"A CLINICAL STUDY AND MANAGEMENT OF CHOLELITHIASIS"

Dissertation submitted to

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TAMIL NADU

With partial fulfillment of the regulations for the award of the degree of

BRANCH - I M.S(GENERAL SURGERY)

APRIL 2019

Government Kilpauk Medical College

Chennai

April -2019

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation thesis **"A CLINICAL STUDY AND MANAGEMENT OF CHOLELITHIASIS**" is a bonafide and genuine research work carried out by me in the Department of General Surgery, Government Kilpauk Medical and Hospital, Chennai-10 under the guidance of our Chief **Prof. Dr.B. SANTHI M.S.,** Government Royapettah Hospital, Kilpauk Medical College, Chennai.

This dissertation is submitted to **THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI** in partial fulfillment of the University regulations for the award of M.S degree (General Surgery) Branch I, examination to be held in APRIL 2019.

Date: DR .R. GOPI,

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DECLARATION

I **Dr. R. GOPI,** declare that this study on **"A CLINICAL STUDY AND MANAGEMENT OF CHOLELITHIASIS"**at the Department of Surgery, Govt. Royapettah Hospital during the period from Febeuary2018 to July 2018. I also declare that this Bonafide work a part of this work was not submitted by me or any others for any award, degree, diploma to any other College, 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.

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LIST OF ABBREVIATIONS

CCK - Cholecystokinin

CBD - Common bile duct

US - Ultrasound

GB - Gallbladder

IDA - Imminodiacetic acid

ESWL - Extra corporeal shock wave lithotripsy

MTBE - Methyl tert butyl ether

OCP - Oral contraceptive pill

ECG - Electro cardio gram

LFT - Liver function test

Lap -Laparoscopy

OPD - Out Patient Department

SGOT - Serum Glutamic Oxaloacetic Transaminase

SGPT - Serum Glutamic Pyruvic Transaminase

BT - Bleeding Time

CT - Clotting Time

PT - Prothrombin Time

SG - Stone in gallbladder

SS - Solitary Stone in gallbladder

MS - Multiple stone in gallbladder

BDS - Common Bile duct stone

CDCA - Chenodeoxycholic acid

UDCA - Ursedeoxycholic acid

TG -Thickening ofgallbladde

Yrs – Years

NS – Not significant

CONTENTS

INTRODUCTION

Among the many distinguished names in Hindu medicine is that of SUSHRUTA, The Father of Indian surger.Hecompiled the surgical knowledge of his time in his classic –SushrataSamhitha‖. It isbelieved that this classicwascompiledbetween800B.C.and400A.D.Hedescribedjaundiceas–pitta-ashmarijanya‖ meaning a jaundice caused by stone in bile.[1]It was also known that such jaundice could be caused by wrongdiet.

6% of prevelance gall stone was found in india.Diagnosis of gallstone is by proper history and physical examination and combining it with appropriate investigations. With the help of ultrasound we can easily identify gall stones.

Because of increase incident of Gall stones and its variable presentations in there is a need for study which can provide prevalence, clinical presentations and management outcomes.

AIMS AND OBJECTIVES

1. To study the age and sexdistribution.

2. To study the various modes ofpresentation.

3. To study safety and efficacy of laparoscopic cholecystectomy in patients of cholelithiasis by comparing with results of open cholecystectomy by comparing use of post-operative pain ,use of analgesia, post-operative hospital stay, wound infection

REVIEW OF LITERATURE

HISTORICAL ASPECTS

Gallstones were described before the modern era of cholecystectomy by Langenbuch in the late 19th century. He widened the understanding of gallstone pathology and performed the first successful cholecystectomy.

First elective cholecystectomy was done by Bobbs in 1867. First successful cholecystectomy was done by Karl Langenbuch on July 15th 1882 in Berlin on a male patient suffering from biliary colic for years. First cholecystojejunostomy for CBD obstruction by Von Winiwarter in 1882 and First successful choledochotomy by Courvoisier in 1882. First successful choledochojejunostomy was done by Sprengel in 1891[7]. First hepaticoduodenostomy by W.J.Mayo in 1905.[8]

First time accurate diagnosis of gallbladder disease was demonstrated by Graham and Cole by oral cholecystography. First PTC was done by Huard and Doxun in 1937.

The Endoscopic Retrograde Cholengiopancreatography was first performed by Mecune in 1937. Mirizzi introduced operative cholangiography in 1937 in Argentina. First gallstones dissolution in man using CDCA was reported by Miyo group in 1972. Makino reported gallstones dissolution by UDCA.

The surgical techniques started to evolve in the late 1800, John Bobbs was an Indiana surgeon and others attempted to perform cholecystolithotomy, removing the stone from the gallbladder and leaving the organ in situ. First percutaneous cholecystolithotomy by Akiyama et al in 1985, Kerlan et al in 1985.Cope et al in 1990 removed the smaller calculi by wire baskets, fragmentation of larger calculi may be done with electro hydraulic or laser mediated intracorporal lithotripsy by Burhenne et al in 1975 and Pinacus et al in 1989.Combined surgical and radiological intervention [mini cholecystostomy] was described by Burhenne et al in 1985.

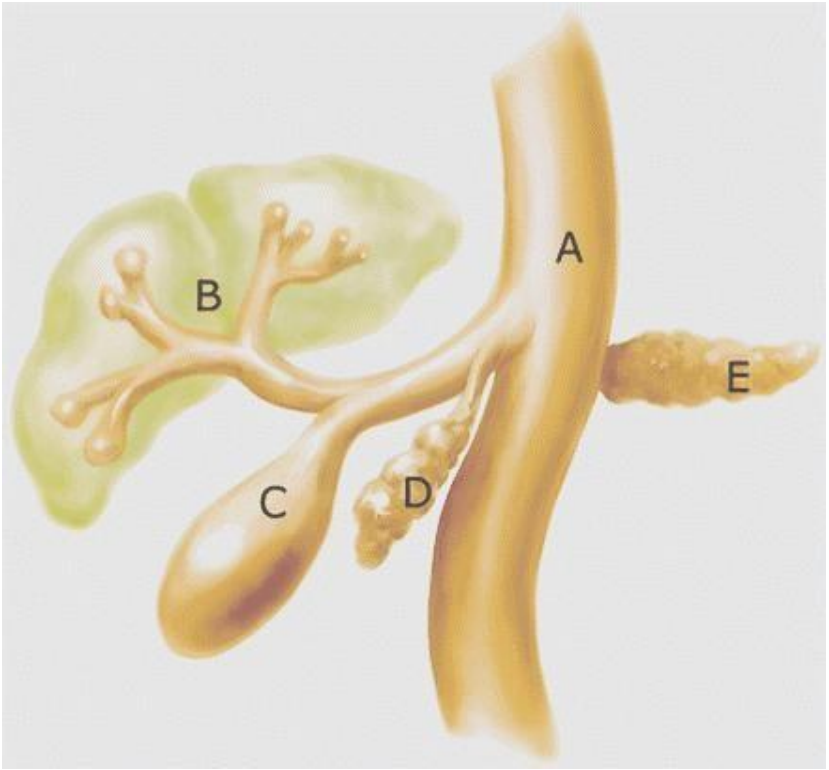
In 1985, first laparoscopicallyassisted cholecystectomy was performed by Muhe in Boblingen,

Cadiere and colleagues reported the first successful clinical implementation of telerobotics in 1998 when they accomplished a laparoscopic cholecystectomy using a prototype of the

Da Vinci robotic surgical system.[10]

EMBRYOLOGY

The genesis of the extrahepatic biliary duct system and gallbladder may, perhaps, be the responsibility distal portion of the hepatic diverticulum. By the end of the 4th week, it has produced the cystic gallbladder primordium. The common bile duct and the hepatic ducts may be seen at theweek. The solid stage of the ducts takes place during the 5th week. The ducts elongate to reach progressively forming at this time. Slow ductal recanalization occurs approximately from the 6th weeks. Human fetal gallbladder contractility in the second half of pregnancy has beenphysiological role is unknown.3



Drawing of the normal embryologic development of the gallbladder and bile ducts illustrates the foregut (*A*), the cranial end of the hepatic diverticulum, which represents pars hepatica (*B*) and the cystic diverticulum (*C*). The ventral (*D*) and dorsal (*E*) pancreas are alsodemonstrated.

SURGICAL ANATOMY

ANATOMY OF EXTRAHEPATIC BILIARY SYSTEM

The gallbladder lies on the underside of the liver in the main liverscissura at the junction of the right and left lobes of the liver. The relationship of the gallbladder to the liver varies between being embedded within the liver substance to being suspended by a mesentry. It is a pear-shaped structure, 7.5–12 cm long, with a normal capacity of about 25–30 mL. The anatomical divisions are a fundus, a body and a neck that terminates in a narrow infundibulum. The muscle fibres in the wall of the gallbladder are arranged in a criss-cross manner, being particularly well developed in its neck. The mucous membrane contains indentations of the mucosa that sink into the muscle coat; these are the crypts of Luschka.

The cystic duct is about 3 cm in length but the length

is variable. The lumen is usually 1–3 mm in diameter. The mucosa of the cystic duct is arranged in spiral folds known as the valves of Heister and the wall is surrounded by a sphincteric structure called the sphincter of Lütkens. The cystic duct joins the supraduodenal segment of the common hepatic duct in 80% of cases, however the anatomy may vary and the junction may be much lower in the retroduodenal or even retropancreatic part of the bile duct. Occasionally, the cystic duct may join the right hepatic duct or even a right hepatic sectorial duct .

The common hepatic duct is usually less than 2.5 cm long and is formed by the union of the right and left hepatic ducts.

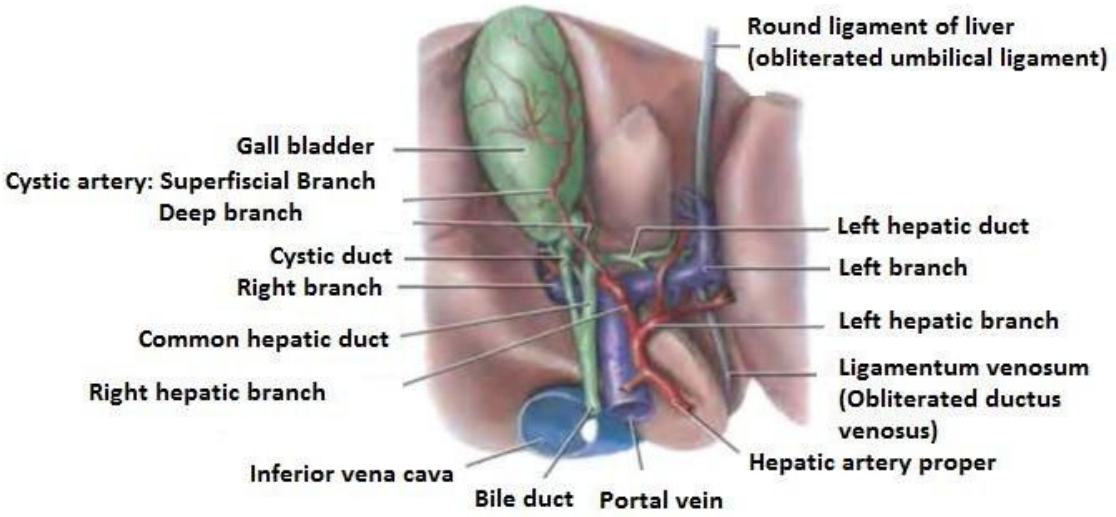
The common bile duct is about 7.5 cm long and formed by the junction of the cystic and common hepatic ducts. It is divided into four parts:

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

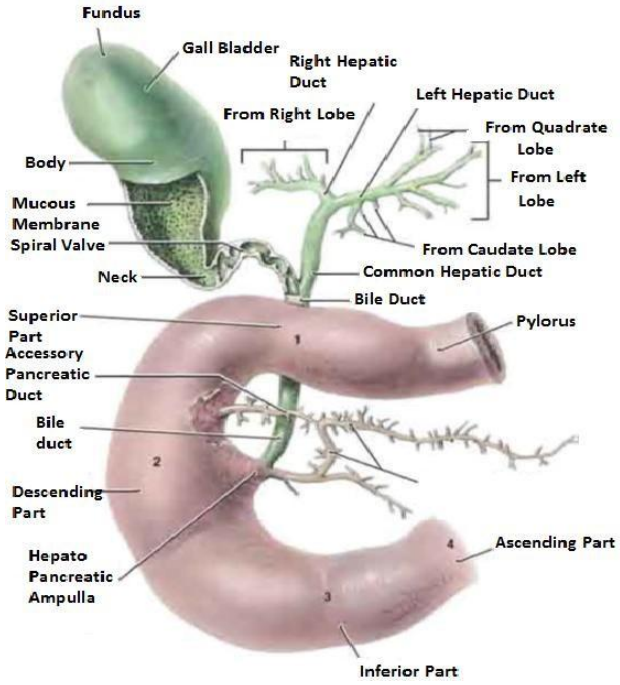
The cystic artery, a branch of the right hepatic artery, usually arises behind the common hepatic duct

Occasionally, an accessory cystic artery arises from the gastroduodenal artery. In 15% of cases the right hepatic artery and/or the cystic artery crosses in front of the common hepatic duct and the cystic duct.

Calot's triangle, or the hepatobiliary triangle, was initially described by Calot as the space bordered by the cystic duct inferiorly, the common hepatic duct medially and the superior border of the cystic artery. This has been modified in contemporary literature to be the area bound superiorly by the inferior surface of the liver, laterally by the cystic duct and the medial border of the gallbladder and medially by the common hepatic duct. It is an important surgical landmark as the cystic artery usually can be found within its boundaries and should be identified by surgeons performing a cholecystectomy to avoid damage to the extrahepatic biliary system.



ANATOMY OF GALL BLADDER, INFERIOR VIEW



anatomy of the gallbladder, biliary radicals,

pancreatic duct and the hepatopancreatic ampulla

BLOOD SUPPLY OF EXTRAHEPATIC BILIARY SYSTEM:

The blood supply to the extrahepatic biliary tree originates distally from gastroduodenal, retroduodenal, and posterosuperiorpancreatoduodenal arteries andproximally from the right hepatic and cystic arteries. These arteries supply the common bile and common hepatic ducts through branches running parallel to the duct in the 3- and 9-o'clock positions. The extrahepatic biliary tree is vulnerable to ischemic injury. To avoid disrupting the fragile inconstant blood supply to the duct, it is important not to strip the investing areolar tissue around it during dissection and isolation. Ischemia of the bile duct will not be readily evident at time of dissection but can result in biliary stricture or leakpostoperatively.

