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A Dissertation On

"A COMPARITIVE STUDY OF OPEN CHOLECYSTECTOMY VERSUS LAPAROSCOPIC CHOLECYSTECTOMY"

Submitted to

The Tamil Nadu Dr. M.G.R. Medical University

In fulfillment of regulations for the award of degree of

M.S., DEGREE EXAMINATION

BRANCH –I M.S., (GENERAL SURGERY)

GOVT.KILPAUK MEDICAL COLLEGE

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

CHENNAI - 10

APRIL-2014

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**COMPARITIVE STUDY OF OPEN CHOLECYSTECTOMY VERSUS LAPAROSCOPIC CHOLECYSTECTOMY"** is a bonafide record of work done by **Dr.A.Sekar** in the Department of General Surgery, Government Kilpauk Medical College Hospital, Chennai-10.

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DECLARATION

I, **Dr.A.Sekar,** solemnly declare that this dissertation, titled " A **COMPARITIVE STUDY OF OPEN CHOLECYSTECTOMY *versus* LAPAROSCOPIC CHOLECYSTECTOMY "** is a bonafide record of work done by me in the Department of General Surgery, Government Kilpauk Medical College, Chennai-10, during the period from July 2012 to Dec 2013 under the guidance of my Unit Chief Prof. D. Nagarajan M.S., Government Kilpauk Medical College Hospital, Chennai-600 010.

This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in fulfillment of the University regulations for the award of M.S. (General Surgery) Branch I, General Surgery Examination to be held in APRIL-2014

Place: Chennai

Date:

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ACKNOWLEDGEMENT

I wish to express my sincere gratitude and thanks to **Prof. P. N. SHANMUGASUNDARAM, M.S.,** *Professor & Head of the Department of General Surgery* for having helped me at every step in completing this study successfully.

I wish to express my sincere gratitude to my **Unit Chief Prof D.NAGARAJAN, M.S.,** *Professor of Department of General Surgery* for having helped me at every step in successfully completing this study. I also wish to express my sincere thanks to my Assistant Professors **Dr.C.Malarvizhi, Dr.A.Rajeswaran** for helping me to complete this study.

I also thank ***Professor and Head of the department of Pathology*** and all other teachers who helped me to do this study in time.

I am also very grateful to our *Dean* **PROF.P.RAMAKRISHNAN, M.D.,DLO.,** for his permission to utilize the clinical material for this study.

I also extend my thanks to all my patients for their full co-operation in successful completion of this study.

CONTENTS

ABSTRACT

TITLE: A COMPARITIVE STUDY OF OPEN CHOLECYSTECTOMY VERSUS LAPAROSCOPIC CHOLECYSTECTOMY

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Introduction : Gall bladder disease is the most common curable disease in female of middle age .Laparoscopic cholecystectomy has rapidly become the choice of elective surgery for the treatment of Cholecystitis even though Open Cholecystectomy remains the main modality of surgery in many centres in India. But to become an alternative to open method, it should be safe, less morbid and it should have the possibility of early return to work better than that of open the procedure. This study compares the open cholecystectomy and laparoscopic cholecystectomy with respect to duration of procedure post operative pain,wound infection,requirement of antibiotics and analgesics period of stay in hospital and return to work

In this study we selected 65 patients with gall bladder diseases and divided in to two groups as GroupA and groupB in a random wise 33 in former and 32 in later group. The source of cases were obtained from Govt KMC & GRH chennai, department of general surgery. All the patients were subjected for USG abdomen, Complete blood count , renal function test , CXR, liver funtion test and MRCP in selected cases. Group a subjected for open cholecystectomy and Group B for laparoscopic cholecystectomy and the above mentioned datas were compared. Pre operative antibiotic given

half an hour prior to surgery is more enough to prevent infection except in conditions like diabetes mellitus association with it. Per rectal Paracetamol suppository is enough to relieve in both the groups . Time taken for laparoscopic surgery is lesser than that for open procedure and the period of hospital stay is also less in laparoscopic surgery.

Keywords: Cholecystectomy, laparoscopy, gall stones, cholecystitis, MRCP

INTRODUCTION

It was a gynecologist who introduced the so called 'endoprocedures' in the field of surgery, his name was ***Kurt Semm***, a German national who used his instruments for the removal of ovaries and myomas in 1970s. Only after a successful laparoscopic appendicectomy by Semm in 1982 the general surgeons realized the importance and started applying these instruments in the field of general surgery. The German surgeon ***Eric Muhe*** while pioneering cholecystectomy came to know that a modification of the instrument is needed for gall bladder removal. Since then rest of the world started doing laparoscopic cholecystectomies but, this was the time when abdominal open surgeries were becoming extensive operations with the concept of *'bigger problems need bigger incisions' and laparoscopic technique was criticized by many as "**a futureless technique, circus surgery, the mediatized show of a tight-rope dancer totally careless of the risks for the patients"** due of its lack of publicity.* It was Professor **Perissat**, a renowned surgeon made laproscopic cholecystectomy to gain its credibility. "***The progenitors of the laparoscopic revolution were few… but they were articulate, and the public was receptive"*** as correctly said by Kevic and now the minimally turned in to minimally access surgery which in turn evolved in to SILS and NOTES….

Aim of study

- A Aim of study is to compare the open cholecystectomy and laparoscopic cholecystectomy with respect to
- Duration of procedure
- Post operative pain
- Wound infection
- Requirement of antibiotics and analgesics
- Period of stay in hospital
- Return to work

REVIEW OF LITERATURE

Historical review

Founder of gall bladder surgery is **Jean Louis Petit**.

In 1420→***Antonio Benevieni***, a pathologist gave the first description of gall stones in a woman who died of abdominal pain.

In 1687→**Stal Pert Von DerWeil** explained gall stones during laparotomy done for an acute abdomen.

In 1743→**Petit** removed gall bladder successfully for the first time.

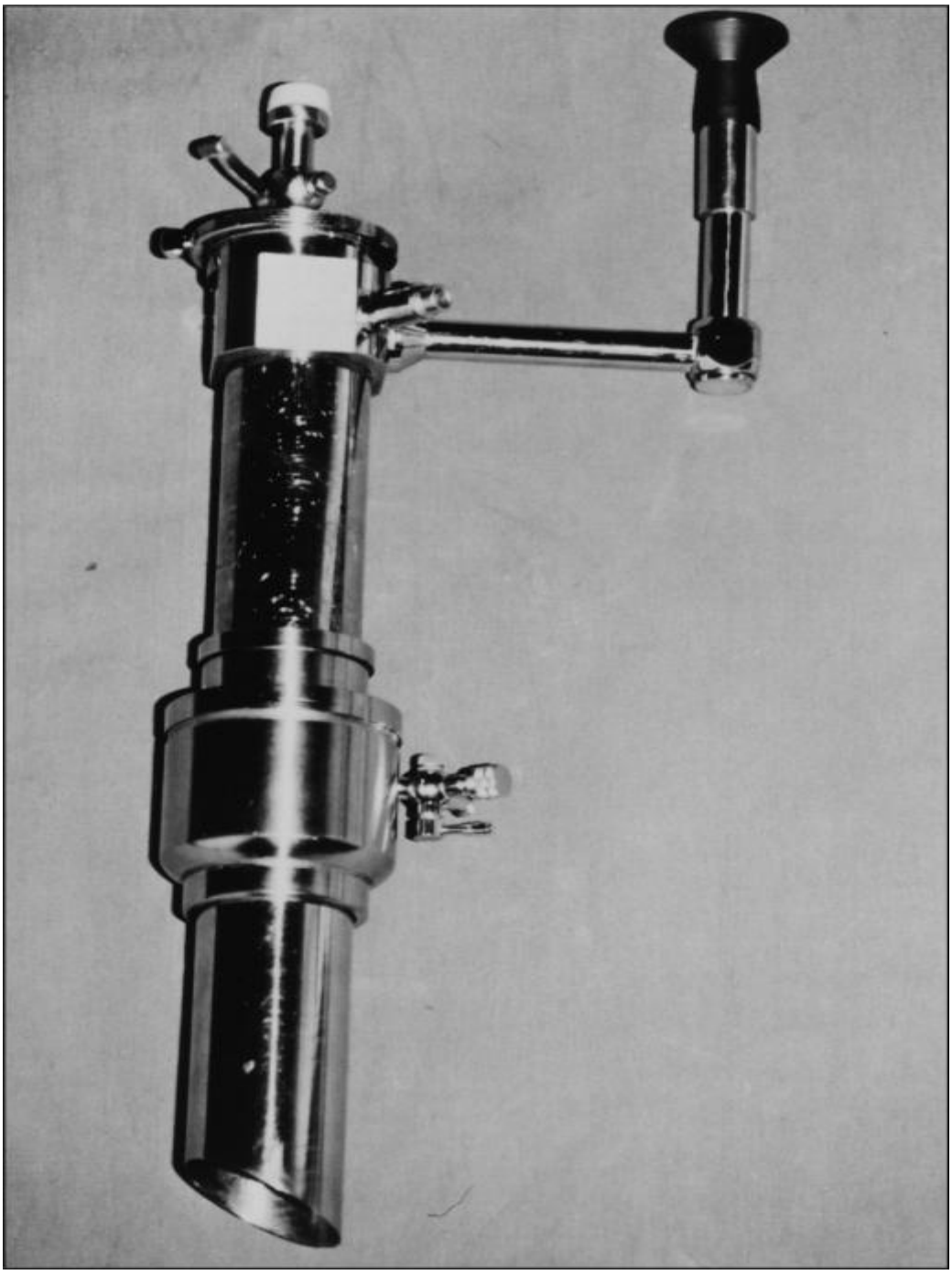
In 1859→ **J.L.W Thudichum** did a 2 staged cholecystectomy.

In 1895→ **Carl Johanan August Langenbuch** did first open cholecystectomy On a 43 yr old male who suffered from gall bladder disease for more than 16 yrs.

In 1987→**Eric Muhe** did the first laparoscopic cholecystectomy in Boblingen, Germany. [23]

In 1985→**Philippe Mouret** in Lyon, France did first laparoscopic cholecystectomy.

In1988→ Americans started doing lap cholecystectomies, since then it started its Journey to take over the conventional open technique. Only about 10% of cholecystectomies were done by laparoscopic method during 1990's which accounted for 88 % during 2006, now almost replacing the open method except in some special situations like gall bladder carcinoma, conversion due to hemorrhage. Revolution of laparoscopy had led the surgeons to have a mobile set up with all the equipments and taking the assistants even to the remote areas to perform laparoscopic cholecystectomies.



Historical laparoscope

Embryology of Gall bladder

Hepatic diverticulum arises from endodermal epithelium of distal end of the foregut during 3rd week of gestation. Proximal end of this diverticulum develops in to liver and the distal end develops in to gall bladder and cystic duct. By 5th week all the elements of biliary system are developed where common hepatic duct and cystic ducts are occluded initially which becomes recanalises later.

The stalk joining these 2 ducts to duodenum becomes common bile duct. By 6th week common bile duct rotates by 180 degrees around duodenum. Sphincter of oddi starts to develop by 10th week and completes by 28th week. Bile pigment starts to form at 13-16 weeks to reach duodenum.

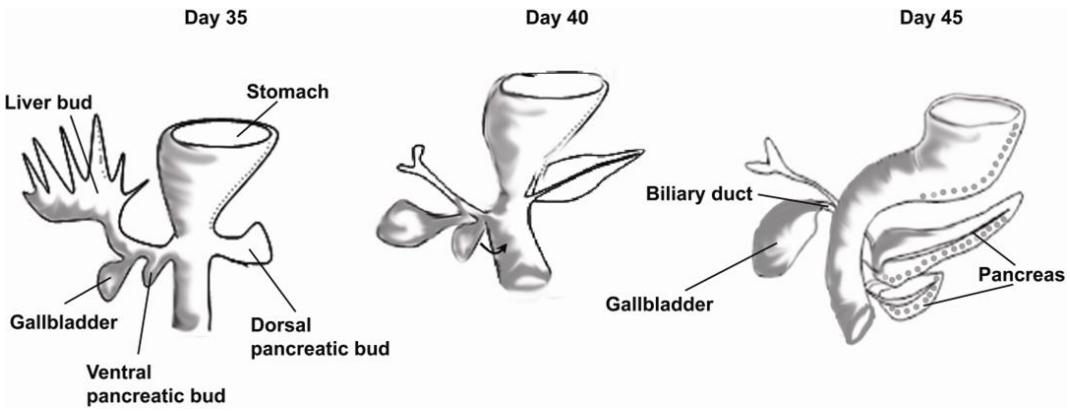


FIGURE 1: DEVELOPMENT OF GALL BLADDER

Developmental anomalies of gall bladder:

Agenesis of the gall bladder [1] occurs due to failure of development of distal part of hepatic diverticulum. Atresia and Hypoplasia are due to maldevelopment and duplication of the gall bladder as well as due to defective revacuolization of the gall bladder which occurs in 1 in 4000 with definite chances of Cholecystitis is seen. Gall bladder Phrygian cap [2] occurs 1%-6% of the population.

Other anomalies are

- Wandering GB
- Multiseptate GB
- Ectopic GB
- Micro gall bladder
- Cholecystomegaly.
- Diverticula of gall bladder.
- Torsion of Gall bladder.

Anatomy of Gall Bladder

The knowledge about the anatomy of biliary system and its anomalies are essential for the safe execution of any surgical planning in gall bladder bed. The normal and abnormal variations of biliary system may lead to major post operative bile duct injuries, especially during laparoscopic procedures. Since, LC has become the gold standard procedure for Gall bladder disease which needs good exposure of calot's triangle and failures are more common when compared to open procedures.

Gall bladder is a Pear shaped organ, located in the gall bladder fossa on the inferior surface of liver covered by peritoneum in all sides except on the fossa. It is 7 to 10cm long with an average capacity to hold 30 to 50 ml of bile as an "*extrahepatic reservoir".* ***Cantlies line***, dividing liver in to right and left runs through the gall bladder. It is mostly extra hepatic but may be intra hepatic in which case it is embedded in the liver parenchyma making the surgical procedure very difficult. Gall bladder is divided in to 4 parts namely,

1) Fundus
2) Body or corpus
3) Infundibulum and

4) Neck

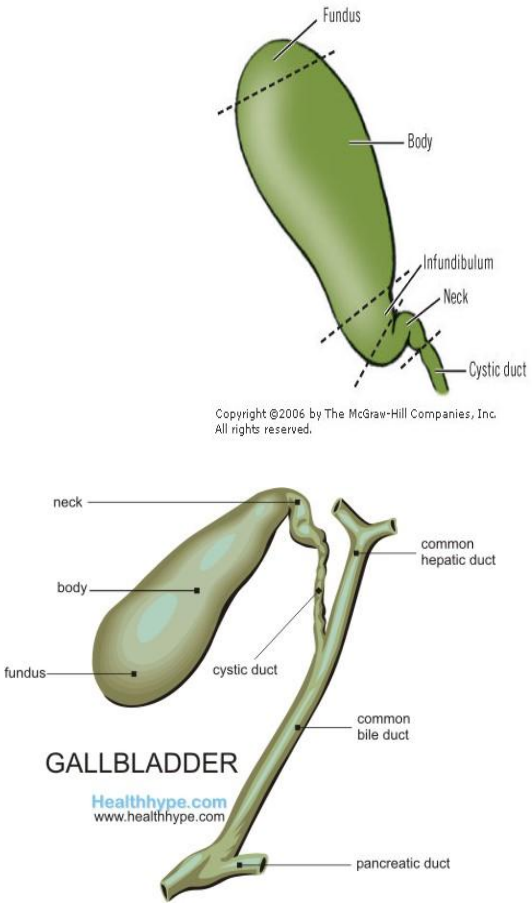


FIGURE: 2 & 3 ANATOMY OF GALL BLADDER

Fundus is that part of GB which projects beyond liver surface. Body lies in the GB fossa of the liver. Hartmann's pouch is that part of Infundibulum which pouches out inferiorly leading to cystic duct. Whenever there is an outflow obstruction GB can expand to hold even 300 to 600 ml of bile.

