A PROSPECTIVE COMPARATIVE STUDY OF MINI LAPAROSCOPIC AND CONVENTIONAL LAPAROSCOPIC CHOLECYSTECTOMY

Submitted by

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B. L. D. E. ((DEEMED TO BE UNIVERSITY)'s

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RESEARCH CENTRE, VIJAYAPUR, KARNATAKA

In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY

In

GENERAL SURGERY

UNDER THE GUIDANCE OF

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LIST OF ABBREVIATIONS USED

CHD	- Common Hepatic Duct
CBD	- Common Bile Duct
LC	- Laparoscopic Cholecystectomy
MLC	- Mini Laparoscopic Cholecystectomy
CLC	- Conventional Laparoscopic Cholecystectomy
USG	- Ultrasonography
RHA	- Right Hepatic Artery
GB	- Gall Bladder
ERCP	- Endoscopic Retrograde Cholangio pancreatography
P/C	- Peri cholecystic collection
Pvalue	- Predictive value

ABSTRACT

BACKGROUND

Cholelithiasisis is the most common biliary pathology, with a prevalence of 10 to 15%. It is symptomatic in approximately 1to2% of patients. NIH consensus development stated that laparoscopic cholecystectomy "Provides a Safe and Effective treatment for most patients with symptomatic gallstones". In about 5 to 10% of laparoscopic cholecystectomy, conversion to open cholecystectomy may be needed for safe removal of gall bladder.[58]

- Mini-laparoscopy was pioneered more than 20 years ago.
- Newer generation mini instruments have recently become available with improved effector tips, a choice of shaft diameters and lengths, better shaft insulation and electrosurgery capability, improved shaft strength and rotation, more ergonomic handles, low-friction trocar options, and improved instrument durability. [11]
- Mini-Lap cholecystectomy is a refinement of LC in which instruments and ports of size ≤ 3mm in diameter are used compared with the standard 5-mm and10-mm sizes used in conventional laparoscopic cholecystectomy. [59]

  Mini Laparoscopic cholecystectomy can be safely performed using 10-mm umbilical, 5-mm epigastric, 3-mm subcostal ports. [59]

  The use of mini- laparoscopic techniques resulted in decreased early post-operative pain, & decreased length of hospital stay, variable operative time. [59]

  Although improved instrument durability and better optics are needed for widespread use of miniport techniques. [59]

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INTRODUCTION

Cholelithiasis is the most common biliary pathology. Gallstones are present in 10 to 15% of the general population and asymptomatic in the majority (>80%).The prevalence of gallstone varies widely in different parts of the world. In India it is estimated to be around 4%.[1]

Changing incidence in India is mainly attributed to westernization and availability of investigation that is ultrasound in both rural and urban areas and due to change in socioeconomic structure.[2]

Approximately 1-2% of asymptomatic patients will develop symptoms requiring cholecystectomy per year.[3]

Cholelithiasis is rare in the first two decades. Incidence gradually increases after 21 years and reaches its peak in 5^{th} and 6^{th} decade. Women are more affected than men in the ratio of 4:1.[4]

In 1992, The National Institute of Health (NIH) [58] consensus development conference stated that laparoscopic cholecystectomy "provides a safe and effective treatment for most patients with symptomatic gallstones."[5]

The advantages of Mini laparoscopic cholecystectomy over Conventional Laparoscopic cholecystectomy are, minimal pain in

postoperative period, cosmesis, minimal hospital stay, earlier return to normal activity.

Laparoscopic cholecystectomy has become the gold standard in the treatment of gallbladder pathology and is replacing open cholecystectomy. The rate of conversion from laparoscopic cholecystectomy to open cholecystectomy is 5 to 10%.[6]

AIMS AND OBJECTIVES OF THE STUDY

To compare the outcome of Mini Laparoscopic Cholecystectomy and Conventional Laparoscopic Cholecystectomy

To determine the outcome of Mini Laparoscopic Cholecystectomy and Conventional Cholecystectomy in laparoscopic surgeries in view of :

1) Mean operative Time

2) Post-operative Pain (by Visual Analogue Scale on day 1,3,5)

3) Stay in the hospital (From the day of surgery to day of discharge)

4) Intra-operative and post-operative complications.

.

RESEARCH HYPOTHESIS

Mini Laparoscopic Cholecystectomy is equally safe, less painful and better cosmetic outcome compared to Conventional Laparoscopic Cholecystectomy.

REVIEW OF LITERATURE

HISTORICAL ASPECTS

The Roman Celsus in his text, De Medicina (translated by W.G. Spencer in 1935), mentioned the liver and described its anatomic location in an accurate form: "The liver, which starts from the actual partition under the precordia on the right side, is concave within (that is on the inferior surface) and convex without; its projecting part rests lightly on the stomach and it is divided into four lobes. Outside its lower part, the gallbladder adheres to it."[7]

Vesalius found (that he had) a hemoperitoneum coming from an abscess which had eroded the portal vein. The gallbladder was yellow and contained 18 calculi. Very light, of a triangular shape with even edges and surfaces everywhere, green by color somewhat blackish. The spleen was very large."[7]

Morgagni published in 1769 an analysis of disease under the title Seats and Causes of Disease, among which are those of the liver and biliary tract.[7]

Vater (1684-1751) was the first to describe the papilla of the duodenum.[7]

Petit introduced the term biliary colic.[7]

1878: Kocher performed a cholecystostomy in two stages.

1971 Glenn,. In the first stage, he packed the wound with gauze to the bottom of the gallbladder, and 8 days later he emptied the residual stones from the gallbladder.

1885: Tait performed first cholecystostomy for gallbladder lithiasis in one stage.

1882: Langenbuch performed first elective cholecystectomy

1882: Von Winiwarter developed Cholecystenterostomy.

1895: Kocher wrote an article on internal choledochoduodenostomy to remove supra-ampullary choledochal calculi.

1897: Kehr placed a rubber tube in the common bile duct through the cystic duct; this was the first systematic use of biliary intubation.

1898: Thornton performed the first removal of a stone from the common bile duct.

1898: MacBurney published his experience with duodenostomy and papillotomy in patients with impacted periampullary calculi.

1898: Buxbaum observed biliary calculi on plain x-rays.

1912: Kehr developed T-tube.

1923: Bakes developed choledochoscopy.

1924: Graham developed oral cholecystography.

1932: Mirizzi developed Postoperative cholangiography.

1937: Mirizzi developed Intraoperative cholangiography.

1989: Dubois in Paris published the first series of laparoscopic cholecystectomies (Dubois et al).[7]

HISTORY OF LAPAROSCOPY AND LAPAROSCOPIC CHOLECYSTECTOMY:

- Laparoscopy (from the Greek, Laparo meaning the flank and Skopein meaning to examine), was first performed in 1901 by George killing of Dresden, Germany using room air filtered through sterile cotton for pneumoperitoneum and a wide cystoscope to veiw the abdominal cavity of dog. The use of carbon dioxide (co2) for pneumoperitoneum was first recommended by Richard Zollikofer of Switzerland in 1924.[8]

- The primary mode of insufflation was the Veress needle which was introduced by Janos Veress of Hungary in 1938.[8]

- In 1933, A German general surgeon, Feowers, was the first to report laparoscopic lysis of abdominal adhesions for the diagnosis of bowel obstructions.[8]

- Kurt Semm incorporated new aspects of fiber optic and used automatic gas insufflator which allowed precise controlled intra abdominal pressure.[9]

- In 1983, Lukichev and colleagues described laparoscopic cholecystectomy for acute cholecystitis.[9]

- In 1985, Muhe of Boblinger, Germany performed the first laparoscopic assisted cholecystectomy.[10]

- In 1987,a French surgeon in Lyon, Phillipe Mouret, performed the first video-laparoscopic cholecystectomy.

MINI LAPAROSCOPIC CHOLECYSTECTOMY

1. When applied to elective laparoscopic cholecystectomy, the practice of mini-laparoscopic instruments results in a slightly longer operative procedure (3-5 minutes), slightly less immediate postoperative pain (in the first 24 hours), and a better early cosmetic result, with no other apparent significant differences.[11]

2. Laparoscopic Cholecystectomy has arose as the gold standard in the management of gall stones. Though it is easier to teach and learn the laparoscopic procedure with the help of magnified visual display, specialized training is a must in case of the laparoscopic technique. On the other hand, mini-laparotomy cholecystectomy does not require any special training (nor any additional / special instruments) Mini-LPC is an alternative to laparoscopic cholecystectomy in the treatment of symptomatic cholelithiasis. Both techniques produce similar results in terms of postoperative complications, hospital stay and postoperative pain except surgical time, which show longer duration of operation in LC. Mini-LPC is seen as acceptable resource in centers where laparoscopic equipment is not available. The approach by mini-LC is an option for surgeons experienced in open surgery and for residents in training in developing country settings with limited resources.[12]

3. Laparoscopy offers a good cosmesis, less postoperative pain and short hospital stay, it takes longer to perform, requires special training and is found to offer no significant advantage over mini-cholecystectomy. [13]

4. Mini Lap is an intuitive, easy-to-learn and reproducible technique and requires small changes from Conventional Lap. As such, Mini Lap may be an attractive alternative, avoiding the cost and complexity.[14]

5. Further randomized trials are needed to determine whether mini laparoscopic techniques truly offer any advantages. Important patient outcomes such as failure of technique, adverse events, cosmesis, and quality of life should be emphasized to determine whether there is any benefit over conventional laparoscopic cholecystectomy.[15]

6. Both MLC and CLC are feasible surgical techniques for day surgery. However, appropriate prevention and prompt management of established post-operative nausea and vomiting and careful patient selection are important aspects for success of short stay approach. If there is a sign of chronic cholecystitis preoperatively, it might be considered as a contraindication for day surgery.[16]

7. Minilaparoscopic cholecystectomy could be a feasible alternative to conventional laparoscopic cholecystectomy in select patients, resulting in less pain and better cosmetic results. Additional well-designed randomized controlled and, if possible, blinded trials, with large sample sizes, are required to confirm this conclusion.[17]

8. Mini lap cholecystectomy is safe and feasible without increased operative risk, with better cosmetic results, less pain, and good acceptance among patients.[18]

ANATOMY

The extra-hepatic biliary tree consists of the right and left hepatic ducts, common hepatic duct, cystic duct and gallbladder and the common bile duct.

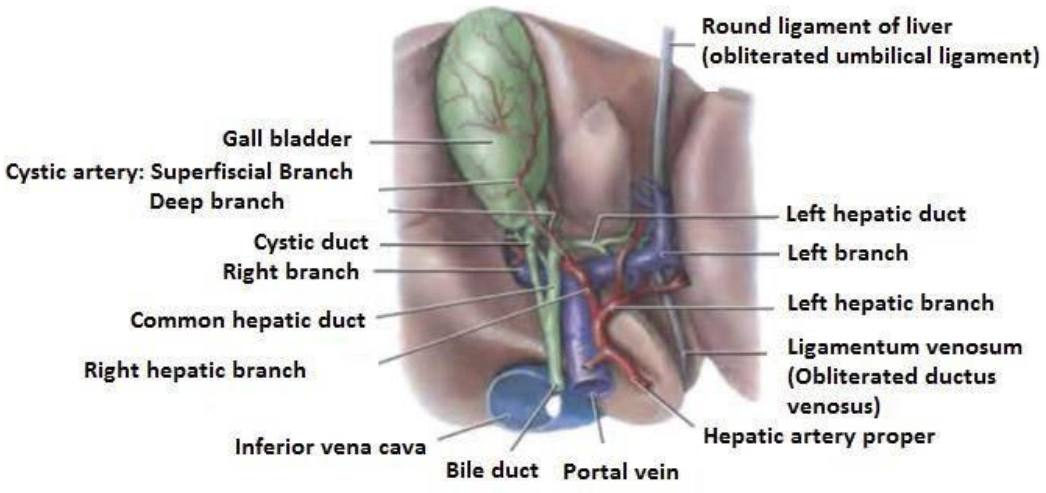


FIGURE1: Showing anatomy of gall bladder, inferior view

GALL BLADDER:

- The gall bladder is a flask-shaped, blind-ending diverticulum attached to the common bile duct by the cystic duct. It usually lies in a shallow fossa in the liver parenchyma covered by peritoneum continued from the liver surface. This attachment can vary widely.[19]
- The gall bladder lies on a fibrous or cystic plate, which is part of the perihilar system of fibrous tissue. The cystic plate attaches directly onto the anterior surface of the right portal pedicle.
- The hepatic parenchyma lies deep to the cystic plate, through which small bile ducts may penetrate to enter the gallbladder. Between the muscularis of the gallbladder and the cystic plate, a thin layer of areolar tissue thickens progressively from the top of the gallbladder downward.

- During dissection of the gallbladder from the liver, the posterior surface of the cystic artery and bile duct will be reached when the areolar tissue is left on the cystic plate. Should dissection be undertaken deep into the cystic plate, the surface to the right portal pedicle may be breached and result in injury to the right portal pedicle structures and the right hepatic duct.

NECK:

Neck lies at the medial end close to the porta hepatis, and almost always has a short peritoneal cover attached to the liver (MESENTERY); this mesentery usually contains the cystic artery.

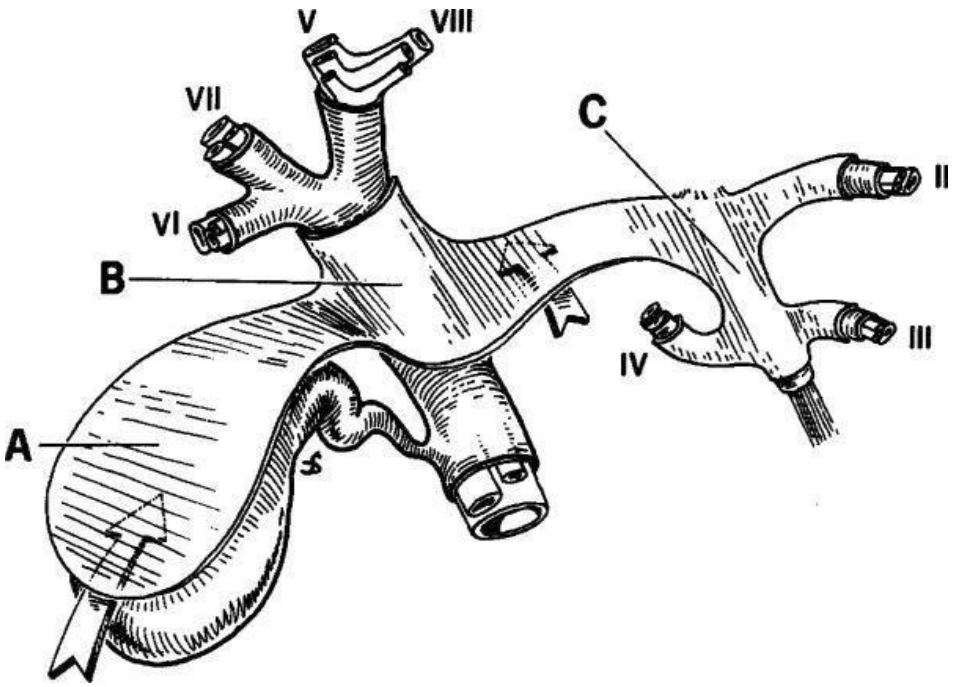


FIGURE 2: The anatomy of the plate system. cystic plate (A) above the gallbladder, the hilar plate (B) above the biliary confluence and at the base of the quadrate lobe, and the umbilical plate (C) above the umbilical portion of the portal vein.

BODY AND FUNDUS:

The body of the gall bladder normally lies in contact with the liver surface. It lies anterior to the 2^{nd} part of the duodenum and the right end of the transverse colon. The fundus lies at the lateral end of the body and usually projects past the inferior border of the liver to a variable length. It often lies in contact with the anterior abdominal wall behind the 9^{th} costal cartilage where the lateral edge of the right rectus abdominis crosses the costal margin. This is the location where enlargement of the gall bladder is best sought on clinical examination.

The fundus of gall bladder may be folded back upon the body of gall bladder: PHRYGIAN CAP.

EXTRAHEPATIC BILIARY TREE CYSTICDUCT

The cystic duct is about 3 to 4 cm in length, passes posteriorly to the left from the neck of gallbladder, and joins the common hepatic duct to form the common bile duct. It almost runs parallel to it and is adherent to common hepatic duct for a short distance before joining it.

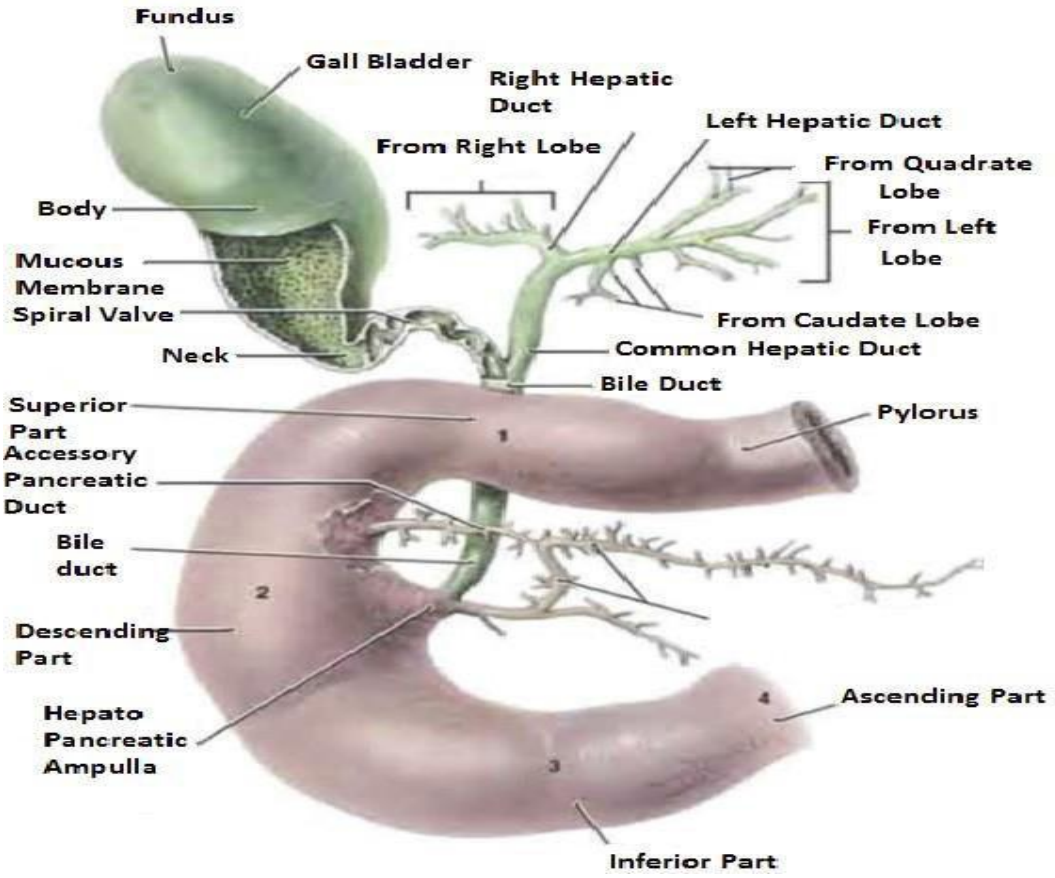


FIGURE 3: Showing the anatomy of the gallbladder, biliary radicals, pancreatic duct and the hepato-pancreatic ampulla.[20]

ANATOMICAL VARIATIONS OF CYSTIC DUCT

The cystic duct occasionally drains into the right hepatic duct in which case it may be elongated, lying anterior or posterior to CHD and joins the right hepatic duct on its left border.[19]

1) The cystic duct lies along the right edge of the lesser omentum, all the way down to the level of the duodenum before the junction is formed. Here cystic duct and common bile ducts are usually closely adherent.

2) The cystic duct may be double or absent in which case gall bladder drains directly into CBD.

3) One or more accessory hepatic ducts occasionally emerge from segment V of the liver and joins either the right hepatic duct the common hepatic duct, the common bile duct, the cystic duct or the gall bladder.

They project obliquely in regular succession, appearing to form a spiral valve when the duct is cut in longitudinal section. When the duct is distended the spaces between the folds dilate and externally it appears twisted like the neck of the gallbladder.

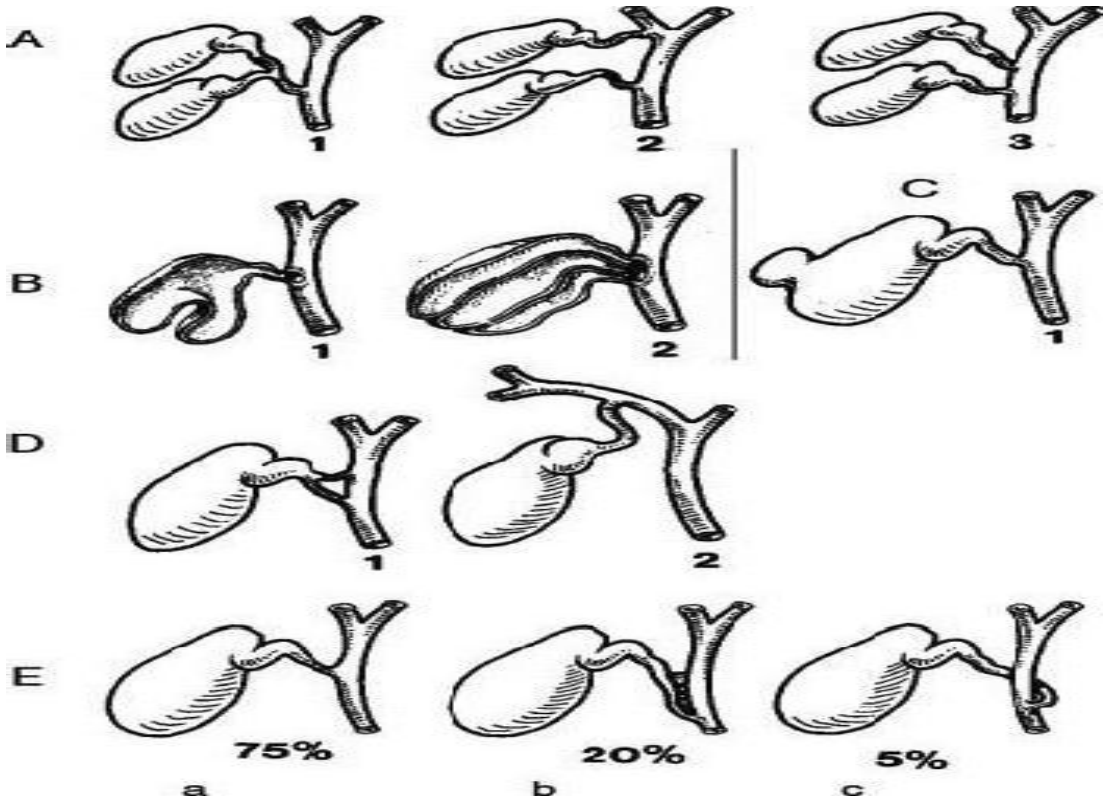


FIGURE 4: Variations in gallbladder and cystic duct anatomy : duplicated gallbladder (A), septum of the gallbladder (B), diverticulum of the gallbladder (C), variations in cystic ductal anatomy (D). Different types of union of the cystic duct and common hepatic duct (E).[21]

HEPATIC DUCTS

The main right and left hepatic ducts emerge from the liver and unite near the right end of the porta hepatis as the common hepatic duct. This descends for about 3 cm before joining cystic duct at an acute angle to form common bile duct.