The gallbladder is supplied by a single cystic artery.. The cystic artery may originate from the left hepatic,common hepatic, gastroduodenal, or superiormesenteric arteries. The cystic arterymay arises from the right branch of the hepatic artery, passes posterior to the common hepatic duct and over the cystic duct to the superior aspect of the gallbladder neck, on which it descends and divides into superficial and deep branches. The cystic artery is an end artery and its occlusion is followed by the gangrene of the gallbladder.

VENOUS DRAINAGE:

Cystic veins draining the gallbladder vary. Those from its superior surface are in the alveolar tissue between the gallbladder and liver, usually entering the through the vertical fossato join the hepatic veins. The remainder form one or two cystic veins which commonly enter the liver either directly or after joining the veins draining the hepatic ducts and upper bile duct. Rarely a single or double cystic veins drain into the right portal branch.

LYMPHATIC DRAINAGE OF GALLBLADDER: Proximally the lymphatic channels of the gallbladder communicate with those of Glisson‘s capsule of the liver which in turn drain into the thoracic duct through several channels.Distally the lymphatics from gallbladder and extrahepatic bile duct drain into the cystic lymph node, which is situated near the cystic artery origin from the right hepatic artery. Efferent vessels from the cystic nodedrain into the nodes along the free border of hepatoduodenalligament and to the superior pancreatic duodenal nodes inferiorly and hilar lymph nodes superiorly. All these in turn drain into celiac lymph nodes.

INNERVATION:Parasympathetic (vagal)

Preganglionic sympathetics and visceral afferent fibers for pain reach the celiac plexus by way of the greater thoracic splanchnic nerves.

Fibers from the right phrenic nerve travel by way of the phrenic, celiac, and hepatic plexuses to reach the gallbladder. Many of these fibers are afferent and may account for the pain referred to the right hypochondrium and radiating to the back between the shoulder blades in some patients with gallbladder diseases.

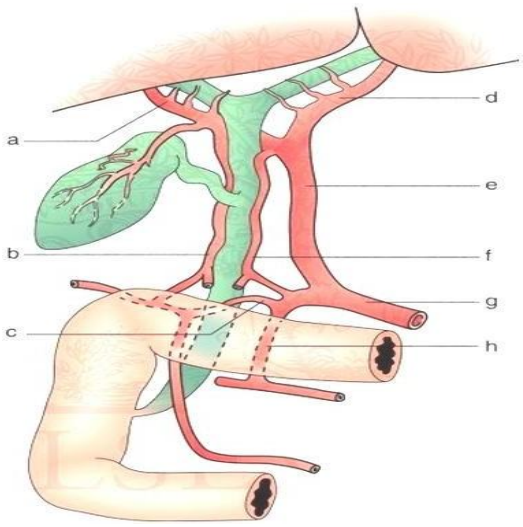
COMMON ANOMALIES AND VARIATIONS

1. Absent gallbladder – extremely rare, autopsy incidence of 0.03% have been reported

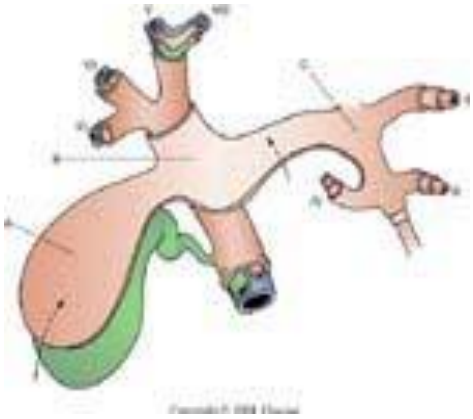
2. Variation in size and shape ofgallbladder.
   - Bilobedgallbladder.
   - Funduldivertculum.
   - Phrygian cap.
   - Hour glassgallbladder

3. Variation inposition
   - Left sided gallbladder, floatinggallbladder.

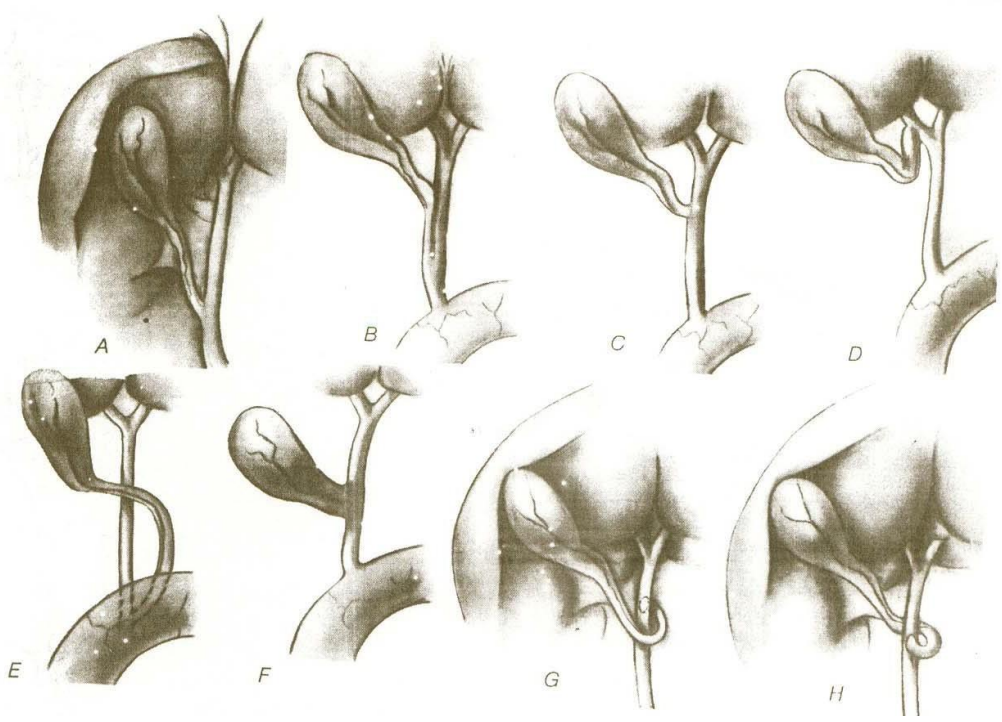
4. Double gallbladder, duplication of gallbladder with two separate cavities and two separate cystic ducts has an incidence of approximately 1 in 4000. Pathological process such as cholelithiasis and cholecystitis may involve one organ while the other isspared.



Blood supply to the common bile duct and common hepatic duct is illustrated: a, right hepatic artery; b, 9 o'clock artery ; c, retroduodenal artery; d, left hepatic artery; e, proper hepatic artery; f, 3 o‘clock artery; g, common hepatic artery; h, gastroduodenal artery.

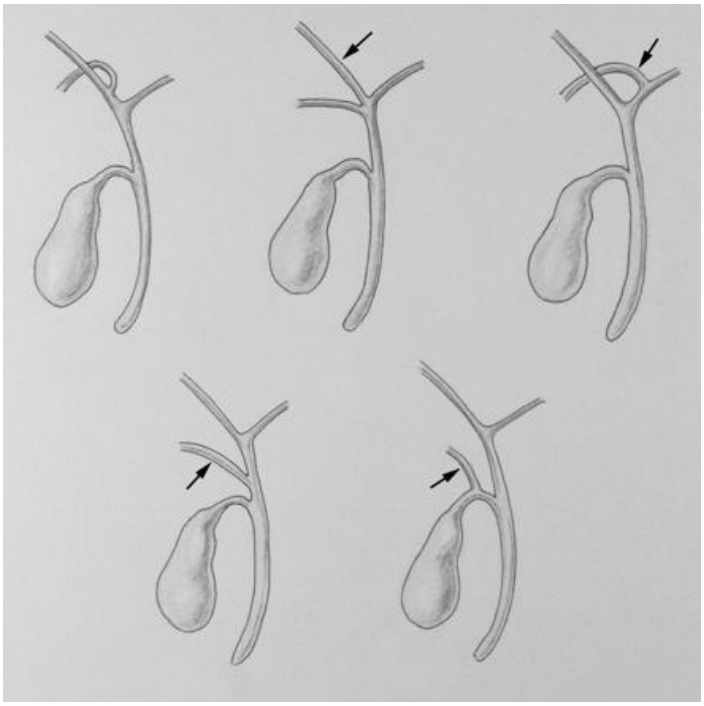


The plate system is illustrated, including the cystic plate between the gallbladder and the liver **(A)** , the hilar plate at the biliary confluence at the base of segment IV **(B)** , and the umbilical plate above the umbilical portion of the portal vein (**C**). The *arrows* show the plane of dissection of the cystic plate for cholecystectomy and the hilar plate for exposure of the hepatic duct confluence and the main left hepatic duct.



Variation of the cystic ducts. A. Low junction between cystic duct and common hepatic duct. B. Cystic duct adherent to the common hepatic duct. C. High junction between cystic duct and common hepatic duct. D. cystic duct drains into the right hepatic duct. E. Cystic duct that joins the common hepatic duct. F. Absence of cystic duct. G. Cystic duct crosses anterior to the common hepatic duct and joins it posteriorly. H. Cystic duct courses posteriorly to common hepatic duct and joins it anteriorly.

Anomalies of the gallbladder. A. Double gallbladder with single cystic duct. B. Bilobed gallbladder. C. Intrahepatic gallbladder. D. Left sidedgallbladder.



Normal and variant biliary ducts. A, Normal biliary tree. B, Trifurcation of biliary duct (arrow). C, Right dorsocaudal branch (arrow) draining into left hepatic duct. D, Aberrant right hepatic duct (arrow) emptying into common hepatic duct. E, Aberrant right hepatic duct (arrow) draining into cystic duct.

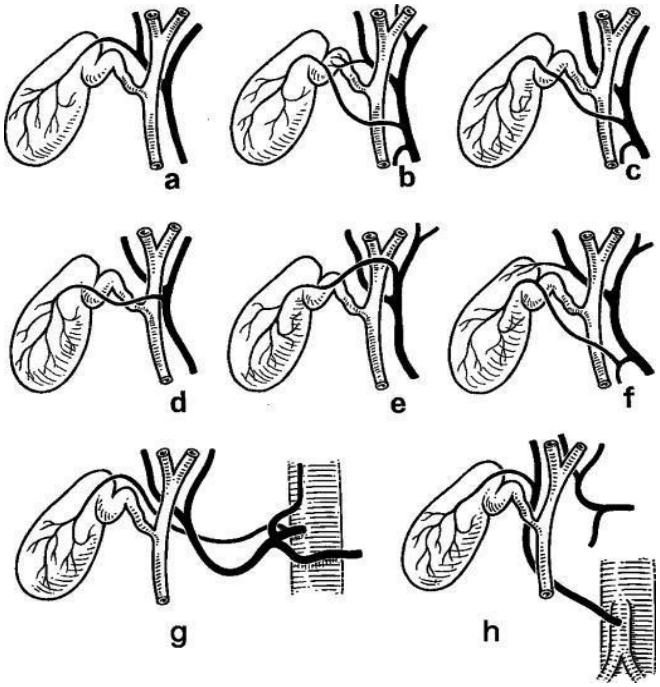
TRIANGLE OF CHOLECYSTECTOMY

Calots defined a triangle of anatomical area formed by the common hepatic duct medially, the cystic duct laterally and the cystic artery superiorly. The present concept of the triangle of cholecystectomy has for its upper limit not the cystic artery.but the inferior surface of the liver. This triangle is of surgical importance because a number of important structures pass through it.Therefore during cholecystectomy there is a need to identify all structure within the triangle to prevent complications.

VARIATIONS IN ARTERIAL ANATOMY

The cystic artery supplies the cystic duct with one or more small arterial branches which have been referred to as Calot‘s arteries by Hugh and Kelly. These branches may be a source of troublesome bleeding during cholecystectomy. Anatomical variations of the hepatic and cystic arteries are recognized in approximately 50% of individuals. Benson and Page has defined 3 surgically significant variations in arterial anatomy.

1. Accessory on double cystic artery – seen in approximately 15%to20%
2. Caterpillar hump of right hepatic artery seen in 5% to 15% of individuals.. This is the most dangerous anomaly, the tortuosity is known as the caterpillar turn or Moynihan‘s hump.
3. Cystic artery may lies ventral to common hepatic or common bile duct.



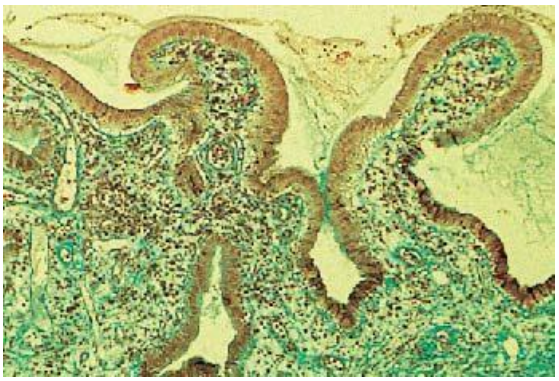
The main variations of the cystic artery: typical course (a); double cystic artery (b); cystic artery crossing anterior to main bile duct (c); cystic artery originating from the right branch of the hepatic artery and crossing the common hepatic duct anteriorly (d); cystic artery originating from the left branch of the hepatic artery (e); cystic artery originating from the gastroduodenal artery (f); cystic artery arising from the celiac axis (g); cystic artery originating from a replaced right hepatic artery (h).

HISTOLOGY

GALLBLADDER

The mucosa is yellowish-brown and elevated into the rugae with a honeycomb appearance. In section, projections of the mucosa into the gallbladder lumen resemble intestinal villi, but these are not fixed structures and the surface flattens as the gallbladder fills with bile.

The epithelium is a single layered columnar epithelium with apical microvilli. Goblet cells are absent. Beneath it is a thin fibromuscular layer composed of fibrous tissue mixed with smooth muscles which are arranged loosely in longitudinal, circular and obliquebundles.



Microscopy of gallbladder wall

PHYSIOLOGY

Bile is made up of bile salts,bile pigments and other substances dissolved in an alkaline medium. About 500 ml is secreted daily. The glucuronides of the bile pigments, bilirubin and biliverdin are responsible for golden yellow colour.