Calot's triangle [8]: Also called as Hepatobiliary ***triangle*** this is bounded by

- Right side- Proximal part of Gall bladder &Cystic duct
- Left side- Common hepatic duct
- Superiorly- Margin of the right lobe of the liver

This triangle *contains cystic artery and cystic lymph nodes*

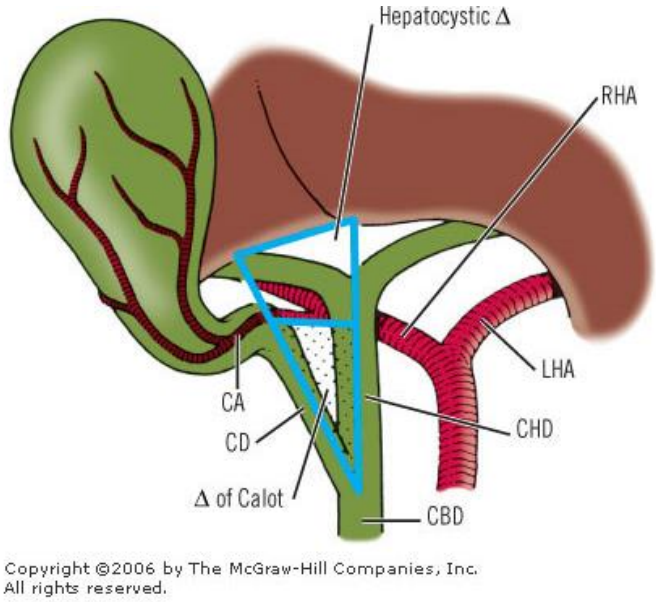


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FIGURE: 4 CALOTS TRIANGLE

Moosman area [9]: This is a circular area of about 30mm in diameter where exactly, CBD, the right hepatic artery, cystic artery and an aberrant hepatic artery are located. As a general rule, no artery more than 0.3 cm in diameter in the triangle will be a cystic artery.

Bile ducts: These include

- Right & Left Hepatic Ducts
- Common Hepatic Duct
- Cystic Duct
- Common Bile Duct

Cystic duct starts from the neck of gall bladder with 3mm diameter and length of about 3cm which is variable. Its mucosa is arranged in a spiral fold known as the ***'valves of Heister'*** and 'sphincter ***of Lutkens'*** which is a sphincteric structure to control bile flow out of gall bladder.

Right and left hepatic ducts joins together to form common hepatic duct which is of 2.5cm in length that finally joins with cystic duct at an acute angle to take part in the formation of CBD.

Common Bile Duct: The length of the CBD is 5.5cm to 15cm [3] and the diameter is 0.4mm to 1.4 cm in adult males and 5cm to 9.5 cm with the diameter between 4–8 mm in females. But the average length of CBD is 7.2cm and diameter of 5.25 mm and has been divided in to 4 parts namely,

- Supra duodenal
- Retro duodenal

- Infra duodenal and Intra duodenal

Supra duodenal CBD: This portion lies along the right border of lesser omentum with portal vein as posterior relation and it is 0.5 to 2 cm in length[4]

Retro duodenal CBD: This part has portal vein on posterior side and common hepatic artery on the lateral side and more over it forms the anterior wall of the "***Foramen of Winslow".***

Infra duodenal or Retro pancreatic CBD: This part passes through the '**Quenu space'** which is bounded by 3 segments of the duodenum and SMV/PV

Intra duodenal CBD: Pierces the duodenum through its medial wall and opens with the main pancreatic duct at the level of the hepato-pancreatic ampulla.

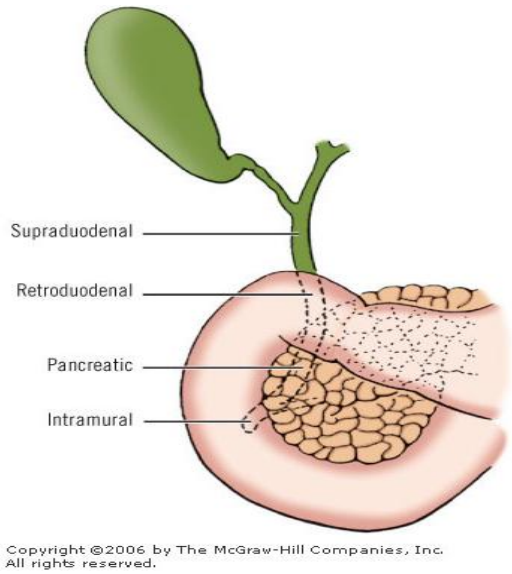

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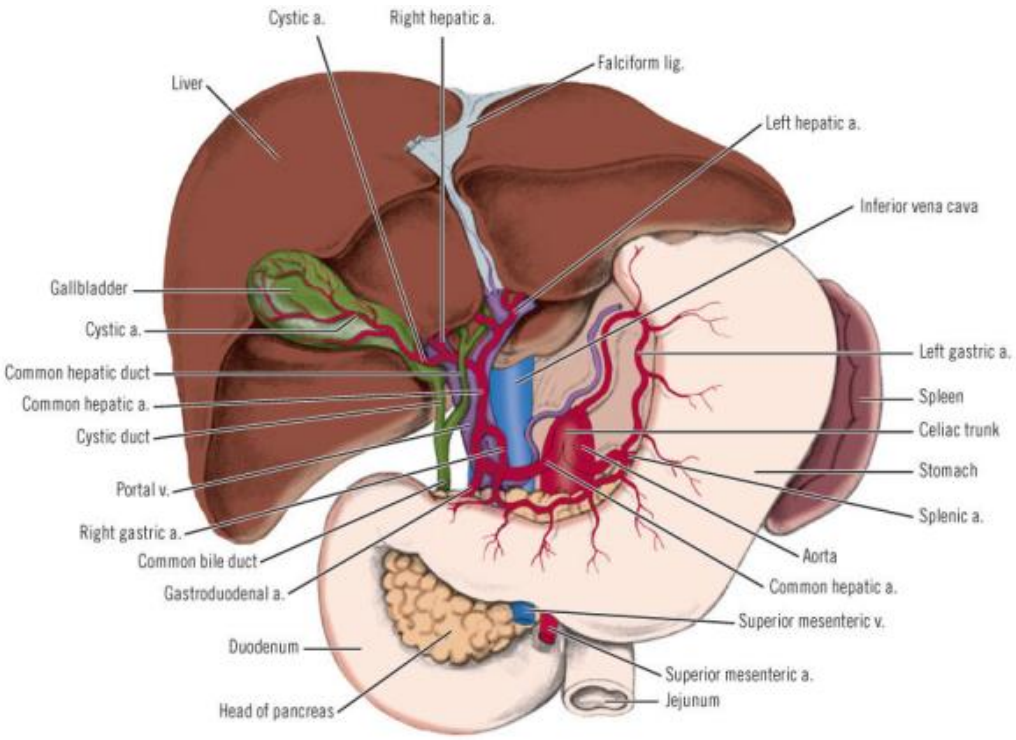
FIGURE 5: PARTS OF COMMON BILE DUCT

Blood supply: The blood supply of gall bladder as follows

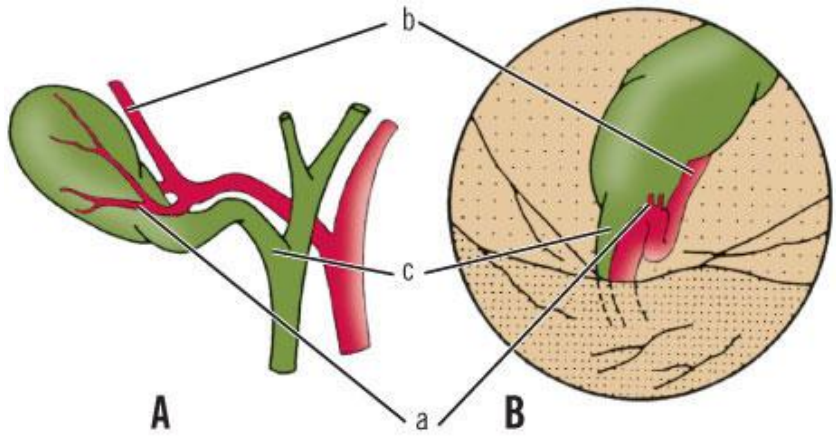
- Gastroduodenal artery
- Hepatic
- Cystic
- Celiac
- Superior mesenteric arteries

This rich blood supply from various arteries safeguard bile duct from injuries during various surgical procedures.



A single Cystic artery which arises from the Right Hepatic Artery seen in 70-80% of cases passes behind the cystic duct[10] and divides in to superficial (passing subserously) and deep branches (passing between gall bladder and gall bladder fossa) to supply gall bladder. As for as biliary tree is concerned part of the duct above the duodenal bulb eg. CBD, cystic duct and CHD is supplied by cystic artery (***calots arteries***) and right hepatic artery where as that part, inferior to duodenal bulb is supplied by posterosuperior, pancreaticoduodenal and gastroduodenal arteries. Moosman described in

cadaveric specimen that cystic artery is of 0.3mm in diameter and no artery with more than 0.3 mm in diameter in the calot's triangle will be cystic artery.



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Anatomical variants of cystic arteries:

Double cystic arteries [5]: cystic artery failure to divide in to branches Occurs in 15% of cases **Caterpillar" or "Moynihan's" hump** (Aberrant right hepatic artery) Originates from the SMA or abdominal aorta passes through the calot's triangle, running parallel to the cystic duct giving origin to multiple

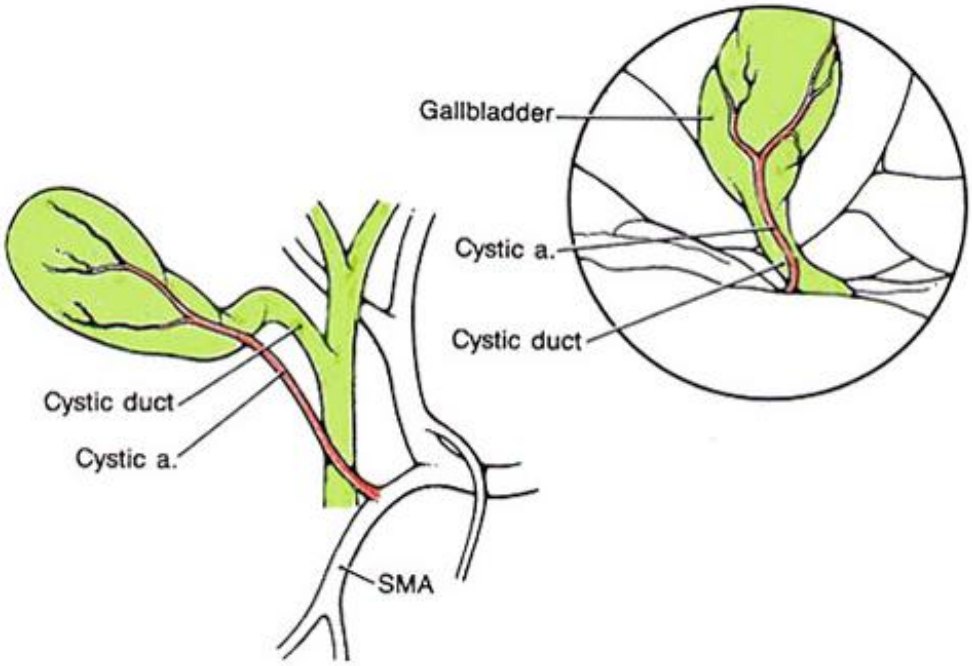
small cystic artery branches rather than a single cystic artery seen in 2-6% of cases.

Left hepatic artery: The cystic artery can arise from the *left hepatic artery*, does not pass through the calot's triangle seen in 1% of cases. Here cystic artery passes through the liver.

Low-lying cystic artery: Arising from gastroduodenal artery and passes inferior to the cystic duct through the cholecysto duodenal ligament outside the calot's triangle. This rare anomaly is seen in 5% of the cases.

Clinical importance:

"*Aberrant course of cystic artery is clinically important during cholecystectomy especially lap procedure, causing a massive hemorrhage that could convert the procedure into open technique or causing great confusion to the surgeon to delineate the arterial structures from the biliary radicals leading to disastrous biliary leak, peritonitis and sepsis. Inadvertent right hepatic artery ligation in cholecystectomy has been associated with liver ischemia, necessitating hepatic lobectomy sometimes".*



Venous drainage:

Venous drainage of the gall bladder varies for open free surface, peritoneal part and hepatic surface accordingly. From the peritoneal surface, one vein usually drains the fundus and body and other veins drains the neck and upper portions of the cystic duct as well as the hepatic ducts. These veins open in to Liver along with veins from CBD.[6] The hepatic surface of the gallbladder is drained by numerous small veins passing through the gallbladder bed that break up into capillaries Within the liver. They do not form a single "cystic

vein." Veins from the hepatic surface drain directly into the liver. Veins on the free surface open directly or follow the hepatic ducts into the liver.

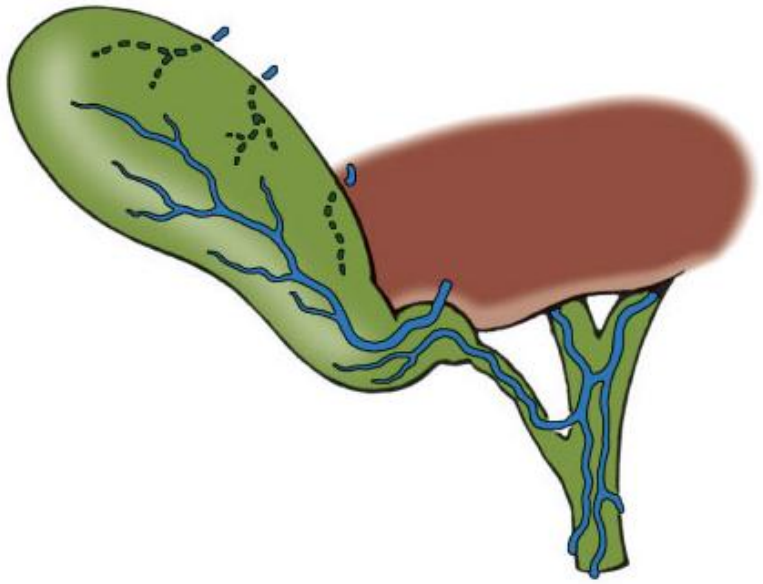


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FIGURE 9: VENOUS DRAINAGE OF GALL BLADDER

Lymphatic Drainage: Subserosal and sub mucosal lymphatic's of the gall bladder drain in to the cystic lymph node of Lund that lies in the junction between cystic duct and common hepatic duct. These lymphatic's finally drain in to the hilum of liver that is acting as a channel for spread of carcinoma gall bladder.

