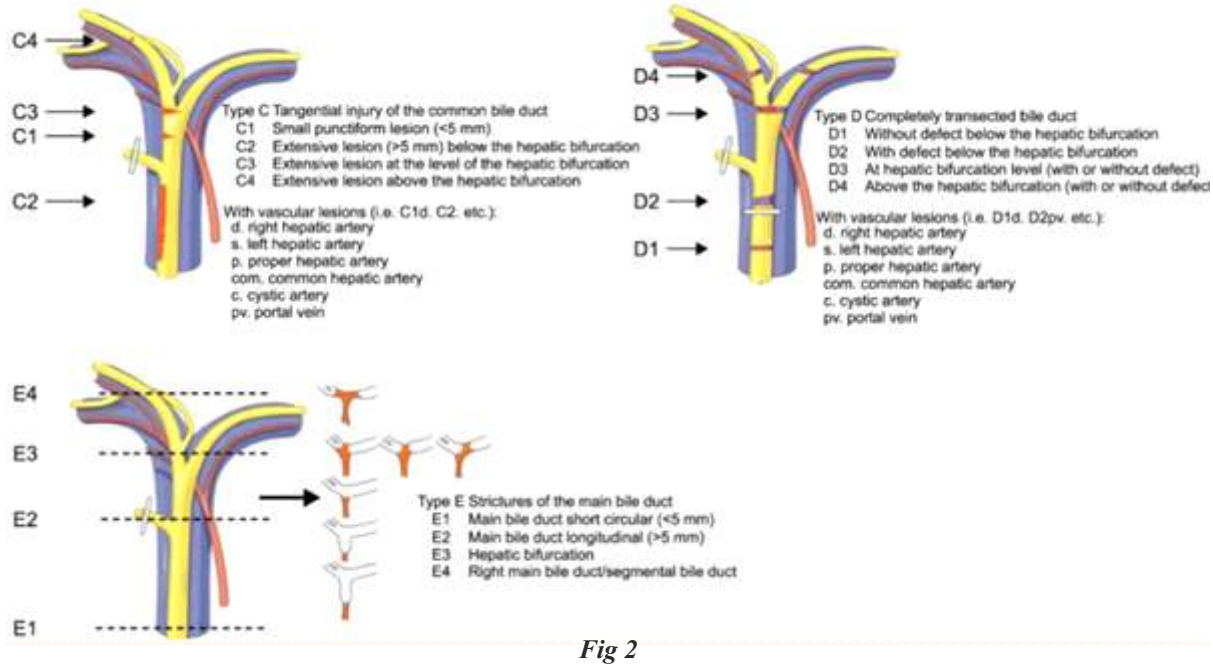


Fig 2

(Adapted from Semantic Scholar, Recent classification of common bile duct injury)

PREVENTION OF BILE DUCT INJURY

In the scientific literature, a number of strategies for preventing iatrogenic biliary tract lesions have been put forth and explained. Table 1 provides a summary of them. Introduced by Strasberg in 1995, the "critical view of safety (CVS)" technique is regarded as the gold standard for safely performing a cholecystectomy with identification of biliary structures during dissection. To meet the CVS, three requirements must be met: 1) The gallbladder's bottom third needs to be detached from the liver bed in order to reveal the cystic plate ;2) the hepatocystic triangle needs to be free of fibrotic and adipose tissues, CBD and CHD must not be exposed ;3) there should only be two structures visible entering the gallbladder. CVS was designed to prevent biliary damage rather than as a means of performing LC.

A method of dissecting the gallbladder from the fundus up to the infundibulum, away from Calot's triangle, is known as "antegrade dissection or fundus first/dome-down technique."[11]

- Critical view of safety (CVS) method
- Infundibular technique
- Antegrade dissection
- Subtotal cholecystectomy
- Anatomic landmarks:
  - Rouviere's sulcus
  - Calot's node
  - B-SAFE method
- Intra-operative cholangiography (IOC)
- Laparoscopic ultrasound (LUS)
- Near-infrared fluorescent cholangiography (NIRF-C)
- Conversion to open surgery

Table 1

CLINICAL PRESENTATION

In the event that an iatrogenic lesion is not identified during surgery, the clinical presentation primarily depends on its size and nature. Non-specific symptoms including nausea, vomiting, bloating, diffuse stomach pain, overall discomfort, and anorexia may indicate the existence of a potential biliary leak. Even when there is a lot of bile in the belly, the patient hardly ever exhibits the clinical signs of biliary peritonitis. Due to the ambiguity of these symptoms, biliary leak development is quite modest. Patients with strictures at the CBD level will exhibit the traditional Charcot's triad of fever, jaundice, and right hypochondrial pain, in situations when the CBD is misdiagnosed as the cystic duct. It is generally advised that any deviation from the typical postoperative course following LC should indicate a potential biliary tract injury. These factors make a thorough clinical assessment of patients crucial. The postoperative result depends critically on the timing of the diagnosis.

EVALUATION

It is crucial to identify BDI as soon as possible. The best results usually result from quick surgical repair following intra-operative detection; however, only 25% to 30% of BDIs are identified during surgery, and not all surgeons do an intra-operative cholangiography [12].

The diagnosis is frequently made within the first six weeks following the procedure or, in certain situations, later (more than six weeks). A proper diagnosis is aided by a thorough clinical evaluation. A diagnosis of any iatrogenic lesion of the biliary tree is supported by the presence of persistent or abnormal abdominal pain in the right hypochondrium, the appearance of fever and jaundice with elevated liver function tests, the leakage of bile from drainage (if placed during surgery), and a picture of biliary peritonitis. Planning therapeutic options and accurately identifying the extent and severity of the injury require radiologic examinations. The initial diagnostic method for locating any liver collections, CBD dilatation, and any related vascular abnormalities is abdominal ultrasonography. The primary test that determines the existence of focal intra- or peri-hepatic fluid collections, ascites, biliary obstruction with upstream dilatation, or long-term consequences of a chronic bile stricture, like lobar hepatic atrophy or indications of secondary biliary cirrhosis, is the abdominal computed tomography (CT) scan. Any accompanying vascular lesions, such as those to the right hepatic artery, can also be found with a CT scan. Endoscopic retrograde cholangiography is used to evaluate the level of the leak and provide therapeutic intervention through stenting.

Magnetic resonance cholangio-pancreatography (MRCP) represents the “gold standard” for the complete morphological evaluation of the biliary tree as it offers detailed information about the integrity of the biliary tract [13]. Using a contrast agent during MRCP not only shows the kind of BDI and the anatomical location of the leakage, but it also directly visualizes the extravasation of contrast material into fluid collections, allowing for the detection of active bile leakage.

MANAGEMENT

Treatment options might vary from straightforward drainage techniques to biliary system regeneration, contingent on the extent and intricacy of the damage sustained.[14] If the patient has any injuries, whether they are discovered right away or later, they can always move to a specialized facility where the doctors and resources are more qualified to handle bile duct injuries.

The Strasberg classification can be used to direct medical care. The biliary system is continuous with a leak from the cystic duct or minor hepatic duct across the liver bed in cases of Strasberg type A injuries. If a drain is already installed, it is possible to monitor the output and assess if the leak closes on its own. By lowering the pressure in the proximal biliary system, endoscopic stenting over the lesion can aid in occulting the leak and facilitating drainage through the biliary system. In situations where choledocholithiasis is retained, sphincterotomy may be required. Washout may require examination if the patient has progressive intraabdominal sepsis or peritonitis. In cases of Type A injuries when there is leaking from the cystic duct stump, interventional radiology's coil embolization of the cystic duct has been described in few cases.[15]

Conservative treatment and close monitoring are appropriate for Strasberg type B injuries exhibiting only minor pain and slight increase in liver function tests. Unnoticed occlusion can result in intrahepatic stones, segmental cholestasis, and liver atrophy in the right lobe. The patient will need a hepaticojejunostomy or percutaneous transhepatic cholangiogram with biliary drainage tube implantation if there is evidence of cholangitis as a result of the obstruction. If atrophy is severe, segmental excision of the affected segments can be required. Because the occlusion causes the proximal biliary segment to become discontinuous with the distal biliary tree, endoscopic therapy of occlusive injuries usually fails.

Similar to type B injuries, Strasberg type C injuries involve a leaking accessory duct rather than an obstructed one. Because the segment proximal to the damage is not in continuity with the biliary system, endoscopic intervention is not an option for either type B or type C injury. Biliary peritonitis can be avoided and spontaneous closure of the subhepatic biliary leak can occur via percutaneous draining of the leak. Rarely, the patient would need a hepatectomy or hepaticojejunostomy.

A medial partial damage to the common bile duct is associated with Strasberg type D injuries. Endoscopic sphincterotomy, stent implantation, and interrupted 5-0 absorbable monofilament suture with a drain left in place can be used to close the defect if the injury is minimal and shows no signs of devascularization. The surgeon should still patch the damage with a drain left in place in case of devascularization since this will prepare the body for an anticipated bile leak. An alternative is to use an endoscopic stent placed with an interventional radiologic drain, particularly for injuries observed during the postoperative phase.

If there is no tension on the anastomosis, Strasberg type E injuries that are identified at the time of injury can primarily be treated in an end-to-end manner. A Y tube that empties into the duodenum or a T-tube that offers external drainage should be repaired above them. The preferred repair method is a Roux-en-Y hepaticojejunostomy if the anastomosis cannot be completed without causing stress. When hepaticojejunostomy is not an option due to friable tissue or extremely dense adhesions, a pedicled omental patch might be employed as a stopgap measure to manage bile leakage until permanent reconstruction [16].

Vascular injuries associated with bile duct injuries can present with hemobilia, abscess, or ischemia, for which the management is angioembolization, percutaneous drainage, or liver resection, respectively [17]

CONCLUSION

BDIs represent a serious complication which can be brought on by cholecystectomy. The primary cause of mistakes resulting in laparoscopic bile duct lesions is a misinterpretation of the biliary anatomy. We appreciate any effort made to lower the risk profile associated with routine cholecystectomy. Early detection, management of any intra-abdominal fluid collection and infection, nutritional balance, a multidisciplinary approach, and surgical repair by a skilled biliary reconstruction surgeon are the essential components of a successful course of treatment.

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CHAPTER - 4

BILIARY STRICTURES

INTRODUCTION

The terms "biliary strictures" or "bile duct strictures" describe areas where the intrahepatic or extrahepatic biliary ductal system narrows. When they constrict, they hinder the bile's normal antegrade flow, leading to proximal dilatation and the clinical and pathological consequences of biliary obstruction. If cancer is suspected in patients with persistent biliary strictures, there is a special problem. Hepatobiliary specialists, endoscopists, surgeons, and interventional radiologists may all have a role in the diagnosis and treatment of patients with biliary strictures.

ETIOLOGY

Strictures in the bile ducts may be acquired or congenital. Congenital strictures are less prevalent than the latter [4]. Additionally, acquired strictures might be categorized as benign or malignant.[5] A variety of benign acquired disorders can cause strictures in the bile duct and account for thirty percent of strictures in the biliary system.[6] Of the various types of benign biliary strictures, the majority are iatrogenic strictures. Bile duct injury resulted by misidentification of the biliary duct for the cystic duct during laparoscopic cholecystectomy can be partial or total. Benign biliary strictures develop as a result of the long-term effects of these lesions.[7] An intraoperative cholangiogram may reduce the risk of damage, particularly in situations with gangrenous cholecystitis or gallbladder empyema.[8][9] When it comes to repairing the bile duct, it is crucial to comprehend the structure of the blood flow that feeds it.[10] Anastomotic biliary strictures are also a recognized side effect following orthotopic liver transplantation.[11] It is also known that anastomotic strictures can develop during a Whipple procedure (4% of cases), which is carried out to treat pancreatic masses or injuries. This is particularly valid for thin-walled ducts with tiny diameter.[1] Although they are uncommon, diseases such as tuberculosis can also cause strictures. On the other hand, cancer is the most frequent cause of biliary strictures.[13] Most malignant biliary strictures are associated with pancreatic head malignancy and cholangiocarcinoma. Additional conditions include lymphoma, hepatocellular carcinoma, gallbladder carcinoma, periampullary malignancy, and metastases to nearby lymph nodes and solid organs.

EPIDEMIOLOGY

Globally, the incidence of biliary strictures is thought to be on the rise primarily because of the iatrogenic bile duct injuries resulting from the widespread practice of laparoscopic cholecystectomy. A number of tactics, such as the critical view of safety, have been proposed to reduce the risk of bile duct injuries and the morbidity that results from bile leaks and strictures.[16] After laparoscopic cholecystectomy, biliary injuries are thought to occur at an incidence of roughly 0.7%. The majority of these wounds are either bile leaks or small wounds. In the pediatric age range, biliary strictures are uncommon.[17] There is no documented variation in the frequency or occurrence of biliary strictures between males and females, however some risk factors, such as chronic pancreatitis caused by alcohol, are more common in men.

PATHOPHYSIOLOGY

The narrowing of a section of the bile duct linked to proximal ductal dilatation is the hallmark of biliary strictures. Serum bilirubin levels rise as a result of bile flow obstruction, exhibiting both clinical and laboratory manifestations of obstructive jaundice. One of the main risk factors for ascending cholangitis is bile stasis.[18]

Benign strictures have smooth, symmetric borders and tapered margins, as shown by magnetic resonance

cholangiopancreatography (MRCP) and endoscopic retrograde cholangiopancreatography (ERCP). Malignant strictures, on the other hand, feature uneven borders and a shouldering of the edges. [11] Unlike benign strictures, which have shorter segments, malignant strictures have longer segments. On cross-sectional imaging enhanced by contrast, malignant strictures Seem to enlarge. [18]

The Strasberg-Bismuth classification can be used to categorize the strictures. Management is guided by classification. [18] [19] [20]

Type E injuries lead to strictures of the hepatic ducts, which are further defined by the proximal extent.

- E1: Common hepatic duct division greater than 2 cm from the bifurcation.
- E2: Common hepatic duct division less than 2 cm from the bifurcation.
- E3: Common bile duct division at the bifurcation.
- E4: Hilar stricture involves confluence and loss of communication between the right and left hepatic duct.
- E5: Involves aberrant right hepatic duct with concomitant stricture of the common hepatic duct

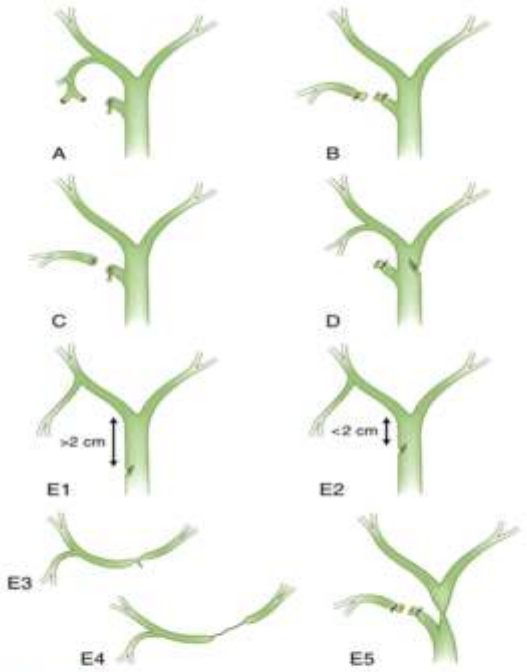

FIG. 55.48 Strasberg classification of postoperative bile duct strictures. (A) Injury to small ducts in continuity with the biliary system with a leak in the duct of Luschka or the cystic duct. (B) Injury to a sectoral duct, causing obstruction of portion of the biliary system. (C) Injury to a sectoral duct with bile leak; leak from a duct not continuous with the biliary system. (D) Lateral injury to the extrahepatic biliary ducts. (E1) Bismuth type 1: injury more than 2 cm from the confluence. (E2) Bismuth type 2: injury less than 2 cm from the confluence. (E3) Bismuth type 3: injury at the confluence; confluence intact. (E4) Bismuth type 4: destruction of the biliary confluence. (E5) Complete occlusion of all bile ducts, including sectoral ducts.

HISTOPATHOLOGY

Histopathological results in biliary strictures typically vary depending on the mechanism or etiological agent. For instance, histology for primary sclerosing cholangitis shows fibro-obliteration and inflammation of the bile ducts either intra- or extrahepatic. [5] Histological evaluation along with cytology or histopathology is mostly necessary in the evaluation of biliary strictures in order to rule out malignancy. [18] A positive histological diagnosis is not required prior to surgery, though, if imaging has confirmed or raised a high suspicion of cancer.

HISTORY AND CLINICAL FEATURE

The stricture's location and etiology determine how it manifests clinically. [11] When a physical examination reveals non-contributory symptoms, biliary strictures can be evidently asymptomatic. Even so, certain patients with biliary strictures will exhibit symptoms of obstructive jaundice, such as dark urine, steatorrhea, pruritus, pale faeces, and yellowing of the skin and mucosal surfaces.[19] Constitutional symptoms including fever, nausea, vomiting, weight loss, and malaise may accompany these. As a consequence of the bile duct stricture, patients may present with an acute abdomen due to cholangitis or a hepatic abscess.[21] . Leukocytosis with fever in the past point to an infectious etiology or stricture sequelae. Be cautious if there is a history of weight loss, back or abdominal pain, or declining performance status as it may indicates possible malignant etiology. In order to narrow down the differential diagnosis or rule it out, it is important to know about any prior history of hepatobiliary surgery, autoimmune disease, pancreatitis, cholelithiasis, or chemotherapy.[11]

More attempts should be made to elicit particular clinical symptoms, such as the Courvoisier and Murphy signs, during the physical examination, since this could also aid in limiting the cause or diagnosis of jaundice. Hard masses in the abdomen may indicate a cancerous disease that has progressed.

EVALUATION

A coagulation profile, a complete blood count, and a liver function test are the primary sources of contributing laboratory results. Increased levels of conjugated bilirubin and liver enzymes (gamma-glutamyl transferase and alkaline phosphatase) may be detected by liver function testing. It is possible for transaminases to be raised in general. It is possible to evaluate some autoimmune etiologies of biliary strictures with immunological investigations. The linkage of laboratory and imaging results with epidemiological and clinical data forms the basis for the appropriate diagnosis and subsequent care.[11] Deviant coagulation profiles and liver function tests may be useful in establishing the minimally invasive study and imaging strategy.[15][22] Similarly, specific immunological markers aid in precise diagnosis and treatment when they are present. A case in point is IgG4-associated sclerosing cholangitis, which manifests as distal or hilar common bile duct (CBD) strictures.

In order to aid in diagnosis and follow-up, individuals with suspected hepatobiliary malignancy may benefit from obtaining assays of CA19-9 and carcinoembryonic antigen (CEA). Notably, these are not unique to biliary cancer. Fine needle aspiration cytology (FNAC) guided by endoscopic ultrasound (EUS) is very sensitive and specific, however it cannot be used to rule out cancer.

In order to provide more information on tumor vascular encroachment and biliary tree obstruction, a multi-detector CT (MDCT) scan and the CT-pancreatic protocol can be used to enhance the utility of computed tomography (CT) scanning, which has a higher sensitivity than trans-abdominal ultrasound for biliary malignancy. Additionally, it can identify biliary obstruction side effects such cholangitis and abscesses.

Locating and measuring the amount of biliary strictures, MRCP can give high-quality cholangiograms. The biliary tree is reconstructed in three dimensions and in cross-section. [5] In cases where ERCP is contraindicated, it aids in directing endoscopic therapy. Additionally, MRCP does not use ionizing radiation, which makes it a better choice than an MDCT scan. On the other hand, diffusion-weighted imaging, or DWI, can increase its sensitivity and specificity, making it comparable to ERCP.

The etiology of biliary strictures can be ascertained by ERCP evaluation, tissue samples for cytology and histology can be provided, and therapeutic procedures such as stenting of obstructed segments from strictures can be facilitated. Nonetheless, it is linked to pancreatitis following ERCP and is being surpassed by more recent methods like as confocal laser endomicroscopy (CLE). Direct peroral cholangioscopy, intraductal ultrasound, and fluorescent in-situ hybridization (FISH) are further cutting-edge technologies that can be helpful in determining the cause of biliary strictures.

The degree of bile leaks and bile damage following cholecystectomy can be determined with the use of a hepatobiliary iminodiacetic acid (HIDA) scan.

TREATMENT

Treatment of the underlying cause and occasionally complications from biliary obstructions constitute the majority of medical care for biliary strictures. Thus, when appropriate, analgesics, empirical antibiotics, and intravenous fluids with or without vasopressors and inotropes for hemodynamic support are frequently started.[24] Treatments designed to lessen the symptoms of rising bilirubinemia are also frequently used. These activities are frequently preparatory to final therapy and supportive in nature.[25] Additional medical interventions could be used to stop more problems from occurring, such as deep vein thrombosis, sepsis, and excessive bleeding brought on by a coagulopathy in the early stages of the healing process.

The goal of the interventions for biliary strictures includes reestablishing patency and avoiding additional procedures.[11][19] There are varying options for operative or interventional management. These options can be accomplished via endoscopy, open surgery, or percutaneously. The etiology and the location of the stricture, as well as the patient's hemodynamic stability and nutritional status, could determine the timing and type of intervention required.[25] The Bismuth classification can further guide the most appropriate approach since it offers a guide to the determination of the level at which healthy biliary tissue is available for repair and anastomosis.[11] Likewise, the Strasberg classification system, which incorporates the presence of a bile leak

and lateral injuries into consideration, can also be useful in some cases in making the choice of the best intervention.[19] Additionally, preoperative determination of malignancy is pivotal in the planning of treatment and avoiding undue exploratory surgery.

Surgical reconstruction has the highest patency rates. To achieve a successful and durable repair, the anastomosis must be per- formed between a minimally inflamed bile duct to intestines in a tension-free, mucosa-to-mucosa fashion. When the anastomosis is within 2 cm of the hepatic duct bifurcation or involves intrahepatic ducts, some evidence suggests that long-term stenting may improve patency. If the bifurcation is involved, stenting of both right and left ducts should be performed. When the reconstruction involves the CBD or common hepatic duct more than 2 cm from the bifurcation, stenting is not necessary; therefore, a preoperatively placed transhepatic drain or intraoperatively placed T tube will provide adequate decompression in the immediate postoperative period.

At the time of operation, the adhesions of the duodenum and colon to the liver should be separated. he porta hepatis can be encircled with a Penrose drain. Although the bile duct should lie on the lateral border of the porta hepatis, preoperatively placed per-cutaneous biliary drainage catheters can assist in the dissection, as the marked fibrosis and inflammatory process may make its identification difficult. If necessary, a small-caliber needle attached to a syringe can be used to aspirate and to identify the bile duct while avoiding inadvertent injury to a vascular structure (Fig. 55.50).

Once identified, above the stricture, only a limited segment of bile duct (<5 mm) is dissected free. Any further dissection of normal duct risks vascular compromise of the segment to be used in the anastomosis. Preservation of as much normal biliary tree as possible remains a goal of the reconstruction. Next, the bile duct can be opened and the percutaneously placed catheters advanced through the incision. At this point, a wire can be used to exchange the catheters for long-term Silastic stents, if appropriate, or the catheters can be left in place for trans anastomotic decompression. The mucosa-to-mucosa anastomosis can be created in an end-to-side fashion to the Roux-en-Y jejunal limb. In the setting of substantial inflammation at the bifurcation, another reconstruction option involves anastomosis of the Roux limb to the left hepatic duct. As noted, the left hepatic duct retains a substantial extra parenchymal length, allowing an anastomosis in this portion of normal duct. Before this section is used for drainage of the entire liver, cholangiography must confirm that the biliary bifurcation is widely patent, thus ensuring drainage of the right lobe across the bifurcation to the left duct system.

OUTCOMES

Successful outcomes can be achieved in patients undergoing biliary-enteric reconstruction after bile duct injury, with many series showing more than 90% of patients free of jaundice and cholangitis. High success rates are generally achieved when injuries are identified early, and patients are referred immediately to experienced centers. In several studies, referral to centers performing complex biliary surgery routinely was associated with better long-term success. Surgical reconstruction provides a durable long-term management strategy. Management of these injuries requires a multidisciplinary management and may need percutaneous techniques as well as surgical reconstruction. Sepsis at the time of reconstruction and biliary cirrhosis are predictors of stricture. In some studies, results were generally better if trans anastomotic stents were used during reconstruction.36 Chronic liver disease and hepatic fibrosis are associated with higher operative mortality and lower success rates. Although a devastating com- plication, management is highly successful and restores health- related quality of life scores to preinjury levels. Complications include recurrent cholangitis, ascending cholangitis, gram-negative septicemia, stone formation, hepatic abscesses, secondary biliary cirrhosis, end-stage liver disease and cholangiocarcinoma.

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CHAPTER - 5

NON MALIGNANT (OTHER THAN CHOLELITHIASIS AND CHOLECYSTITIS) AND MALIGNANT DISEASES OF THE GALL BLADDER

Cholesterolosis ('strawberry gallbladder')

The buildup of fatty materials can also create yellow patches on a reddened lining, giving the gallbladder a strawberry-like appearance, hence the name "strawberry gallbladder."

Cholesterolosis may sometimes be associated with gallstones.

Cholecystitis glandularis proliferans (adenomyomatosis)

Adenomyomatosis is a benign condition that affects the gallbladder. It's characterized by:

- Overgrowth of the inner lining (mucosa)
- Thickening of the muscular wall
- Formation of cyst-like structures within the gallbladder wall
- Polypoid projections (protrusions resembling polyps) from the inner lining
- Intramural diverticulae (pouches within the wall) in diffuse adenomyomatosis

While not typically considered a precancerous condition, adenomyomatosis is often associated with gallstones (cholelithiasis). These gallstones can lead to complications such as:

- Intramural abscesses (collections of pus) within the gallbladder wall
- Extramural abscesses (collections of pus) outside the gallbladder wall
- Fistula formation (abnormal connections between organs)

Clinical features of cholecystoses:

1. Fever
2. Pain in right hypochondrium
3. Asymptomatic
4. Dyspepsia

Diagnosis of cholecystoses:

- USG: in adenomyomatosis, The most common finding is focal or diffuse thickening of the gallbladder wall. This thickening can be irregular and may sometimes appear as a layered pattern.

  Other findings:

  - Presence of cystic spaces within the thickened wall.
  - "Comet tail artifacts" - echogenic streaks extending posteriorly from the gallbladder wall.
  - "Twinkle artifact" - tiny, bright echoes within the gallbladder wall.
- in cholecysterolosis, Focal cholesterolosis: May appear as small, hyperechoic (brighter) nodulesless than 1mm in diameter, giving the lining a coarse and granular appearance.Polypoid cholesterolosis:

Larger, polypoid protrusions from the gallbladder wall.

Management: surgery is the definitive management as medical management is only symptomatic. Laparoscopic cholecystectomy is the standard of care.

Gallbladder polyps

Clinical presentation:

Gallstone polyps are often found unexpectedly during abdominal imaging tests. Most are harmless (cholesterol polyps, adenomyomas, inflammatory polyps, adenomas, or others). However, there's a small chance they might be cancerous (adenocarcinoma being the most common type)

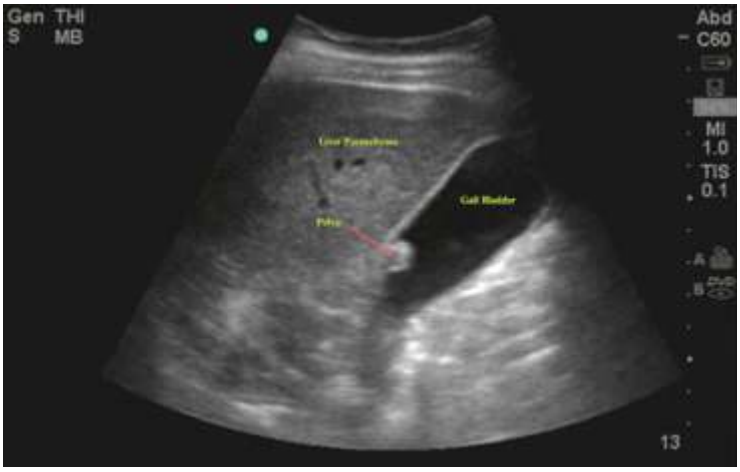
Diagnosis:

USG is the investigation of choice for GB polyps.

- Presence of a mass: A well-defined mass projecting into the lumen (inner space) of the gallbladder is the most basic indicator.
- Size: The size of the polyp is measured in millimeters (mm). Generally, polyps smaller than 10mm are less concerning.
- Number: USG can reveal if there are single or multiple polyps present.
- Location: The location of the polyp within the gallbladder (e.g., fundus, body, neck) may be noted.
- Pedunculated vs. Sessile: Ultrasound can sometimes differentiate between polyps with a stalk (pedunculated) and those with a broad attachment to the gallbladder wall (sessile). Pedunculated polyps are generally less worrisome.
- Echogenicity: This refers to the brightness of the polyp compared to surrounding tissue. Most polyps are similar or slightly hyperechoic (brighter) than the liver tissue.
- Posterior acoustic shadowing: Unlike gallstones, polyps typically don't cause this specific shadowing behind the mass on ultrasound.

Management:

Surgery to remove the gallbladder (cholecystectomy) is considered for people with symptoms or certain risk factors. These risk factors include having gallstones, a condition called primary sclerosing cholangitis (PSC), biliary colic (abdominal pain from gallstones), pancreatitis (inflammation of the pancreas), polyps larger than 10mm, polyps in people over 50, or flat polyps (sessile) with a thickened gallbladder wall exceeding 4mm.



Gall Bladder Polyp on USG; Emory School of Medicine, Emory

Xanthogranulomatous cholecystitis

XGC is an uncommon inflammatory disease affecting the gallbladder. It's more frequent in females and certain areas like India and Japan. The cause isn't fully understood, but it likely involves bile leaking into the gallbladder wall. This leak can be caused by ruptures in tiny channels or ulcers in the lining. The leaked bile triggers an inflammatory response with buildup of specific cells, scar tissue, and inflammation.

Clinical presentation:

Usually asymptomatic, however may present as a gall bladder mass on examination. Symptoms of pain in the right hypochondrium and fever may be present. Dyspepsia is seen in 37% of patients.

Diagnosis:

Diagnosing XGC can be challenging because:

- Symptoms are often general, mimicking other gallbladder problems.
- Ultrasound (USG) may show a thickened gallbladder wall, sometimes with darker areas inside.
- CT scans can reveal specific features, but these can be confused with gallbladder cancer.

Ultrasound (USG):

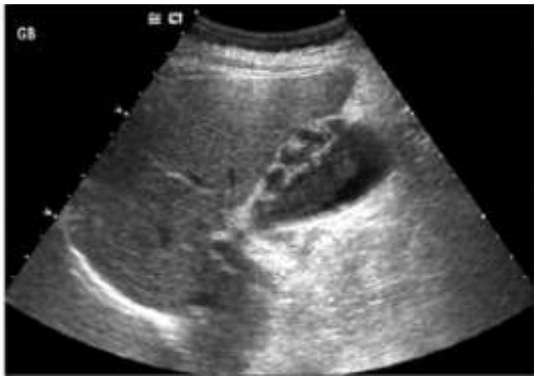
1. Thickened gallbladder wall: This is a common finding in many gallbladder conditions, but in XGC, the thickening may be more irregular or patchy compared to a smooth thickening seen in other diseases.
2. Intramural hypoechoic nodules or bands: These are areas that appear darker than the surrounding liver tissue on the ultrasound. They are a more characteristic finding of XGC, but not always present.
3. Gallstones: While not always there, gallstones can sometimes be seen within the gallbladder lumen on ultrasound.

CT scan:

- 5-20 millimeter (mm) hypoattenuating nodules: These appear as darker areas within the thickened gallbladder wall on a CT scan.
- Poor/heterogeneous contrast enhancement: After contrast dye is injected, the inflamed tissue in XGC may not take up the dye uniformly, resulting in a patchy or uneven enhancement.
- Early enhancement of the adjacent liver parenchyma: Similar to acute cholecystitis, there might be early enhancement of the liver tissue next to the gallbladder.

Surgery is often the best option for both diagnosis and treatment.

Due to the difficulty in distinguishing XGC from cancer before surgery, open surgery to remove the gallbladder (cholecystectomy) is frequently recommended. During surgery, a rapid tissue analysis (frozen section) can help tell the difference between the two. This ensures the doctor removes the entire gallbladder if necessary.



Xanthogranulomatous cholecystitis; Semantic Scholar, xanthogranulomatous cholecystitis-appearance on USG

Gall Bladder Cancer

Gallbladder cancer (GC) is a rare malignancy but represents almost 50% of all biliary tract cancer. Biliary cancers are highly fatal malignancies with a 5-year survival rate of 17.6% (2007-2013). The prognosis of gallbladder cancer is poor due to the aggressive tumor biology, late presentation, complicated anatomic position, and advanced stage at diagnosis. Locally advanced and metastatic disease is treated with palliative chemotherapy. Conversely, early stage is potentially curative with surgical resection followed by adjuvant therapy.

Etiology:

Chronic inflammation is the most critical risk of gallbladder cancer.

1. history of gallstones (cholelithiasis), and the risk increases with gallstone size, chronicity, and burden of symptoms
2. Porcelain gallbladder, a calcification of the gallbladder
3. gallbladder polyps
4. biliary cysts
5. abnormal pancreaticobiliary anatomy
6. carcinogens causing gallbladder cancer include (e.g., methyldopa, isoniazid), work exposure (e.g., methylcellulose, radon), and lifestyle (e.g., cigarette smoking, obesity, high carbohydrate intake).
7. Chronic primary sclerosing cholangitis
8. inflammatory bowel disease can also lead to gallbladder cancer.

Clinical features:

1. vague symptoms such as abdominal pain, nausea or vomiting, indigestion, weakness, anorexia, loss of appetite, weight loss
2. obstructive jaundice and associated symptoms: clay colored stool, yellowish discoloration of sclera, high colored urine
3. Physical examination may demonstrate jaundice, right upper quadrant pain, or Courvoisier sign (non-tender palpable gallbladder with jaundice
4. Hepatomegaly, abdominal palpable mass, ascites, and bowel obstruction on physical examination are presentations that indicate advanced metastatic stage.

Diagnosis:

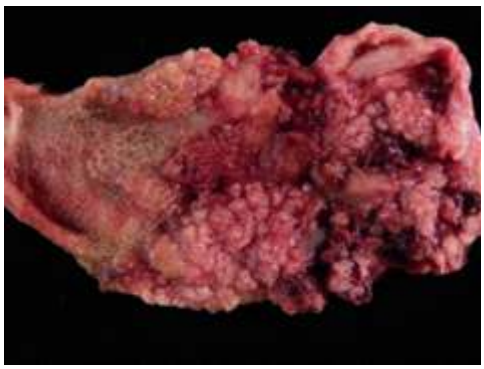
1. Complete blood count, basic chemistry panel, and a liver function test. Results may reveal an unspecific cholestatic pattern caused by bile obstruction requiring decompression.
2. Ultrasonography (US) and computed tomography (CT) are usually the initial imaging studies.
3. Endoscopic ultrasonography (EUS) is considered more accurate than ultrasound (76%) and useful in differential diagnosis to detect histological neoplasia correctly (97%). EUS will provide a valuable tumor-stage description with invasion depth and local lymphadenopathy.
4. Dynamic magnetic resonance (MR) and MR cholangiopancreatography (MRCP) can help assess disease extent more accurately and properly identify unresectable candidates with hepatoduodenal ligament invasion, vasculature encasement, and/or lymph node involvement.
5. Tumor markers, such as carcinoembryonic antigen and carbohydrate antigen19-9, are frequently elevated but considered non-diagnostic due to lack of specificity (CA 19-9 92.7% versus 79.2% CEA) and sensitivity (CA 19-9 50% vs. 79.4% CEA).

Management

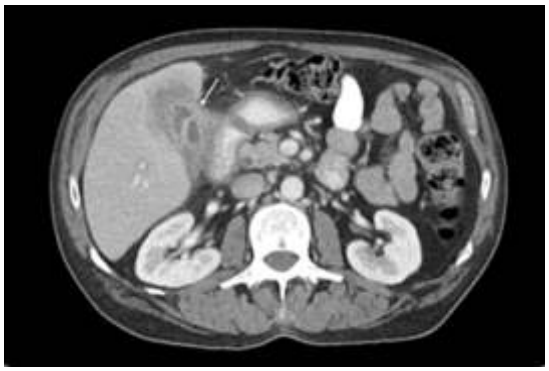
- Surgery is the only curative treatment for patients with Stage II or less and no contraindications (see evaluation). Surgical resection of gallbladder cancer will include cholecystectomy with a marginal hepatectomy with regional lymphadenectomy or common bile duct resection (extended organs may require removal). For gallbladder cancer incidentally found on cholecystectomy pathological specimen with stage T2 or higher, the recommendation is to return for further exploration and re-resection.
- Postoperative chemotherapy should be offered within 8 to 12 weeks and requires baseline laboratory and imaging to re-stage disease before therapy initiation. Adjuvant therapy should be offered to

patients with a resected pathological specimen report of T2 or higher, node-positive, and margin positive, preferably for six months adjuvant chemotherapy (ACT) or alternative four months with concurrent adjuvant chemoradiation (ACRT)

- Neoadjuvant chemotherapy can be offered followed by surgery and adjuvant chemotherapy. Neoadjuvant regimens are laid down by the National Comprehensive Cancer Network and the recommended regimens include Gemcitabine and Cisplatin combination chemotherapy, and oral capecitabine therapy. Patient are offered surgery after 6 cycles as NACT alone is not a cure for gall bladder cancer.



Gall Bladder Cancer, morphology; Pathology Outlines, adapted from UPenn School of Medicine



Gall Bladder Cancer on CT (white arrow); International Contrast Ultrasound Society, 2016

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CHAPTER - 6

RARE DISEASES OF GALLBLADDER

Mirizzi Syndrome-

Refers to common hepatic duct obstruction caused by an extrinsic compression from an impacted stone in the cystic duct or Hartmann's pouch of the gallbladder. Occurs in approximately 0.1% of patients with gallstones. Found in 0.7 to 2.5 percent of cholecystectomies.

CLASSIFICATION

Type I : external compression of the bile duct by a large stone or stones impacted in the cystic duct or in the Hartmann's pouch.

Type II : cholecystobiliary fistula resulting from erosion of the bile duct wall by a gallstone, the fistula must involve less than one-third of the circumference of the bile duct.

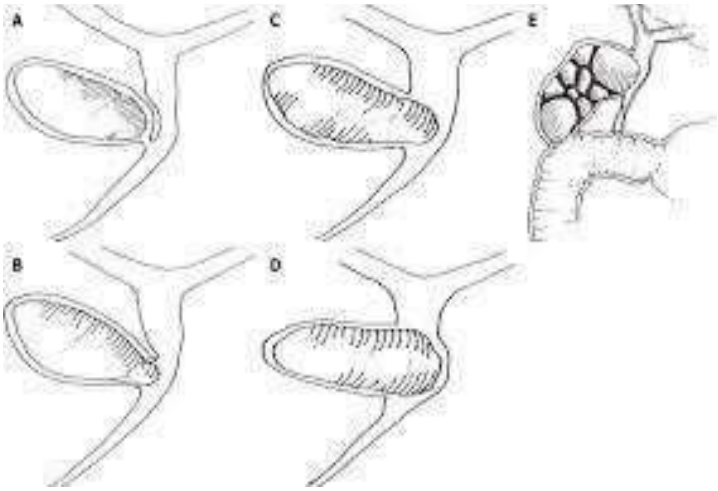
Type III : cholecystobiliary fistula involving up to two-thirds of the bile duct circumference.

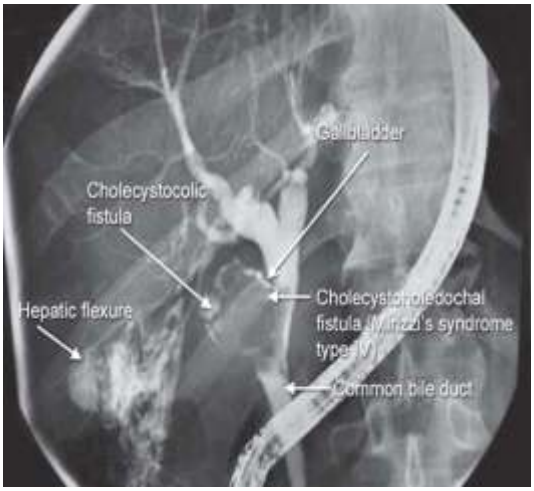
Type IV : cholecystobiliary fistula with complete destruction of the bile duct wall with the gallbladder completely fused to the bile duct forming a single structure with no recognizable dissection planes between both biliary tree structures.

Mirizzi type V, includes the presence of a cholecystoenteric fistula together with any other type of Mirizzi.

Type Va : includes a cholecystoenteric fistula without gallstone ileus.

Type Vb : cholecystoenteric fistula complicated by gallstone ileus.





Treatment

- The treatment of Mirizzi syndrome is surgical
- The surgical treatment of Mirizzi syndrome avoids a truly standardized approach and must be individualized depending on the stage of the disease and the expertise of the surgical team
- Subtotal cholecystectomy – Type 1,2 and 3

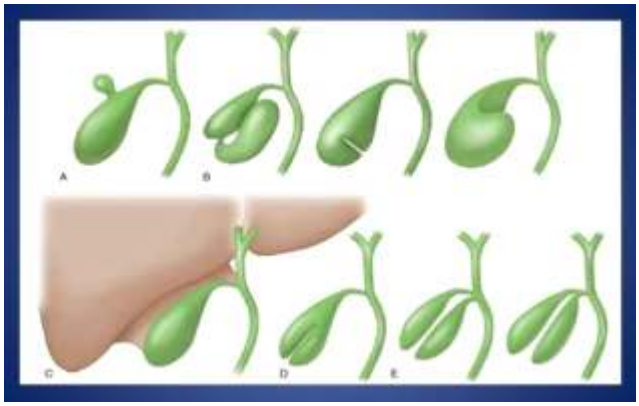
- If a fistula is present (Mirizzi III and IV), besides partial cholecystectomy, a biliary-enteric anastomosis could sometimes be performed between theduodenum and bile duct or between the bile duct and a loop of jejunum *en- Y-de-Roux* Type I - total/ subtotal cholecystectomy
- Type II - subtotal cholecystectomy + choledocoplasty
- Type III - subtotal cholecystectomy + choledocoplasty +/- bilioenteric Anastomosis
- Type IV - bilioenteric anastomosis (A hepaticojejunostomy *en-Y-de- Roux* is preferred)
- Type V-
  - A - Subtotal/total cholecystectomy, primary repair over the bilioenteric fistula
  - B - Treat the acute condition first. Underlying cause 3 months later

CLASSIFICATION GALL BLADDER ANOMALIES

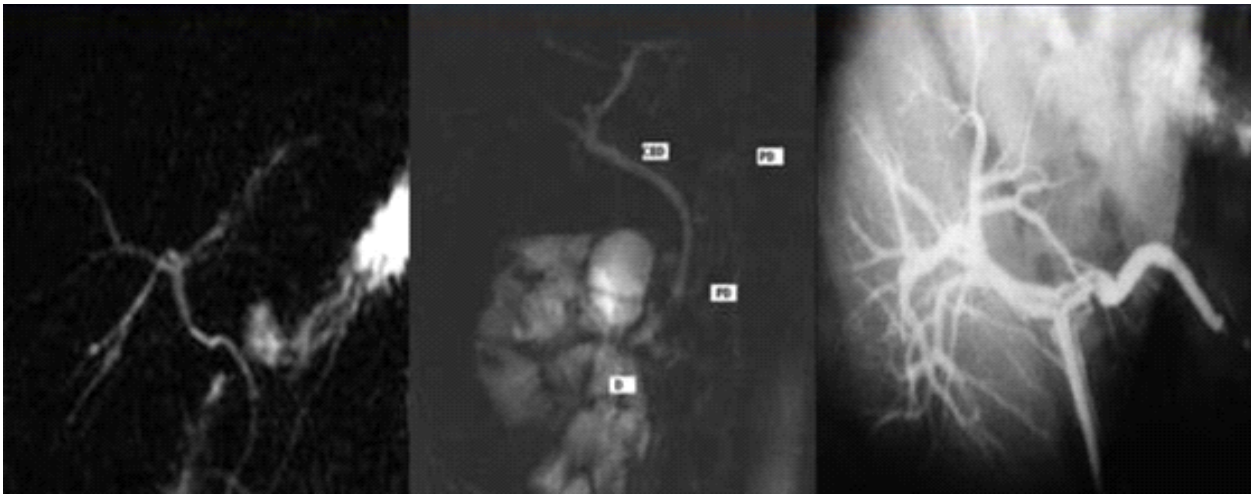
Category	Anomaly
Number	Agenesis, duplication
Position	Ectopic
Shape	Folds and septa, diverticula
Miscellaneous	Heterotopias, wandering gallbladder, congenital adhesions

AGENESIS OF GALL BLADDER

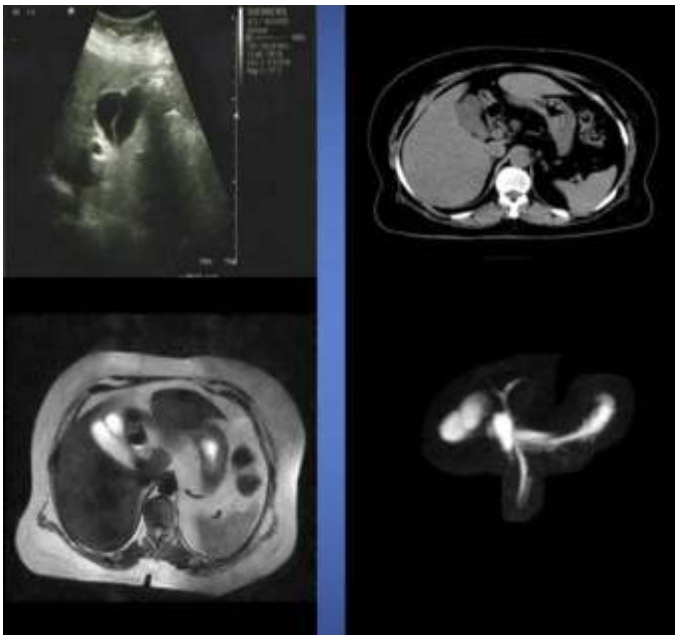
- Congenital absence of the gallbladder is a rare, usually asymptomatic, anatomical variation which is caused by failure of development of caudal division of the primitive hepatic diverticulum or failure of vacouliuzation after the solid phase of embryonic development.
- Nearly 2/3 of adult patients with AGB have biliary tract & etrahepatic biliary calculi are reported in 25%- 50% of pts.



(Adapted from Bailey and love 28th edition)



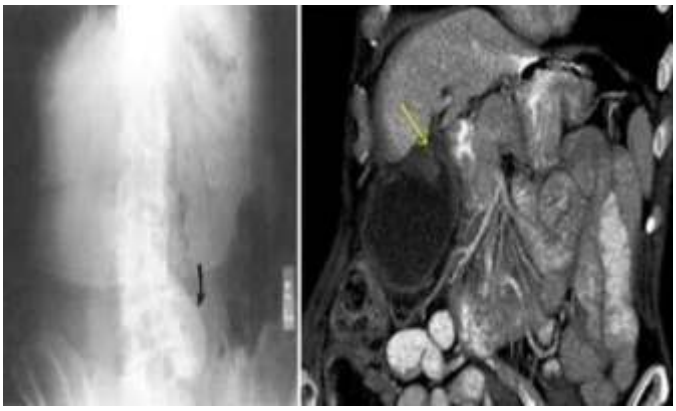
Duplication of gall bladder



- Occurs in about 1 in 4000 people.
- Caused by incomplete re- vacuolization of the primitive gall bladder, resulting in persistent longitudinal; septum that divides the gall bladder lengthwise/ occurrence of separate cystic buds

Wandering gall bladder

- Gall bladder may "disappear" in to the pelvis on upright radiographs or wander in front of the spine or to the left of the abdomen.
- Rarely, the gall bladder can herniate through the foramen of winslow in to the lesser sac.
- The herniation can be intermitten and may be responsible for abdominal pain.



Torsion

Three unusual anatomic situation give rise to torsion of gall bladder, and they all produce twisting of an unusually mobile gall bladder on a pedicle:

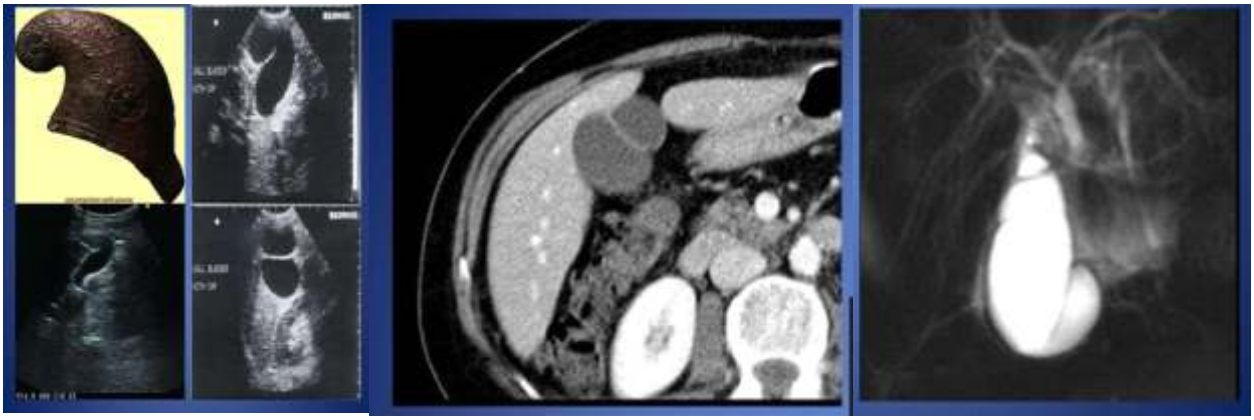
A GB that is completely free of mesenteric or peritoneal investments except for its cystic duct and artery.

A long GB mesentery sufficient to allow twisting.

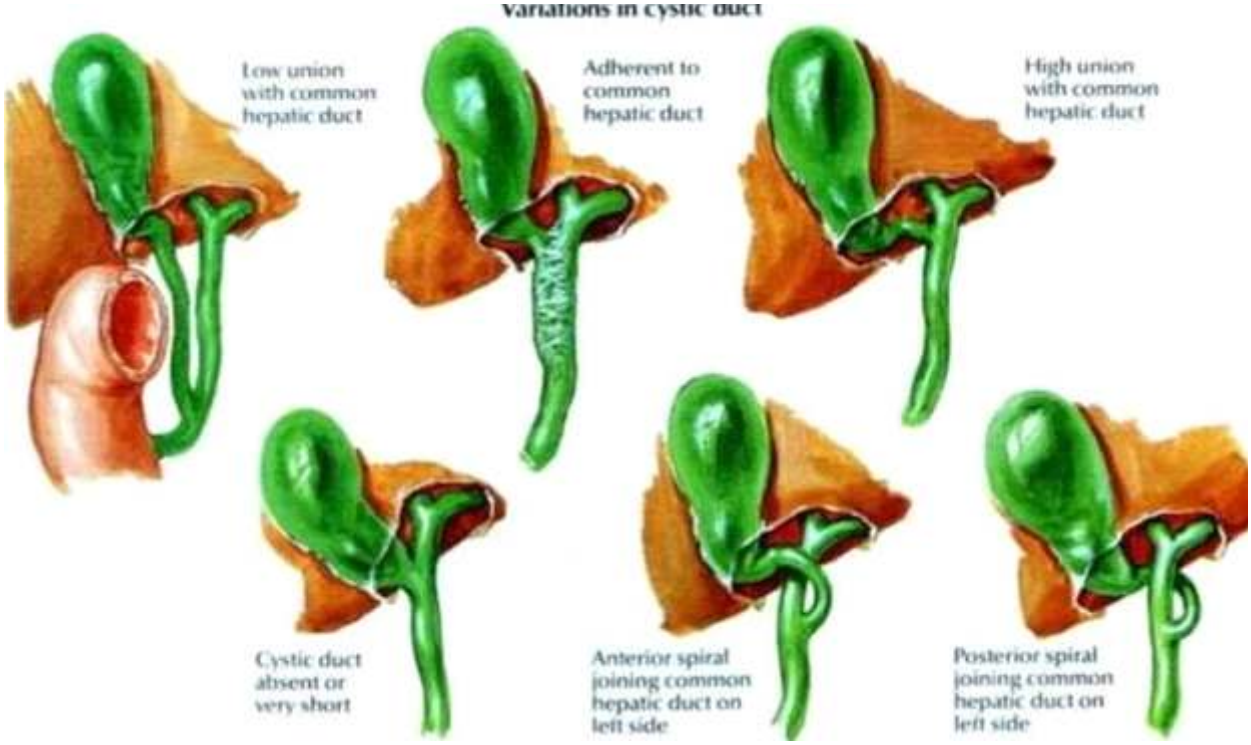
The presence of large stones in the gall bladder mesentery. Most cases of gall bladder torsion occur in women.

Anomalies of gall bladder shape

- Phrygian cap
- Diverticula
- Multiseptate gall bladder



Variation of cystic duct



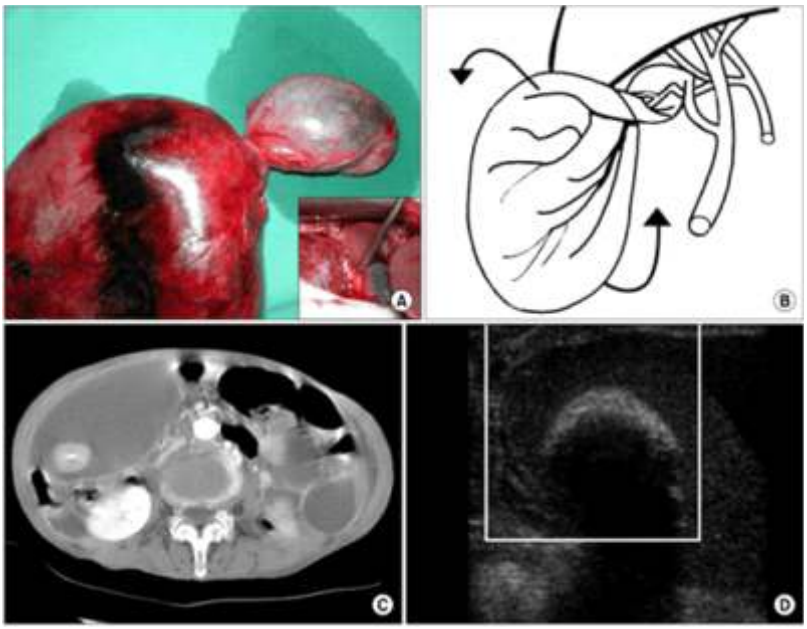
Adapted from Netter images, Variations of extrahepatic Bile ducts

Gall Bladder Volvulus

First described as a case of "floating gallbladder" in 1898, gallbladder volvulus is a rare condition with an incidence of only 1 in 365,520 hospital admissions.

This phenomenon occurs when the gallbladder rotates on its axis , with subsequent interruption of its blood supply and flow of bile.

Emergent cholecystectomy is necessary due to the risk of perforation, bilious peritonitis, and hemodynamic instability. The mortality rate associated with gallbladder volvulus approaches six percent; however, with surgical intervention, the prognosis is excellent.



(Adapted from Semantic Scholar, A literature review of the gall bladder torsion along the axis of gall bladder body, H..Kwon, S.Kim)

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