Composition of hepatic bile

Water	97.0 %
Bile salts	0.7%
Bile pigments	0.2%
Cholesterol	0.06 %
Inorganic salts	0.7%
Fatty acids	0.15 %
Lecithin	0.1%
Fat	0.1%
Alkaline phosphatase	-------

REGULATION OF BILIARY SECRETION:

The tone of sphincter of Oddi decreases when food enters mouth. Fatty acids and amino acids in the duodenum release CCK, which cause gallbladder contraction. Substances that cause contraction of gallbladder are called cholagogues.The production of bile is increased by stimulation of the vagus nerves and by the hormone secretin, which increases water and HCO3- content of bile. Substances that increase the secretion of bile are choleretics. Bile salts themselves are important choleretics.A gallbladder helps in fluid transport and its regulation. Studies have demonstrated that the gallbladder concentrates hepatic bile by selective reabsorption of bile constituents. Sodium and chloride ion are absorbed from the gallbladder by both active and transport mechanism, water absorption is thought to be passive. The secretion of the water and the electrolyte by the gallbladder mucosa is an active process which can take place against hydrostatic and osmoticgradients.

There are number of gastrointestinal peptides such as cyclic AMP, vasoactive intestinal peptides and other peptides such as glucogon, cholecystokinin, neurotensin, bombaysin, motilin and stomatostatin. During fasting the normal bile gallbladder absorbs fluid at a rate corresponding to one third of fasting gallbladder volume. After feeding there is a reversal of the direction of gallbladder transport from net absorption to net secretion into the gallbladder lumen. The net water transport across the gallbladder may be influenced by both humoral and autonomic nerves.

The flow of bile into the gallbladder is modulated by hepatic secretory pressure, Sphincter of Oddi and cystic duct resistance. Only 50% of the secretory hepatic bile enters the gallbladder during fasting, the remaining bile passes into the duodenum. The CCK causes rise in the intra luminal gallbladder pressure to about a cm of water above that in the CBD, resulting in the secretion of bile into theduodenum.

Protein entering the duodenum also produces gallbladder contraction but carbohydrates produces only minimum effect. Cholecystokinin released from the proximal small intestine produces gallbladder contraction.The gallbladder contraction induced by the intraduodenal influence of fat correlates directly with the level of circulatingCCK.

The role of autonomic nerve in regulating gallbladder volume is not clear.

PATHOGENESIS OF GALLSTONES

Gallstones can be divided into three main types: cholesterol,pigment (brown/black) or mixed stones. In the USA andEurope 80% are cholesterol or mixed stones, whereas in Asia80% are pigment stones. Cholesterol or mixed stones contain51–99% pure cholesterol plus an admixture of calcium salts,bile acids, bile pigments and phospholipids.Cholesterol, which is insoluble in water, is secreted fromthe canalicular membrane in phospholipid vesicles. Whethercholesterol remains in solution depends on the concentrationof phospholipids and bile acids in the bile, and on the type ofphospholipid and bile acid. Micelles formed by the phospholipid hold cholesterol in a stable thermodynamic state. Whenbile is supersaturated with cholesterol or bile acid concentrationsare low, unstable unilamellar phospholipid vesicles form,from which cholesterol crystals may nucleate, and stones may form. The process of gallstone formation is complex and many areas remain unclear. Obesity, high-caloricdiets and certain medications (e.g. oral contraceptives) canincrease secretion of cholesterol and supersaturate the bile,increasing the lithogenicity of bile. Resection of the terminalileum, which diminishes the enterohepatic circulation, willdeplete the bile acid pool and result in cholesterol supersaturation.Nucleation of cholesterol monohydrate crystals frommultilamellar vesicles is a crucial step in gallstone formation. Abnormal emptying of the gallbladder may aid the aggregationof nucleated cholesterol crystals; hence, removing gallstoneswithout removing the gallbladder

inevitability leads togallstone recurrence.

Pigment stone is the name used for stones containing<30% cholesterol. There are two types: black and brown.Black stones are largely composed of an insoluble bilirubinpigment polymer mixed with calcium phosphate and calciumbicarbonate. Overall, 20–30% of stones are black. The incidence rises with age. Black stones are associated with haemolysis,usually hereditary spherocytosis or sickle cell disease. Forreasons that are unclear, patients with cirrhosis have a higherinstance of pigmented stones.Brown pigment stones contain calcium bilirubinate, calciumpalmitate and calcium stearate, as well as cholesterol.Brown stones are rare in the gallbladder. They form in the bile duct and are related to bile stasis and infected bile. Stoneformation is related to the deconjugation of bilirubin deglucuronideby bacterial β-glucuronidase. Insoluble unconjugatedbilirubinate precipitates. Brown pigment stones arealso associated with the presence of foreign bodies within thebile ducts such as endoprostheses (stents) or parasites such as*Clonorchis sinensis*and *Ascarislumbricoides*.

EPIDEMIOLOGY OF GALLSTONES

Gallstones are a major cause of morbidity and mortality throughout the world.

AGE

At least 10 percent of adults have gallstones. The prevalence varies with age, sex, and ethnic group. There is an increasing prevalence with age, after the age of 40 about 10 to 15 percent of men and 20 to 40 percent of women have gallstones.

SEX

Female: male ratio of about 2:1 in the younger age groups and the risk of gallstones are also associated with a history of childbearing, estrogen-replacement therapy, and oral-contraceptive use, but not diabetes mellitus.

PLACE

The prevalence of gallstones is especially high in the Scandinavian countries and Chile. North Indians have 7 times higher occurrence of gall stone as compared with south Indians

OBESITY

It is higher in markedly obese persons and in those who lose weight rapidly.There is little agreement about the effect of dietary components on the risk of gallstones. Fasting is normally associated with increased biliary cholesterol saturation and this phenomenon more accentuated in obesity.Obesity also reduces gallbladder emptying.

ESTROGEN AND CHOLESTEROL LOWERING AGENTS

Excess estrogen from pregnancy, hormone replacement therapy, or birth control pills appears to increase cholesterol levels in bile and decrease gallbladder movement, both of which can lead to gallstones. Drugs that lower cholesterol levels in the blood actually increase the amount of cholesterol secreted in bile. This in turn can increase the risk of cholesterol gallstones. Clofibrate increases biliary cholesterol and results in formation of the gall stone.

DIABETIS MELLITUS

Gallbladder atony consequent upon an autonomic neuropathy may favour stone formation in super saturated bile. It has been stated that the diabetes patients have higher incidence of gallstone disease and are particularly prone to complications from there stone.

FASTING

Fasting decreases gallbladder movement causing the bile to become over concentrated with cholesterol, which can lead to gallstones. No clear relationship has been proved between diet and gallstone formation.

CIRRHOSIS OF THE LIVER

Patients with cirrhosis have 3 times greater risk for gallstones than the normal people. The stones are usually of pigment type and probably results from the chronic haemolysis.

VAGOTOMY

Early clinical study suggested that truncalvagotomy was associated with two fold increase in the incidence of gallstones; other studies have failed to confirm this hypothesis. Ultrasonography suggested that truncalvagatomy is associated with dilated gallbladder.[6] Nerve fibres from both vagal nerves merge to form the hepatic plexus which supplies parasympathetic motor nerves to the extra hepatic biliary system.

TOTAL PARENTERAL NUTRITION AND GALLSTONE FORMATION

Symptomatic gallstone disease forms in approximately 45% of patients who are maintained on long term TPN.

INFLAMMATORY BOWEL DISEASE

Patients with ileal dysfunction which is more saturated with cholesterol and patients with jejuno-ileal operation are associated with increased risk of gallstone formation.

Whentheileumisdiseasedorremovedabsorptionofbilesaltsisimpairedandasignificant loss of bile salts will occur, as result of loss of bile salts there will be relative increase in cholesterol leading to the gall stone formation.

MISCELLANEOUS

The prevalence of gallstones in thalassaemia is about 10%, in sickle cell disease is 10% to 40%, and in hereditary spherocytosis is 43% to 66%. Pigment gallstones are reported in 58% of patients with homozygous sickle disease and in 17% of the patients with heterozygous type. Hormonal changes during pregnancy and alteration of gallbladder motility by progesterone are thought to be responsible for the development of gallstones in women. There is no increased risk of morbidity if surgical therapy for biliary disease is carried out in the secondtrimester.There is a controversy over an association between the gallstone and colorectal cancer and gastric cancer.[58] There is an association between hiatal hernia and diverticular disease of the colon and gallstone.

CLINICAL MANIFESTATION

PRESENTATION

Two thirds of gallstones are asymptomatic. Risk factors for stones becoming symptomatic are smoking and parity.Asymptomatic gall stone disease has a benign natural course; the progression of asymptomatic to symptomatic disease is relatively low, ranging from 10 to25%.Some patients may present with non specific symptoms. Stones may cause acute or chronic cholecystitis, biliary colic, pancreatitis or obstructive jaundice.

The most common presenting symptom is intermittent pain below the right ribcage. Pain might radiate to the back, and to the shoulder . Nausea, with or without vomiting and dyspepsia might be present. Certain foods, especially those with high fat content, can provoke symptoms. The patient might experience episodes of acute abdominal pain, called biliary colic. Attacks may be separated by weeks, months, or even years. Once a true attack occurs, subsequent attacks are much more likely. William et al have confirmed biliary pain as the main symptom of gallstone disease. He also confirmed that the gallbladder itself, without stones can produce pain and this pain is relieved in 77% of patients by cholecystectomy.

OBSTRUCTIVE JAUNDICE

Large stone in Hartmann's pouch compresses the common hepatic duct (Mirrizi's syndrome).

Gallstones may also interfere with the flow of digestive fluids secreted from the pancreas into the small intestine, leading to pancreatitis. Prolonged blockage of any of these ducts can cause severe damage to the gallbladder, liver, or pancreas, which can be fatal.

Charcot‘s triad (right upper quadrant pain, fever, and intermittent jaundice) is associated with common bile duct obstruction and cholangitis. Additional symptoms, such as alterations in the mental status and hypotension, indicate Raynaud‘s pentad, a harbinger of worsening ascending cholangitis. Other conditions like peptic ulcer, pancreatitis and hiatus harnia should be ruled out in patient presenting with dyspepsia.

PHYSICAL SIGNS

Discomfort might be elicited on deep palpation of the right upper quadrant of the abdomen. Murphy‘s sign (pain on palpation of the right upper quadrant when the patient inhales) might indicate acute cholecystitis. Other signs of cholecystitis include fever and tachycardia. An enlarged gallbladder may be palpated when a mucocele or empyema of the gallbladder is present, the gallbladder is felt as tense globular swelling projecting downwards and lateral to the right rectus abdominus muscle. Hyperaesthesia between the 9th and 11th ribs posteriarly on the right side is present in acute cholecystitis, is called as Boas‘s sign.

Complete or partial obstruction of the common bile duct. Manifests as jaundice. Severe hemorrhagic pancreatitis occurs in 15% patients and carries a high mortality rate because of multisystem organ failure. In a few patients, the hemorrhagic pancreatic process and bleeding into the fascial plane induce discoloration around the umbilicus (Cullen sign) or the flank (Grey-Turner sign).

COMPLICATIONS OF GALLSTONES

In the gall bladder:

- Acutecholecystitis
- Chroniccholecystitis
- Gangrene
- Perforation
- Empyema
- Mucocele(hydrops)
- Carcinoma

In the bile ducts:

- Obstructivejaundice
- Cholangitis
- Acutepancreatitis

In the intestine:

- Acute intestinal obstruction (_gallstoneileus)

MIRIZZI'S SYNDROME: Mirizzi‘s syndrome is a rare complication of chronic cholecystitis and cholelithiasis occurs in less than 1%. Originally described by Kehr and Ruge, it is later named by Mirizzi in 1948. Mirizzi and others described the presence of four features including a parallel cystic duct; gallstones impacted in the neck of gallbladder or cystic duct, causing common hepatic obstruction; and recurrent cholangitis. It was thought to be a functional disorder of a putative sphincter within the common bile duct. It is now well known that jaundice is caused by external compression of the common bile duct. McSherry in 1982 subclassifiedMirizzi syndrome into two types based on ERCP. Type I being an extensive compression of the common bile duct by a stone in the cystic duct or Hartmann‘s pouch and type II being a cholecystocholedochal fistula resulting from calculus erosion into the common bile duct. Based on this Bear et al suggested a standardized surgical approach to Mirizzi syndrome. A unifying classification has subsequently been proposed by Casendes. Type I lesions are those with external compression of the common bile duct, in type II lesion a cholecystobiliary fistula is present with erosion of less than $1/3^{rd}$ circumference of the bile duct . In type III the fistula involves upto $2/3^{rd}$ of the circumference , type IV being a complete destruction of the common bile duct wall . The patients usually present with acute or chronic cholecystitis or obstructive jaundice. This anatomical distortion does not allow a definitive preoperative diagnosis by ultrasound. A high index of suspicion is required to arrive at a correct diagnosis preoperatively. Direct cholangiography is more informative than USG for diagnosis though USG the best screening procedure. PTC and ERCP are important in comforming the diagnosis.

CARCINOMA GALLBLADDER: Malignant change in the gallbladder is the fifth commonest cause of carcinoma in the gastrointestinal tract. The male to female ratiois 1:4. The majority of cases are associatedwithgallstones.

CHOLANGITIS: Acute cholangitis, which usually presents as a combination of fever, rigors and jaundice (Charcot's triad), is a serious and potentially lethal condition. It is caused by complete or partial biliary obstruction in combination with ascending infection of the biliary tree.Acutesuppurative cholangitis is a rare subgroup in which pus is under tension within the biliary tree, causing a profound illness with Gram-negative septicemia. It requires high-dose antibiotics and urgent decompression of the bile ducts. This may be achieved by passing a small plastic catheter via an endoscope through the Ampulla of Vater above the site of the obstruction.

BILIARY PANCREATITIS: Acute pancreatitis is caused by gallstones in 13-19% of patients. Small gallstones are particularly liable to cause this complication and there is now evidence that an attack is due to the impaction or passage of a stone through the Ampulla of Vater - the data have been obtained from routine examination of feces or from endoscopic appearances. Diagnosis is based on an elevated serum amylase, the ultrasonic demonstration of gallbladder stones and the absence ofalcohol ingestion. Theacceptedmanagement plan for the majority of patients withpancreatitis of biliary origin is early ERCP, with or without endoscopic sphincterotomy and laparoscopic cholecystectomy

GALLSTONE ILEUS: Gallstone ileus accounts for 1-4% of all cases of intestinal obstruction, with its incidence rising with age of patients. There is often a long delay between onset of symptoms (usually abdominal pain, vomiting, and boweldistension) and proper treatment, with a simple enterolithotomy as one ofthechoice. Atypical gallstone ileus may present as a complication of acute cholecystitis, with continuous vomiting as the only symptom of a subileus.