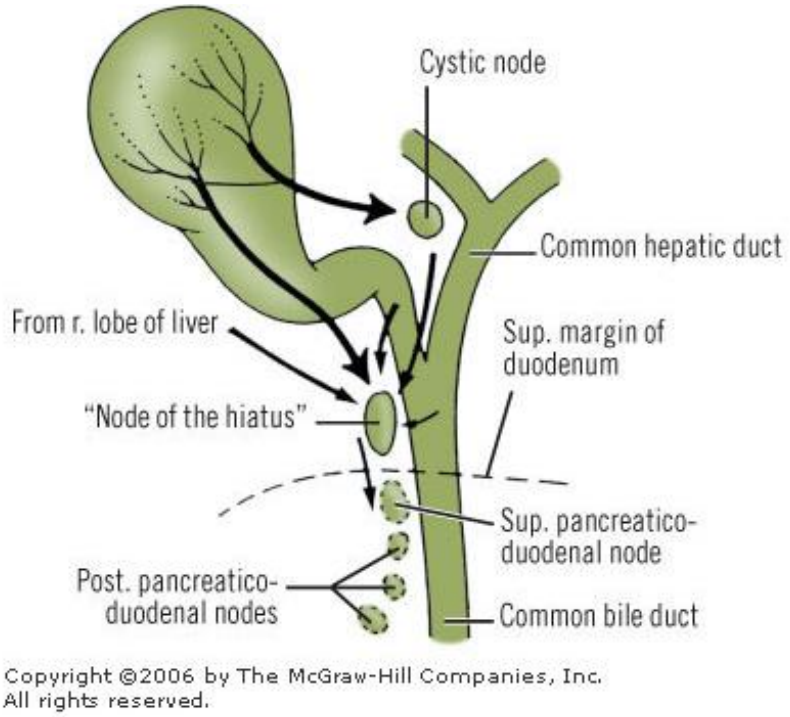

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FIGURE 10: LYMPHATIC DRAINAGE OF GALL BLADDER

As for as the lymphatic drainage is concerned Ito et al, has divided into 3 pathways.[7]

1. ***Cholecystoretropancreatic pathway-***

   Is the main pathway which terminates in retroportal lymphnodes.

2. ***Cholecystoceliac pathway-***

   Terminates in celiac lymphnodes found on hepatoduodenal ligament.

3. ***Cholecystomesentric pathway*** –Passing left and front of portal vein and terminates at superior mesenteric Lymph nodes, close to the left renal vein all the pathways converge with the abdomino-aortic lymph nodes.

Physiology of bile secretion:

Function of gall bladder is *storage, concentration* and *supply*ing bile whenever the fatty meal is ingested. Bile is secreted by the Hepatocytes, stored in the gall bladder and released in to the intestine whenever Cholecystokinin (CCK) is secreted by the intestinal mucosa in relation to food intake. Gall bladder fills in the retrograde mechanism due to increased tonic activity of sphincter of Oddi [11] and increased pressure within the common bile duct. As already mentioned gall bladder can expand up to 10 times of its original capacity to hold more than 300ml to 600ml. Bile that is emptied by the gall bladder is also 10 times more concentrated than that secreted by the liver, aided by active NaCl transport system by the epithelium. The primary bile salts are cholic acid and deoxycholic acid which are reabsorbed in the terminal ileum with net loss of less than 5% in stool every day.

Components of bile:

Water	97%
Bile salts	0.7%
Bile pigments	0.2%
Cholesterol	0.06%
Inorganic salts	0.7%
Fatty acids	0.15%
Phosphatidylcholine	0.2%
Fat	0.1%
Alkaline phosphatase	….

Gall bladder disease:

- Cholelithiasis –symptomatic
- Acute Cholecystitis
- Chronic Cholecystitis
- Acalculous Cholecystitis
- Empyema of gall bladder

- Mucocele of gall bladder

Cholelithiasis:

Stones are formed due to changes in the concentration process that increases the calcium and cholesterol components followed by decreased motility and bile stasis. The process of gall stone formation is complex and the pathophysiology is unclear. In India 80% of stones are pigment stones, especially brown pigment. Predisposing factors for the gall stone diseases are-

- Obesity
- Rapid weight loss
- Pregnancy
- Diabetes mellitus
- TPN & Octreotide use

These conditions are associated with increased hepatic secretion of cholesterol [4] and are a major risk factor for the development of gall stones especially cholesterol stones. About 500 to 1000ml of bile is secreted per day, majority of which is reabsorbed by the entero-hepatic circulation. Gall stones can be

symptomatic or asymptomatic, when the symptoms are absent in the presence of documented gall stones.

Pathogenesis of gall stone formation:

Gall stone disease is polygenic and many factors contribute in its Pathogenesis, the primary factor is the excess biliary cholesterol in comparison to the solubilizing bile salts or phospholipids. Other factors are increased intestinal loss of bile salts and cholesterol. Genetics also plays a role in stone formation. Adenosine triphosphate–binding cassette (ABC) transporter which regulates the transport of cholesterol is located in the canalicular membranes of hepatocytes and the genetic disorders associated with this gene also leads to gall stone formation. One more genetic problem is increased expression of ***Niemann-Pick C1 –like protein*** which also plays a role in pathogenesis. A mutation in the ***ABCB11*** may lead to progressive intrahepatic cholestasis and gall stone formation.

Supersaturation and increased lithogenicity are playing a vital role in pathophysiology of cholesterol gallstone formation. Nucleation of cholesterol monohydrate crystals from the multilamellar vesicles is the crucial step

here.[15] Obesity, OCP and high caloric diet pour more amount of cholesterol in to the bile and ultimately all these lead to thick bile and dysmotility of gall bladder. So in gall stone disease removing the stones alone will not cure the disease unless gall bladder is removed.

Types of gall stones: There are three types namely

- Cholesterol stones
- Pigment stones
- Mixed stones

Differences between cholesterol and pigment stones are:

Cholesterol stones	Pigment stones
>70% cholesterol	<30% of cholesterol
Contains calcium salts, Bile acids , Bile pigments &Phospholipids	Calcium bilirubinate, Calcium palmitate, Calcium stearate & Cholesterol
Formed in gall bladder	Formed in bile duct
Abnormal motility is the cause	Causes - Bile stasis & infection & FB's

Pigment stones:

Two types of pigment stones are encountered, they are ***brown*** and ***black.*** **Brown pigment stones** are more common in East Asia which are formed in the bile Ducts [1] because of bile stasis and bile infection and are usually less than 1 cm in diameter. They are brownish-yellow, soft, and often mushy. They are composed of *calciumbilirubinate,* [14]*calcium palmitate* and *calcium stearate* and varying amounts of cholesterol and protein and usually associated with chronic bacterial infection of the bile ducts and causing dangerous cholangitis. When the deconjugation of bilirubin deglucronide occurs this leads to stone formation by the action of Beta glucronidase which is a component of bacteria and deposition of insoluble unconjugated bilirubinate on the foreign bodies or parasites.

Bacteria & parasites forming stones are

- Escherichia coli
- Bacteroides
- Clonorchis sinensis and
- Ascaris lumbricoides

Black pigment stones are formed within the gall bladder and they are sterile and usually small, brittle, black, and sometimes speculated and composed of calcium bilirubinate mixed with calcium carbonate and calcium phosphate. In over all, about 20-30% of stones are black. Usually associated with chronic hemolytic disorders like sickle cell disease,[12] Hereditary spherocytosis but the gall bladder dysmotility & bile stasis *do not play* a role in the stone formation. It is more common in western countries. These stones are very rare in young age but, the incidence increases progressively as the age advances [3].

Cholesterol stones

In western countries 80% of the gall stones are cholesterol stones. But in India it constitutes very less when compared to pigment stones. It is more common in multiparous women due high exposure to the progesterone levels during pregnancy. Because progesterone leads to decreased gall bladder contractility with subsequent stasis and concentration of bile resulting in stone formation. In nulliparous women it causes gall stones by pouring more amount of cholesterol due to high concentration of estrogen. Hereditary factors also play about 25 % of role in the stone formation. Formation of stone alone is not sufficient to produce symptoms but it has to obstruct the bile duct

lumen to become symptomatic. Stasis and bacterial invasion can cause infection.

Risk factors include:

- Obesity
- Pregnancy
- Gallbladder stasis
- Drugs
- Heredity

The metabolic syndrome of truncal obesity, insulin resistance, type II diabetes mellitus, hypertension, and hyperlipidemia are associated with increased hepatic cholesterol secretion and is a major risk factor for the development of cholesterol gallstones.

Complications of gall stones:

Most of the gall stones are asymptomatic and does not need treatment until they complicate. Impaction of gall stones in gall bladder, bile duct or in the intestine may complicate the clinical picture

In the gall bladder:

- Silent stones
- Acute Cholecystitis
- Chronic Cholecystitis
- Gangrene & perforation
- Empyema
- Mucocele
- Carcinoma[16]

In the bile ducts:

- Obstructive jaundice
- Cholangitis
- Acute pancreatitis

In the intestine:

Gall stone ileus **(Bouveret's syndrome):** Sometimes small CBD stones can migrate downwards in to the duodenum and ileum causing intestinal obstruction . Sometimes it can causes acute pancreatitis also by obstructing the infra duodenal CBD. Obstruction by gall stones in the duodenum or in small bowel may predisposes to inflammation. Because of recurrent

inflammatory process with fibrosis the gall bladder shrinks. In the absence of resolution of inflammation gallbladder may perforate leading to peritonitis and empyema of gall bladder. If the stone is obstructing the bile duct which can be either due to a primary stone that is formed within the CBD or slipping from the cystic duct (secondary stone) may cause obstructive jaundice or inflammation extending in to intrahepatic biliary radicals leading to a devastating cholangitis.

Cholecystitis:

This is inflammation of the gall bladder due to various causes. Can be divided in to 1) Acute Cholecystitis **2)** Chronic Cholecystitis 3) acute on chronic Cholecystitis

Acute cholecystitis

This is " acute inflammation of the gall bladder with edema and subserosal hemorrhage due to calculi obstructing in the cystic duct". if the obstruction is not relieved it may lead to acute gangrenous cholecystitis. Patients present

with fever, pain and tenderness in right subcostal region with positive " Murphy's sign".

Chronic cholecyctits

This is "recurrent attacks of biliary colic due to cystic duct obstruction by a calculi but without causing acute cholecystitis can cause some inflammation and scarring the neck of the gall bladder and cystic duct". Pain due to chronic cholecystitis generally does not last more than a few hours. If it exceeds 24 hrs with fever it suggests acute or acute on chronic cholecystitis.

It is secondary to gallstones in 90 to 95% of cases where as in less than 1% of the cases a *tumor* may obstruct the cystic duct. *Obstruction of the cystic duct by a gallstone is the initiating event* [17] that leads to gallbladder distention, inflammation, and edema of the gallbladder wall. Degree of inflammation is probably related to the duration of obstruction. Initially, acute cholecystitis is an inflammatory process, mediated by lysolecithin, as well as bile salts and platelet-activating factor. Secondary bacterial contamination leads to gallbladder wall thickening and reddish with subserosal hemorrhages. Pericholecystic fluid is present and the mucosa may show hyperemia and

patchy necrosis. In severe cases, about 5 to 10%, the inflammatory process progresses to ischemia and necrosis of the gallbladder wall. In some cases the gallstone is dislodged and the inflammation resolves. [18]

Courvoisier's law:

Jaundice in a case of cholecystitis is rare and in the setting of obstructive jaundice if the gall bladder is palpable it is not due to stone disease rather may be due to a growth obstructing the carcinoma head of the pancreas or of CBD.

Acalculous cholecystitis: This is acute inflammation of the gallbladder without gallstones, developing in critically ill patients in the intensive care unit who is on parenteral nutrition with extensive burns, major operations, sepsis, multiple traumas, and prolonged illness with multiple organ system failure. The gallbladder distended with bile stasis and ischemia occurs, showing edema of the serosa and muscular layers, with patchy thrombosis of arterioles and venules. [19,20] The signs and symptoms are similar to acute calculous cholecystitis, with right upper quadrant pain and tenderness, fever and leukocytosis..

Clinical features:

Patient may present with upper abdominal pain more on the right subcostal region radiating to the back or to the shoulder with H/O dyspepsia, fever and vomiting. Pain usually follows a fatty meal, severe in nature for first half an hour but lasting for 1 to 5hrs and ***"Murphy's sign"*** is positive. In between two attacks patient's clinical picture improves until the next attack. When the pain lasts more than 24 hours, an impacted stone in the cystic duct or acute cholecystitis should be suspected. An impacted stone will result in what is called *hydrops of the gallbladder*. The bile gets absorbed, but the gallbladder epithelium continues to secrete mucus and the gallbladder becomes distended with mucinous material. Presence of jaundice should always suspect biliary stone obstruction. During attacks there is guarding, rigidity on the right subcostal region and sometimes a mass will be palpable due to omentum covering the gall bladder. This process may be limited by slippage of stone back in to the gall bladder. Such episodes occur repeatedly for a few weeks with complete symptom free period for some months.

Investigations :

USG abdomen:

Is the first choice of investigation and it is non invasive. It clearly defines the stone and its site with size within the biliary tree. Also gives details of gall bladder wall thickness and size of the CBD.

Endoscopic USG:

This is an ultrasound with an endoscope for detecting stones and growth in the periampullary region.

LFT:

Serum bilirubin, AST, ALT, Alkaline phosphatase and proteins. BT, CT with PT, INR.

MRCP:

It is becoming standard for evaluation of anatomy of the biliary system which tells clearly about the aberrant right hepatic artery and the biliary duct anomalies. Here contrast is not necessary which is very useful in patients with compromised renal function.

ERCP:

Nowadays this is used mainly as a therapeutic aid to relieve obstruction in case of obstructive jaundice caused by stone in CBD and also in stricture. Because it causes acute cholangitis and pancreatitis, its usage as a diagnostic aid is restricted.

Operative Biliary Endoscope:

It is also called as '*Choledochoscopy'* which is a flexible endoscope passed in to the CBD to identify stone and to remove it under direct vision.