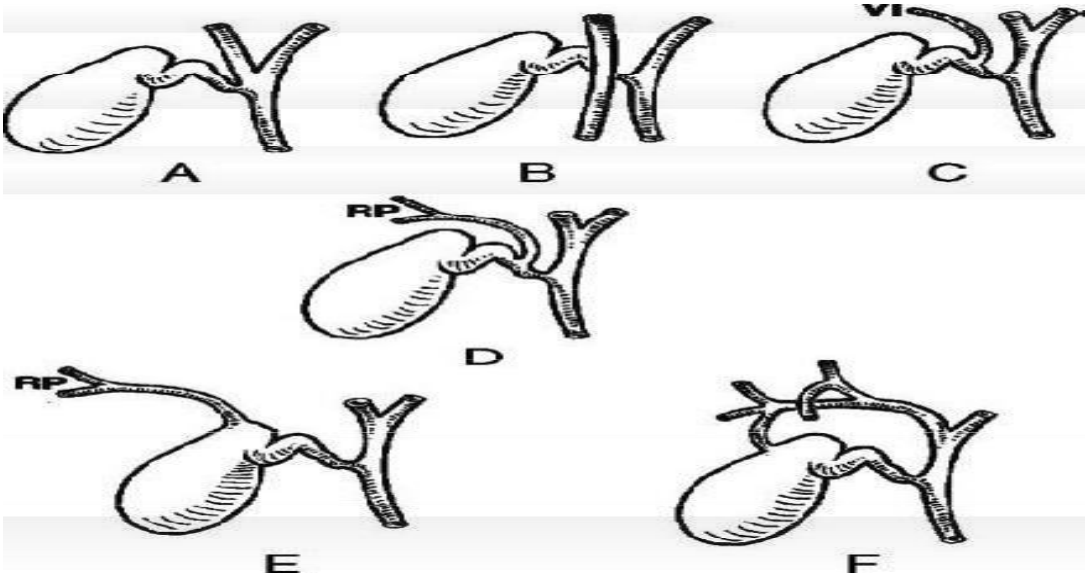


FIGURE 5: The variations of ectopic drainage of the intrahepatic ducts into the gallbladder and cystic duct. A, cystic duct into the biliary confluence. B, cystic duct into the left hepatic duct associated with no biliary confluence. C, segment VI duct into the cystic duct. D, right posterior sectorial duct into the cystic duct. E, distal part of the right posterior sectorial duct into the neck of the gallbladder. F, proximal part of the right posterior sectorial duct into the body of the gallbladder.[21]

COMMON BILE DUCT

Common bile duct is formed near the porta hepatis, by the junction of the cystic and common hepatic ducts. It is usually between 6 and 8 cm in length and about 6 mm in diameter in adults. It descends posteriorly and to the left, anterior to epiploic foramen, in the right border of lesser omentum. It lies anterior and to the right of portal vein and to the right of the hepatic artery. The duct may lie close to the medial wall of the second part of the duodenum or as much as 2 cm from it.

HEPATOPANCREATIC AMPULLA (OF VATER)

It is formed by the union of CBD and pancreatic duct before entering the 2nd part of the duodenum. Circular muscles usually surround the lower part of the CBD (bile duct sphincter), and frequently also surround the terminal part of the main pancreatic duct (pancreaticduct sphincter) and the hepatopancreatic ampulla (sphincter of oddi).

CALOT'S TRIANGLE - CHOLECYSTOHEPATIC TRIANGLE

The near triangular space formed between the cystic duct, common hepatic duct and the inferior surface of the segment V of the liver is commonly referred to as Calot's triangle. It is enclosed by double layer of peritoneum which forms the short mesentery of the cystic duct, it is perhaps better described as a pyramidal space with one apex lying at the junction of the cystic duct and fundus of the gallbladder, one at the porta hepatis and two closer apices at the attachment of GB to the liver bed. The base of the triangle thus lies on the inferior surface of the liver.[22]

CONTENTS OF THE CALOT'S TRIANGLE[22]

1) Cystic artery.
2) Cystic lymph node (**Calot's node)**.
3) Lymphatics from the GB.
4) 1 or 2 cystic veins.

5) Autonomic nerves to the GB.

6) Adipose tissue.

7) May contain any accessory ducts which drain into GB from liver.

VASCULAR SUPPLY AND LYMPHATIC DRAINAGE

CYSTIC ARTERY

The cystic artery usually arises from the right hepatic artery. It usually passes posterior to the common hepatic duct and anterior to the cystic duct to reach the superior aspect of the neck of the gallbladder. It divides into superficial and deep branches, superficial branches ramifies on the inferior aspect of the gallbladder, the deep branches on the superior aspect. These arteries anastomose over the surface of the body and fundus. The cystic artery is an end artery and its occlusion is followed by the gangrene of the gall bladder.

ANATOMICAL VARIATIONS

1) May arise from common hepatic artery, sometimes from the left hepatic artery or rarely from the gastro duodenal or superior mesenteric arteries. In this case it may cross anterior (or less commonly posterior) to CBD or CHD to reach gallbladder.
2) An accessory artery may arise from the common hepatic artery or one of its branches.
3) The cystic artery often bifurcates close to its origin to give rise to two arteries supplying the gallbladder.

4) Multiple fine arterial branches may arise from the parenchyma of the liver (segment IV or V) and contribute to supply the body particularly when the GB is substantially intrahepatic. The cystic artery gives rise to multiple fine branches which supply the common and lobar hepatic ducts and the upper part of the CBD.

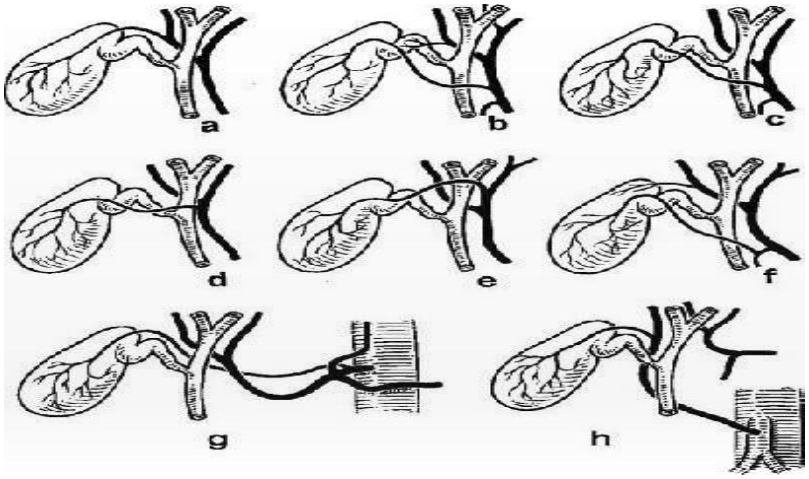


FIGURE 6: Variations of the cystic artery: typical course (a); double cystic artery (b); cystic artery crossing anterior to main bile duct (c); originating from the right branch of the hepatic artery and crossing the common hepatic duct anteriorly (d); originating from the left branch of the hepatic artery (e) originating from the gastro duodenal artery (f); arising from the celiac axis (g); originating from a replaced right hepatic artery (h)

DUCTAL ARTERIES :

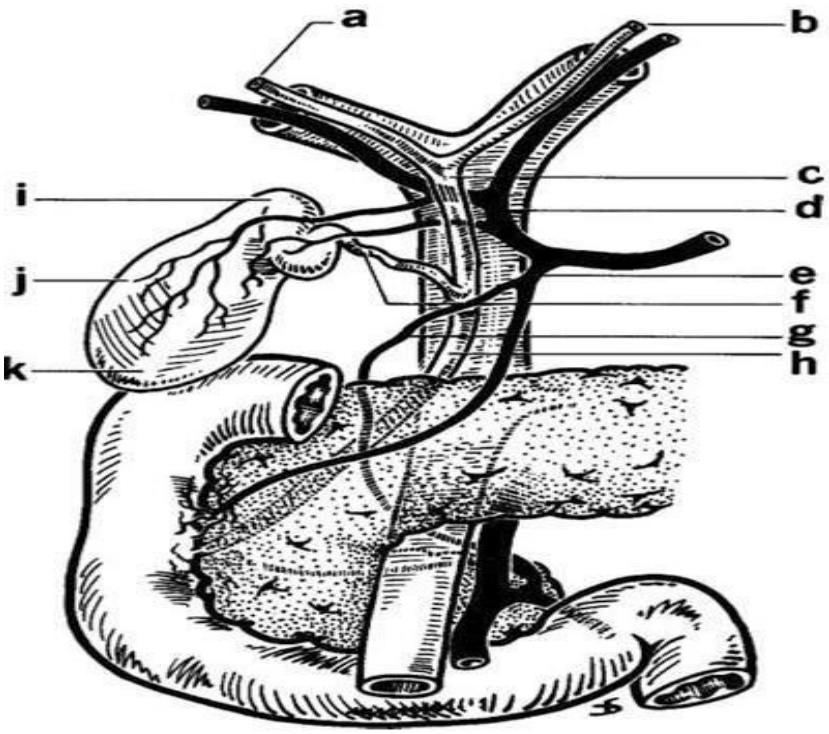


FIGURE 7: Bile duct blood supply. Note the axial arrangement of the vasculature of the supra duodenal portion of the main bile duct and the rich network enclosing the right and left hepatic ducts: right branch of the hepatic artery (a); 9 o'clock artery (b); retro duodenal artery (c); left branch of the hepatic artery (d); hepatic artery (e); 3 o'clock artery (f); common hepatic artery (g); gastroduodenal artery (h).[21]

- The common bile duct and hepatic ducts are supplied by a fine network of vessels, which lie in close proximity to the ducts themselves.

  - Disruption of the network during surgical exposure of the bile ducts over a long length frequently causes chronic ischemia and stenosis.
  - Anterior to the CBD, 2 to 4 ascending vessels arise from the retro duodenal branch of the gastro duodenal artery. 3 to 4 descending

branches of the right hepatic and cystic arteries arise as this vessels pass close to the lower CHD.

- These descending and ascending arteries form long narrow anastomotic channels along the length of the duct called medial and lateral trunks.
- Posteriorly, a retroportal artery often arises from the coeliac axis, superior mesenteric artery or one of its major branches close to its origin from the aorta.
- It contributes to the arterial network supplying the supraduodenal part of bile duct system. It runs upward on the posterior surface of the portal vein.

CYSTIC VEINS

Those arising from the superior surface of the body and neck lie in the areolar tissue between the gall bladder and the liver and enter the liver parenchyma to drain into the segmental portal veins.

LYMPHATICS

Numerous lymphatic vessels run from the submucosal and subserosal plexuses on all aspects of the gall bladder and cystic duct. Those on the hepatic aspect of the gallbladder connect with the intrahepatic lymphatics. The remainder drain into the cystic node, which usually lies above the cystic duct in the tissue of Calot's triangle. This node, and some lymphatic channels which bypass the cystic node, drain into a node lying in the anterior border of the free edge of the lesser Omentum.

INNERVATION

The gall bladder and the extrahepatic biliary tree are innervated by branches from the hepatic plexuses. The retroduodenal part of the CBD also has contribution from the pyloric branches of vagus, which also innervate the smooth muscles of the hepatopancreatic ampulla.

- **REFERRED PAIN**

  In common with other structures of foregut origin, pain from stretch of CBD or gallbladder is referred to the central epigastrium. involvement of overlying somatic peritoneum produces pain which is more localized to the right quadrant.

❖ EMBRYOLOGY

- The liver primodium appears in the middle of the third week as an outgrowth of the endodermal epithelium at the distal end of the foregut. This outgrowth, the hepatic diverticulum or the hepatic bud consists of rapidly dividing cells that penetrate the septum transversum, that is the mesodermal plate between the pericardial cavity and the stalk of the yolk sac. While the hepatic cells continue to penetrate the septum, the connection between the hepatic diverticulum and the foregut (duodenum) narrows forming the bile ducts.
- On day 26, a distinct endodermal thickening appears on the ventral side of the duodenum just caudal to the base of the hepatic diverticulum and buds into ventral mesentery.[23]

This cystic diverticulum will form the GB and the cystic duct. Cells at the junction the hepatic and cystic duct proliferate and form the CBD. In the 10th week of development the weight of liver is approximately 10% of the total body weight due to large number of sinusoids and large nests of proliferating cells, which produce red blood cells and white blood cells. It lies between the hepatic cells and the wall of the vessels. Approximately at 12th week of life liver begins to produce bile, which is dark green in colour.

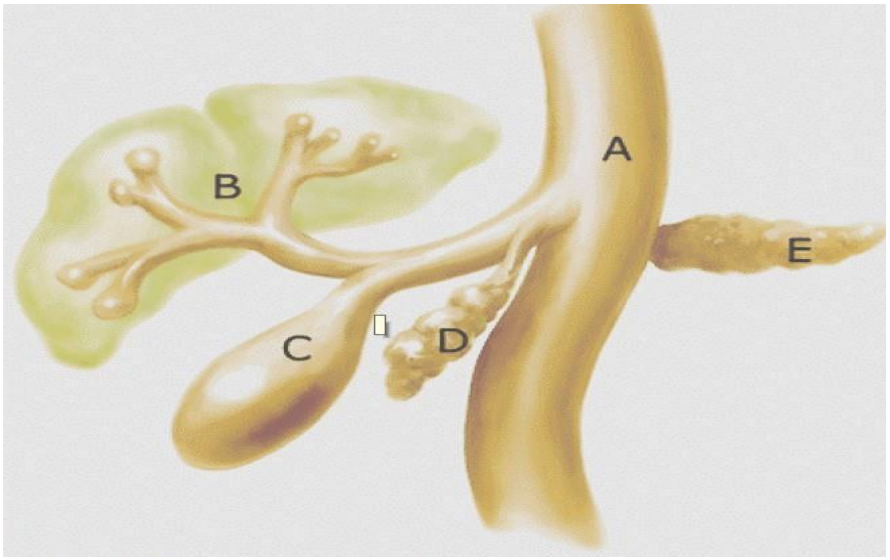


FIGURE 8: Illustrating the foregut (A), the cranial end of the hepatic diverticulum which represents Pars hepatica (B) and the Cystic diverticulum (C). The ventral (D) and dorsal (E) pancreas are also demonstrated[23]

- **HISTOLOGY GALLBLADDER**
  - The mucosa is yellowish-brown and elevated into minute 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. Basally, the spaces between epithelial cells are dilated. Many capillaries lie beneath the basement membrane. Beneath it is a thin fibromuscular layer composed of fibrous tissue mixed with smooth muscles which are arranged loosely in longitudinal, circular and oblique bundles.

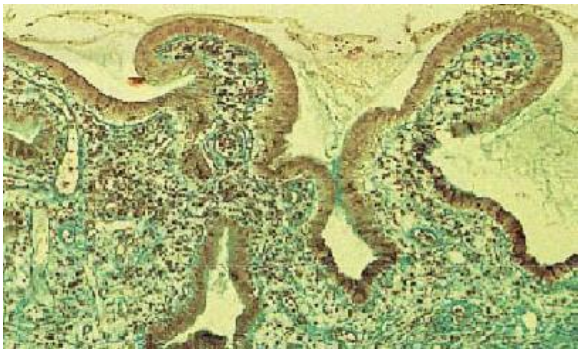


FIGURE 9: Microscopy of gall bladder wall

BILE DUCTS:

The larger biliary ducts have external fibrous and internal mucous layers. The former is fibrous connective tissue which contains variable amount of connective tissue which contain variable amount of longitudinal, oblique and circular smooth muscles. The epithelial covering is columnar and contains many tubuloalveolar mucous glands.

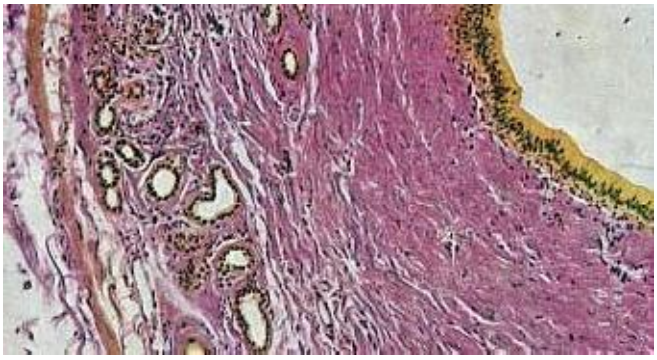


FIGURE 10: Microscopy of common bile duct

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. Entire pool recycles twice per meal and 6 to 8 times per day

Table 1: 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	-------

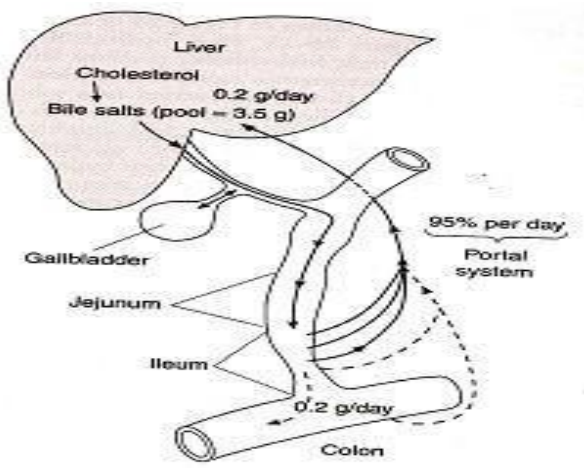


FIGURE 11: Showing enterohepatic circulation of bile salts.

BILIRUBIN METABOLISM AND EXCRETION

Most of the bilirubin in the body is formed by the breakdown of hemoglobin. it is bound to cytoplasmic proteins. It is conjugated to glucuronic acid by UDP-glucuronyl transferase, This diglucuronide is water soluble and is transported actively against concentration gradient into bile canaliculi.

A small amount of bilirubin glucuronide escapes into blood, where it is bound to albumin and excreted in urine.The intestinal mucosa is relatively impermeable to conjugated bilirubin but is permeable to unconjugated bilirubin and to urobilinogen. Small amounts of urobilinogen enters the general circulation through portal circulation and is excreted in urine.

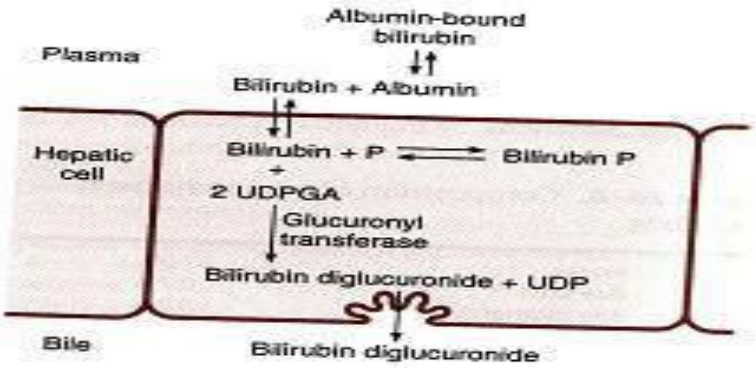


FIGURE 12: Metabolism of bilirubin in liver. p-intracellular binding protein, udpga-uridine diphosphate glucuronic acid, udp-uridine diphosphate.

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 gall bladder contraction. Substances that cause contraction of gallbladder are called cholagogues.

PATHOGENESIS:

In the west, about 80% are cholesterol stones, containing more than 50% of crystalline cholesterol monohydrate. The remainder are composed predominantly of bilirubin calcium salts and are designated pigment stones.

CHOLESTROL STONES

Cholesterol is rendered soluble in bile by aggregation with water soluble bile salts and water insoluble lecithin, both of which act as detergents. When cholesterol concentration, exceed the solubilizing capacity of bile (supersaturation).

1) Bile must be supersaturated with cholesterol: this appears to be a primary defect, mediated by abnormal regulation of hepatic mechanisms for delivering cholesterol to bile. The excess free cholesterol is toxic

to gallbladder, penetrating the wall and exceeding the ability of the mucosa to detoxify it by esterification. Gallbladder hypo motility ensues. Muscular stasis appears to result both from intrinsic neuromuscular dysmotility and decreased response neuromuscular response to CCK.[24]

2) Gallbladder hypomotility promotes nucleation.[24]
3) Cholestrol nucleation in bile is accelerated: due to shift in balance between antinucleating and pronucleating proteins and presence of micro precipitates of inorganic or organic calcium salts.
4) Mucus hypersecretion in the GB traps the crystals, permitting their aggregation into stones.