INVESTIGATIONS

IMAGING STUDIES

ULTRASOUND

Sonography was introduced in the mid 1970‘s. Ultrasound (US) is the simplest and most reliable method for diagnosis of gallstones· US is the most sensitive and specific test for the detection ofgallstones.

US provide information about the size of the common bile duct and hepatic duct and the status of liver parenchyma and the pancreas. Thickening of the gallbladder wall and the presence of pericholecystic fluid are radiographic signs of acute cholecystitis. The limitation of ultrasound in stone detection apart from very small stones, are the difficulty in estimating the size and number of stones and in detecting whether they arecalcified.

Endoscopic ultrasonography is useful for detecting small gallbladder stones missed on transabdominol imaging, especially those located in the neck of the gallbladder, where duodenal gas can obscure the image when scanningpercutaneously.

Multiple gallstones noted within the gallbladder.

ABDOMINAL RADIOGRAPHY

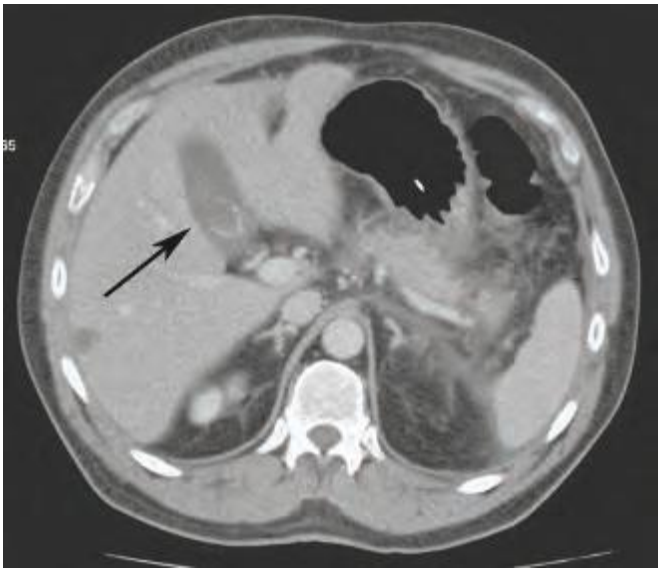
Upright and supine abdominal radiographs are of limited value in identifying gallstones. Approximately 10% of gallstones are radiopaque and can be visualized on plain x-ray. Plane film may show a lucent triradiate appearance resembling a symbol of a Mercedes Benz automobile.

RADIOISOTOPES SCAN

HIDA scan identifies an obstructed gallbladder (eg, gallstone impacted in the neck of the gallbladder).HIDA scan is the most sensitive and specific test for acute cholecystitis. A poorly contracting gallbladder (biliary dyskinesia) might cause the patient's symptoms, and HIDA scan makes the diagnosis.

COMPUTED TOMOGRAPHY

CT scanning often is used in workup of abdominal pain without specific localizing signs or symptoms. CT scanning is not a first-line study for detection of gallstones.



Computed tomography scan demonstrating a gallstone within the gallbladder

PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY

With fluoroscopic , guidance a needle (the Chiba or Okuda needle)is advanced into the liver through the image guidance. The stylet is then removed, and contrast is injected and the needle is slowly withdrawn until contrast starts entering a biliary radical. This investigation is used to know the site of biliary obstruction, dilated bile ducts, biliary enteric or biliary cutaneous fistulas, level of bile leak, and to place the biliary stents.

MAGNETIC RESONANCE IMAGING

Gallstones are easily identifiable on T2W images as well as on MRCP sequences. Choledocholithiasis can be effectively imaged by MRCP.

MRCP can also be obtained to evaluate if retained stones are present post cholecystectomy. If no stones are present on the MRCP this may negate the need for an ERCP.

ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY

This technique remains widely used as both a diagnostic and a therapeutic modality. Using a side-viewing endoscope the ampulla of Vater can be identified and cannulated. Injection of water-soluble contrast directly into the bile duct provides excellent images of the ductal anatomy and can identify causes of obstruction such as calculi or malignant strictures The commonest significant complications are biliary leakage, sepsis and hemorrhage. The complications of ERCP include acute pancreatitis. Necrotising pancreatitis is a very severe complication. The most serious complication is the development of post procedure cholangitis.

Endoscopic retrograde cholangiopancreatography:

partial occlusion of the bile duct by a malignant stricture (arrow).

PEROPERATIVE CHOLANGIOGRAPHY

There are various types of cholangiography performed intraoperatively, in the most common type the cystic duct is opened and a fine polythene catheter is passed through it into the common bile duct for 3 cm. A ligature is tied around the cystic duct and catheter which prevents leakage. The catheter is filled with physiological saline before insertion, so that there is no air bubbles present which would have a similar radiographic appearance to radiolucent gallstone. Instrument and packs which might obscure the radiograph are removed. The table is tilted to 10 degrees right side so that the spine and ducts are not super imposed and 20 degrees head down to ensure contrast flows into the liver. 3 injections of 25% Hypaque are given. A radiographic exposurefollowseachinjection.Anormalcholangiogramissufficientevidentt hatexploration of the CBD is unnecessary. Failure of contrast to enter the duodenum may be due to sphincter spasm rather than organic lesion.

T tube cholangiography is performed 10 days to 14 days after choledochotomy via the T tube. The absence of stones and a normal flow of bile into the duodenum indicate that the T tube can beremoved.

OPERATIVE BILIARY ENDOSCOPY

At the operation a rigid or flexible fibre optic endoscope can be passed into biliary tree. Stone can be identified and removed under direct vision.

TREATMENT

There are various treatments available for treatment of gallstones but cholecystectomy still remains the gold standard.

NON INVASIVE TREATMENT OF GALLSTONES [68]

1. Oral dissolutiontherapy
2. Extracorporeal shock wave lithotripsy.(ESWL)

MINIMALLY INVASIVE GALLBLADDER PROCEDURE

1. Percutaneouscholecystostomy
2. Contact dissolutiontherapy
3. Percutaneouscholecystolithotomy
4. Laparoscopiccholecystectomy

INVASIVE PROCEDURE

1. Open cholecystectomy.

MINIMALLY INVASIVE GALLBLADDER PROCEDURE

PERCUTANEOUS CHOLECYSTOSTOMY.

Percutaneous cholecystostomy is an intervention that may be used in moribund patients who cannot tolerate immediate surgery, particularly in elderly people and high risk patients, for the treatment of acute cholecystitis, empyema and the perforated gallbladder with localized abscess formation. Drainage of the gallbladder produces symptomatic relief. Drainage tube kept and fixed followed by cholecystectomy.

CONTACT DISSOLUTION THERAPY

Various solutions are effective in dissolving stones within the biliary tree when direct asses to the stone are available via a tube. These substances are likely to be effective only in dissolving cholesterol stones. Success with such an approach depends upon the compound‘s ability to dissolve the main constituents of the stone and good contact with thestone.

SODIUM CHLORATE

MONO OCTANOIN

METHYL TERT BUTYL ETHER

PERCUTANEOUS CHOLECYSTOLITHOTOMY

Percutaneous cholecystolithotomy was originally described in 1985 in poor risk patients with acute cholecystitis. After dilatation of the transhepaticcholecystostomy tract, stones are removed in one to five sessions using baskets or forceps. The standard approach is transhepatic, but large cannula which makes stone extraction easier, cause unnecessary trauma to the liver. It is performed under general anaesthesia or local anaesthesia with intravenous sedation and . Percutaneous cholecystography is performed and then the fundus of the gallbladder is punctured with a Kellett needle, using a combination of ultrasononographic and fluoroscopic guidance. A guide is placed in the gallbladder for the entire procedure as a safety measure. The track is dilated to 28-30Fr using Teflon and telescopic metal dilators before inserting an Amplatz Teflon sheath. The gallbladder is inspected with a rigid cholecystoscope and stones of up to 10mm in diameter are flushed out or removed with the forceps. Stones that are too large to pass through the Amplatz sheath can be fragmented by intracorporeal electrohydraulic, laser or ultrasound lithotripsy and removed piecemeal. At the end of the procedure, a Foley‘s catheter is introduced through the Amplatz sheath and placed on free drainage for 10 days. A tubogram is performed and the catheter is removed provided that the biliary tree is free of stones and there is no intraperitoneal extravasation ofcontrast.

CHOLECYSTECTOMY

We must bear in mind that the gallbladder must be removed not because it contains stone, but because it forms them.

Patients having cholelithiasis with acute cholecystitis require cholecystectomy, unless this is impossible because of the patient's

medical condition.

Prophylactic cholecystectomy for gallstones has been recommended in specific groups, such as children, because symptoms develop in almost all patients. It has also been recommended in patients with gallstones and sickle cell disease, because the symptoms of gallstones can mimic those of sickle cell crisis, and elective cholecystectomy is much safer than emergency cholecystectomy in this group.

Incidental cholecystectomy for cholelithiasis is often performed concomitantly with surgery for morbid obesity, in view of the high incidence of symptomatic gallstones during rapid weight loss.

OPEN SURGICAL CHOLECYSTECTOMY.[6]

First described in 1882 by Langenbuch, open cholecystectomy (OC) has been the primary treatment of gallstone disease for most of the past century.The greatest drawbacks to open cholecystectomy are the resulting pain and weeks of disability. Antibiotics prophylaxis is employed, a single dose being given at the time of anaesthetic premedication.

Preparation for operation

After appropriate history taking and assessment of thepatient's fitness for the procedure, a full blood count andbiochemical profile should be performed to exclude anaemiaand to identify abnormal liver function. A blood coagulationscreen should be checked if there is a history of jaundice or liver function is abnormal. Prophylactic antibiotics should beadministered either with the premedication or at the time ofinduction of anaesthesia. A second-generation cephalosporin is appropriate. Subcutaneous heparin or antiembolicstockingsshould be prescribed. The patient must sign a consentform to indicate that he or she is fully aware of the procedure being undertaken, alternative options and the risks involvedand complications that may occur.

ANAESTHESIA

General anaesthesia

POSITION OF PATIENT

The patient is placed in supine position

INCISION

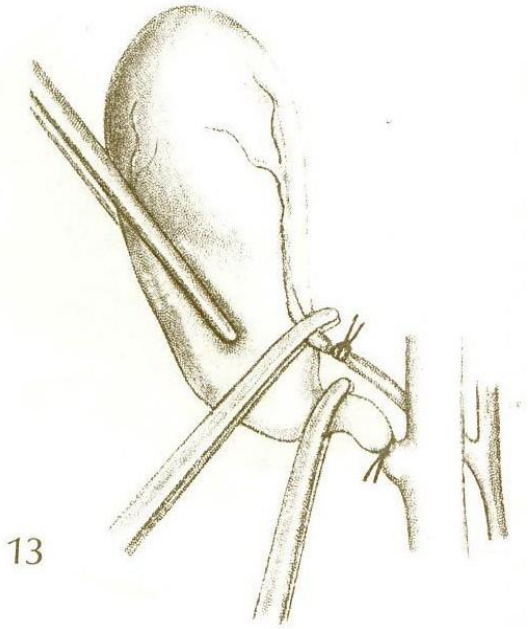
Right Kocher‘s right subcostal, upper paramedian, or a midline incision.

Usually right Kocher‘s incision is placed and deepened rectus opened peritoneum entered. Retractors placed in position to retract liver and colon. Decompression of gallbladder done when its distended. Meticulous dissection carried out to identify cystic duct, cystic artery, CBD to avoid injury. Cystic duct and artery clamped cut ligated and gall bladder dissected from its bed.

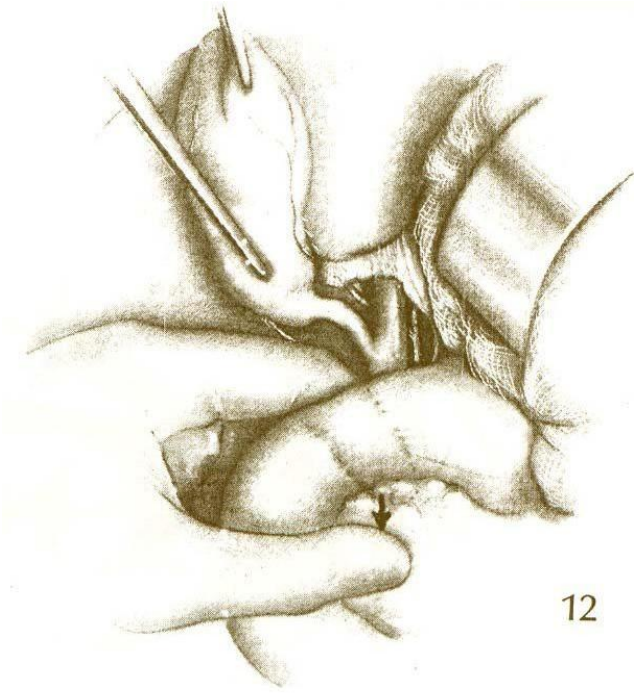
In fundus first method fundus grasped and dissected from its bed and triangele of safety viewed.

In acute cholecystitis subtotal cholecystectomy performed to avoid CBD and vital structure injury.

The surgeon introduces the left index finger into the foramen of Winslow and thoroughly palpatesforcalculiinthecommonbileduct.The subhepatic space is irrigated with warm saline and all irrigation fluid is evacuated. The incision is usually closed in one or two layers. The skin can be closed with skinstaples.



Ligation of cystic duct and arter



Palpation of the lower end of CBD and pancreas

Post operative complications:

Wound infection , bleeding, lower lobe atlectasis, electrolye imbalance, bile duct injury.

Postcholecystectomy syndrome refers to the persistence of symptoms referable to the biliary tract after cholecystectomy. As currently defined, the syndromes exclude those patients whose symtoms are due to organic disease outside the biliary tract.

There is a female preponderance, particularly in the 40-50 years age group. A careful evaluation and a full investigation of the biliary tract including an ERCP are advisible in all patients with persistence or recurrence of symptoms after cholecystectomy. The common causes of post- cholecystectomy syndromesare:

- Retained or recurrentcalculi
- Gall bladder/cystic duct remnants
- Bile duct strictures and unrecognized iatrogenicinjuries.
- Injuries [choledochoduodenal fistula]
- Papillary stenosis and biliarydyskinesia.

LAPAROSCOPIC CHOLECYSTECTOMY

The laparoscopic procedure has many advantages it has early recovery, less postoperative pain, short hospital stay, but wide learning curve as compare to open cholecystectomy.

TECHNIQUE OF LAPAROSCOPIC CHOLECYSTECTOMY

Consent:-

Informed written consent is obtained for laparoscopic procedure, its complications and the need, if necessary for conversion to open cholecystectomy.