Biliary Radionuclide Scanning (HIDA Scan)

This is a non invasive evaluation of the gallbladder, bile ducts, and duodenum. Both anatomy and function of gall bladder are well studied by this method. ^{99m}-Technetium-labeled derivatives of dimethyl imino diacetic acid (HIDA) which is injected intravenously and cleared by the Kupffer cells in the liver, and excreted in the bile. This is done in fasting state where the uptake by the liver is detected within 10 minutes and the gall bladder, biliary radicals in 60 minutes. This is mainly used for diagnosing acute cholecystitis, which shows a prompt filling of the common bile duct and duodenum and non visualization of gall bladder.

Other investigations are :

- Renal function test with serum electrolytes
- Complete blood count with HB
- CXR PA VIEW
- ECG

TREATMENT

Treatment of gall stone diseases

All gall stones need not be treated by surgery.

I. Non operative medical treatment

II. SURGICAL TREATMENT

Medical management: Options available are dissolution with oral bile salt therapy, contact dissolution and ESWL.

Surgical options:

Since the introduction of laparoscopic cholecystectomy, the number of Cholecystectomies performed in India has increased with exceptions like coagulopathy, severe chronic obstructive pulmonary disease, end-stage liver disease and congestive heart failure. Nowadays because of better instruments and operative technique laparoscopic cholecystectomy has become the treatment of choice and the conversion rate for elective laparoscopic cholecystectomy has come down to 5%, but with acute cholecystitis it is as high as 30%.

The most common indication for cholecystectomy are,

- Biliary colic due to cholelithiasis and chronic cholecystitis
- Obstruction of the cystic duct by a gallstone causing acute cholecystitis
- Pancreatitis and Cholangitis caused by Stone obstructing the pancreatic duct and/or distal CBD and causing pancreatitis and Cholangitis.

Methods of Cholecystectomies

A. Open Cholecystectomy

B. Laparoscopic Cholecystectomy

Open Cholecystectomy:

Indications are

- Poor cardiac or pulmonary reserve
- Suspected gall bladder
- General peritonitis
- Penetrating trauma
- Fistulous disease of gall bladder
- Previous upper abdominal surgery
- Cirrhosis of liver with PHT
- 3rd trimester of pregnancy

Incision:

- Right Sub costal or Kocher (most commonly employed) or
- Right Transverse or
- Mayo Robson incision

Position:

Supine with head end up and right side up, preferably a sand bag under right Shoulder

Methods:

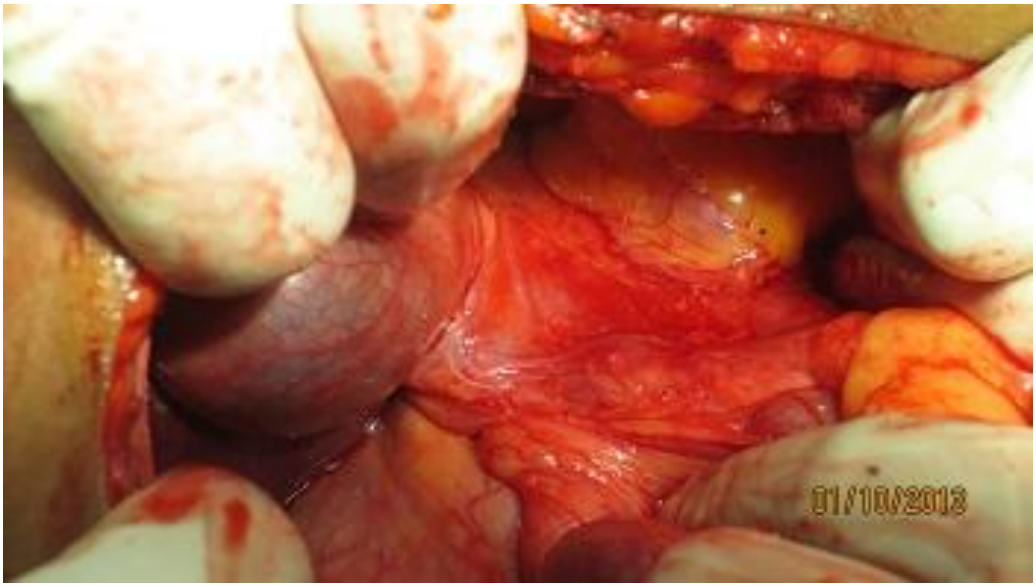
- Duct first method → when calots triangle area is clear
- Fundus first → when the calots triangle is not clearly defined due to acute inflammatory pathology

Procedure:

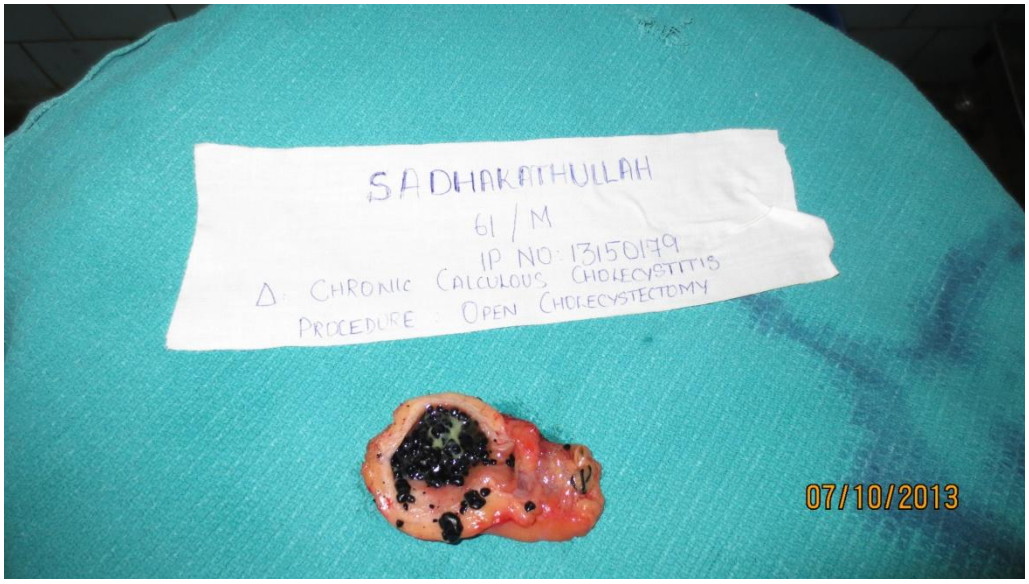
After opening the abdomen it is explored to exclude other intra-abdominal pathology and a hand is passed over the right lobe of the liver to allow air to enter the subphrenic space. Moist pads are kept inferiorly to push colon downwards, one to push the stomach medially and one more pack for the assistant to give traction caudally so that the cystic duct is stretched and the calots triangle area is clearly visualized. When the gall bladder is tense with more bile it can be decompressed to avoid uncontrolled spillage of infected bile during dissection. The fundus of the gallbladder is grasped with a Kelly clamp and retracted caudally and an another to grasp Hartmann's pouch and pulled away from the cystic duct.

Dissection around Calot's area:

The peritoneum overlying Calot's triangle is incised and the dissection is continued close to the gallbladder, displacing the cystic node and the fatty areolar tissues to expose the cystic duct and artery.



Picture : showing calot’s region



Picture: removed gall bladder

The cystic artery is identified and traced onto the gallbladder far enough and confirmed that it is not right hepatic artery and divided after ligating in two place with 2-Vicryl.Now by blunt dissection cystic duct is identified and ligated with a silk in three places close to the gall bladder and transected. Then the fundus is held with a sponge holding forceps and by applying a mild traction towards right and upwards, gall bladder is released from the liver surface with a monopolar diathermy cautery.

Laparoscopic Cholecystectomy

Laparoscopic Instrument:

- High-quality video laparoscope
- 300-W light source
- High-resolution monitors
- Veress needle
- High-flow carbon dioxide insufflator
- Four Trocars (two 10-mm Trocars and two 5-mm Trocars)
- Monopolar Electrode L-hook
- Suction and Irrigation
- Fine-tipped dissector
- Two gallbladder graspers
- Large gallbladder extractor
- Pair of scissors
- Clip Applicator
- A 10-mm stone retrieval grasper
- Bi polar cautery
- Endo pouches (to collect gall stones)
- LT 200 & LT 300 clips

A good operating team members are very much needed to execute a successful laparoscopic cholecystectomy. It should include

- A well trained surgeon
- First assistant surgeon with equivalent skills
- Camera operator
- A scrub Nurse

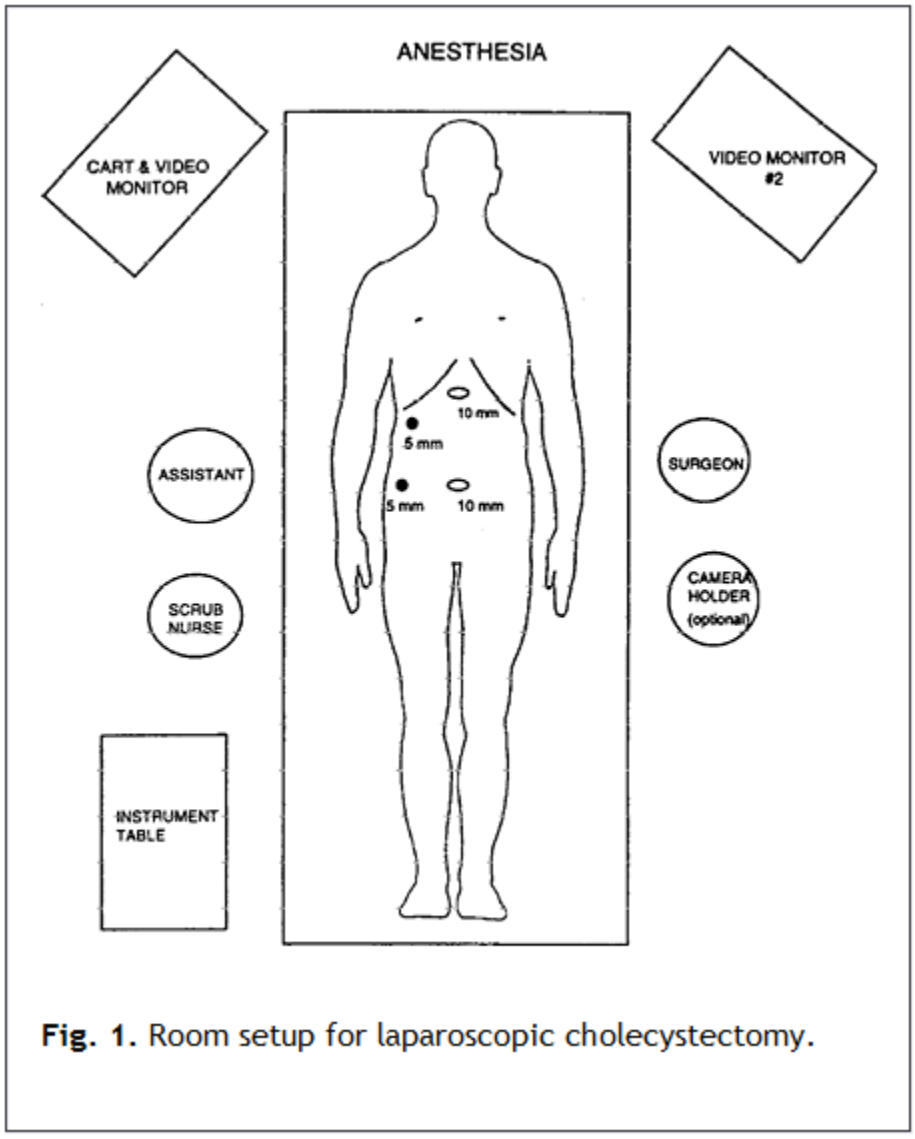


Fig. 1. Room setup for laparoscopic cholecystectomy.

Preoperative antibiotic: Inj. Cefotaxime 50mg /kg body weight, IV stat.

Anaesthesia **:** ETGA

Position of surgeons **1)** Surgeon stands to the patient's left

2) First assistant stands to the patient's right

3) Camera operator stands at the surgeon's left

Pneumoperitoneum **:** I) Closed method (veress needle)

II) Open method (Hasson method)

Carbon dioxide pneumoperitoneum at 15 mm Hg pressure is the agent of choice for many reason like low toxicity, low risk of gas embolism, rapid reabsorption, low cost and ease of use. Ideal insufflator should be able to deliver 8 to 10L/min with a minimum acceptable flow rate of 6L/min. It regulates flow rate, monitors intra abdominal pressure and stops delivering carbon dioxide whenever the pressure exceeds predetermined level of 12 to 15mm Hg.

Procedure :

After induction of anesthesia, positioning and draping, pneumoperitoneum is created with Veress needle through umbilicus and attached to the insufflators and look for uniform distension of the abdomen, make tapping over abdomen. Flow rate is allowed up to 15mmHg is achieved. Entering the

abdominal cavity by direct vision and the trocar introduced through which carbon dioxide is insufflated to create pneumoperitoneum.

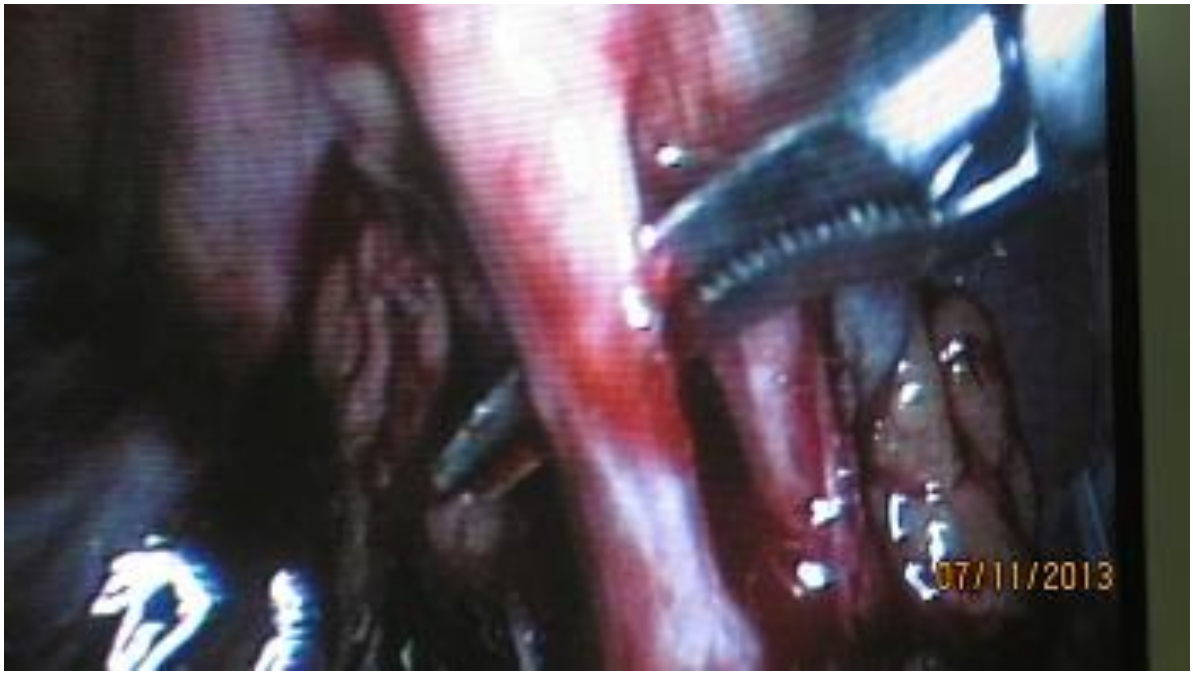
Placement of Trocars:

With the help of an assistant who lifts and holds the abdominal wall ,a 10 mm trocar introduced through umbilical wound in to the peritoneal cavity by screwing movement. Then either a 0-degree or 30-degree telescope is passed through this and thorough laparotomy of the abdomen including calots area is done. The advantages of 30 –degree telescope is that it can be used to view posterior wall of gall bladder, CBD and calots triangle. Next, an 10mm trocar passed through the epigastrium at the right of the falciform ligament. The third trocar is a 5-mm trocar is placed in the midclavicular line 2 to 3 cm below the costal margin and the fourth trocar in the anterior axillary line.

Dissection of calots triangle area

Usually the first assistant uses a traumatic grasper to hold the fundus of the gall bladder and retracts it towards the right dome of diaphragm while the second assistant grasps infundibular part and retracts towards right lateral side. This aids in the better visualization of the calots triangle. Even after this if Porta hepatis is not clearly seen then 5^{th} trocar can introduced from

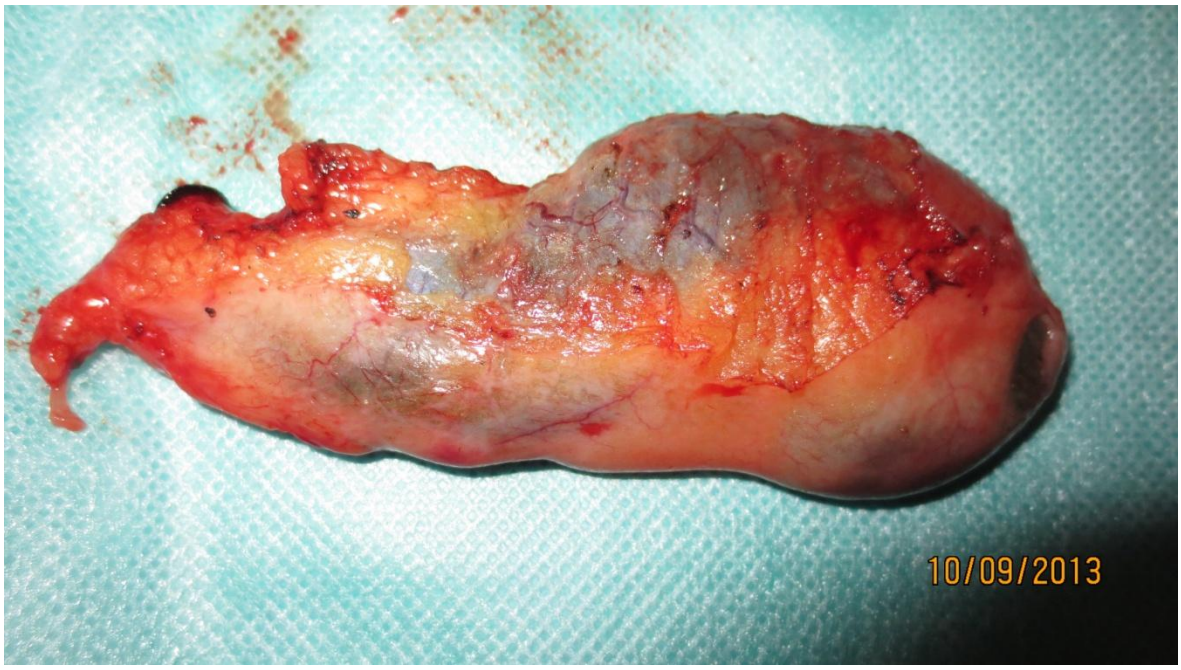
patients left side. Now the surgeon can dissect the peritoneum covering of the area by using a retract infundibulam with his left hand. The peritoneum is teased towards the common bile duct till cystic duct, cystic artery and calots lymph nodes are identified. If 30-degree telescope is used, dissection on the posterior aspect of the cystic duct is easier. Once this is done, two clips proximal and one clip distally close to the gall bladder is applied and transected with a scissors.



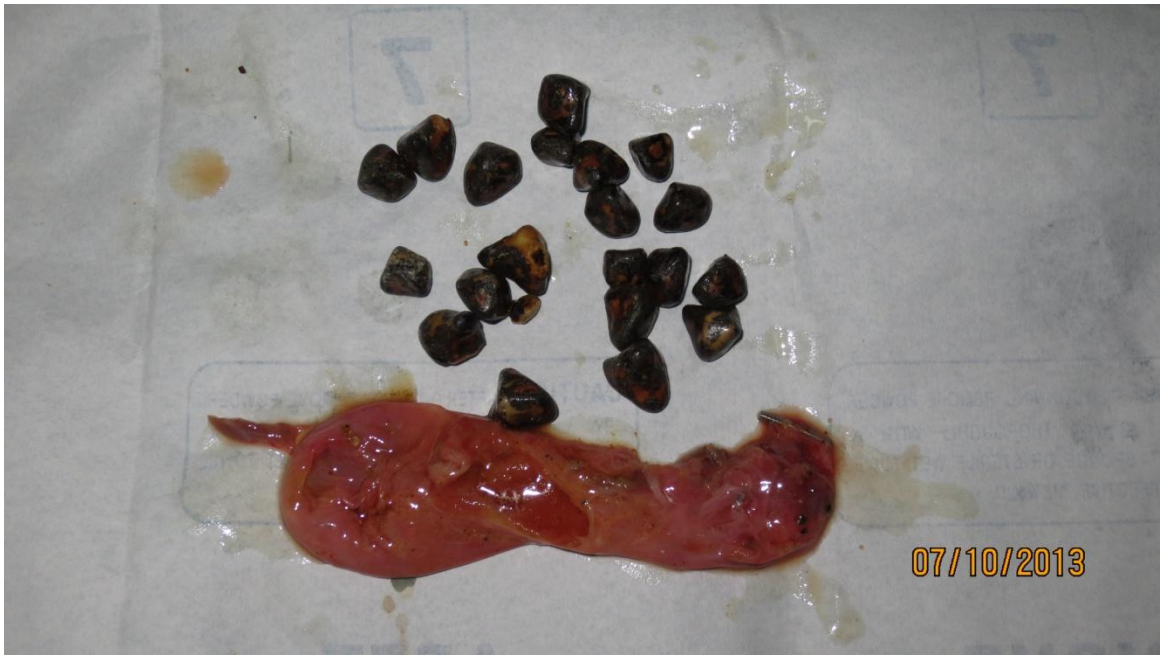
Then, cystic artery also clipped in the same way and disconnected. Dissection of the gall bladder from the liver surface is done with monopolar cautery. If bleeding occurs this can be cauterized.

Removal of the Gallbladder:

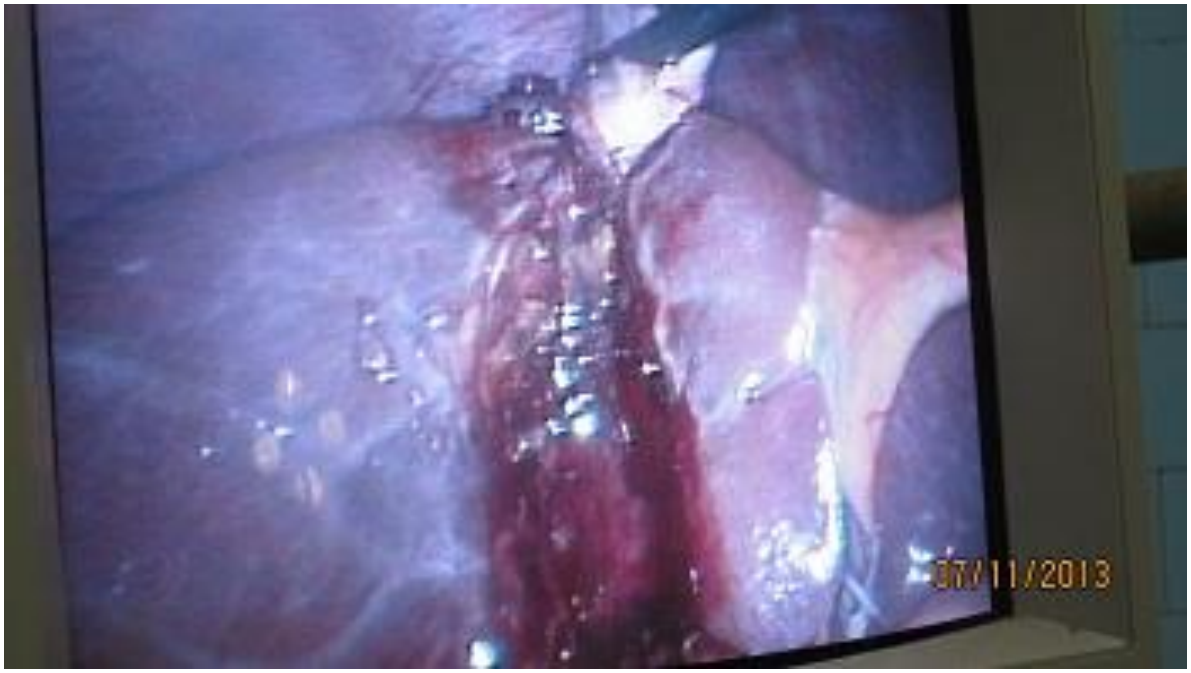
The telescope is passed through the epigastric trocar, the gallbladder is grasped with a 10-mm grasper introduced through the umbilicus, and the gallbladder and trocars are removed. If there is any difficulty in delivering the gallbladder suctioning of the bile can be done to reduce the size and then gall bladder is removed. If the stones are large enough to create problem, the fascial muscle plane is enlarged with a Kelly clamp or the fascial incision is enlarged to remove it. If there is any spillage of the stones, these can be retrieved in a sac and removed.Warm saline can be used to wash the peritoneal cavity and the blood clots must be removed as much as possible. In case of doubt regarding the hemostasis and bile leak, the drain can be kept and brought out through one of the 5 mm port wound site.



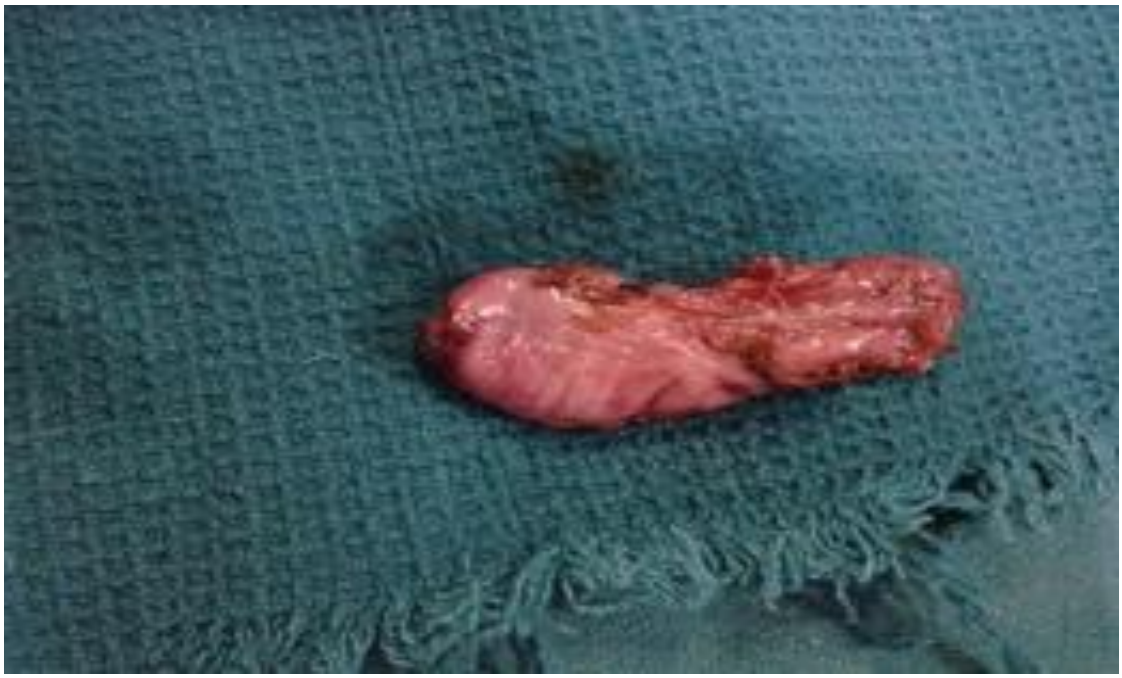
Picture : showing removed gall bladder

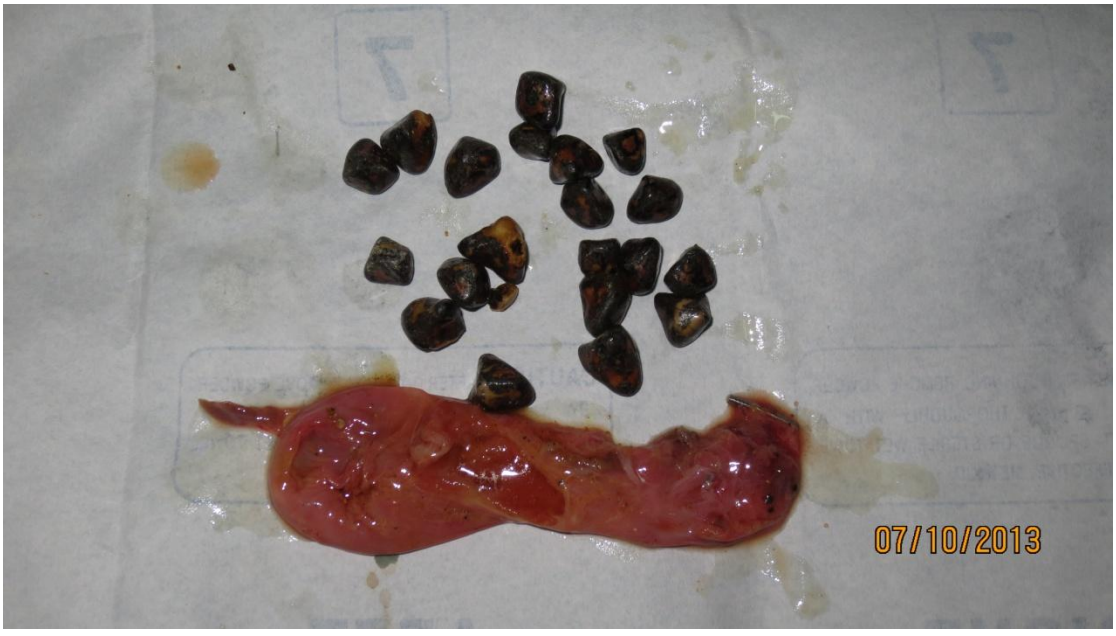


Picture: removed gall bladder



Picture : gall bladder bed after its removal





Picture : showing gall bladder and stones

CLOSURE:

Under the vision all the ports are removed one by one and the pneumo peritoneum is let out. The rectus sheath is closed with 2-0 Vicryl in ‘figure of 8 suture’ at 10mm port sites and skin closed with subcuticular stitch and sterile- strips placed.