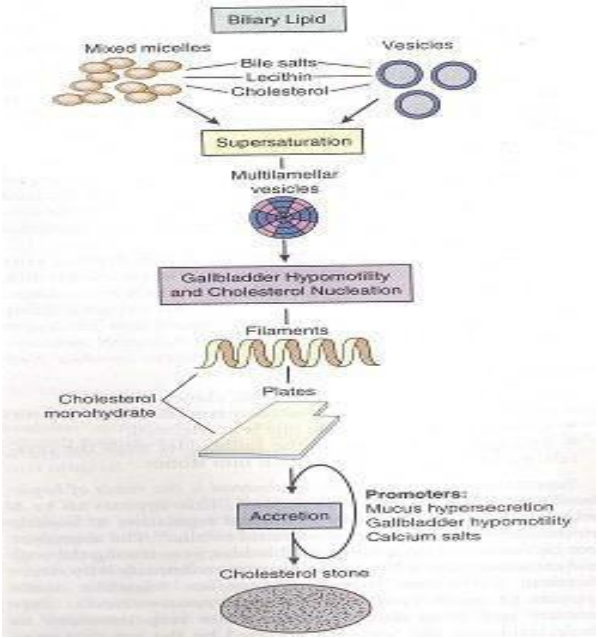


FIGURE 13: Schematic representation of four contributory factors for cholelithiasis: supersaturation, gallbladder hypomotility, crystal nucleation and accretion within the gallbladder mucous layer.[24]

TABLE 2: Superimposed conditions that exacerbate defective GB emptying and cholesterol stone formation

Prolonged fasting	Total parentral nutrition
Pregnancy	Spinal cord injury
Rapid weight loss	

PIGMENT STONES

Pigment stones are complex mixtures of abnormal insoluble calcium salts of unconjugated bilirubin along with inorganic calcium salts. Infection of biliary tract with E.coli or ascaris lumbricoids or by the liver flukes opisthorchis sinensis leads to release of microbial β-glucuronidase, which hydrolyses bilirubin glucuronides to unconjugated bilirubin.[24]

MORPHOLOGY

CHOLESTEROL STONES

Arises exclusively in GB and are composed of cholesterol ranging from 100 to 50%. Pure cholesterol stones are pale yellow, round to ovoid and have a fine granular, hard external surface which on transection reveals a glistening radiating crystalline palisade. With increasing proportions of calcium carbonate, phosphates and bilirubin, the stones exhibit discolouration and may be lamellated and gray white to black on transection.[7,24]

Most often multiple stones are present that range upto several centimeters in diameter. Surfaces of multiple stones may be rounded or faceted, owing to tight

apposition. Stones composed largely of cholesterol are radiolucent; sufficient calcium carbonate is found in 10 to 20% of cholesterol stone to render them radiopaque.

PIGMENT STONES

Are classified as black and brown stones. Black pigment stones are found in sterile gallbladder bile, and brown in infected intrahepatic and extrahepatic ducts.

Mucin glycoproteins act as binding proteins in both cholesterol and pigment stones.

THE NATURAL HISTORY OF GALLSTONES

In 1992, it was estimated that 10% to 15% of the adult population in the United States had gallstones, about 1 million patients are newly diagnosed annually. Gallstones are the most common digestive disease.[25]

EPIDEMIOLOGY:

Gallstones are most common gastrointestinal illness with a prevalence of 11 to 36% in autopsy reports. Only first degree relatives of the patients with gallstones and obesity (BMI >30 kg/m^2) have been identified as strong risk factors for the development of symptomatic gallstone disease.[26]

TABLE 3: Risk factors for gallstones

Obesity	First degree relatives
Rapid weight loss	Drugs: Ceftriaxone, postmenopausal estrogens, total parenteral nutrition
Childbearing	Ethnicity: Native American(Pima Indian) , Scandinavian
Multiparity	Ileal disease, resection or bypass
Female sex	Increasing age

CLINICAL PRESENTATION

Most patients remain asymptomatic from their gallstones. Although the mechanism unclear, some patients develop symptomatic gallstones with biliary colic caused by a stone obstructing the cystic duct. Only 1% to 2% of asymptomatic individuals with gallstones develop serious symptoms or complication related to their gallstones per year; therefore only about 1% require cholecystectomy. Once symptomatic, patients tend to have recurring symptoms, usually repeated episodes of biliary colic. Nonspecific gastrointestinal symptoms develop in 10 to 30% of patients and 5 to 10% of patients develop classic biliary symptoms.

BILIARY COLIC

Acute obstruction of the gallbladder by calculi results in biliary colic, a common misnomer because the pain is not colicky in the epigastrium or right upper quadrant.

Biliary colic is a constant pain that builds in intensity and can radiate to the back, interscapular area or right shoulder. The pain is described as a band-like tightness of the upper abdomen that may be associated with nausea and vomiting.[27]

This is due to a normal gallbladder contracting against a luminal obstruction, such as a gallstone impacted in the neck of the neck of the gallbladder, the cystic duct or the CBD. The pain is most commonly triggered by fatty foods, but it can also be initiated by other types of food or even occur spontaneously. An association with meal is present in only 50% of patients, and in these patients, the pain often develops more than 1 hour after eating.

INVESTIGATIONS

ROUTINE BLOOD INVESTIGATIONS

Includes complete haemogram, renal function tests and ECG

LIVER FUNCTION TEST[27]

Biliary colic, in the absence of gallbladder pathology orcommon bile duct obstruction don't produce abnormal laboratory values. Obstructive choledocholithiasis have raised direct bilirubin and elevated alkaline phosphatase levels.[27]

Leukocytosis predominantly neutrophils are present in a Cholecystitis and cholangitis.

PT-INR : Prolonged PT is present in liver dysfunction which needs to be normalized before surgery.

❖ **INVESTIGATIONS / INTERVENTIONS:**

Investigations or interventions required in this study are routine standardized procedures for postoperative follow-up:

1. Complete blood count.
2. Liver Function Tests.
3. Urine – sugar, albumin and microscopy.
4. Random blood sugar, FBS, PPBS, Blood urea, Serum creatinine, Blood grouping.
5. Electro-cardio-gram and Chest X-ray (when age of patient is >35yrs, or if necessary).
6. Ultrasonography of abdomen, (CT/CECT) if required.
7. Human Immunodeficiency disease, Hepatitis Band Hepatitis C serum markers.

IMAGING STUDIES

PLAIN RADIOGRAPHS

Only about 15% of gallstones contain enough calcium to render them radiopaque and therefore visible on plain abdominal films. Plain films are important to exclude perforated ulcer with free intraperitoneal air, bowel obstruction with dilated loops, or right lower lobe pneumonia.

ULTRASONOGRAPHY

An ultrasound is the initial investigation of any patient suspected of disease of the biliary tree. Abdominal ultrasound is a part of routine evaluation in patients with cholelithiasis and has a sensitivity of >98% and sensitivity of >95%.[28] In addition to identifying gallstones, ultrasound can also detail signs of cholecystitis such as thickening of the gallbladder wall, pericholecystic fluid, and impacted stone in the neck of the gallbladder. Dilation of the extrahepatic (>10 mm) or intrahepatic (>4 mm) bile ducts suggests biliary obstruction.[5]

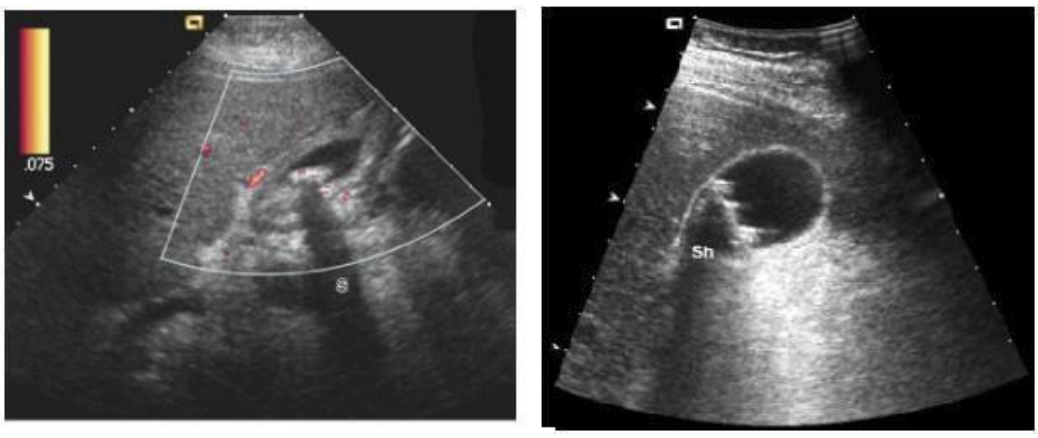


FIGURE 14: A, Echogenic foci in the gallbladder with acoustic shadowing (S) are characteristic of gallstones. In this patient, the gallbladder wall is thickened, but not hypervascular. Features suggest chronic cholecystitis. B, Multiple stones are layered in the dependent portion of the gallbladder, but the wall is not thickened.[29]

ORAL CHOLECYSTOGRAPHY

Identifies filling defects in a visualized, opacified gallbladder after oral administration of a radio-opaque compound that passes into the gallbladder.[28] It is contraindicated in patients with vomiting, biliary obstruction, jaundice, or hepatic failure.

COMPUTED TOMOGRAPHY

CT identifies gallstones within the biliary tree and gallbladder with a sensitivity of only about 55% to 65%. This is because both gallstone and bile are isodense and stones are identified only if they are calcified.[30]

SCINTIGRAPHY

Scintigraphy is useful to visualize the biliary tree, assess liver and gallbladder function. Nonvisualization of the gallbladder at 2 hours after injection is reliable evidence of cystic duct obstruction. Biliary scintography followed by CCK administration is helpful for documenting biliary dyskinesia when gallbladder contraction accompanies biliary track pain in patients without evidence of stones (CCK hepatobiliary 2,6- dimethyl-iminodiacetic acid (HIDA).[31]

INTRAOPERATIVE CHOLANGIOGRAPHY

The first operative cholangiogram was reported in 1936 by Micken. Mirizzi in 1937 performed the first cystic duct cholangiography and this procedure remains the most accepted method for performing (IOC) today.[32]

TECHNIQUES Cystic duct cholangiography. Gall bladder Cholangiography.

Table 4 : Indications for routine IOC.

Detection of unsuspected CBD stones
To detect anomalous anatomy
Presence of accessory duct
Short cystic duct
Identification of iatrogenic injury

COMPLICATIONS OF GALLSTONES[33]

1) Acute cholecystitis
2) Chronic calculus cholecystitis
3) Choledocholithiasis with or withoutcholangitis
4) Gallstone pancreatitis
5) Gallstone ileus
6) Gallbladdercarcinoma.

MANAGEMENT OF CHOLELITHIASIS

The non operative management of gall stones has long fascinated physicians. the idea of dissolving gall stones attracted early interest with Durande in 1782. In 1975, Makino reported gall stone dissolution by administering ursodeoxycholic acid.

EXTRACORPOREAL SHOCK WAVE LITHOTRIPSY(ESWL)

ESWL is in use since 1986. It is used to fragment stones. Patient selection is very crucial for success and are selected according to criteria.

The criteria are functioning of gall bladder and stone should be

i. Cholestrol stone
ii. Less than 3 in number
iii. Less than 3 cm.

Recurrence rate is 5-7% at 12 months and 15% at 24 months.

MEDICAL MANAGEMENT

Ursodiol (urosodeoxy cholic acid) constitutes less than 5% of total bile salt pool.[34]

CLINICAL USES

1) Dissolution of small cholesterol gallstones in patients with symptomatic gallstones who refuse cholecystectomy or who are poor surgical candidates. At a dosage of 10 mg/kg/day for 12 -24 m, dissolution occurs in upto 50% of patients with small (<5-10 mm) non calcified gallstones.[35]
2) Prevention of gallstones in obese patients undergoing rapid weight loss therapy.[35]
3) At a dosage of 13-15mg/kg/d is helpful for patients with early stage primary biliary cirrhosis, reducing liver function abnormalities and improving liver histology.

ADVERSE EFFECTS

i. Ursodiol is practically free ofserious adverse effects.

ii. Bile salt induced diarrhea is uncommon.

PREOPERATIVE PREPARATION

1) Blood coagulation should be normalized in patients with prior, by giving vitamin K (IM in 3 doses).

2) A prophylactic antibiotic either with premedication or at the time of anesthesia induction is given. A second generation cephalosporin is appropriate.[36]

3) Subcutaneous heparin or antiembolic stocking are used to prevent deep vein thrombosis.

OPEN CHOLECYSTECTOMY

Indications

1) Poor pulmonary or cardiac reserve
2) Suspected or known gallbladder cancer
3) Cirrhosis and portal hypertension.
4) Third-trimester pregnancy
5) Combined procedure
6) Conversion from laparoscopic approach

A short right upper transverse incision is made centered over the lateral border of the rectus muscle-kocher's incision.[36]

The gallbladder is appropriately exposed and packs placed on the hepatic flexure of the colon, the duodenum, and the Lesser omentum to clear view of the anatomy of the porta hepatis.

This packs are retracted using the left hand of the assistant, or a stabilized ring retractor is used to keep the pack in position. A duval forceps is placed on the infundibulum of the gallbladder and the peritoneum overlying calot's triangle is stretched. [36]

The calot's triangle is dissected to expose the cystic duct and the cystic artery.[36] These are confirmed by tracing them to enter the gallbladder. The cystic artery is ligated and cut. The cystic duct is then ligated and divided. A suction drain is placed before closure.

When there is doubt about anatomy, a fundus first or retrograde cholecystectomy dissecting on the gallbladder wall down to the cystic duct can be helpful. [36]

LAPAROSCOPIC CHOLECYSTECTOMY

LC is one of the most common surgeries performed and has replaced open cholecystectomy. In 1992, The National Institute of Health (NIH) [58] consensus development conference stated that laparoscopic cholecystectomy "provides a safe and effective treatment for most patients with symptomatic gallstones.[5]

INDICATIONS [37]

a) **Symptomatic cholelithiasis**

i) Biliary colic: Once the patient experience symptoms, there is a greater than 80% chance that they will continue to have symptoms. There is also a finite risk of disease related complications such as acute cholecystitis, gallstone pancreatitis and choledocholithiasis.

ii) Acute cholecystitis.

iii) Gallstone pancreatitis.

b) **Asymptomatic cholelithiasis**

Patient with asymptomatic gallstone have less than 20% chance of ever developing symptoms, and the risks associated with prophylactic operation outweigh the potential benefit of surgery in most patients. [38]

Therfore, prophylactic cholecystectomy is recommended in :

1. Sickle cell disease[39]
2. Total parenteral nutrition
3. Chronic immunosuppression.
4. No immediate access to health care facilities (eg: missionaries, military personal, peace corps workers, relief workers).
5. Incidental cholecystectomy for patients undergoing procedures for other indications.

c) Acalculous cholecystitis or biliary dyskinesia

d) Gallbladder polyps >1 cm in diameter.

e) Porcelain gallbladder.

CONTRAINDICATION TO LAP CHOLECYSTECTOMY

ABSOLUTE

1) Unable to tolerate general anesthesia.
2) Refractory coagulopathy
3) Suspicion of carcinoma[39]

In porcelain gallbladder and potentially curable GB malignancy, due to persistent concerns[39] with adequacy of resection and reports of port site metastasis associated with the use of minimally invasive surgical technique for treatment of intra-abdominal malignancies.[39]

RELATIVE

1) Previous upper abdominal surgery[39]
2) Cholangitis
3) Diffuse peritonitis with hemodynamic compromise
4) Cirrhosis and /or portal hypertension.

   Brittle, friable liver that may be difficult to retract in cephalad direction, associated coagulopathy and due to abnormal portosystemic venous shunts in portal hypertension.

5) Cholecystoenteric fistula.
6) Chronic obstructive pulmonary disease.
7) Pregnancy.

Due to unknown effect of Co^2 on foetus-therefore avoided in first trimester. Open insertion of port or location of initial port in right upper quadrant to avoid damage to uterus. Maintenance of pneumoperitoneum to <12 mm of hg and maternal hyperventilation with monitoring of pco2 is needed to avoid fetal acidosis. [39]

PATIENTS LIKELY TO REQUIRE CONVERSION

a) Multiple prior operations due to difficulty in safe access to peritoneal cavity.

b) Acute severe cholecystitis: Due to difficult dissection secondary to inflammation, adhesions or edema.

c) Acute pancreatitis: Difficult visualization of calot's triangle due to edematous pancreatic head.

d) Abnormal anatomy: Higher likelihood of biliary/vascular injury.

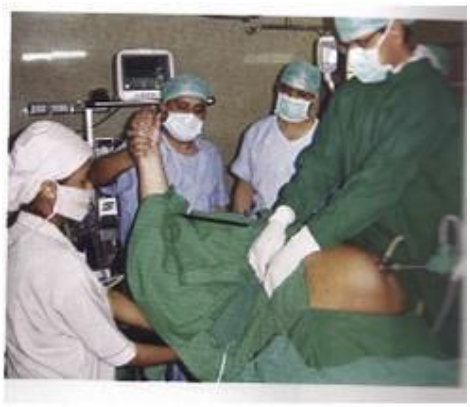
e) Cirrhotic liver: Higher likelihood of liver injury and haemorrhage.

f) Third trimester pregnancy: Higher likelihood of uterine injury during access.

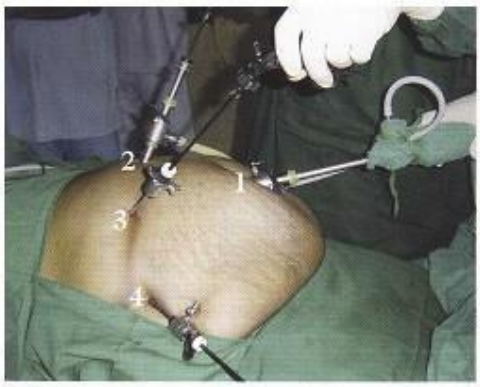
g) Morbid obesity: Difficulty in access and dissection.

h) Evidence of generalized peritonitis. Septic shock from cholangitis.

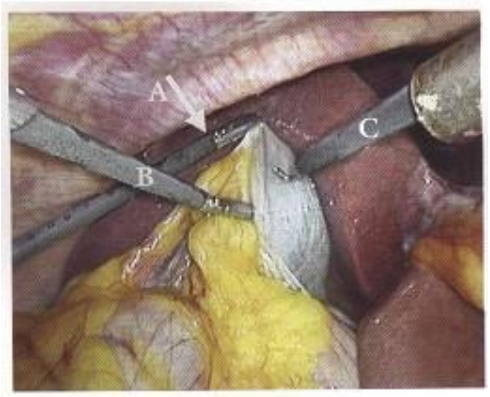
FIGURE 15: Showing steps of laparoscopic cholecystectomy



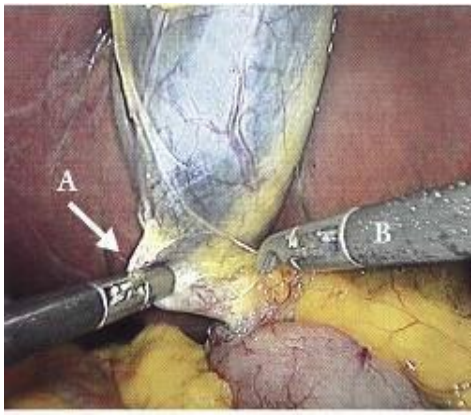
STEP 1: Patient Position



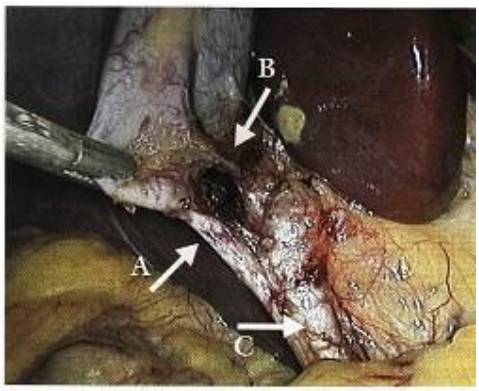
STEP 2: Port Placement



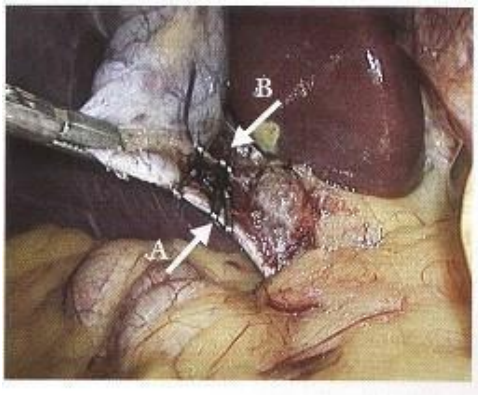
STEP 3: Exposure of Porta Hepatis



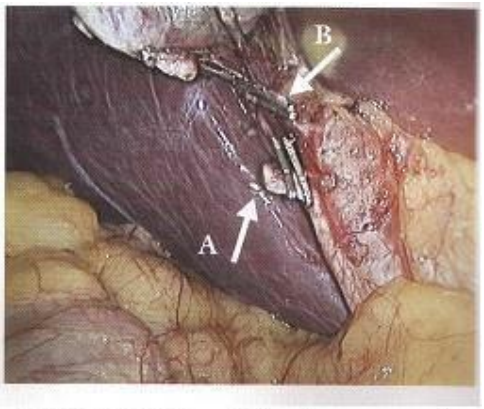
STEP 4 : Dissection of Calot's Triangle



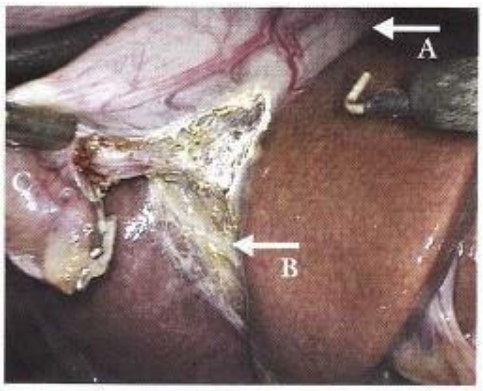
STEP 5: Identification of Cystic Duct(A), Cystic Artery(B) and CBD(C)



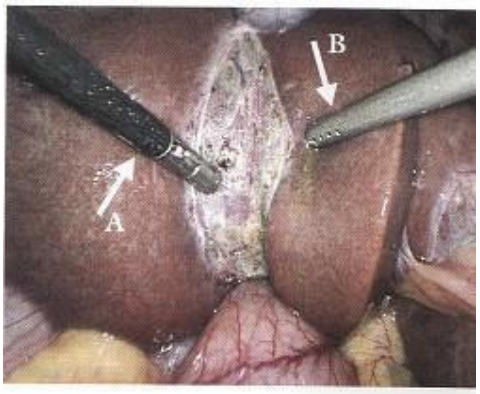
STEP 6: Clipping of Cystic artery and Cystic duct



STEP 7: Division of Cystic artery and Cystic duct

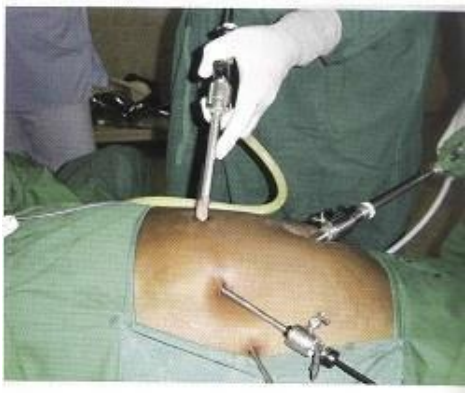


STEP 8: Detachment of GB from the Liver bed



STEP 9: Inspection of Liver bed

STEP 10: Etraction of GB



STEP 11: Extraction of the entire unit

APPROACHES

A) NORTH AMERICAN APPROACH

The patient is kept in supine in anti-trendlenburg position (15 degree head up tilt) with left lateral tilt (15-20 degree).this ensures that the bowel and Omentum falls down and medially, away from the operative site. The operating surgeon and camera surgeon stand on the left of the patient while the assistant surgeon stands on the right of the patient. Two monitors are placed at 10'0 and 2'0 clock position.[40]

PORT PLACEMENT

Ports are placed by screwing motion. Second hand is used to prevent inadvertent plunge of the trocar. The assistant should provide counter traction on the abdominal wall during placement of the first trocar.