Anaesthesia:-

It is done under endotracheal general anaesthesiawith monitoring of end tidal carbon dioxide and pulse oximetry is mandatory.

Position:-

Reverse trendlenberg posture sligately rotated to left.

Procedure:-

Pneumoperitoneum created using verres needle through subumbilicalincisiononcepneumoperitoneum created 10mm trocar placed or open technique under direct vision.
30degree telescope introduced
Other ports are introduced under vision:-

- The right lateral 5mm port in the anterior axillary line
- The epigastric 10mm port
- The sub costal mid-clavicular 5mm port

Fundus of gallbladder grssped and retracted towards shoulder for adequate visualization.

Dissection and skeletonisation of cystic duct and cystic artery—Further

dissection is commenced by division of the peritoneal fold between Hartmann‘s pouch and liver.critical view of safety viewed The cystic artery is identified and cystic duct is identified clipped and divided

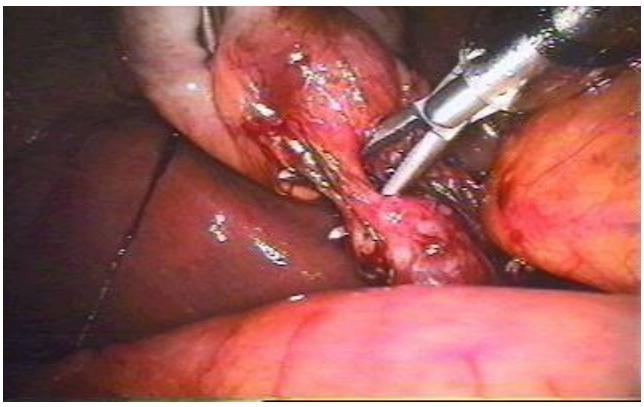
GB is dissected off the liver bed. And takenout via epigastric port.

A drain is inserted through the lateral trocar and positioned in the sub hepatic region. Closure sheath is sutured with vicryl/ prolene. All skin incisions are closed and the drainage tube is connected to the bottle and covered withdressing.

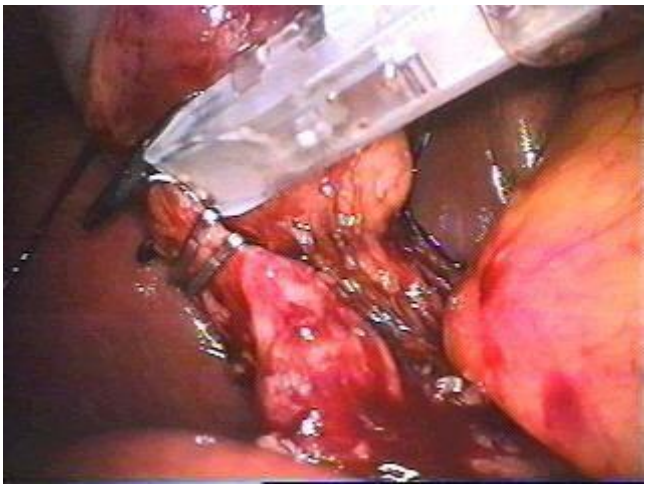
The gall bladder is opened and examined and sent for histopathology.

PortPlacement

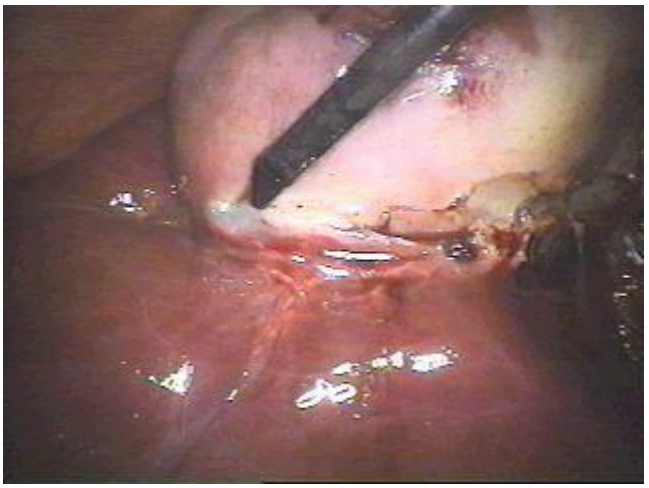
Calotstriangle



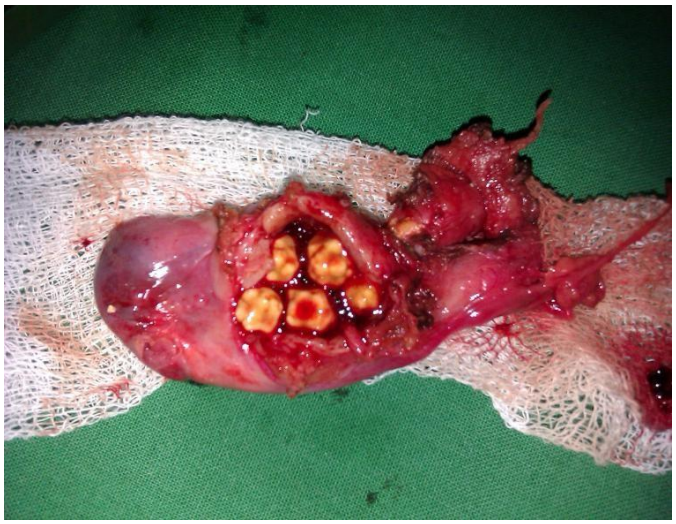
Dissection of cystic duct



Application of clips to Cystic duct



Dissection of GB from liver bed



Gall bladder with stones

COMPLICATIONS OF LAP CHOLECYTECTOMY

A) HEMORRHAGE

TROCAR SITEBLEEDING

Trocar site bleeding can be prevented bymsubcutaneous vessel in subcutaneous tissue should be avoided during insertion.

SUDDEN AND PULSATILE BLEEDING IN CALOT‘STRIANGLE

Bleeding in the Calot‘s triangle can be prevented by careful dissection and proper application of clip to cystic artery.

GALLBLADDER FOSSABLEEDING

GB fossa bleeding can be controlled by bipolar diathermy, packing the site with gel foam.

b) PERFORATION OFGB

In acute cholecystitis perforation may occur due to inflammatory edema. Managed with saline irrigation and suctioning , extraction ofspilledstones.

Drain should be kept inside.

c) OCCULTCARCINOMA

In cases suspected to have carcinoma intraoperatively, frozen section is sent and if frozen section is positive for carcinoma, then conversion to open technique is considered and radical surgery with excision of port sites done.

d)POST OPERATIVE BILELEAK

Post operative bile leak can occur due to injury to the CBD, the right hepatic duct or accessory bile duct.The diagnosis can be confirmed by USG or ERCP.

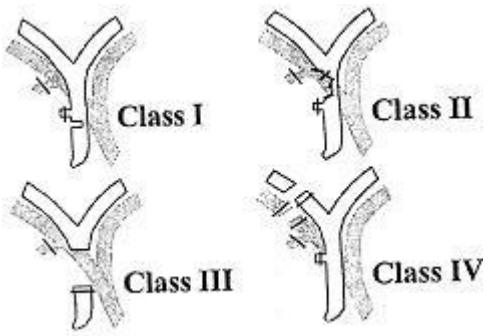
If drain is placed most of the minor leak will heal with expectant management. In some persistent cases, it may be advisable to decrease the intraductal pressure by nasobiliary drainage, endoscopic spincterotomy or transpapillary stenting.

e)BILE DUCTINJURY

Incidence of CBD injury during LC exceeds that of open cholecystectomy i.e0.5% vs 0.2%.[123] Reasons for the increase in injury during LC included loss of hepatic information, incorrect traction forces to the gallbladder and injudicious use of cautery inside of the triangle of Calot. Risk factors that increase the risk of CBD injury include acute cholecystitis, aberrant anatomy. The most common anatomic variant is an aberrant right hepatic duct.

CLASSIFICATION

The Stewart-Way classification is derived from analysis of a series of



LC-associated CBD injuries.

Stewart- Way classification of bile duct injury

It is managed by biliary enteric anastomosis. This is to prevent cholangitis and biliary strictures.

f) BOWELINJURY

Injury to bowel can occur during trocar insertion or dissection in the right upper quadrant, especially when using electrosurgical devices. The jejunum, ileum and colon can be injured by Veress needle and trocars, while duodenum is likely to be injured during dissection. Any structure fixed to the under surface of the umbilicus like the urachus or a meckel‘s diverticulum is more susceptible to injuryduringacess.

g) WOUND INFECTION AND INCISIONALHERNIA

The risk of wound infection following laparoscopic cholecystectomy is less than 1% and the risk of incisional hernia is 0.5%.

h)DIAPHRAGMATIC INJURY

Diaphragmatic injury may be due to either cautery or by mechanical puncture by an instrument.

i)PANCREATITIS

j)PNEUMOPERITONEUM RELATEDCOMPLICATIONS

Embolism

vasovagal reflex,

cardiac arrhythmias and

hypercapniaacidosis.

ADVANTAGES AND DISADVANTAGES OF LC COMPARED TO OC

Advantages and disadvantages of lc compared to oc

ADVANTAGES	DISADVANTAGES
Less post operative pain	Lack of depth perception
Smaller incision	View controlled by camera operator
Better cosmesis	More difficult to control hemorrhage
Shorter hospitalization	Decreased tactile discrimination (haptics)
Earlier return to full activity	Potential CO_2 insufflation complications
Decreased total costs	Adhesions/inflammation limit use
	Slight increase in bile duct injury

CONVERSION In 5-10% of cases, conversion to open cholecystectomy may be needed for safe removal of gallbladder.

MATERIALS AND METHODS

This dissertation titled as –A clinical study and management of cholelithiasis‖ was done at GovtRoyapettah Hospital, Kilpauk medical College and Hospital, Chennai-10.

About 53 consecutive cases were admitted, examined, investigated and operated during the period of Jan 2018 to June 2018. Detailed history of all the 53 cases were taken according to the proforma approved by the guide. All patients underwent detailed examination and Investigationsdone.Risk and complications of the condition as well as surgery was explained to the patients, consent was taken.

In this study some patients underwent open cholecystectomy and some of the patients underwent lap cholecystectomy

Inclusion criteria: 1. Symptomatic gall stones disease with or without complications like

a. acute and chroniccholecystitis
b. mucocele of the gallbladder
c. empyema of the gallbladder
d. perforation
e. pancreatitis

2. asymptomatic gall stones of size more than2.5cm
3. patients with stones both in the gall bladder and the common bileduct

Exclusion criteria:

1. Primary CBD stones with outgallstones
2. Comorbid conditions like cardiac disease and renal failure
3. Asymptomatic gallstones of size less than2.5cm
4. Gall bladder stones with congenital malformations of the biliary tree or stricture of theCBD.

SURGICAL PROCEDURE:

Open Cholecystectomy: A sub costal muscle transection incision was used for open cholecystectomy; the length of the incision was tailored to the individual patient and kept to the minimum necessary to allow safe and adequate access to the gall bladder. Dissection was started at Calot‘s triangle and proceeded antegradely towards the fundus. ―Fundus first method‖ was used in case of dense adhesions where anatomy of Calot‘s triangle was not clear. Based on clinical investigation and operative criteria, exploration of the CBD was done.

Laparoscopic Cholecystectomy: Laparoscopic cholecystectomy was performed with the operating surgeon on the left side of the table. Pneumoperitoneum was created using Veress needle and by Hassan‘s technique in some cases. It involved two 10mm and two 5mm trocars. Peritoneal cavity was visualized and any adhesions if present were released. Calot‘s triangle was visualized and dissection was carried out

by means of electrocautery and the cystic duct and artery were secured with titanium clips. At the completion of the operation, a sub hepatic drain was inserted as required in both the groups. All patients were administered NSAID‘s and anti-emetics as required. Patients were allowed liquids once bowel sounds returned. Patients were discharged from the hospital once they were fully mobilized and able to tolerate a normal diet and pain relief was adequate. Pain in the post op period was rated for each patient using a Visual Analogue Scale (from 0 to 5).
Data was collected prospectively and included in patient‘s demographics.

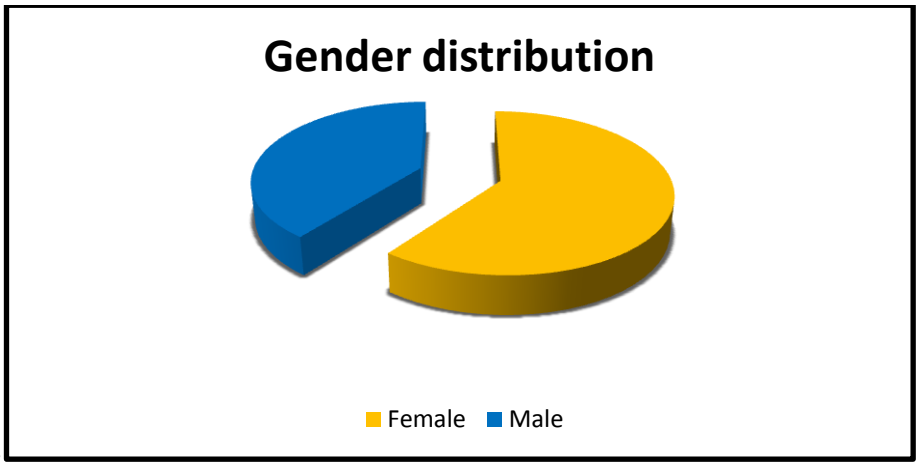
Laboratory results, operative findings, requirement for conversion to open cholecystectomy, operating time (from incision to closure), operative complications, duration of post operative pain, analgesic administration and length of hospital stay along with post-operative complications if any were recorded.
Patients were encouraged to resume work and normal daily activity as soon as possible. Evaluation of return to normal work was made during an OPD appointment 4 weeks aftersurgery.