Challenges

Hydrops of the Gall bladder: Some times the gall bladder may be too tense due to hydrops of the Gall bladder that cannot be grasped, in which it has to bed decompressed by cholecystostomy. Once reasonable quantity of bile is let out of it and sucked out the puncture site can be grasped with a grasper.

Wide mouthed cystic duct:

This is more challenging because of the short and wide cystic duct where application of clips will reach a cross the cystic duct and even it is done, the risk of narrowing of the CBD is always there.

Other techniques for closing the cystic duct:

- If the duct is long and wide, it can be transected and an Endo loop can be applied to the cystic duct stump.
- Two ties can be passed around the cystic duct in continuity and secured with

Extra corporeal knotting techniques.

The cystic duct is transected with an endoscopic stapling device, or it is simply divided and over sewn with an intra corporeal suturing technique.

Post operative care:

Before extubation, PARACETAMOL 250mg suppository per rectally used for pain relief. Parenteral antibiotic should be continued for the procedure exceeding 3Hrs and with comorbid illness like DM. Started on oral feeds after 6 hrs for all the cases of laparoscopic procedure but for open cases kept under NPO for 24 hrs and then orals started after confirming the return of bowel sounds.

Conversion to open method

The aim of conversion to an open method is to avoid complications and it must be decided before its occurrence. The rate of conversion is 5% in the hands of an experienced hepato biliary surgeon. Indications for conversion are

- Dense adhesion on the calots area
- Necrotic gall bladder
- Carcinoma gall bladder
- Difficult dissection of neck of gall bladder leading to hemorrhage
- Delay in identifying the junction of gall bladder with cystic duct within 30 minutes due to any reason

Risk factors are

- Male gender
- Obesity
- Cholecystitis
- Choledochlithiasis

Complications of Laparoscopic Cholecystectomy

- Veress needle or trocar injury to mesentery, duodenum or bowel
- CBD injury causing stricture and obstructive jaundice
- Bleeding
- Bladder and Ureter injury
- Bile leak
- Infection
- Cholangitis leading to septicemic shock
- Infection
- Subphrenic abscess
- Gall stone spillage
- Deep vein thrombosis
- Pancreatitis
- Conversion to open procedure
- Ileus
- Subcutaneous edema & emphysema
- Biliary fistula
- Port site hernia

How to manage complications?

Percentage of serious complications of laparoscopic cholecystectomy is less than 2%. They are

- Intestinal injury –May be due to abdominal access, adhesiolysis or dissection of gall bladder away from duodenum. If accidental injury occurs due to veress needle (Incidence is 0.2%) it need not be repaired where as cautery injuries should be repaired in two layers.

- Vasculo biliary injury – Occurs during abdominal access due to inadequate insufflations of CO2 or excessive thrusting of the trocar given by surgeon. If an unexplained hypotension or retroperitoneal hematoma is found that is an indication for immediate laparotomy.

- Excessive bleeding from the porta hepatis- if it is minor bleeding clips can be applied where as major bleeding necessitates conversion to open method. Bleeding from the gall bladder bed can be controlled by fulguration of the bleeding site.

- Spillage of stones in the presence of infected bile is an indication for conversion to open method

CBD injury:

Occurs commonly in laparoscopic procedure than in open method. The incidence during earlier period was more but now it has come down to 0.5%. The reasons for higher incidence in laparoscopic procedure are,

- Incorrect traction forces to the gall bladder
- Lack of knowledge about the anatomy of biliary system
- Injudicious use of cautery on the calots triangle area

Steps taken to avoid CBD injury during the procedure:

- Use of 30 degree telescope and a high quality imaging equipment.
- Make the cystic duct perpendicular to CBD by pushing the fundus towards right dome of diaphragm and infundibular traction towards more laterally. Dissect the cystic duct at the place where it joins with gall bladder

'Critical view of safety' -all the tissues except for cystic artery and cystic duct should be cleared off in the triangle of calot so that these two structures entering the gall bladder can be clearly seen before ligating them.

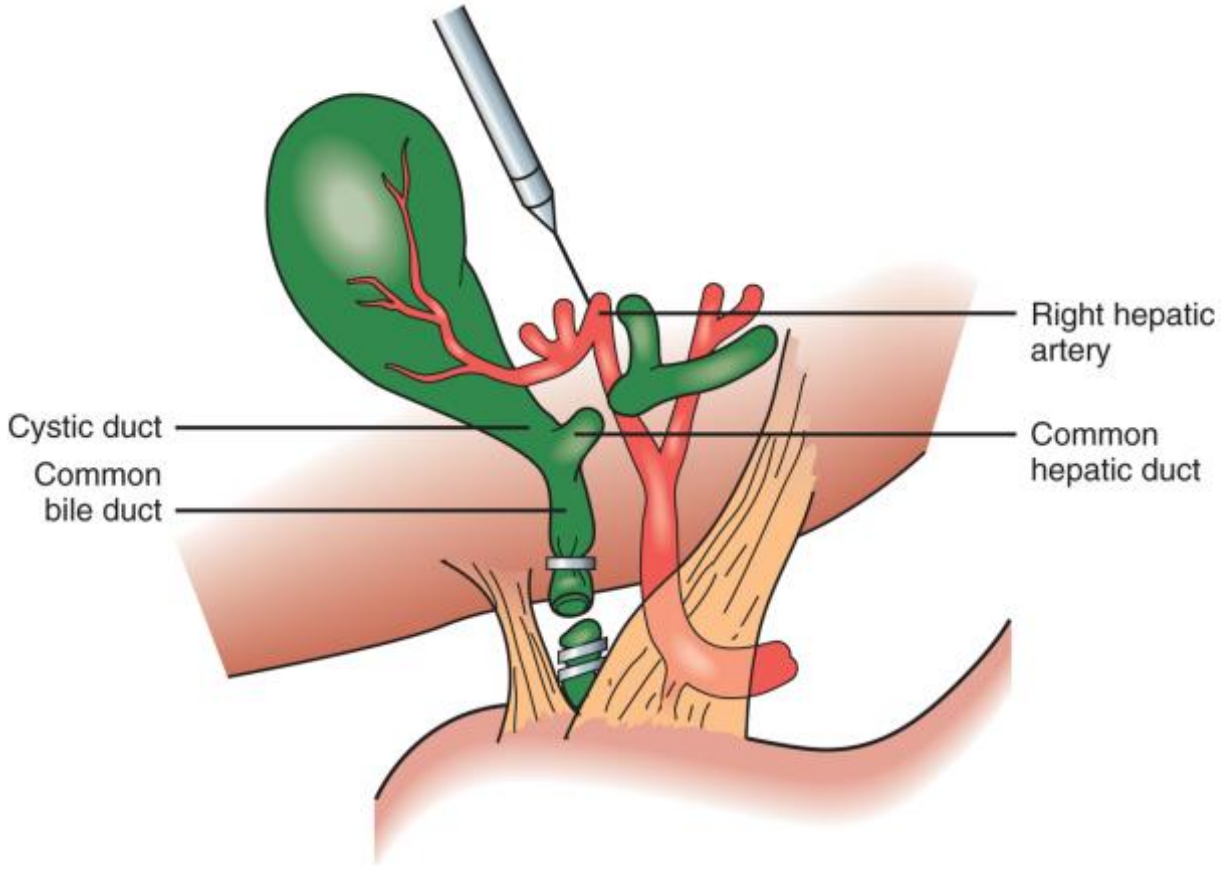
Management of bile duct injury

Intra operative diagnosis

Class I & II injuries can be repaired primarily with or without T-tube drainage where as class III & IV are managed with Hepatico jejunostomy.

Post operative diagnosis

Minor leaks can be managed by percutaneously placed catheter alone. If it is <300 ml /day, it is called as low out put drainage and it usually resolves in 5-7days.If it is >300 ml/day, it is called as high output drainage for which ERCP is the treatment of choice. Other methods are sphincterotomy and transpapillary stenting



Picture : A classic bile duct injury

MATERIALS AND METHODS

Source:

The patients admitted in surgical wards in Govt.Kilpauk Medical College Hospitals including Govt.Royapettah Hospitals, Chennai with signs and symptoms of gall bladder diseases. This study was conducted from JULY 2012 to DECEMBER 2013.

Methods of collecting data;

A proforma for study of patients with features of cholecystitis was used. The presentation, clinical features, investigations, diagnosis and the management of both the open and laparoscopic cholecystectomy were documented. The patient related factors such as age, sex, co-morbid illness and past history were recorded. In the past history the H/O jaundice, previous episodes of similar illness were recorded.

Routine investigations;

Hemoglobin, White cell count, Differential count, Blood urea, Serum creatinine, Blood sugar, ECG in all leads, CXR and Urine examination were done for all cases.

Special investigations;

Liver function test-serum bilirubin, alkaline phosphatase, total protein, Ultra sonogram abdomen- for GB stones, wall thickness and to R/O CBD stones, MRCP- to know the aberrant biliary duct and cystic arteries and Lipid profile.

Inclusion criteria

Following patients are included for treatment,

- Calculus cholecystitis
- Acute cholecystitis
- Chronic cholecystitis
- Acalculous cholecystitis with physical status 1 and 2 (stable vitals)
- Polyps of gall bladder

All the patients with evidence of stone identified in USG, presenting with minimum of one episode of attack and fit to undergo elective surgery were included in this study group.

Exclusion criteria

Patients with following conditions were excluded from this study

- History or investigations suggestive of CBD stones
- Empyema of gall bladder
- On immune suppressive drugs
- Gross obesity
- Mucocele of gall bladder
- Malignancy of gall bladder
- History of prior abdominal surgery
- Age less than 30yrs
- Patients age above 70yrs (symptom recurrence rate is only 15%)
- Ruptured gall bladder
- Physical status 3 &4

Sample size

Totally 65 patients divided in to two groups, 33 in Group A and 32 in Group B admitted from the period since July 2012 to December 2013.

GROUP A- Patients with open cholecystectomy

GROUP B- Patients with laparoscopic cholecystectomy

A day before surgery all the patients were kept NPO for about 10hrs, nasogastric tube insertion and bladder catheterization done according to the individual need. Then prophylactic antibiotic was given 30 minutes before the commencement of surgery. After anesthesia Cholecystectomy done and before extubation irrespective of the type of surgery all the patients were given MOL suppository 250mg per rectally for pain relief.

Injectable analgesics and NSAIDS were avoided as far as possible and pain was assessed by VISUAL ***ANALOGUE SCALE*** (from 0 to 5). A full course of Intravenous antibiotics (Inj.Cefotaxime) continued in patients with co-morbid illness. Nasogastric tube and Foleys catheter were removed by next day. Orals started after the return of the bowel sounds, encouraged to resume work as soon as possible with post operative advice to come for follow up weekly for one month

Comparision of results

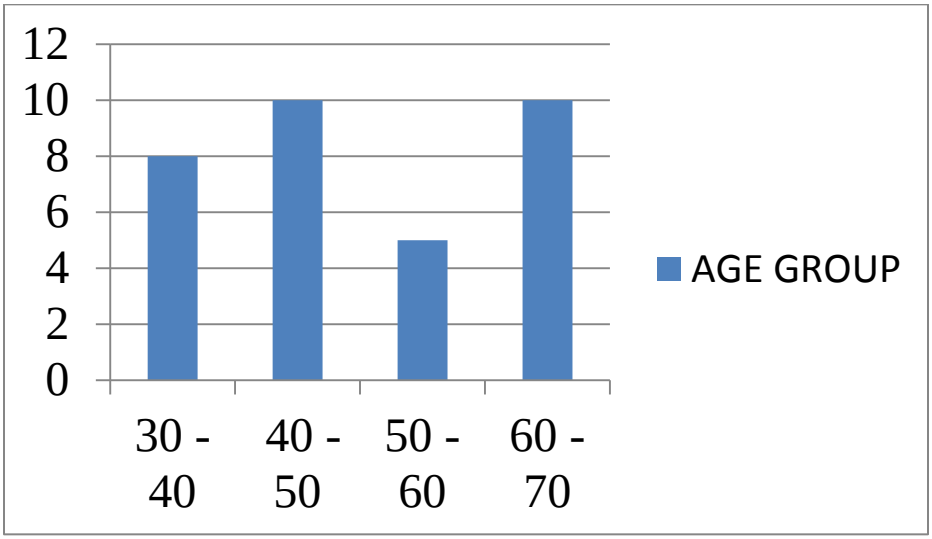
Both the groups were analysed based on age, duration of surgery, post operative recovery , requirement of antibiotic and analgesics , complications and hospital stay.

Age analysis

Table:1 Age Distribution In The Study Group A

Age group (in years)	Number of subjects	Percentage
30 – 40	8	24.2
40 – 50	10	30.3
50 – 60	5	15.2
60 – 70	10	30.3
Total	33	100

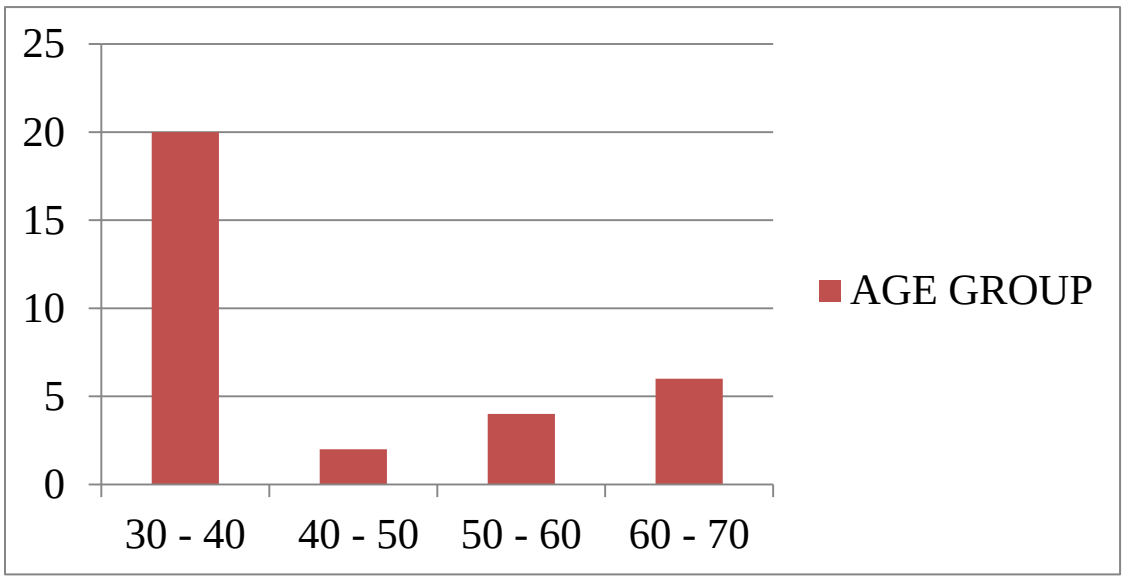
In Group A, symptomatic gall stone disease patients underwent elective open cholecystectomies were between 40 and 50 yrs and 60 and 70 yrs. Where as in Group B patients between 30 to 40 yrs of age underwent laparoscopic cholecystectomies due to the fact that these patients were free of comorbidities and ability to withstand general anesthesia well when compared to patients above 50yrs of age.



Graph 1: Age Distribution of the Study Group

Table 2: Age distribution in Group B patients

Age group (in years)	Number of subjects	Percentage
30 – 40	20	62.5
40 – 50	2	6.2
50 – 60	4	12.5
60 – 70	6	18.8
Total	32	100

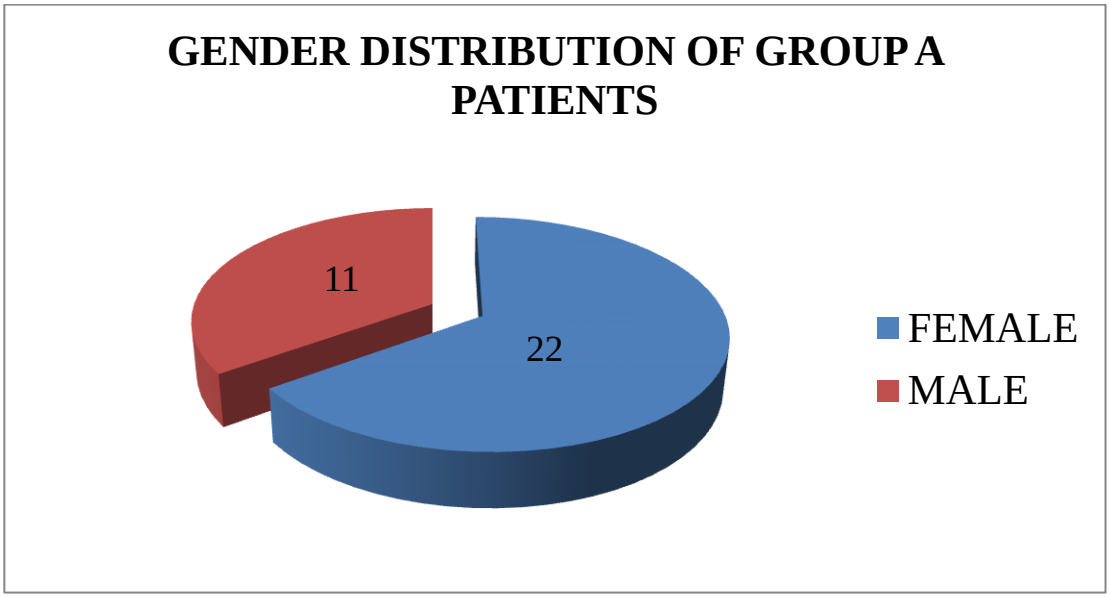


GRAPH 2: Age distribution of Group A patients

Table 3: Gender Distribution in the Study Group A

Gender	Number of subjects	Percentage
Male	11	33.3
Female	22	66.7
Total	33	100

The gender distribution in our study shows 2/3rd of population belongs to female sex.

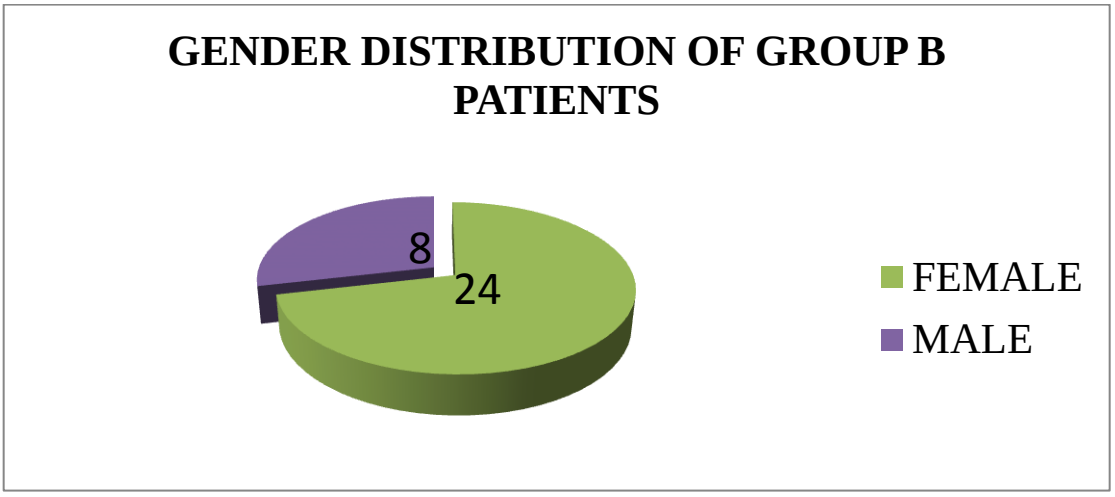


Graph 3: Gender Distribution in the Study Group A

Since gall bladder disease is more common in female than that of male gender, dominance of female observed in both the methods.

Table 4: Gender distribution of Group B patients

Gender	Number of subjects	Percentage
Male	8	25
Female	24	75
Total	32	100



Graph4: Gender Distribution in the Study Group B

Table 5: Comparison of duration of both surgeries

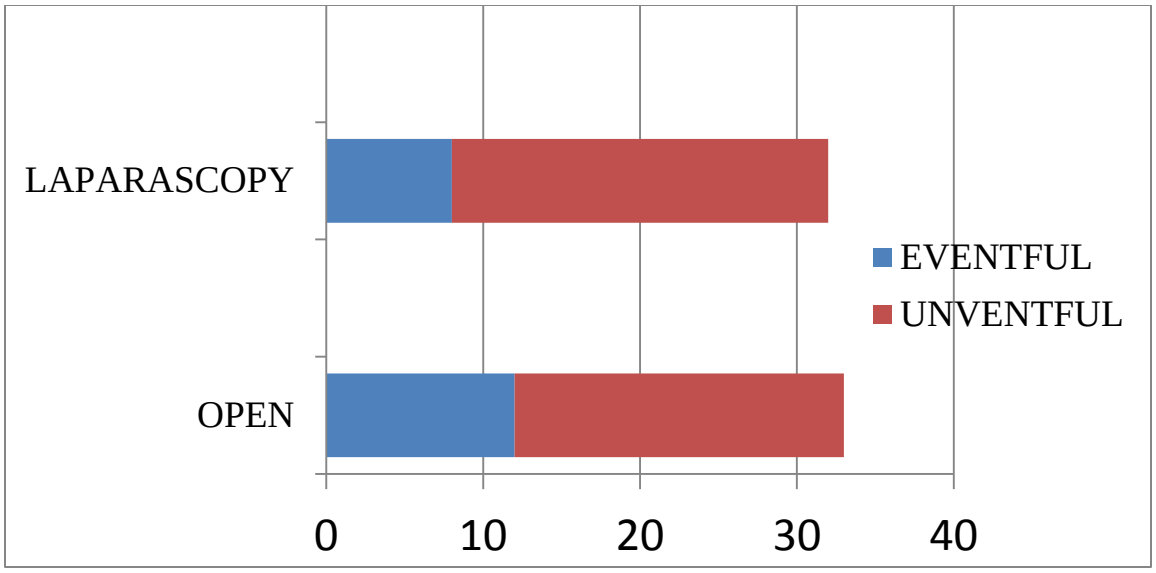
Surgery	Mean duration± SEM	T value	P value
Open Cholecystectomy	89.48 ± 3.61	6.08	0.001
Lap Cholecystectomy	62.41 ± 2.57		

The average time taken for laparoscopic cholecystectomy was much lesser (62.41 minutes) than that taken for the open method which is 89 minutes and48 seconds.

Table 6: Comparison of post op recovery in both surgeries

Surgery	Post op recovery		Chi square value	P value
	Uneventful	Eventful		
Open Cholecystectomy	21	12	0.98	0.321
Lap Cholecystectomy	24	8		

The post operative recovery between laproscopic and open cholecystecytomy does not show any statistical difference between either groups.



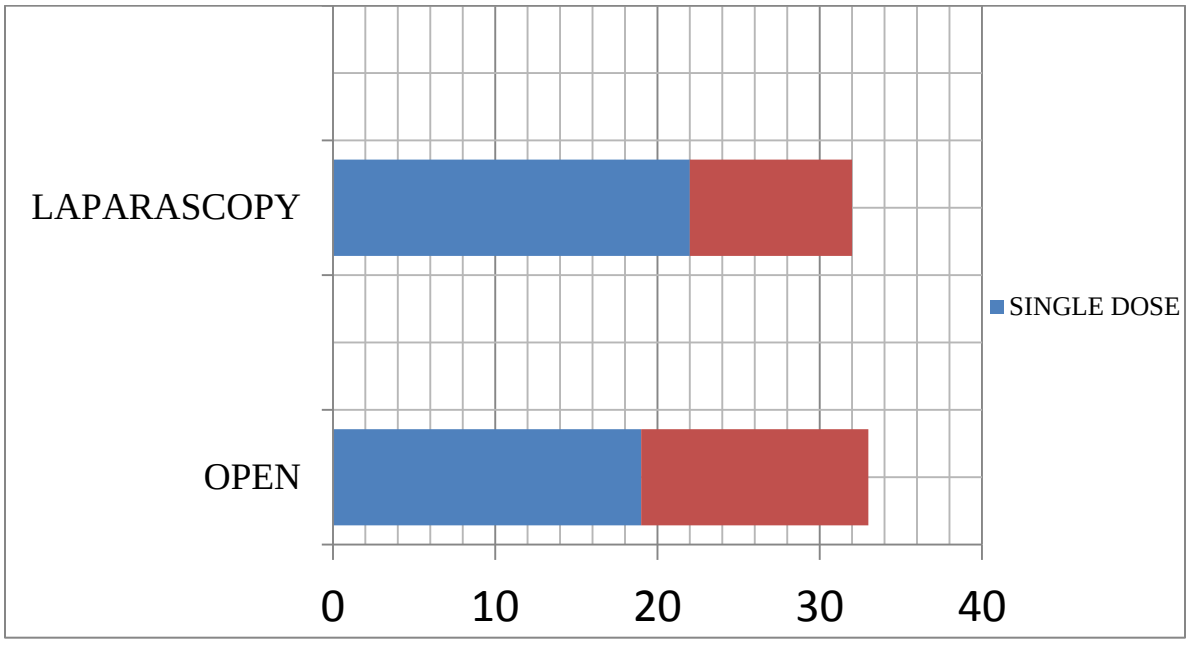
Graph 5: Post Op Recovery In Both Groups

The above mentioned stastical variable from our study does not show major significant difference in post operative recovery between open and laparoscopic procedures.

P = 0.321 (not significant)

Table 7: Comparison of antibiotic usage in both surgeries

Surgery	Antibiotic used		Chi square value	P value
	Single dose	Full course		
Open Cholecystectomy	19	14	0.87	0.35
Lap Cholecystectomy	22	10		



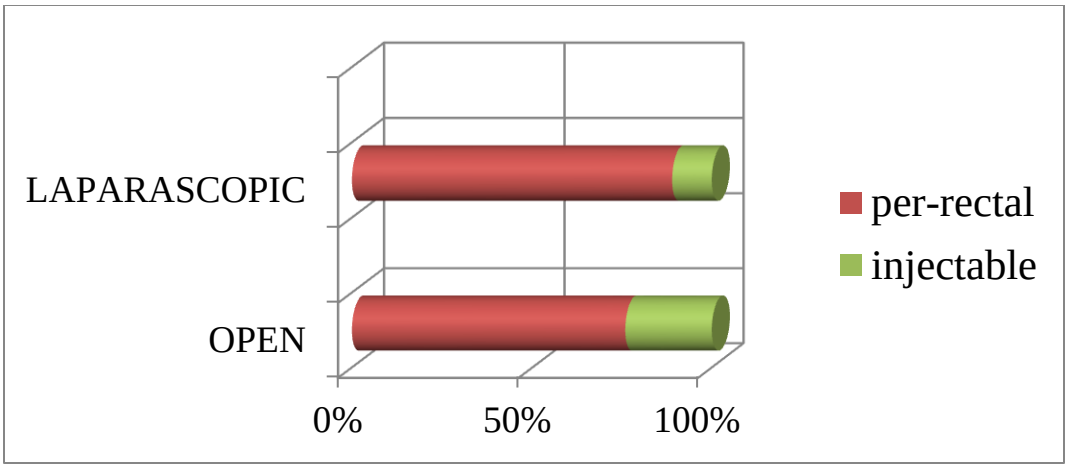
Graph 6: Post Operative Antibiotic Requirement In Both Groups

Since recommendation of antibiotic was based on the associated comorbidity our study shows no major difference in its usage in both the groups.

Table 8: Comparison of analgesic usage in both surgeries

Surgery	Analgesic used		Chi square value	P value
	Perrectal suppository	Injectable analgesics		
Open Cholecystectomy	15	18	5.91	0.015
Lap Cholecystectomy	24	8		

Requirement of stronger parental analgesics are not needed for group B patients while per rectal paracetamol alone is insufficient in group A patients.



Graph 7: Route of Analgesic Requirement

Table 9:Comparison of period of stay in both surgeries

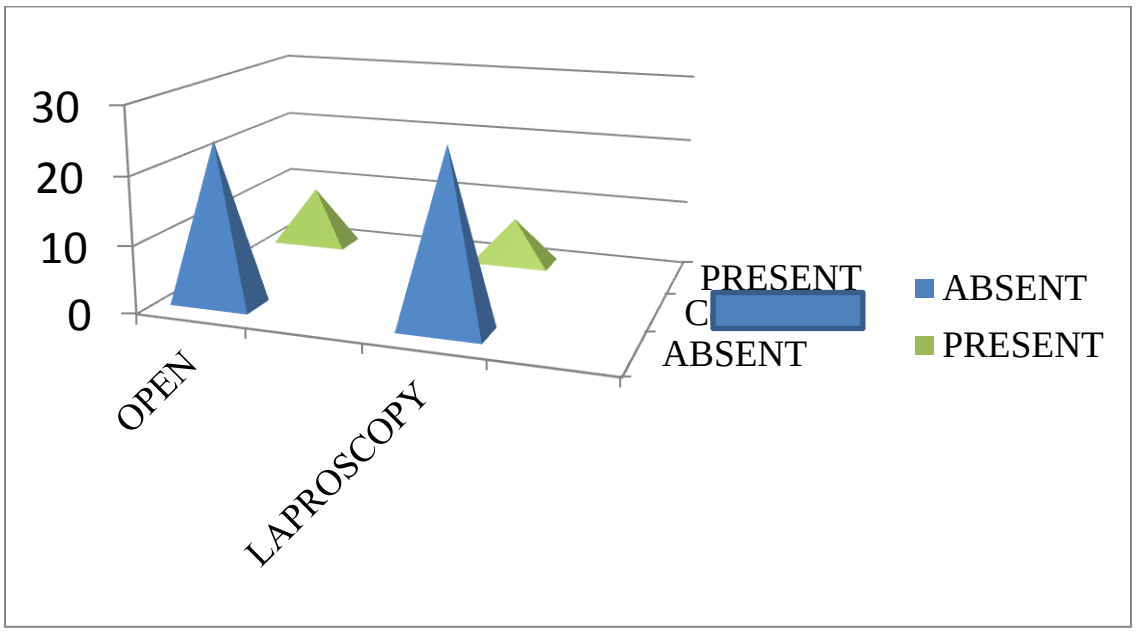
Surgery	Mean period of stay	T value	P value
Open Cholecystectomy	7.88 ± 0.50	9.095	0.001
Lap Cholecystectomy	2.95 ± 0.19		

The mean period stay in the Hospital in Laparoscopic surgery was 2.95 days where as in open method it was 7.88 days

Table 10:Comparison of occurrence of complications in both surgeries

Surgery	Complications		Chi square value	P value
	Present	Absent		
Open Cholecystectomy	9	24	0.33	0.565
Lap Cholecystectomy	7	26		

P value < 0.05 is significant



Graph 8: Occurance Of Complications In Both Groups

During our study we observed that occurrence of wound infection was little bit high in open method but since the sample size was small we could not assess all other complications in detail. One important reason was its association with diabetes mellitus but for overall it does not significance between both the groups

Analysis of the results

Total number of patients analysed for this study was 65 among which 33 taken for open cholecystectomy and 32 patients were taken up for laparoscopic cholecystectomy with 2 patients converted to open due to adhesions found in the calots region.

- In Group A, symptomatic gall stone disease patients underwent elective open cholecystectomies were between 40 and 50 yrs and 60 and 70 yrs. Where as in Group B patients between 30 to 40 yrs of age underwent laparoscopic cholecystectomies due to the fact that these patients were free of comorbidities and ability to withstand general anesthesia well when compared to patients above 50yrs of age.

- The gender distribution in our study shows 2/3rd of population belongs to female sex. Since gall bladder disease is more common in female than that of male gender, dominance of female observed in both the methods.

- During our study we observed that occurrence of wound infection was little bit high in open method but since the sample size was small we could not assess all other complications in detail. One important

reason was its association with diabetes mellitus but for overall it does not significance between both the groups

- The average time taken for laparoscopic cholecystectomy was much lesser (62.41 minutes) than that taken for the open method which is 89 minutes and48 seconds.

- The mean period stay in the Hospital in Laparoscopic surgery was 2.95 days where as in open method it was 7.88 days. This is mainly due to the more trauma given to the adjacent organs by means of traction given in open method.

- The post operative recovery between laproscopic and open cholecystecytomy does not show any statistical difference between either groups.

Discussion and Conclusion

This study proves that laparoscopic cholecystectomy is the surgical treatment of choice due to the following reasons

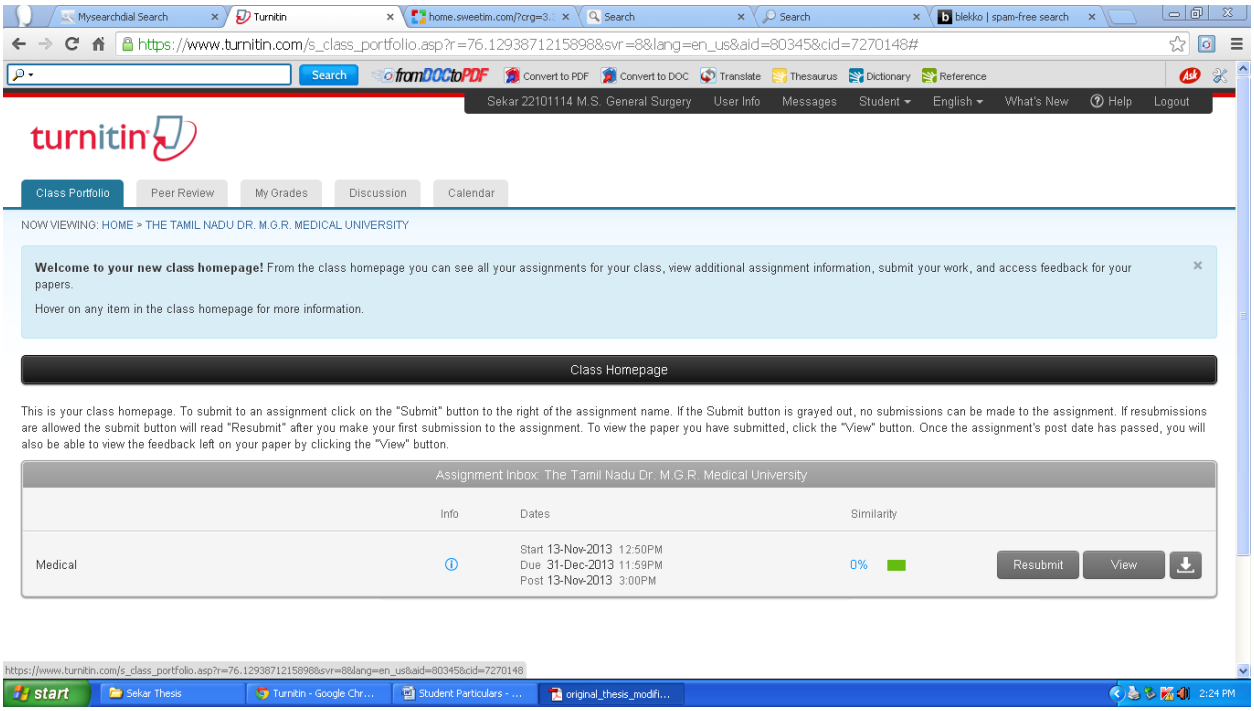
The time taken for laparoscopic procedure is less and in an experienced hand the entire procedure can be end within 30 minutes. So that prolonged anaesthesia and its complications like MI, Respiratory distress and ventilator support can be avoided.

Lesser need of analgesic especially powerful analgesics like diclofenac or tramadol in the form of Injectable.

If the associated co morbid illnesses like DM are absent the need of full course of antibiltics is definitely not needed.

Wound infection also very less compared to open procedure.

Patient can be treated as Day care basis , admission and discharge on the same day if preoperative work has been done earlier.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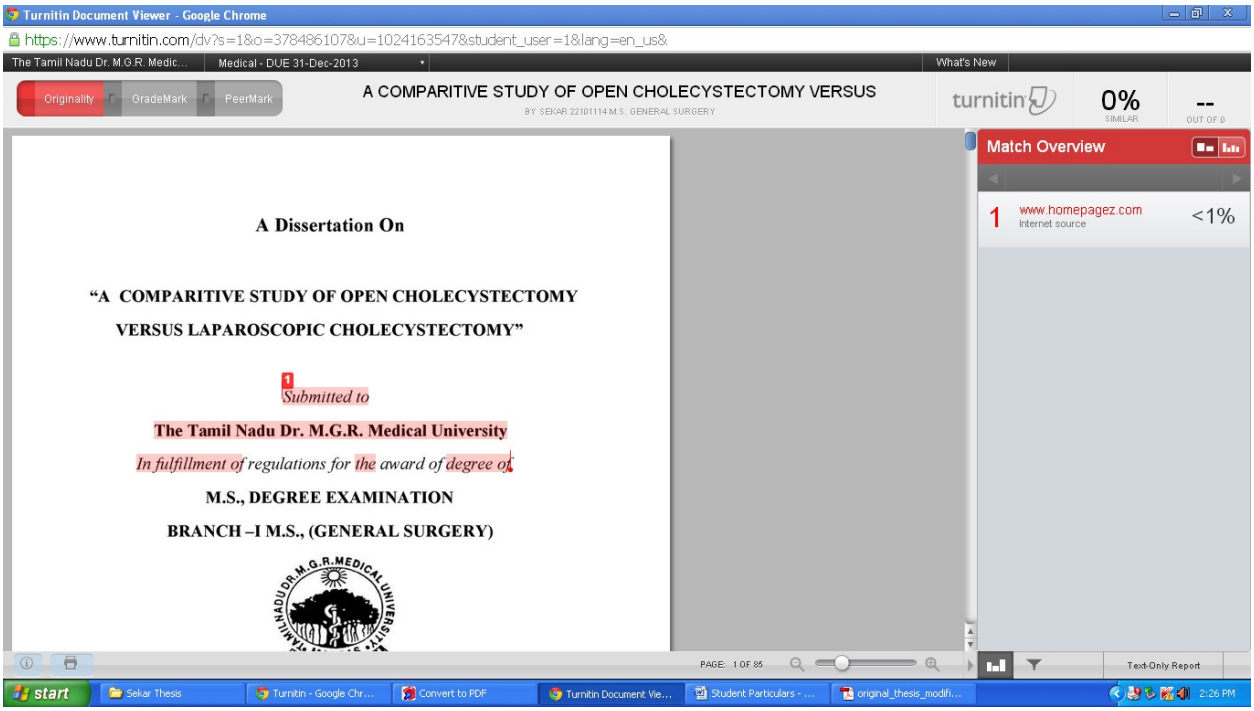
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GROUP A

NAMES	AGE	SEX	IP NO	Time taken	Comorbidity	Post op Recovery	Antibiotic	Analgesics	period of Stay	Complications	Return to regular work
Lilly	33	2	21777	120	DM	0	2	1	6	0	25
Ras avathy	45	2	22252	128	DM	0	2	1	5	1	20
Subhashini	31	2	23250	100	NIL	0	1	1	4	0	30
Velu	35	1	22212	105	NIL	0	1	1	8	0	18
Meenambigai	35	2	28667	70	NIL	0	1	2	10	0	21
TajilNisha	51	2	29741	110	DM/SHT/OBESITY	1	2	2	7	1	18
Mohan	31	1	31303	95	NIL	0	1	1	5	0	14
Dhanalakshmi	35	2	1301693	83	OBESITY	1	1	1	7	0	21
Jaghadhambal	44	2	1302111	125	DM/OBESITY	1	2	1	10	1	45
Krishnaveni	58	1	1304079	50	DM	1	2	1	5	0	30
Thangaiyan	65	1	1312361	77	SHT	1	1	1	5	0	21
Kumari	50	2	1330994	85	OBESITY	1	1	1	8	0	14
Narayanan	60	1	1333010	115	DM	1	2	2	13	1	20
Lakshmi	55	2	1311778	50	NIL	0	1	1	6	0	16
Anjali	70	2	1335242	68	DM	1	2	2	12	1	30
Geetha	45	2	1334959	74	HBsAg +ve	0	2	2	8	0	14
Sadhakdulla	61	1	1315079	85	DM/IHD	1	2	1	8	0	14
Radha	31	2	1326589	86	NIL	0	1	1	4	0	14
Saraswathy	47	2	1314568	65	NIL	0	1	1	4	0	21
Dhanalakshmi	41	2	3709	63	NIL	0	1	1	8	0	23
Palayam	65	1	3247	105	DM	0	2	2	10	1	30
Balamurugan	48	1	6144	100	NIL	0	1	1	7	0	18
Renuka	50	2	7041	88	OBESITY/DYSLIPEDEMIA	1	1	2	12	0	17
Philip Krishnan	64	1	110963	86	DM	1	2	1	11	1	15
Seema	45	2	111291	68	NIL	0	1	1	7	0	14
Muniyammal	60	2	104623	72	DM/OBESITY	0	2	1	14	0	35
Saradha	50	2	102881	75	NIL	0	1	1	6	0	16
Jayanthi	35	2	105968	78	NIL	0	1	1	5	0	18
Shanmugamaniya	61	1	110174	108	DM/HBsAg+ve	1	2	2	10	0	28
Rajeshwari	65	2	114805	111	DM/OBESITY	0	2	1	13	1	30
Kuppammal	65	2	3906	102	NIL	0	1	1	7	0	21
Selvathai	65	2	5794	98	OBESITY	0	1	1	10	1	28
Balamurugan	63	1	110811	108	SHT/IHD	0	1	1	5	0	24

NAME	AGE	SEX	IPNO	Duration of Surgery	Comorbidity	Post op recovery	Antibiotic	Analgesic	Period of Hospital stay	Complications	Return to regular work	conversion
Godhavari	68	2	1302995	77	DM/SHT	0	1	2	4	0	15	
Sheela	37	2	1312361	78	NIL	0	1	1	3	0	12	
Kavitha	36	2	1306910	66	DM	1	2	1	4	1	18	
Lakshmi	37	2	1320370	40	NIL	1	1	1	2	1	10	
Natarajan	70	1	1315201	54	DM	0	2	1	3	1	11	YES
Vatsala	37	2	1329343	62	FAMILY	0	1	1	2	0	10	
Kauserbanu	35	2	1316044	65	NIL	0	1	2	2	0	7	
Eswari	32	2	1322710	54	NIL	1	1	1	3	1	60	
Sherkhan	53	1	1334655	63	DM/SHT	0	2	2	4	0	14	
Hamsavalli	33	2	1731	55	NIL	1	1	1	2	0	14	
Pushpa rani	65	2	10088	78	DM/OBESITY	1	2	2	5	1	12	
Latha	32	2	847	90	OBESITY	0	1	1	2.5	0	10	
Johnson	63	1	132462	65	SHT	0	1	1	3	1	14	
Usharani	31	2	880	70	NIL	0	1	1	1.5	0	13	
Selvi	32	2	113453	58	NIL	0	1	1	2	0	9	
Mebin	31	1	1329345	42	NIL	0	1	1	3	0	10	
Vimala	38	2	106107	62	DM/OBESITY	1	2	1	3	0	10	YES
Anjamma	45	2	106277	45	OBESITY	0	1	1	6	0	14	
Arun	42	1	1323101	46	DM	0	2	2	4	1	13	
Vinayagamurthy	62	1	108213	103	F/H OF CA GB	0	1	2	3	0	7	
Kanchana	60	2	106330	66	DM	0	2	1	5	0	9	
Loganayagi	60	2	102574	65	DM/OBESITY	0	2	2	3	0	8	
Thamarai	33	2	106963	75	NIL	0	1	1	2	0	11	
Seetha	55	2	108786	56	DM	0	2	1	3	0	10	
Arunkumar	33	1	107787	67	NIL	0	1	2	2	0	7	
Srinivasan	70	1	109033	55	SHT	0	1	1	1.5	0	7	
Lakshmi	33	2	109331	77	NIL	0	1	1	2	0	7	
Deepa	30	2	111125	45	NIL	0	1	1	3	0	7	
Deepa	33	2	111107	48	OBESITY	0	1	1	3	0	8	
Deivakani	35	2	112703	62	NIL	0	1	1	2	0	14	
Malarvizhi	33	2	1549	70	OBESITY	1	1	1	3	0	11	
Sridevi	32	2	3470	38	DM		2	1	3	0	10	
											GROUP B	
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