10 mm port is placed in the midline,[41] usually through the umbilicus. Sub-umbilical position preferred in patients with cirrhosis due to the presence of dilated, tortuous anastomotic veins in the periumbilical region, visceroptic liver, hepatomegaly and in patients with pendulous abdomen.

If a previous abdominal surgery has been performed through a vertical midline incision, abdomen in insufflated through a site adjacent to the umbilicus, and a primary 5 mm trocar is placed in the right upper quadrant. The 10 mm trocar is then placed under direct vision, avoiding the adhesions from previous operation, under direct vision through a 5 mm telescope passed through 5 mm port.

Pneumoperitoneum is created through Hasson technique if previous surgery prevent primary puncture through the umbilicus.[42]

Another 10 mm port is placed in the epigastrium starting from the midline and angling toward the gallbladder, at the level of the inferior edge of the liver and to the right of falciform ligament.

If it is placed too high, segment IV of the liver will impede the ability to get to the gallbladder.[42]

A 5 mm trocar is placed 2 to 3 cm below the costal margin in the midclavicular line. The fourth, a 5 mm trocar is generally placed in the anterior axillary line, several centimeters below the fundus of the gallbladder, but its position is variable.[42]

B) FRENCH/EUROPEAN APPROACH

The patient is in semi- lithotomy antitrendlenburg position with leg in allen stirrups such that the thighs are almost parallel to the ground. The operating surgeon stands between the legs of the patient with the camera surgeon on the right of the patient and the assistant on the left of the patient.[43]

PORT PLACEMENT

A camera port is placed at umbilicus, 5 mm epigastric port is placed to allow retraction by assistant, 10 mm right hand working port is placed in left hypochondrium or in the midline between the camera port and the epigastric port and the left hand working port (5 mm) is placed in the right hypochondrium.

ADDITIONAL PORT

a) Left lumbar 5 or 10 mm port for three prong or flat blade retractor for downward traction of the colon, Omentum and duodenum. This maneuver gives wide exposure of the hilum.

b) 5 mm port midway between epigastric and right midclavicular ports for lifting the quadrate lobe using blunt tipped retractors (French technique), eg in liver cirrhosis, left lobe gallbladder.

PNEUMOPERITONEUM

LC is generally performed with a carbondioxide pneumoperitoneum at a pressure of 15 mm of Hg pressure. Other gases like helium and argon are being tried.[42]

TECHNIQUES:

a) VERESS NEEDLE TECHNIQUE[42]

In veress needle technique; pneumoperitoneum is generally created by sliding a veress needle through the umbilicus, confirming its position by allowing saline to run through the needle from a plungerless syringe, and then attaching the needle to tubing from carbon dioxide. Once it is confirmed to be intra- abdominal, the flow rate can be increased until 15 mm Hg of pressure is attained. [42]

b) OPEN (HASSON) LAPAROSCOPY TECHNIQUE

In open technique, abdominal cavity is entered under direct vision. Once the peritoneal cavity is entered, the initial trocar is inserted and its position is secure with two stay sutures. The abdominal cavity can then be insufflated with carbon dioxide. [42]

STEPS

A) PATIENT PREPARATION, EQUIPMENT AND ANAETHESIA

EQUIPMENT

a) High-quality videoscope with a 300 w light source be coupled to two high-resolution monitors. [42]

b) High-flow carbon dioxide insufflator.

c) Four trocars: 2-10 mm trocars and 2-5 mm trocars.

d) Hand instruments: Monopolar electrode c-hook with suction and irrigation, a fine tipped dissector, two gallbladder grasper, a large gallbladder extractor, scissors and a hemoclip applier.

e) 10 mm stone retrieval grasper.

f) Micro scissor, a specialized cholangiogram clamp and a 4 or 5 mm French catheter to perform cholangiogram. [42]

ANAESTHESIA TECHNIQUE[42]

Generally, nitric oxide is avoided to minimize the likelihood of bowel distention. Intravenous fluids must be run carefully as the insensible fluid losses through the closed abdomen are minimized and pneumoperitoneum is a strong stimulator of antidiuretic hormone. End tidal pco2 is monitored to check for hypercarbia and acidosis secondary to carbon dioxide pneumoperitoneum.

Narcotics are used in smaller doses and powerful antiemetic is used to lessen postoperative nausea. Once the patient is anesthetized and intubated, a foley catheter, sequential compression devices and orogastric tube are generally placed. [42]

North American approach is generally followed.

B) <u>EXPOSURE OF PORTA HEPATIS</u>

The fundus[25] of the gallbladder is held with a ratchet grasper and retracted by the assistant in a cranial direction, which lifts the right lobe of the liver and exposes the calots triangle and hilum of the liver. Adhesions to the underside of the liver and bladder are carefully taken down beginning near the fundus and proceeding down towards the neck.

C) DISSECTION OF THE CHOLECYSTOHEPATIC TRIANGLE (CALOTS TRIANGLE)

In tensely distended GB, it may be decompressed in two ways- percutaneous veress needle aspiration or the midclavicular trocar is

introduced into the fundus of the gallbladder directly and content aspirated. An atraumatic grasper is introduced through the left hand working port to hold the infundibulum and retract it downwards and to the right. Using a Maryland's forceps introduced through the epigastric port, the peritoneum of the infundibulum is held and breached by using small bursts of cautery current. Peritoneum on anterior and posterior aspect are stripped down. The infundibular grasper in moved inferolaterally and superomedially (flag technique) to aid the dissection of anterior and posterior surface of calot's triangle.[45]

D) IDENTIFICATION OF THE CYSTIC DUCT AND ARTERY

Methods for ductal identification in laparoscopic cholecystectomy

i) Infundibular or infundibular-cystic technique: In this method the cystic duct is isolated by dissection on the front and the back of the triangle of calot's and once isolated it is traced on to the gallbladder.

ii) Critical view of safety triangle: method requires complete dissection of the cholecystohepatic triangle and separation of the base of the gallbladder infundibulum from the liver bed. When this view is achieved, the two structures entering the gallbladder can only be cystic duct and artery. It is not necessary to see the common bile duct.

Cystic duct is identified at the junction of gallbladder (SAFETY ZONE) and followed down for adequate length for cholangiography. It is not necessary to identify and dissect cystic duct CBD junction (DANGER ZONE).[46]

Cystic artery is identified along with its anterior and posterior branches by blunt dissection.[46] The cystic node sometimes overlies the cystic artery. Both the cystic duct and artery are clipped, two clips on the cystic duct side and one on the gallbladder side. Before clipping, the cystic duct the stones in the cystic duct are milked back to GB, Artery is divided before the duct but in certain cases duct is divided first to give exposure to the artery. In case of an impacted cystic duct stone, the cystic duct is clipped at its junction with GB and a partial cut is made just distal to the clip and impacted stone milked back and extracted. [46]

E) DETACHMENT OF GB FROM THE LIVER BED

The GB can be detached from the liver bed using a spatula with monopolar cautery, hook with monopolar cautery, scissors with monopolar cautery or harmonic scalpel. Care should be taken to stay away from the porta hepatis and the liver bed and to avoid perforation of the gallbladder.[47] Traction and counter traction facilitate dissection. Any inadvertent spillage of bile or stones from the GB during the procedure should be immediately controlled by applying clips, pre-tied loops or reapplying the grasping clamp. [46]

Spilled bile is immediately sucked and stone removed. Prior to complete detachment of the gallbladder, the liver bed is inspected for adequate hemostasis or bile leak. The cystic duct remnant and cystic artery stumps are examined. Minor oozing from liver bed is controlled

with cauterizing and continuous suction irrigation. Once complete hemostasis is achieved GB is separated completely.[46]

F) EXTRACTION OF THE GB

Extraction of the GB can be done through umbilical or epigastric port. Epigastric port is preferred because:

a. No need to change camera port.
b. Facilitates thorough rinsing to avoid port tract infection
c. By extending skin incision, the fascial opening can be easily dilated and majority of GB extracted
d. Fascial opening closed easily by cutaneous approach.
e. Better cosmetic appearance.

A claw shaped gallbladder extraction forceps is introduced and used to grasp the neck of the GB. If GB is too distended the neck is pulled out through the skin incision, small nick made and bile suctioned and stones crushed using sponge holder. If the GB is thick preventing its extraction the fascial incision is enlarged. Infected or necrotic GB or a GB with suspicion of carcinoma is placed in a sterile bag before extraction to reduce port site infection.

G) FINAL INSPECTION AND IRRIGATION

After GB is extracted, the epigastric port is replaced and surgical site inspected for bleeding. A thorough wash is given to the GB bed, Morrison's pouch, paracolic gutter and perihepatic areas with saline which is later suctioned.

Venous ooze is controlled from the liver bed by

i. Gelatin sponge soaked in hemostatic solution.
ii. Use of harmonic ball application.
iii. Rarely intracorporeal suturing.
iv. Argon plasma coagulator.

H) DRAINGE AND CLOSURE

If drain is needed a 14F Redivac tube is placed through 5mm trocar site-lateral most port. Trocars are removed under direct vision to check for bleeding from trocar site. Pneumopritoneum evacuated and subcuticular stitch/skin clip/dermabond.

COMPLICATIONS

A) HEMORRHAGE

i) TROCAR SITE BLEEDING

Trocar site bleeding can be prevented by control of bleeding following skin incision and before inserting trocar.

Any subcutaneous vessel in subcutaneous tissue should be avoided during insertion.

Detection:

The blood may run down the abdominal wall or drip down the instruments into the operative field.[47]

Management: Pressure over the site of bleeding by tilting the trocar. Injection of epinephrine 1:10000 in the vicinity of the bleeding site.

Screwing in the anchoring device of a disposable trocar may compress and stop the bleeding. Suture ligation.

ii) HAEMORRHAGE DUE TO BLUNT DISSECTION OF ADHESIONS can be managed with electrocautery.

iii) SUDDEN AND PULSATILE BLEEDING IN CALOT'S TRIANGLE

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

Management: Retraction of the GB is released and the GB is gently pushed into the calot's triangle to obtain temporary respite during which additional port is placed between the umbilical and the epigastric ports, by repeated suction and irrigation, the blood is cleared from the operative field and the bleeding vessel is precisely identified and clipped.

iv) GALLBLADDER FOSSA BLEEDING

GB fossa bleeding can be controlled by electrocautery, packing the site with hemlock soacked gel foam, figure of eight stitch in case of spurter from liver parenchyma.

b) PERFORATION OF GB

GB perforation seen in acute cholecystitis and while detaching GB from the liver bed. This can be prevented by confining to the areolar tissue between the GB and the liver bed during dissection and decompression of the gall bladder if distended.

TABLE 5: Clinical presentation secondary to gallstone spillage

INFECTIVE	CUTANEOUS	MECHANICAL
Liver abscess	Sinus	Intestinal obstruction
Retrohepaticabscess	Port tract infection	
Subhepatic abscess	Granuloma formation	
Retroperitoneal abscess	Colocutaneous fistula	
Loin abscess		
Pelvic abscess		

Management:

Plenty irrigation and suction will remove majority of small stones while larger ones are removed using laparoscopic tissue pouch. Drainage catheter is placed. Perforated site must be closed with pre-tied ligature or by holding with the grasper.

c) DIFFICULTY IN EXTRACTION OF THE GALLBLADDER

Seen in gallbladder containing large stones and those with thick wall. In GB containing large stones, the GB is placed in an

endobag, the neck retrieved out through the abdomen and stones are crushed and removed. In GB with thickened wall, the GB is placed in an endobag and extracted.

d) OCCULT CARCINOMA

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.

e) POST OPERATIVE BILE LEAK

Post- operative bile leak can occur due to injury to the CBD, the right hepatic duct or accessory bile duct. This can be prevented by correct identification of the cystic duct and artery, minimum use of electrocautery in calot's triangle dissection and appropriate choice of laparoscopic subtotal cholecystectomy. Postoperative bile leak should be suspected in patients with fever, tachycardia and upper abdominal pain and tenderness persisting or appearing unexpectedly. The diagnosis can be confirmed by USG or ERCP.

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

f) BILE DUCT INJURY

Incidence of CBD injury during LC exceeds that of open cholecystectomy ie 0.5% vs 0.2%.[21] 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.[48]

PREVENTION

i) Use a 30 degree laparoscope and high-quality imaging equipment.

ii) Apply firm cephalic traction to the fundus and lateral traction to the infundibulum so that the cystic duct is perpendicular to the CBD.[49,50]

iii) Dissect the cystic duct where it joins the gallbladder.

iv) Expose the "critical view of safety" prior to dividing the cystic duct.[51]

v) Convert to open procedure if the infundibulum cannot be mobilized or bleeding or inflammation obscures the triangle of calot.

Perform routine intraoperative cholangiography. It is managed by biliary enteric anastomosis. This is to prevent cholangitis and biliary strictures.

g) BOWEL INJURY

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 undersurface of the umbilicus like the urachus or a meckel's diverticulum is more susceptible to injury during access.The rate of bowel injury between 0 and 0.4% has been reported in various studies.[43]

h) WOUND INFECTION AND INCISIONAL HERNIA

The risk of wound infection following laparoscopic cholecystectomy is less than 1% and the risk of incisional hernia is 0.5%.[23] Use of a retrieval bag for extraction of GB and closure of all port sites larger than 8mm may avoid these complications.

i) DIAPHRAGMATIC INJURY

j) PANCREATITIS.

k) PNEUMOPERITONEUM RELATED COMPLICATIONS

Pneumoperitoneum related complications include carbon dioxide embolism, vasovagal reflex, cardiac arrhythmias and hypercapnia acidosis.[39] Hypercapnia and acidosis are due to absorption of carbon dioxide from the peritoneal cavity. Sudden increases in $Paco_2$ may be related to port slippage and extraperitoneal or subcutaneous diffusion of co_2. It is managed by desufflating the abdomen for 10 to 15 min. If reinsufflation results in recurrent hypercapnia, then change the insufflations gas or convert to open. Carbon dioxide embolism is characterized by unexplained hypotension and hypoxia. Characteristic mill wheel murmur is detected on auscultation. This is produced due to the contraction of right ventricle against the blood gas

interface. There is an exponential decrease in end tidal co_2 due to complete right ventricular outflow obstruction.[52]

It is managed by immediate evacuation of pneumoperitoneum and placement of the patient in left lateral decubitus, head down (Durant) position.[53] This allows the co_2 bubble to float to the apex of the right ventricle, where it is less likely to cause right ventricular outflow obstruction. Patient is hyperventilated with 100% oxygen.

CONVERSION

In 5-10% of cases, conversion[6] to open cholecystectomy may be needed for safe removal of gallbladder, the risk factors for conversion were male sex, obesity, cholecystitis and choledocholithiasis.

ADVANTAGES AND DISADVANTAGES OF MINI LC COMPARED TO CONVENTIONAL LC

TABLE 6:

ADVANTAGES	DISADVANTAGES
Less post operative pain	Difficulty in Obese patients
Smaller incision	Slightly more operative time
Better cosmesis	Slight increase in bile duct injury
Shorter hospitalization	
Earlier return to full activity	
Decreased total costs	

RISK FACTORS OF DIFFICULT LAPAROSCOPIC CHOLECYSTECTOMY

i. CLINICAL RISK FACTORS

a) Stocky male patients due to difficulty in initial port placement

b) Multiparous women with flabby abdomen

c) Previous upper abdominal surgery

d) Cirrhosis of liver

e) Present or previous acute cholecystitis or acute pancreatitis

f) Previous treatment: percutaneous drainage / cholecystostomy

ii. ULTRASOUND CRITERIAS

a) Thick walled gallbladder (>4mm)

b) Contracted (nonfunctioning) gallbladder

c) Packed stones and large calcified GB.

d) Polyp or mass lesion without acoustic shadow

e) Evidence of acute cholecystitis:- impacted stones

f) Edematous gallbladder wall

g) Pericholecystic fluid collection

h) Emphysematous cholecystitis.

i) Subphrenic collection.

j) Intraperitoneal fluid collection due to perforated GB Hepatomegaly.

k) Cirrhosis of liver.

l) Portal vein thrombosis with cavernoma.

PROBLEMS IN DIFFICULT CHOLECYSTECTOMY ACCESS PROBLEMS

a) ADHESIONS

Post-operative adhesions: In lower abdominal scars, the veress needle is inserted at the site of proposed epigastric port. The umbilical port is inserted under visual guidance. In open appendicectomy scar, Hasson method is the ideal technique for creating pneumo peritoneum. In case of upper abdominal scars present in the midline or right Para median position, the left subcostal veress needle insertion (palmer's point) is used to create pneumoperitoneum.[55] Conversion rate as high as 25% has been reported in patients with extensive upper abdominal adhesions.

Inflammatory adhesions: is usually due to acute cholecystitis or acute severe pancreatitis. These adhesions can easily be removed using suction nozzle. But if the adhesions are organized then sharp dissection is done.[54]

b) INCISIONAL HERNIA

In cases of lower abdominal incisional hernias, appropriate repair could be accomplished after completing laparoscopic cholecystectomy either by open or laparoscopic technique.

c) OBESITY

The veress needle insertion and the insertion of first trocar is difficult. Cystic artery and cystic duct are covered with thick fat hence dissection is difficult.

d) CIRRHOSIS

Due to adhesions with increased vascularity, difficult traction of liver, inadequate exposure of hilum, high risk of GB bleed and high risk hilum.[25]

CONCOMITANT PATHOLOGY

a) MUCOCOELE

Mucocoele is difficult to retract and apply grasping forceps. It is managed by decompression of the GB, using toothed forceps for retraction of GB, removal of the impacted stone either by dislodging into the GB or through an incision over the cystic duct after applying distal clip.

b) GANGRENOUS GB

Due to difficulty in grasping, loss of tissue plane, difficulty in exposure of calot's triangle, performance of intraoperative cholangiogram is difficult, spillage of stones and infected bile; gangrenous GB is difficult to operate.

c) EMPYEMA

d) SCLEROATROPIC GB

The GB is contracted, fibrosed and densely covered with extensive adhesions. Adhesions of the duodenum and the colon are very common and access to calot's triangle is difficult due to fibrous scarring.[54]

e) MIRRIZZI'S SYNDROME

LC is difficult in Mirrizzi's syndrome due to contracted GB with extensive adhesions, CBD may be mistaken for cystic duct and chances of CBD injuries are more and if fistula is not recognized during surgery, biliary peritonitis may occur. [54]

f) PORCELAIN GB

The prevalence of Porcelain GB in cholecystectomy specimen ranges from 0.06% to 0.8%. Decompression of the gallbladder and traction is difficult due to calcified wall. Toothed forceps can be used for cranial traction of the GB. Calcification of the cystic duct may require endosuturing or application of endoloops to the cystic duct.

g) CHOLECYSTOENTERIC FISTULAS

Cholecystoenteric fistula is an incidental finding in 0.5 to 0.7% of cases of laparoscopic cholecystectomy for biliary disease. The diagnosis i s suspected by the presence of air in GB. Problems arise due to difficulty in identification of the anatomy, difficulty in performing cholangiography and due to the requirement of intracorporeal suturing for closure ofperforation.

h) ACUTE BILIARY PANCREATITIS

Difficulty in performing LC in acute biliary pancreatitis is due to- extensive adhesions, inflammatory phlegmon at the head of pancreas, edematous cystic duct and hepatoduodenal ligament, presence of ascites, pseudocyst pancreas in retrogastric position.

NEWER APPROACHES IN LAPAROSCOPIC CHOLECYSTECTOMY

a) GASLESS LAPAROSCOPIC CHOLECYSTECTOMY:

Gasless LC is especially useful in patients with cardiorespiratory problems. Here the abdominal wall is lifted mechanically allowing an adequate space for laparoscopic surgery.

b) SPA (SINGLE PORT ACCESS) CHOLECYSTECTOMY.

METHODOLOGY

▶ SOURCE OF DATA:

a) All patients admitted in Surgical Department in B.L.D.E.(Deemed to be University) Shri B.M. Patil Medical College, Hospital and Research Centre admitted for symptomatic Cholelithiasis/ Cholecystitis.

b) Period of study is from October 2016 to May 2018.

SAMPLING : (Prospective, interventional study)

- A Prospective Randomized Trial conducted by Yuri W. Novitsky, et al, titled "Advantages of Mini Laparoscopicand Conventional Laparoscopic Cholecystectomy was taken as reference study". [59]

- Considering the average standard deviation at permissible error the calculated

Sample Size is 66. i.e, **33** in each group.

Formula for estimating sample size

n = Sample size to be estimated.

$Z\ \alpha$ = Z value error, e = permissible error, σ =standard deviation.

Determination of sample size (n).

With 99% Confidence Interval, 95% Power and using cosmetic results,

Sample size is 66.

Z α = Z value error

$N = (Z\alpha + Z\beta) \times 2 \times (S.D.)^2/(M.D.)^2$

Z α = Z value at α level = 99% Z β= Z value at b level = 95%

S.D. =Common Standard Deviation (38.8±1.7 and 33.4±5.7)

M.D. = Difference between two parameters.

Hence **n=66** and 33 cases will be included in each group.

Statistical Analysis Data will be analysed using :

- Mean ± S.D.

- Student 'T' Test / Mann Whitney 'U' Test.

- Chi-Square Test./ Fisher's Exact Test

- Correlation Co-efficient if necessary.

METHOD OF COLLECTION OF DATA :

a) The patients will be allocated to study subgroup ie.,

b) Patients admitted for Cholecystectomy will be included in the study and allocated to study and control group alternatively.

c) Detailed history will be taken and thorough clinical examination and investigations will be performed for all the patients in both the study and control groups.

d) A Proforma will be used to collect all the relevant data from the patients pre, intra and post operatively.

e) All cases will be followed up to discharge and subsequently for a follow up of three months.

f) Following evaluation the patient will be subjected to laparoscopic cholecystectomy and time taken, biliary / stone spillage, injury to duct/ artery or conversion were noted.. Post operatively cases were followed up for any complication. Drain was removed between 2^{nd} and 5^{th} post OP day depending on the drainage, and Suture removal was done 8^{th} post OP day. All cases were followed up for any recurrent symptoms.

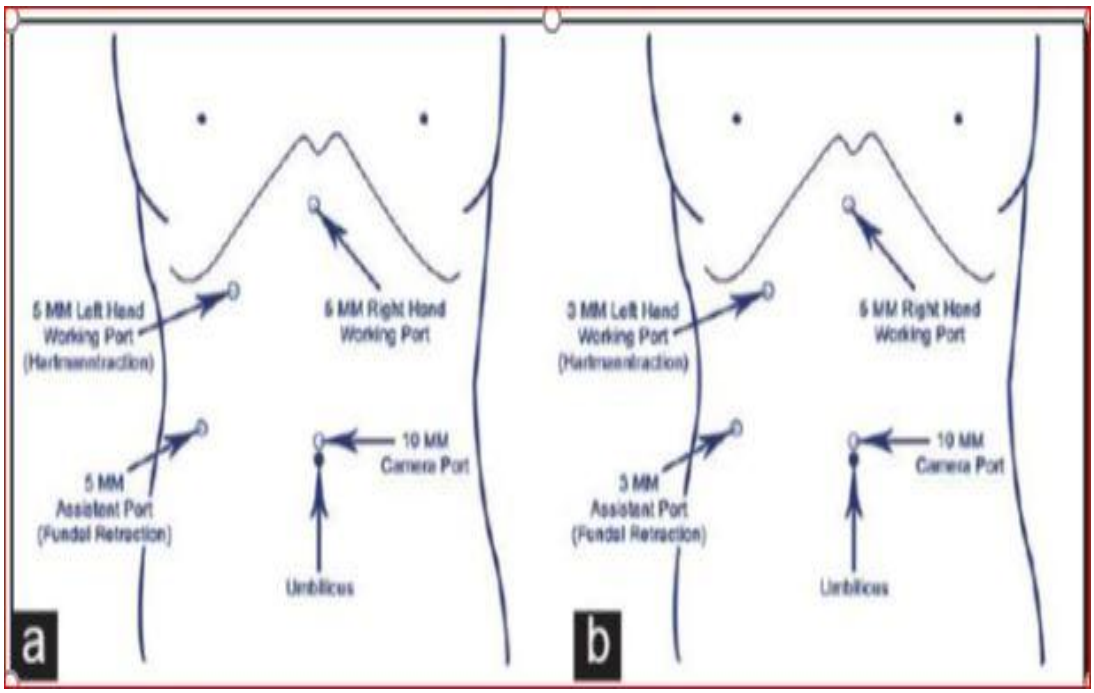
▶ INCLUSION CRITERIA :

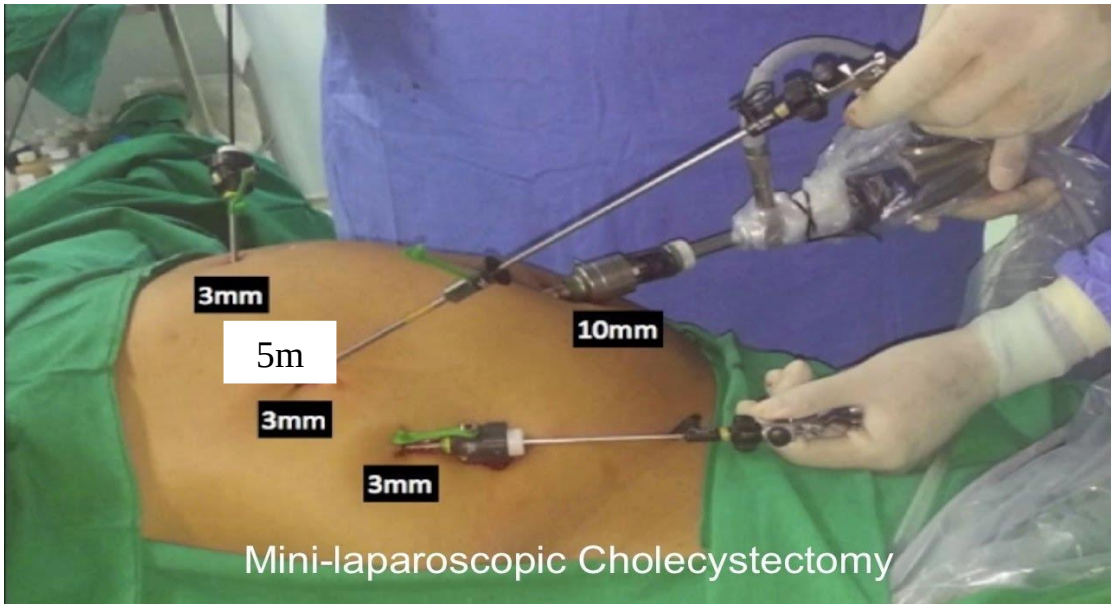
1) All Patients posted for Laparoscopic Cholecystectomy.

▶ EXCLUSION CRITERIA :

1) Age older than 70 years, (with uncontrolled diabetes and hypertension).
2) Patients with Liver or coagulation disorders.
3) Acute Cholecystitis with / without Phlegmon formation.
4) Empyema/ Gangrenous Gall Bladder.

Conventional Lap and Mini Lap cholecystectomy Port site Insertions :



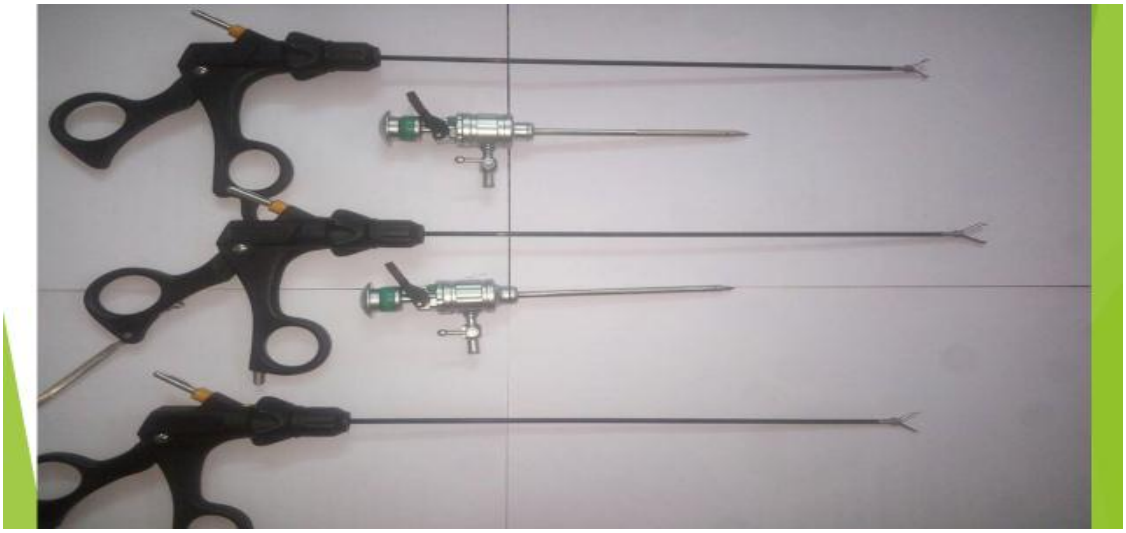


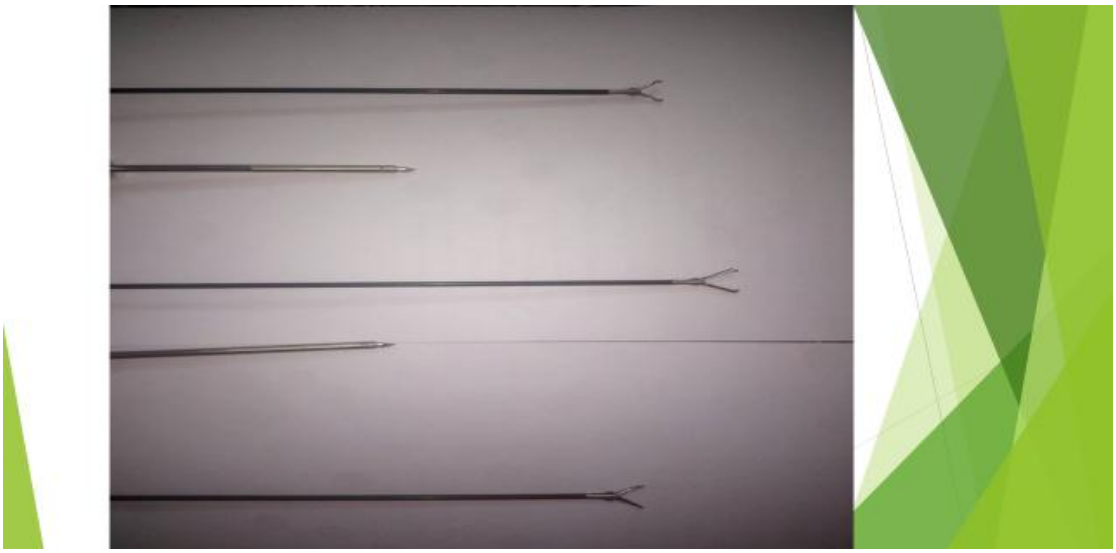
- Port site and instrument used at each site

Port site	C-LC	M-LC
Umbilical Port (Camera Port)	10 mm	10 mm
Epigastric Port (working port)	10 mm	5 mm
Subcostal Port (working port/Hartman traction)	5 mm	3 mm
Lateral Port (Fundal retraction)	5 mm	--/spinal needle/3mm

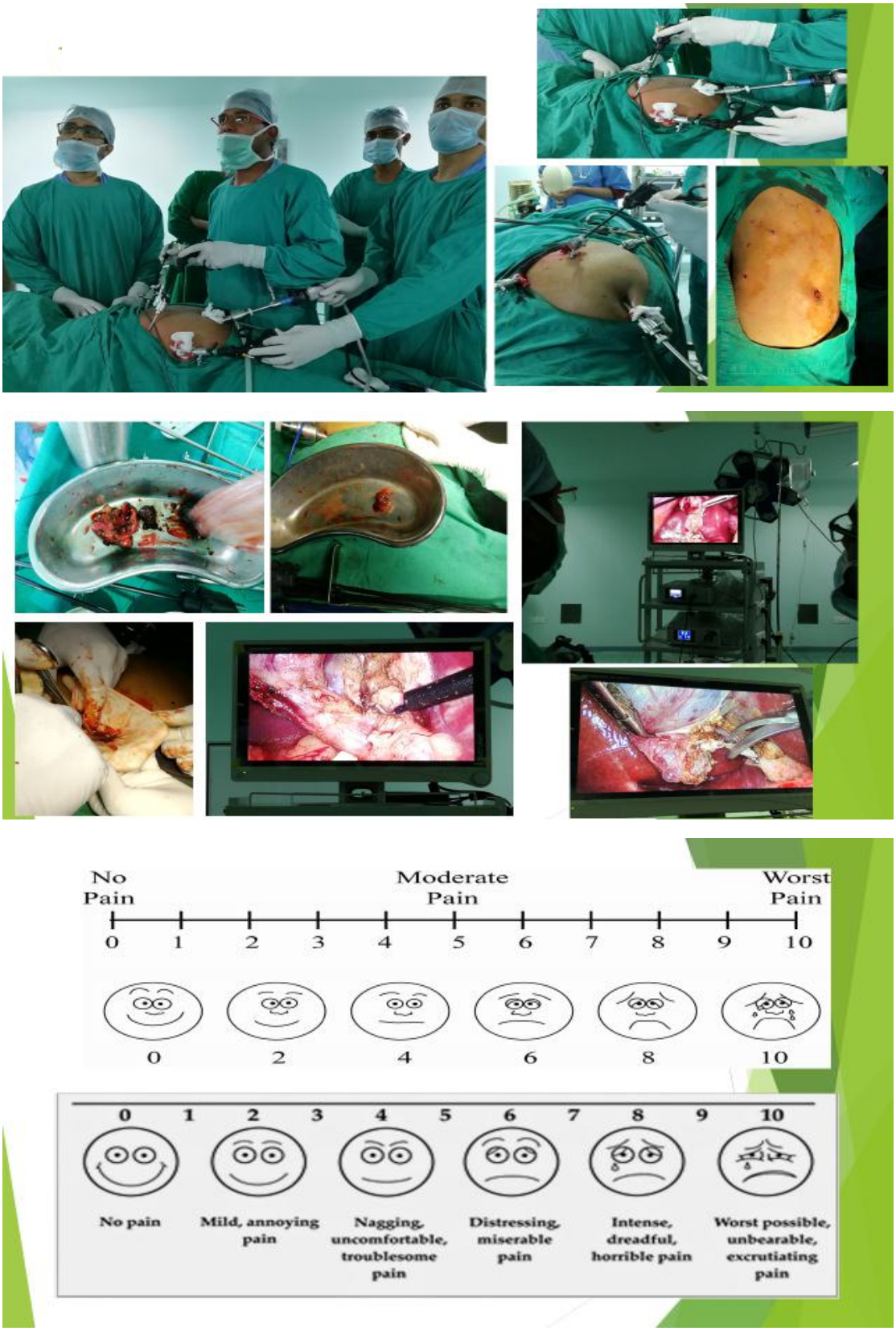
Stryker, Stryker Babcocks,

Mini Alligator Forceps





Mini Laparoscopic Instruments. (3mm) Maryland Grasper and Babcocks.



Visual analogue scale used for Pain scoring in my study.

RESULTS

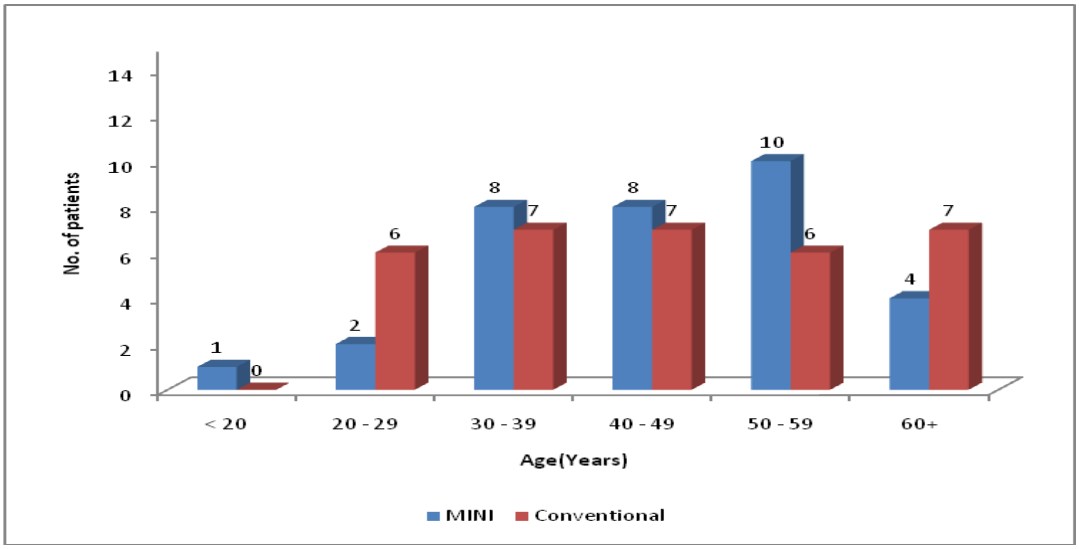
This study included 66 cases that were studied prospectively over a period of 23 months, from October 2016 to August 2018.

AGE DISTRIBUTION

In the present series the youngest patient was 18 yrs of age and the oldest was 65 yrs of age. Majority of the patients in the present series were in the age group of 31-40 yrs of age.

Table 7 : Distribution of patients according to Age

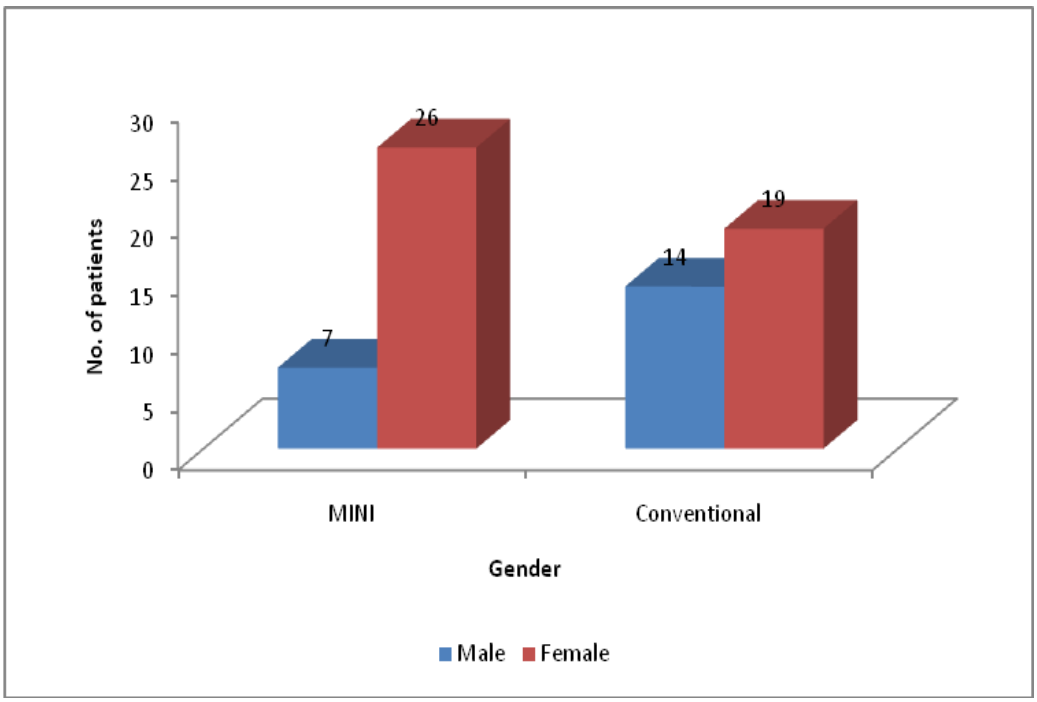
Age(Years)	MINI N(%)	Conventional N(%)
< 20	1(3)	0
20 - 29	2(6)	6(18.2)
30 - 39	8(24.2)	7(21.2)
40 - 49	8(24.2)	7(21.2)
50 - 59	10(30.3)	6(18.2)
60+	4(12.1)	7(21.2)
Total	33(100)	33(100)



- Among 33 of MLC patients below age 20 is one, & among 33 of CLC Patients, none.
- In the age group of 20-29 in MLC, there are 2 patients, in CLC there are 6 patients, age group of 30-39 MLC – 8 Patients, in CLC – 7 patients.
- In age group 40-49, MLC -8 patients, in CLC- 7 patients.
- 50-59 age group, in MLC -10 patients , in CLC – 6 patients.
- 60-69 age group, in MLC – 4patients , in CLC – 7 patients.

Table 8: Distribution of patients according to Gender

Gender	MINI N(%)	Conventional N(%)	Chi square test
Male	7(21.2)	14(42.4)	P=0.0643 NS
Female	26(78.8)	19(57.6)	
Total	33(100)	33(100)	



According to gender, among 33 patients of MLC – 7 males, 26 female patients. In 33 patients of CLC – 14 males , 19 female patients.

Table 9: Distribution of patients according to LFT

LFT		MINI N(%)	Conventional N(%)
Normal		33(100)	33(100)
Total		33(100)	33(100)

LFT is normal in all MLC and CLC patients.

Table 10 : Distribution of patients according to No. of calculi

No. of calculi	MINI N(%)	Conventional N(%)	Chi square test
MULTIPLE	9(27.3)	9(27.3)	P=01604 NS
SLUDGE	7(21.2)	6(18.2)	
SOLITARY	13(36.4)	9(27.3)	
SOLTARY ,SLUDGE	1(3.0)	0	
MULTIPLE, SLUDGE	1(3.0)	9(27.3)	
Polyp	2(3.)0	2(3)	
-	3(9.1)	1(3)	
Total			

		Frequency	Percent
Valid	-	2	6.1
	MULTIPLE	9	27.3
	MULTIPLE+SLUDGE	7	21.2
	SLUDGE	6	18.2
	SOLITARY	9	27.3
	Total	33	100.0

	Frequency	Percent
MULTIPLE, SL	1	3.0
-	3	9.1
MULTIPLE	9	27.3
SLUDGE	7	21.2
SOLITARY	12	36.4
SOLTARY ,SLUDGE	1	3.0
Total	33	100.0

Among 33 of MLC Patients

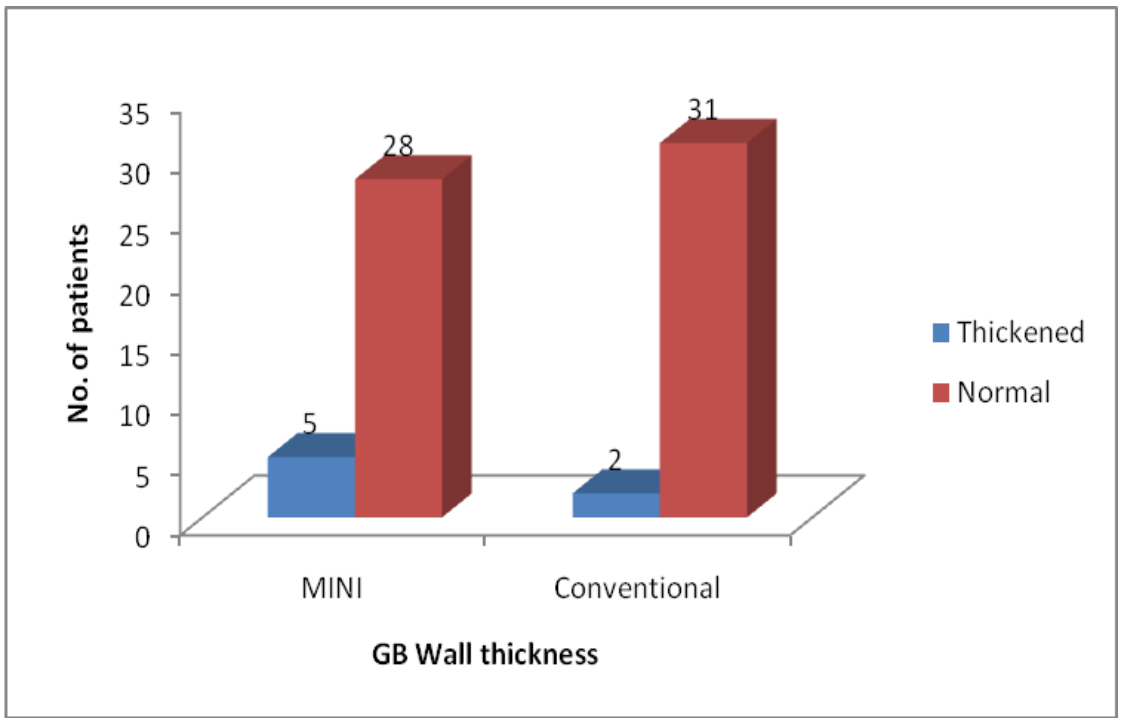
Multiple stones seen in – 9 patients, Sludge in 7 patients, Solitary stone in 13 patients, Solitary stone with Sludge in 1 , multiple stones with sludge in 1, Polyp in 2 patient.

Among 33 of CLC Patients

Multiple stones seen in – 9 patients, Sludge in 6 patients, Solitary stone in 9 patients, Solitary stone with Sludge in 0 , multiple stones with sludge in 9, Polyp in 2 patient.

Table 11 : Distribution of patients according to GB wall thickness

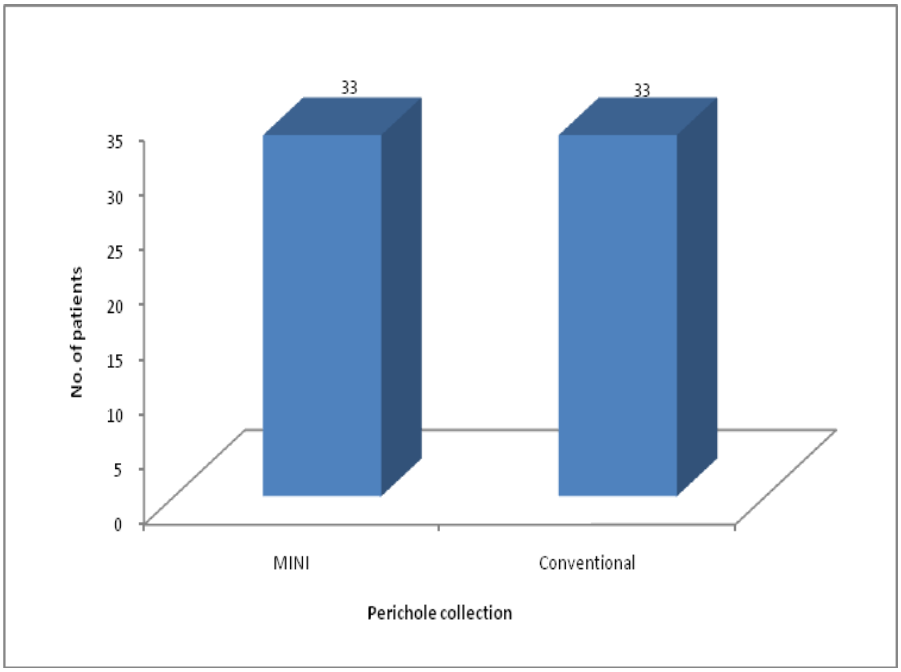
GB wall thickness	MINI N(%)	Conventional N(%)	Chi square test
Thickened	5(15.2)	2(6)	P=0.2304 NS
Normal	28(84.8)	31(94)	
Total	33(100)	33(100)	



- In MLC (33) Patients, GB wall thickening is seen in 5 patients, normal in 28 patients.
- In CLC (33) Patients, GB wall thickening is seen in 2 patients, normal in 31 patients.

Table 12 : Distribution of patients according to Perichole collection

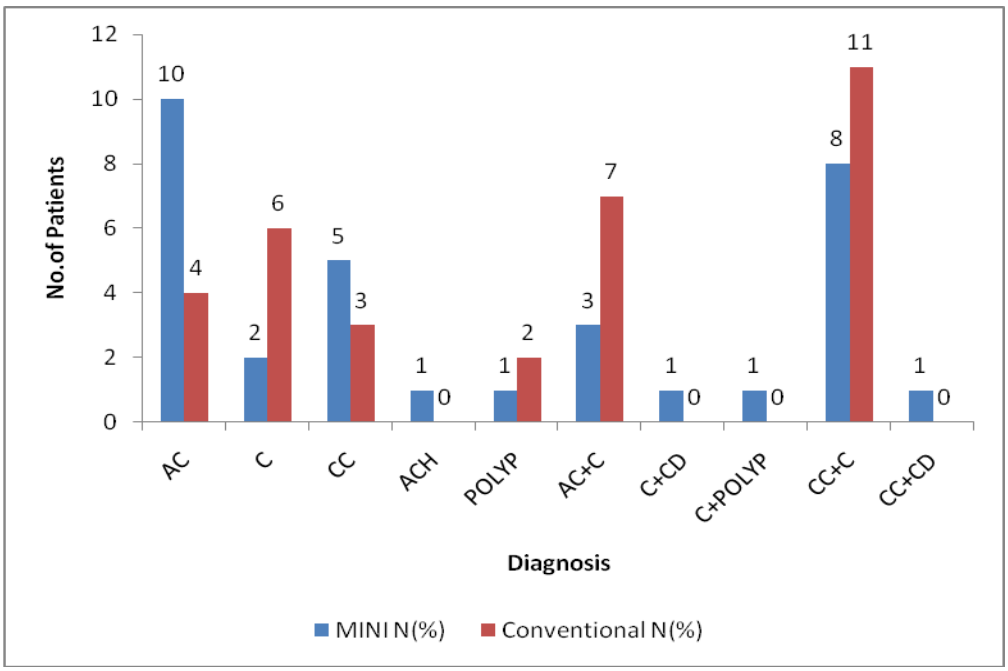
Perichole collection	MINI N(%)	Conventional N(%)
Absent	33(100)	33(100)
Total	33(100)	33(100)



No Pericholic collection is seen in Both MLC and CLC patients.

Table 13 : Distribution of patients according to Diagnosis

Diagnosis	MINI N(%)	Conventional N(%)	Chi square test
AC	10(30.3)	4(12.1)	P=0.2443 NS
C	2(6.1)	6(18.2)	
CC	5(15.2)	3(9.1)	
ACH	1(3)	0	
POLYP	1(3)	2(6.1)	
AC+C	3(9)	7(21.2)	
C+CD	1(3)	0	
C+POLYP	1(3)	0	
CC+C	8(24.2)	11(33.3)	
CC+CD	1(3)	0	
Total	33(100)	33(100)	

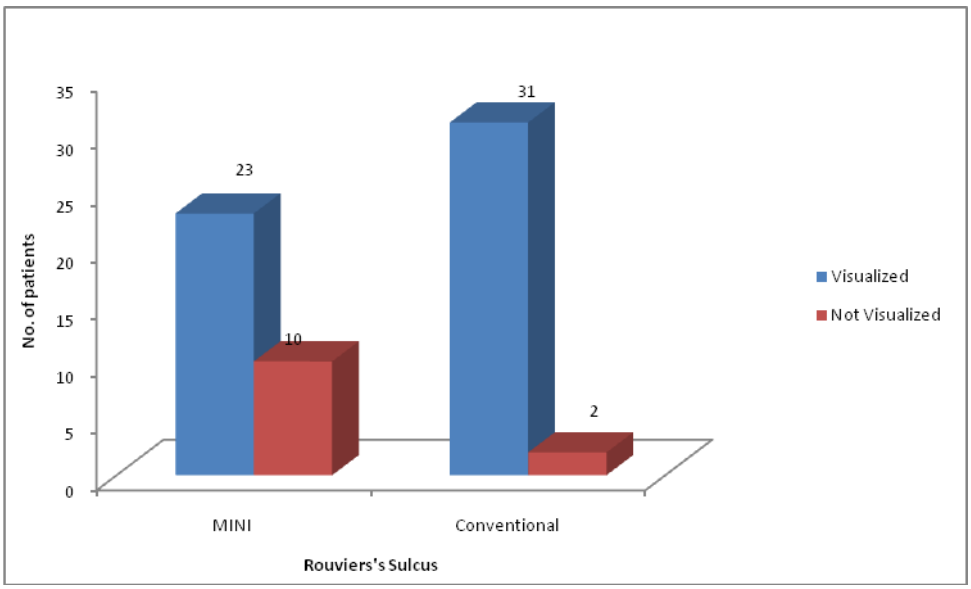


Among 33 of **MLC** Patients, 33 of **CLC** Patients.

Acute Cholecystitis	10	4
Chronic Cholecystitis	5	3
Cholelithiasis –	2	6
Acute Cholelithiasis	1	0
Polyp	2	2
(Acute Cholecystitis + Cholelithiasis)	3	7
(Cholelithiasis + Choledocholithiasis)	1	0
Cholelithiasis + Polyp	1	0
(Chronic Cholecystitis + Cholelithiasis)	8	11
(Chronic Cholecystitis + Choledocholithiasis)	1	0

Table 14 : Distribution of patients accordingI Intra-op Rouvier's sulcus

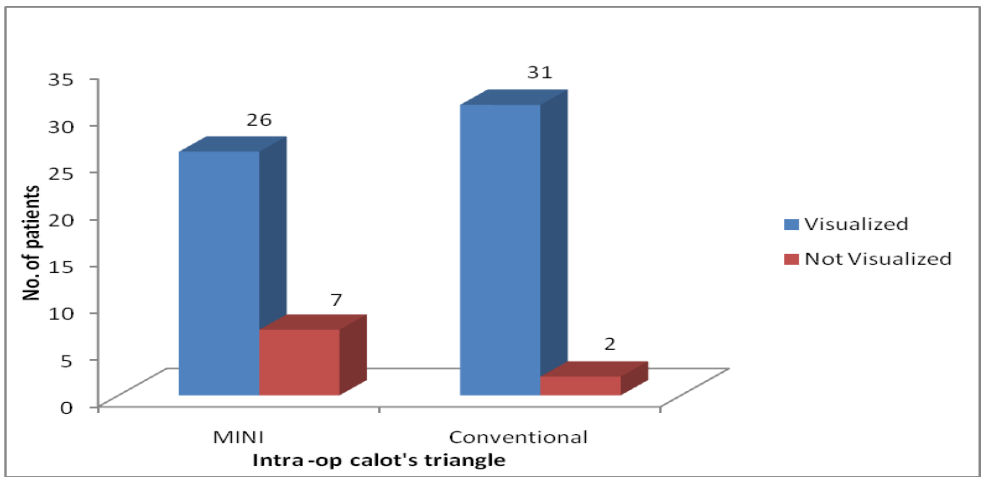
Intra-op Rouvier's sulcus	MINI N(%)	Conventional N(%)	Chi square test
Visualized	23(67.7)	31(91)	P=0.0107*
Not Visualized	10(30.3)	2(6)	
Total	33(100)	33(100)	



- Among 33 of MLC Patients Intra-op Rouvier's sulcus visualized in 23 and not visualized in 10 patients. In 33 of CLC Patients Intra-op Rouvier's sulcus visualized in 31 and not visualized in 2 patients.

Table 15 : Distribution of patients according Intra-op Calot's triangle

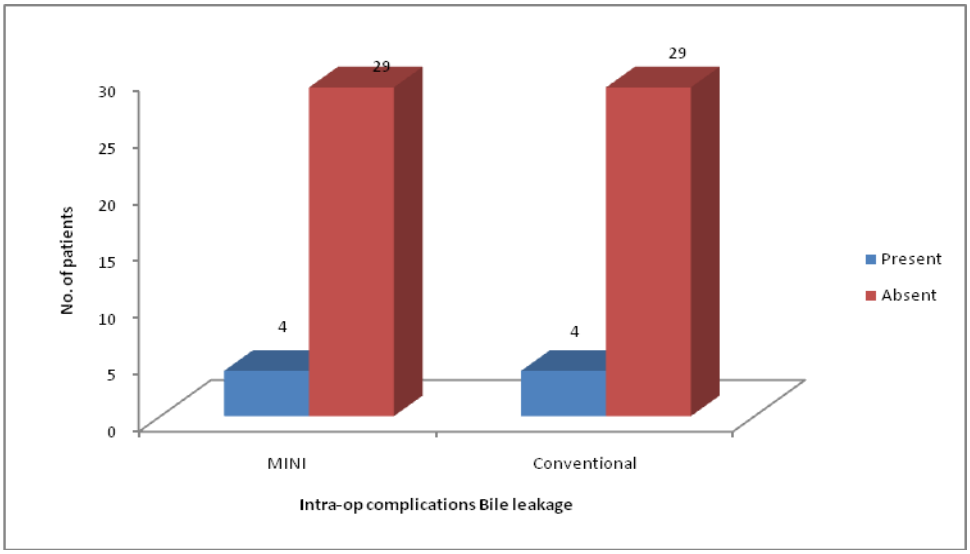
Intra-op Calot's triangle	MINI N(%)	Conventional N(%)	Chi square test
Visualized	26(78.8)	31(91)	P=0.0729 NS
Not Visualized	7(21.2)	2(6)	
Total	33(100)	33(100)	

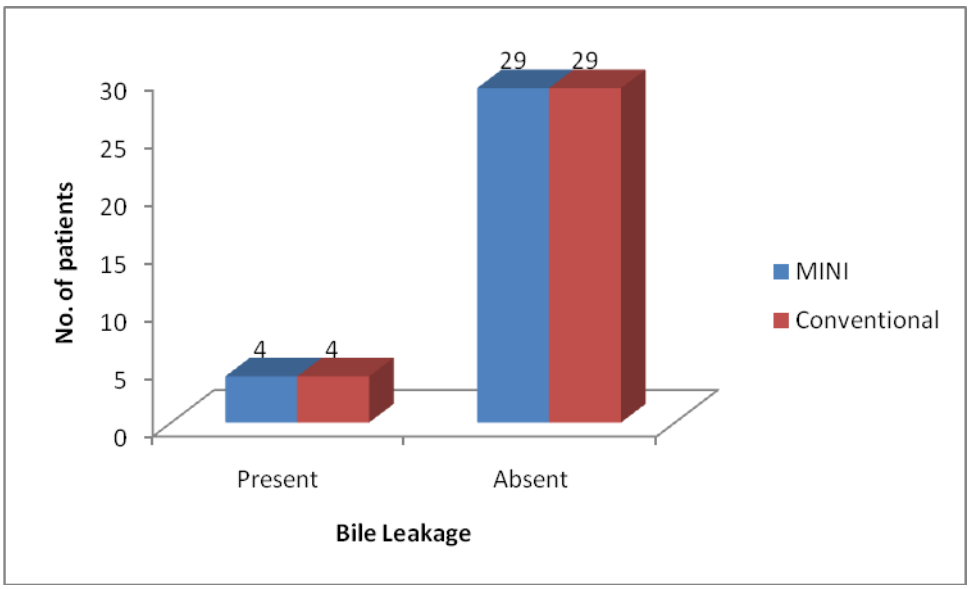


Among 33 of MLC patients intraop Calots triangle is visualize in 26 patients and not visulised in 7 patients, In 33 of CLC Patients Intra-op Calots triangle visualized in 31 and not visualized in 2 patients.

Table 16 : Distribution of patients according Intra-op complications Bile leakage

intra-op complications Bile leakage	MINI N(%)	Conventional N(%)	Chi square test
Present	4(12)	4(12)	P=1.00 NS
Absent	29(88)	29(88)	
Total	33(100)	33(100)	

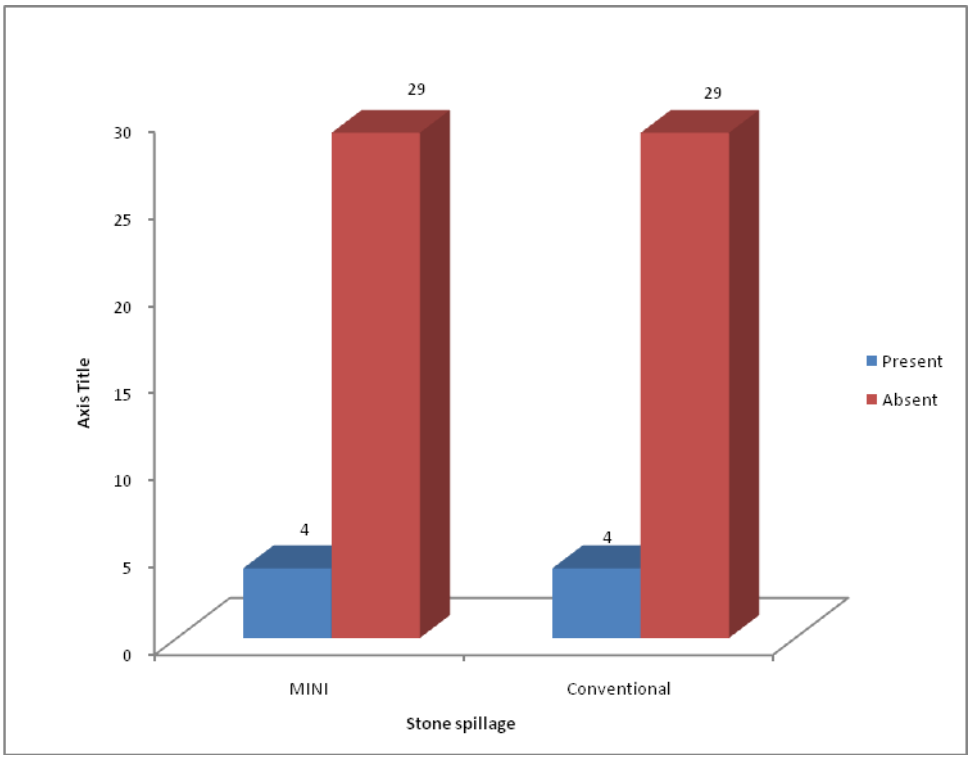




I**ntra-op Bile leakage** is seen in 4 patients, absent in 29 in both MLC and CLC Groups.

Table 17 : Distribution of patients according Intra-op complications Stone spillage

Intra-op complications Stone spillage	MINI N(%)	Conventional N(%)	Chi square test
Present	4(12)	4(12)	NS
Absent	29(88)	29(88)	
Total	33(100)	33(100)	



I**ntra-op Stone spillage** is seen in 4 patients, absent in 29 in both MLC and CLC Groups.

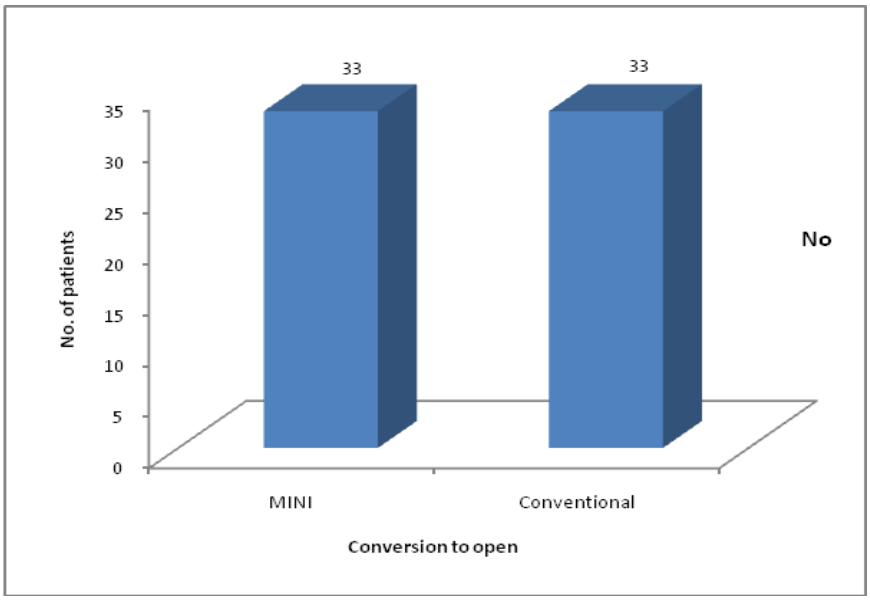
Table 18 : Distribution of patients according intra-op complications CBD INJURY

Intra-op complications CBD INJURY	MINI N(%)	Conventional N(%)	
Yes	0	0	NS
No	33(100)	33(100)	
Total	33(100)	33(100)	

No intra-op CBD Injury is noted in both MLC and CLC Groups.

Table 19 : Distribution of patients according Conversion to open

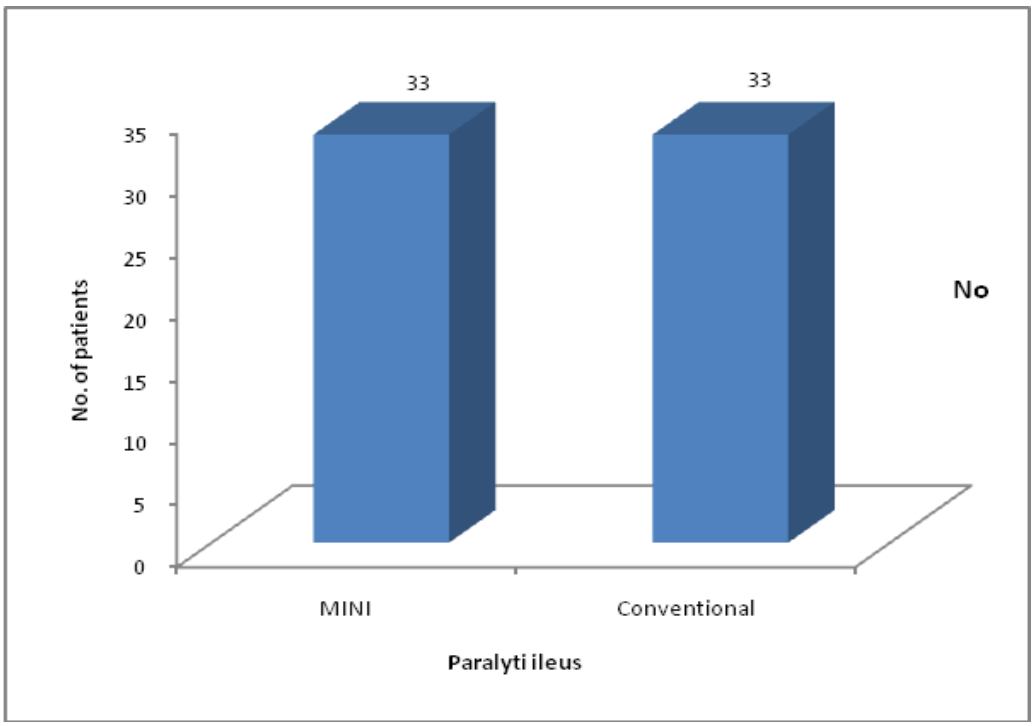
Conversion to open	MINI N(%)	Conventional N(%)
Yes	0	0
No	33(100)	33(100)
Total	33(100)	33(100)



No conversion to open is observed in both MLC and CLC Patients.

Table 20 :Distribution of patients according Paralytic ileus

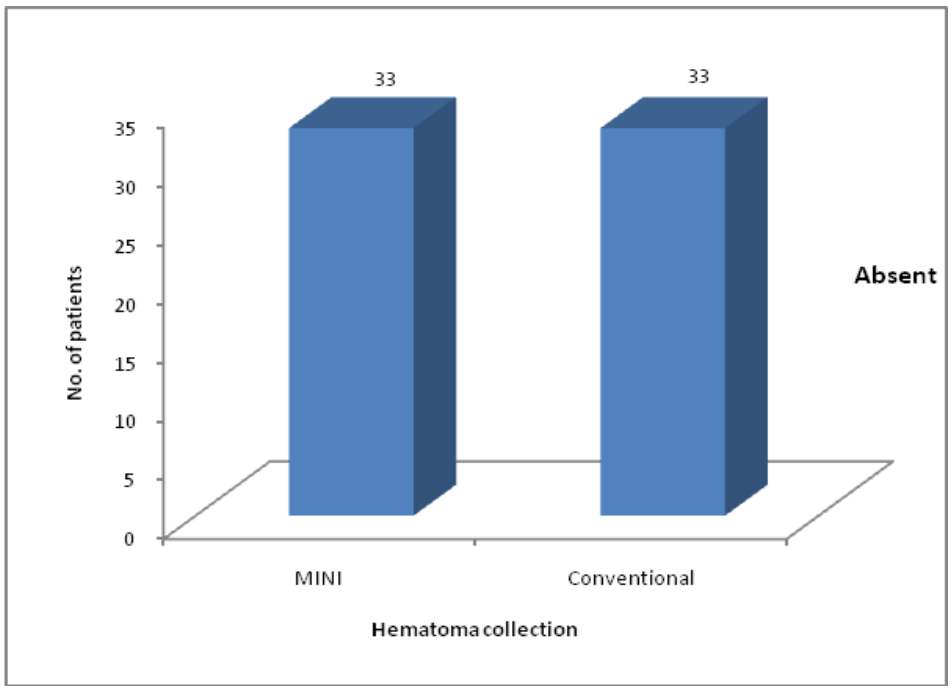
Paralytic ileus	MINI N(%)	Conventional N(%)
Yes	0	0
No	33(100)	33(100)
Total	33(100)	33(100)



NO Paralytic Ileus is seen in both MLC and CLC Patients.

Table 21: Distribution of patients according Hematoma collection

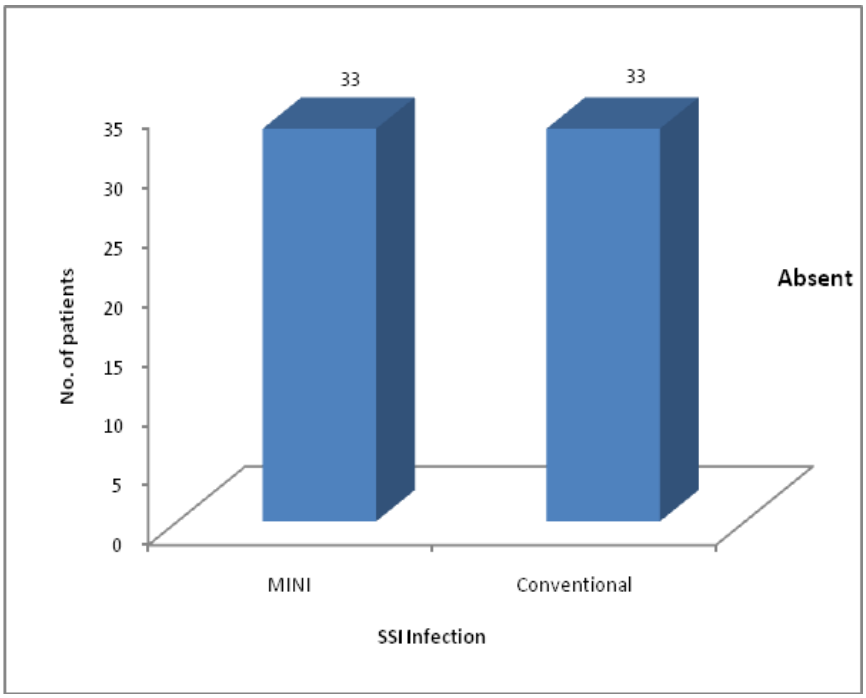
Hematoma collection	MINI N(%)	Conventional N(%)
Present	0	0
Absent	33(100)	33(100)
Total	33(100)	33(100)



NO **Hematoma collection** is seen in both MLC and CLC Patients.

Table 22 : Distribution of patients according SSI Infection

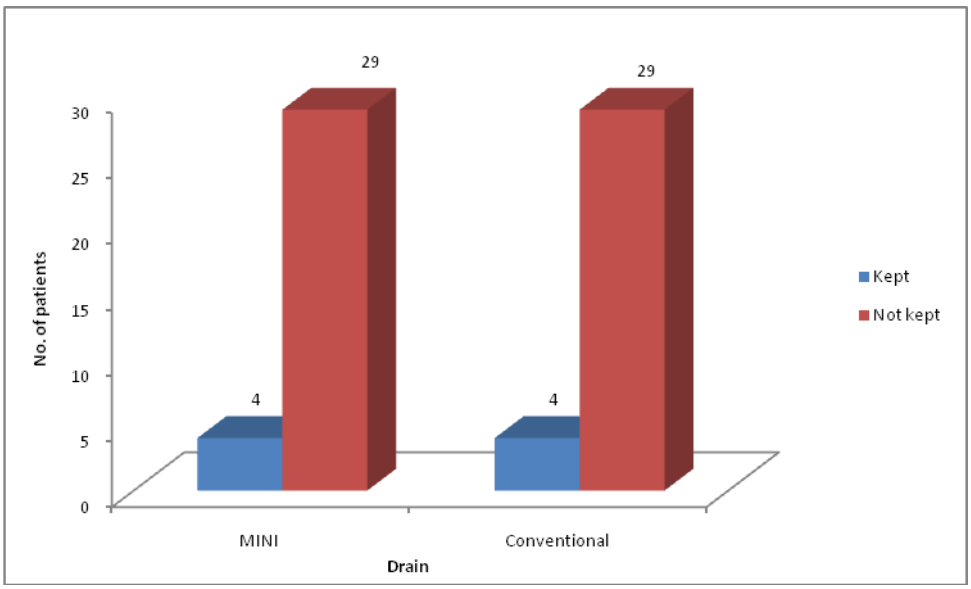
SSI Infection	MINI N(%)	Conventional N(%)
Present	0	0
Absent	33(100)	33(100)
Total	33(100)	33(100)



NO **SSI Infection** is seen in both MLC and CLC Patients.

Table 23 : Distribution of patients according Drain

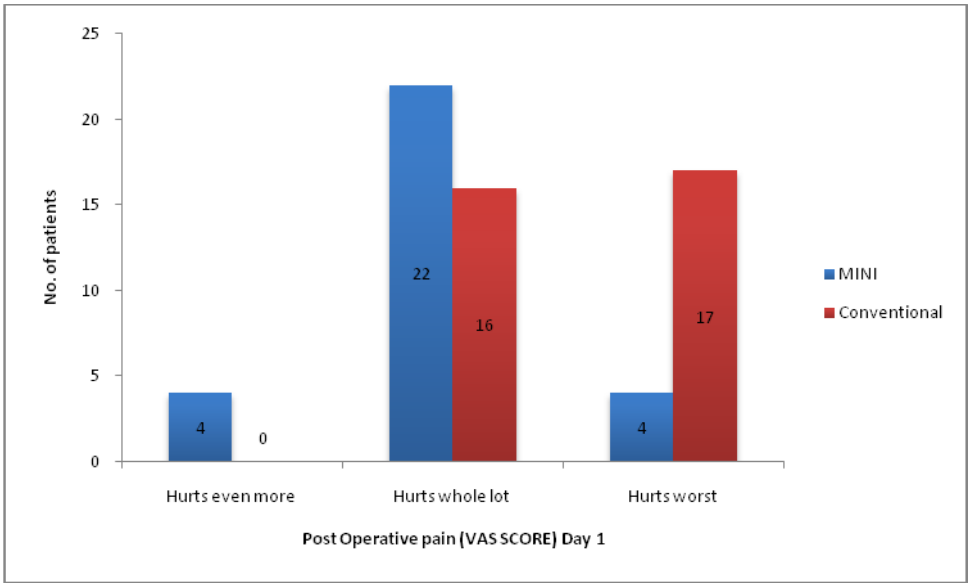
Drain	MINI N(%)	Conventional N(%)	Chi square test
Kept	4(12)	4(12)	P=1.000 NS
Not kept	29(88)	29(88)	
Total	33(100)	33(100)	



Drain is kept in 4 patients, not kept in 29 in both MLC and CLC Groups.

Table 24 : Distribution of patients according Post operative pain (VAS SCORE) Day 1

Post operative pain (VAS SCORE)	MINI N(%)	Conventional N(%)	Chi square test
3	4(12)	0	P=0.0016*
4	22(66.7)	16(48.5)	
5	4(12)	17(51.5)	
Total	33(100)	33(100)	

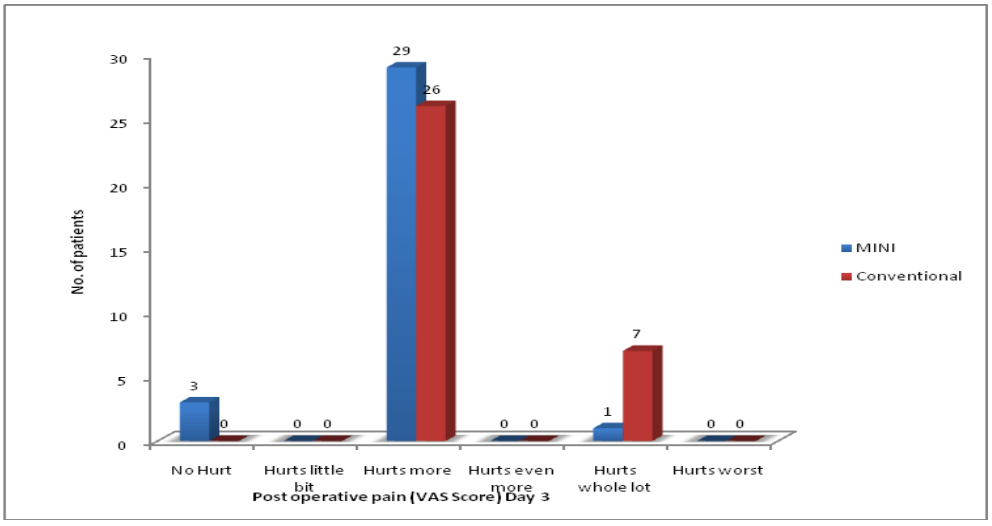


Post op pain (VAS Score) on day 1

- In among 4 MLC Patients score is 3 and no pain in patients of CLC Group was noted,
- In among 22 MLC Patients score is 4 and 16 CLC Patients pain score is 4,
- In among 4 MLC Patients score is 5 and 17 CLC Patients pain score is 5,

Table 25 : Distribution of patients according Post operative pain (VAS SCORE) Day 3

Post operative pain (VAS SCORE)	MINI N(%)	Conventional N(%)	Pooled chi square test
0	3(9)	0	P=0.001*
1	0	0	
2	29(88)	26(78.8)	
3	0	0	
4	1(3)	7(21.2)	
5	0	0	
Total	33(100)	33(100)	



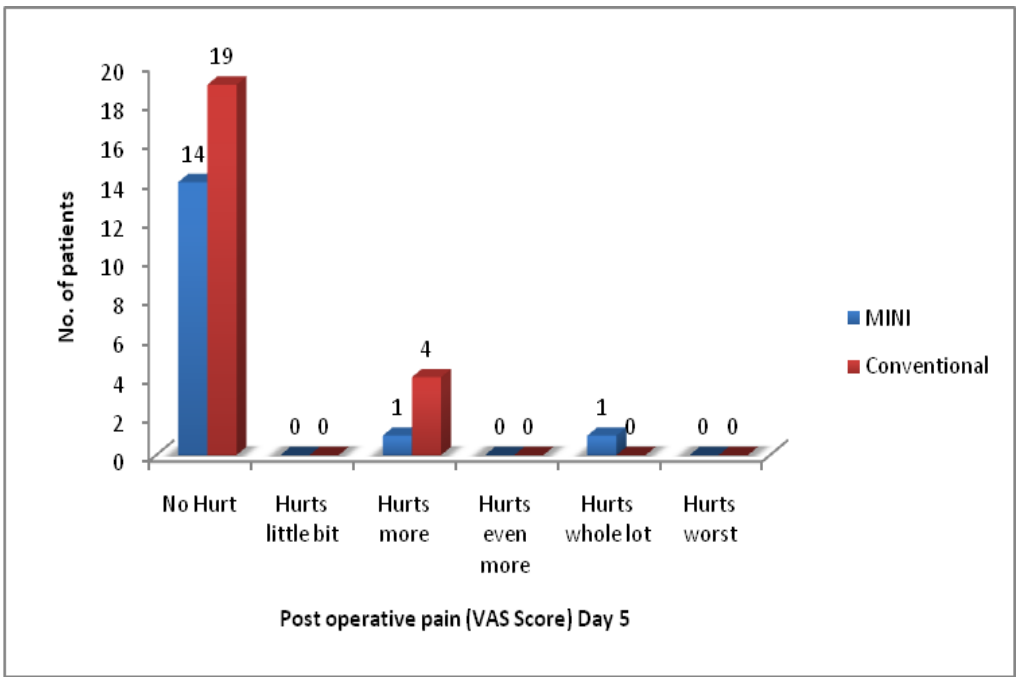
Post op pain (VAS Score) on day 3

- In among 3 MLC Patients score is 0 and no pain in patients of CLC Group was noted,
- No patients in both MLC , CLC Groups have VAS score 1
- In among 29 MLC Patients score is 2 and 26 CLC Patients pain score is 2,
- No patients in both MLC , CLC Groups have VAS score 3
- In among 1 MLC Patient score is 4 and 1 CLC Patients pain score is 4,
- No patients in both MLC , CLC Groups have VAS score 5, on Post-op Day 3.

Table 26 : Distribution of patients according Post operative pain (VAS SCORE)

Day 5:

Post operative pain (VAS SCORE)	MINI N(%)	Conventional N(%)	Pooled chi square test
0	14(42.4)	19(57.6)	P=0.1202 NS
1	0	0	
2	1(3)	4(12.1)	
3	0	0	
4	1(3)	0	
5	0	0	
Discharged	17(54.5)	10(30.3)	
Total	33(100)	33(100)	



Post op pain (VAS Score) on day 5

- In among 14 MLC Patients have VAS score is 0 and 19 CLC Patients pain score is 0,
- No patients in both MLC , CLC Groups have VAS score 1, on Post-op Day 5.
- 1 MLC Patient VAS score is 2 and 4 CLC Patients pain score is 2.
- No patients in both MLC , CLC Groups have VAS score 3 on Post-op Day 5
- 1 MLC Patient VAS score is 4 and none in CLC Group have VAS Score 4.
- No patients in both MLC , CLC Groups have VAS score 5, on Post-op Day 5.

Descriptive Statistics (MINI)

	N	Minimum	Maximum	Mean	Std. Deviation
Age	33	18	65	44.06	12.872
operative time (mins)	33	30	75	54.00	13.604
Post-op Pain (VAS) DAY1	33	3	5	4.09	.579
Post-op Pain (VAS) DAY3	33	0	4	1.88	.696
Hospital Stay	33	1.5	4.0	2.712	.6850
Post-op Pain (VAS) DAY5	15	0	2	0.13	0.516

In MLC Patients minimum operative time is 30 minutes and maximum is 75 minutes. Hospital stay in MLC Patients, minimum is 1.5 days, and maximum is 5 days.

Descriptive Statistics(Conventional)

	N	Minimum	Maximum	Mean	Std. Deviation
Age	33	22	65	43.67	13.249
operative time (mins)	33	30	70	46.97	10.312
Post-op Pain (VAS) DAY1	33	4	5	4.52	.508
Post-op Pain (VAS) DAY3	33	2	4	2.42	.830
Post-op Pain (VAS) DAY5	23	0	2	.35	.775
Hospital Stay	33	2.0	5.0	3.470	.8286

Minimum Operative time in CLC Patients is 30 minutes, and maximum is 70 minutes. Hospital stay in CLC Patients, minimum is 2 days, and maximum is 5 days.

Table 27 : Comparison of variables between MINI and Conventional groups

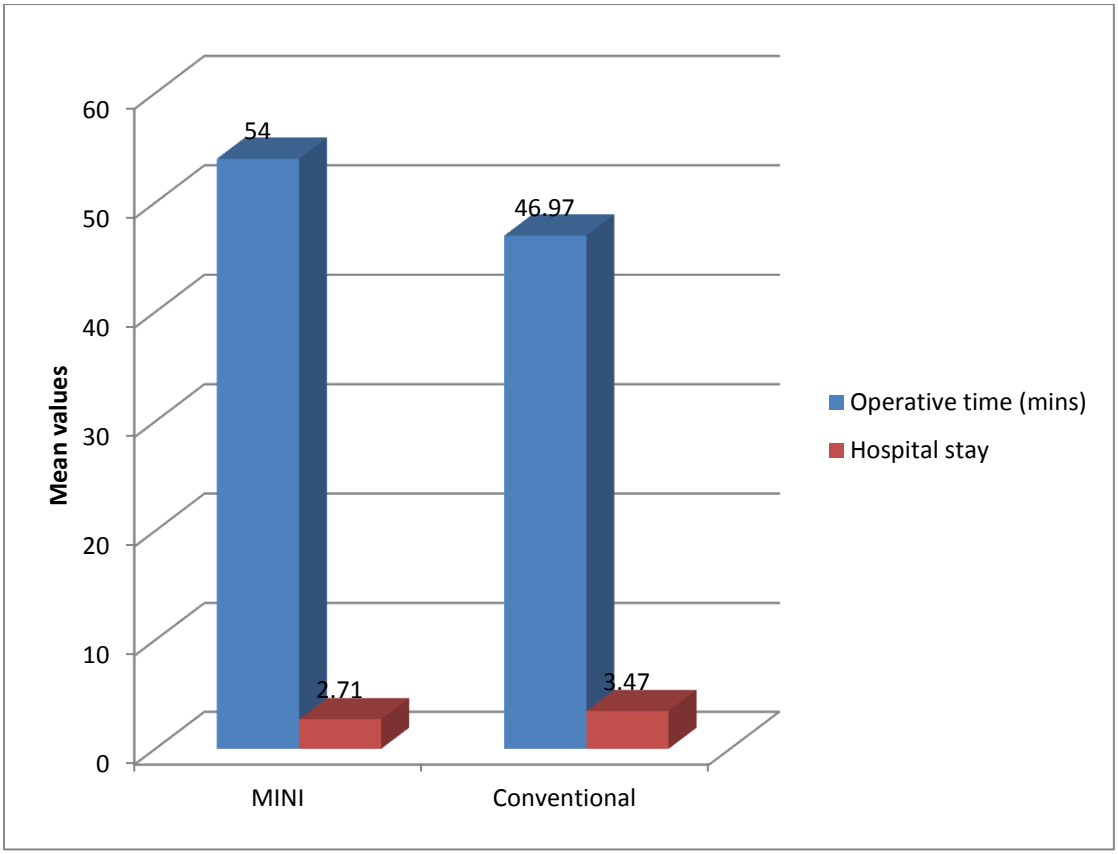
Variables	MINI Mean±SD	Conventional Mean±SD	Student t test
Age	44.06±12.9	43.67±13.25	P=0.901 NS
Operative time (mins)	54.00±13.60	46.97±10.31	P=0.021*
Hospital stay	2.71±0.69	3.47±0.83	P=0.0001*

Table 28 : Comparison of post operative pain (VAS Score) between MINI and Conventional groups

Post Operative time (VAS score)	MINI Mean(Median)±SD	Conventional Mean(Median)±SD	Mann Whitney U test
Day 1	4.09(4.0)±0.58	4.52(5)±0.51	P=0.004*
Day3	1.88(2.0)±0.69	2.42(2)±0.83	P=0.006*
Day 5	0.133(0)±0.52	0.35(0)±0.78	P=0.595 NS

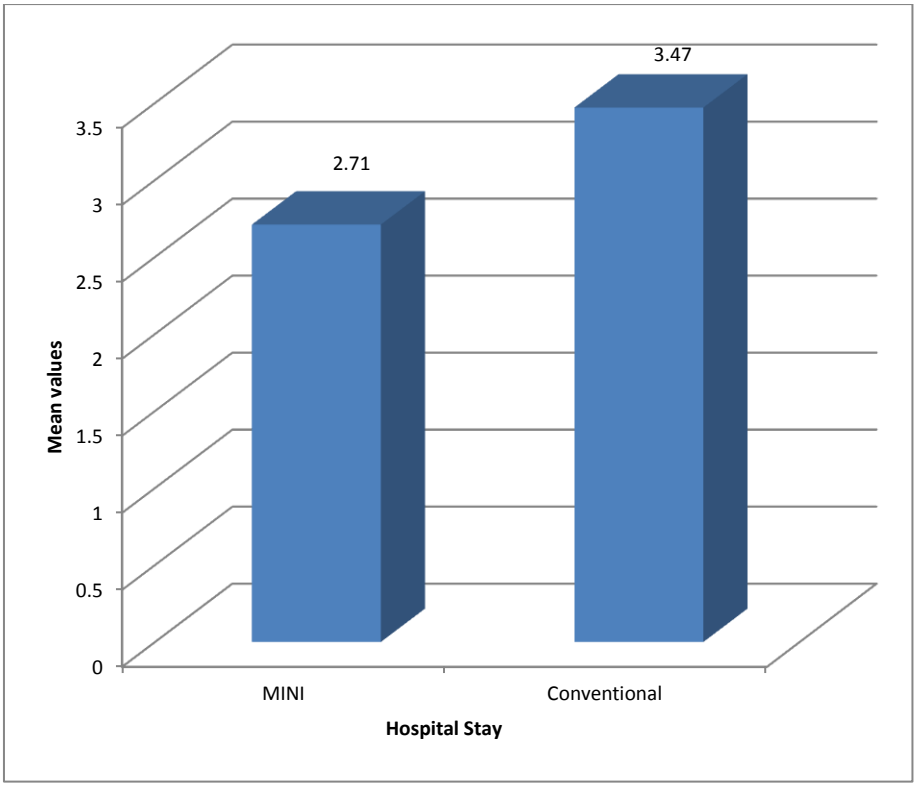
Table 29 : Comparison of Hospital stay between MINI and Conventional groups

Variables	MINI Mean±SD	Conventional Mean±SD	Student t test
Age	44.06±12.9	43.67±13.25	P=0.901 NS
Operative time (mins)	54.00±13.60	46.97±10.31	P=0.021*
Hospital stay	2.71±0.69	3.47±0.83	P=0.0001*



1. Mean Operative time in MLC(33) Patients is 54 minutes and the duration of hospital stay is 2.71 days.
2. Mean Operative time in CLC(33) Patients is 47 minutes and the duration of hospital stay is 3.47 days.

Variables	MINI Mean±SD	Conventional Mean±SD	Student t test
Hospital stay	2.71±0.69	3.47±0.83	P=0.0001*



HISTOPATHOLOGICAL EXAMINATION

24 (13+11) cases were detected as Acute Cholecystitis, 26 (13+13)cases were reported as chronic cholecystitis. 35 cases (11+24) were detected as cholelithiasis, and 4 Gall bladder polyp.

No case of malignancy of the GB was detected.

HISTOPATHOLOGIC EXAMINATION	No. OF CASES (M-LC + C-LC)
Chronic cholecystitis	26(13+13)
Acute cholecystitis	24(13+11)
Cholelithiasis	35(11+24)
Gall bladder polyp	4(2+2)
Malignancy	0

FOLLOW UP

All patients were followed up for a period of 1 month and no significant complication was noted.

DISCUSSION

AGE DISTRIBUTION :

- In my study majority of the patients in the present series were in the age group of 31-40 yrs of age, and 80% 0f the patients came under the age group from 18-65 years.

- According to my study age is not a single significant predictor because majority of the patients had easy cholecystectomy irrespective of age.

SEX DISTRIBUTION :

In the present series, out of 66 patients 45 were females and 21 were male patients. The male : female ratio is 1:2.14 (**P=0.0643 NS)**

- According to my study, Obese patients had a little difficult Mini Lap cholecystectomy because of instruments which are slender and of lower caliber ,chances of bending of instruments is more, and also difficulty in reaching gall bladder because of excessive omental fat.

  1 Patient with Choledocholithiasis having deranged LFT, that patient subjected to ERCP and after 6 weeks interval cholecystectomy done which was easy.

- It stated that Gall stone disease with deranged LFT need further investigation and delayed cholecystectomy also influences the per operative outcome.

ULTRASONOGRAPHY :

Ultrasound was done as a routine investigation in all the patients.

The sonologic criteria used to diagnose gall stones were acoustic shadowing of the opacities in the gall bladder and change in the position of the opacity with the change in patient position.

a) 48 patients had stones in gallbladder, 5 patients had GB wall thickening measuring 6-8 mm.

b) 26 patients had multiple calculi, 22 had solitary calculus,

c) 4 patients had gall bladder Polyp, 22 patients had Sludge.

d) 24patients has Acute Cholecystitis, 26 patients has Chronic Cholecystitis

e) No pericholecystic collection is noted.

- LFT is normal in all MLC and CLC patients except in 1 patient MLC group who had Choledocholithiasis.

Among 33 of MLC Patients

- Multiple stones seen in – 9 patients, Sludge in 7 patients, Solitary stone in 13 patients, Solitary stone with Sludge in 1 , multiple stones with sludge in 1, Polyp in 2 patient.

Among 33 of CLC Patients

- Multiple stones seen in – 9 patients, Sludge in 6 patients, Solitary stone in 9 patients, Solitary stone with Sludge in 0 , multiple stones with sludge in 9, Polyp in 2 patient. **(P=01604 NS).**

- In MLC (33) Patients, GB wall thickening is seen in 5 patients, normal in 28 patients.
- In CLC (33) Patients, GB wall thickening is seen in 2 patients, normal in 31 patients. (**P=0.2304 NS**)

No Pericholic collection is seen in Both MLC and CLC patients.

Among 33 of **MLC** Patients, 33 of **CLC** Patients.

Acute Cholecystitis	10	4
(Chronic Cholecystitis + Cholelithiasis)	8	11

In my study, Chronic cholecystitis with cholelithiasis are more i.e., 19 patients. Next to CC+C, Acute cholecystitis are more i.e., 14 patients. **(P=0.2443 NS)**

- Among 33 of MLC Patients Intra-op Rouvier's sulcus visualized in 23 and not visualized in 10 patients. In 33 of CLC Patients Intra-op Rouvier's sulcus visualized in 31 and not visualized in 2 patients. (**P=0.0107*)**

- Among 33 of MLC patients intraop Calots triangle is visualize in 26 patients and not visulised in 7 patients, In 33 of CLC Patients Intra-op Calots triangle visualized in 31 and not visualized in 2 patients. (**P=0.0729 NS)**

- Intra-op Bile leakage is seen in **4** patients, absent in 29 in both MLC and CLC Groups**. (P=1.00 NS)**
- Intra-op Stone spillage is seen in **8** patients**.**
- No intra-op CBD Injury is noted in both MLC and CLC Groups.
- No conversion to open is observed in both MLC and CLC Patients.
- NO Paralytic Ileus is seen in both MLC and CLC Patients.
- NO **Hematoma collection** is seen in both MLC and CLC Patients.
- NO **SSI Infection** is seen in both MLC and CLC Patients.
- Drain was kept in 8 patients, in both MLC and CLC Groups.

❖ Distribution of patients according **Post operative pain :**

- **Post op pain (VAS Score) on day 1**
  - In among 4 MLC Patients score is 3 and no pain in patients of CLC Group was noted,
  - In among 22 MLC Patients score is 4 and 16 CLC Patients pain score is 4,
  - In among 4 MLC Patients score is 5 and 17 CLC Patients pain score is 5,
  - **(P=0.0016*)** P value is less than 0.05, and is found to be significant.
- **Post op pain (VAS Score) on day 3**

In among 3 MLC Patients score is 0 and no pain in patients of CLC Group was noted,

- No patients in both MLC , CLC Groups have VAS score 1
- In among 29 MLC Patients score is 2 and 26 CLC Patients pain score is 2,
- No patients in both MLC , CLC Groups have VAS score 3
- In 1 MLC Patient score is 4 and 1 CLC Patients pain score is 4,

- No patients in both MLC , CLC Groups have VAS score 5, on Post-op Day 3.
- **(P=0.001*)** P value is less than 0.05, and is found to be significant

➢ **Post op pain (VAS Score) on day 5**

- In among 14 MLC Patients have VAS score is 0 and 19 CLC Patients pain score is 0,
- No patients in both MLC , CLC Groups have VAS score 1, on Post-op Day 5.
- 1 MLC Patient VAS score is 2 and 4 CLC Patients pain score is 2.
- No patients in both MLC , CLC Groups have VAS score 3 on Post-op Day

  5.
- 1 MLC Patient VAS score is 4 and none in CLC Group have VAS Score 4.
- No patients in both MLC , CLC Groups have VAS score 5, on Post-op Day 5.
- **(P=0.1202 NS)** P value is more than 0.05, and is found to be Not significant.

Post-op Pain Score on day1 and day3 is less in MLC Patients, when compared to CLC Patients.

- In MLC Patients minimum operative time is 30 minutes and maximum is 75 minutes.
- Minimum Operative time in CLC Patients is 30 minutes, and maximum is 70 minutes.
- Mean Operative time in MLC(33) Patients is **54 minutes**.
- Mean Operative time in CLC(33) Patients is **47 minutes** .
- Mean Operative time in MLC(33) Patients is **more** (**54minutes**) as compared to Operative time (**47 minutes**) in CLC Patients (33). (**P=0.021***) P value is found to be significant.
- Hospital stay in MLC Patients, minimum is 1.5 days, and maximum is 5 days.
- Hospital stay in CLC Patients, minimum is 2 days, and maximum is 5 days.
- MLC(33) Patients, Mean duration of hospital stay is **2.71 days**.
- CLC(33) Patients, Mean duration of hospital stay is **3.47 days**.

Hospital Stay is less in MLC Patients i.e., 2.71 days , when compared to 3.47 days, in CLC group.

(P=0.0001*) P value is found to be significant.

In case of Cosmesis, No much difference is Noted.

HISTOPATHOLOGICAL EXAMINATION

24 (13+11) cases were detected as Acute Cholecystitis, 26 (13+13)cases were reported as chronic cholecystitis. 35 cases (11+24) were detected as cholelithiasis, and 4 Gall bladder polyp.

No case of malignancy of the GB was detected.

HISTOPATHOLOGIC EXAMINATION	No. OF CASES (M-LC + C-LC)
Chronic cholecystitis	26(13+13)
Acute cholecystitis	24(13+11)
Cholelithiasis	35(11+24)
Gall bladder polyp	4(2+2)
Malignancy	0

Follow Up :

All patients were followed up for a period of 1 month and no significant complication was noted.

POST-OPERATIVE TREATMENT

- Nasogastric aspiration till the patient recovered from appearance of bowel sounds and passage of flatus in few cases.
- I-V fluids continued till oral liquid diet was started, ie following removal of Ryle's tube in selective cases.
- Broad spectrum antibiotic for 5 days.
- Analgesics as and when required.
- Drainage tube was removed between 1^{st} and 5^{th} post OP day in selective cases.

CONCLUSIONS

According to my study

1) Pain was the predominant symptom seen in all (100%) the patients.

2) Mini Laparoscopic cholecystectomy can be safely performed using 10-mm umbilical, 5-mm epigastric, 3-mm subcostal ports. [59]

3) The use of mini- laparoscopic techniques resulted in decreased early post-operative pain, & decreased length of hospital stay, variable operative time. [59]

4) Although improved instrument durability and better optics are needed for widespread use of miniport techniques. [59]

5) The conversion rate from Laparoscopic Cholecystectomy to open cholecystectomy was zero.

6) There was no incidence of port site infections.

SUMMARY

- Cholelithiasis is the most common biliary pathology. Gall stone are present in10 to 15% of the general population and asymptomatic in the majority of them, of about >80%.[57]

- Approximately 1-2% of asymptomatic patients will develop symptoms requiring cholecystectomy every year, making it one of the most common operations performed.[57]

  In 1992, The National Institute of Health (NIH) consensus development Conference stated that laparoscopic cholecystectomy "Provides a safe and effective treatment for most patients with symptomatic gallstones". [58]

- Laparoscopic cholecystectomy (LC) has become the gold standard operation for patients with gallbladder disease since the first hepatic case performed successfully by Mouret in 1987.[60]

- Mini-laparoscopy was pioneered more than 20 years ago.

- Newer generation mini instruments have recently become available with improved effector tips, a choice of shaft diameters and lengths, better shaft insulation and electrosurgery capability, improved shaft strength and rotation, more ergonomic handles, low-friction trocar options, and improved instrument durability. [11]
- Mini-Lap cholecystectomy is a refinement of LC in which instruments and ports of size ≤ 3mm in diameter are used compared with the standard 5-mm and10-mm sizes used in conventional laparoscopic cholecystectomy. [59]

- The perceived advantages of the MiniLap technique are wounds that heal leaving imperceptible scars, reduced postoperative analgesic use, potential reduced risk of trocar hernias, lower incidence of wound complications, a smaller sheath that makes the introduction smooth and effortless decreasing the risk of intra-abdominal lesions, with a high satisfaction rate and possibly a faster recovery. [61]

- Whether the use of mini instruments, particularly newer generation instruments, offers advantages for laparoscopic cholecystectomy is the subject of this review. [11]

- The use of smaller instruments during laparoscopic cholecystectomy (LC) has been proposed to reduce postoperative pain and improve cosmesis. [59]

- However, despite several recent trials, the effects of the use of miniaturized instruments for (LC) are not well established. [59]

- We hypothesized that LC using miniports (M-LC) is safe and produces less incisional pain and better cosmetic results than LC performed conventionally (C-LC).[59]

- Mini Laparoscopic cholecystectomy can be safely performed using 10-mm umbilical, 5-mm epigastric, 3-mm subcostal .The use of mini- laparoscopic techniques resulted in decreased early post-operative pain, and decreased length of hospital stay, variable operative time.[62]
- Although improved instrument durability and better optics_are needed for widespread use of miniport techniques.[59]

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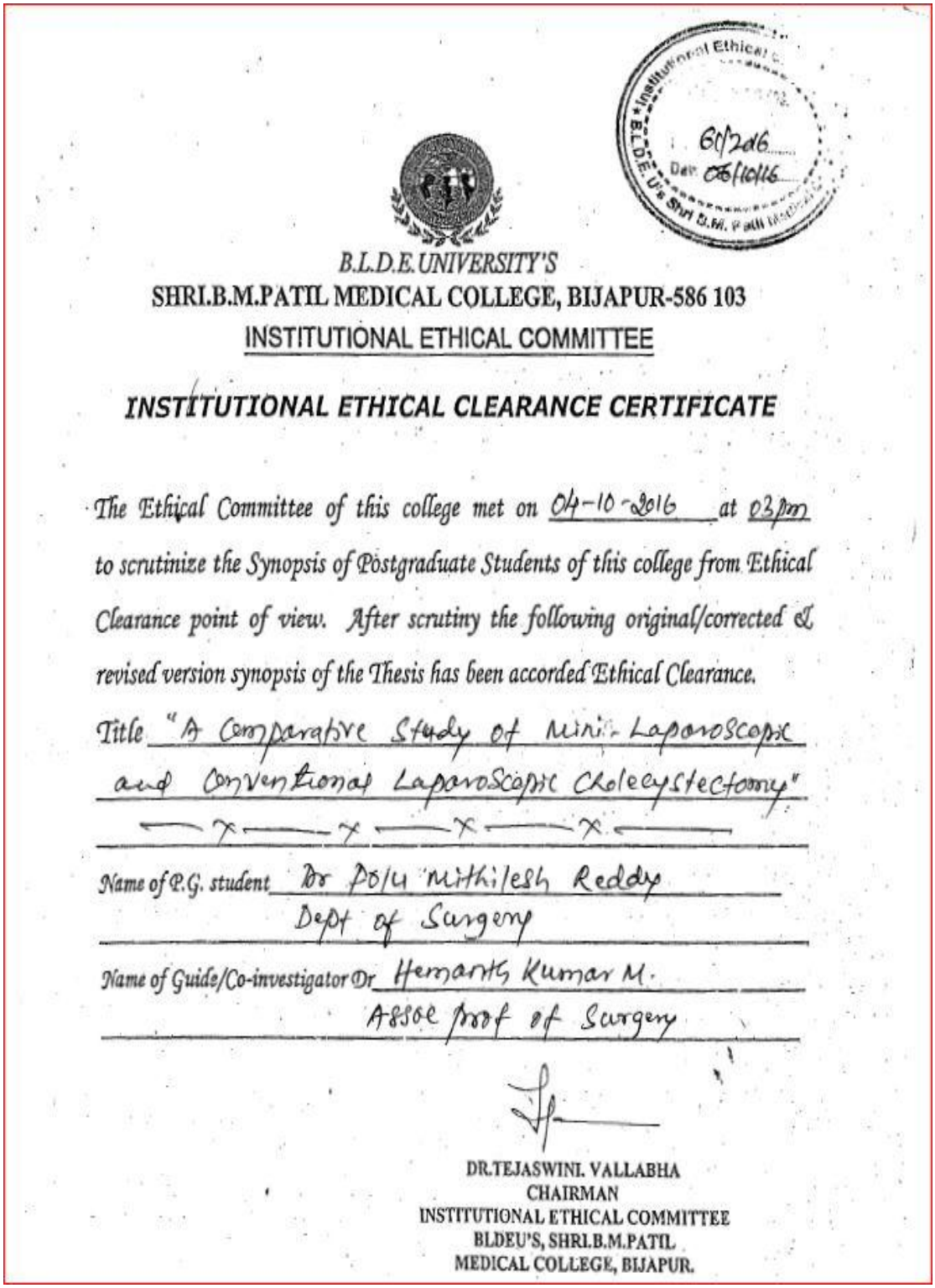
ANNEXURES

ETHICAL CLARENCE CERTIFICATE



B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 04-10-2016 at 03pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title "A Comparative Study of Mini - Laparoscopic and Conventional Laparoscopic Cholecystectomy"

Name of P.G. student Dr Polu Mithilesh Reddy
Dept of Surgery

Name of Guide/Co-investigator Dr Hemanth Kumar M.
Assoc Prof of Surgery

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

SAMPLE INFORMED CONSENT FORM

TITLE OF THE PROJECT	:	A PROSPECTIVE COMPARATIVE STUDY OF MINI LAPAROSCOPIC AND CONVENTIONAL LAPAROSCOPIC CHOLECYSTECTOMY.
PG GUIDE	:	**DR. HEMANTH KUMAR M.** **M.S., DMAS** M.S. General Surgery ASSOCIATE PROFESSOR Department of Surgery
PRINCIPAL INVESTIGATOR	:	**Dr. POLU MITHILESH REDDY**

PURPOSE OF RESEARCH:

I have been informed that this study will help in Comparing Mini Laparoscopic And Conventional Laparoscopic Cholecystectomy. I have also been given a free choice of participation in this study. This study will help in proper management of patients having Intestinal anastomotic leakages.

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomforts during the examination or during my treatment. This is mainly the result of my condition and the procedures of this study are not expected to exaggerate these feelings which are associated with the usual course of treatment.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigations research file.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study to Dr. Polu Mithilesh Reddy in the Department of General Surgery who will be available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that Dr. Polu Mithilesh Reddy may terminate my participation in the study after he has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me. But, no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks to the best of my ability in pts own language.

Date Dr Hemanth Kumar M (Guide) Dr Polu Mithilesh Reddy (Investigator)

Witness signature :

1)

DATE

2)

DATE

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. Polu Mithilesh Reddy has explained to me the purpose of research, the study procedure, that I will undergo and the possible discomforts as well as benefits that I may experience in my own language. I have been explained all the above in detail in my own language and I understand the same. Therefore, I agree to give consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to signature) Date

PROFORMA

SL NO

NAME

AGE IP NO

SEX UNIT

RELIGION DOA

OCCUPATION DOO

ADDRESS DOD

WEIGHT BMI

Complaints:

HISTORY OF PRESENT ILLNESS

SYSTEMIC SYMPTOMS :

PAST HISTORY:

PERSONAL HISTORY: SMOKER/ALCOHOLIC

GENERAL PHYSICAL EXAMINATION

BUILT: WELL/MODERATE/POOR

NOURISHMENT: WELL/MODERATE/POOR

PALLOR

ICTERUS

CYANOSIS

CLUBBING

KOILONYCHIA

PEDAL EDEMA

GENERAL LYMPHADENOPATHY

VITALS:

TEMPERATURE:

PULSE

RESPIRATORY RATE

BLOOD PRESSURE:

LOCAL EXAMINATION:

INSPECTION –

PALPATION –

PERCUSSION –

AUSCULTATION –

SYSTEMIC EXAMINATION:

PER ABDOMEN:

PER RECTAL:

RESPIRATORY SYSTEM

CARDIOVASCULAR SYSTEM

CENTRAL NERVOUS SYSTEM

CLINICAL DIAGNOSIS:

LABORATORY TESTS

HB%

TOTAL COUNT

DIFFERENTIAL COUNT

N/L/E/B/M

LIVER FUNCTION TEST

URINE ROUTINE:

RBS

B.UREA

S.CREATININE

HIV

HBsAg

HCV

CHEST X RAY:

ULTRASONOGRAPHY OF ABDOMEN AND PELVIS& CT (IF REQUIRED):

OPERATIVE PROCEDURE (DATE AND TIME):

INTRA-OPERATIVE FINDINGS:

1. MEAN OPERATIVE TIME
2. OPERATIVE FINDINGS / COMPLICATIONS
3. BILIARY LEAKAGE
4. STONE SPILLAGE
5. COMMON BILE DUCT INJURY
6. ROUVIERE's SULCUS(VISIBLE / NOT)
7. CALOTS'S TRIANGLE (CRITICAL VIEW OF SAFETY)
8. DRAIN PLACEMENT (PLACED OR NOT)

POST OPERATIVE ASSESSMENT:

1. POST OPERATIVE PAIN
2. PARALYTIC ILEUS
3. HEMATOMA/COLLECTION
4. BLEEDING
5. POST OPERATIVE SURGICAL SITE INFECTIONS.
6. INCISIONAL HERNIA.

Conversion to open -

Reason for conversion -

POST-OPERATIVE PERIOD

Drain removal –

Suture removal --

Wound infection/ hemarrhage / Bile leak / Prolonged ileus / Retained stone

FOLLOW UP

All patients were followed up for a period of one month.

KEY TO MASTER CHART

Alb	- Albumin
ALP	- Alkaline Phosphatase
AC	- Acute cholecystitis
ACH	- Acute Cholelithiasis
BMI	- Body Mass Index
BiT	- Total Bilirubin
BiD	- Direct Bilirubin B/S Spillage
	- Bile/Stone Spillage
CBD	- Common Bile Duct
C	- Cholelithiasis
CC	- Chronic Cholecystitis
CD	- Choledocholithiasis
CLC	-Convention LC
DM	- Diabetes Mellitus
Epg	- Epigastrium
ERCP	- Endoscopic Retrograde Cholangiopancreatography
GB	- Gall Bladder
GPE	- General Physical Examination

Hb	- Haemoglobin
HTN	- Hypertension
Lap.	- Laparoscopy
LC	- Laparoscopic Cholecystectomy
LFT	- Liver Function Test
MLC	- Mini Laparoscopic cholecystectomy
M/C	- Multiple Calculi
Mi	- Mixed diet
NS	- Nothing Significant
N	- Normal
P/A	- Per Abdomen
PT-INR	- Prothrombin International Normalized Ratio
POD	- Post Operative Day
Po	- Polyp
RHC	- Right Hypochondrium
S/C	- Solitary Calculus
S/I/C	- Solitary Impacted Calculus
SGOT	- Serum Glutamic Oxaloacetic Transaminase
SGPT	- Serum Glutamic Pyruvate Transaminase
Sl.No.	- Serial Number