OBSERVATIONS & RESULTS

Our study is the prospective study of Clinical Study and Management of Cholelithiasis. It included 53 cases, presenting with symptoms of gall stones, confirmed with imaging techniques and admitted to the department of surgery of Government Royapettah Hospital, Kilpauk Medical College and Research Hospital, Chennai. Duration of study was from Jan 2018 to June 2018

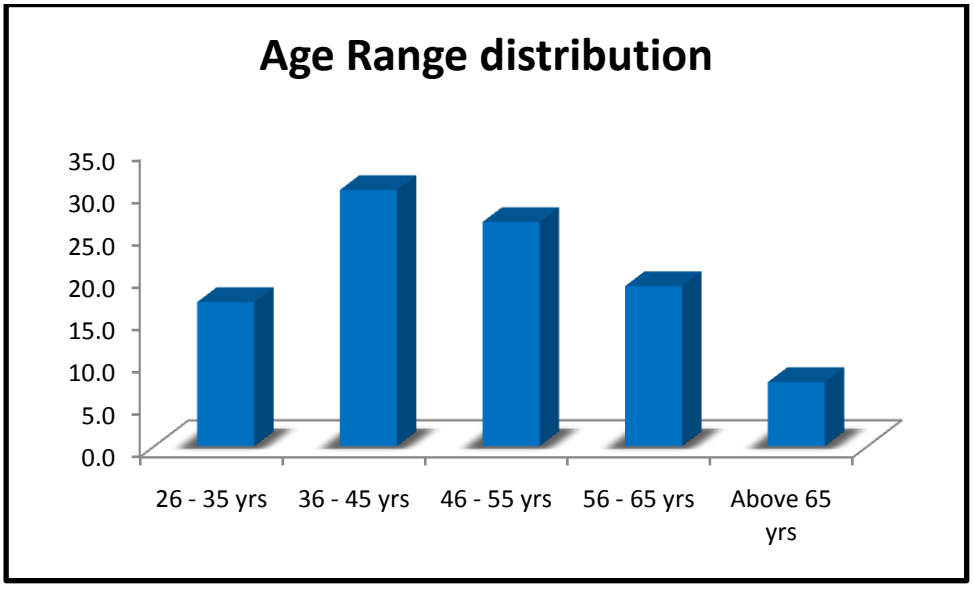
Table : Sex wise distribution of the cases in our study.

		Frequency	Percent
Valid	Female	32	60.4
	Male	21	39.6
	Total	53	100.0



In the present study 32 (60.4%) patients were female and 21 (39.6%) patients were male. The study shows that gallstones disease is a common problem in female population. The female to male ratio is 1.52:1.

Age Range Groups:



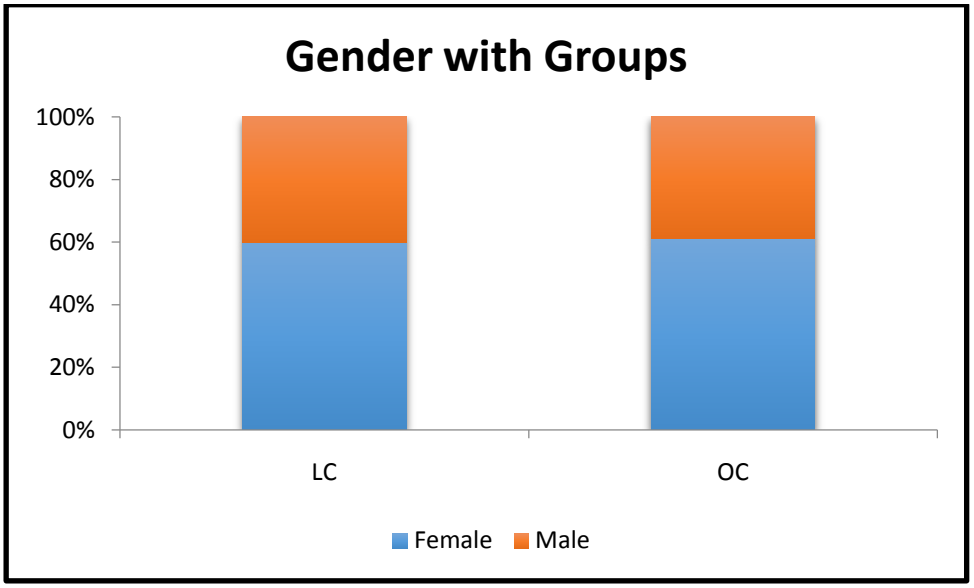
In this study there was a increased incidence of cholelithiasis in the 4th decade, even though no age group was exempt from the disease process

CLINICAL SYMPTOMS:

Right upper quadrent pain is the most common symptom present in 52(98.1%) cases. Fever in 14 (26.4%) cases, vomiting and dyspepsia seen in 22(41.5%) cases . Jaundice present in 4(&.5%)cases.

P - Value	** Highly Significant at P ≤ .01
P - Value	* Significant at 0.01 < P ≤ .050
P - Value	# No Significant at P >.050

	LC	OC
Female	60.0%	61.1%
Male	40.0%	38.9%



PER OP COMP * Groups

Crosstab

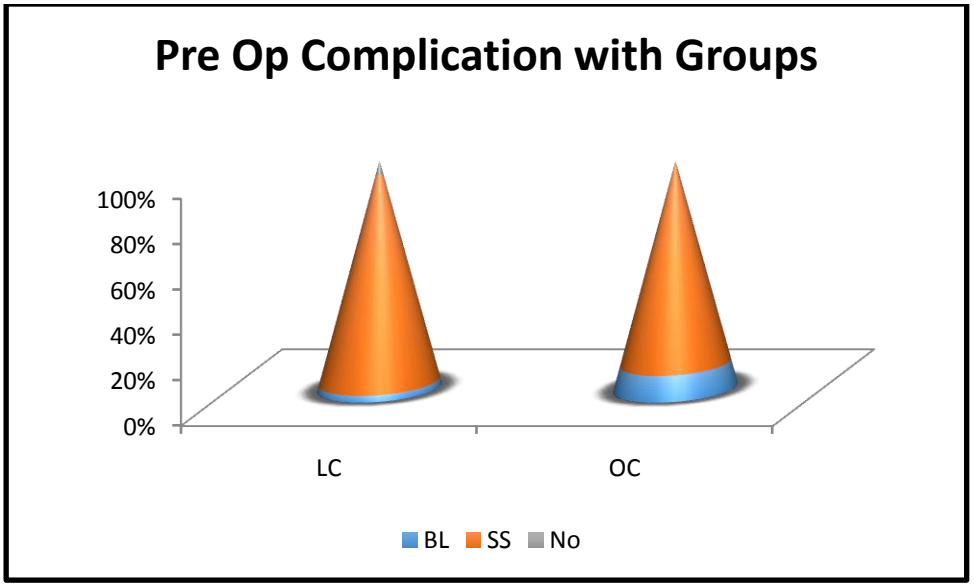
			Groups		
			LC	OC	Total
PER OP COMP	BL	Count	1	2	3
		% within Groups	2.9%	11.1%	5.7%
	No	Count	32	16	48
		% within Groups	91.4%	88.9%	90.6%
	SS	Count	2	0	2
		% within Groups	5.7%	0.0%	3.8%
Total		Count	35	18	53
		% within Groups	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.468[a]	2	.291
Likelihood Ratio	2.999	2	.223
N of Valid Cases	53		

a. 4 cells (66.7%) have expected count less than 5. The minimum expected count is .68.

	LC	OC
BL	2.9%	11.1%
SS	91.4%	88.9%
No	5.7%	0.0%



Age range * Groups

Crosstab

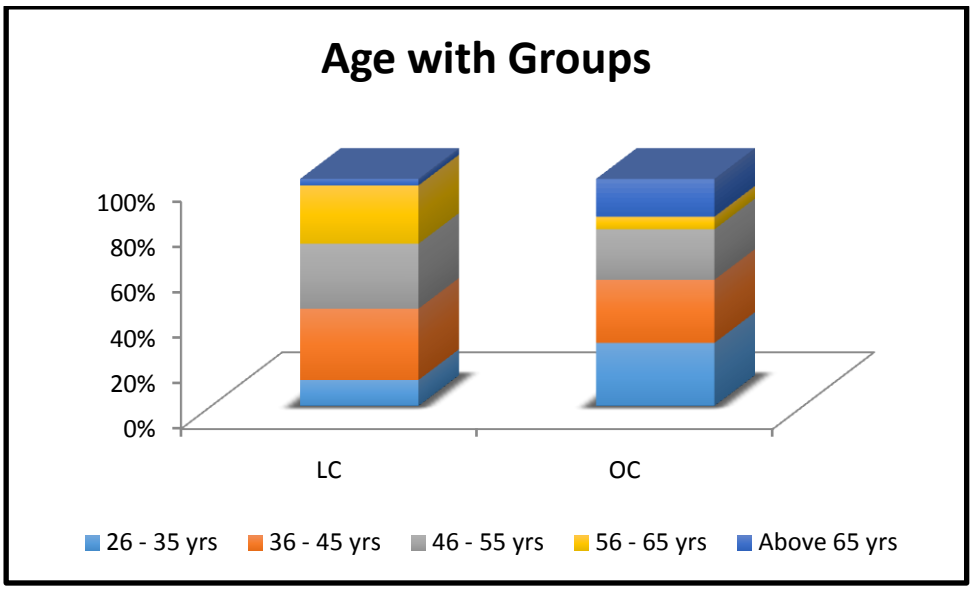
			Groups		
			LC	OC	Total
Agerange	26 - 35 yrs	Count	4	5	9
		% within Groups	11.4%	27.8%	17.0%
	36 - 45 yrs	Count	11	5	16
		% within Groups	31.4%	27.8%	30.2%
	46 - 55 yrs	Count	10	4	14
		% within Groups	28.6%	22.2%	26.4%
	56 - 65 yrs	Count	9	1	10
		% within Groups	25.7%	5.6%	18.9%
	Above 65 yrs	Count	1	3	4
		% within Groups	2.9%	16.7%	7.5%
Total		Count	35	18	53
		% within Groups	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	7.669[a]	4	.104
Likelihood Ratio	7.931	4	.094
Linear-by-Linear Association	.394	1	.530
N of Valid Cases	53		

a. 5 cells (50.0%) have expected count less than 5. The minimum expected count is 1.36.

	LC	OC
26 - 35 yrs	11.4%	27.8%
36 - 45 yrs	31.4%	27.8%
46 - 55 yrs	28.6%	22.2%
56 - 65 yrs	25.7%	5.6%
Above 65 yrs	2.9%	16.7%



T-Test

Group Statistics

Groups		N	Mean	Std. Deviation	Std. Error Mean
Age	LC	35	48.43	10.556	1.784
	OC	18	47.06	13.549	3.194
DURATION	LC	35	88.43	16.214	2.741
	OC	18	103.89	25.003	5.893
VAS	LC	35	2.60	.695	.117
	OC	18	3.28	.669	.158
PAIN DUR	LC	35	4.00	1.237	.209
	OC	18	5.17	1.465	.345
ANALGESICS	LC	35	5.06	1.830	.309
	OC	18	6.94	1.626	.383
Days in	LC	35	8.09	2.832	.479
Hospital	OC	18	11.44	3.166	.746
RETURN TO	LC	35	9.49	2.737	.463
NORMAL	OC	18	13.17	3.330	.785
WORK					

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of M				
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	St Di
Age	Equal variances assumed	2.277	.137	.407	51	.686	1.373	
	Equal variances not assumed			.375	27.910	.710	1.373	
DURATION	Equal variances assumed	6.921	.011	-2.721	51	.009	-15.460	
	Equal variances not assumed			-2.379	24.574	.025	-15.460	
VAS	Equal variances assumed	.820	.369	-3.406	51	.001	-.678	
	Equal variances not assumed			-3.447	35.594	.001	-.678	
PAIN DUR	Equal variances assumed	1.527	.222	-3.053	51	.004	-1.167	
	Equal variances not assumed			-2.890	29.741	.007	-1.167	
ANALGESICS	Equal variances assumed	.019	.892	-3.687	51	.001	-1.887	
	Equal variances			-3.832	38.251	.000	-1.887	

	not assumed						
Days in Hospital	Equal variances assumed	1.768	.190	-3.928	51	.0005	-3.359
	Equal variances not assumed			-3.788	31.225	.001	-3.359
RETURN TO NORMAL WORK	Equal variances assumed	2.993	.090	-4.305	51	.0005	-3.681
	Equal variances not assumed			-4.040	29.111	.000	-3.681

Groups = LC

Statistics[a]

		Age	DURATION	VAS	PAIN DUR	ANALGESICS	Days in Hospital	I N
N	Valid	35	35	35	35	35	35	
Mean		48.43	88.43	2.60	4.00	5.06	8.09	
Median		50.00	90.00	3.00	4.00	5.00	7.00	
Std. Deviation		10.556	16.214	.695	1.237	1.830	2.832	
Range		39	70	3	5	9	12	
Minimum		28	60	1	3	3	4	
Maximum		67	130	4	8	12	16	

a. Groups = LC

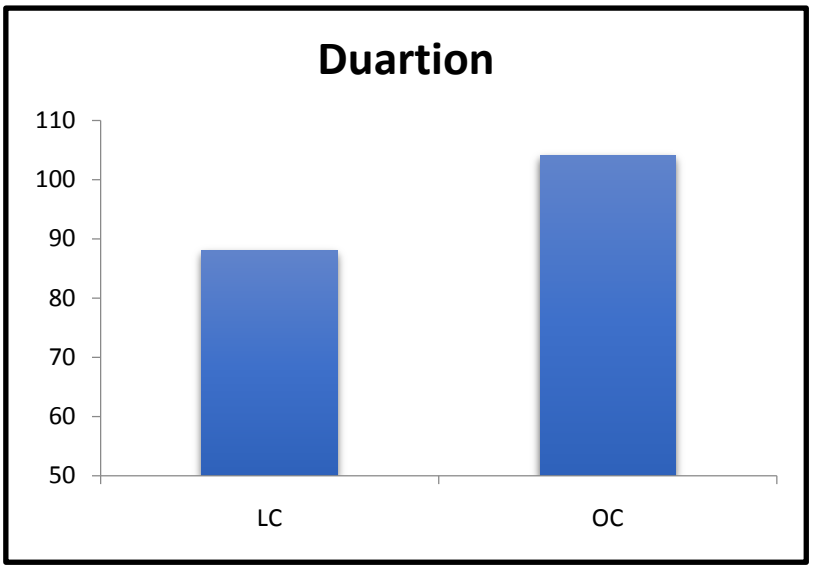
Groups = OC

Statistics[a]

		Age	DURATION	VAS	PAIN DUR	ANALGESICS	Days in Hospital	I N
N	Valid	18	18	18	18	18	18	
Mean		47.06	103.89	3.28	5.17	6.94	11.44	
Median		43.50	100.00	3.00	5.00	7.00	10.00	
Std. Deviation		13.549	25.003	.669	1.465	1.626	3.166	
Range		42	80	3	5	5	8	
Minimum		28	70	2	3	5	8	
Maximum		70	150	5	8	10	16	

