A STUDY ON CALCULOUS CHOLECYSTITIS IN GRH MADURAI

DISSERTATION SUBMITTED FOR

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THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled “A STUDY ON CALCULOUS CHOLECYSTITIS IN GRH MADURAI” submitted by Dr. P. SUDHA to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S Degree Branch– I (General Surgery) is a bonafide research work were carried out by her under direct supervision & guidance.

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DECLARATION

I Dr. P. SUDHA declare that, I carried out this work on, "A STUDY ON CALCULOUS CHOLECYSTITIS IN GRH MADURAI" at the Department of Surgery, Govt. Rajaji Hospital during the period of December 2007 to October 2009. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, diploma to any other University, Board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S degree examination in General Surgery.

Place : Madurai
Date :
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INTRODUCTION

Gall stones are among the most common gastro intestinal illness requiring hospitalization and frequently occur in young, otherwise healthy people. Most patient remains asymptomatic from their gallstones. Although the mechanism is unclear, some patients develop symptomatic gall stones with biliary colic caused by a stone obstructing the cystic duct.

In my study, I have analysed the prevalence and incidence of calculous cholecystitis diseases. Age incidence, clinical features and investigatory procedure and types of management for gall stone disease.
HISTORICAL ASPECTS

The earliest case of calculous cholecystitis dates back to 21st Egyptian Dynasty (108 s – 945Bc) having been discovered in the mummy of a priestess of Amen. Later, Plinky described the rare anomaly of double gall bladder, and then Soranus of Ephesus, described jaundice and the associated sign of extrahepatic obstruction, including acholuric stools, dark urine and itching. Gall stones were first described by Alexander Trallianus a Greek physician who wrote about calculi in the bile duct.

The first cholecystostomy is credited to John Stongn Bobbs, in Indianapolis on June 15, 1867. His patient, a 32 years old woman, had a large abdominal mass, which proved to be the gall bladder filled with clear serous fluid and gallstones.

Carl Langenbuch of Berlin performed the first cholecystectomy in June 1882, using the aseptic technique that Joseph Lister had initiated in 1886 the first cholecystectomy in the United States was performed by Justus Curvoisier of Basel performed the first successful cholecystostomy
in 1890 and made general contributions to the understanding of bile duct obstruction.

Cholecystography was developed by Graham and Cole in 1924. Operative Cholangiography was described in 1932, Cholangiography by percutaneous transhepatic and Endoscopic retrograde routes has been available since 1950. The applications of ultrasonography computed tomography, choledochoscopy and interventional radiologic techniques computed tomography, choledochoscopy and interventional radiologic techniques to the diagnosis and management of biliary tract diseases home occurred in past 2 decades.
AIM OF THE STUDY

This study comprises of 82 consecutive cases of cholecystitis admitted and treated in Madurai Medical College.

The aim of the study is to analyze the following

1. The incidence and prevalence of calculous cholecystitis disease in and around this region as represented in Madurai Medical College.
2. To study the age and sex incidence
3. To study the clinical features and investigatory procedures that help in diagnosis.
4. To compare and evaluate the management of gall stone disease.
5. To study the nature of procedure and their outcome.
6. To evaluate the post operative complication and their outcome.
SURGICAL ANATOMY

Gall Bladder is located on the visceral surface of the Liver, at the plane dividing the right lobe from the medial segment of the left lobe and surrounded by the connective tissue of the Glison’s capsule. It is a pear shaped organ about 7 to 10cms long. It has an average capacity of 50ml, but it is capable of distending upto 50 times.

The gall bladder is divided into the fundus, body and the neck. The fundus is rounded & usually projects below the inferior margin of the liver, where it comes in contact with the anterior abdominal wall at the level of the tip of the ninth costal cartilage. The body lies in contact with the visceral surface of the liver and is directed upward, backward and to the left. The neck becomes continuous with the cystic duct which turns into the lesser omentum to join the right side of the common hepatic duct, to form the bile duct. The peritoneum completely surrounds the fundus of the gall bladder and binds the body and neck to the visceral surface of the liver.
The right and left hepatic ducts emerge from the right and the left lobes of the liver in the porta hepatic. After a short course, the hepatic ducts unite to form the common hepatic duct. The common hepatic duct is about 4 cms long and descends within the free margin of the lesser omentum. It is joined on the right side by the cystic duct from the gall bladder to form the bile duct.

The cystic duct is 3-4 cms long and its diameter is 1-3 millimeters. The cystic duct contains 5-12 crescent shaped folds of mucosa similar to those seen in the neck of the Gall bladder called the Spiral value of Heistner.

**THE BILE DUCT**

The bile duct is formed near the porta hepatis, by the junction of the cystic duct and the common hepatic duct. It is usually about 7.5 cms long and 6mm in diameter. It descends posteriorly and slightly to the left, anterior to the epiploic foramen, at the Right border of the lesser omentum, in from and to the right of the portal vein and to the right of the hepatic artery proper. It passes behind the first part of the duodenum with
the gastroduodenal artery to its left and then runs in a groove on the superolateral part of the posterior surface of the head of the pancreas, anterior to the IVC and sometimes embedded in the pancreatic tissue.

The Common bile duct ends in the papilla of Vater in the second part of the duodenum surrounded by the spincter of Oddi.

**BLOOD SUPPLY OF THE GALL BLADDER**

- Cystic artery which is usually a branch of Right hepatic artery
- Drained by small veins which enter directly from the gall bladder into the liver. A large cystic vein, follows the cystic artery and drains into the Portal vein.

**LYMPHATIC DRAINAGE**

Majority of the lymph from the subserosal and sub mucosal layers drain into the cystic lymph node of Lund. Efferents from this go to the hilum of the liver. Lymphatics from the gall bladder also pass directly into the liver to connect with subscapsular lymph channels, of the liver.
Other lymphatics drain into lymphnodes in the porta hepatis, then to preaortic lymph nodes

**NERVE SUPPLY**

**SYMPATHETIC:** From coeliac ganglia. On stimulation it inhibits the contraction of the gallbladder.

**PARASYMPATHETIC**

From hepatic branch of anterior vagus, stimulation of which cause contraction of gall bladder and relaxation of the sphincter of Oddi.

**HEPATOCYSTIC TRIANGLE, TRIANGLE OF CALOT**

Formed by the gall bladder and cystic duct to the right common hepatic duct to the left and the margin of the right lobe of the superiorly, the triangle originally described by Calot defined the upper boundary as cystic artery.

**DEVELOPMENTAL ABNORMALITIES OF GALL BLADDER**

1. Longitudinal septate gall bladder.
2. Double gall bladder with common serosa and common cystic duct.
3. Double gall bladder with separate serosa and cystic ducts.
4. Persistent communications between the gall bladder and bile ducts in liver
5. Phrygian cap deformity.

VARIATIONS IN THE BILE DUCTS

1. The common hepatic and cystic ducts lie parallel, being joined by connective tissue before common bile duct is formed.
2. The common hepatic duct and cystic ducts join just before the common bile duct enters the duodenum.
3. The cystic duct joins the common hepatic duct on its left.
4. Accessory right hepatic duct.
5. Absence of cystic duct – the common hepatic duct enters the gall bladder and the common bile duct leaves it.
6. The right hepatic duct joins the neck of the gall bladder.
SURGICAL PHYSIOLOGY

The prime function of the biliary tract is to convey bile from the liver where it is formed in the duodenum. Along the way bile is stored and concentrated in the gall bladder until it is required. Bile helps in the digestion of certain food stuffs and acts as a major excretory pathway.

About 500 ml to 1000 ml of bile are secreted by human hepatocytes every day. The main constituent is water along with bile acids, bile pigments cholesterol, phospholipids and all the inorganic ions found in plasma. The pH of bile duct is generally above 7 and inorganic ions are normally present in concentrations slightly higher than in plasma with the exception of chloride which is usually lower.

COMPOSITION OF BILE

<table>
<thead>
<tr>
<th></th>
<th>HEPATIC</th>
<th>GALLBLADDER</th>
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<tbody>
<tr>
<td>Na (mEq/l)</td>
<td>160</td>
<td>270</td>
</tr>
<tr>
<td>K(mEq/l)</td>
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<td>10</td>
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<tr>
<td>Cl(mEq/l)</td>
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<td>HCO3(mEq/l)</td>
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<tr>
<td>Component</td>
<td>Value</td>
<td>Reference Value</td>
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<tr>
<td>-------------</td>
<td>-------</td>
<td>-----------------</td>
</tr>
<tr>
<td>Ca (mEq/l)</td>
<td>4</td>
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</tr>
<tr>
<td>Mg (mEq/l)</td>
<td>2</td>
<td>-</td>
</tr>
<tr>
<td>Bilirubin (mEq/l)</td>
<td>1.5</td>
<td>15</td>
</tr>
<tr>
<td>Protein (mEq/l)</td>
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<td>-</td>
</tr>
<tr>
<td>Bile Acids (mEq/l)</td>
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<td>150</td>
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<tr>
<td>Phospholipids (mEq/l)</td>
<td>8</td>
<td>40</td>
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<tr>
<td>Cholesterol (mEq/l)</td>
<td>4</td>
<td>18</td>
</tr>
<tr>
<td>Total solids (mEq/l)</td>
<td>-</td>
<td>125</td>
</tr>
<tr>
<td>pH (mEq/l)</td>
<td>7.8</td>
<td>7.2</td>
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**BILE ACIDS**

These are steroid molecules formed from Cholesterol by the hepatocytes and major pathway of cholesterol excretion in the body.

```
CHOLESEROL

PRIMARY BILE ACID | CHOLIC ACID | CHENODEOXYCHOLIC

SECONDARY BILE ACID | DEOXYCHOLIC ACID | LITHOCHOLIC ACID

TERTIARY BILE ACID | URSODEOXYCHOLIC ACID
```
1/3 to ¼ of bile acid pool is lost everyday or converted into secondary bile acids.

CONTROL OF BILE FLOW

Bile flow within the biliary system is regulated by three factors.

Hepatic secretion

Gall bladder contraction and

Bile duct sphincteric resistance

ENTEROHEPATIC CIRCULATION OF BILE ACIDS

Liver secretes 20 – 30 gms of bile acids per day. Total body pool of bile acid at any time is between 3 and 5 gm. This pool circulates twice each meal(i.e.) 6-8 times per day.

After a meal about 98% of the bile salts entering the intestine are absorbed in the ileum, return through the portal vein to the liver, where they are cleared, conjugated feed back mechanism.

The rate limiting enzyme is cholesterol 7 alpha hydroxylase mediate bile acid synthesis. This activity is inversely proportional to the
enterohepatic circulation of bile acid. Only 5% enter the systemic circulation.

In colon, bacteria deconjugate bile acids 1/3 to ¼ primary bile acid pool is lost or converted by anaerobic bacteria to secondary bile acids. About 500 to 700 mg bile acid is lost in faeces.
FORMATION OF GALL STONE

Gall stones are among the most common gastro intestinal illness, requiring hospitalization and frequently occur in young otherwise healthy people with prevalence of 11% to 36% in autopsy report.

Gall stone pathogenesis :

Metabolic :

Cholesterol is synthesized in liver. Its solubility is determined by relative concentration of cholesterol, bile salts and lecithin. Altered level of cholesterol, lecithin and bile salts in bile reduces the micelle concentration in the bile leading to precipitation of insoluble cholesterol. Hence stone formation (Lithogenic bile) Normal ratio of the bile salt and lecithin to cholesterol is 25:1 and ratio below 13 : 1 leads to precipitation of cholesterol. Insoluble cholesterol is within the soluble micelle which is formed by lecithin and bile salts. If cholesterol component increases bile gets supersaturated and inadequate micelle makes insoluble cholesterol to undergo crystallisation and cholesterol monohydrate stone formation (Admiron’s triangular hypothesis)
Any condition which increases the cholesterol secretion in the bile or reduces the bile salt concentration causes cholesterol stone formation.

II Infection and Infestation:

Bacteria like E coli, Salmonella
Parasite like clonarchis sinensis and Ascaris lumbricoides are often associated.

III - Bile stasis

Occurs due to oestogen therapy, pregnancy, vagotomy and in patients who are on long term intravenous fluids or TPN.

IV – Others

Increased bilirubin production due to any of the cause of haemolysis as in hereditary spherocytosis sickle cell anaemia thalasaemia, malaria, cirrhosis.

Altered GB function:

Stasis
Poor emptying
Poor absorption
Infection

Supersaturated bile

Female

Fertile

Fat

Forty

High calone

Altered entero hepatic circulation

Ileal resection

Ileal disease

Altered bowel transit time

Altered bowel flora

Cholestyramine

Deoxycholate

CLASSIFICATION ACCORDING TO CHEMICAL COMPOSITION

1) Cholesterol stone

2) Mixed stone

3) Pigment stone
**CHOLESTEROL STONES**

Their formation in the gall bladder is preceded by the formation of biliary studge. 75% of gall stones are Cholesterol stones in western hemisphere. They contain protein matrix, Cholesterol, bile pigment and varying amount of calcium carbonate and palmitate. They do not harbor bacteria (10%) and not associated with infected bile. They are often radiolucent but cast strong acoustic shadows on USG. They are often multiple and medium sized. When solitary attain large size and have radiating crystalline cross-sectional appearance.

**PIGMENT STONES**

Two types

- Black pigment stone
- Brown pigment stone

**1. BLACK PIGMENT STONES**

They are formed in the Gallbladder. They are composed of bilirubin polymers without calcium, varying amount of cholesterol and a matrix of organic material. Associated infection is present in 20%
patients. They are multiple, small, irregular dark green to black in colour. Haemolytic states predispose to formation of black pigment stone.

2. BROWN PIGMENT STONES

Forms in the bile duct associated with infection of the biliary tract. In 98% of these stones bacteria is present inside. They contain calcium bilirubinate, calcium palmitate and only small amount of Cholesterol.

MIXED STONES

Have varying proportion of all three stones forming constituents of bile.

   e.g.) Cholesterol, bile pigment and calcium.

Effect of Gall stone :

a) In the gall bladder

i) Silent asymptomatic stones occurs in 10% of males and 20% of females.

ii) Biliary coli with periodicity (commonest presentation)

iii) Acute cholecystitis

iv) Chronic cholecystitis

v) Empyema of the gall bladder
vi) Mucocele of gall bladder  

vii) Perforation causing biliary peritonitis  

viii) Carcinoma gall bladder  

b) In CBD  

  Secondary CBD stones  
  Cholangitis  
  Pancreatitis  
  Mirizzi syndrome  

c) In the intestine  

  Cholecystoduodenal fistula causing gallstone ileus
ACUTE CALCULOUS CHOLECYSTITIS

Pathophysiology:

Acute cholecystitis or related to gall stones in 90% to 95% of cases. Obstruction of the cystic duct leading to biliary colic is the initial event in acute cholecystitis and the cystic duct remains obstructed, the gall bladder distends and gall bladder wall becomes inflamed and edematous. Initially acute cholecystitis is an inflammatory process with thickened and reddish wall with subserosal hemorrhage. The mucosa may show hyperemia and patchy areas of necrosis. In most common scenario the gall stone dislodges and the inflammation will gradually resolve.

Clinical presentation:

Right upper quadrant pain

Fever, nausea, vomiting

In Physical Examination:

- Right upper quadrant tenderness and guarding
- Guarding and rigidity sometimes
- A mass, the gall bladder and adherent omentum is palpable
Murphy’s sign - Inspiratory arrest on deep palpation in the right upper quadrant

Mild leucocytosis

Mild elevation of Serum bilirubin, alkaline phosphatase, transaminases and amylase present

**Investigations:**

Ultra sound is the most useful radiographic test for diagnosing acute cholecystitis with sensitivity and specificity of 85% to 95%.

Shows - Presence of thickening of gall bladder wall (6 mm)
- Pericholecystic fluid collection
- Gall bladder distension
- Impacted stone
- and a sonographic murphysign

**Plain X ray Abdomen:**

10% of the gall stones are radio opaque

**Differential Diagnosis:**

1. Duodenal ulcer perforation

2. Acute pancreatitis
3. Acute appendicitis
4. Acute pyelonephritis
5. Acute pneumonia, myocardial infection
6. Ruptured ectopic pregnancy

**Chronic calculous cholecystitis:**

Ongoing inflammation with recurrent episodes of biliary colic or pain from cystic duct obstruction is referred to as chronic cholecystitis.

Although the pathologic changes in the gall bladder can vary, repeated attacks, scarring and nonfunctioning gall bladder are the rule. Histologically, chronic cholecystitis is characterized by an increase in subepithelial and subserosal fibrosis and mononuclear cell infiltrate.

**Clinical features:**

The primary symptoms of chronic cholecystitis is pain, often referred to as biliary colic. The pain is constant and usually last 1-5 hrs. The attack usually last for more than 1hr. But subsides by 24 hrs if persists longer than 1 day acute cholecystitis is likely the underlying etiology.
The physical examination and liver function tests are usually normal in patients with chronic cholecystitis, particularly if they are pain free.

**Investigations:**

- An abdominal USG is the standard diagnostic investigation – gall bladder wall will be thickened with posterior acoustic shadow.
- LFT
- Total count may be raised if there is an acute recurrent infection.

**EMPYEMA WITH GALL BLADDER:**

- It is a type of acute cholecystitis where in the gall bladder is filled with pus. In 30% cases pus may be sterile
- It also can occur in a pre-existing mucocele of the gall bladder where it gets infected.
- It is commonly observed in impacted stones.

**CHOLEDOCHOLITHIASIS**

Choledocholithiasis is the most common cause of obstructive jaundice and cholangitis. May be
1. Primary stone – formed primarily in the CBD and are Brown Pigment stones. Primary stones are rare.

2. Secondary stone – The stone migrate from the gall bladder

3. Retained stones – those discovered within 1 year of cholecystectomy are mostly those missed during surgery.

4. Recurrent stones – stones formed denova in the CBD > 1 year.

Patients with CBD stone may present as

1) Acute cholangitis
2) Acute suppurative cholangitis
3) Acute pancreatitis
4) Painless progressive jaundice
5) Biliary colic
6) Other complications:
   a. Biliary cirrhosis
   b. Hepatic abscess
   c. Bilary duct stricture
CLINICAL FEATURES

It depends on the site of the stone. A stone is situated in the gallbladder may remain asymptomatic lifelong. But when it tries to move out of the gallbladder may get obstructed at the neck of the gallbladder resulting in cholecystitis and dull aching continuous pain. If the gallbladder contracts against obstruction, colicky pain in the right hypochondrium or back will result. The obstruction at the neck may become relieved and the stone may fall back into the GB or pass into the CBD.

In the CBD, if the stone passes out of obstruction it will merely produce mild pain, fever, and jaundice. But if it is obstructed surgical jaundice will result.

Charcot’s triad :

Intermittent pain,

Intermittent fever,

Intermittent jaundice may ensure.

It is due to transient attacks of cholangitis.
Reynold’s Pendad :

Persistent pain
Persistent fever
Persistent jaundice
Shock (toxicity) and
Altered mental status

Investigations :

Bio Chemical

1. Increased levels of Alkaline phosphatase and Gamma glutamyl transpeptidase.
2. Increased levels of Bilirubin (conjugated)
3. Mildly increased level of SGOT and SGPT

Radiological Investigations :

USG :

Dilated CBD
Intrahepatic biliary radicals dilatation
Features of cholecystitis
CT Scan:

Identifies pathology in the liver, pancreas and gall bladder

Lymphnode involvement

Presence of growth can be seen.

ERCP:

The diagnostic and therapeutic mode of investigation. If bilirubin is more than 10mg% and preoperative biliary stenting is contemplated, ERCP is used.

With side viewing endoscope the ampullary region may be seen and if any growth is found it can be biopsied.

Both bile duct and pancreatic duct may be cannulated and dye injected to visualize both the bile duct and the pancreatic duct.

Brush cytology during ERCP may be done.

It may show site of obstruction in CBD with proximal dilatation.

MRCP:

If Bilirubin is less than 10mg% and preoperative stenting is not contemplated MRCP is the investigation of choice.
Non invasive investigation

Gives very good delineation of biliary tree and pancreatic duct.

Any pathological lesion in the bile duct and pancreatic duct may be diagnosed.

7. PERCUTAEOUS TRANSHEPATIC CHOLANGIOGRAPHY

It is used for visualization of the biliary tract in jaundiced patients and can be modified to allow percutaneous transhepatic drainage and insertion of endoprosthesis. The accuracy of PTC in detecting level and cause of biliary obstruction averages 90%. The procedure is carried out under sedation using Chiba needle (22G) under fluoroscopic guidance. With the use of MRCP, PTC has limited role.

Complications of PTC and ERCP

1. Bacteraemia
2. Biliomia
3. Haemorrhage
4. Bile embolization
5. Intrahepatic arterio portal fistula.
6. Pneumothorax.
7. Contrast reactions.
CLINCHING THE DIAGNOSIS

1. ACUTE CHOLECYSTITIS

- Fat, fertile, female or fifty
- H/o flatulent dyspepsia and belching
- Pain in right hypochondrium radiating to the inferior angle of the right scapula or the top of right shoulder.
- Nausea, retching, vomiting, pyrexia, elevated pulse rate.
- Jaundice may be present.
- Charcot’s triad – pain, jaundice and fever.
- Tenderness and rigidity.
- Gall bladder is hardly palpable.
- Elevated WBC count.
- Rise in Serum bilirubin may be present.
- Oral Cholecystography is contra indicated.
- Ultra sonogram and radioactive scanning are helpful in diagnosis.
2. CHRONIC CHOLECYSTITIS

- Feeling of distension
  - Pain over right hypochondrium often radiating to the inferior angle of right scapula.

The pain becomes worse after taking fatty foods.

- Nausea is common.
- Flatulent dyspepsia is common.
- Jaundice may be present and it is usually due to associated cholangitis or obstruction.
- Murphy’s sign
- Oral cholecystography, ultrasonogram and CT are diagnostic.
MANAGEMENT

1. ACUTE CHOLECYSTITIS

- Intra venous fluid and Electrolyte replacement
- Nasogastric suction.
- Systemic antibiotics
- Parenteral analgesia
- Nothing by mouth to reduce the cholecystokinin release from the upper small bowel in order to minimize gall bladder stimulation.

The timing of surgery is decided by the severity of the attack.

Indications for emergency surgical intervention.

1. Progression of the disease despite conservative treatment
2. Failure to improve within 24 hours especially in patients >60 years
3. Presence of an inflammatory mass in the right hypochondrium.
4. Detection of gas in the gall bladder/biliary tract
5. Established generalized peritonitis.
SEVERE PROGRESSIVE DISEASE

The exact procedure depends on the operative findings.

In patients with a tense empyema, preliminary decompression of gall bladder contents using a Mayo Ochsner suction trocar canula followed by cholecystectomy. At times the precarious condition of the patient precludes a lengthly operation or the anatomy may be obscured by the inflammatory mass as to render the cholecystectomy hazardous. In these situations a cholecystostomy should be performed.

Subtotal cholecystectomy is performed as an alternative approach to cholecystectomy. In this procedure, the posterior wall of the gallbladder is left in situ, attached to the liver, and the cystic duct is secured from within the gall bladder lumen by a purse string suture.

A. DELAYED CHOLECYSTECTOMY

Patient is conservatively managed during the acute episode with discharge of the patient after complete resolution of the attack. Subsequently the patient is admitted 6 weeks later for an elective surgery (open cholecystectomy / lap cholecystectomy).
**CHRONIC CHOLECYSTITIS**

The treatment of chronic cholecystitis is surgical – cholecystectomy. Peroperative cholangiography should be considered as an integral part of cholecystectomy. The cholangiographic findings, the presence of jaundice together with the operative appearances, dictate the need for exploration of the CBD. Routine uneventful cholecystectomy without exploration of common bile duct does not require the insertion of a subhepatic drain, but most biliary surgeons recommended drainage for the following.

1. Difficult cholecystectomy
2. Early cholecystectomy in acute cholecystitis
3. In patients who require common bile duct exploration laparoscopic cholecystectomy is now firmly established as the gold standard therapy for symptomatic gall stone disease.
CHOLECYSTECTOMY

INDICATIONS

All pathological conditions of the gall bladder viz.

1. cholecystitis,
2. cholelithiasis,
3. Empyema of gall bladder,
4. carcinoma.

PREOPERATIVE MEASURES

1. A high carbohydrate diet to ensure adequate stores of glycogen. In very ill patients, high carbohydrate intravenous infusions are employed.
2. Adequate preoperative intravenous hydration
3. Broad spectrum antibiotic therapy is used in the acute case and where common bile duct exploration is anticipated.
   If the patient is jaundiced, then the following additional preoperative measures are to be undertaken.
4. If prothorombin time is prolonged, it should be corrected before operation by the administration of vitamin K, 10mg/day by i.m.
injection. If there is fresh frozen plasma made available for the preoperative period.

5. Longer course of higher parenteral antibiotics

6. Renal tubular function is compromised to a greater or lesser degree in jaundiced patients due to a direct action of bilirubin on the tubules and to a degree of vascular shunting in the kidney leading to relative cortical ischaemia. Adequate preoperative hydration is essential to minimize the risk of acute renal failure, and frequently obviates the need for preoperative mannitol infusion.

7. Adequate preoperative carbohydrate intake and hydration, combined with measures to control infection and minimize hemorrhage should reduce considerably the risk of this severe complication.

1. OPEN CHOLECYSTECTOMY

POSITION OF THE PATIENT

Supine on an operating table which can accept X ray cassettes for peroperative cholangiography with a 15° tilt.
INCISIONS

- Right paramedian
- Kocher’s subcostal
- Mayo robson (Hockey stick)
- Right upper quadrant transverse
- Upper midline.

PRELIMINARY EXPLORATION

The entire biliary system is explored. The stomach & duodenum examined to rule out associated ulcers, the pancreas to exclude chronic pancreatitis, pancreatic calculi or small carcinoma.

PRINCIPAL METHODS

1. Duct first method: The cystic duct and the artery are dissected first and divided, after which the gall bladder is removed.

2. Fundus first method: The dissection starts from the fundus of the gall bladder and gradually proceeds towards the cystic duct which is divided last of all.
RISKS OF THE OPERATION

1. The common bile duct and the right hepatic artery may be injured.

2. An undetected duct may escape and lead to biliary peritonitis.

3. Haemorrhage due to slipping of ligature of the cystic artery.

4. Accumulation of bile due to biliary leakage from some unknown duct leading to Waltman-Walter syndrome, manifested by upper abdominal pain or chest pain associated with tachycardia and a persistent low blood pressure. If there is any suspicion, an urgent ultrasound scan followed by emergency re-exploration of the abdomen is carried out.

Indications for CBD exploration:

After open cholecystectomy

1. Ultrasound shows stone in CBD

2. Palpable stone in CBD

3. CBD diameter > 10 mm

4. Recent H/o Jaundice and with raised serum alkaline phosphatase level.

5. On table cholangiogram shows stone

6. When in doubt.
Management of CBD Stones:

After the removal of gall bladder on table cholangiogram is done through cystic duct using soluble iodine dye to see any stones in CBD.

After choledochotomy, stones are removed using Desjardin’s choledocholithotomy forceps.

‘T’ tube is then placed in CBD and kept for 14 days.

After 14 days post operative ‘T’ tube cholangiogram is done to see for free flow of dye into the duodenum, so that ‘T’ tube can be removed.

If ‘T’ tube cholangiogram shows persistent stone it can be extracted after 6 weeks, through the basket (Dornea) catheter (fogarty) through the track or through a choledochoscope. Refaced stones can be removed through ERCP.

Methods to confirm the removal of T tube

Clamp the T tube, after 10-14 days and observe 48 hours for development of pain, jaundice and fever.

Confirm free flow of dye in T tube cholangiogram.
Management of retained CBD stones:

- Small stones may spontaneously pass down
- Heparinised saline or bile acid flushing through the T tube (Wash 250 ml of normal saline with 25,000 IV heparin for 5 days)
- Burhenne technique – after 6 weeks one T tube track gets matured, track if needed is dilated using graduated dilators. Either using Dormia basket or Forgarty catheter or choledochoscope, stone is removed through T tube tract under fluoroscopic guidance (C-ARM)
- ERCP and stone removal in 3 weeks.

2. LAPAROSCOPIC CHOLECYSTECTOMY

Laparoscopic cholecystectomy has become widely accepted as the procedure of choice for patients with symptomatic cholelithiasis. Laparoscopic cholecystectomy clearly has been shown to be associated with decreased pain, shorter hospitalization, a reduced period of post operative disability and decreased hospital costs.

PATIENT SELECTION

Thin female patients with small stones in a thin walled non obstructed gall bladder and with no previous abdominal surgery.
PREOPERATIVE PATIENT MANAGEMENT

One hour prior to surgery, the patient empties the urinary bladder, starts primary pain relief with a suitable NSAID and receives pre-medication. There is no need for routine urethral catheterization or nasogastric intubation.

DISSECTION TECHNIQUE

Gall bladder retraction, correct tissue tension and the ability to rotate Calot’s triangle to work both anteriorly and posteriorly are the keys to good dissection technique.

The basic choice lies between the American approach and the French approach. For the American approach, the patient is placed supine, both the fundus of the gall bladder and the neck of the gall bladder are fixed and retracted and the main working port is in the epigastrium.

In the French approach, the patient is in the Lloyd Davis position with surgeon operating from between the legs. The fundus of the gall bladder lies free while retracting is upon the neck of the gall bladder with counter traction on the porta hepatis.
RULES OF DISSECTION – THE EDINBURGH METHOD

1. Initial display of the biliary anatomy
2. Skeletalization
3. Final display of biliary anatomy
4. Controlled response to bleeding
5. Stay out of the liner
6. Know when to stop

EQUIPMENT

1) Video Equipment
2) Laparoscopic Instruments
   These include instruments to create and maintain a pneumoperitoneum, those used for grasping cutting and dissecting tissue, those used for ligating, clipping or suturing vessels and others structures and those used to maintain a clear field, such as suction and irrigation devices.
3) Energy source

   Electrocoagulators, diathermy units lasers, adequate dissection instruments, the harmonic scalpel and ultrasonic dissection instruments.
OPERATIVE TECHNIQUE

1) Preparation and positioning
2) Establishing pneumoperitoneum
3) Primary port placement
4) Secondary port placement
5) Dissection of Calot’s Triangle
6) Cholangiography
7) Dissection of Gallbladder from liver bed
8) Extraction of gall bladder
9) Closure.

PEROPERATIVE DIFFICULTIES

1. The Pneumoperitoneum may be difficult to create because of previous surgery with adhesions to the scars.
2. Dense adhesions around an inflamed gall bladder. If there is much inflammation around the Calot’s triangle a safe way to proceed is to dissect around the back of Hartman’s pouch and elevate this from its liver bed.
3. Anatomical variation of the cystic duct, especially of the short cystic duct and the cystic duct draining directly into the right hepatic duct.

4. Variations of the cystic artery.

5. Rupture of the gall bladder leading to escape of bile and stones.

ADVANTAGES OF LAPAROSCOPIC OVER OPEN CHOLECYSTECTOMY

1. Shorter hospital stay
2. Far less post operative pain
3. Early return to work
4. Less scars
5. Lesser incidence of wound infections
6. Lesser wound related complications
7. Rapid postoperative mobilization with fewer incidences of chest infections and deep vein thrombosis.
8. Greatly diminished contact with patients’ blood and other body fluids which in turn reduce the risk of viral disease transmission.
Carcinoma Gall Bladder:

- It is more common in India and Asian countries
- It is common in females and elderly. Male : Female ratio is 1:3

Etiology:

- 90% of carcinoma gall bladder is associated with gall stones
- Choledochal cyst,
- Gall bladder polyp > 1 cm in size or more than 3 in number
- Chronic typhoid carriers
- Porcelain gall bladder
- Nitrosamines

Treatment:

Cholecystectomy with resection of liver segments IV and V – extended cholecystectomy with perihepatic nodal clearance.

Hemi hepatectomy with cholecystectomy with nodal clearance.

Prognosis:

Poor prognosis

5 year survival is only 5%
ETIOLOGY
RISK FACTORS FOR PREVALENCE OF CHOLECYSTITIS

• Female sex

• Obesity

• Advanced age

• Genetics and ethnic factors

• Diet highly refined, fiber depleted, rich in fat

• Diabetes mellitus

• Ileal disease and resection

• Haemolytic states

• Infection of biliary tract

• Parasitic infestations

• Cirrhosis

• Cystic fibrosis

• Pregnancy

• Drugs - Clofibrate

- Thiazide diuretics

- Oral contraceptives
MATERIALS AND METHODS

The study material consist of 82 cases of Cholecystitis in all the seven surgical units of our department of Madurai Medical College Hospital, Madurai during the period from December 2007 to October 2009.

All cases of pain abdomen admitted in the surgical ward were carefully and thoroughly examined to arrive at a clinical diagnosis. A preformed proforma was carefully filled up giving particular importance to the duration of illness, general and special investigations, and wherever possible, histopathological examination of the specimen was carried out.

The following procedures were adopted according to the condition of the patient, ie.

1. Conservative management,
2. Open cholecystectomy,
3. Open cholecystectomy with T-tube drainage,
4. Laparoscopic cholecystectomy.
If selected cases, preoperative, peroperative and post operative clinical and operative photographs were taken. All the patients were followed up in the immediate post operative period and in the subsequent period ranging from 3 months till the end of the study period.
OBSERVATION AND RESULTS

Table -1 : Age Incidence

<table>
<thead>
<tr>
<th>Age in years</th>
<th>No.of Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>21 – 30 years</td>
<td>16</td>
<td>19.4</td>
</tr>
<tr>
<td>31 – 40 years</td>
<td>15</td>
<td>18.1</td>
</tr>
<tr>
<td>41 – 50 years</td>
<td>28</td>
<td>34.1</td>
</tr>
<tr>
<td>51 – 60 years</td>
<td>10</td>
<td>12.1</td>
</tr>
<tr>
<td>61 – 70 years</td>
<td>11</td>
<td>13.3</td>
</tr>
<tr>
<td>71 – 80 years</td>
<td>2</td>
<td>3.0</td>
</tr>
<tr>
<td>Total</td>
<td>82</td>
<td>100</td>
</tr>
</tbody>
</table>

In my study, increased incidence of gall stone disease was between 41 – 50 years.
<table>
<thead>
<tr>
<th>Sex Incidence</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>21 – 30 years</td>
<td>5</td>
<td>11</td>
</tr>
<tr>
<td>31 – 40 years</td>
<td>3</td>
<td>12</td>
</tr>
<tr>
<td>41 – 50 years</td>
<td>7</td>
<td>21</td>
</tr>
<tr>
<td>51 – 60 years</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>61 – 70 years</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>71 – 80 years</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>22</td>
<td>60</td>
</tr>
</tbody>
</table>

Females have increased incidence to have gall stone disease.
Table -3: Clinical Presentation

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Clinical Presentation</th>
<th>No.of Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Right hypochondrial pain</td>
<td>76</td>
<td>92.7</td>
</tr>
<tr>
<td>2</td>
<td>Jaundice</td>
<td>12</td>
<td>14.6</td>
</tr>
<tr>
<td>3</td>
<td>Right hypochondrial tenderness</td>
<td>65</td>
<td>79.3</td>
</tr>
<tr>
<td>4</td>
<td>Associated with fever</td>
<td>22</td>
<td>26.8</td>
</tr>
<tr>
<td>5</td>
<td>Gall bladder palpable</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In my study, almost all the patients have right hypochondrial pain and right hypochondrial tenderness.
### Table -4 : Pathology

<table>
<thead>
<tr>
<th>Pathology associated with</th>
<th>No.of Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calculous with CBD stone</td>
<td>11</td>
<td>13.4</td>
</tr>
<tr>
<td>Calculous without CBD stone</td>
<td>71</td>
<td>86.6</td>
</tr>
<tr>
<td>Total</td>
<td>82</td>
<td>100</td>
</tr>
</tbody>
</table>

Out of 82 patients, only 11 patients have associated CBD stones

### Table -5 : Associated Diseases

<table>
<thead>
<tr>
<th>Associated Diseases</th>
<th>No.of Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Silent Stones</td>
<td>6</td>
<td>7.3</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>12</td>
<td>14.6</td>
</tr>
</tbody>
</table>
### Table -6 : Investigations

<table>
<thead>
<tr>
<th>Investigations</th>
<th>No.of Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated Serum bilirubin</td>
<td>9</td>
<td>11.0</td>
</tr>
<tr>
<td>Elevated serum alkaline phosphate</td>
<td>7</td>
<td>8.5</td>
</tr>
<tr>
<td>Elevated serum cholesterol</td>
<td>49</td>
<td>59.7</td>
</tr>
</tbody>
</table>

### Table -7 : Ultraonogram Findings

<table>
<thead>
<tr>
<th>Findings</th>
<th>No.of Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gall stones</td>
<td>71</td>
<td>86.6</td>
</tr>
<tr>
<td>Gall stones with CBD stones</td>
<td>11</td>
<td>13.4</td>
</tr>
<tr>
<td>Total</td>
<td>82</td>
<td>100</td>
</tr>
</tbody>
</table>

Almost 86.6% of patients shows gall stones and 13.4 % shows CBD stones.
# Table 8: Management

<table>
<thead>
<tr>
<th>Management</th>
<th>No.of Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Open cholecystectomy</td>
<td>59</td>
<td>71.9</td>
</tr>
<tr>
<td>Lap. Cholecystectomy</td>
<td>23</td>
<td>28.0</td>
</tr>
<tr>
<td>Cholecystectomy with CBD explo</td>
<td>11</td>
<td>13.4</td>
</tr>
<tr>
<td>Emergency conservative</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In my study, 59 patients were treated with open cholecystectomy and 23 patients with lap. Cholecystectomy and 11 patients with CBD exploration and none of the patient was taken for emergency surgery.
In this study 84% of stones were mixed type, 5 were pure cholesterol and 11 were pigment stones.
The overall prevalence in my study was found to be 9% and this correlate with prevalence in South India and United States.
DISCUSSION

The study included 82 consecutive cases of cholecystitis, who were admitted and treated in all the four surgical units of Govt. Rajaji Hospital Madurai Medical College, Madurai. The overall incidence of cholecystitis in our hospital was 0.97%.

The patient’s details were entered in a typed proforma with necessary details for the study preoperatively, and were followed up post operatively till the time of discharge of the patients and for two months henceforth. The rest of the data was provided by the medical records officer.

The incidence, age and sex incidence, urban and rural incidence, associated diseases, bacteriological status of bile, the common clinical presentations, the different types of management were analysed and discussed in relevance to each of the patients. The various investigations that were available in our Institution have been used, which include biochemical and radiological investigations. The radiological investigations included plain radiographs, ultrasonography, MRCP, T-tube cholangiography and CT scan.
The various treatment options were considered for each of the cases and each was provided the best optional treatment available in our institution. The incidence and the further management of the post operative complications that occurred are also discussed.

The incidence of cholecystitis is found to be increased in our institution during the past few years. The age range of the patients admitted and treated for cholecystitis was between 21 and 80 years and the mean age was 43 years.

The incidence of cholecystitis was more in females than in the males, the ratio being 2:1. There were 53 females and 27 male cases of cholecystitis in the study.

The commonest presenting symptom was right hypochondrial pain which was seen in 76 patients i.e. 92.7%. Jaundice was present in 12 patients. 22 patients presented with associated pyrexia, 4 patients had previous history of peptic ulcer surgery. Murphy’s was positive in 65 of the 82 cases which amounted to 79.3%. There was a palpable gallbladder in 6 patients.
The cholecystitis was the calculous type in 71 patients, 11 patients with calculous cholecystitis had associated common bile duct stones.

There was associated chronic duodenal ulcer with gastric outlet obstruction in 3 patients. 6 patients had associated diabetes mellitus, 9 patients presented with elevated serum bilirubin, 7 had elevated serum alkaline phosphatase, 49 patients had elevated serum cholesterol.

Ultrasonography was done for all the patients. 71 patients had visualized gallstones on ultrasonography, and 11 patients presented with associated common bile duct stones diagnosed ultrasonographically. Out of 6 patients three patients were done MRCP.

2 patients had conservative management which was later followed by elective cholecystectomy. 59 cases had elective open cholecystectomy. 23 patients had laproscopic cholecystectomy done. Cholecystectomy with common bile duct exploration and T-tube drainage was done for 11 patients. At the end of 14 days ‘T’ tube was clamped and the patient was observed for pain after that ‘T’ tube cholangiogram was done and after confirming no residual stones. ‘T’ tube was removed.
6 patients had post operative wound infection. 2 patients had post operative wound infection, 2 patients had biliary fistula which was dealt with conservatively. 1 patient had retained common bile duct stones.
SUMMARY

In my study of 82 patients increased incidence of age for gall stone disease was between 41-50 years and females was found to have gall stone disease than males and almost most of the patient have right hypochondrial pain and tenderness and only 6 patients have been incidently diagnosed to have gall stones and they were preceeded with cholecystectomy with incisional hernia.

Out of 82 patients 72 patients have only gall stones and 11 patients have associated CBD stones and those patients was proceeded with cholecystectomy with CBD exploration.

59 patients was treated with open cholecystectomy and 23 patients with lap cholecystectomy.

Outcome of overall prevalence of gall stone disease is correlated with prevalence in South India and United States.
CONCLUSION

1. The overall incidence of cholecystitis was 9% out of all admission in my study period.

2. The age incidence varies between 21-80 years with a peak incidence in the age group of 41-50 years

3. Female preponderance of 2 : 1 was observed in my study. While the Indian studies showed a more incidence in males the western showed a more male preponderance

4. Mixed stones are the most common type of gall stone.

5. Common presenting symptom was right hypochondrial pain. The commonest sign was Right hypochondrial tenderness.

6. Ultrasonogram was the investigation of choice because of its simplicity, safety, repeatability and accuracy, it was used in all the cases in my study.

7. Elective open cholecystectomy was the widely followed method of surgical treatment.

8. The increasing trend towards laproscopic cholecystectomy was obvious in my study. The trend was due to shorter hospital stay, lesser post operative pain and faster recovery.
ACUTE CHOLECYSTITIS
DISTENDED GALLBLADDER AND CBD
CHRONIC CHOLECYSTITIS
GALL BLADDER
SPECIMEN OF GALLBLADDER WITH GALL STONES
T-TUBE DRAINAGE
CYSTIC DUCT LIGATED
PORTS IN LAPAROSCOPIC CHOLECYSTECTOMY
LAPAROSCOPIC CHOLECYSTECTOMY
ULTRASOUND PICTURE OF GALLSTONES
EFFECT OF GALLSTONES

- Perforation (Hartmann’s)
- Mirizzi syndrome
- CBD stone
- Pancreatitis
- Carcinoma
- Abscess
- Perforation (fundus)
- Gallstone ileus
SEX DISTRIBUTION

- 21 – 30 years: 5 Male, 11 Female
- 31 – 40 years: 2 Male, 12 Female
- 41 – 50 years: 7 Male, 21 Female
- 51 – 60 years: 2 Male, 8 Female
- 61 – 70 years: 6 Male, 6 Female
- 71 – 80 years: 2 Male, 2 Female

Legend: Male in blue, Female in purple
PROFORMA

S.No. Ward Address

Name

Age/sex

IP No.

Date of admission

Date of surgery

Date of discharge

Complaints

• Right hypochondrial pain
• Radiating to shoulder and back
• Nausea / vomiting
• Relation with fatty food
• Flatulent dyspepsia
• Fever
• Jaundice

Past history

• Diabetes mellitus / hypertension / asthma
H/o Typhoid

H/o ileal disease / resection / bypass surgery

**Drug History**

- H/o Oral contraceptive pills
- H/o cholestyramine therapy
- H/o clofibrate
- H/o TPN

**Personal History**

- Mixed diet
- High fat

**Menstrual History**

**Marital History**

**General Examination**

- Height
- Weight
- Pulse rate
- Blood pressure
- Jaundice
- Anemia
Clinical Examination

Inspection

- Abdomen shape
- Moves with respiration
- Visible lump

Palpation

- Warmth
- Tenderness
- Guarding / Rigidity
- Palpable gall bladder
- Signs of peritonitis
- Associated signs of pancreatitis
- Other findings

Investigations

- Urine
  - Haemoglobin
    - Albumin
    - Sugar, deposit
- Total count
- Differential count
- ESR
- Blood urea sugar creatinine
- LFT
- Lipid profile
- X-ray abdomen erect
- Ultrasound abdomen and pelvis
- Upper GI Endoscopy

**Treatment**

- Conservative
- Surgery
  - Open/Lap
- Operative findings
- Biopsy report
- Biochemical analysis of stone

Follow up
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na</td>
<td>Sodium</td>
</tr>
<tr>
<td>K</td>
<td>Potassium</td>
</tr>
<tr>
<td>Cl</td>
<td>Chlorides</td>
</tr>
<tr>
<td>HCo$_3$</td>
<td>Bicarbonate</td>
</tr>
<tr>
<td>Ca</td>
<td>Calcium</td>
</tr>
<tr>
<td>Mg</td>
<td>Magnesium</td>
</tr>
<tr>
<td>TPN</td>
<td>Total Parental Nutrition</td>
</tr>
<tr>
<td>USG</td>
<td>Ultrasonogram</td>
</tr>
<tr>
<td>CBD</td>
<td>Common Bile Duct</td>
</tr>
<tr>
<td>MRCP</td>
<td>Magnetic Resonance cholangio pancreaticogram</td>
</tr>
<tr>
<td>PTC</td>
<td>Percutaneous trans hepatic Cholangiography</td>
</tr>
<tr>
<td>ERCP</td>
<td>Endoscopic retrograde Cholangiogram</td>
</tr>
</tbody>
</table>


5. Lee Mc Gregor’s Synopsis surgical anatomy.


7. Ashby BS. Acute and recurrent torsion of the gallbladder. BrJ Surg 1965;52:


