A STYDY OF POST CHOLEYCYSTECTOMY BILIARY LEAKAGE AND ITS MANAGMENT

Dissertation submitted to

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

In partial fulfilment of the requirements for the award of the degree of

M.S DEGREE EXAMINATION

(GENERAL SURGERY)

INSTITUTE OF GENERAL SURGERY

TAMIL NADU DR.M.G.R.MEDICAL UNIVERSITY

CHENNAI – 600 001

MAY 2020

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled '**A STYDY OF POST CHOLEYCYSTECTOMY BILIARY LEAKAGE AND ITS MANAGEMENT'** is a bonafide and genuine research work carried out by me under the guidance of **Prof. DR.P.THANGAMANI.,M.S,** Institute of General Surgery, Madras Medical College,Chennai-03.

This dissertation is submitted to **THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY CHENNAI** in fulfilment of the degree of M.S. General Surgery examination to be held in **April 2020.**

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ACKNOWLEDGEMENTS

I like to thank to all my coollegues and teachers and my professors and parents who stood with me in all my tough situations.

I thank every person involved in made this manuscript from inception to publication which I submitted.

I am most thankful to **Prof JEYANTHI MD** , Dean, Madras Medical College and Hospital for the opportunity to conduct this study in the Department of General Surgery, Madras medical college Chennai.

My gratitude to my guide and mentor **Prof.P.THANGAMANI.MS.,** Institute of General Surgery, Madras Medical College, who has inspired me during my training as a post graduate student.

I am very grateful to **Prof KANNAN.MS.,** Director of the Institute of General Surgery for the encouragement and unrestricted permission to view the ethical works and publications. I also acknowledge the invaluable advice and counseling received from **DR.K.SENTHILKUMAR.,MS,DR.SIVAN, DR.PRASANNA.,MS,MCH.**.This study would have not been possible without the support of my fellow post graduates and interns who have been a source of help in need.

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INTRODUCTION

Laparoscopic cholecystectomy is now considered the gold standard for the treatment of symptomatic gallstone disease. The preferred treatment for symptomatic gallstone disease is cholecystectomy. Open cholecystectomy remained the preferred surgical option for patients with symptomatic gallstone disease until the advent of laparoscopic cholecystectomy in the late 1980s. Because early results were so promising regarding decreasing pain, costs, and hospital stay, rapid adoption of the laparoscopic technique ensued worldwide (Escarce et al, 1995; Legorreta et al, 1993; Nenner et al, 1994; Steiner et al, 1994). Shortly thereafter, there was a rising concern that the new laparoscopic technique was associated with an increased risk for common bile duct (CBD) injuries .After this was recognized, increased efforts in awareness, education, and training have decreased the risk.

The problem seems was more acute in developing nations such as India. While objective data may be lacking , the most tertiary care institutions was seeing a larger number of patients with bile duct injuries. In the open cholecystectomy era the incidence of bile duct injury was 0.1% to 0.2%. An increase in iatrogenic bile duct injury from 0.1% to 0.2% up to 0.4% .

Through the early 1990s it was thought that the high rate of this complications was because of a learning curve associated with laparoscopic procedures. However , this high rate has reached a plateau instead of declining , and subsequent publications have shown that a high rate of bile duct injury is an inherent problem of the laparoscopic procedure.

A late repair is preferable by most of larger studies because an early repair has a higher risk of developing biliary stricture [7-8], whereas Schmidt et al prefer an early repair because it decreases hospital stay, pain, and inconvenience.[9]. When endoscopic techniques are not effective, different surgical reconstructions are performed. The goal of surgical treatment is to allow good bile flow to the alimentary tract. To be aware of the presence of postoperative benign biliary strictures and find a best approach for treatment,we have analyzed the outcome of surgical treatment of 41 patients by classified according to Bismuth classification systems.

The present study also evaluated clinicopatholgical factors influencing hepaticojejunostomy following laparoscopic cholecystectomy. Gallstones are common in the west(6). Gallstones even though most are asymptomatic sometimes end up with life threatening complications. In united states more than 750 gallbladder removal surgeries are performed each year (7). It costs in excess than 4.5 billion

dollar, each year in the United states for hospitalization and treatment gall bladder calculi and its complications. The presence of gall bladder calculi differ between people of age,various ethinicity, and gender. It is known well that various mechanisms also influence the gall bladder removal surgeries rates since there is a minimal correlation to prevalence. The rate of gall bladder surgeries varies upon the instituiton,protocols regarding severity and surgeons attitude

.In the late 1980s several reports showed an rise in cholecystectomy rates of around 22% due to introduction of minimally invasive techniques like laproscopy and become in some places as a day care surgery. As a result, even minor changes in indications for gall bladder removal surgeries have a major impact on health care costs (11). cholecystectomy benefits most of symptomatic patients.

Title	**“A STUDY OF POST CHOLEYCYSTECTOMY ARY LEAKAGE AND ITS MANAGEMENT”**
Aims and Objectives	The aim of this study is to know the outcome of post cholecystectomy biliary leaks and its management
Study Centre	Institute of General Surgery, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai
Durationof Study	September 2017 to September 2019
Study Design	Observational study (Prospective and Retrospective)
Sample Size	100
Inclusion Criteria	All cases admitted to surgical wards with biliary leakage after cholecystectomy above 12 years of age.
Exclusion criteria	Patient undergoing cholecystectomy as a part of some primary operation like Whipple’s operation, biliary-enteric anastomosis are excluded from this study.
Materials & methods	Pre-operative workup with ultrasound abdomen, LFT and other biochemical investigations were done and diagnosis was established. The outcome of surgery and its most common post-operative complication - biliary leak was studied with following parameters: Post-operative diagnosis, Surgery - Lap or Open, Incision, Method of approach – Fundus or Classical method, CBD exploration, Intra-operative findings, Drains used, Post-operative symptoms, Amount of collections, Removal of drains, USG – Sub hepatic collections, ERCP etc.

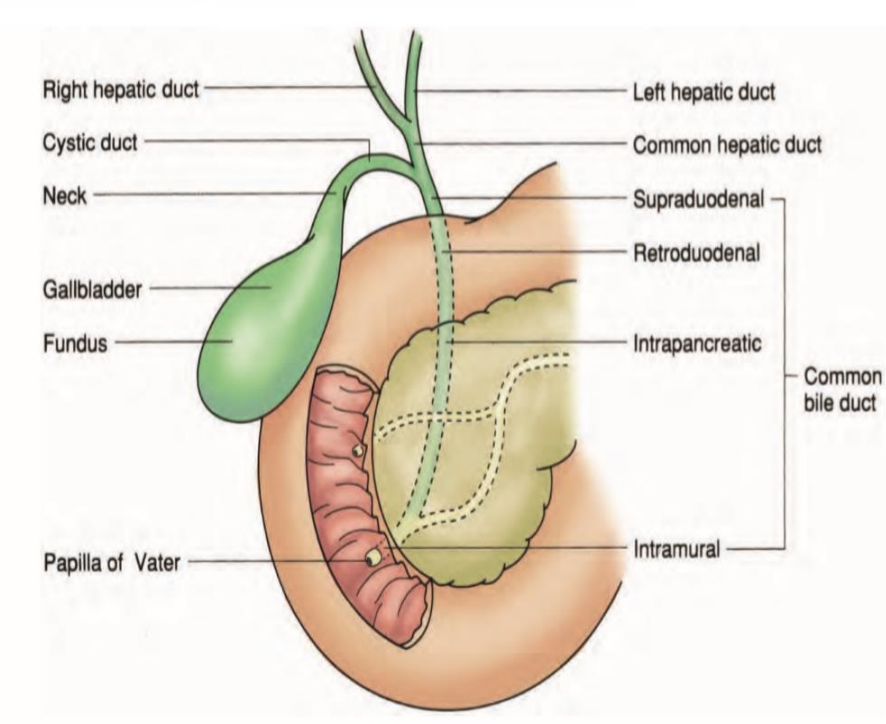
AIMS & OBJECTIVES

Laparoscopic cholecystectomy was one of the the mainstay, of treatment for gallstones since its introduction in1987.The advantages of laparoscopic cholecystectomy compared to open surgery is well documented. Some patients complain of symptoms even after complete removal of gallbladder with calculi which lasts even years after the procedure.Persistent postcholecystectomy symptoms' was more accurate description.There is wide variation in number of patients with symptoms after surgery.Many have reported relief in symptoms upto 95% (67) but there is poor description of patients who have reported relief . Moreover, the symptoms before and after the procedure was never analysed. Study aimed to evaluate the various modalities of treatment and their outcome in biliary leakage following cholecystectomy. The aim of this study is to know the outcome of post cholecystectomy biliary leaks and its management.

Anatomy

` The gallbladder(GB) is a situated under the segments IVB and V of the liver. The upper surface is related close to the gallbladder fossa in liver and lower part is covered by peritoneum. Fundus is the projecting part of gall bladder that is visible beyond liver anteriorly, and it continuous to form rest of gall bladder that is present in gallbladder fossa

. On CT, the neck of GB is visualized in more superior cuts than the body. The infundibulum is the part continuous to form the cystic duct.. The outpouching of this infundibulum forms what is called the Hartmann pouch. The triangle is surrounded by the liver above, cystic duct below and common hepatic duct medially. Cystic artery is and important component.The duodenum and lesser curvature of stomach was connected by lesser omentum and free edge of the lesser omentum is called hepatoduodenal ligament.Epiploic foramen or foramen of Winslow behind the hepatoduodenal ligament preceds to omental bursa.



The common bile duct is formed by the union of the cystic and common hepatic ducts. The common bile duct is approximately 8 cm in length, but, like the hepatic duct, it varies in length according to the point of union of the cystic duct and the common hepatic duct. The normal

diameter of the common bile duct was ranges from 4 to 9 mm. The common bile duct is considered enlarged if the duct diameter more than 10 mm. The upper third, or supraduodenal portion, of the common bile duct courses downward in the lesser mentum, anterior to the portal vein and right of the proper hepatic artery. The distal common bile duct join towards the main pancreatic duct in the head of pancreas to form a common pathway. It then continues in the duodenal walls medial aspect and opens finally into middle of second part of duodenum in the ampulla of vater in the medial aspect.

A semicircular fold of mucous membrane attaches the papilla in the upper aspect. The sphincter of oddi prevent the reflux of duodenal juices into common bile and pancreatic duct. It is composed of smooth muscle. Other sphincters include sphincter of Boyden.

The Common hepatic artery was main supply of the biliary system. It starts from celiac artery at it origin and runs over upper aspect of pancreatic body and divides terminally right and left hepatic artery.Gastrodudodenal artery is a branch of hepatic artery. Common bile duct perceives vascular supply through the common hepatic artery & its branches, gastric vessels and superior pancreaticoduodenal arteries.

The gallbladder drain into the Lymph node of Lund. It was present near cystic artery in triangle of calot major lymphatic flow is to right of hepaticoduodenalThe sentinel lymph node of gall bladder is not the node of lund.Malignancy of Gall bladder may go directly porta hepatic nodes of the hepatoduodenal ligament.Subserosal lymph drainage can receive to subcapsular lymphatic of liver. .

REVIEW OF LITERATURE

Gallstones is a extremely common condition, occurring in approximately 10% to 20% of the adult population.

Strasberg et al reported a 0.3% incidence of injuries in a literature review of open cholecystectomies. Laparoscopic cholecystectomy was considered the gold standard for the surgical treatment of gallstone disease will results in less postoperative pain, better cosmesis, shorter hospital stays, and less disability when compared with open cholecystectomy. Worldwide various studies documented a dramatic increase in bile duct injuries associated with the laparoscopic approach, ranging from 0.4% to 1.2%[10,11]. It has long been thought that the high rate of this complications was because of the so called - learning curve effect.

A large recent report showed that an initial decline in injuries was not sustained at the end of the study [12] . Hence, it is clear that the incidence of bile duct injury has stabilized above the historically accepted rate for open cholecystectomy. Laparoscopic cholecystectomy–associated bileductinjury(LC-BDI) continues to be a clinical problem with significant morbidity for patients. These preventable injuries can be devastating, increasing the morbidity, mortality, and medical cost, while decreasing the patient's quality of life. Biliary injuries will always exist, and we need to be aware of the best methods to avoid, evaluate, and treat

HISTORY

The presence of gall bladder calculi was essential before surgery in current scenario.[23]. Earlier plain x ray was only investigation to detect cholelithiasis. However, for this calcified stones were required and its seen not more than 15 % of calculus .Oral cholecystography introduced in 1924 by graham & Cole (24). This became the "Gold standard" until 1970s when ultrasonography (USG) replaced it. Non-invasiveness, ability to detect all kinds of stones and possibility to examine other organs in the abdomen are advantages of Ultrasonography. Except 6 hours of fasting are needed ,Hence suitable for emergency situations. In 1891, Calot described a triangular anatomic region formed by the common hepatic duct medially, the cystic duct laterally, and the cystic artery superiorly.6 Calot's triangle is considered by most to comprise the triangular area with an upper boundary formed by the inferior margin of the right lobe of the liver, rather than the cystic artery .. Thus, an understanding of biliary anatomy must include these adjacent organs as well as their embryology. Similiar anomalies of the biliary tract and the associated vasculature are common and result from arrested or abnormal development during embryonic growth. Biliary physiology also is closely associated with the liver where bile was formed with the pancreas as the sphincter of Oddi regulates the flow of both bile and pancreatic juice.

EPIDEMIOLOGY

The prevalence among adults is 20% for women and 10-15% for men in Europe and North America (Caucasians). Age, gender and ethnicity are the most important factors affecting prevalence (2).

India was included along with countries with a low incidence of gallstones from many texts and journals published in the west. The prevalence of gallstone disease was different parts of India. In 1966 an epidemiological study in Indian railway employees done by malhotra shows that compared to South Indian employees, north Indians had seven times higher prevalence of gallstones.

A very high and increasing prevalence was reported in epidemiological studies done in northern states such as jharkand (7-11). Khuroo from Kashmir reported a prevalence of 6.12% (male 3% and females 9.9%) the prevalence increasing progressively to reach a peak in the sixty's(10).Multiparous women have a much higher prevalence than nulliparous (10). Nil relation were found with diet, ,socioeconomic status or obesity(8)

With high prevalence of gallstones, high number of gall bladder carcinoma was also observed (11). The pathogenesis of gallbladder cancer albeit only in a small number of patients with gallstone disease

was not well understood. The duration of cholelithiasis and A high concentration of glucourso deoxycholate were postulated. However the incidence of malignancy in gallbladder with calculi over years remains a rare event (12) as proved by a recent prospective study. Migrants from India to the other countries have a higher incidence of mortality from gallbladder malignancy as compared to native populations of the areas (13).

Sharma and associates from BHU said there was an about 84 times higher risk of developing gallbladder carcinoma in patients who were bile culture positive for typhoid bacilli in their study of 390 patients (15). The role of prophylactic cholecystectomy in patients (with gallstones) from high risk areas for gallbladder cancer was not understood.but should be individualized (13). factors like age, geography, race, size of stone help to find suitable cases for prophylactic cholecystectomy.

PATHOGENESIS

The size of gall bladder calculi may vary from millimeters to as large a few centimeters . more than 15% of people have stones which have migrated to common bile duct (30).

Gallstones are classified as :

1. Cholestrol stones
2. Pigment stones-Either brown or black

3. Mixed stones

Cholesterol was the most common type of gallstone, which comprises 55-97 % of the dried component of the calculi. Other constituents may include fatty acids, triglycerides, proteins, polysaccharides, as well as calcium bilirubinate, calcium bicarbonate and calcium carbonate.

The major components of bile are :

1. bile acids (salts),
2. phospholipids
3. cholesterol.

Bile salts organised from cholesterol are the two primary bile acids – cholic and chenodeoxycholic acid. As Cholesterol was only slightly soluble in aqueous solutions, a bile salt micelle makes it soluble along with phospholipids and lecithin.

Bile salts are secreted to the duodenum and reabsorbed through the ileum Enterohepatic circulation of bile acids along with adequate bile acids in the liver from cholesterol was sufficient to keep the cholesterol in solution.

Mechanisms in the genesis of cholesterol gallstones:

1. Supersaturation of bile with cholesterol.

2. Nucleation of cholesterol monohydrate with subsequent crystallization and stone growth
3. Gallbladder stasis – delayed emptying
4. Decreased Enterohepatic circulation –increased loss of bile acids (eg: ileal resection / Crohns disease).

Cholesterol gallstones changes in the composition of bile. Increase in the composition of a normal biliarycomponent bilirubin or cholesterol the solubility, a decrease in the solubilizing component or both leads to gallstones. An added pathogenetic factor will be dysmotility of the gallbladder. All these produce a nidus ,an insoluble becomes sequestrated and aggregate to form a calculus. Cholesterol stones one of the most common and contribute to more than 80% of the stones. Cholesterol with cholesterol monohydrate crystals agglutinated by a mucin glycoprotein matrix, unconjugated bilirubin and small amounts of calcium phosphate are components of these stones.

Gallbladder sludge is probably creation of gallstones. Suspension of cholesterol monohydrate crystals, calcium carbonate, calcium bilirubinate and calcium phosphate form the sludge

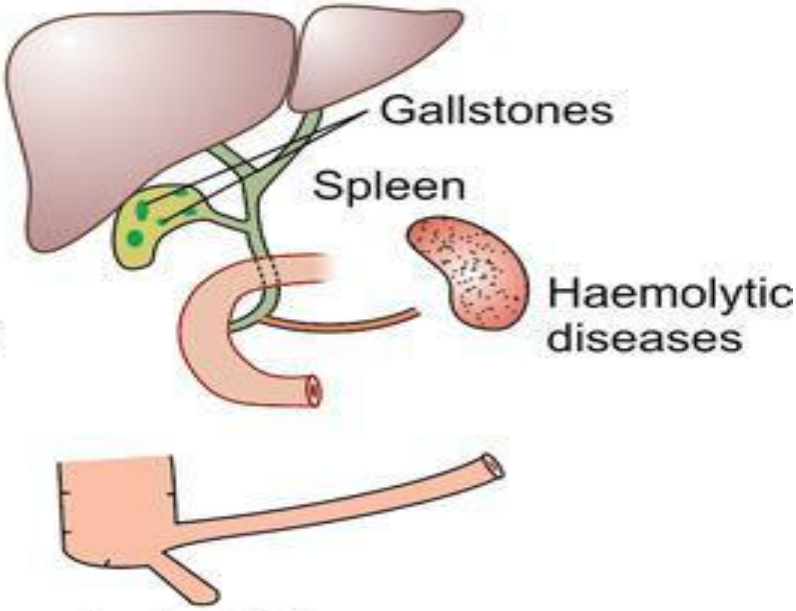
It may either go on and form stones or just disappear. The Cholesterol in the body is acquired both from diet and liver synthesis .Mucin secreted by the gallbladder epithelium is an important factor as Supersaturated bile alone does not lead to gallstones. Though crystals develop in supersaturated bile need for a pronucleating agent to form gallstone which is the mucin.

Brown pigment stones was formed by stasis and infection in the biliary system. Black pigment stones were common in patients with hemolytic diseases (hereditary spherocytosis, sickle cell anemia, and Thalassaemia) and liver cirrhosis (25-29). Other constituents may include fatty acids, triglycerides, proteins, polysaccharides, as well as calcium bilirubinate, calcium bicarbonate and calcium carbonate.

RISK FACTORS

Altered GB function
- Stasis
- Poor emptying
- Poor absorption
- Infection

Supersaturated bile
- Female
- Fertile
- Fat
- Forty
- High calorie



Altered enterohepatic circulation
- Ileal resection
- Ileal diseases
- Altered bowel transit time
- Altered bowel flora
- Cholestyramine
- Deoxycholate

Hormones

Jorgensen et al proved that estrogen therapy and parity (43) was an important factor for variation in presence of gallstones between males and female. Scragg et al found OCP as dependent factor due to age 45, the greatest risk in females less than 30 years of age. The enhancing effect of hormonal therapy on gall bladder calculi development found in a large study involving 22000 people (46). The chance of surgery was almost double in the people with estrogen intake as with the those who are placebo.

Parity

Even though some studies one europe could not verify this finding (49),(50), many say than higher risk with number of pregnancy and child-bearing,(42-44),(47),(48)

BMI

This relation is usually more in women than men. Many studies have proved than this is an important factor (7, 42, 43,51-54) .Obesity increases cholesterol secretion in bile, which is an major factor in gall bladder calculi formation. The prophylactic removal of gall bladder after bariatric surgery such as gastric bypass since there is an high chance gall

bladder calculi formation because to sudden weight loss 53,as around 45% of the patients form calculi.

Genetic

The connection between gall bladder calculi occurrence in a family is shown in many studies (5, 55, 56,). In India, People in north are more prone for gall bladder calculi. This may also be an reason for the large occurrence among Indians (48). A report done in south America proved that cholesterol lithogenic genes are present in native population of chile and people of spanish origin in that country. But many factors were represented high in these patients. For example, the women/men ratio is more .The frequency of obesity and parity were also similar.

Age

The incidence of gallstones increases with age across all ethnic groups, becoming 4 to 10 times more likely in individuals older than 40 wth a very low rate among infants and children .Uncommon <20(exception: Mexican American girls).

Gender

Female/male ratio highest in the youngest; narrows to2:1 after age 50 Female gender is a risk factor for developing gallstones, surpassing males in the incidence of gallstones and the chance of having surgery by 2 : 1 or 3 : 1 in most studies

Socioecomic factors

The Italian MICOL report(51) showed that male with higher income and education had higher rates of gall bladder calculi formation. However in a english report ,lower strata people had more chance of gall bladder calculi formation. An North american study showed that women of mexicans emigrating to USA had higher incidence of gall bladder calculi than white women. However , after controlling for factors the presence among females inversely (47).

Family history

Higher risk in first-degree relatives of gallstone patients

Alcohol

The Consumption of alcohol was also related inverse to the risk of gall bladder calculi (78). Lesser presence of gall bladder calculi (54, 60, 76, 77) with symptoms (48) and no symptoms (75) are related with intake of alcohol. But, the relation was absent when intake was not more than 2

days in a week (79). A European report said there s no relation of gall bladder calculi with alcohol (80).

Tobacco

There are varying studies regarding as smoking is factor or is it not. The reduced production of prostaglandin and more production of mucus are the reason in people who smoke (59) is considered as the main reason. Stampfer et al. said that in females who consume cigarettes in large amounts (>30 cigarettes/24 hrs) it is an independent risk factor (60). In a large english study, cigarettes consumption was considered as an important factor for formation gall bladder calculi (58).

In an Australian study, they said that even early smokers had an increase in risk of developing gall bladder calculi with symptoms.

Metabolic disorders

Diabetes mellitus, overweight and increased cholesterol are with high risk for gall bladder calculi formation which shows that gall bladder calculi is component of metabolic syndrome (62, 63) The increased secretion of cholesterol in bile which oversaturated it is an reason.Insulin resistance is cause for such secretion. Large number of reports have proved this theory (51, 64-67), However it is still controversial since many other reports had not found this conclusion (63, 68, 69).

Diet

Diabetic patients are more prone for infections and gall stones

Spinal Cord Injury

Abnormal gallbladder motility may be a factor

Inflammatory bowel disorder-Chron's disease

This cause excreted of cholesterol in a bile which is oversaturated leading to increased gallstone formation. Another one also showed a connection between gall bladder calculi and Chron's disease in relation to site, number of affected segments(73)

However the rate of gall bladder removal surgeries have not increased after removal of affected ileum which is surprising (74). Hence, gall bladder removal in asymptomatic cases is not justified.

Non steroidal anti-inflammatory agents

21 years ago in a study it was concluded that gall bladder calculi formation could be reduced by the intake of non steroidal anti-inflammatory agents (70). This however could not be proved in a large study with intake aminosalicylic acid of more than 1g/24 hrs (71).

Exercise

The mechanism of exercise in reducing the formation of gall bladder calculi is unknown. The effect of physical activity in reducing time for movement of food in colon is associated with dehdroxylation of bile salts and an higher contraction of gallbladder (81). Many studies found no such relation. (82-84). Leitzman et al,found no relation between gall bladder calculi in males (80).In a study > 55 000 females that physical activity reduces the incidence of gall bladder calculi surgeries (86).

Genetics

Excess cholesterol load in the smooth muscle of the gallbladder can affect signaling via the cholecystokinin A receptor and contribute to the development of gallstones by inducing bile stasis. Intestinal cholesterol/bile salt absorption is tightly controlled, and genes responsible for cholesterol absorption (NPC1L1, Niemann-Pick C1–like protein) or cholesterol efflux (ABCG5/G8) have been implicated in the development of gallstones in certain populations because of intestinal bile salt loss. Single-gene mutations resulting in hemolysis increase the incidence of black stones because of excess of unconjugated bile acids forming polymerized calcium bilirubinate stones.

SYMPTOMS

We recognize four types of gallbladder disease:

1. Asymptomatic gallstone
2. Symptomatic gallstone
3. Pain abdomen from another etiology such as peptic ulcer, with asymptomatic gallstones
4. Cholecystitis with no gallstones

Asymptomatic gallstone disease

Only 20 % patients actually develop symptoms after follow up over a period of twenty years. Gall bladder calculi which do not cause symptoms do not require surgery except in certain situations.without it appearing before. Other complications such CBD obstruction due to calculi or malignancy are not seen frequently. Ransohof et al. saw no reason for gall bladder removal in patients with no symptomatic stones (78). GREPCO-group in Europe (77) found that cholecystectomies caused no difference in minor dyspeptic symptoms. Only In less than 2 % per year of patients with gallstones with no symptoms developed serious complications. In a study of 120 people with no symptoms due to gall bladder calculi followed up for 11 years and the chance of forming complication was no more than 3%. Biliary colic is an important symptoms and patients rarely develop complications.

Symptomatic gall stones

The chance of symptomatic cure after surgery will be very low for patients with preoperative dyspeptic symptoms.however,72% of patients will feel improved after the procedure.This suggests that gall bladder calculi could have caused those dyspeptic symptoms(7-9).If the pain is of biliary type then 95% chance of cure were reported after follow up of upto a year(10,11).

Borly et al (12) studied whether factors present before surgery could be used to predict postop symptomatic over a period of 24 months. Of the Only 78 patients completed the study out of 100,and of them 70 were females. 20 patients continued to complain of pain abdomen after the surgery and were found to have by high dyspepsia score before surgery,

Lorusso et al (13) studied whether poor outcome after surgery in patients with gallstones without any complication could be due to psychological causes and symptoms of dyspepsia . 52 (42 women, 10 men) patients were studied 2 weeks before the surgery and a year after surgery.

Pediatric gallstones

The distribution of gallstone types in children differs from the adult population, with cholesterol stones being the most common type of stone in adults and black pigment stones being the most common type in children.

Black pigment stones make up 48% of gallstones in children. They are formed when bile becomes supersaturated with calcium bilirubinate, the calcium salt of unconjugated bilirubin. Black pigment stones are commonly formed in hemolytic disorders and can also develop with parenteral nutrition.

Calcium carbonate stones, which are rare in adults, are more common in children, accounting for 24% of gallstones in children .

Cholesterol stones are formed from cholesterol supersaturation of bile and are composed of 70-100% cholesterol with an admixture of protein, bilirubin, and carbonate. These account for most gallstones in adults but make up only about 21% of stones in children.

Brown pigment stones are rare, accounting for only 3% of gallstones in children, and form in the presence of biliary stasis and bacterial infection. They are composed of calcium bilirubinate and the calcium salts of fatty acids and occur more often in the bile ducts than in the gallbladder.

The remaining portion of gallstones in children consists of protein-dominant stones, which make up about 5% of gallstones in these patients.

Microliths are gallstones smaller than 3 mm; can form within the intrahepatic and extrahepatic biliary tree; may lead to biliary colic, cholecystitis, and pancreatitis; can persist after cholecystectomy; and are difficult to diagnose as they are often missed on ultrasonography. Biliary sludge is made up of precipitates of cholesterol monohydrate crystals, calcium bilirubinate, calcium phosphate, calcium carbonate, and calcium salts of fatty acids, which are embedded in biliary mucin to form sludge.

Treatment for simple cholelithiasis is symptomatic treatment. Surgical removal of asymptomatic gallstones is currently not standard practice.

INVESTIGATIONS

Liver function test

It has been shown in small studies that liver enzymes are often deranged after LC because of pneumoperitoneum and raised intra-abdominal pressure. Biekel et al showed that only 41(3.9%) of 1034 patients had deranged LFT, in whom it was because of choledocholithiasis. Hence any derangement of LFT after LC should be carefully followed up with a high index of suspicion for choledocholithasis or a bile duct injury.

Ultrasound

Ultrasound is the best first-line imaging for intra-abdominal fluid collecctions . It is also important to image a potential biloma with ultrasound because , it is also used to target the bilioma for percutaneous drainage (PCD) .

Pitfalls : It is observer –dependent investigation and may miss small bilomas and abscesses.

Nuclear scan:

HIDA is valuable in assessing incomplete strictures, and isolated sectoral hepatic duct strictures [45].Although the anatomical delineation is not very clear, it quite accurate in demonstrating bile leaks. Newer agents mebrofenin is excreated even with very high serum bilirubin unlike HIDA, DISIDA which not excreated in jaundiced patients .

Pitfalls :

A block in the lower bile duct caused by a stone may be interpreted as a complete transection .A high output biliary fistula may lead to the tracer draining out mainly throught the fistula with very little gut activity, even if there is biliary continuity.

COMPUTED TOMOGRAPHY:

CT is the best initial study which helps to localize the level of dutal obstruction also identifies fluid collections or ascites, vascular damage, and lobar atrophy.

Cholangiography

Delineation of the proximal biliary tree in the form of complete cholanigogram is extremely important for the successful repair of a stricture. PTC (percutaneous transhepatic cholangiography) provides information in selecting patients with for appropriate reconstructions. After the advent of MRCP, its role has lessened, but it still remains an important tool in refractory cholangitis .It allows serial ballon dilations and upsizing of stents that can start from 8F upto 22F.

ERCP

ERCP is occasionally valuable in the precise diagnosis of complete proximal bile duct and is appropriate for patients with suspicion of papillary stenosis . ERCP also plays a role in partial or lateral injury to the common duct [46]. Thus the role of ERC as a cholangiogram is limited only to low and incomplete biliary strictures.

MRCP

Magnetic resonance cholangiography is the investigation of choice in patient with biliary strictures. Magnetic resonance cholangiopancreatography is a useful tool in bile duct injuries [47]. It shows the exact anatomy of the stricture, the entire biliary system and specifically helps in delineating first and second order bile duct strictures which a standard PTBD gram may not be able to demonstrate unless multiple punctures are made. Associated hepatolithasis may be better shown by MRCP. [48-50]. The interpretation of the images in the MRCP may be hampered by the presence of biloma. Also collapsed bile ducts in presence of an on going external biliary fistula. [51]. Computed tomographicangiorgraphy (CTA) is often quick was to assess for a suspected biloma, hepatic abscess, and the condition of the hepatic vessels. If a pseudoaneurysm is observed , it should be tackled by conventional angiography and angioembolization before the biliary stricture is repaired.

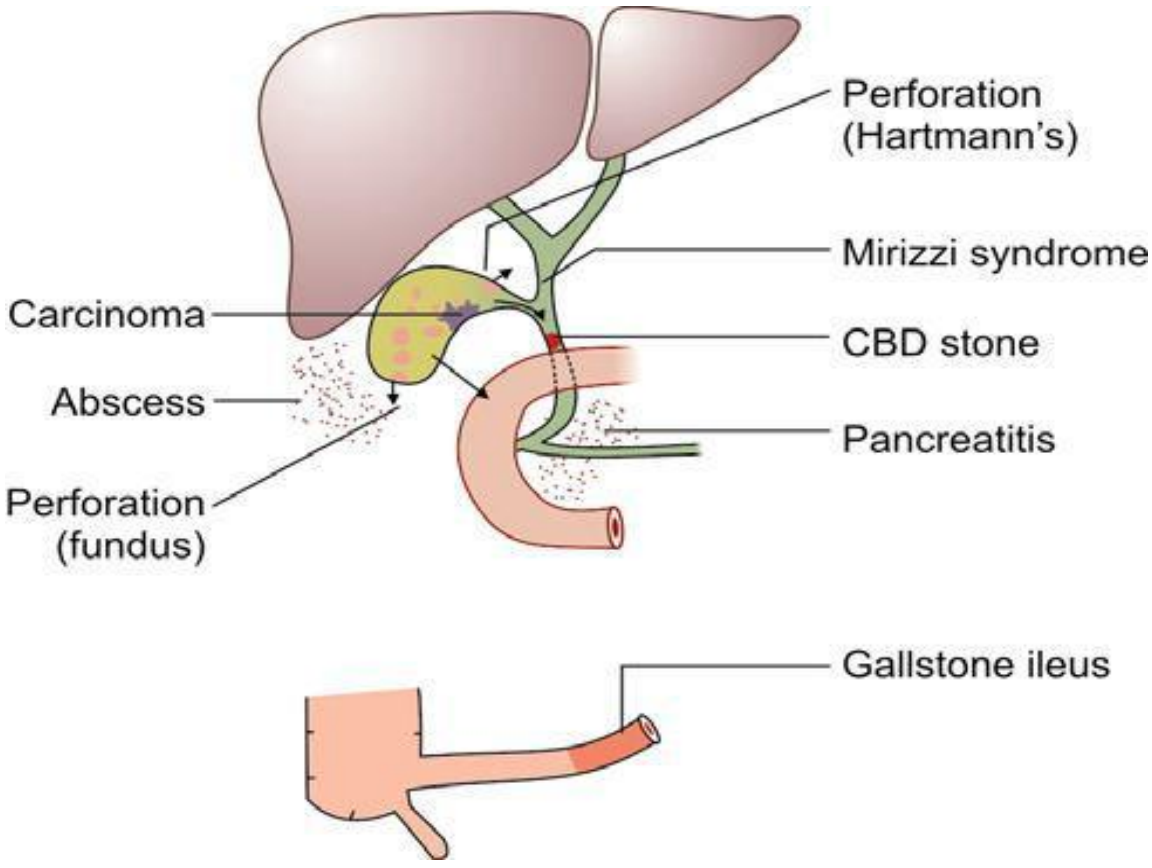
PREOPERATIVE PREPARATION:

Operative repair of bile duct injuries needs more careful precise assessment for bile duct injuries which are recognized at the time of initial cholecystectomy. Cholangitis is a frequent occurrence in patients with bile duct strictures which is treated with appropriate antibiotics

according to bile culture sensitivity report . Anemia should be correctedcoagulation defects, with prolongation of the prothrombin time. treated with vitamin K or fresh frozen plasma. Malnurished patients are feed with enteral feedings through nasogastric or nasojejunal catheter

Parenteral nutrition may be needed who are intolerant to enteral feeding .The preoperative management of complications must be addressed before biliary reconstruction can be considered. If cholangitis occurs without any response to antibiotics needs immediate biliary drainage

COMPLICATIONS



Acute cholecystitis

An acute inflammation occurs when there is occlusion to bile flow from gall bladder for a long time. The patients suffers from toxic symptoms like elevated temperature and localized right hypochondrial tenderness.Blood investigations show an elevated total count. The presence of gall bladder pathology should be confirmed using USG or CT after clinical diagnosis. Imaging shows gallbladder with edema and thickening suggestive of acute inflammation. Mild jaundice may be seen due to edema. Marisa's syndrome or CBD calculi should be considered if jaundice becomes severe (79) . Surgery should be considered immediately in such situations without delay of more than six hours (80).

Jaundice

Gallbladder calculi can cause occlusion to flow of bile if they migrate into CBD and cause obstruction. Such cases will not have palpable gall bladder according to Courvoisier's law. Rarely, an stone in the Hartman´s pouch may compress the common bile duct. In the above mentioned cases, the patient has jaundice.Cholangitis presence is variable. Such cholangitis has a mortality rate pretty high unless the biliary drainage is done. Age, neurological disease and peripapillary diverticula are all identified as risk factors in these scenarios (81).

Chronic cholecystitis

It is a shrunken, chronically inflamed gall bladder which is no distending and non functioning. It has rokitansky-Aschoff sinuses which are nothing but mucosal clefts

Mucocele

Collection of sterile mucous after all bile is absorbed forms mucocele.It occurs due to obstruction of gall bladder in Hartman's pouch.

Empyema

It is a type of acute cholecystitis where the gall bladder is filled with pus.More common in diabetics and immunosupressed individuals. Preexisting Mucocele could turn into empyema.Patient will be toxic with fever and localized tenderness. Requires urgent treatment.

Acute pancreatitis

Calculi of smaller sizes or microlithiasis, are major cause of acute gallstone pancreatitis. Majority of patients (around 90%) have a mild form that is treated with IV fluids. Not more than 5 days is required for recovery. The rest 10% develop the necrotising pancreatitis which is more aggressive and has chance of translocation of microorganisms from gut to which was before an no septic inflammation . Such patients require intensive care. Unfortunately, no causative treatment is available for the already developed pancreatitis .

If there is cholangitis due to occlusion by calculi, Emergency ERCP with sphincterotomy must be performed (82). If not, prophylactic cholecystectomy should take place as soon as possible after the acute episode (83).

Gallstone ileus

A fistulous connection can develop between gallbladder and small intestine or stomach.. If its big, then calculi can occlude the small intestine causing obstruction. This causes no more than 4% of all causes with small intestine obstruction (81).

This incidence of gallstone illeus is more in people >60 years of age and more seen in females.(82).It's a diagnosis that is hard to arrive. The patient presents with symptoms of small intestine obstruction. Sometimes air in the biliary tract is seen along with signs of intestinal obstruction and rarely calculi that is calcified in the small intestine. Very rarely, a stone penetrates into the stomach, where it is trapped, causing the so called Bouveret´s syndrome(154, 155),with signs of intermittent GOO.

Gallstones in the gallbladder

↓

Cholecystitis

↓

Suppuration and adhesion over the duodenal wall

↓

Communication of gallbladder into the duodenum

↓

Gallstones pass into the duodenum forms a *bolus (‘Rolling stone gather mass’)*

↓

Blocks narrow part in the ileum.

↓

Gallstone ileus

Gallbladder carcinoma

Gallbladder calculi is an important risk factor in development of malignancy (81). Cholangiocarcinoma has a high mortality in later stages (82). Therefore, some suggest prophylactic removal of asymptomatic gallbladder with calculi. But, one few people with calculi (0.25 %)

develop this carcinoma. Hence, there is a general consensus not to treat asymptomatic gallstones, since the mortality associated with surgery is at least at the same level as that of gallbladder carcinoma (83).

INDICATIONS FOR CHOLECYSTECTOMY

The risk of developing complications to gallstone disease must be included during the evaluation of cholecystectomy (55), chance of complication and, obviously, the relief from preoperative symptoms(85), the cost for society must also be taken into consideration. Rates of gall bladder removal surgeries differs among various countries

In screening studies there is an relation between biliary colic (upper right abdominal quadrant pain) and occurrence of gallbladder calculi (86), making biliary colic the important indicator for gallbladder calculi (87). But, various studies denote that single event of pain may not be succeeded by more events in a reasonably span of time. So waitful watching is required in cases with just single episodes

Therefore guidelines suggest surgery to patients with multiple episodes of symptoms or a complication resulting from calculi (86). When the stones are symptomatic, some even recommend operation without delay in order to minimize costs and complications.

Prophylactic Cholecystectomy Considered for

High risk for gallbladder cancer Gallstones larger than 3 cm in diameter

Porcelain gallbladder (calcification in the wall)

Gallbladder polyps larger than 12 mm Carriers of Salmonella typhi

Working for prolonged periods in remote parts of the world with poor medical facilities

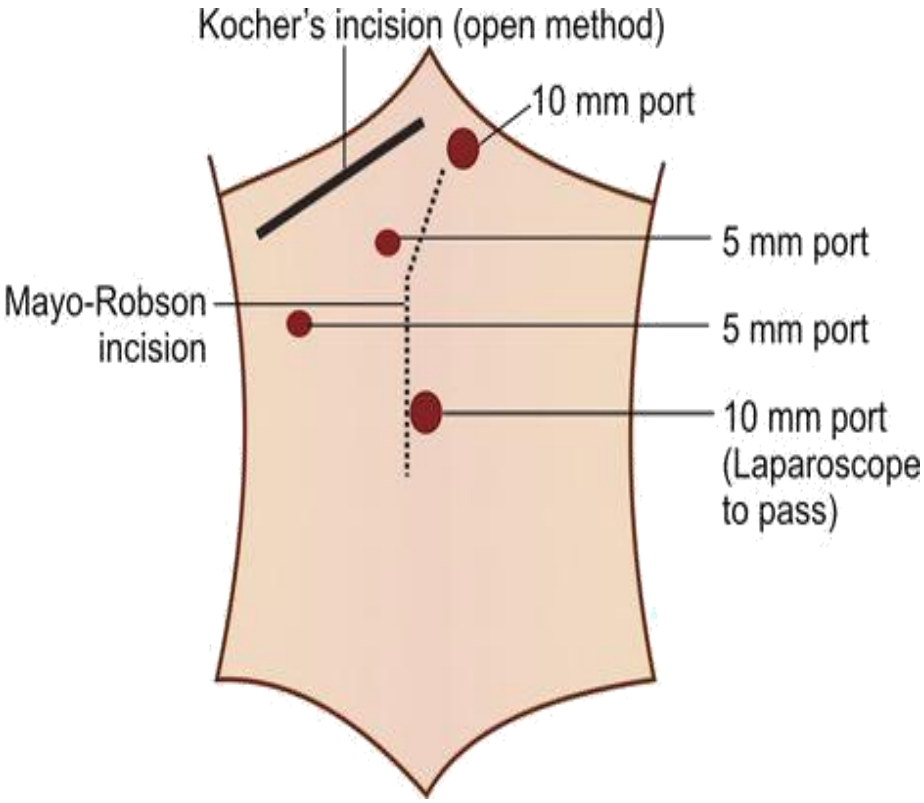
TREATMENT

OPTION	STONE CLEARENCE	COMMENT
Laparoscopic Cholecystectomy	100 %	Standard of care, Invasive, General anesthesia
ERCP/ Sphincterotomy	70 %	In selected cases. Advanced age & pregnancy
ESWL	70%	Rarely helpful
Oral bile acid dissolution		Not ideal in symptomatic cases

Laparoscopic Cholecystectomy

Under general anesthesia and patient is placed in supine position.4 trocars are inserted into the peritoneum. CO2 is used to insufflate the abdomen by either closed method (Verees needle) or open method. Incision then made near umbilicus for ten millimeter trocar.The camera is passed through the umbilical port.. 3 more ports (epigastrium, mid-clavicular line, and flank) are inserted under vision. The operation is performed and visualized through a monitor with magnified images.

The flank or lateral port is used for an atraumatic grasper to grasp the fundus and retract it upward. The epigastric port is used for the dissection by a dissector, hook or scissors The mid clavicular port is used to hold the infundibulum with an atraumatic grasper, and pull towards the right lower quadrant.The cystic duct and artery are identified and separated after hepatoduodenal ligament dissection. Both of them are then clipped and cut. The GB is then separated off the gallbladder fossa. The GB is then brought out through the umbilical incision. (18)



Single incision laparoscopic Surgery (SILS) in cholecystectomy

SILS is an advanced technique in which surgeon uses an single umbilical port for access hence it is also called as one port umbilical surgery (OPUS).

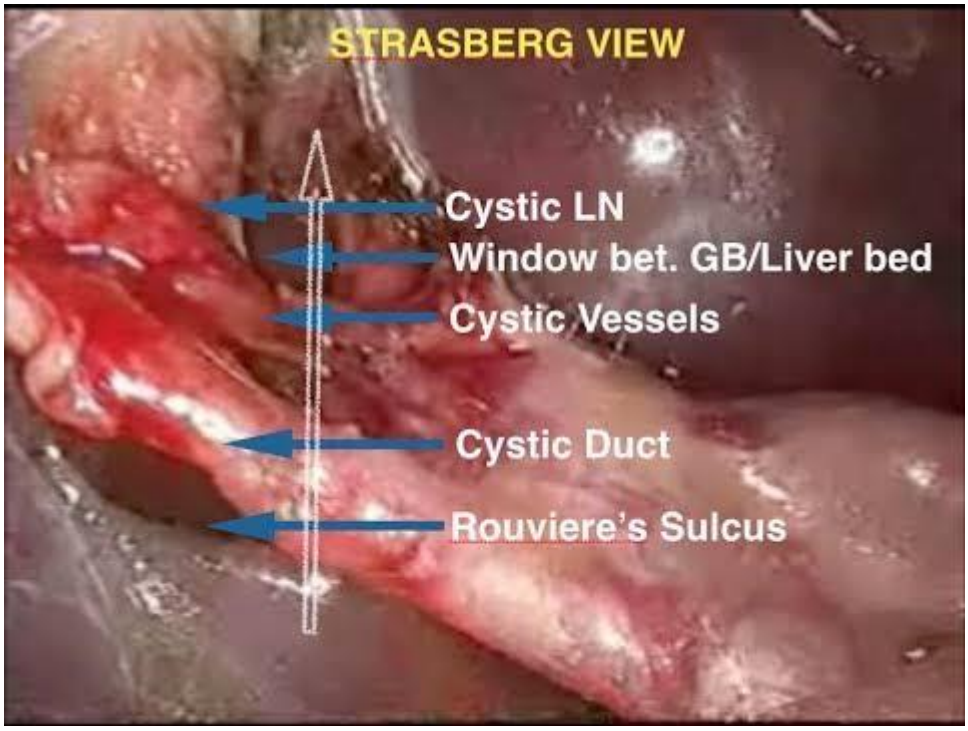
It is done using general anesthesia and requires advanced umbilical trocars which is large but should also allow harmonic scalpel. working instruments, flexible scope, rotatory instruments, handles, 2.4 cm umbilical incision made vertically and dissection is done by open method to enter peritoneal cavity. Port through which a 10 mm telescope is passed and 2 instruments of 5 mm for work is used. Instruments are angled and flexible to meet the ergonomic principles to certain extent.

Dissection of gallbladder is done in similar fashion like four port technique. Specimen is easily retrieved through umbilical port as it is wide enough. If difficulty arises any time one can add additional ports as required.

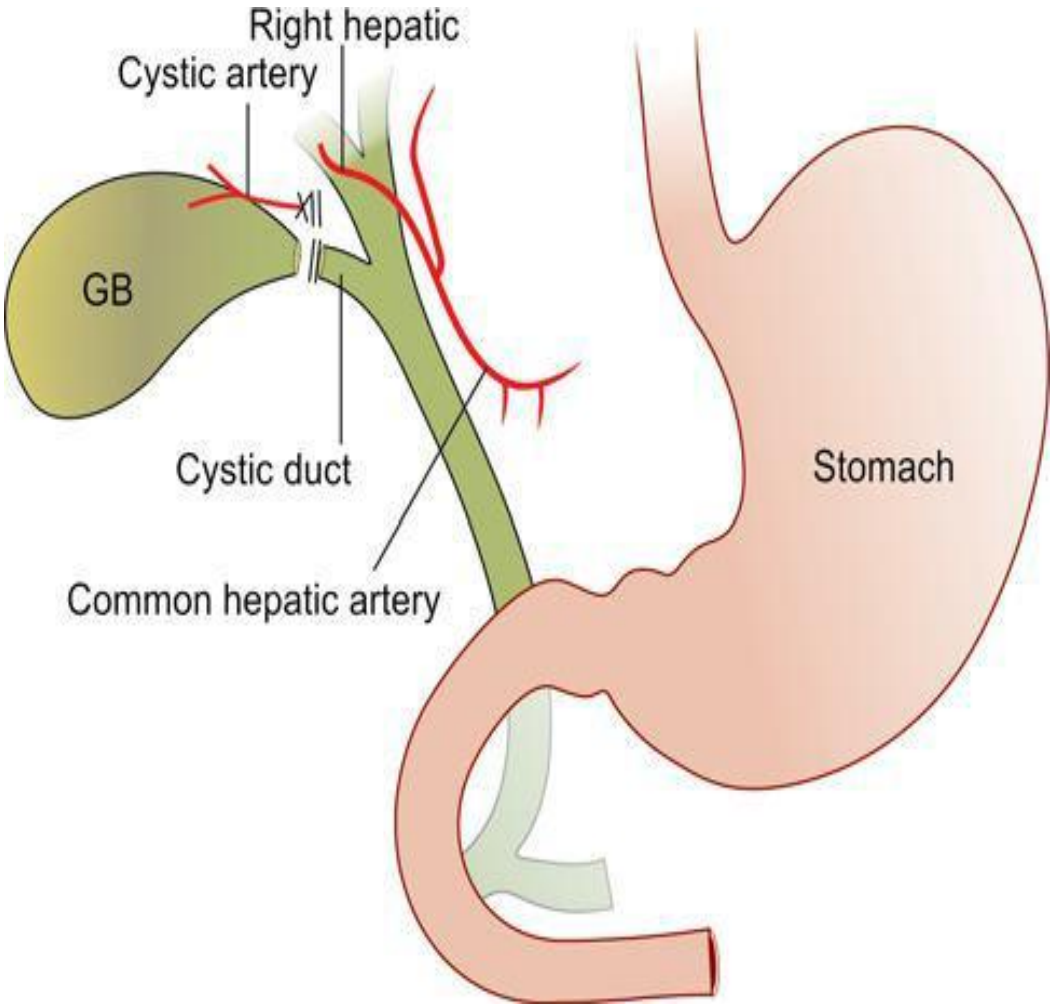
Open Cholecystectomy

The patient under a general anesthesia and is supine position and draped under aseptic precautions. Two types of incision cane be used: Either an subcostal or kocher's incision or upper midline incision. The dissection of GB is similar as done in laparoscopy.

Advantages of laparoscopy is there is less postoperative pain, Early recovery and early discharge. But there is higher chance of surrounding biliary system injuries as compared to open procedures (26)



COMPLICATIONS OF CHOLECYSTECTOMY



As with any invasive surgery, cholecystectomy carries certain risks. Operation-specific complications include surgical site infection, bile duct and bowel injury, Bile leak, vascular injury and vascular-biliary injuries. Medical complications include myocardial infarction, pneumonia, urinary tract infection and venous thromboembolism. While rare, death may result from any of these or other rarer complications.

The frequency of surgical site infection ranges from 1% to 10% and the risk is related to surgical approach (laparoscopic or open cholecystectomy) and the degree of contamination (e.g. gangrenous cholecystitis, leakage of infected bile, occurrence of a bile duct or bowel injury) (37) More specifically, when characterized according to the Center for Disease Control's levels of 14 surgical site infection,

Bile leaks and bile duct injuries represent a spectrum of injury to the biliary tract. Strasberg et al. proposed a classification system most applicable to the laparoscopic era that classifies injuries based on the length, circumference and level of the injury involved and whether the main duct (common hepatic and common bile duct) versus an accessory or the cystic duct are injured (39). With respect to associated morbidity and impact on quality of life, injury to the biliary tract can be considered in two broad categories : bile leaks and bile duct injuries requiring operative intervention. Leaks may result from injury to a side branch of the biliary tree, the cystic duct stump or a non-circumferential injury to

the main ductal system. These may be managed with endoscopic cholangiopancreatography and stent placement, possibly in association with percutaneous drainage (40). Major bile duct injuries require operative repair or reconstruction of the biliary tract and are therefore associated with the greatest morbidity (40). In fact bile duct injuries are associated with reduced long term survival and are a major cause of litigation against general surgeons (40-43). Leaks occur in approximately 1-3% of laparoscopic cholecystectomies whereas injuries complicate only 0.3-0.5% of cholecystectomies (41,44-48). In addition, vasculo- biliary injuries have recently received greater attention and refer to an extreme case of major bile duct injury that occurs in conjunction with injury to a hepatic artery and/or portal vein (49,50). This devastating injury accounts for only 2% of major bile duct injuries (51).The frequency of bowel injuries is not well characterized but results from dissection of the colon, small bowel or duodenum adhered to an inflamed gallbladder or from inadvertent cautery burn. Primary repair is generally possible if recognized early; otherwise, patients will present later with peritonitis necessitating a return visit to the operating room. A small proportion of cholecystectomies for symptomatic gallstones lead to medical complications. Based on data from the National Surgery Quality Improvement Program (NSQIP) of the American College of Surgeons, a procedure-specific registry, medical complications such as myocardial

infarction, pneumonia, urinary tract infection and venous thromboembolism respectively occur in 0.2%-1%, 0.4%-4%, 0.7%-2% and 0.2%-1% patients respectively (37). The frequency of such complications might reasonably be expected to be higher in patients operated on for acute cholecystitis.

POSTCHOLECYSTECTOMY SYNDROME

Post cholecystectomy syndrome (PCS) is nothing but failure of relief of preoperative symptoms after cholecystectomy .These symptoms can just be continuation of old or newly started ones. PCS also be due the absence of gallbladder after surgical removal (eg, gastritis and diarrhea).

PCS reportedly occurs in around 10-15% of patients. The cause of the mild GI symptoms is due to bile. Loss of this reservoir function of the gallbladder changes flow of bile and the Enterohepatic circulation of bile. This pathogenesis however is fully not understood. Early reports on PCS was mainly on changes in anatomy that were grossly or at least microscopically seen during exploratory surgery. Improvements in technology and imaging studies have yielded an improved understanding of biliary tract disorders.

This has affected the preoperative workup of patients with suspected gallbladder disease as well as those with PCS, making functional disorders of the biliary tract (including irritable sphincter).

The most common causes are:

Anatomy	**Etiology**
Gall bladder remnant and cystic duct	Residual or reformed gallbladder Stump cholelithiasis Neuroma
Liver	Fatty infiltration of liver Hepatitis Hydrohepatosis Cirrhosis Chronic idiopathic jaundice Gilbert disease Dubin-Johnson syndrome Hepatolithiasis Sclerosing cholangitis Cyst
Biliary tract	Cholangitis Adhesions Strictures Trauma Cyst

Periampullary	Malignancy and cholangiocarcinoma Obstruction Choledocholithiasis Fistula Dilation without obstruction Hypertension or nonspecific dilation Dyskinesia Sphincter of Oddi dyskinesia, spasm, or hypertrophy Sphincter of Oddi stricture Papilloma Cancer
Pancreas	Pancreatitis Pancreatic stone Pancreatic cancer Pancreatic cysts Benign tumors
Esophagus Stomach	Aerophagia Diaphragmatic hernia Hiatal hernia Achalasia Bile gastritis Peptic ulcer disease Gastric cancer
Duodenum	Adhesions Duodenal diverticula Irritable bowel disease
Small bowel Colon	Adhesions Incisional hernia Irritable bowel disease Constipation Diarrhea Incisional hernia

	Irritable bowel disease
Vascular	intestinal angina Coronary angina
Bone	arthritis
Other	Adrenal cancer Thyrotoxicosis 20% organ other than hepatobiliary or pancreatic Foreign bodies, including gallstones and surgical clips

CLASSIFICATIONS SYSTEMS

Many classification systems have attempted to sort BDI for planning management and prognostication . None of the systems is ideal in all respects .

BISMUTH CLASSIFICATION

The classifications of bismuth was used because of the ease of management and operative risk stratifications. It is often used to describe established bile duct injury.[40]

TABLE : BISMUTH CLASSIFICATION

Type	Anatomy
1	CHD: stump (more than 2cm)
2	CHD stricture : stump more than 2cm
3	Stricture without any residual common hepatic duct with intact confluence
4	Hepatic duct confluence destruction : both right and left hepatic ducts separated
5	Involvement of aberrant right sectoral hepatic duct alone or with CHD stricture

STRASBERRG CLASSIFICATION

Strasberg proposed a comprehensive system that incorporate sbismuth's scheme and also on the basis of review of the literature on laparoscopic cholecystectomy. [41]

TABLE : STRASBERG CLASSIFICTION

TYPE	ANATOMY
Type A	Minor leaks from the cystic duct
Type B	Occlusion of a part of the biliary tree
Type C	Bile leakage from a duct not in communication with the common bile duct
Type D	Lateral injuries to the extrahepatic common bile duct
Type E	Circumferential injury to the extrahepatic bile duct ;
Type E-1	CHD Stump > 2 cm
Type E-2	CHD Stump < 2 Cms
Type E-3	No CHD Stump
Type E-4	Confluence involved with separation of right and left systems
Type E-5	Injury to an aberrant right segmental duct with or without injury to the CHD/CBD

TABLE – 6 : CLASSIFICATION OF BILE LEAK, CIRCUMFERENCE INVOLVED AND DUCT INJURED FOR BILE DUCT INJURY

Class	Description	Types	Diagnosed by
B	Bile leak	By- Yes (Open Duct) Bn–No (ligated / clipped duct)	Bile in drain/ on aspiration, isotope scan, cholagniography (ERC, MRC)
C	Circumstances involved	Cf-full circumstances (transaction or excision)	Isotope scane, cholangiography (ERC, MRC), operative findings.
D	Duct injured	DS-significant duct (CBD, CHD, RHD, right sectoral or segmental duct) Di-insignificant duct (cystic duct, sub-segmental duct, sub-versical duct)	Cholangiography (ERC, MRC, operative findings.

TECHNICAL APPROACHES TO BILIARY REPAIR:

END-TO-END BILE DUCT REPAIR.

The technique used for reconstruction was excision of the stricture with end-to-end anastomosis [60-62]. For a transected bile duct, primary suture repair or end-to-end primary repair is not done. Patients who underwent end to end anastomosis had high failure rate.Thus end-to-end repair has limited role in benign biliary strictures management.

BILIO-ENTERIC REPAIR

Reconstruction and repair methods include also biliary bypass with Roux-en-Y hepaticojejunostomy or rarely hepaticoduodenostomy, closure over T-tube, removal of the obstructing clip, and endoscopic or percutaneous transhepatic dilation and stenting. Successful bile duct enteric reconstruction is dependent on several factors:

1. Adequate preoperative assessment of biliary anatomy
2. Exposure of proximal, healthy bile ducts with adequate blood supply
3. The repair must include all injured and strictured ducts to ensure adequate drainage of the entire liver, and control of bile leakage.
4. Use of a healthy segment of intestine that can be brought to the anastomosis without tension (most often a Roux-en-Y jejunal limb)
5. Tension-free biliary mucosa-to-bowel mucosa anastomosis

TABLE – 7 :TYPE OF INJURY AND TECHNICAL OF REPAIR

Type (Starsberg) of Injury	Technique of repair
TYPE A	Do not require reconstruction
TYPE B	Usually not discovered intraoperatively
TYPE C	Roux-en-Y hapatico-jejunostomy Ligation / clipping of duct (if small < 3 mm), delayed repair may be required in future
TYPE D	Simple suture closure Suture closure over a simple Stent / T-tube
E1, E2	End-to-end repair over a T-tube/internal stent Roux-en-Y hepatico-jeunostomy
E3, E4, E5	Roux-en- Y Hepatico-jeunostomy Need for two stomas in some E4 and E5

TECHNIQUE:

Incision and Exposure

A right subcostal incision with or without an midline extension provides adequate exposure .Use of self retaining retractors like Thompson gives good exposure . Dissection should begin in the subhepatic area, and the hepatic flexure of the colon mobilized completely. A choledochoduodenal or duodenal wall rent occurring requires repair.

The dissection has to be meticulous to prevent injury to the adherent colon and to reach the hepatic hilum."

Since the distal duct generally cannot be used for anastomosis attempts to identify the distal bile duct are unnecessary; and such attempts risks injury to the hepatic artery and portal vein as it is usually encased in dense scar tissue.[34]

APPROACH TO THE LEFT HEPATIC DUCT :

It is safe to divide the fibrous tissue between the bile duct and the liver (hilarplate) to expose the bifurcation of the hepatic duct as well as the extrahepatic left hepatic duct. This dissection is greatly facilitated by placing a curved retractor to elevate segment in an anterocephaladdirection. This region is likely to be free of adhesions from previous surgery.

APPROACH TO THE RIGHT HEPATIC DUCT:

By effectively lowering the hilar plate,a plane of dissection is developed at the porta hepatis. The confluence may be exposed using above approach . If the extrahepatic course of the right hepatic duct is too short to be visualized , hepatotomies may be made to expose the right portal pedicle.

It is important to correlate the pre-operative cholangiogram with the intra-operative findings as this is an important factor affecting a successful outcome.

ANASTAMOTIC TECHNIQUE

A standardized technique of hepaticojejunostomy is applied .This technique is of particular utility for high anastomoses, where duct mobility and size are limited.

A 60-cm Roux-en-Y limb of jejunum is prepared about 20 cm from duodenal flexure . A generous length (15 to 20mm) of healthy bile duct is need for anastomosis keeping in mind the inevitable postoperative contracture.

If the right anterior and posterior sectoral ducts are isolated , a septoplasty can be done to join them to make a common stoma.

Two or more stomas may, however, be required in a high stricture. Anastomotic construction begins with an anterior row of 3-0 or 4-0 absorbable sutures, working from left to right. It is important not to injure any of the hepatic arteries as the blood of the bile duct might have been compromised by a non- apparent arterial injury during the index cholecystectomy..Complicated procedures such as an intrahepatic cholangiojejunostomy(to the segment III or V ducts, Longmire procdure (hepaticojejunostomy to the cut surface of the liver), smith's mucosal graft, porto-enterosotmy , etc may be thought of as alternatives in high and difficult strictures but not recommended as they are all associated with ha higher risk of anastomotic stricuture and recurrence. A liver resection is best alternative in such cases.

USE OF STENTS:

The routine use of transanastomotic stents in all cases is not recommended. In case the anastomosis looks precarious (thin walled friable ducts, sutures cutting through) and an anastomotic leak is anticipated, a transcutaneous biliary drain can be placed retrogradely through the bile duct and into the liver parenchyma using a probe wire, a vascularized flap of omentum can also be used to protect the anastomosis. Transanastomotic stents are retained for several months and may sometimes be required for 6-12 months.

A liver biopsy (wedge or needle) is obtained in all patients to rule out a hepatic parenchymal cause of continued hyperbilirubinemia postoperatively. A sub-hepatic drain is usually left in situ.

HEPATIC RESECTION.

Patients who have concomitant vascular injury or undergone prior unsuccessful repairs or long-standing cholangitis often develop sectional duct strictures or interruptions between the right-sided and left-sided biliary tree, effectively precluding biliary-enteric revision. These patients land up in hemi atrophic liver .Such patients requires formal liver resection for removal of atrophied liver . Hepatic resection is usually indicated in refractory bilary stricture.

LIVER TRANSPLANTATION.

Transplantation is occasionally indicated for vascular injury leading to biliary cirrhosis [68-69]. .Orthotopic liver transplantation also considered in patients with secondary biliary fibrosis resulting from longstanding biliary obstruction progressing to cirrhosis .

PORTAL HYPERTENSION :

Reported incidence of portal hypertension at the time of referral is 10% to 20% of patients [70-71]. Bleeding esophageal varices, hypersplenism or ascites, render the overall prognosis far worse [72-73].

Collateral venous channels and dense adhesions make dissection difficult It is preferable to attempt stenting or balloon dilation in seriously ill patients with jaundice and portal hypertension than to proceed to immediate definitive repair [74-78]. A portosystemic shunt was performed and the hepatic hilum is approached again after an interval of 3-6 months in patients with portal hypertension creation of roux loop of jejunum amy be associated with blood loss: a simple loop with a distal jejuno-jejnostomy (Braun) may be used.

MATERIALS & METHODS

Period of Study

September 2017 to September 2019

Place of Study

Institute of General surgery

Madras medical college

Chennai-10

Inclusion Criteria :

All cases admitted to surgical wards with biliary leakage after choleycystectomy both lap and open after 12 years of age.

Exclusion criteria :

Patient undergoing cholecystectomy as a part of some primary operation like Whipple's operation, biliary-enteric anastomosis are excluded from this study.

SAMPLE SIZE: 100

STUDY METHODOLOGY:

Seventy patients were evaluated for laparoscopic cholecystectomy between september 2017 to September 2019. All of them were planned to undergo laparoscopic cholecystectomy electively or underwent urgent surgery after admission to hospital for acute emergency relating to gallstones. USG was done in all patients to confirm the diagnosis of cholelithiasis .The patients were excluded from the study if they underwent open procedure or not able to fill the proforma. Cholecystectomy indications and investigations done for surgery were obtained. : 10 cases of biliary leaks were studied from 100 open cholecystectomies and 100 laparoscopic cholecystectomies from 2013 January to 2014 January. Pre-operative workup with ultrasound abdomen, LFT and other biochemical investigations were done and diagnosis was established. The outcome of surgery and its most common post-operative complication - biliary leak was studied with following parameters: Post-operative diagnosis, Surgery - Lap or Open, Incision, Method of approach – Fundus or Classical method, CBD exploration, Intra-operative findings, Drains used, Post-operative symptoms, Amount of collections, Removal of drains, USG – Sub hepatic collections, ERCP etc.

RESULTS

1.AGE OF THE PATIENT:

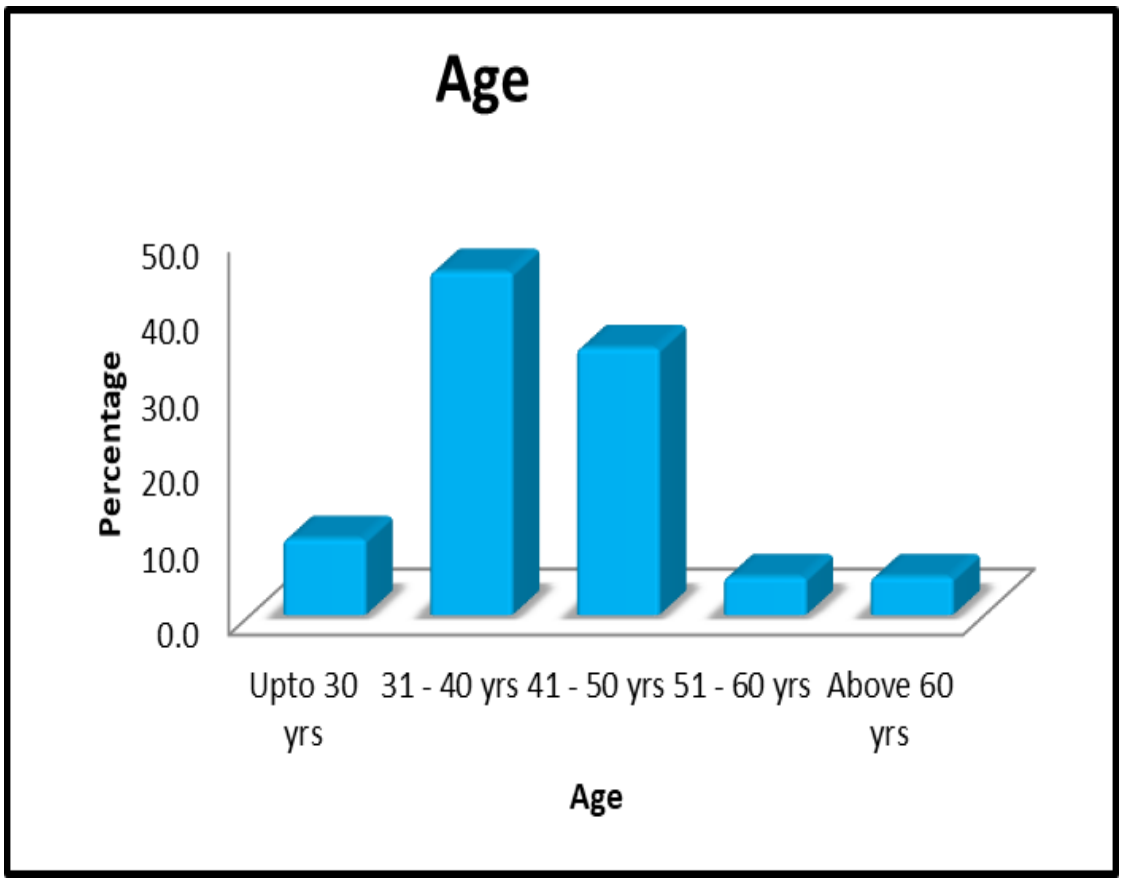
Among 100 persons,10% were under 30 years ,45% were under 40 years, 355 under 50 years.thus most of the patients in my study were between 30 to 50 years of age**.**

TABLE 1

Age	Frequency	Percent
Upto 30 yrs	10	10.0
31 - 40 yrs	45	45.0
41 - 50 yrs	35	35.0
51 - 60 yrs	5	5.0
Above 60 yrs	5	5.0
Total	100	100.0

AGE DISTRIBUTION%

GRAPH 1



2.SEX OF THE PATIENT:

In my study female patients were 66% and male patients were 34%.female are pre4dominant in this group.

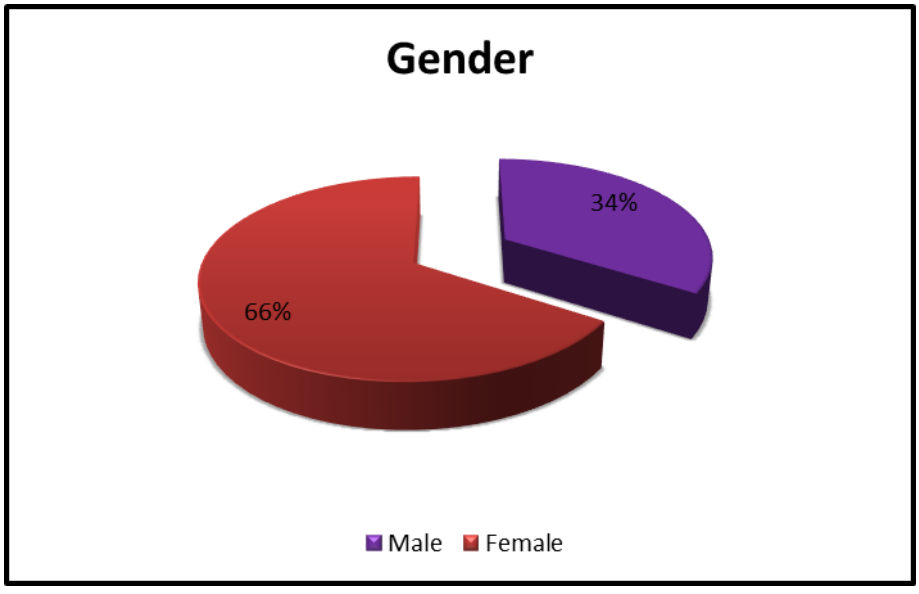
SEX

	Frequency	Percent
Male	34	34.0
Female	66	66.0
Total	100	100.0

Table 2

GENDER DISTRIBUTION

GRAPE 2



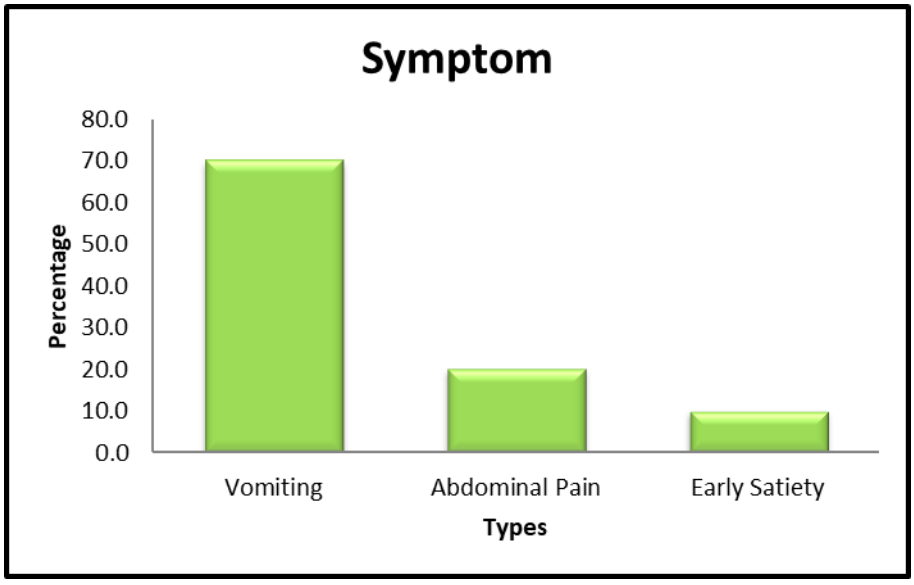
3.SYMPTOMS

In my study vomiting ,early satiety,abdominal pain were analysed in this group study.early satiety and vomiting were most common symptoms**.**

Table 3

	Frequency	Percent
Vomiting	70	70.0
Abdominal Pain	20	20.0
Early Satiety	10p	10.0
Total	100	100.0

Graph 3

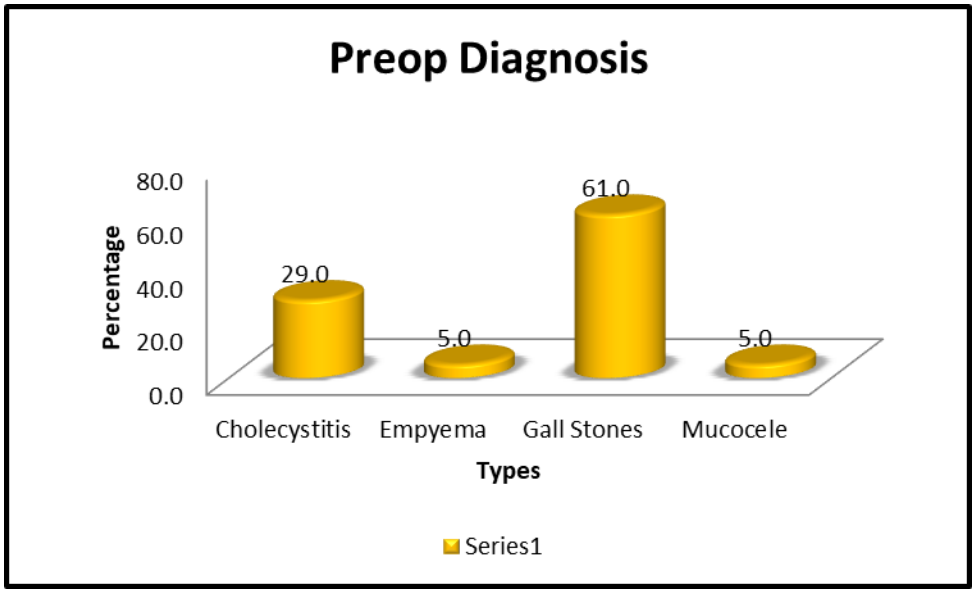


4.**PREOP DIAGNOSIS**

In my study gallstones was the most commonly indication for lap choleycystectomy.choleycystitis was most found to be 29% and gall stones 61%.

DIAGNOSIS	FREQUENCY	PERCENT
Cholecystitis	29	29.0
Empyema	5	5.0
Gall Stones	61	61.0
Mucocele	5	5.0
Total	100	100.0

TABLE 4



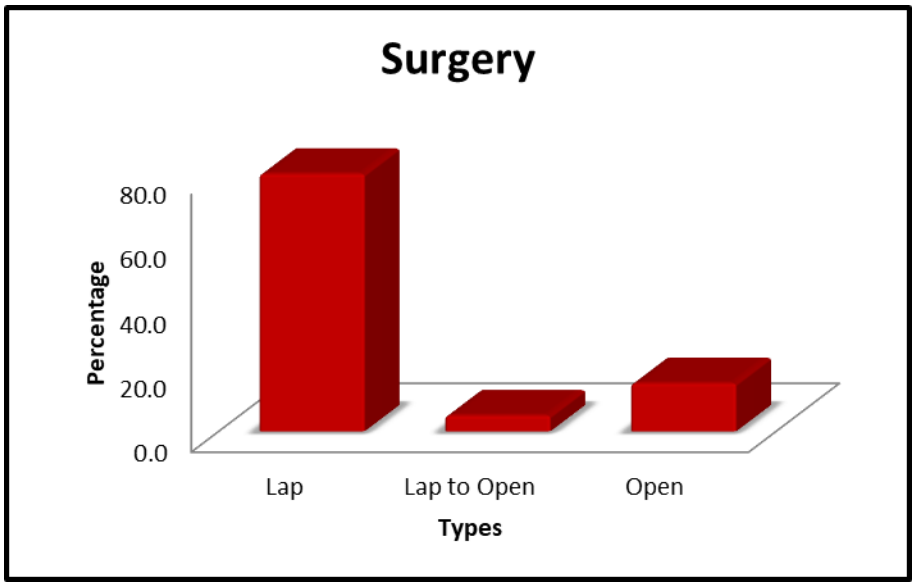
GRAPH 4

5.**SURGERY**

Total patients were 100 patients included in this study out of 100, 55 %underwent lap cholycystectomy and 40 %were underwent open choleycystectomy

	Frequency	Percent
Lap	55	55.0
Lap to Open	5	5.0
Open	40	40.0
Total	100	100.0

Table 5



Graph 5

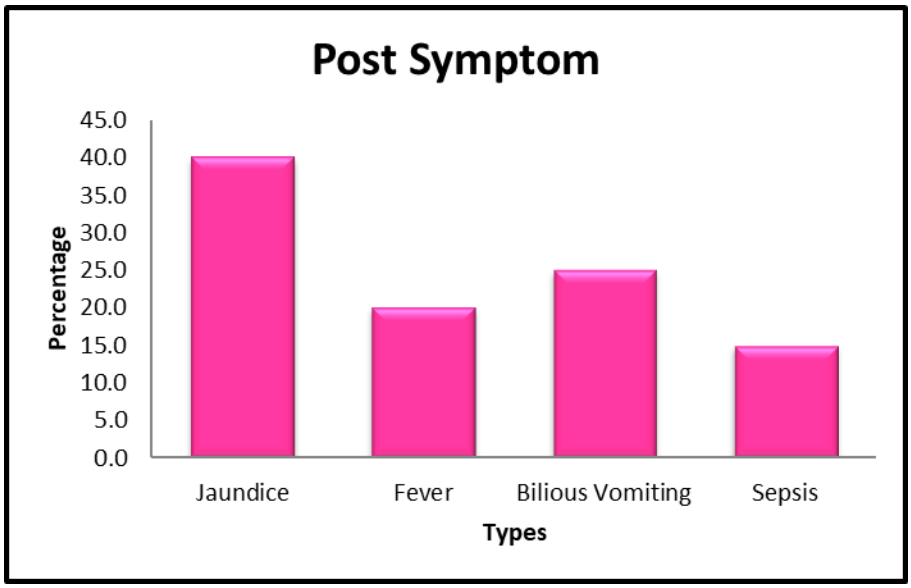
6. POST OPERATIVE SYMPTOMS

The most common post operative symptoms were found to be jaundice, pain fever, vomiting.

POST SYMPTOM

	Frequency	Percent
Jaundice	40	40.0
Fever	20	20.0
Bilious Vomiting	25	25.0
Sepsis	15	15.0
Total	100	100.0

Table 6



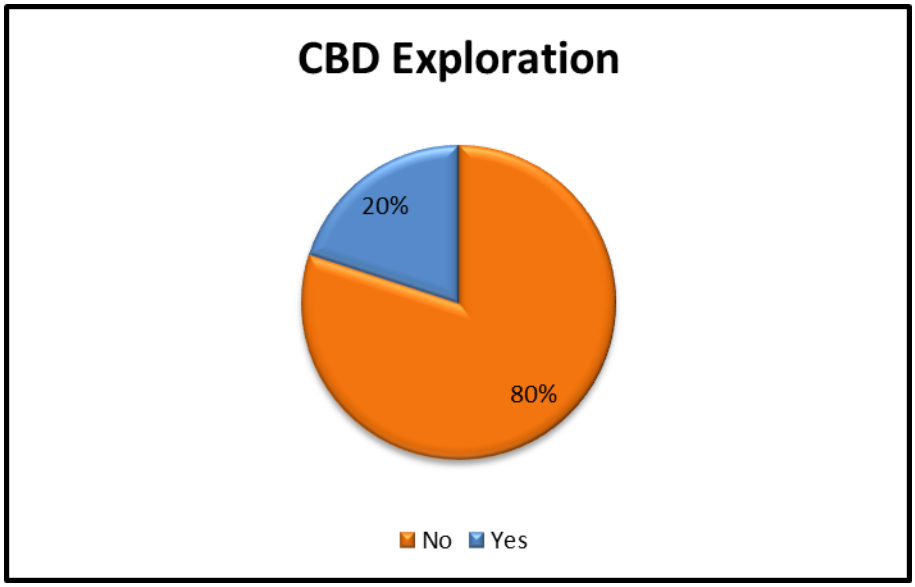
Graph 6

7. CBD EXPLORATION

In my study CBD exploration was done for 20% out of 100.

	Frequency	Percent
No	80	80.0
Yes	20	20.0
Total	100	100.0

Table 7



Graph 7

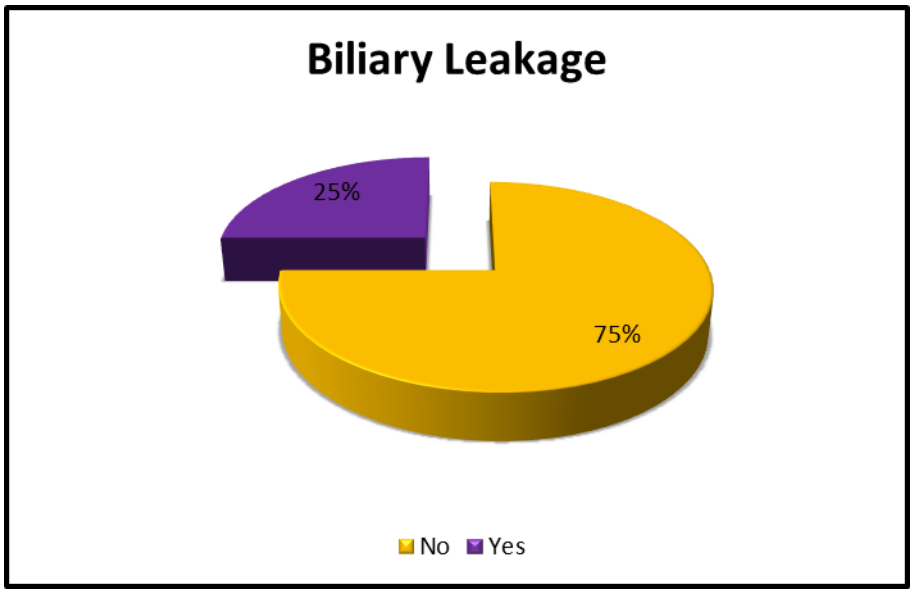
8.BILIARY LEAKAGE

Out of 100 patients post choleycystectomy biliary leakage was observed in 25% of patients.

TABLE

	Frequency	Percent
No	75	75.0
Yes	25	25.0
Total	100	100.0

Table 8



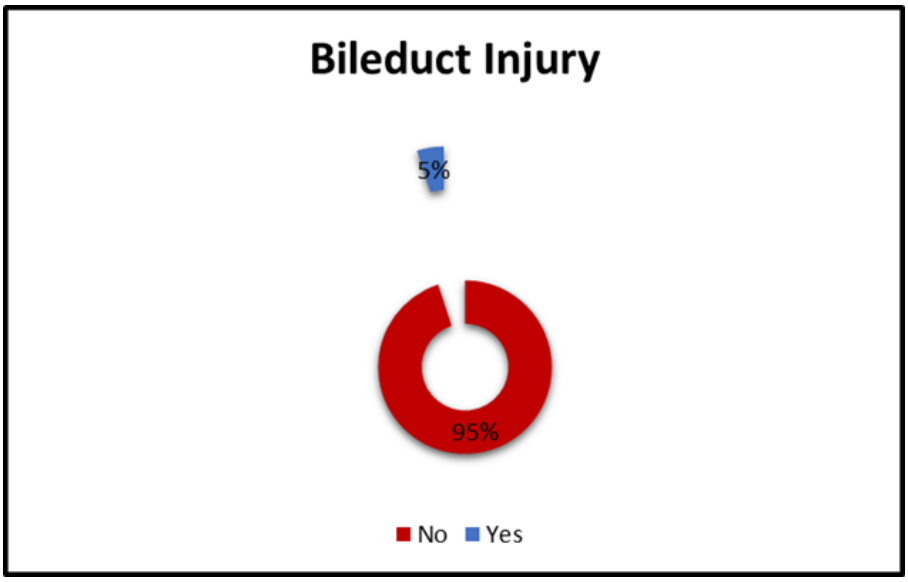
GRAPE 8

9. BILEDUCT INJURY

In my study bile duct injury was observed only 5% of patients out of 100% patients.

Table 9

	Frequency	Percent
No	95	95.0
Yes	5	5.0
Total	100	100.0



GRAPE 8

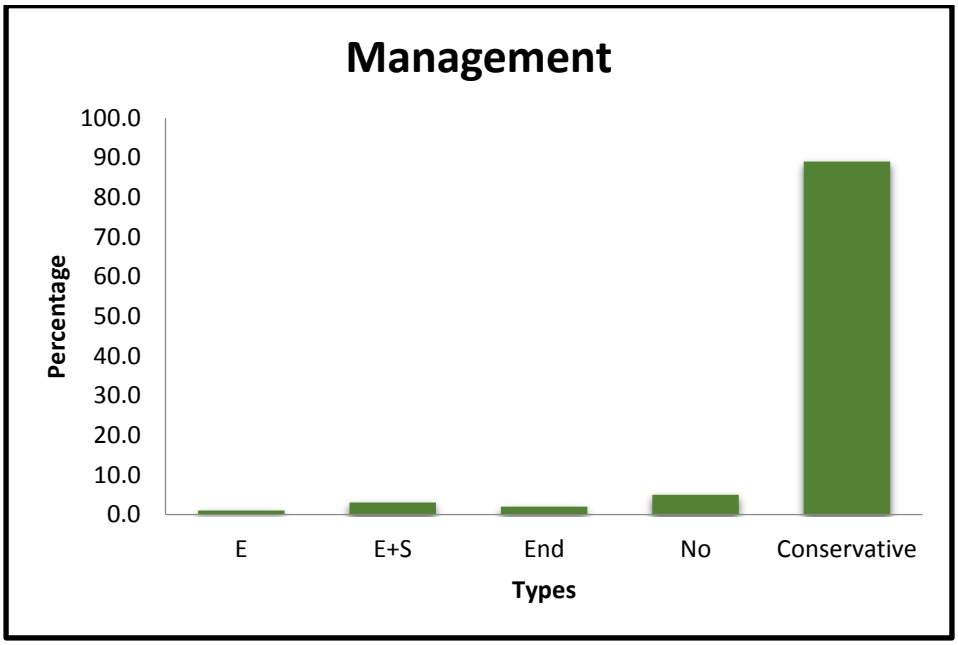
10.MANAGMENT

In my study post choleycystectomy biliary leakage was managned conservatively by89% and sphinterotomy done was 5% and bile duct repair was done by 2% and stenting done was 3%.reoperation was done for 1%

Table 10

	Frequency	Percent
REOPERATION	1	1.0
STENTING	3	3.0
BILEDUCT REPAIR	2	2.0
SPHINTEROTOMY	5	5.0
CONSERVATIVE	89	89.0
TOTAL	100	100.0

GRAPH 10



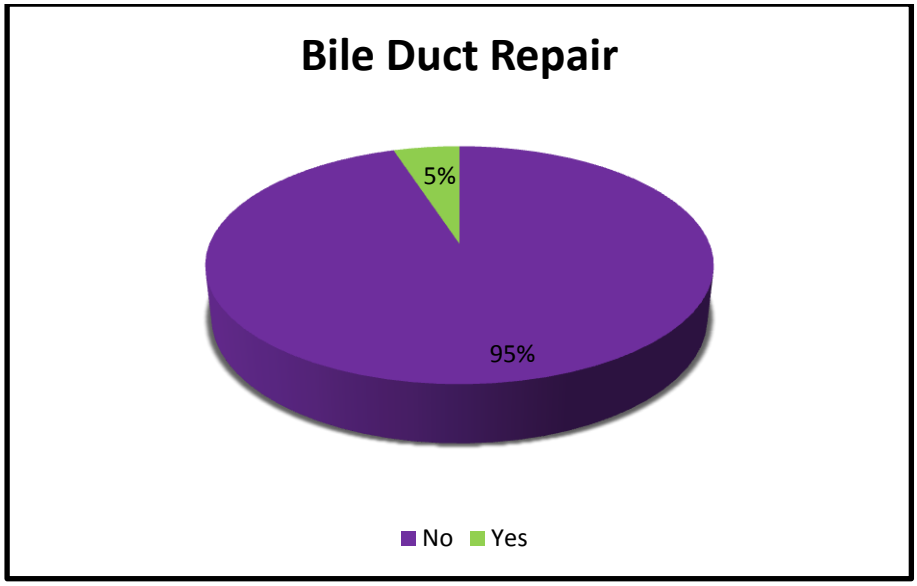
11.BILEDUCT REPAIR

In my study bile duct repair was observed only 5% of patients out of 100% patients.

TABLE 11

BILE DUCT REPAIR

	Frequency	Percent
No	95	95.0
Yes	5	5.0
Total	100	100.0



GRAPH 11

12. MORTALITY

MORTALITY

	Frequency	Percent
NO	100	100.0

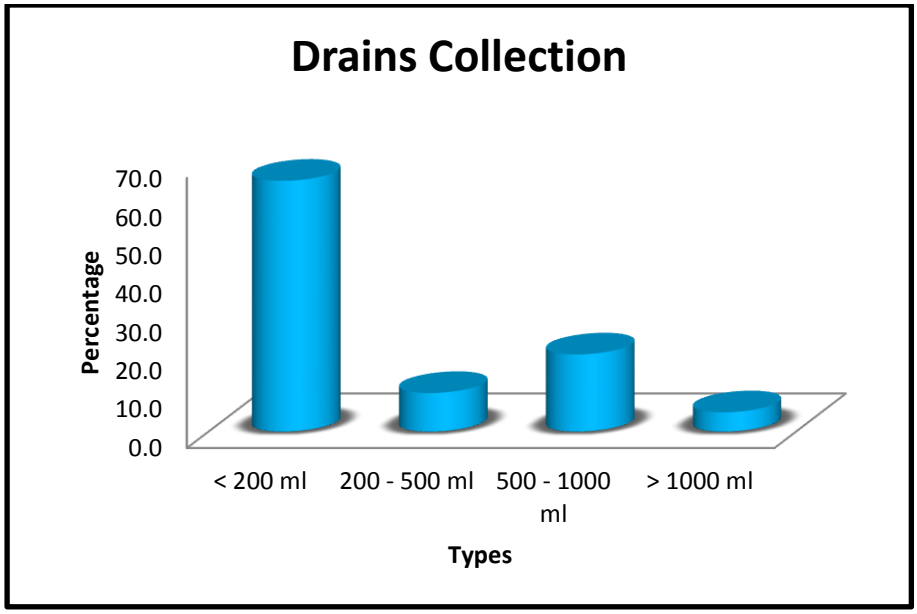
13.DRAIN COLLECTION

TABLE 13

DRAINS COLLECTION

	Frequency	Percent
< 200 ml	65	65.0
200 - 500 ml	20	20.0
500 - 1000 ml	10	10.0
> 1000 ml	5	5.0
Total	100	100.0

GRAPH 13



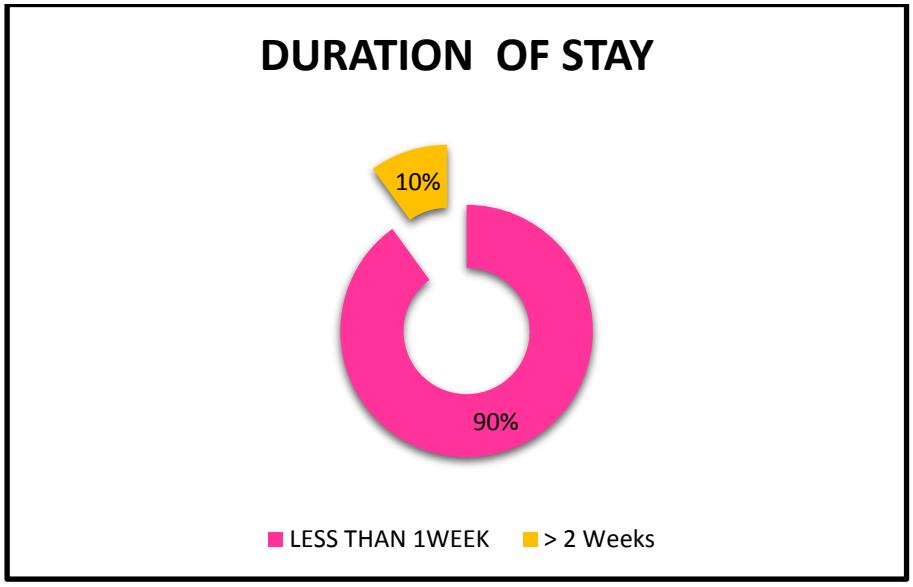
DESCRIPTIVE STATISTICS

	N	Minimum	Maximum	Mean	S.D
AGE	100	26.0	64.0	41.100	9.0448
TB	100	3.0	5.0	4.175	3.8326
DB	100	3.0	5.0	3.160	3.7592
ALP	100	12.0	224.0	105.700	56.1915
SGOT	100	10.0	266.0	105.800	75.9144
DRAINS COLLECTION	100	15.0	1350.0	276.750	356.9097
HOSPITAL STAY	100	8.0	18.0	11.350	2.3414

14.DURATION OF HOSPITAL STAY

	Frequency	Percent
LESS THAN 1WEEK	90	90.0
> 2 Weeks	10	10.0
Total	100	100.0

TABLE 14



GRAPH 14

DISCUSSION

Operative repair is the gold standard management of post laparoscopic bile duct stricture .The aim of the definite management of patients is to establish an uninterrupted bile flow into the gastrointestinal tract. Experience from large volume centers has consistently shown successful results following bilio-enteric drainage in more than 90% patients[52]

The results of surgical repair depend on the timing of the repair, previous failed attempts, the level of injury, associated vascular injuries, and the presence of infection at that time of the repair. The most important initial step for success of a bile duct stricture repair is the expertise and experience of the team performing it . Chronic cholecystitis appeared to be the most common cause in the bile duct injury than acute cholecystitis of the 25 cases. 52% (13) found to have chronic cholecystitis as compared to the acute cholecystitis with 20% (5 cases). Onse study showed acute cholecystitis is the most common cause of bile duct injury. This controversy is due to increased number of cases of chronic cholecystitis as they delay in approaching for treatment in G.M.C. when compared to the western patients getting the earliest possible treatment.

TIMING OF SURGERY:

Timing for repair requires careful evaluation and depends highly on the patient's clinical condition, time from injury , hepatic function, type of injury (biliary fistula vs. obstruction), comorbidities, extent of inflammation, nutritional status, and presence of infection or abscess. Patients present at a median of 3 days after laparoscopic cholecystectomy (1 day to 93 weeks).[53] .

EARLY APPROACH :

Patients who present early after surgery, and show no signs of sepsis, intraabdominal collections, or vascular injury, should be considered for early repair within 72 hours. These patients have simpler injuries .Strasberg type A injuries, cystic duct leaks, leaks from a duct of Luschka can be managed with endoscopic sphincterotomy and the placement of a biliary stents (typically 8- to 10-French. plastic stent). Strasberg type D injuries can also be approached in the early postoperative period. These injuries are also amenable to endoscopic sphincterotomy and stenting.Immediate repair can performed if expertise is available.

In case of a partial transection of the common bile duct recognized at the time of initial surgery, primary repair over a T-tube gives good outcome. Fine, monofilament, absorbable sutures should be used for the repair and the T-tube brought out via common bile duct at a distant site away from the repair site . One study reported a restricture rate of nearly 100% for end-to-end repairs of the common bile duct especially if the injury is secondary to the use of cautery or results in complete transection of the duct .[14] These patients managed with a biliary-enteric anastomosis as later described. However, Stewart and Way indicate success of repair does not depend on timing of repair but rather depends on eradication of bilioma, , use of a single layer end-to-side hepaticojejunostomy with fine absorbable suture, and severity of Stewart-Way injury class.[54]

DELAYED APPROACH

Walsh et al indicate that early stricture repair has a higher stricture rate than delayed repair (19% vs. 8%).[55]. Sahajpal et al report a high stricture rate in patients repaired between 72 hours and 6 weeks of the injury.[56] . Wait for atleast 6-8 weeks after the bile duct injury, may allow sepsis better controlled, and allow patient recovery from the acute injury with better understanding of level and type of injury.

Operative repair cannot be completed within 72 hours injury because of patient condition inability to complete radiographic workup, delay of repair is often advocated.[57]. This approach results in dense adhesions, making definitive repair difficult.[58] .Regardless of timing of repair, intra-abdominal sepsis and patient condition must be stabilized before repair of complex injuries.Some of the biliary injuries not require biliary-enteric anastomosis. Because 31% to 54% successfully managed with closure over a T-tube or endoscopically stented, endoscopic or percutaneous management of the injuries should be considered in Strasberg type A and type D e duct injuries. But for major bile duct injuries, 93% will require surgical reconstruction with biliary-enteric anastomosis. Biliary colic was the most reliable symptom for gallstones.It has high negative predictive value or absence of pain makes the patient less operable. Biliary colic is defined as pain in upper abdomen either in epigastric or right hypochondrial regions , Ranging from minutes to upto 5 hours with radiation of pain to the tip of right shoulder (102). Eventhough sleep of the patient is disturbed.He will still be ambulant. Exacerbation of pain after food is present. There is absence of elevated temperature or local tenderness as in acute cholecystitis.vomiting and nausea may be reported.

Acute Cholecystitis patients will often have history of biliary pain in acute cholecystitis,lasting for > 3 hours, associated with fever and right upper quadrant tenderness (Murphy's sign).Chronic Chlolecystitis patients most episodic epigastric, right upper quadrant pain lasting for more than 30 minutes. Patients, may present with complications of gallstones-pancreatitis, choledocholithiasis and cholangitis May be asymptomatic or present with biliary colic, acute cholangitis or pancreatitis. Acute Cholangitis was a emergency condition tat Patients will present with Charcot's triad- right upper quadrant pain, fever and jaundice.

CONCLUSION

The anatomy of biliary tree will be highly variable in normal subjects also. Accessory bile ducts will cause bile leak not only intraoperatively and postoperatively for prolonged periods. The bile leakage from accessory bile ducts can be seen by intraoperative cholangiograms only. laparoscopic cholecystectomies, use of intraoperative cholangiograms must be identify the source of bile leakage.

Postoperative bile leakage will be managed conservatively if the amount of bile leak is less than 250ml/24 hrs although it might take weeks to settle. In our experience, a structured stepwise approach to the management of uncommon complications such as bile leaks is advantageous. Alternatively, they can be transferred to centres where minimally invasive expertise is routinely available. Most of the biliary leakage can be managed conservatively.Female patients are more prone for gall stones particularly am0ong age group 40 to 50 years of age. Laproscopic choleycystectomy was one of the gold standard procedure foe symptomatic gall stones.

PROFOMA

Demographic Profile :-

1.	Name	:	
2.	Age	:	/yrs
3.	Sex	:	M / F
4.	Occupation	:	
5.	Income	:	
6.	IP Number	:	
7.	Date of Admission	:	
8.	Date of Surgery	:	
9.	Date of Discharge	:	
10.	Length of Hospital Stay	: Total-	/ days, Post op-/days

Clinical Details :-

1. **Symptoms** :

Right hypochondriac pain / Anorexia / Weight loss / Jaundice / Fever / UGI bleed

Duration of symptoms

2. Jaundice / Blood Transfusion
3. Alcohol / Smoking
4. Co-mobidities
5. Previous Surgery
6. Significant Family History
7. Clinical Examination details with per examination details – lymphadenopathy, Biloma.
8. Details of any pre-operative treatment – Open / Lap cholecystectomy
    - Biloma Drainage by Per-Cutaneous / Lap
    - Bilary operation history before this admission
9. Interval between trauma & referral

Details of preoperative Investigations :-

1. Complete haemogram and basic urine analysis.
2. Blood sugar, renal function test with electrolytes.
3. Liver function test.
4. Viral markers status (HBs Ag and Anti - HCV).
5. Findings of Radiological imaging (CXR, USG with Doppler, CECT / MRI).Dilatation of Bile Duct above Stricture

Intraoperative Parameters :-

1. Type of Surgery – lap or open choleycystectomy.
2. Associated Vascular injury
3. Bile duct injury
4. Operative time
5. Blood loss
6. Blood transfusion – no of transfusions

Post op complications :-

1. Mortality
2. Haemorrhage
3. Cholangitis
4. Bile leak & bilioma

5. Wound complications
6. Pulmonary, cardiac and renal complications
7. Miscellaneous

Follow up details :-

1. Duration of Follow up – assessment of Symptoms / signs.
2. LFT
3. Radiological imaging
4. Mortality – cause and duration after surgery.

நோயாளி சம்மத படிவம்

நுண்துளை அறுவைசிகிச்சை மூலம் பித்தப்பையை அகற்றியபிறகு வரும் பித்தக்குழாய் சுருக்கத்திற்கு மேற்கொள்ளப்படும் அறுவை சிகிச்சையின் முடிவுகளை நிர்மாணிக்கும் காரணிகள்

நோயாளியின் பெயர் .....

வயது ..... வருடங்கள் அல்லது பிறந்த தேதி .....

நோயாளியை தொடர்பு கொள்ளும் முகவரி .....

.....

.....

நோயாளியின் தொலைபேசி எண்.

நோயாளியின் இன்சியல்ஸ் ..... பாலினம் ஆண் ..... பெண் .....

		பங்கேற்பவரின் இன்சியல்/பெரு விரல் பதிப்பு
1)	மேல் குறிப்பிடப்பட்டுள்ள ஆய்வின் ..... தேதியிட்ட நோயாளிகளுக்கான செய்தி நான் படித்திருக்கிறேன் மற்றும் புரிந்திருக்கிறேன்/ விவரிக்கப்பட்டுள்ளேன். கேள்விகள் கேட்கவும் அனுமதி வழங்கப்பட்டுள்ளேன் என நான் உறுதி செய்கிறேன்.	
2)	இந்த ஆய்வில் பங்கேற்பது என் சொந்த விருப்பப்படியே என நான் புரிகிறேன். மேலும் என் மருத்துவ சிகிச்சை கவனிப்பு அல்லது சட்ட பூர்வ உரிமைகளுக்கு பாதிப்பு ஏற்படாமல் நான் எந்த நேரத்திலும் விலகிக் கொள்ளலாம் என்பதை புரிகிறேன்.	
3)	எத்திக்ஸ் கமிட்டி மற்றும் ரெகுலேட்டரி அதாரிட்டீஸ்க்கும் நான் இந்த ஆய்விலிருந்து விலகினாலும் தற்போதைய மற்றும் எதிர்கால இந்த ஆய்வு சார்ந்த என் உடல்நல குறிப்புகளை என் அனுமதியின்றி பார்க்க முடியும் என நான் அறிகிறேன்.	
4)	இந்த ஆய்வில் கிடைக்கப்பெறும் குறிப்புகள் மற்றும் முடிவுகளை உபயோகப்படுத்த தடை செய்ய மாட்டேன் என சம்மதிக்கிறேன். ஆனால் அவைகள் விஞ்ஞானம் சம்மந்தப்பட்டவைகளுக்கு மட்டும் பயன் உள்ளதாக இருக்க வேண்டும்.	
5)	மேற்கூறிய ஆய்வில் பங்கேற்க நான் சம்மதிக்கிறேன்.	

ஆய்வில் பங்கேற்பவர் / சட்டபூர்வமாக
ஏற்கப்பட்ட நபர் கையொப்பம் அல்லது
பெருவிரல் பதிவு

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ABBREVIATIONS

CBD – Common bile duct

LC – Laparoscopic cholecystectomy

GB- Gall bladder

GS-Gallstones

USG-Ultrasonography

ERCP-Endoscopic retrograde cholangiopancreatography

MRCP-Magnetic resonance cholangiopancreatography

MRI-Magnetic resonance imaging

PCS-Post-cholecystectomy syndrome

AC-Acute Cholecystitis

CL-Choledocolithiasis

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
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CERTIFICATE OF APPROVAL

To
Dr.S.Prabhakaran
Post Graduate in MS General Surgery
Institute of General Surgery
MMC/Chennai

Dear Dr.S.Prabhakaran,

The Institutional Ethics Committee has considered your request and approved your study titled "**A STUDY OF POST CHOLEYCYSTECTOMY BILIARY LEAKAGE AND MANAGEMENT**" - **NO.17122017**

The following members of Ethics Committee were present in the meeting hold on **12.12.2017** conducted at Madras Medical College, Chennai 3

1. Prof.P.V.Jayashankar	:Chairperson
2. Prof.R.Narayana Babu,MD.,DCH., Dean,MMC,Ch-3	: Deputy Chairperson
3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4. Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch	: Member
5. Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3	: Member
6. Prof.A.Pandiya Raj,Director, Inst. of Gen.Surgery,MMC	: Member
7. Prof.Shanthy Gunasingh, Director, Inst.of Social Obstetrics,KGH	: Member
8. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai	: Member
9. Prof. Susila, Director, Inst. of Pharmacology,MMC,Ch-3	: Member
10.Prof.K.Ramadevi,MD., Director, Inst. of Bio-Chemistry,MMC,Ch-3	: Member
11.Prof.Bharathi Vidya Jayanthi,Director, Inst. of Pathology,MMC,Ch-3	: Member
12.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
13.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist
14.Thiru K.Ranjith, Ch- 91	: Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

2019/10/31 16:18

URKUND

Urkund Analysis Result

Analysed Document:	post chole biliary leakage_1.docx (D58004956)
Submitted:	10/31/2019 4:51:00 PM
Submitted By:	prabhumedsparkz@gmail.com
Significance:	3 %

Sources included in the report:

A CLINICAL STUDY AND MANAGEMENT OF CHOLELITHIASIS.docx (D42444299)
gall stones.docx (D42511652)
THESIS (1).docx (D28013430)
THESIS.docx (D22568330)
dr selva kumar thesis.docx (D57881944)
https://jamanetwork.com/journals/jamasurgery/fullarticle/406203
https://www.slideshare.net/EricMyrzahanov/anatomy-of-gall-bladder-and-excretory-bile-ducts
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https://www.imaios.com/en/e-Anatomy/Anatomical-Parts/Cystohepatic-triangle
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Instances where selected sources appear:

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SNO	NAME	AGE	SEX	SYMPTOMS	PREOPERATIVE DIAGNOSIS	SURGERY LAP/ OPEN CHOLY	POST SYMPTOM	L	F	T		CBD EXPLRATION	DRAIN USED	BILIARY LEAKAGE	DRAINS COLLECTION	BILEDUCT INJURY	CONSERVATIVE	HOSPITAL STAY	BILE DUCT REPAIR	MORTALITY
								TB	DB	ALP	SGOT									
1	Arulselvi	35	2	1	GALL STONES	LAP	1	10	1.3	49	21	NO	NO	NO	15ML	NO	YES	10	NO	NO
2	Alamelu	28	2	2	CHOLECYSTITIS	OPEN	2	1.2	0.7	114	129	YES	YES	NO	150ML	NO	YES	18	NO	NO
3	Zareena	38	2	1	GALL STONES	LAP	4	4	17	100	111	NO	YES	NO	50ML	NO	YES	10	NO	NO
4	Ganapathy	64	1	1	CHOLECYSTITIS	OPEN	3	2	1	86	90	YES	YES	YES	1350ML	YES	NO	12	YES	NO
5	Chinaponnu	45	2	1	GALL STONES	LAP	1	3	3.9	97	96	NO	YES	NO	100 ML	NO	END	10	NO	NO
6	Rathidevi	48	2	1	GALL STONES	LAP	3	1.7	3	12	10	NO	YES	NO	50ML	NO	YES	10	NO	NO
7	Jayanthi	32	2	2	CHOLECYSTITIS	OPEN	1	3.4	5	95	57	NO	NO	YES	800ML	NO	YES	9	NO	NO
8	Bhoopathy	40	1	1	GALL STONES	LAP	1	5.4	3	89	86	NO	YES		50ML	NO	YES	11	NO	NO
9	Komala	49	2	2	CHOLECYSTITIS	OPEN	3	1.3	4.9	49	51	YES	YES	YES	750ML	NO	YES	10	NO	NO
10	Krishnaveni	35	2	1	GALL STONES	LAP	2	0.7	1	97	76	NO	YES	NO	100ML	NO	YES	10	NO	NO
11	Lakshmi	58	2	2	CHOLECYSTITIS	OPEN	1	17	3	110	25	YES	YES	NO	200ML	NO	YES	12	NO	NO
12	Mahalakshmi	40	2	1	GALL STONES	LAP	2	1	9	130	80	NO	YES	NO	100ML	NO	YES	10	NO	NO
13	Patachi	45	2	1	CHOLECYSTITIS	OPEN	4	3.9	1.1	95	75	NO	YES	YES	700ML	NO	END	10	NO	NO
14	Ravi	40	1	1	GALL STONES	LAP	3	3	1.2	93	100	NO	YES	NO	50ML	NO	YES	16	NO	NO
15	Ravi	42	1	1	GALL STONES	LAP	1	5	1.1	45	42	NO	YES	NO	50ML	NO	YES	12	NO	NO
16	Sekar	42	1	3	EMPYEMA	OPEN	1	3	1.3	67	89	NO	YES	NO	350ML	NO	YES	11	NO	NO
17	Senthil Kumar	33	1	1	GALL STONES	LAP	2	4.9	2.0	224	234	NO	NO	YES	600ML	NO	E	13	NO	NO
18	Shantha	45	2	3	MUCOCELE	OPEN	4	1	1.2	217	244	NO	YES	NO	20ML	NO	YES	11	NO	NO
19	Sivasakthi	26	2	1	GALL STONES	LAP	3	3	1.3	222	266	NO	YES	NO	20ML	NO	YES	8	NO	NO
20	Suriyakala	37	2	1	GALL STONES	LAP TO OPEN	1	9	1.2	123	234	NO	YES	NO	50ML	NO	YES	14	NO	NO

SNO	NAME	AGE	SEX	SYMPTOM	PREOP DIAGNOSIS	SURGERY LAP/ OPEN	POST SYMPTOM	L	F	T		CBD EXPLRATION	DRAIN USED	BILIARY LEAKAGE	DRAINS COLLECTION	BILEDUCT INJURY	MANAGMENT	HOSPITAL STAY	BILE DUCT REPAIR	MORTALITY
								TB	DB	ALP	SGOT									
21	Arjun	35	2	1	GALL STONES	LAP	1	10	1.3	49	21	NO	NO	NO	15ML	NO	YES	10	NO	NO
22	Mani	28	2	2	CHOLECYSTITIS	OPEN	2	1.2	0.7	114	129	YES	YES	NO	150ML	NO	YES	18	NO	NO
23	Muniyammal	38	2	1	GALL STONES	LAP	4	4	17	100	111	NO	YES	NO	50ML	NO	YES	10	NO	NO
24	Balu	64	1	1	CHOLECYSTITIS	OPEN	3	2	1	86	90	YES	YES	YES	1350ML	YES	NO	12	YES	NO
25	Vikram	45	2	1	GALL STONES	LAP	1	3	3.9	97	96	NO	YES	NO	100 ML	NO	YES	10	NO	NO
26	Vijay	48	2	1	GALL STONES	LAP	3	1.7	3	12	10	NO	YES	NO	50ML	NO	YES	10	NO	NO
27	Seetha	32	2	2	CHOLECYSTITIS	OPEN	1	3.4	5	95	57	NO	NO	YES	800ML	NO	E+S	9	NO	NO
28	Surendr	40	1	1	GALL STONES	LAP	1	5.4	3	89	86	NO	YES		50ML	NO	YES	11	NO	NO
29	Prabhu	49	2	2	CHOLECYSTITIS	OPEN	3	1.3	4.9	49	51	YES	YES	YES	750ML	NO	YES	10	NO	NO
30	Manikam	35	2	1	GALL STONES	LAP	2	0.7	1	97	76	NO	YES	NO	100ML	NO	YES	10	NO	NO
31	Naranayasamy	58	2	2	CHOLECYSTITIS	OPEN	1	17	3	110	25	YES	YES	NO	200ML	NO	YES	12	NO	NO
32	Tejas	40	2	1	GALL STONES	LAP	2	1	9	130	80	NO	YES	NO	100ML	NO	YES	10	NO	NO
33	Stephan	45	2	1	CHOLECYSTITIS	OPEN	4	3.9	1.1	95	75	NO	YES	YES	700ML	NO	END	10	NO	NO
34	Vikrapandfi	40	1	1	GALL STONES	LAP	3	3	1.2	93	100	NO	YES	NO	50ML	NO	YES	16	NO	NO
35	Muniyammal	42	1	1	GALL STONES	LAP	1	5	1.1	45	42	NO	YES	NO	50ML	NO	YES	12	NO	NO
36	Prema	42	1	3	EMPYEMA	OPEN	1	3	1.3	67	89	NO	YES	NO	350ML	NO	YES	11	NO	NO
37	Ponmalar	33	1	1	GALL STONES	LAP	2	4.9	2.0	224	234	NO	NO	YES	600ML	NO	END	13	NO	NO
38	Banumathiu	45	2	3	MUCOCELE	OPEN	4	1	1.2	217	244	NO	YES	NO	20ML	NO	YES	11	NO	NO
39	Mariyammal	26	2	1	GALL STONES	LAP	3	3	1.3	222	266	NO	YES	NO	20ML	NO	YES	8	NO	NO
40	cinna	37	2	1	GALL STONES	LAP TO OPEN	1	9	1.2	123	234	NO	YES	NO	50ML	NO	YES	14	NO	NO

SNO	NAME	AGE	SEX	SYMPTOM	PREOP DIAGNOSIS	SURGERY LAP/ OPEN	POST SYMPTOM	L	F	T		CBD EXPLRATION	DRAIN USED	BILIARY LEAKAGE	DRAINS COLLECTION	BILEDUCT INJURY	MANAGMENT	HOSPITAL STAY	BILE DUCT REPAIR	MORTALITY
								TB	DB	ALP	SGOT									
41	Thenmozhi	35	2	1	GALL STONES	LAP	1	10	1.3	49	21	NO	NO	NO	15ML	NO	YES	10	NO	NO
42	Vadivel	28	2	2	GB PERFORATION	OPEN	2	1.2	0.7	114	129	YES	YES	NO	150ML	NO	YES	18	NO	NO
43	Valliammal	38	2	1	GALL STONES	LAP	4	4	17	100	111	NO	YES	NO	50ML	NO	YES	10	NO	NO
44	Verrammal	64	2	1	CHOLECYSTITIS	OPEN	3	2	1	86	90	YES	YES	YES	1250ML	YES	NO	12	YES	NO
45	Yamunabai	45	2	1	GALL STONES	LAP	1	3	3.9	97	96	NO	YES	NO	100 ML	NO	YES	10	NO	NO
46	Venkamesh	48	1	1	GALL STONES	LAP	3	1.7	3	12	10	NO	YES	NO	50ML	NO	YES	10	NO	NO
47	Madhavan	32	1	2	CHOLECYSTITIS	OPEN	1	3.4	5	95	57	NO	NO	YES	800ML	NO	YES	9	NO	NO
48	Pethang	40	1	1	GALL STONES	LAP	1	5.4	3	89	86	NO	YES		50ML	NO	YES	11	NO	NO
49	Bazilath	49	2	2	CHOLECYSTITIS	OPEN	3	1.3	4.9	49	51	YES	YES	YES	750ML	NO	YES	10	NO	NO
50	Chandrabose	35	1	1	GALL STONES	LAP	2	0.7	1	97	76	NO	YES	NO	100ML	NO	YES	10	NO	NO
51	Geetha	58	2	2	CHOLECYSTITIS	OPEN	1	17	3	110	25	YES	YES	NO	200ML	NO	YES	12	NO	NO
52	Ibhrahim	40	1	1	GALL STONES	LAP	2	1	9	130	80	NO	YES	NO	100ML	NO	YES	10	NO	NO
53	Indirani	45	2	1	CHOLECYSTITIS	OPEN	4	3.9	1.1	95	75	NO	YES	YES	700ML	NO	E+S	10	NO	NO
54	Viinnarasi	40	1	1	GALL STONES	LAP	3	3	1.2	93	100	NO	YES	NO	50ML	NO	YES	16	NO	NO
55	Venkatachalapathy	42	1	1	GALL STONES	LAP	1	5	1.1	45	42	NO	YES	NO	50ML	NO	YES	12	NO	NO
56	Usharani	42	2	3	EMPYEMA	OPEN	1	3	1.3	67	89	NO	YES	NO	350ML	NO	YES	11	NO	NO
57	Selvaraj	33	1	1	GALL STONES	LAP	2	4.9	2.0	224	234	NO	NO	YES	600ML	NO	YES	13	NO	NO
58	Sagadurna	45	2	3	MUCOCELE	OPEN	4	1	1.2	217	244	NO	YES	NO	20ML	NO	YES	11	NO	NO
59	Muthukrishnan	26	2	1	GALL STONES	LAP	3	3	1.3	222	266	NO	YES	NO	20ML	NO	YES	8	NO	NO
60	Jothimani	37	2	1	GALL STONES	LAP TO OPEN	1	9	1.2	123	234	NO	YES	NO	50ML	NO	YES	14	NO	NO

SNO	NAME	AGE	SEX	SYMPTOM	PREOP DIAGNOSIS	SURGERY LAP/ OPEN	POST SYMPTOM	L	F	T		CBD EXPLRATION	DRAIN USED	BILIARY LEAKAGE	DRAINS COLLECTION	BILEDUCT INJURY	MANAGMENT	HOSPITAL STAY	BILE DUCT REPAIR	MORTALITY
								TB	DB	ALP	SGOT									
61	Venkat	35	1	1	GALL STONES	LAP	1	10	1.3	49	21	NO	NO	NO	15ML	NO	YES	10	NO	NO
62	Pradeep	28	1	2	CHOLECYSTITIS	OPEN	2	1.2	0.7	114	129	YES	YES	NO	150ML	NO	YES	18	NO	NO
63	Thivagar	38	1	1	GALL STONES	LAP	4	4	17	100	111	NO	YES	NO	50ML	NO	YES	10	NO	NO
64	Vijayakumar	64	1	1	CHOLECYSTITIS	OPEN	3	2	1	86	90	YES	YES	YES	1350ML	YES	NO	12	YES	NO
65	Hasan kadhar	45	1	1	GALL STONES	LAP	1	3	3.9	97	96	NO	YES	NO	100 ML	NO	YES	10	NO	NO
66	Fazlur	48	1	1	GALL STONES	LAP	3	1.7	3	12	10	NO	YES	NO	50ML	NO	YES	10	NO	NO
67	Praveen	32	1	2	CHOLECYSTITIS	OPEN	1	3.4	5	95	57	NO	NO	YES	800ML	NO	YES	9	NO	NO
68	nishar	40	1	1	GALL STONES	LAP	1	5.4	3	89	86	NO	YES		50ML	NO	YES	11	NO	NO
69	Verrammal	49	2	2	CHOLECYSTITIS	OPEN	3	1.3	4.9	49	51	YES	YES	YES	750ML	NO	YES	10	NO	NO
70	Yamunabai	35	2	1	GALL STONES	LAP	2	0.7	1	97	76	NO	YES	NO	100ML	NO	YES	10	NO	NO
71	Venkamesh	58	2	2	CHOLECYSTITIS	OPEN	1	17	3	110	25	YES	YES	NO	200ML	NO	YES	12	NO	NO
72	Madhavan	40	2	1	GALL STONES	LAP	2	1	9	130	80	NO	YES	NO	100ML	NO	YES	10	NO	NO
73	Pethang	45	2	1	CHOLECYSTITIS	OPEN	4	3.9	1.1	95	75	NO	YES	YES	700ML	NO	YES	10	NO	NO
74	Bazilath	40	11	1	GALL STONES	LAP	3	3	1.2	93	100	NO	YES	NO	50ML	NO	YES	16	NO	NO
75	Chandrabose	42	2	1	GALL STONES	LAP	1	5	1.1	45	42	NO	YES	NO	50ML	NO	YES	12	NO	NO
76	Geetha	42	2	3	EMPYEMA	OPEN	1	3	1.3	67	89	NO	YES	NO	350ML	NO	YES	11	NO	NO
77	Ibhrahim	33	1	1	GALL STONES	LAP	2	4.9	2.0	224	234	NO	NO	YES	600ML	NO	YES	13	NO	NO
78	Indirani	45	2	3	MUCOCELE	OPEN	4	1	1.2	217	244	NO	YES	NO	20ML	NO	YES	11	NO	NO
79	Viinnarasi	26	2	1	GALL STONES	LAP	3	3	1.3	222	266	NO	YES	NO	20ML	NO	YES	8	NO	NO
80	Venkatachalapathy	37	2	1	GALL STONES	LAP TO OPEN	1	9	1.2	123	234	NO	YES	NO	50ML	NO	YES	14	NO	NO

SNO	NAME	AGE	SEX	SYMPTOM	PREOP DIAGNOSIS	SURGERY LAP/ OPEN	POST SYMPTOM	L	F	T		CBD EXPLRATION	DRAIN USED	BILIARY LEAKAGE	DRAINS COLLECTION	BILEDUCT INJURY	MANAGMENT	HOSPITAL STAY	BILE DUCT REPAIR	MORTALITY

								TB	DB	ALP	SGOT									
81	Pachaiyammal	35	2	1	GALL STONES	LAP	1	10	1.3	49	21	NO	NO	NO	15ML	NO	YES	10	NO	NO
82	Pechii	28	2	2	CHOLECYSTITIS	OPEN	2	1.2	0.7	114	129	YES	YES	NO	150ML	NO	YES	18	NO	NO
83	Thangam	38	2	1	GALL STONES	LAP	4	4	17	100	111	NO	YES	NO	50ML	NO	YES	10	NO	NO
84	Cinnathayee	64	2	1	CHOLECYSTITIS	OPEN	3	2	1	86	90	YES	YES	YES	1350ML	YES	NO	12	YES	NO
85	Selvi	45	2	1	GALL STONES	LAP	1	3	3.9	97	96	NO	YES	NO	100 ML	NO	YES	10	NO	NO
86	Thennarasi	48	2	1	GALL STONES	LAP	3	1.7	3	12	10	NO	YES	NO	50ML	NO	YES	10	NO	NO
87	Porkodi	32	2	2	CHOLECYSTITIS	OPEN	1	3.4	5	95	57	NO	NO	YES	800ML	NO	E+S	9	NO	NO
88	Ponmanii	40	2	1	GALL STONES	LAP	1	5.4	3	89	86	NO	YES		50ML	NO	YES	11	NO	NO
89	poolandevi	49	2	2	CHOLECYSTITIS	OPEN	3	1.3	4.9	49	51	YES	YES	YES	750ML	NO	E	10	NO	NO
90	Verrammal	35	2	1	GALL STONES	LAP	2	0.7	1	97	76	NO	YES	NO	100ML	NO	YES	10	NO	NO
91	Yamunabai	58	2	2	CHOLECYSTITIS	OPEN	1	17	3	110	25	YES	YES	NO	200ML	NO	YES	12	NO	NO
92	Venkamesh	40	2	1	GALL STONES	LAP	2	1	9	130	80	NO	YES	NO	100ML	NO	YES	10	NO	NO
93	Madhavan	45	2	1	CHOLECYSTITIS	OPEN	4	3.9	1.1	95	75	NO	YES	YES	700ML	NO	YES	10	NO	NO
94	Pethang	40	1	1	GALL STONES	LAP	3	3	1.2	93	100	NO	YES	NO	50ML	NO	YES	16	NO	NO
95	Bazilath	42	1	1	GALL STONES	LAP	1	5	1.1	45	42	NO	YES	NO	50ML	NO	YES	12	NO	NO
96	Chandrabose	42	1	3	EMPYEMA	OPEN	1	3	1.3	67	89	NO	YES	NO	350ML	NO	YES	11	NO	NO
97	Geetha	33	1	1	GALL STONES	LAP	2	4.9	2.0	224	234	NO	NO	YES	600ML	NO	YES	13	NO	NO
98	Ibhrahim	45	2	3	MUCOCELE	OPEN	4	1	1.2	217	244	NO	YES	NO	20ML	NO	YES	11	NO	NO
99	Indirani	26	2	1	GALL STONES	LAP	3	3	1.3	222	266	NO	YES	NO	20ML	NO	YES	8	NO	NO
100	Viinnarasi	37	2	1	GALL STONES	LAP TO OPEN	1	9	1.2	123	234	NO	YES	NO	50ML	NO	YES	14	NO	NO

AGE	
SEX	1 MALE
	2 FEMALE
SYMPTOMS	1 VOMITING
	2ABDOMINAL PAIN
	3.EARLY SATIETY
	4FEVER
	5DYSPEPSIA
SURGERY	LAP CHOLEYCYSTECTOMY
	OPENHOLEYCYSTECTOMY

POST OP SYMPTOMS	1 JAUNDICE
	2 FEVER 2
	3 BILIOUS VOMNITING
	4SEPSIS
	5PERITONITIS
	6 CHOLANGITIS
MANAGMENT	
YES	CONSERVATIVE
E	E-ENDOSCOPIC SPHINTEROTOMY
E+S	SPHINTEROTOMY+STENTING
	BILE DUCT REPAIR

ONSET OF SYMPTOMS	1 TO 7TH DAY
	8 TO 15 TH DAY
DRAIN COLLECTION	MINIMAL
	<200
	200 TO 500
	500 TO 1000
	>1000 ML