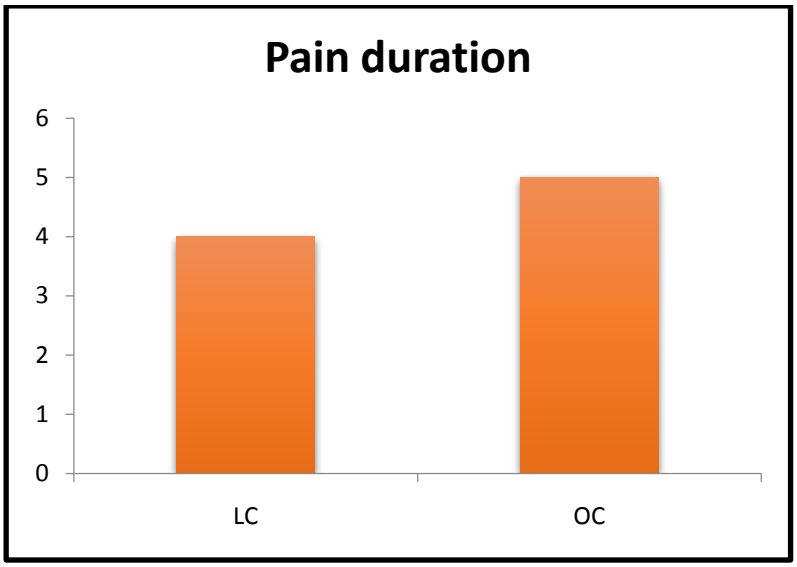
a. Groups = OC

Duration of procedure compared with both groups



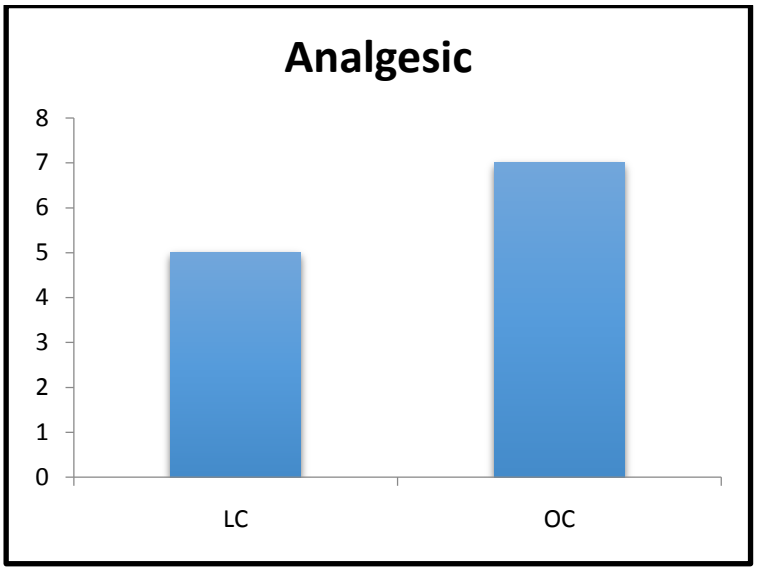
	Duartion
LC	88mins
OC	104mins

Postoperative pain duration



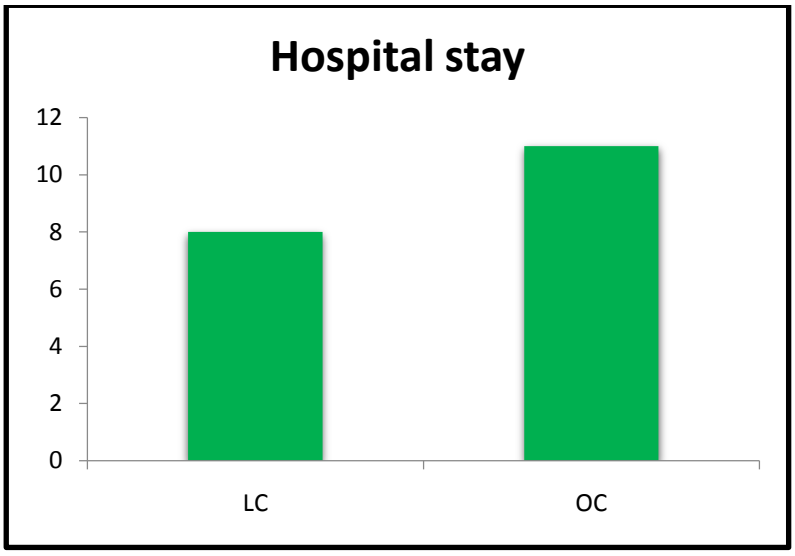
	Pain duration
LC	4
OC	5

Postoperative use of analgesics



	Analgesic
LC	5
OC	7

Postoperative hospital stay



	Hospital stay
LC	8
OC	11

All cases were managed by one or the other surgical procedure as mentioned below. 6 patients had other co-morbid conditions like diabetes mellitus, hypertension, COPD.

Open cholecystectomy was done in 18 (34%) patients while 35 (66%) patients underwent laparoscopic cholecystectomy. Of the 18 open cholecystectomies, Kocher‘s right subcostal incision was used in 17 cases. Abdomen was opened in midline for 1 case of acute cholecystitis (75 year old male) who didn‘t improve on conservative management and had developed features of peritonitis. In 2 (9.5%) cases, Lap was converted to open cholecystectomy due to denseadhesions.

Common bile duct stones were managed with cholecystectomy and common bile duct exploration with T- tube insertion in 2 patients following failure of ERCP stone extraction. In 1 case ERCP successfully removed the CBD stone and later patient underwent elective laparoscopic cholecystectomy. On table T- tube cholangiogram was done in first 2 patients.

Mild to moderate adhesions were noted in 30% of cases and in 10% thick fibrous adhesions noted. In 10% Calot‘s triangle was found difficult to dissect. In 90% of cases classical porta to fundus dissection (retrograde) was used except in 10% where Calots‘s triangle was obscured.

Intraoperative anomalies found was short and thick cystic duct in 2 cases.

40 (80%) cases had fibrosed, contracted and thickened
gallbladder, 9(18%) cases had inflamed gallbladder
1(2%) cases had empyema of
gallbladder and

The median duration of operative procedure was 103 (70-130min) for OC and 88min (60-130min) for LC. The difference was not found to be significant. The main complications noted per operatively were bile leak (1patient (5.7%) in LC and 1 patients (11.1%) in OC group) and stone spillage (1 (5%) in LC and 1 (3%) in OC). There was no instance of CBD injury in either group.

Post operative complications were minimal. 1 patient in laparoscopic and 2 patients in open had surgical site infection.

The VAS was median Grade 3.8 in OC group as compared to median Grade 2.6 in LC group. The pain was more in the initial 3 days in both groups and it lasted for median duration of 5 days in OC group compared to 4 days in LC group. The NSAID‘s were used for more days in OC group (median-6days) compared to LC group (median-4 days).

DISCUSSION

Our study is the prospective study of Clinical study and management of Cholelithiasis. It included 53 cases presenting to the OPD of Department of Surgery with symptomatic gall stones, confirmed by imaging studies and admitted for further management in GovtRoyapettah Hospital, Kilpauk Medical College and Research Hospital, chennai. Duration of study was from Jan 2018 to October 2018

Age wise distribution of the cases included in our study

Our study has shown that most common age group affected by symptomatic gall stones was the 4^{th} decade, even though no age group was exempted from the disease process. This was correlated well with studies conducted by Herman's (4^{th} decade) and Rushad's (4^{th} – 5^{th} decade)series.

comparison of age-wise distribution of the cases.

Age Group in years (yrs)	Present study	%	Herman et al	%	Rushad et al	%
11-20	0	0%	25	1.60%	00	0
21-30	03	5.6%	92	5.90%	04	3.33%
31-40	13	24%	226	14.60 %	36	30%

41-50	19	35.8%	325	21%	30	25%
51-60	11	20.7%	473	30.60%	29	24.16%
>60	07	13.2%	352	23.57%	21	17.5%

In this study cases fall between 15 – 85 Yrs. There is an increased incidence in the 4 & 5th decade with the maximum incidence in the 4thdecade.

Similar incidence is seen in the studies of Herman et al (5th decade).[129] Hanif.[130] series showed peek incidence in 5th decade. In western studies the peak incidence is inthe5th&6thdecades.Theriseinthepeakageofincidenceisduetochangeinthe dietary factor. Similar findings are noted in the studies of Ganeyet al.[131] and Moreaux et al.[132]

Sex wise distribution of cases included in our study

:Sex wise comparison of the cases.

Sex	No. of cases	%	Battachary 's series	%	Alokshar ma series	%
Female	32	60.4%	65	71.4	41	70
Male	21	39.6%	26	28.6	17	30

In the present study 32 (60.4%) out of 53 cases were female while the rest 21 (39.6%) were male. Battacharya.[133] series showed 71.4% were female,

28.6% were male. Similar sex preponderance in the favour of females were noted by A.P.Tamhankar.[134], Ganey et al.[131,] Major Alok Sharma et al.[135,] series showed that 70% were female & 30% were male.

Clinical presentation of different cases included in our study.

Our study has shown that all of the patients presented with pain in the right upper quadrant followed by vomiting (44%), dyspepsia (38%). Others also had fever and jaundice.

Similar findings were also observed in the studies conducted by Ganey and AlokSharma.

TABLE : comparison of clinical presentation of the cases.

Sl.No	Clinical Presentation	Present study		AlokSharma[137]		Ganey[133]	
		Number of cases	% of cases	Number of cases	% of cases	Number of cases	% of cases
1	Pain	52	98.1%	58	100%	987	95%
2	Vomiting	22	41.5%	48	82.8%	576	55.6%
3	Dyspepsia	22	41.5%	5	8.62%	222	21%
4	Fever	14	26.4%	NA	00	92	09%
5	Jaundice	04	7.5%	3	5.17%	101	10%

Pain was the predominant symptoms in the present study involving- 98% patients. The commonest site of pain was in the Rt. Hypochondrium, & the next commonest site was Epigastrium. 5 (10%) patients complained of pain radiating to the back. 38 (76%) patients had chronic Recurring pain and 10 (20%) patients had acute onset of pain. pain was colicky in nature. Similar presentations were noted in the series of Alok Sharma, Ganey, Goswitz et al[115]. 44% (22 patients) of cases in the present series had nausea/vomiting.

Vomiting was spontaneous, occurred mostly during the attack of pain.

Vomiting in this study was almost similar to Ganey et al series. In the present study 4 (8%) patient had jaundice. The cause of the jaundice was stone in the common bile duct in 3 (6%) patients, 1(2%) patient had features of cholangitis. The common bile duct were explored in 2 (4%) patients & stone were removed. 38% (19 patients) of patient had dyspepsia. The dyspepsia was relieved after these patients underwent Cholecystectomy. The incidence of dyspepsia in present series was higher to Ganey series and Alok Sharma series. Fever was present in13 (26%) cases in the present study. Fever was due to acute cholecystitis in 10 (20%) patients and secondary tocholingitis due to biliary obstructionin 4(8%) patients

Clinical signs of different cases included in the present study.

TABLE 23: comparison of different clinical signs.

Clinical signs	K.L. Kapoor et al[136]	Karl A et al[137] (N=1261)	Present study
Tenderness in RH/epigastrium	89.6%	96%	96%
Icterus	2.10%	10%	8%
Mass	6.20%	4%	2%

Tenderness in the Rt. Hypochondriam was present in all patients which was similar to Kapoor et al & Karl et al studies, guarding was present in 15 (30%) patients. A positive Murphy‘s sign present in 8 (16%) patients. A Globular mass was felt in 1 (2%) patient who had empyema of the gall bladder. Icterus was present in 4 (8%) patients.

INVESTIGATIONS

All the patients underwent routine hematological & biochemical investigations. The hemoglobin of patients ranged from 8 to 15 gm %. Serum bilurubin was raised in 8 patients, there bilurubin levels ranged form 1.8 to 5 mg %.Alkaline phosphatase was raised in 8 patients

Ultrasound Imaging study of the cases included in the present study. Ultrasound scanning was done in all patients. All 50 patients had stones in the gall bladder, 3 (6%) patient had stones in both gall bladder & common bile duct. 64%(32 patients) had multiple stones in gall bladder, 36% (18 patients) had solitary stones in gall bladder.

Thickening of gall bladder was present in 16% (8 cases) of the cases. Dilatation of the common bile duct more than 1 cm was present in 3 (6%) patients. Many of the features in my study were similar to studies of major Alok Sharma etal.

TABLE No. 24: Comparison of Ultrasound Imaging study of the cases.

Sl.No	Imaging findings	Number of cases	% of cases	Alok Sharma[1] [38] Series	%
1	Stones in gallbladder	53	100%	57	98.3
2	Solitary stone	13	24.5%	15	26.3
3	Multiple stone	27	50.9%	42	73.7
4	Gall stone with Bile duct stone	2	3.8%	3	5.2
5	Dilated bile duct	1	1.9%	10	17.2
6	Gall bladder wall thickening	8	16%	3	5.2
7	Mass	0	0%	1	1.7

Management of the cases included in the present study.

All cases were managed by one or the other surgical procedure. All patients who were diagnosed with chronic cholecystitis were operated electively by open or laparoscopic cholecystectomy.10 Patients who presented with acute severe symptoms were initially managed by Nil by mouth, nasogastric tube, broad spectrum antibiotics and analgesics.3 patients underwent laparoscopic cholecystectomy after 3 days.2 patients underwent emergency open cholecystectomy of which one was gangrenous gall bladder with perforation and one was empyema gallbladder. Another patient who had palpable mass was taken for laparoscopic cholecystectomy but had to be converted to open surgery because of dense adhesions. It was confirmed to be empyema of gall bladder and patient underwent partial cholecystectomy. Other 5 patients were treated conservatively and interval cholecystectomy was done after 6 weeks of which 4 patients underwent open cholecystectomy and 1 patient underwent lap which was again converted to open with partial cholecystectomy due to dense adhesions. Three patients who had cholelithiasis with choledocholithiasis were managed with cholecystectomy with CBD exploration and T-tube insertion in two patients following failure of ERCP stone extraction. In one case ERCP successfully removed the CBD stone and the patient later underwent elective laparoscopic cholecystectomy.

Laparoscopic vs open cholecystectomy

Traditional cholecystectomy is an integral part of every surgical training programme and is performed by most general surgeons. The advent of

laparoscopic cholecystectomy has created an excitement and a flurry of activity in the medical community.

This study showed that morbidity rate is more with open cholecystectomy than laparoscopic cholecystectomy.

The operating time was almost equal in both the procedures, 103min (60-150min) for OC and 88min (60-130min) for LC, slightly more mean time in Open Cholecystectomy was due to dense adhesions and in patients requiring CBD exploration & T-tube insertion.

As experience is gained, an operating time of about 50 min can be achieved during laparoscopic cholecystectomy. This –learning curve‖ represents adapting to operating in the 2-D screen, becoming familiar with the instrumentation and becoming accustomed to the technique.

In this study, there were no major complications and several minor ones. There was no peri-operative mortality and no CBD injury. The complications observed per operatively were bile leak, stone spillage and blood loss which were found to be comparable in both the groups. Post operative complications were surgical site infection, one in each procedure.

The VAS was significantly less for LC group [Grade2 (median) for LC and Grade3 (median) for OC; $p< 0.01$]. Kum[120] also found a mean VAS score of 4 v/s 5.17 between LC and OC. The pain duration (median 3 days for LC and median 5.5 days for OC patients; $p < 0.01$) and the duration of analgesics used (median 5 days for LC and median 7 days for OC patients; $p<0.01$) also were significantly less in laparoscopic group patients. This was due to the lesser incision size in LC. Other studies have also shown similar results.[120,119]

The two most beneficial aspects of LC are, the short hospital stay and the rapid recovery. In this study, the median duration of hospital stay was 7.5 days for LC group and 12 days for OC group. The difference was found to be statistically significant ($p<0.01$). This was also confirmed in various other series.[118]

The time taken to return to normal work was also less in LC cases than OC cases.

CONCLUSION

- The highest age incidence of cholelithiasis was in the 4th decade, eventhough no age group was exempt from the disease process.
- The incidence of cholelithiasis was more infemales.
- The commonest symptom was painabdomen.
- The commonest sign wastenderness.
- Ultrasonogram is the imaging modality ofchoice.
- The most common complication of gallstone disease was chroniccholecystitis.
- Laparoscopic cholecystectomy is a safe and effective treatment for most patients with symptomaticgallstones.
- One should not hesitate to convert to an OC if significant adhesions or inflammation are identified duringlaparoscopy.
- Technically, the dissection of the cystic artery and cystic duct is very precise and less peri operative blood loss in laparoscopictechnique.
- The antibiotic usage in LC is comparatively lesser than that ofOC.

- The degree of post operative pain and its duration isless.

- The amount of analgesic requirement is less inLC.

- LC patients tolerate oral feeds earlier and are mobilizedfaster.
- The duration of hospital stay is less and patients can be discharged quickly from thehospital.

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KEY TO MASTER CHART

IP No	In patient number
RUQ	Right Upper Quadrant
Dysp	Dyspepsia
Prev. Hist	Previous History
OCP‘s	Oral Contraceptive Pills
RHC	Right hypochondriac
STB	Serum total bilirubin
SGPT	Serum Glutamyl Phosphate Transaminase
ALP	Alkaline Phosphatase
TC	Total Cholesterol
LC	Laparascopic cholecystectomy
OC	Open Cholecystectomy
CBDE	Common Bile Duct Exploration
HPR	Histopathology Report
F	Female
M	Male
+	Positive /Present
-	Negative /Absent
A C	Acute Cholelithiasis
Ch. c	Chronic Cholecystitis
CC	Calculus cholecystitis
APD	Acid peptic disease
OJ	Obstructive jaundice
N	Normal
MC	Multiple Calculus
Symb C	Symptomatic Cholelithiasis
CBDS	Common Bile Duct Stones
GBC	Gall Bladder calculus
DIHBR	Dilated intra hepatic bile radical
SC	Solitary Calculus
GB	Gall Bladder
DGB	Distended Gall Bladder
ACC	Acute calculus cholecystitis
OC	Laparoscopic converted to Open Cholecystectomy
GCC	Gangrenous Cholecystitis
CCC	Chronic Calculus Cholecystitis
ACC	Acute Calculus Cholecystitis
ACCC	Acute on Chronic Calculus Cholecystitis
BL	Bile leakage
SSI	Surgical site infection
TLC	Total leucocyte count
PMN	Poly morphonuclear cells
ST	Stormy recovery
ERCP	Endoscopic Retrograde Cholangiopancreatography
SS	Stone spillage

MASTER CHART

SL. No.	NAME				Complaints						RF		Signs				DIAGNOSIS	imaging	Treatment										HPR	Complication	Days in Hospital	RETURN TO NORMAL WORK	Outcome
		IP.NO.	Age	Sex	RUQ Pain	Fever	Vomiting	Jaundice	Dysp	Prev. Hist	OBESITY	OCP's	Febrile	Icterus	Tender RHC	Mass in RHC		Ultrasound	LC/OC	DURATION	ERCP	CBDE	T Tube Drain	PER OP COMP	VAS	PAIN DUR	ANALGESICS					
1	Thiyagarajan	3771	42	M	+	+	+	+	-	-	-	-	+	+	+	-	A C	MC+TG+ASC	OC	130	-	-	No	BL	4	5	7	GCC	NIL	15	16	UR
2	Jamudurin	2373	43	F	+	-	-	-	+	+	+	-	-	-	+	-	Ch.c	MC	LC	90	-	-	NO	-	3	4	5	CCC	NIL	9	10	UR
3	Nagesh	2310	34	M	+	-	-	-	-	+	-	-	-	-	+	-	Ch.c	MC	LC	100	-	-	NO	-	2	3	3	CCC	NIL	5	6	UR
4	Jeyanthi	5778	38	F	+	-	+	-	+	-	+	-	-	-	+	-	Ch.c	SC	LC	80	-	-	NO	-	3	4	5	CCC	NIL	9	10	UR
5	Rajendran	7629	64	M	+	+	+	+	+	-	+	-	-	+	+	-	C+CDL	C+CDL+IHBR	OC	150	√	√	Ö	-	3	8	8	CCC	NIL	16	18	UR
6	Valliyammal	7168	60	F	+	-	-	-	-	+	+	-	-	-	+	-	Ch.c	SC	LC	100	-	-	NO	-	3	4	5	CCC	NIL	11	12	UR
7	Thilagavathy	11202	40	F	+	-	-	-	-	+	-	-	-	-	+	-	Ch.c	MC	LC	70	-	-	NO	-	2	3	5	CCC	NIL	10	11	UR
8	pushpa	14428	55	F	+	-	+	-	-	+	-	-	-	-	+	-	Ch.c	MC	OC	75	-	-	NO	-	3	4	6	ACCC	NIL	10	11	UR
9	Dhanalakshmi	16811	36	F	+	-	-	-	+	+	-	-	-	-	+	-	Ch.c	SC	LC	90	-	-	NO	-	2	4	5	CCC	NIL	9	10	UR
10	Lakshmi	16826	48	F	+	-	+	-	-	+	-	-	-	-	+	-	Ch.c	MC	LC	95	-	-	NO	-	3	4	6	CCC	NIL	10	11	UR
11	Asaithambi	18713	33	M	+	+	+	-	+	-	+	-	-	-	+	-	A C	MC+TG	OC	100	-	-	NO	-	4	5	7	ACCC	NIL	13	14	UR
12	Radhakrishnan	19087	55	M	+	-	-	-	-	+	-	-	-	-	+	-	Ch.c	SC	LC	90	-	-	NO	-	3	4	5	CCC	NIL	9	10	UR
13	Padmavathi	18296	28	F	+	-	-	-	+	+	+	-	-	-	+	-	Ch.c	SC	LC	110	-	-	NO	-	3	3	5	CCC	NIL	6	7	UR
14	Kanaga	26675	58	F	+	+	-	-	+	+	-	-	-	-	+	-	A C	SC+TG+DGB	LC	90	-	-	NO	-	3	3	4	ACCC	NIL	6	7	UR
15	Subramani	25692	50	M	+	-	-	-	+	-	+	-	-	-	+	-	Ch.c	SC	LC	115	-	-	NO	-	4	7	10	CCC	BL	16	17	UR
16	Papa	25198	70	F	+	+	+	+	+	-	-	-	+	+	+	-	C+CDL	C+CDL+DCBD	OC	140	√	√	Ö	-	3	5	7	CCC	NIL	15	18	UR
17	Sabetha	24788	66	F	+	-	-	-	-	-	+	-	-	-	+	-	Ch.c	MC	OC	110	-	-	NO	-	3	4	5	CCC	NIL	10	11	UR
18	Fathima	25718	42	F	+	+	+	-	-	-	-	-	+	-	+	-	A C	MC	OC	80	-	-	NO	-	3	4	6	ACCC	NIL	9	10	UR
19	Gayathri	24085	33	F	+	-	-	-	-	-	+	-	-	-	+	-	Ch.c	MC	OC	100	-	-	NO	-	3	4	5	CCC	NIL	9	10	UR
20	Saminathan	24394	67	M	+	-	+	-	-	-	+	-	-	-	+	-	Ch.c	SC	LC	100	-	-	NO	-	2	3	4	CCC	NIL	5	7	UR
21	Thilagavathy	23786	34	F	+	-	-	-	-	-	-	-	+	-	+	-	Ch.c	MC	OC	85	-	-	NO	-	3	4	6	CCC	NIL	8	9	UR
22	Megala	23334	45	F	+	+	+	-	-	+	+	-	-	-	+	-	A C	MC+TG	OC	120	-	-	NO	-	4	5	7	ACC	NIL	9	10	UR
23	Malar	22285	40	F	+	-	-	-	+	-	-	-	-	-	+	-	Ch.c	MC	LC	90	-	-	NO	-	3	7	8	CCC	SSI	12	14	SR
24	Malliga	23430	34	F	+	-	-	-	+	-	-	-	-	-	+	-	Ch.c	SC	OC	95	-	-	NO	-	3	5	6	CCC	NIL	9	10	UR
25	Gowsar	23208	33	M	+	-	+	-	-	+	+	-	-	-	+	-	Ch.c	SC	LC	90	-	-	NO	-	2	3	4	CCC	NIL	5	6	UR
26	Murugan	2237	40	M	+	-	+	-	+	-	+	-	-	-	+	-	Ch.c	MC	LC	130	-	-	NO	-	3	4	5	CCC	NIL	8	9	UR
27	Gnanavel	21663	68	M	+	-	-	-	-	-	-	-	-	-	+	-	Ch.c	MC	OC	70	-	-	NO	-	3	3	5	CCC	NIL	8	10	UR
28	Shanthi	21538	54	F	+	-	+	+	-	-	-	-	-	+	+	-	C+CDL	MC+CDL+DCBD	LC	80	√	-	NO	-	2	4	4	CCC	NIL	10	12	UR
29	Sasikala	20347	32	F	+	-	+	-	-	-	+	-	-	-	+	-	Ch.c	MC	LC	70	-	-	NO	-	2	3	4	CCC	NIL	5	7	UR
30	Govindammal	20140	60	F	+	-	-	-	-	-	-	-	-	-	+	-	Ch.c	MC	LC	75	-	-	NO	-	2	3	4	CCC	NIL	6	7	UR
31	Shanvasbegam	20534	37	F	+	+	+	-	-	-	+	-	-	-	+	-	A C	SC+DGB+TG	OC	100	-	-	NO	BL	4	8	10	ACC	SSI	15	17	UR
32	Ramya	17615	55	F	+	-	-	-	-	-	+	-	-	-	+	-	Ch.c	SC	OC	70	-	-	NO	-	2	4	6	CCC	NIL	10	12	UR

33	Kalyani	18439	60	F	+	-	-	-	-	-	-	-	-	-	+	-	Ch.c	MC
34	Rani	19686	63	F	+	-	-	-	+	-	+	-	-	-	+	-	Ch.c	SC
35	Yahudhunisha	19321	50	F	+	-	-	-	+	-	-	-	-	-	+	-	Ch.c	MC
36	Venkatraman	18524	50	M	+	-	-	-	-	-	-	-	-	-	+	-	Ch.c	MC
37	Thenmozhi	18974	40	F	+	-	-	-	-	-	+	-	-	-	+	-	Ch.c	MC
38	Kannan	18951	45	M	+	-	+	-	-	+	-	-	-	-	+	-	Ch.c	MC
39	Lakshmi	17592	36	F	+	-	-	-	-	-	+	+	-	-	+	-	Ch.c	MC
40	Murugan	17395	47	M	+	-	+	-	-	+	-	-	-	-	+	-	Ch.c	MC
41	Muthulakshmi	18158	51	F	-	-	-	-	+	-	+	-	-	-	+	-	Ch.c	MC
42	Mamula	17201	39	F	+	+	-	-	+	+	-	-	+	-	+	-	A C	MC+TG
43	Mani	15137	63	M	+	+	+	-	-	+	-	-	-	-	+	-	A C	MC
44	Kandhasamy	12468	55	M	+	-	-	-	+	-	+	-	-	-	+	-	Ch.c	MC
45	Rajendran	14875	53	M	+	+	+	-	-	-	-	-	-	-	+	-	A C	SC+TG+D
46	Lilly	14632	43	F	+	-	+	-	-	-	+	-	-	-	+	-	Ch.c	SC+DGE
47	Kanniyammal	10619	58	F	+	-	-	-	-	+	+	-	-	-	+	-	Ch.c	MC
48	Murugaiyan	13801	55	M	+	+	+	-	+	-	-	-	+	-	+	+	AC	SC+TG
49	Durai Raj	14944	58	M	+	-	+	-	-	-	+	-	-	-	+	-	Ch.c	MC
50	Chitra	194675	42	F	+	+	-	-	+	+	-	-	-	-	+	-	Ch.c	SC
51	Meshak	13690	28	M	+	+	-	-	+	+	+	-	-	-	+	-	AC	MC+TG
52	Raja	17301	48	M	+	-	-	-	+	+	-	-	-	-	-	-	Symb C	MC
53	Ranjith	18249	64	M	+	-	-	-	+	-	-	-	-	-	-	-	Ch c	SC

PATIENT CONSENT FORM

Study detail:

"A clinical study and management of cholelithiasis"

Study centre : GOVT ROYAPETTAH HOSPITAL, CHENNAI

Patients Name:

Patients Age :

Identification

Number :

Patient may check (／)
these boxes

I confirm that I have understood the purpose of procedure for the above study. I had the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I hereby make known that I have fully understood the use of above surgical procedure, the possible complications arising out of its use and the same was clearly explained to me and also understand that this technique is a new method of treatment of patella fractures and this study is done to know the usefulness of the same in management of patella fractures ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well-being or any unexpected or unusual symptoms. ☐

I hereby consent to participate in this study. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests.

Signature/thumb impression:

Patients Name and Address: place date

Signature of investigator :

Study investigator's Name :

Place:

Date: