Dissertation On

"EFFECT OF IRRIGATION OF GALL BLADDER BED AND TROCAR SITES WITH BUPIVACINE IN MANAGEMENT OF POST OPERATIVE PAIN IN PATIENTS UNDERGOING LAPAROSCOPIC CHOLECYSTECTOMY IN TERTIARY CARE CENTRE"

Dissertation submitted

in partial fulfilment of the requirements for the degree of

M.S.DEGREE -BRANCH-I

GENERAL SURGERY

STANLEY MEDICAL COLLEGE

THE TAMILNADU Dr.MGR MEDICAL UNIVERSITY

CHENNAI-TAMILNADU

MAY 2020

CERTIFICATE

This is to certify that, the dissertation titled **"EFFECT OF IRRIGATION OF GALL BLADDER BED AND TROCAR SITES WITH BUPIVACINE IN MANAGEMENT OF POST OPERATIVE PAIN IN PATIENTS UNDERGOING LAPAROSCOPIC CHOLECYSTECTOMY IN TERTIARY CARE CENTRE"** is the bonafide work done by

Dr.D.SATHISH KUMAR Postgraduate student (2017-2020) in the Department of General Surgery,Government Stanley Medical College and Hospital, Chennai under my guidance and supervision ,in partial fulfillment of the requirements of The Tamilnadu Dr.MGR Medical University,Chennai for the M.S.Degree Branch I General Surgery Examination tobe held in May 2020.

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DECLARATION

I **Dr.D.SATHISH KUMAR** solemnly declare that this dissertation titled

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done by myself in the Department of General Surgery ,Government Stanley Medical College Hospital, Chennai under the guidance and supervision of our unit chief **Prof. Dr.R.Manivannan,M.S,** and Our Head of the department **Prof. Dr.T.Sivakumar,M.S.**

I also affirm this work was not submitted by myself or any others for any award, degree to any other University either in India or elsewhere.This is submitted to The Tamilnadu Dr.M.G.R Medical University,Chennai in Partial fulfilment of the rules and regulations for the award Master of Surgery Degree Branch I (General Surgery).

Place:

Date: **Dr.D.SATHISH KUMAR**

CERTIFICATE

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Guide and Supervisor

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ETHICAL COMMITTEE APPROVAL LETTER

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CONTENTS

INTRODUCTION

Laparoscopic cholecystectomy is the gold standard treatment for benign gallbladder disease. It is characterized by a short hospital stay and an quick return to regular activity.

Strategies to manage the different intraabdominal surgical pathologies with a laparoscopic approach offer a significant advantage compared with the conventional technique.

Laparoscopic cholecystectomy , surgical outcome in terms of reduced pain and convalescence compared to conventional cholecystectomy. However, the postoperative pain is significant. Pain management with multiple analgesic and opioids has been reported with variable success.

The pain in the open cholecystectomy is a parietal pain. In laparoscopic cholecystectomy, pain is derived from multiple situations: incision pain (somatic), deep intraabdominal pain (visceral), and shoulder pain (visceral pain due to phrenic nerve irritation).

In 17 % to 41 % of the patients, pain being the main cause for staying overnight in the hospital the day of surgery 2–7 and the primary reason why the patients have a longer convalescence.Because postoperative pain after laparoscopic surgery is

complex mechanism,specialists suggest that effective analgesic treatment should be a multimodal support.

It should also include blocking the sensitive afferents (infiltrating the skin with a local anesthetic before any incision), irrigating a local anesthetic in the peritoneal cavity, providing the patient with fluids and electrolytes.

AIMS AND OBJECTIVES

The aim of this study was to evaluate the use of the irrigation of a local anesthetic, such as bupivacaine, at the surgical bed for postoperative pain reduction.

REVIEW OF LITERATURE

Historical Background

Open cholecystectomy,first performed by Carl Langenbuch in 1882, has been the primary treatment of gallbladder disease through the early 1990s.

In 1985, the first endoscopic cholecystectomy was performed by Erich Mühe of Böblingen, Germany.

Shortly thereafter, pioneers in France and the United States coupled a CCD video camera with a laparoscope to allow the entire surgical team to view the operative field and performed cholecystectomies with laparoscopic equipment.

Since then,laparoscopic cholecystectomy has been adopted around the world, and subsequently been the gold standard for the management of cholelithiasis.

In 1992, the National Institutes of Health (NIH) Consensus Development Conference concluded that lap cholecystectomy provides a safe and effective treatment for patients with symptomatic gallstones.

SURGICAL ANATOMY

The gallbladder is a pear-shaped organ which lies on the inferior surface of the liver at the junction of the left and right hepatic lobes between Couinaud's segments IV and V. The gallbladder varies from 7 to 10 cm in length and from 2.5 to 3.5 cm in width.

The gallbladder's volume varies considerably, being large during fasting states

and small after eating. A moderately distended gallbladder has a capacity of 50 to 60 ml of bile but may become much larger with certain pathologic states.

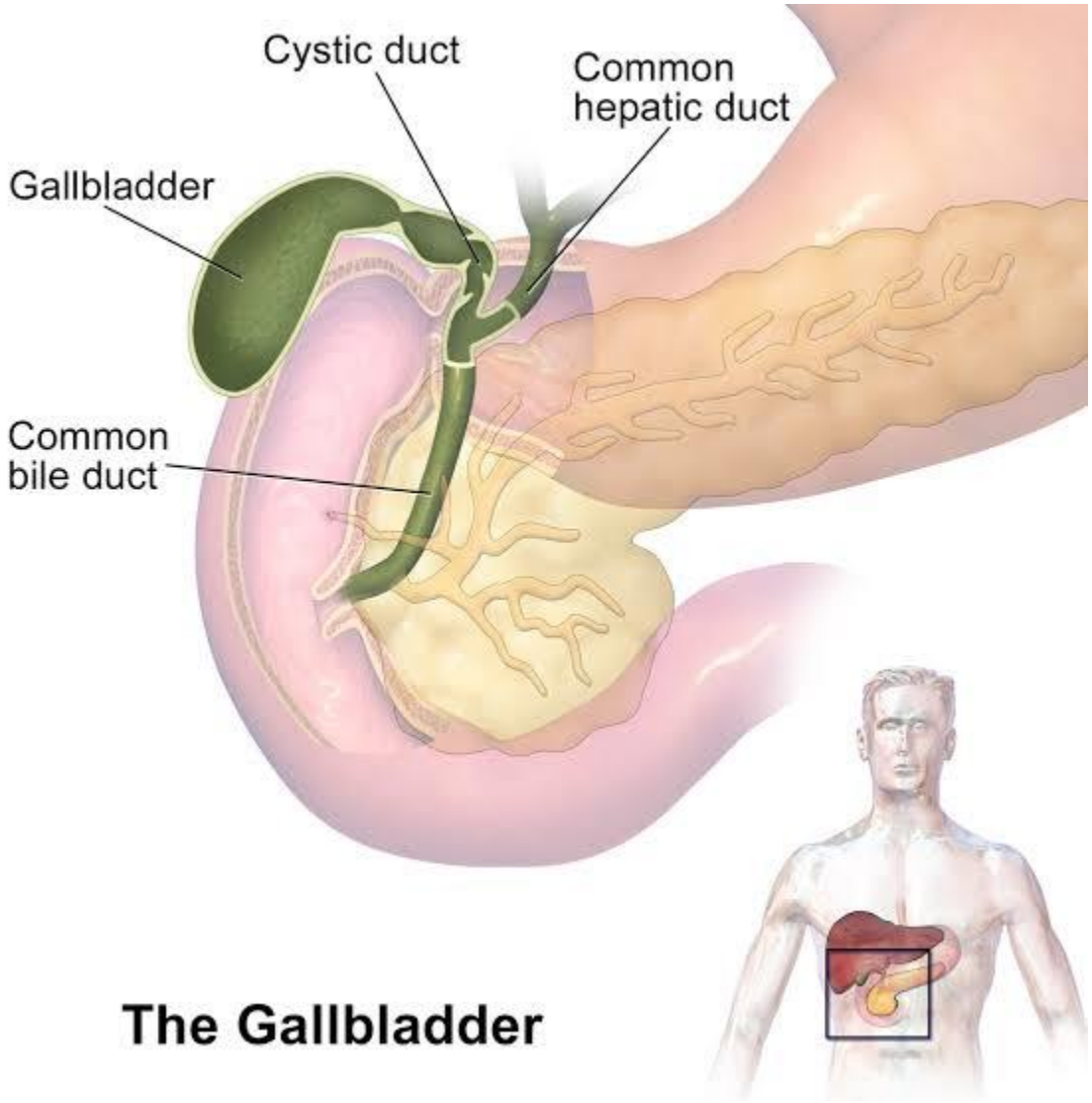


Fig.1 : The Gallbladder

The gallbladder has been divided into four areas: the fundus, body, infundibulum, and neck. Hartmann's pouch is an asymmetrical bulge of the infundibulum

that lies close to the gallbladder's neck. The neck points in a cephalad and dorsal direction to join the cystic duct.(1)

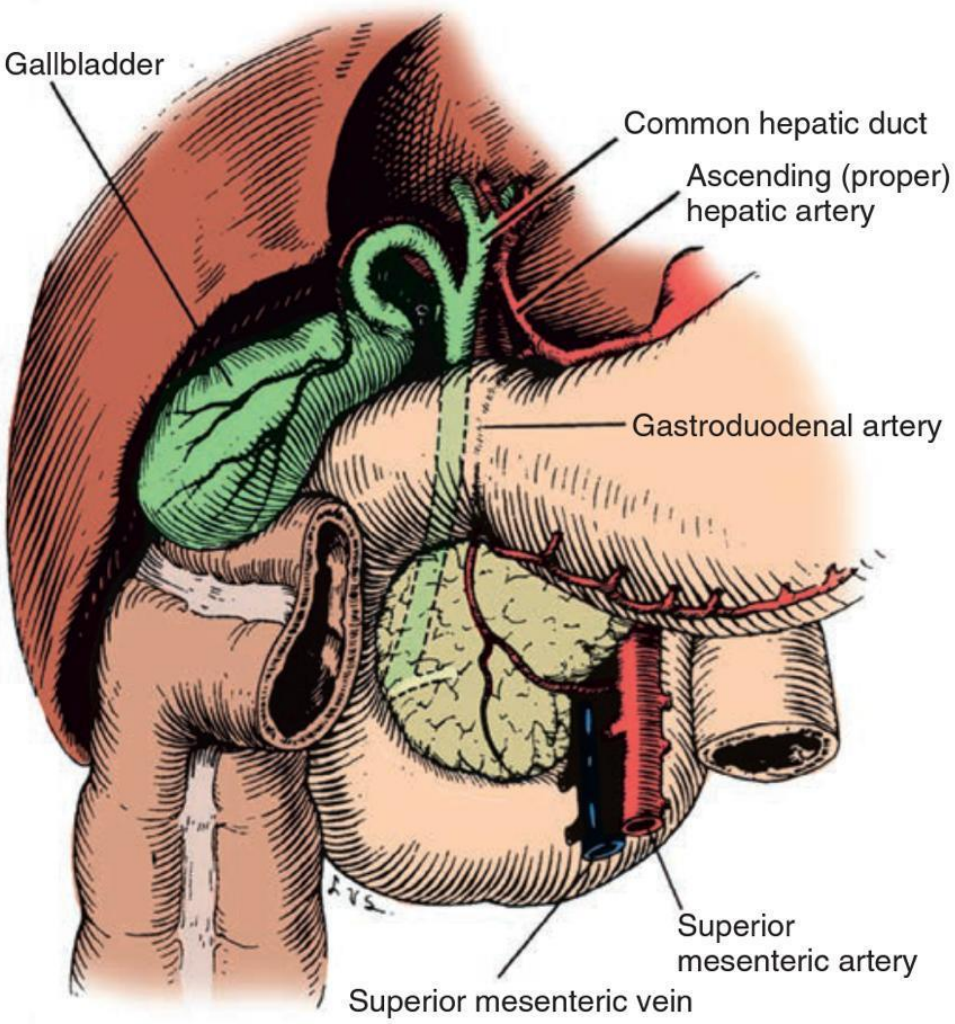


Fig.2 :Anatomic relationships of gall bladder(1)

The gallbladder wall consists of five layers. The innermost layer is the epithelium, and the other layers are the lamina propria, smooth muscle, perimuscular subserosal connective tissue, and serosa. The gallbladder has no muscularis mucosa or

submucosa. Most cells in the mucosa are columnar cells, and their main function is absorption. These cells are aligned in a single row, with slightly eosinophilic cytoplasm, apical vacuoles, and basal or central nuclei.

The lamina propria contains nerve fibers, vessels, lymphatics, elastic fibers, loose connective tissue, and occasional mast cells and macrophages. The muscle layer is a loose arrangement of circular, longitudinal, and oblique fibers without well-developed layers. Ganglia are found between smooth muscle bundles. The subserosa is composed of a loose arrangement of fibroblasts, elastic and collagen fibers, vessels, nerves, lymphatics, and adipocytes.

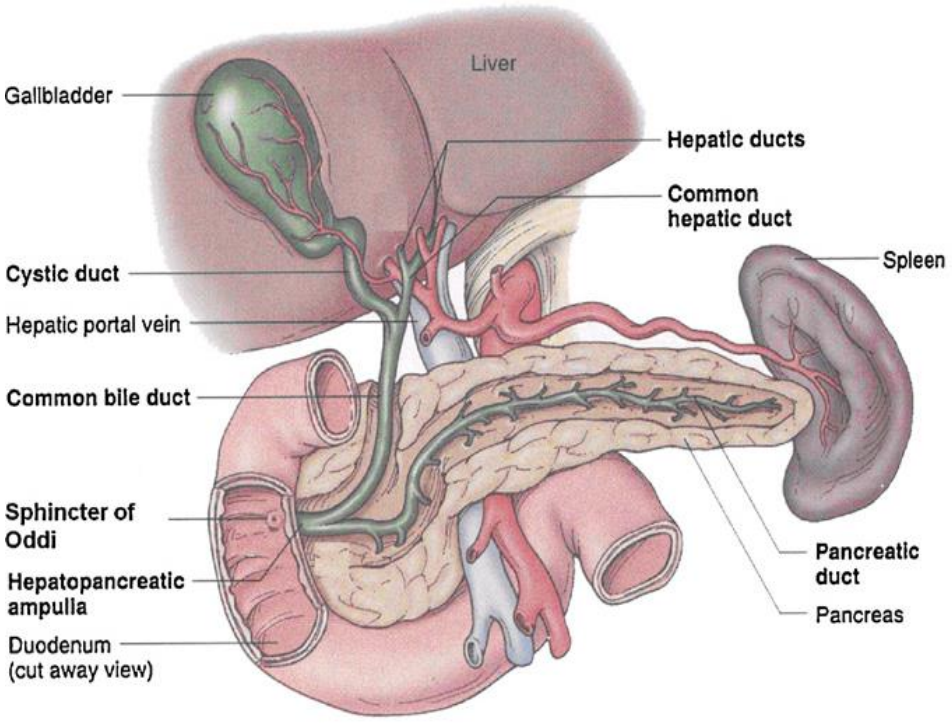


Fig.3 :Biliary Tract

Rokitansky-Aschoff sinuses are invaginations of epithelium into the lamina propria, muscle, and subserosal connective tissue. These sinuses are present in about 40% of normal gallbladders and are present in abundance in almost all inflamed gallbladders. The ducts of Luschka are tiny bile ducts found around the muscle layer on the hepatic side of the gallbladder. They are found in about 10% of normal gallbladders and have no relation to the Rokitansky-Aschoff sinuses or to cholecystitis.

The cystic duct arises from the gallbladder and joins the common hepatic duct to form the common bile duct. The length of the cystic duct is variable, averaging between 2 and 4 cm. The cystic duct usually courses downward in the hepatoduodenal ligament to join the lateral aspect of the supraduodenal portion of the common hepatic duct at an acute angle.

Occasionally, the cystic duct may join the right hepatic duct, or it may extend downward to join the retroduodenal duct. In addition, the cystic duct may join the common hepatic duct at a right angle, may run parallel to the common hepatic duct, or may enter the common hepatic duct dorsally, on its left side, behind the duodenum, or, rarely, may enter the duodenum directly. The cystic duct contains a variable number of mucosal folds, similar to those found in the neck of the gallbladder. Although referred to as valves of Heister, these spiral folds do not have a valvular function. Variations in the length and course of the cystic duct and its point of union with the common hepatic duct are common.

In 1891, Calot described a triangular anatomic region formed by the common hepatic duct medially, the cystic duct laterally, and the cystic artery superiorly.Calot's triangle is considered by most to comprise the triangular area with an upper boundary formed by the inferior margin of the right lobe of the liver, rather than the cystic artery .A thorough appreciation of the anatomy of Calot's triangle is essential during performance of a cholecystectomy because numerous important structures pass through this area. In most instances, the cystic artery arises as a branch of the right hepatic artery within the hepatocystic triangle.

A replaced or aberrant right hepatic artery arising from the superior mesenteric artery usually courses through the medial aspect of the triangle, posterior to the cystic duct. Aberrant or accessory hepatic ducts also may pass through Calot's triangle before joining the cystic duct or common hepatic duct. During performance of a cholecystectomy, clear visualization of the hepatocystic triangle is essential with accurate identification of all structures within this triangle. (1)

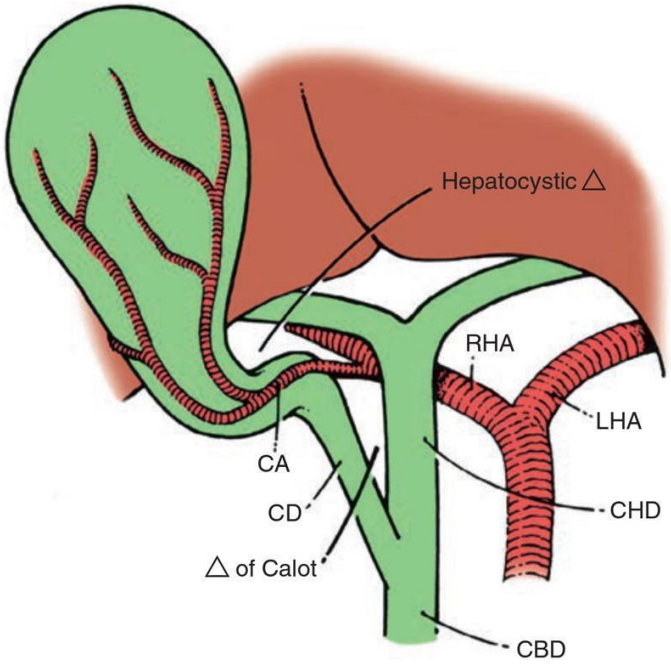


Fig.4 : The triangle (Δ) of Calot and the hepatocystic triangle. The two triangles differ in their upper boundaries. The upper boundary of Calot's triangle is the cystic artery (CA), whereas that of the hepatocystic triangle is the inferior margin of the liver. CBD, common bile duct; CD, cystic duct; CHD, common hepatic duct; LHA, left hepatic artery; RHA, right hepatic artery

GALLBLADDER FUNCTION

The main function of the gallbladder is to concentrate and store hepatic bile during the fasting state, thus allowing for its coordinated release in response to a meal. To serve this overall function, the gallbladder has absorptive, secretory, and motor capabilities.

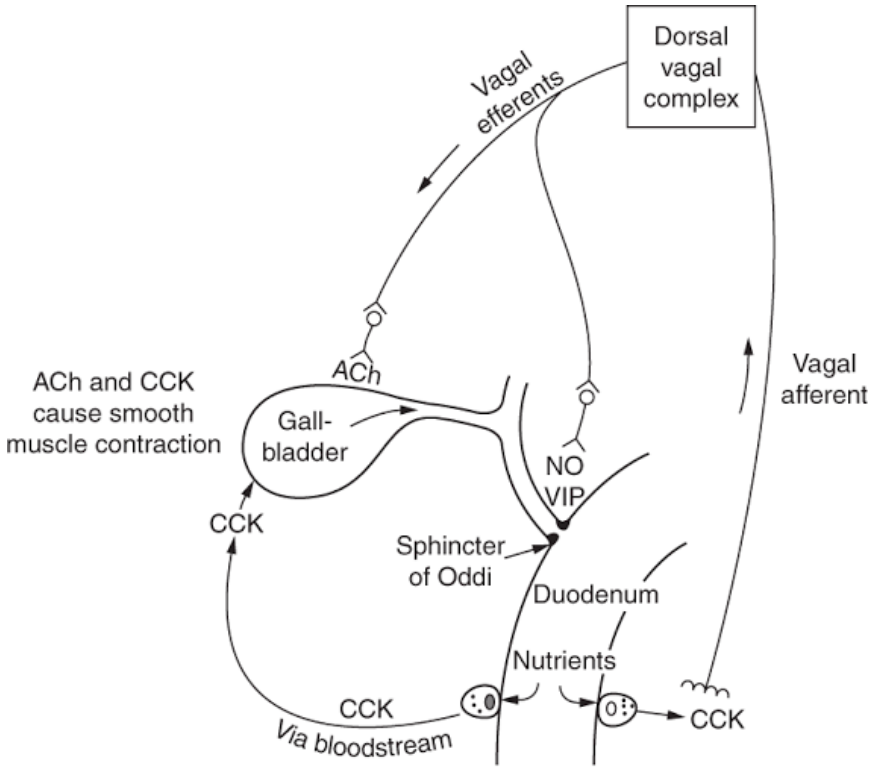


Fig.5: Gall Bladder Function

As a result of active absorption, the gallbladder stores concentrated bile that re-enters the distal bile duct and is secreted into the duodenum in response to a meal. In addition to absorption and concentration, the gallbladder's mucosa actively secretes glycoproteins and hydrogen ions.

Secretion of mucus glycoproteins occurs primarily from the glands of the gallbladder neck and cystic duct. The resultant mucin gel contains an important part of the unstirred layer (diffusion-resistant barrier) that separates the gallbladder cell membrane from the luminal bile. This mucus barrier may be very important in protecting

the gallbladder epithelium form the strong detergent effect of the highly concentrated bile salts found in the gallbladder. However, considerable evidence also suggests that mucin glycoproteins play a role as a pronucleating agent for cholesterol crystallization.

The gallbladder epithelium transports hydrogen ions to decrease in gallbladder bile pH through a sodium-exchange mechanism. Acidification of bile promotes calcium solubility, thereby preventing its precipitation as calcium salts. The gallbladder's normal acidification process reduces the pH of entering hepatic bile from 7.5 to 7.8 down to 7.1 to 7.3.(2)

Absorption

The gallbladder's mucosa has the greatest absorptive capacity per unit of any structure in the body. Bile is usually concentrated fivefold by the absorption of water and electrolytes. The gallbladder epithelium concentrates bile by Active Na-Cl transport.

Water is passively absorbed in response to the osmotic force generated by solute absorption.Both calcium and cholesterol solubilities affected by the concentration of bile. The concentration of calcium in gallbladder bile, which is an important factor in gallstone pathogenesis, is influenced by serum calcium, hepatic bile calcium, gallbladder

water absorption, and the concentration of organic substances such as bile salts in gallbladder bile.Although the gallbladder mucosa does absorb calcium, this process is not nearly as efficient as for sodium or water.

As the gallbladder bile gets concentrated, several changes occur in the bile's capacity to solubilize cholesterol. The solubility of the micellar fraction is increased, but stability of phospholipids-cholesterol vesicles is greatly decreased.

Because cholesterol crystal precipitation occurs mainly by vesicular rather than micellar mechanisms, the net effect of concentrating bile is an increased tendency to nucleate cholesterol.Absorption of organic compounds also occurs; lipid solubility is the main determinant of movement across the gallbladder mucosa.

However, the absorption of bilirubin, cholesterol, phospholipids, and bile salts is low compared with that of water. Thus, these organic compounds are significantly concentrated by normal absorptive process that occurs in the gallbladder.

Unconjugated bile salts are absorbed more easily than conjugated bile salts and may actually damage the gallbladder's mucosa, causing a nonselective increase in

absorption of other solutes. Thus, increased absorption of unconjugated bile salts, caused by bacterial deconjugation or mucosal inflammation, may impair cholesterol solubility and therefore promote cholesterol gallstone formation.(3)

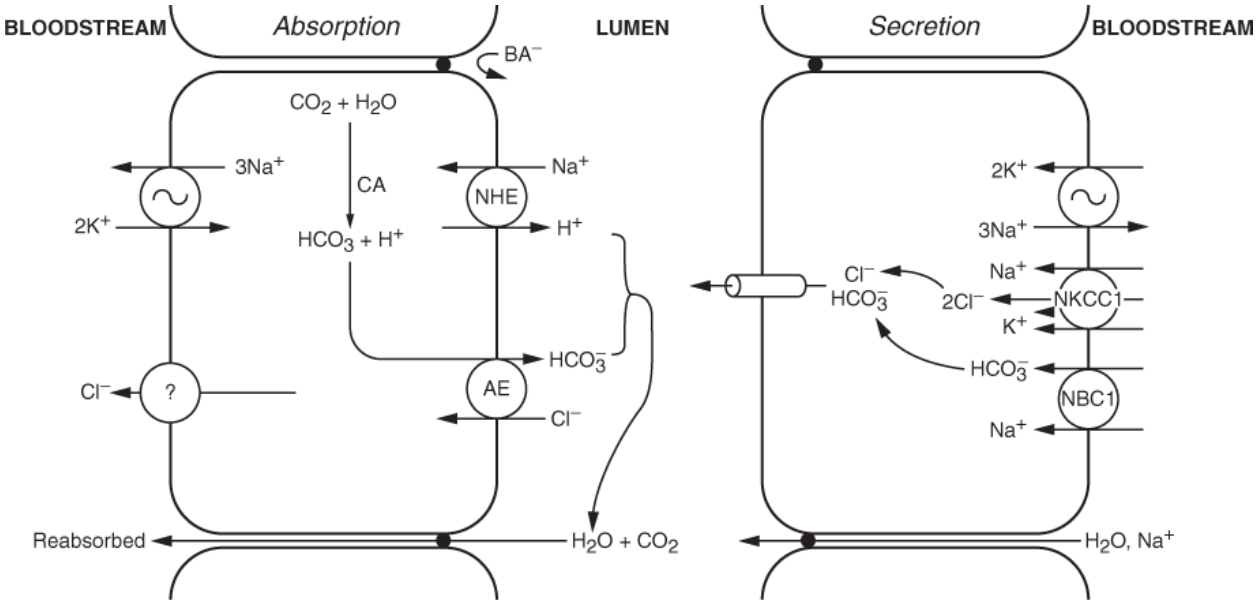


Fig.6 : Absorptive and Secretion of Bile

Secretion

The gallbladder's epithelial cells secrete at least two important products into its lumen: glycoproteins and hydrogen ions. Prostaglandins play an important role as

stimulants of gallbladder mucin secretion. Furthermore, mucin glycoproteins are key pronucleating agents for cholesterol crystallization.

The acidification of bile occurs by the transport of hydrogen ions by the gallbladder epithelium, probably through a sodium-exchange mechanism.

Acidification of the bile improves calcium solubility, thereby prevents its precipitation as calcium salts. The gallbladder's normal acidification process lowers the pH of gallbladder bile, which normally varies from approximately 7.1 to 7.3.

Compared with gallbladder bile, the bile secreted by the liver is slightly alkaline, pH 7.5 to 7.8, so that excess losses of hepatic bile may cause metabolic acidosis.

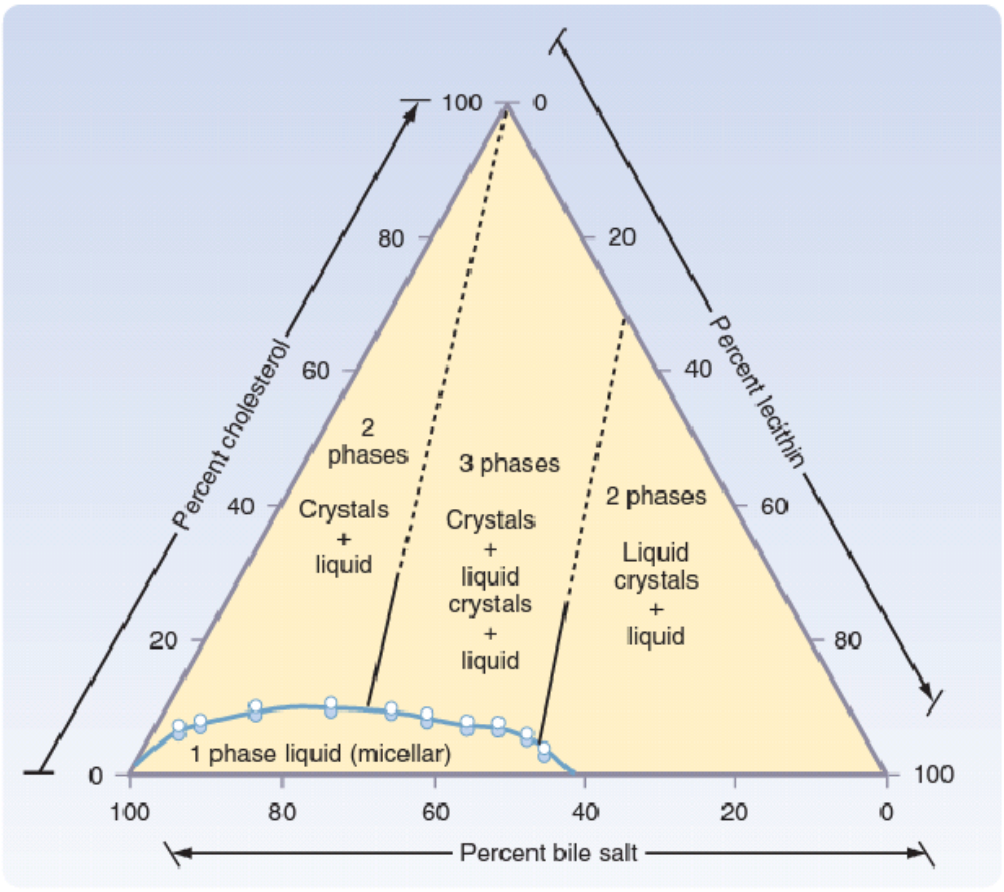


Fig.7 : Proportion of Bile

Motility

Gallbladder filling is increased by tonic contraction of the ampullary sphincter, which maintains the constant pressure in the common bile duct (10 to 15 mm Hg). However, the gallbladder does not simply fill passively and continuously during fasting. Instead, periods of filling are punctuated by brief periods of partial emptying (10% to 15% of its volume) of concentrated gallbladder bile which are coordinated with

each passage through the duodenum of phase III of the migrating myoelectric complex (MMC). This process is mediated, at least in part, by the hormone motilin.

Following meal, the release of stored bile from the gallbladder requires a coordinated motor response of gallbladder contraction and sphincter of Oddi relaxation. One of the main stimuli to the gallbladder emptying is the hormone, cholecystokinin, which is released from the duodenal mucosa in response to a meal. When stimulated by eating, the gallbladder empties 50% to 70% of its contents within 30 to 40 minutes, then Gallbladder refills gradually over the next 60 to 90 minutes.

Many other hormonal and neural pathways are also necessary for the coordinated action of gallbladder and sphincter of Oddi. Defects in gallbladder motility, which increases the residence time of bile in gallbladder, plays a central role in the pathogenesis of gallstones.

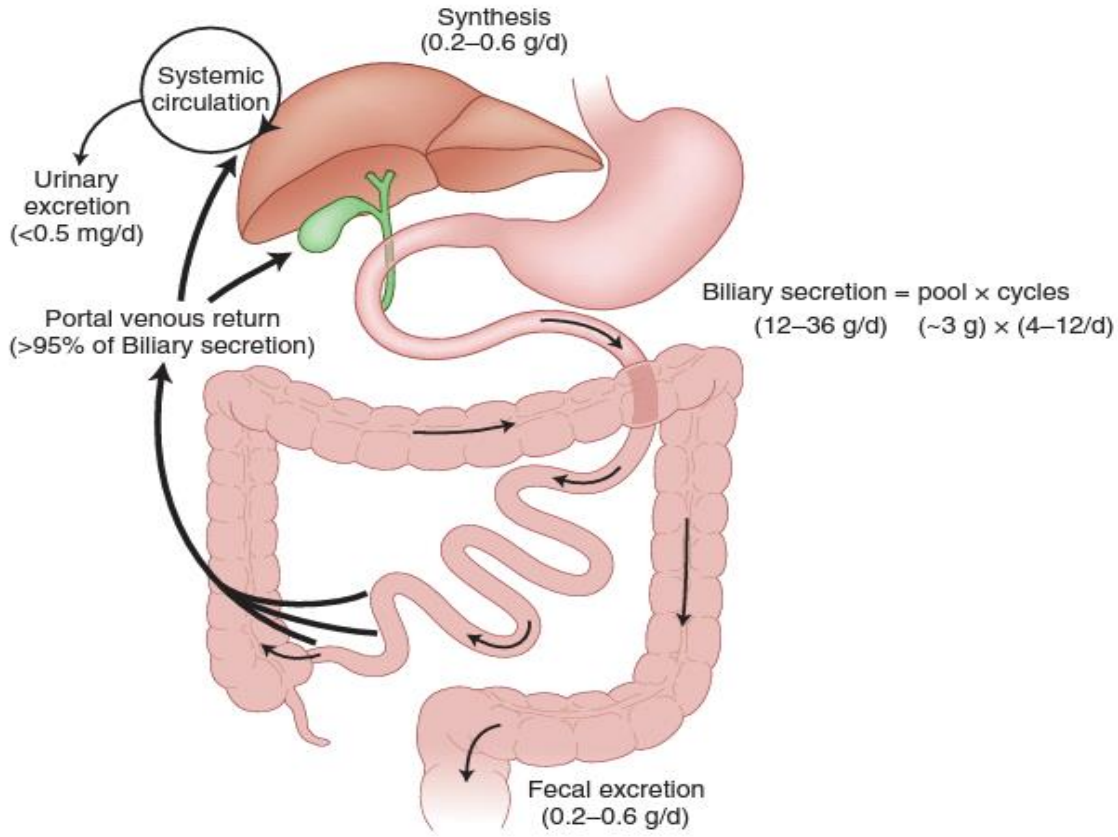


Fig.8:Enterohepatic circulation

ETIOLOGY AND PATHOGENESIS OF GALLSTONES

Gallstones represent an inability to maintain certain biliary solutes, primarily cholesterol and calcium salts, in a solubilized state. Classification of gallstones is by its cholesterol content as either cholesterol or pigment stones.

Pigment stones, further classified as either black or brown. Pure cholesterol stones are not common (10%), with most cholesterol stones containing calcium salts in their center, or nidus. In the United States, 70% to 80% of gallstones are cholesterol, and black pigment stones account for most of the remaining 20% to 30%.

Biliary sludge refers to the mixture of cholesterol crystals, calcium bilirubinate granules, and a mucin gel matrix. It is most commonly found in prolonged fasting states or with the use of parental nutrition. The finding of the macromolecular complexes of mucin and the bilirubin suggests that sludge may serve as the nidus for the gallstone pathogenesis.

Cholesterol Gallstones

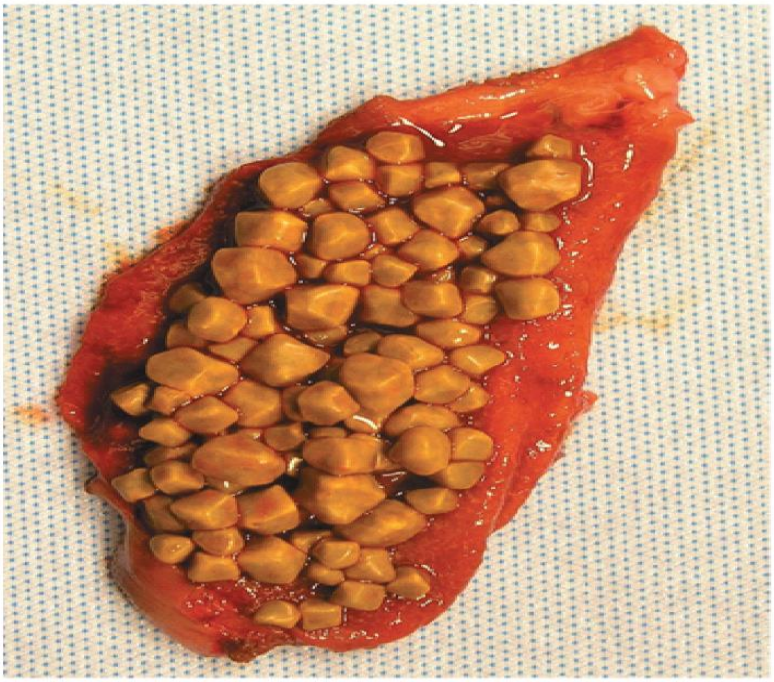


Fig.9 : Cholesterol Gallstones

The pathogenesis of cholesterol gallstones involves three stages:

1. Cholesterol supersaturation in bile
2. Crystal nucleation
3. Stone growth

Gallbladder mucosal and motor function plays a key role in gallstone formation. To maintain cholesterol in solution is the formation of micelles, a bile salt–phospholipid-cholesterol complex, and cholesterol-phospholipid vesicles. If cholesterol production is in excess, these large vesicles also exceed their capability to transport the cholesterol, and crystal precipitation may occur.

One third of biliary cholesterol is transported in micelles, but the cholesterol-phospholipid vesicles carry the majority of biliary cholesterol. By plotting the percentages of each component on triangular coordinates, the micellar zone in which cholesterol is completely soluble can be demonstrated . In the area above the curve, bile is supersaturated with cholesterol, and precipitation of cholesterol crystals can occur. (4)

Pigment Gallstones

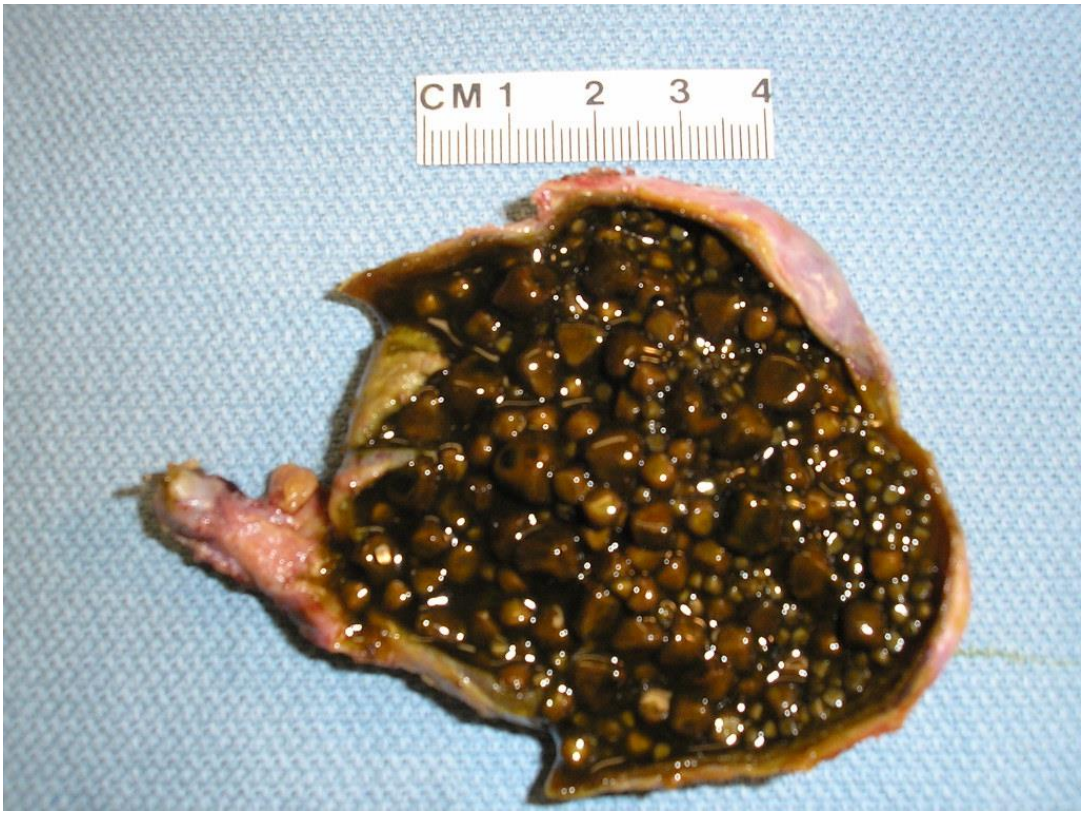


Fig.10: Pigment Gallstones

Pigment stones consists of less than 20% cholesterol and are dark owing to the presence of calcium bilirubinate. Otherwise, black and brown pigment stones have less in common and be considered as separate entities.

Black pigment stones are small and tarry, and are frequently associated with hemolytic conditions such as hereditary spherocytosis and sickle cell disease or cirrhosis. In hemolytic states, the bilirubin load and concentration of unconjugated bilirubin increases.

Cirrhosis may lead to increased secretion of unconjugated bilirubin. These stones are usually not associated with infected bile and are located almost exclusively in the gallbladder. Black stones account for a high percentage of gallstones in Asian countries such as Japan compared with the Western hemisphere.(4)

Brown pigment stones are soft and are typically found in bile ducts, especially in Asian populations. Brown stones often contain more cholesterol and calcium palmitate and occur as primary common duct stones in Western patients with disorders of biliary motility and associated bacterial infection.

Bacteria producing slime such as E. coli secrete β-glucuronidase that causes enzymatic hydrolysis of soluble conjugated bilirubin glucuronide to produce insoluble free bilirubin, which then precipitates with calcium.

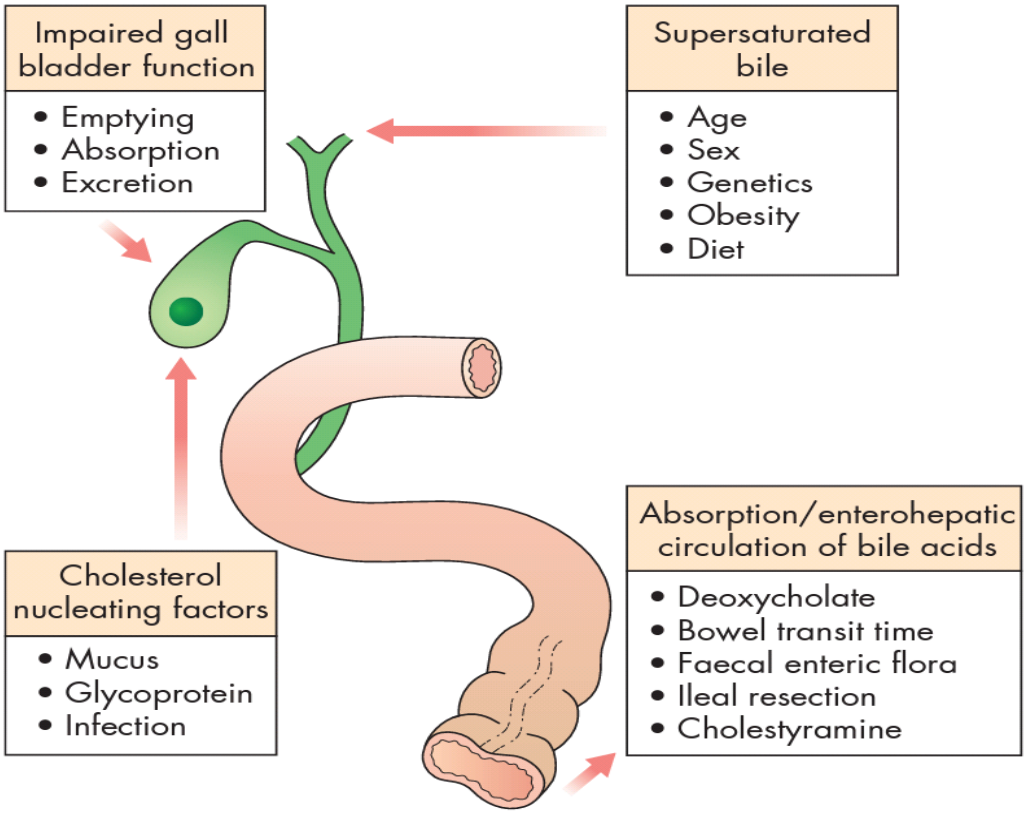


Fig.11 : Factors associated with gallstone formation.(5)

CLINICAL FEATURES AND INVESTIGATIONS

Gallstones may remain symptomatic, being detected incidentally as imaging is performed for other symptoms. If symptoms occur, patients typically complain of right upper quadrant or epigastric pain, which may radiate to the back. This may be described as colicky, but more often is dull and constant.

Other symptoms include dyspepsia, flatulence, food intolerance, particularly to fats, and some alteration in bowel frequency. Biliary colic is typically present in 10–25 per cent of patients. This is described as a severe right upper quadrant pain which ebbs and flows associated with nausea and vomiting. Pain may radiate to the chest. The pain is severe and last for minutes or even several hours. Frequently, the pain starts during the night and wakes the patient. Minor episodes of the same discomfort may occur intermittently during the day.

Dyspeptic symptoms also present and be worse after such an attack. As the pain subsides, the patient condition improves and is able to eat and drink again, often only to suffer further episodes. It is of interest that a patient may have several episodes of this nature over a period of a few weeks and then no more trouble for some months.

Jaundice may result if the stone migrates from the gall bladder and obstructs the common bile duct. Rarely, a gallstone can lead to bowel obstruction (gallstone ileus).(5)

Investigations

A diagnosis of gallstone disease is based on the history and physical examination with confirmatory radiological studies, such as transabdominal ultrasonography and radionuclide scans.

When patients with suspected diseases of the gallbladder, a complete blood count and liver function tests are routinely requested. An elevated white blood cell (WBC) count may indicate or raise suspicion of cholecystitis. If associated with an elevation of bilirubin, alkaline phosphatase, and aminotransferase, cholangitis should be suspected. Cholestasis, an obstruction to bile flow, is characterized by an elevation of bilirubin (i.e., the conjugated form), and a rise in alkaline phosphatase. Serum aminotransferases may be normal or mildly elevated. In patients with biliary colic or chronic cholecystitis, blood tests will typically be normal.

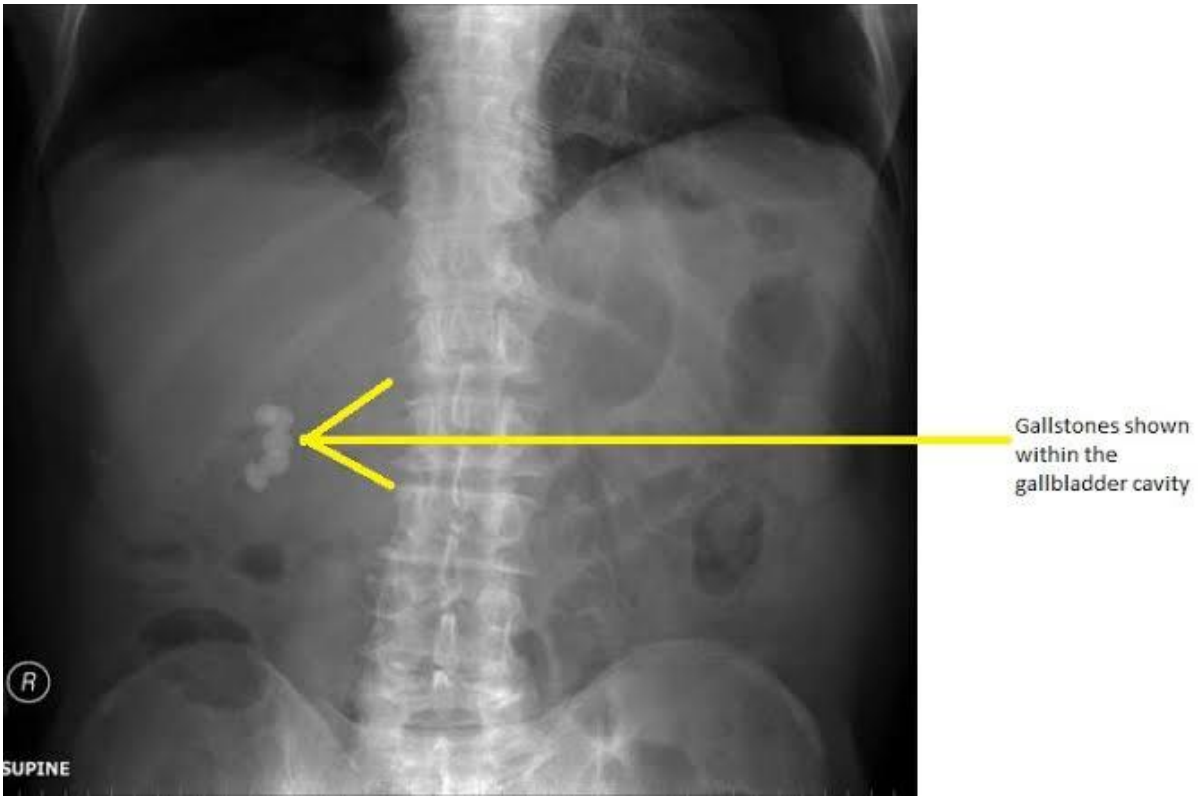


Fig.12 : Plain X-Ray film

A Plain abdominal x-ray films shows gall stones(calcified).Because 10 percentage of gallstones are calcified,xrays has limited usefulness.

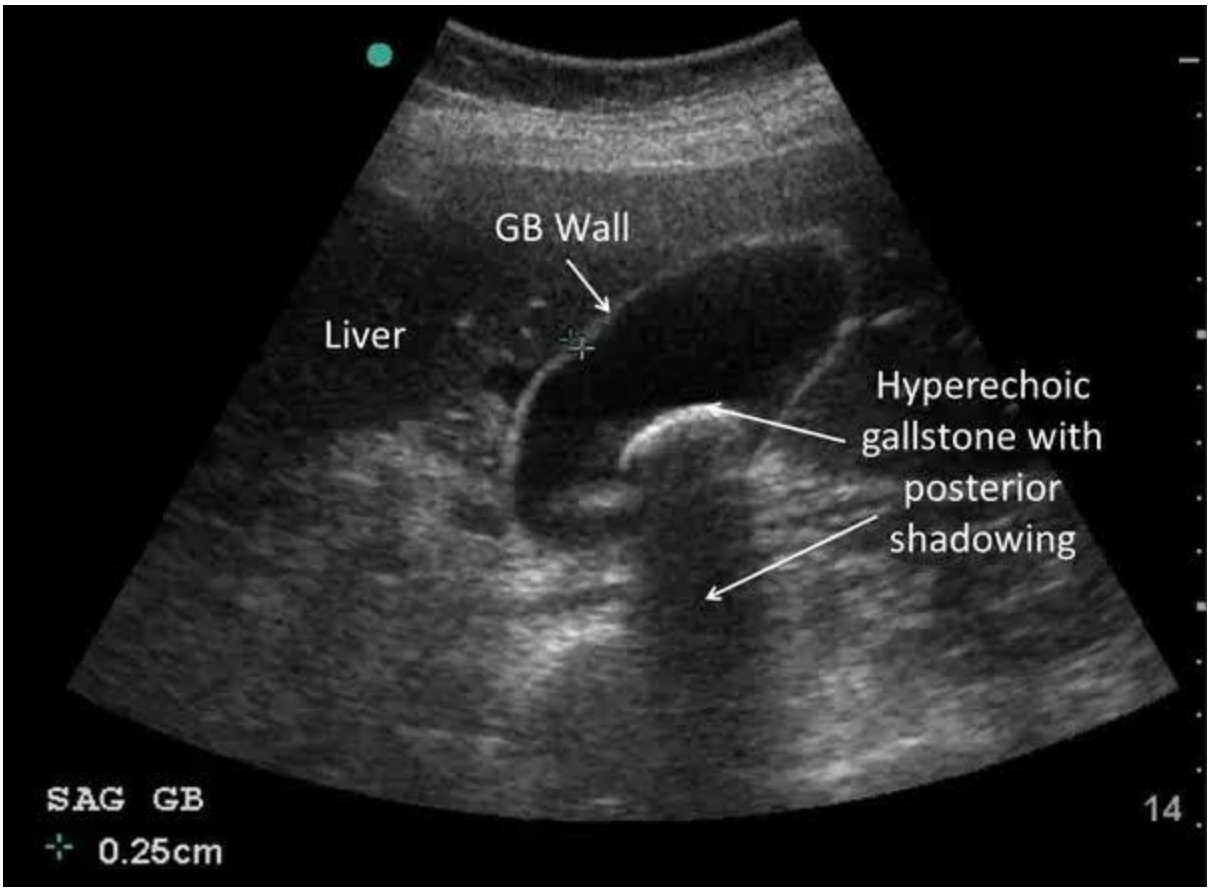


Fig.13: USG showing Gallstone

An ultrasound is the initial investigation of any patient suspected of disease of the biliary tree. It is noninvasive, painless, does not submit the patient to radiation, and can be performed on critically ill patients.

It is dependent upon the skills and the experience of the operator, and it is dynamic (i.e., static images do not give the same information as those obtained during the ultrasound investigation itself). Adjacent organs can frequently be examined at the same time. Obese patients, patients with ascites, and patients with distended bowel may be difficult to examine satisfactorily with an ultrasound.

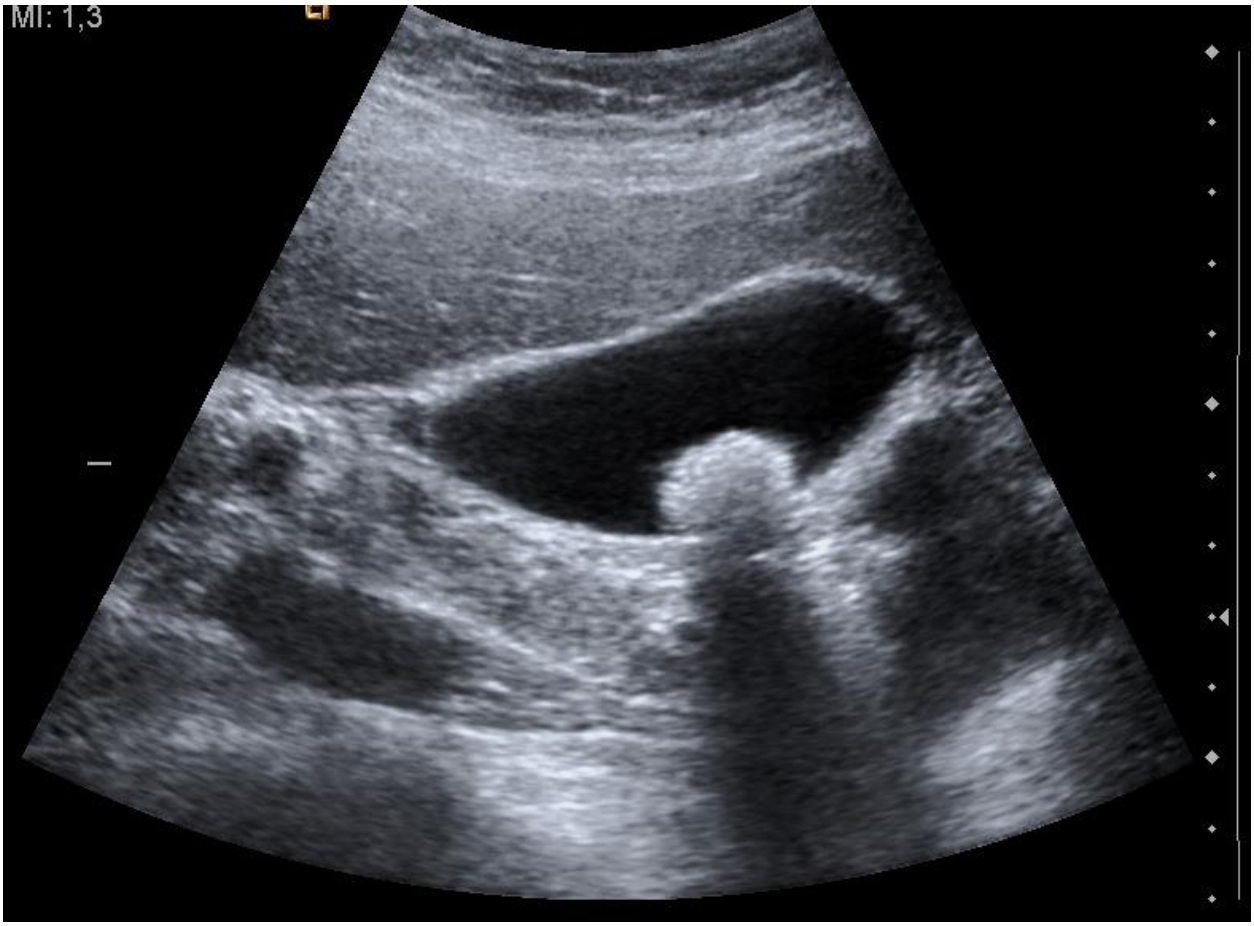


Fig.14:Acoustic shadowing

Ultrasound will show stones in the gallbladder with sensitivity and specificity of >90%. Stones are acoustically dense and reflect the ultrasound waves back to the ultrasonic transducer. Because stones block the passage of sound waves to the region behind them, they also produce an acoustic shadow.

Stones move with changes in position. Polyps may be calcified and reflect shadows, but do not move with change in posture. Some stones form a layer in the gallbladder; others a sediment or sludge.

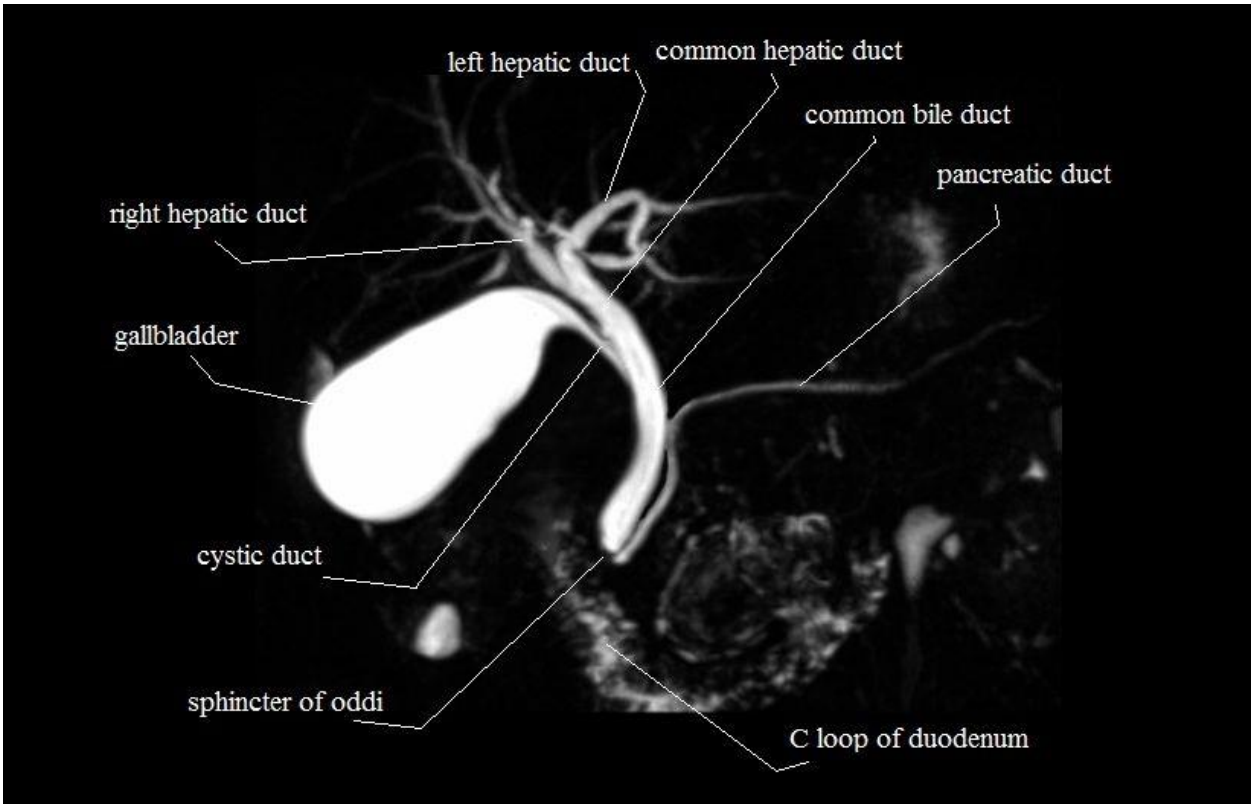


Fig .15: Normal MRCP film

The most significant contribution in the use of noninvasive imaging for management of patients with suspected biliary disease is MRI. MRCP has played an increasing role in the evaluation of gall stones.

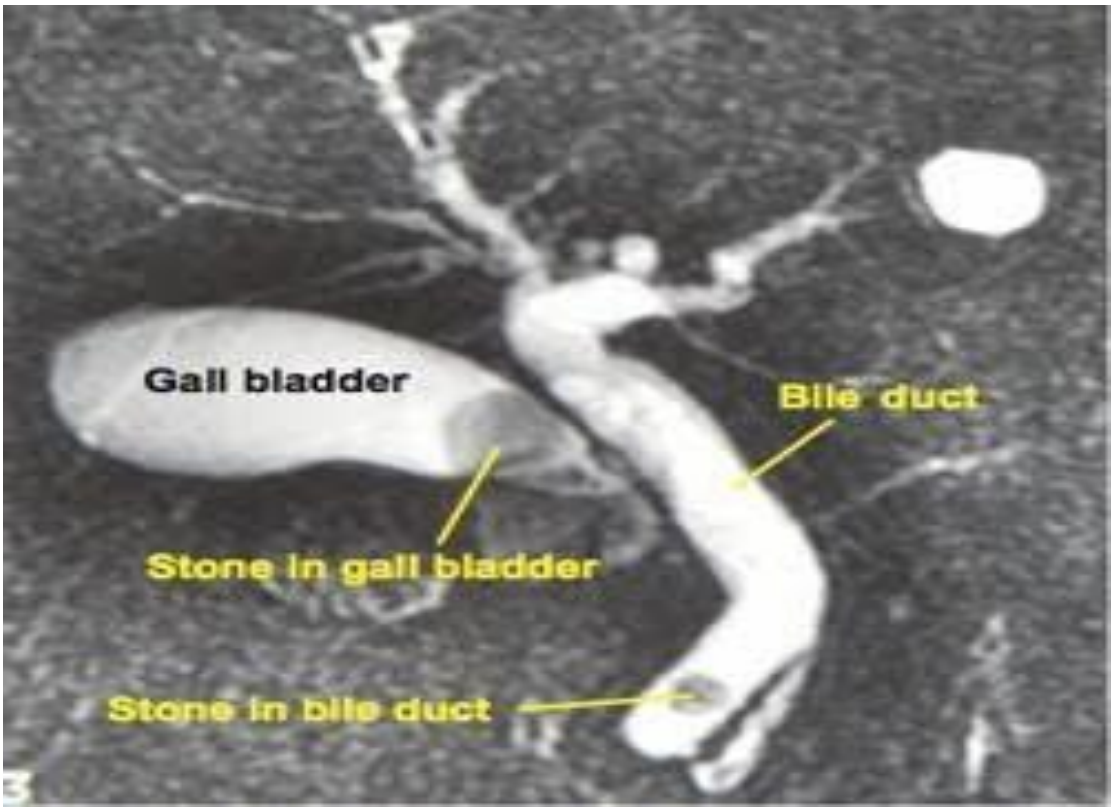


Fig 16: MRCP showing Gallstone

Though more expensive than US or CT, MRCP is considered an accurate, noninvasive technique for evaluation of the biliary tract. MRCP uses heavily T2-weighted sequences to show bile ducts as high-signal-intensity structures.

Many MRI techniques (i.e., pulse sequences) have been described to generate high-resolution images.MRCP may be useful in the evaluation of congenital disorders and benign and malignant biliary obstruction and for patients with failed or incomplete ERCP or PTC.This is a valuable noninvasive means to visualize the biliary tree before therapeutic intervention or surgery.

In the acute phase, the patient may have right upper quadrant tenderness that is exacerbated during inspiration by the examiner's right subcostal palpation (Murphy's sign). A positive Murphy's sign suggests acute inflammation and may be associated with a leukocytosis and moderately elevated liver function tests.

A mass may be palpable as the omentum walls off an inflamed gall bladder. Fortunately in the majority of cases, the process is limited by the stone slipping back into the body of the gall bladder and the contents of the gall bladder escaping by way of the cystic duct. Thus adequate drainage of the gall bladder is achieved and enables the inflammation to resolve.

If resolution does not occur, an empyema of the gall bladder may result. The wall may become necrotic and perforate, with development of localised peritonitis. The abscess may then perforate into the peritoneal cavity with a septic peritonitis – however, this is uncommon, because the inflamed gall bladder is usually localised by omentum which contains the perforation.A palpable, non-tender gall bladder (Courvoisier's sign) portends a more sinister diagnosis. This usually results from a distal common duct obstruction secondary to a peripancreatic malignancy.

Rarely, a non-tender, palpable gall bladder results from complete obstruction of the cystic duct with reabsorption of the intraluminal bile salts and secretion of uninfected mucus secreted by the gall bladder epithelium leading to a mucocoele of the gall bladder.(5)

MANAGEMENT

Medical management

Two bile acids, Ursodeoxycholic acid and Chenodeoxy cholic acid which inhibits HMG CO-A Reductase enzyme ,thereby decreasing cholesterol saturation of bile.

While waiting for surgery, or if surgery has to be postponed, the patient should be advised to avoid dietary fats and large meals.

Laparoscopic cholecystectomy

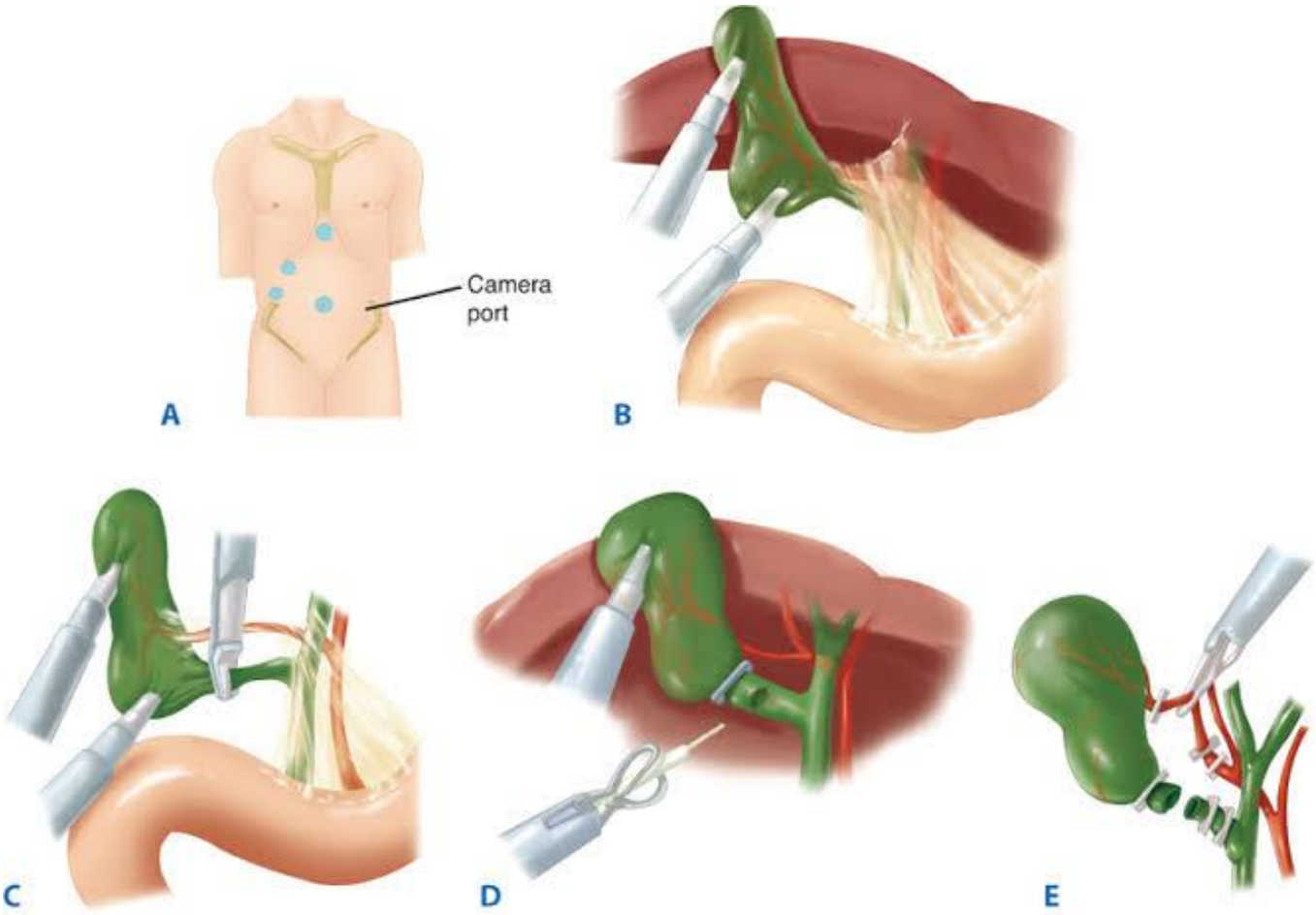


Fig.17: Laparoscopic cholecystectomy

Laparoscopic cholecystectomy is the procedure of choice for the majority of patients with gall bladder disease. The patient is placed supine on the operating table.(6)

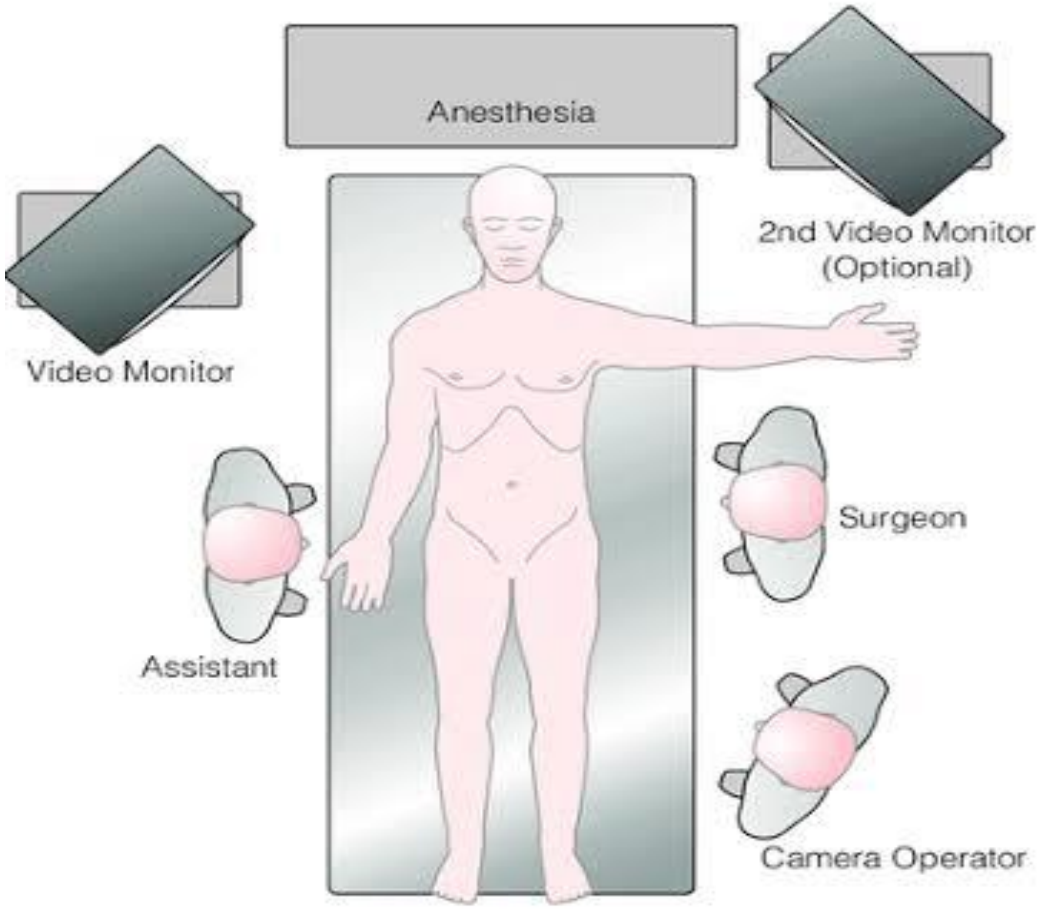


Fig.18 : Operating Setup

- Following induction and maintenance of general anaesthetic, the abdomen is prepared in a standard fashion.

- Pneumoperitoneum is established by an open subumbilical cutdown with direct visualisation of the peritoneum to place the initial port. This port will function as the camera port.

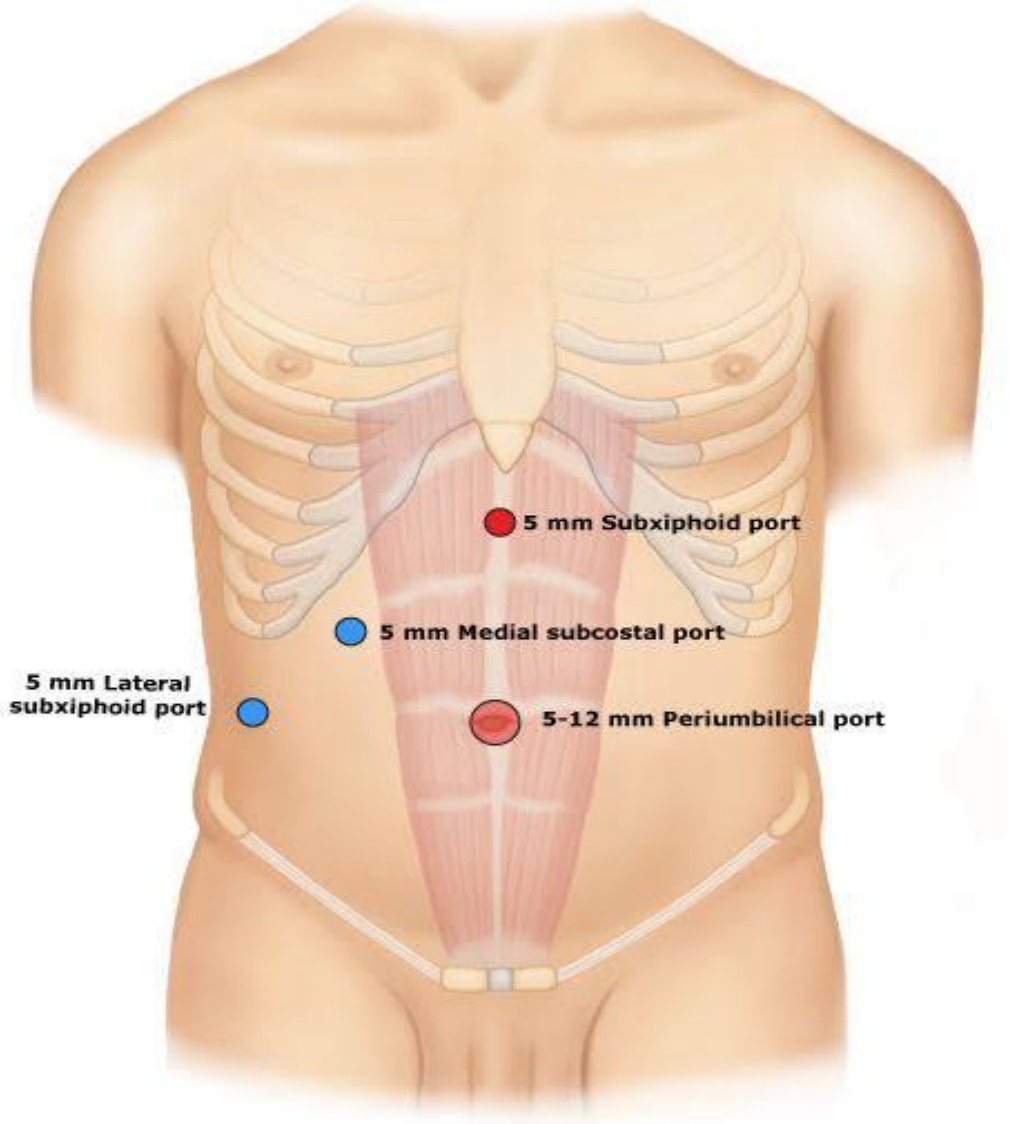


Fig.19 : Port placement

- An angled telescope (30°) is preferred.Additional operating ports are inserted in the subxiphoid area and in the right subcostal area.

- The patient is placed in a reverse Trendelburg position slightly rotated to the left. This exposes the fundus of the gall bladder which is retracted towards the diaphragm.

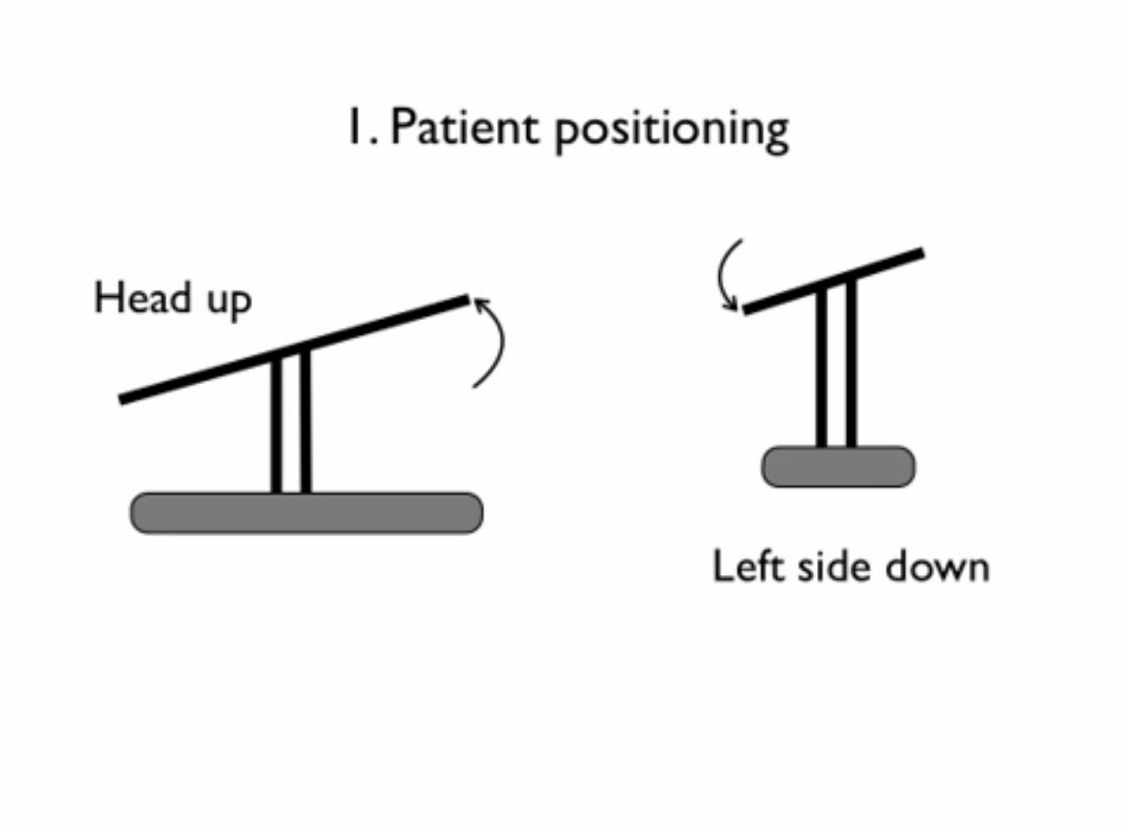


Fig.20 : Positioning of the patient

- The neck of the gall bladder is then retracted towards the right iliac fossa exposing Calot's triangle.

- The key is the identification and safe dissection of Calot's triangle .

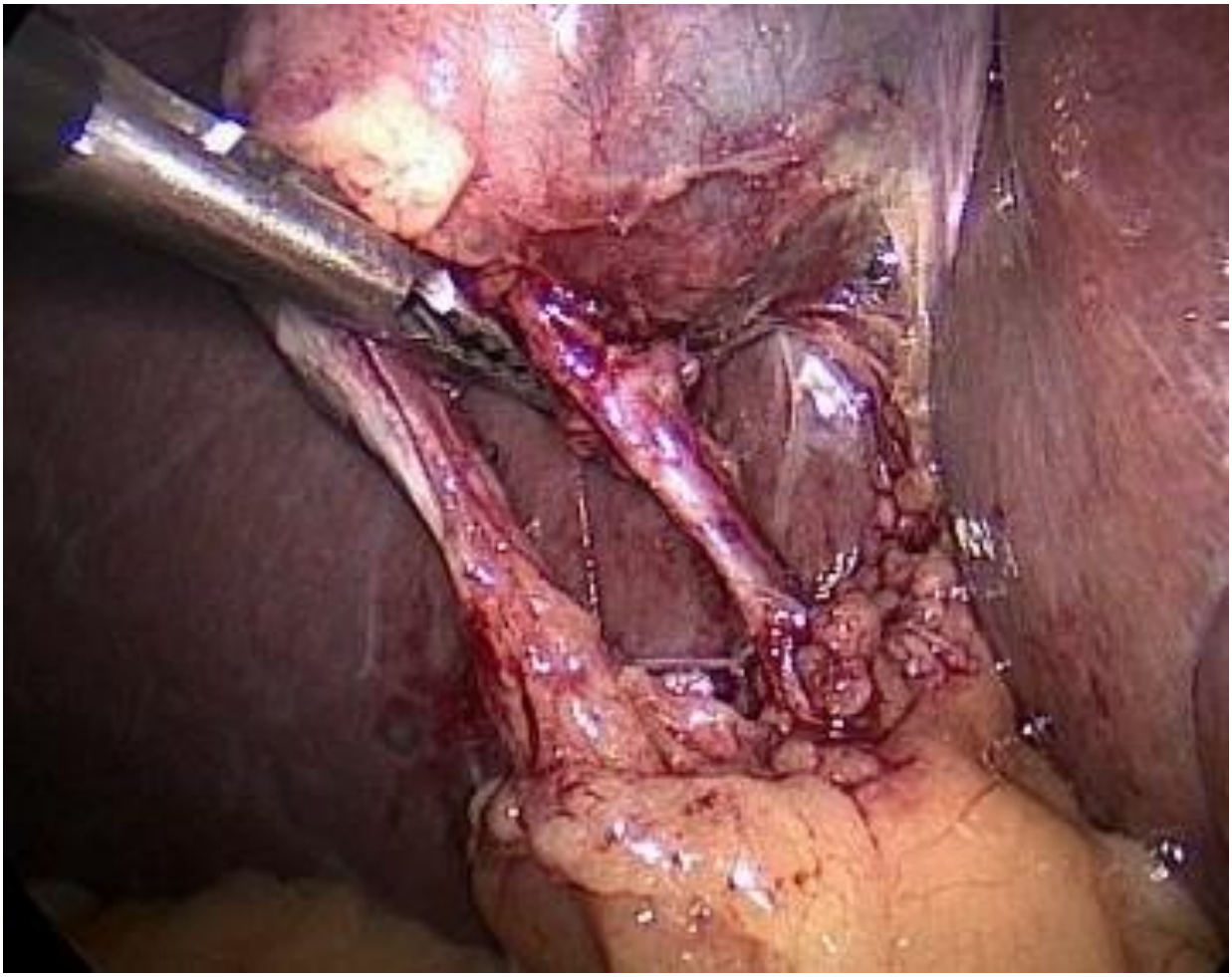


Fig.21:Calots triangle

- This area is laid widely open by dividing the peritoneum on the posterior and on the anterior aspect.

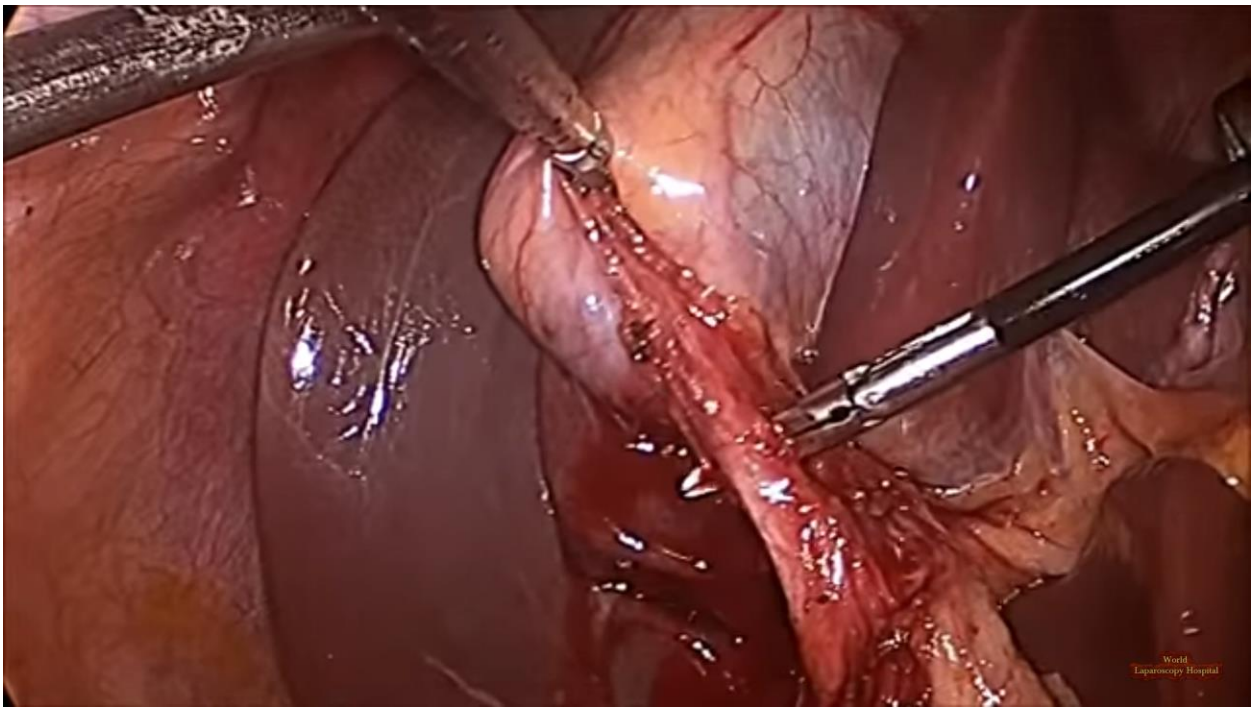


Fig.22 : Creating window between cystic duct and artery

- The cystic duct is carefully defined, as is the cystic artery.

- The gall bladder is separated from the liver bed for about 2 cm to allow for confirmation of the anatomy.

- Once the anatomy is clearly defined and the triangle of Calot has been laid widely open, the cystic duct and artery are clipped and divided.

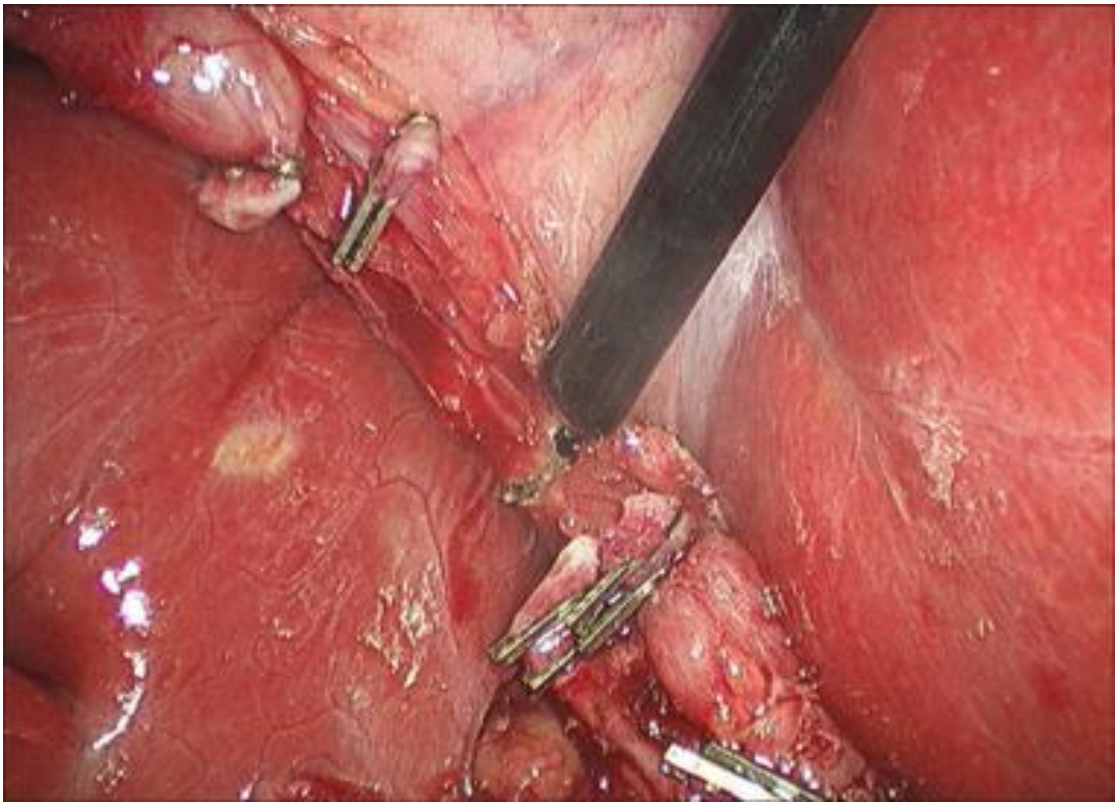


Fig.23 : Clipping and dividing the cystic duct

- The gall bladder is then removed from the gall bladder bed by sharp or cautery dissection and once free removed via the subxiphoid port.

Indications for Laparoscopic cholecystectomy

1)Symptomatic cholelithiasis

- Biliary colic
- Acute cholecystitis
- Gallstone pancreatitis

2) Asymptomatic cholelithiasis

- Sickle cell disease
- Total parenteral nutrition
- Chronic immunosuppression
- No immediate access to health care facilities (e.g., missionaries, military personnel, peace corps workers, relief workers)
- Incidental cholecystectomy for patients undergoing procedure for other indications

3) Acalculous cholecystitis (biliary dyskinesia)

4) Gallbladder polyps >1 cm in diameter

5) Porcelain gallbladder

Contraindications for Laparoscopic cholecystectomy

Absolute

- Unable to tolerate general anesthesia
- Refractory coagulopathy
- Suspicion of gallbladder carcinoma

Relative

- Previous upper abdominal surgery
- Cholangitis

- Diffuse peritonitis
- Cirrhosis and/or portal hypertension
- Chronic obstructive pulmonary disease
- Cholecystoenteric fistula
- Morbid obesity

POST OPERATIVE COMPLICATIONS

- Post operative pain
- Hemorrhage
- Bile duct injury
- Bile leak
- Retained stones
- Pancreatitis
- Wound infection
- Incisional hernia
- Pneumoperitoneum related: CO_2 embolism Vaso-vagal reflex Cardiac arrhythmias Hypercarbic acidosis
- Trocar related: Abdominal wall bleeding, hematoma Visceral injury Vascular injury(7)

POST OPERATIVE PAIN

The definition of the Pain by Taxonomy Committee of International Association for study of Pain (IASP) as "The unpleasant sensory and emotional experience associated with an actual or potential tissue damage or described in terms of such damage".

Postoperative pain is considered as a form of acute pain due to surgical trauma with an inflammatory reaction and initiation of an afferent neuronal barrage. It is a combined constellation of several unpleasant sensory, emotional and mental experiences precipitated by the surgical trauma and associated with autonomic, endocrine, metabolic,physiological and behavioral responses.(8)

Pathways of pain:

Pain is conducted along three neuron pathway that transmits the noxious stimuli from periphery to cerebral cortex.

- A first order neuron (cell body in dorsal root ganglion) transmits pain from a peripheral receptor to the dorsal horn of the spinal cord

- A second-order neuron located in the dorsal horn of the spinal cord sends axons which crosses the midline to ascend in the spinothalamic tract to the thalamus.

- A third-order neuron in the Thalamus projects its fibers to the post central gyrus (via the internal capsule).

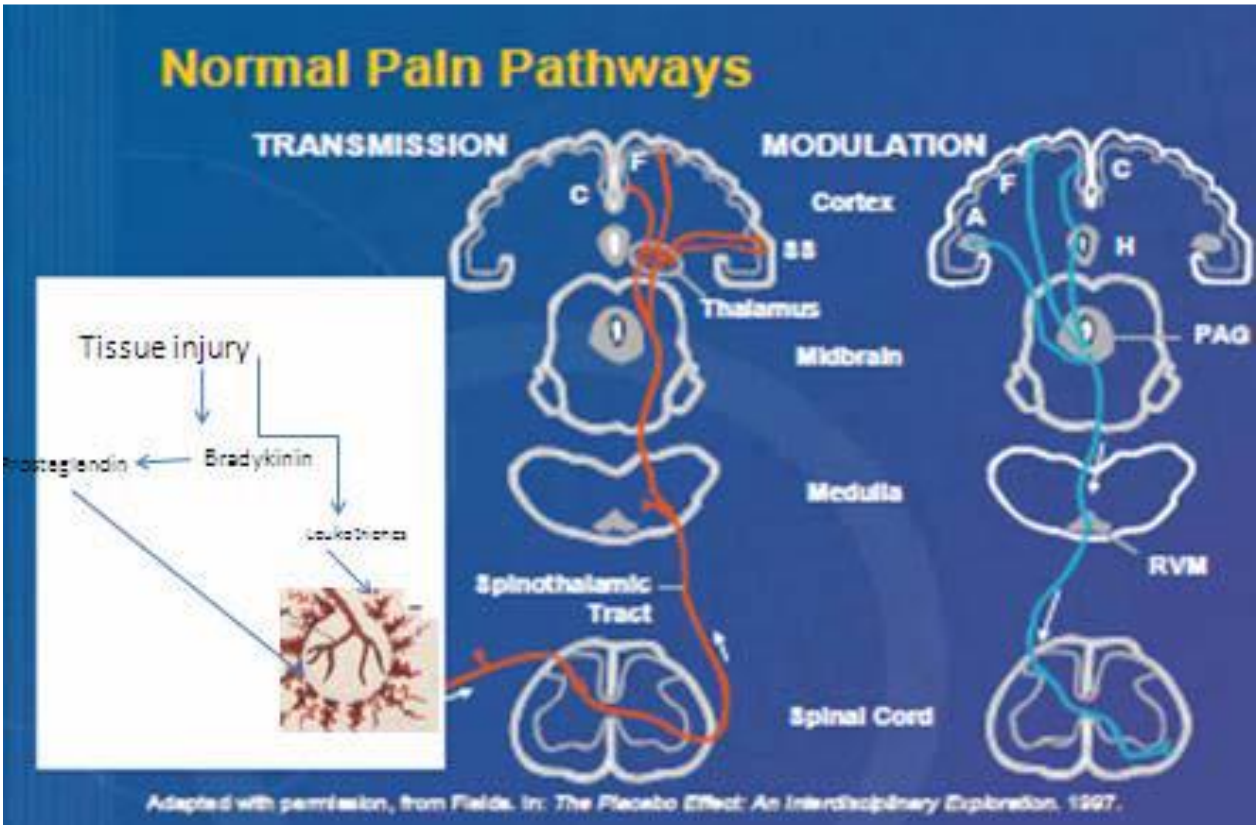


Fig.24 : Normal Pathways of Pain

Postoperative pain can be classified as acute pain and chronic pain.Acute pain is experienced immediately after surgery (less than 7 days) and pain that lasts more than 3 months after the injury is considered to be chronic pain.

Acute and chronic pain can arise from cutaneous, deep somatic or visceral structures.

Acute pain is of two types:

1. Somatic Pain:

a. Superficial somatic pain - arising from skin, subcutaneous tissue, mucous membrane. It is sharp pricking and well localized.

b. Deep somatic pain - arises from muscles, tendons, joint and bones. It has dull aching quality and less well localized. Both the intensity and duration of pain affects the degree of localization.

2. Visceral Pain:

It is due to disease or abnormal function of an internal organ or its covering which is poorly localized, dull and vague, may be colicky, cramping, or squeezing.

Multiple mechanisms are involved in the generation of nociception in patients who underwent laparoscopic cholecystectomy. These include the following causations: traumatic destruction of somatic free nerve endings secondary to abdominal incision, parietal peritoneal distention, disruption of visceral nerve endings in the gallbladder bed, release of endogenous inflammatory
cytokines, phrenic nerve irritation, irritation of peritoneum from blood, bile spillage, or by carbon dioxide, and somatoform or psychogenic causes

The major advantage of local instillation maybe related to its direct nociceptic inhibition of free nerve endings injured in the gallbladder bed, its gradual peritoneal absorption into the systemic circulation, and the lack of systemic toxicities associated with direct systemic administration of NSAIDs.(9)

ASSESSMENT OF PAIN

It is vital element in effective postoperative pain management. Specific pain assessment scales are used to quantify pain.The patient's own report is the most useful tool. The intensity of pain should therefore be assessed as far as possible by the patient as long as he/she is able to communicate and express what pain feels like.A number of different patient self-assessment scales are available.

Visual analogue scale (VAS) :

VAS is the most common used method to assess pain which was first described in 1966. It is a very simple scale, used in pain research. It is a 10 cm line with two anchor points of 'no pain' and 'worst pain imaginable' which is self assessed by patient. The position of the mark on the line measures how much pain the subject experiences.

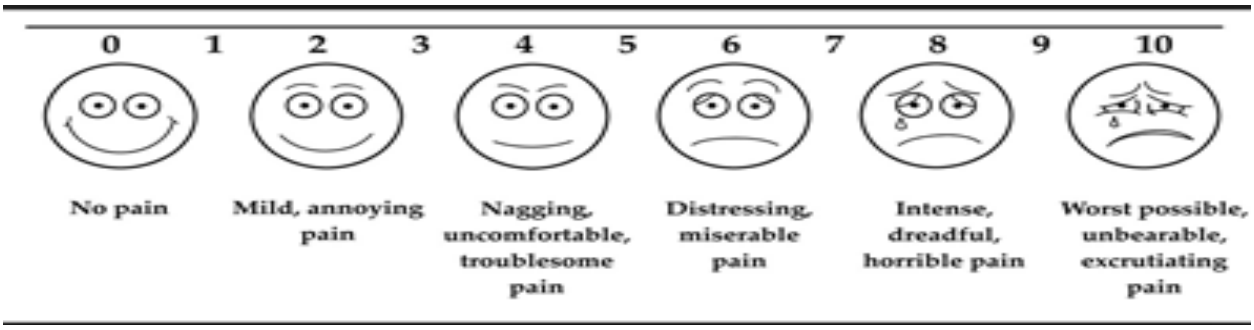


Fig.25: Wong-Baker Faces Pain Rating Scale and Visual Analogue Scale

Facial expressions:

A pictogram of six faces with different expressions from smiling or happy through to tearful. This scale suits for patients where communication is a problem, such as children, elderly patients, confused patients or patients who do not speak the local language.

Numerical rating scale (NRS):

It is similar to the visual analogue scale with the two anchors of ‘no pain’ and ‘worst pain as from 0 to 10 (making an 11-point scale), assessed by patient.

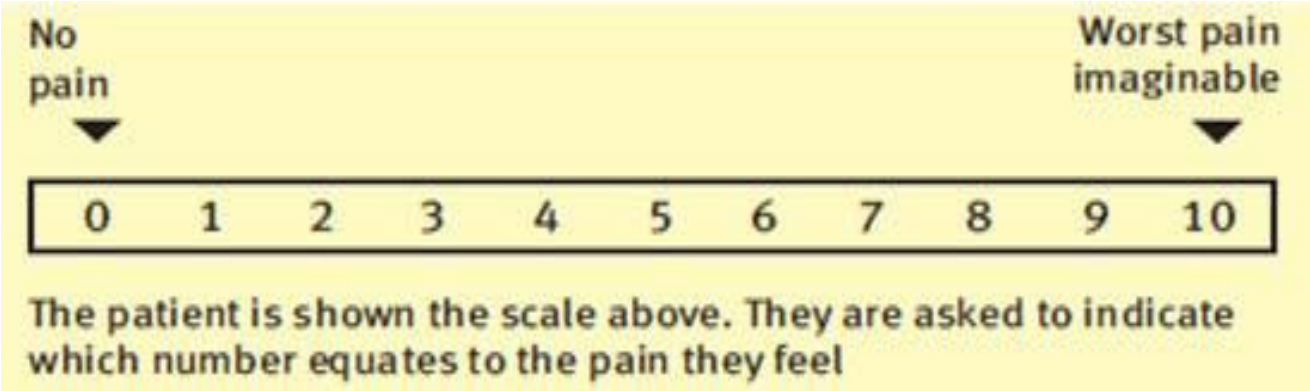


Fig.26 : Numerical Pain Scale

Verbal rating scale (VRS):

It consists of four points: no pain, mild pain, moderate pain, and severe pain. It is easy to use and can be used in the mildly cognitively impaired, but it is insensitive to small changes in pain intensity.

The preoperative personality assessment is also helpful in assessing the patient’s Psychological background and his psycho reactions to surgery and the pain that follows it.

The VRS and NRS are the most frequently used assessment tools in the clinical setting while the VAS scale is primarily used as a research tool.

PHARMACOLOGY OF BUPIVACAINE

Fig.27 : 0.25% Bupivacaine

Bupivacaine hydrochloride is an amino-amide local anaesthetic synthesized by EKENSTAM et al in 1957 and first used by L.J. Telivuo in 1963.It is available as racemic mixtures of the R & S enantiomers (50:50).(10)

PHYSICOCHEMICAL PROPERTIES

- Pk 8.1
- % Ionized at PH 7.4 83
- Partition Coefficient (Lipid Solubility) 3420
- % Protein binding 95

PHARMACOKINETICS

- Volume of distribution (l/Kg) 1.02
- Clearance (l/Kg/hr) 0.41
- T ½ (hour) 3.5

METABOLISM & CLEARANCE

Metabolism prinicipally by microsomal enzymes located in the liver.Pathways for metabolism of bupivacaine include aromatic hydroxylation,N-dealkylation, amide hydrolysis, and conjugation.Bupivacaine undergo renal excretion.

PHARMACODYNAMICS

EFFECT ON CARDIOVASCULAR SYSTEM

Bupivacaine produces dose related effect on the heart.

 In purkinje fibres and ventricular muscles - depresses the rapid phase of depolarization

 Bupivacaine has high arrhythmogenic potential because of incomplete restoration of sodium channel availability in between action potentials.

EFFECT ON CENTRAL NERVOUS SYSTEM

When bupivacaine given by intravenous infusion a general pattern of increasing signs and symptoms of toxicity is discernible.These signs and symptoms are Numbness of tongue and mouth, light headedness, tinnitus, visual disturbances, muscular twitching, irrational conversation, unconsciousness, grand-mal convulsions and apnea.

EFFECT ON RESPIRATORY SYSTEM

When bupivacaine is systemically absorbed it stimulates the ventilator response to carbon dioxide.

EFFECT ON GASTRO INTESTINAL SYSTEM

When bupivacaine is given as epidural infusion either continuously or intermittently is associated with increased plasma concentrations of transaminases enzyme.

DRUG DOSAGE

The maximal dose of bupivacaine is 2 - 2.5mg/kg.

Strength - 0.125% - 0.75% with or without epinephrine.

CLINICAL USES

1.Spinal anaesthesia

2. Epidural & Caudal anaesthesia

3.Continuous epidural anaesthesia

4. Peripheral nerve block

5. Infiltration anaesthesia

SIDE EFFECTS

1.Allergic reactions

2.Systemic toxicity due to excessive plasma and tissue concentrations.

LOCAL ANAESTHETIC SYSTEMIC TOXICITY

Local anaesthetic systemic toxicity (LAST) range from mild systemic symptoms to cardiovascular symptoms like hypertension, hypotension,tachycardia, bradycardia , ventricular arrhythmia leading to cardiac arrest and central nervous system symptoms like perioral numbness,seizure, respiratory depression,coma and death.

Magnitude of the problem depends on ,

1.Dose administered into the tissues

2. Vascularity of the injection site

3. Presence of epinephrine in the solution

4. Physicochemical properties of the drug.

TREATMENT

A. If signs and symptoms of LAST occur, then adequate and effective airway management is crucial to preventing hypoxia and acidosis, which are known to potentiate LAST.

B. If seizures occur, they should be rapidly halted with benzodiazepines. If benzodiazepines not available, small doses of propofol or thiopental are acceptable.

- Although propofol can stop seizures, large doses further depress cardiac function; propofol should be avoided when there are signs of cardiovascular compromise. If seizures persist inspite benzodiazepines, small doses of succinylcholine or similar neuromuscular blocker administered to minimize acidosis and hypoxemia.

- If cardiac arrest occurs, consider standard Advanced Cardiac Life Support with the following modifications:

- If epinephrine is used, small initial doses (10—100 ug boluses in the adult) are preferred.

- Vasopressin is not recommended.

- Avoid calcium channel blockers and ß-adrenergic receptor blockers.

- If ventricular arrhythmias develop, amiodarone is preferred.

C. Lipid emulsion therapy

- Administer at the first signs of LAST, after airway management

Dosing: 1.5 mL/kg 20% lipid emulsion bolus and 0.25 mL/kg per minute of infusion, continued for at least 10 minutes after circulatory stability is attained

- If circulatory stability is not achieved, consider re-bolus and increasing infusion to 0.5 mL/kg per minute.

- Approximately 10 mL/kg lipid emulsion for 30 minutes is recommended as the upper limit for initial dosing

- Propofol should not be used as a substitute for lipid emulsion.

- Failure of response to lipid emulsion and vasopressor therapy should prompt institution of cardiopulmonary bypass (CPB). Because there can be considerable lag in

beginning CPB, it is reasonable to notify the closest facility capable of providing it when cardiovascular compromise is first identified during an episode of LAST.(10)

PROFORMA

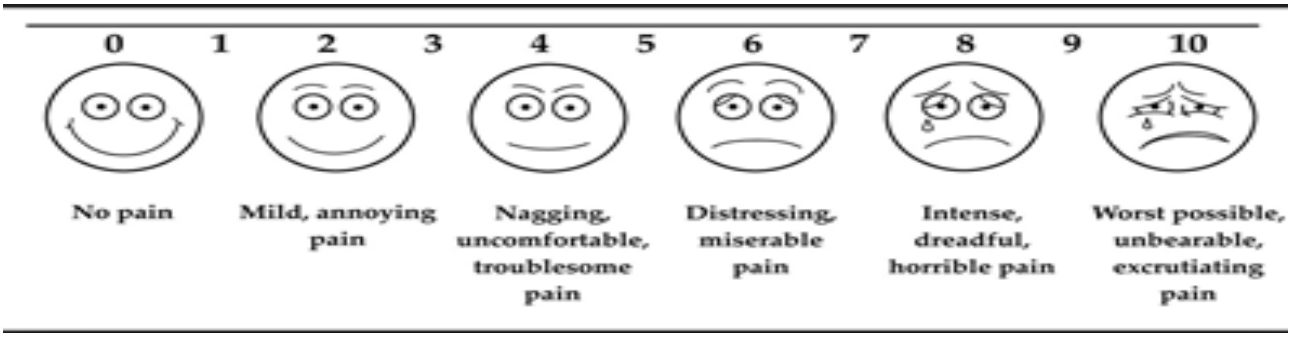
NAME :

AGE/SEX :

IP.NO :

DURATION OF SURGERY :

VISUAL ANALOG SCALE :



PAIN AFTER SURGERY AT VARIOUS INTERVALS :

GROUP	30 MINS AFTER SURGERY	1HOUR AFTER SURGERY	2HOURS AFTER SURGERY	3HOURS AFTER SURGERY	4HOURS AFTER SURGERY	6HOURS AFTER SURGERY	12HOURS AFTER SURGERY	24HOURS AFTER SURGERY
CONTROL								
STUDY								

RESCUE ANALGESIA NEEDED :

MATERIALS AND METHODS

PLACE OF STUDY

Department of general surgery –Govt.Stanley medical college &hospital

DURATION

1 Year

STUDY DESIGN

Randomised control trial

SAMPLE SIZE: 60

Control group(A)-30 (Those who receive only trocar site bupivacaine)

Study group(B)-30 (Those who receive both trocar site and gallbladder bed bupivacaine)

INCLUSION CRITERIA:

- Patients undergoing elective laparoscopic cholecystectomy
- ASA I or II physical status
- Age 15-60

EXCLUSION CRITERIA:

- Patients undergoing emergency laparoscopic cholecystectomy
- ASA III or IV Physical status
- Age <15 and >60
- Those who cannot understand VAS.

METHODOLOGY:

- To obtain written informed consent from all subjects before enrolment in the study.

- To provide knowledge aboutVAS to all patients.

- To randomize the patients into two groups of 30 patients each,control and study group.

- In this study all patients will undergo standard operative method with a 4-trocar technique and achieve pneumoperitoneum with use of Veress needle through a periumbilical incision and maintain at 14mm Hg during entire surgical procedure.

- After removal of gallbladder,secure hemostasis at the surgical bed.

- Insert feeding tube through right subcoastal port and irrigate surgical bed with 20ml of normal saline in control group and 20ml of 0.25%(50mg) bupivacaine in study group.

- Keep the patient in Trendelenburg position with right lateral tilt to facilitate dispersion of drug solution in the sub-hepatic region for 5 minutes,then remove gas,instruments and trocars.

- DT is not used in both control and study groups.

- Inject 20ml of 0.25% bupivacaine subcutaneously to trocar sites for all patients.

- To assess the postoperative pain using VAS scale at 1,2,3,4,6,12 and 24 hours in the postoperative ward.

- If patient experiences pain equal to or more than 5 in VAS scale,give inj.Tramadol 2mg/kg.

- The length of time between extubation and the first request of analgesia to be noted-Rescue Analgesia Needed

- To compare the postoperative pain between two groups at different time intervals

ANALYSIS AND RESULTS

Statistical analysis was performed using the Microsoft (MS) Office Excel Software (Microsoft Excel, Redmond, Washington: Microsoft, Computer software). Results were expressed as mean,variance, number and percentage (%). Data were analysed using post-hoc analysis method.

Normally, distributed data were assessed using unpaired student's t-test (for comparison of parameters among groups). Comparison was carried out using Chi-square test with a P value reported at 95% confidence level. Level of significance used was P= 0.05

AGE DISTRIBUTION

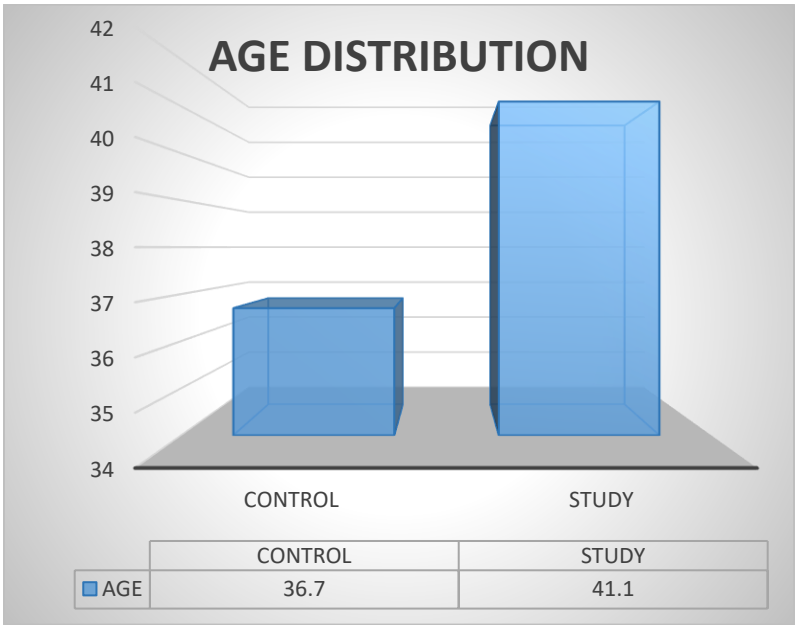


Fig.28: Age distribution

Cholelithiasis is characteristically a disease of middle-aged.The mean age in our study was 36.7 years(control) and 41.1 years(study).

GENDER DISTRIBUTION

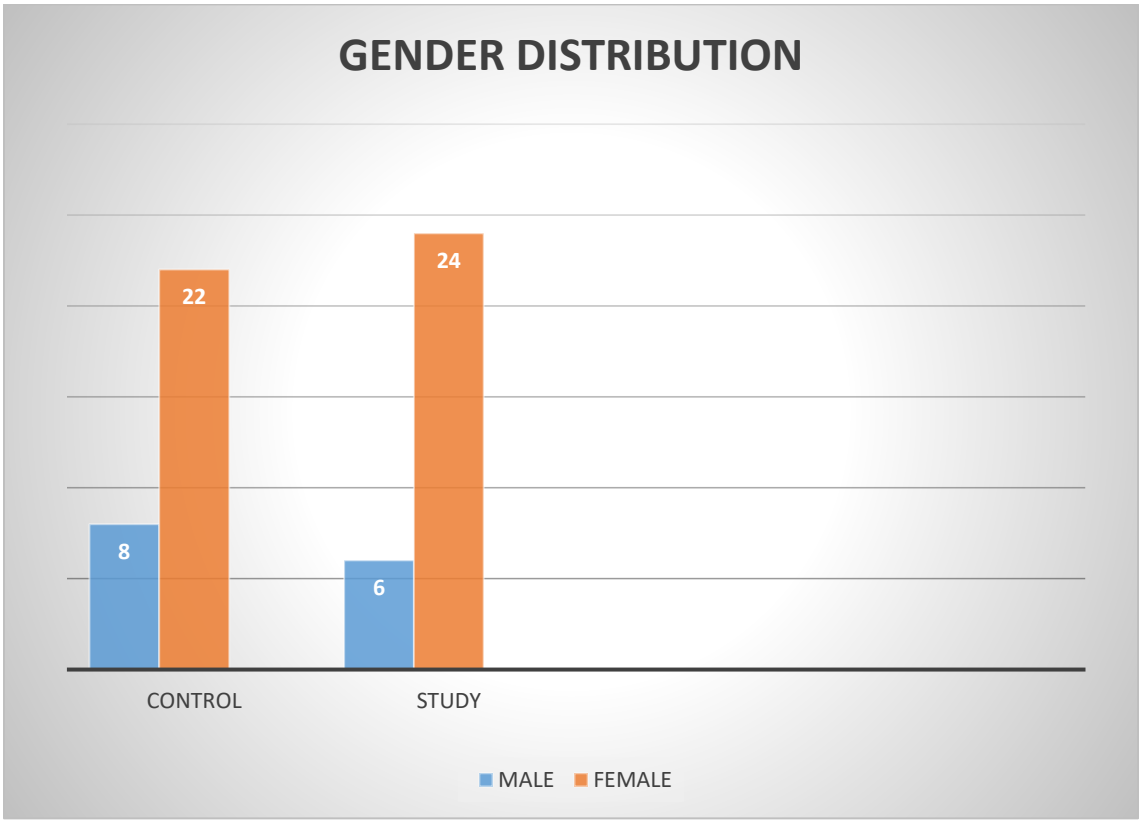


Fig.29: Gender distribution

The number of females in the study was more compared to that of males in both the groups; overall this may be explained by the fact that the disease has a female preponderance.

DURATION OF SURGERY

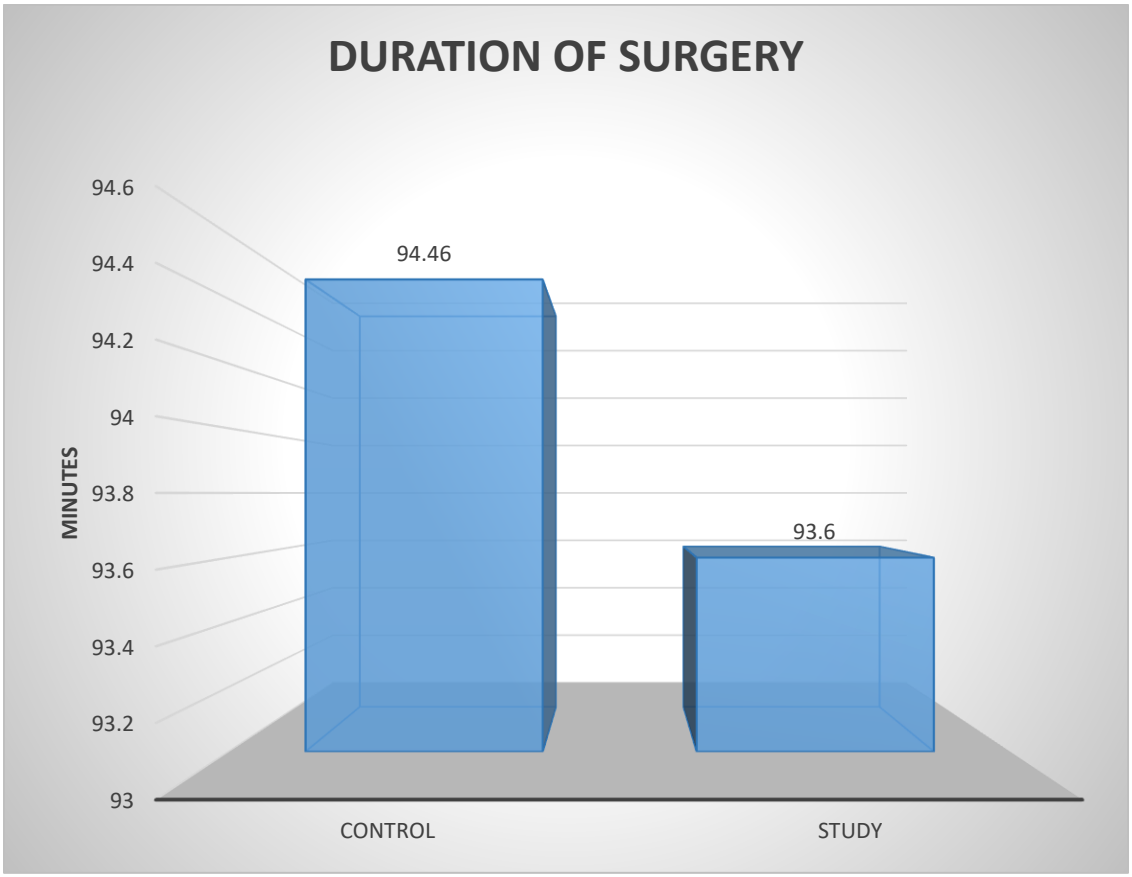
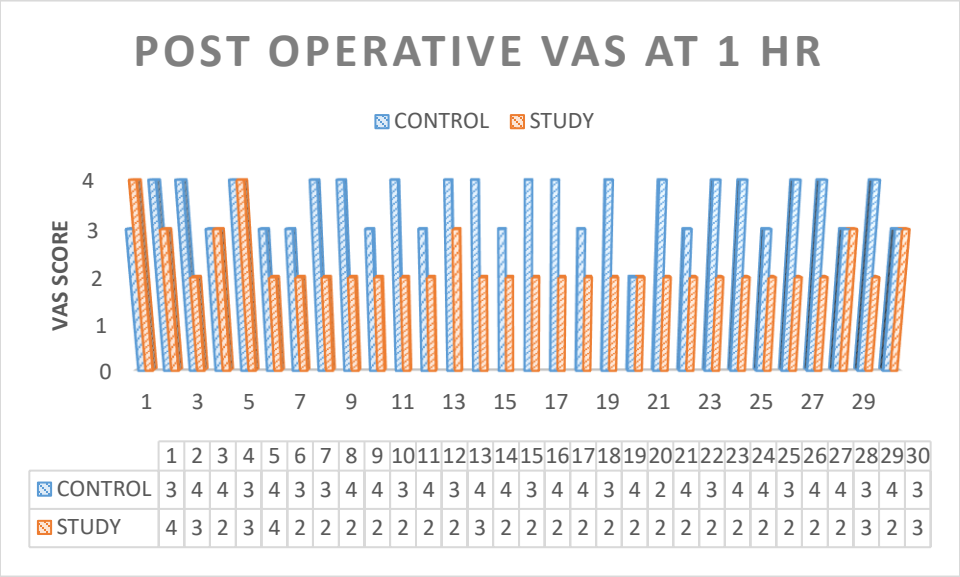


Fig.30:Duration of surgery

The duration of surgery in both groups is comparable, control (94.46mins) and study (93.6mins) indicates that there is no delay in surgery because of instillation of bupivacaine intraperitoneally.

POST OPERATIVE VAS AT DIFFERENT INTERVALS



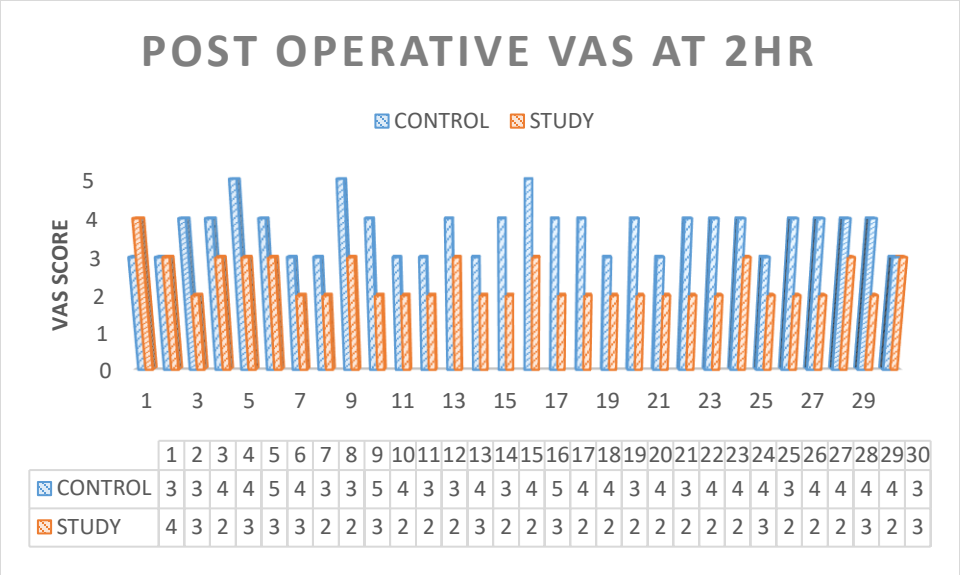
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
CONTROL	3	4	4	3	4	3	3	4	4	3	4	3	4	4	3	4	4	3	4	2	4	3	4	4	3	4	4	3	4	3
STUDY	4	3	2	3	4	2	2	2	2	2	2	2	3	2	2	2	2	2	2	2	2	2	2	2	2	2	2	3	2	3

Fig.31: Postoperative VAS at 1hr

t-Test: Two-Sample Assuming Unequal Variances

	CONTROL	*STUDY*
Mean	3.533333333	2.3
Variance	0.326436782	0.355172414
Observations	30	30
Hypothesized Mean Difference	0	
df	58	
t Stat	8.182261899	
P(T<=t) one-tail	1.5166E-11	
t Critical one-tail	1.671552762	
P(T<=t) two-tail	3.0332E-11	
t Critical two-tail	2.001717484	

The post operative pain measured by visual analog scale at 1 hr showed mean value of 3.5 (control) and 2.3 (study) ,variance of 0.32 (control) and 0.35 (study) , p-value of <0.05 which is significant.



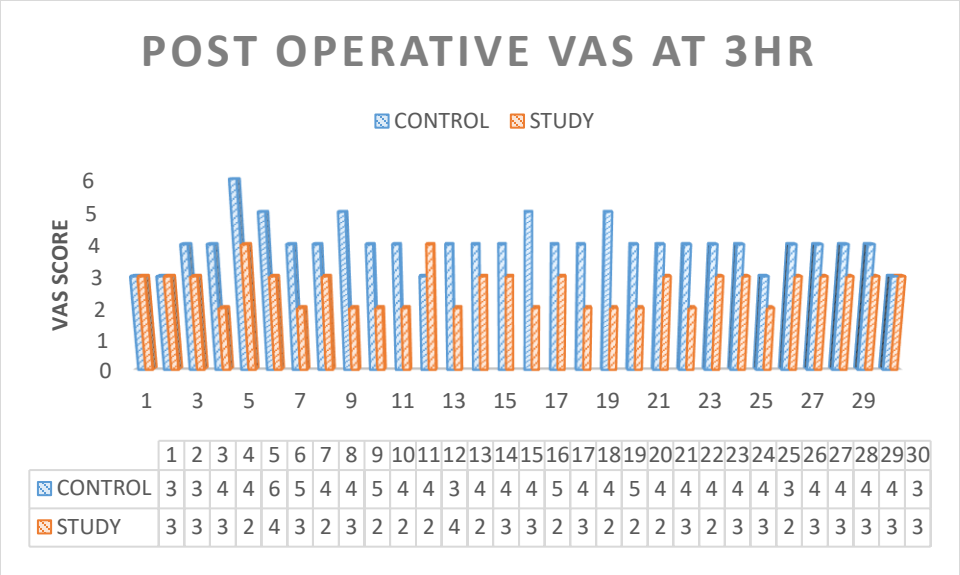
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
CONTROL	3	3	4	4	5	4	3	3	5	4	3	3	4	3	4	5	4	4	3	4	3	4	4	4	3	4	4	4	4	3
STUDY	4	3	2	3	3	3	2	2	3	2	2	2	3	2	2	3	2	2	2	2	2	2	2	3	2	2	2	3	2	3

Fig.32: Postoperative VAS at 2hr

t-Test: Two-Sample Assuming Unequal Variances

	CONTROL	*STUDY*
Mean	3.733333333	2.4
Variance	0.409195402	0.317241379
Observations	30	30
Hypothesized Mean Difference	0	
df	57	
t Stat	8.568414178	
P(T<=t) one-tail	3.94123E-12	
t Critical one-tail	1.672028888	
P(T<=t) two-tail	7.88246E-12	
t Critical two-tail	2.002465459	

The post operative pain measured by visual analog scale at 2 hr showed mean value of 3.7 (control) and 2.4 (study) ,variance of 0.4 (control) and 0.31(study) ,p-value of <0.05 which is significant.



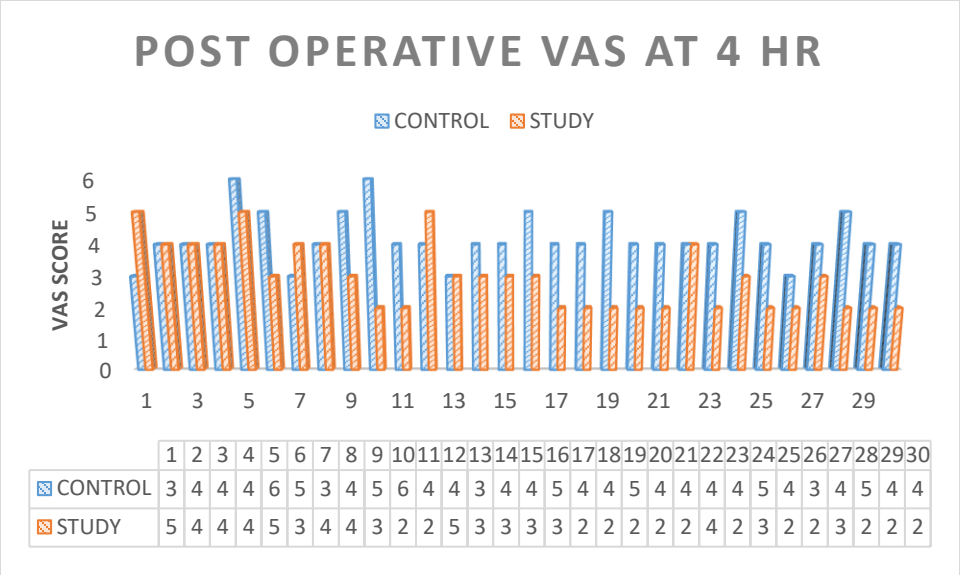
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
CONTROL	3	3	4	4	6	5	4	4	5	4	4	3	4	4	4	5	4	4	5	4	4	4	4	4	3	4	4	4	4	3
STUDY	3	3	3	2	4	3	2	3	2	2	2	4	2	3	3	2	3	2	2	2	3	2	3	3	2	3	3	3	3	3

Fig.33: Postoperative VAS at 3hr

t-Test: Two-Sample Assuming Unequal Variances

	CONTROL	*STUDY*
Mean	4.033333333	2.666666667
Variance	0.447126437	0.367816092
Observations	30	30
Hypothesized Mean Difference	0	
df	57	
t Stat	8.29200849	
P(T<=t) one-tail	1.12718E-11	
t Critical one-tail	1.672028888	
P(T<=t) two-tail	2.25436E-11	
t Critical two-tail	2.002465459	

The post operative pain measured by visual analog scale at 3 hr showed mean value of 4 (control) and 2.6 (study) ,variance of 0.44 (control) and 0.36 (study) ,p-value of <0.05 which is significant.



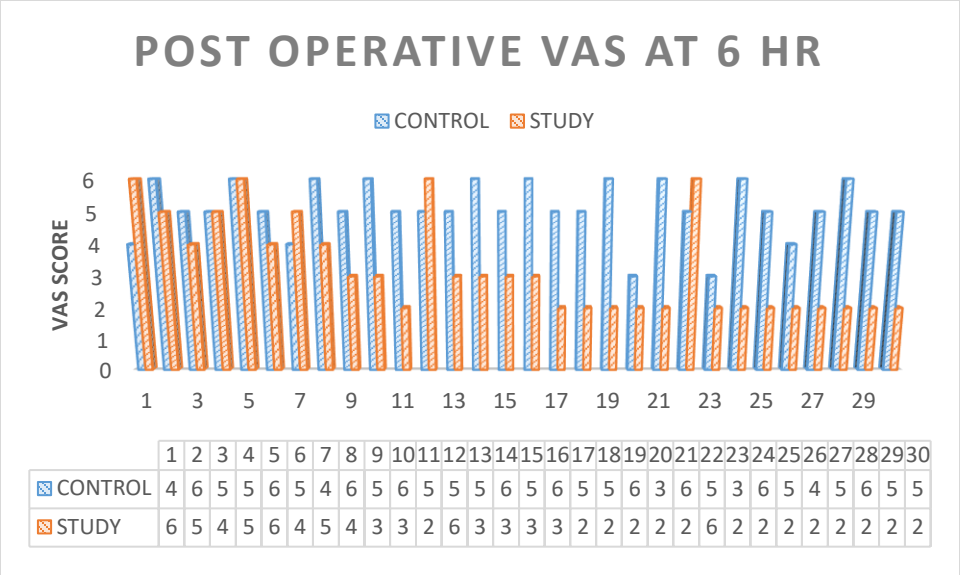
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
CONTROL	3	4	4	4	6	5	3	4	5	6	4	4	3	4	4	5	4	4	5	4	4	4	4	5	4	3	4	5	4	4
STUDY	5	4	4	4	5	3	4	4	3	2	2	5	3	3	3	3	2	2	2	2	2	4	2	3	2	2	3	2	2	2

Fig.34: Postoperative VAS at 4hr

t-Test: Two-Sample Assuming Unequal Variances

	CONTROL	*STUDY*
Mean	4.2	2.966666667
Variance	0.579310345	1.067816092
Observations	30	30
Hypothesized Mean Difference	0	
df	53	
t Stat	5.263536366	
P(T<=t) one-tail	1.30424E-06	
t Critical one-tail	1.674116237	
P(T<=t) two-tail	2.60849E-06	
t Critical two-tail	2.005745995	

The post operative pain measured by visual analog scale at 4 hr showed mean value of 4.2 (control) and 2.9 (study) ,variance of 0.57 (control) and 1.06 (study) ,p-value of <0.05 which is significant.



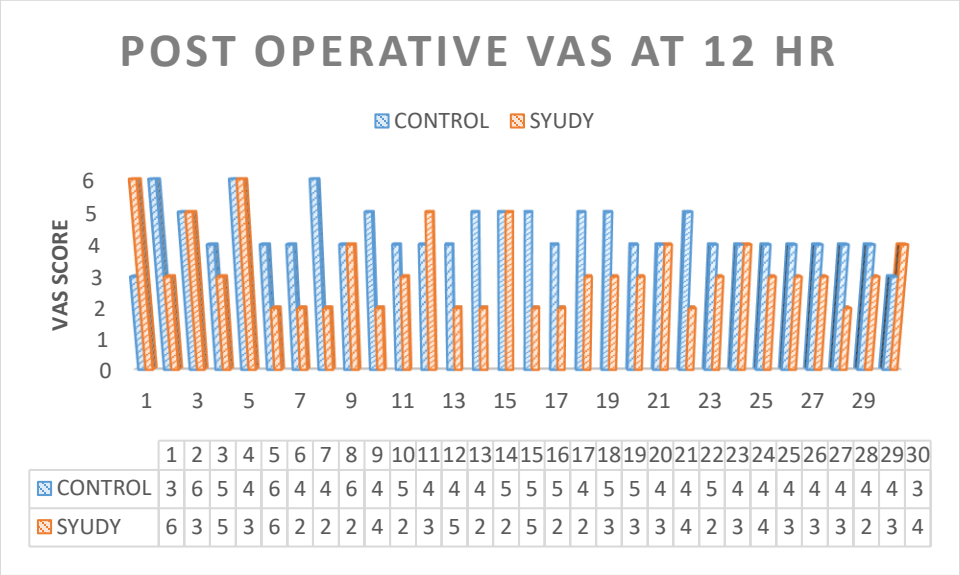
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
CONTROL	4	6	5	5	6	5	4	6	5	6	5	5	5	6	5	6	5	5	6	3	6	5	3	6	5	4	5	6	5	5
STUDY	6	5	4	5	6	4	5	4	3	3	2	6	3	3	3	3	2	2	2	2	2	6	2	2	2	2	2	2	2	2

Fig.35: Postoperative VAS at 6hr

t-Test: Two-Sample Assuming Unequal Variances

	CONTROL	*STUDY*
Mean	5.1	3.233333333
Variance	0.713793103	2.185057471
Observations	30	30
Hypothesized Mean Difference	0	
df	46	
t Stat	6.005020369	
P(T<=t) one-tail	1.42301E-07	
t Critical one-tail	1.678660414	
P(T<=t) two-tail	2.84602E-07	
t Critical two-tail	2.012895599	

The post operative pain measured by visual analog scale at 6 hr showed mean value of 5.1 (control) and 3.2 (study) ,variance of 0.71 (control) and 2.18 (study) ,p-value of <0.05 which is significant.



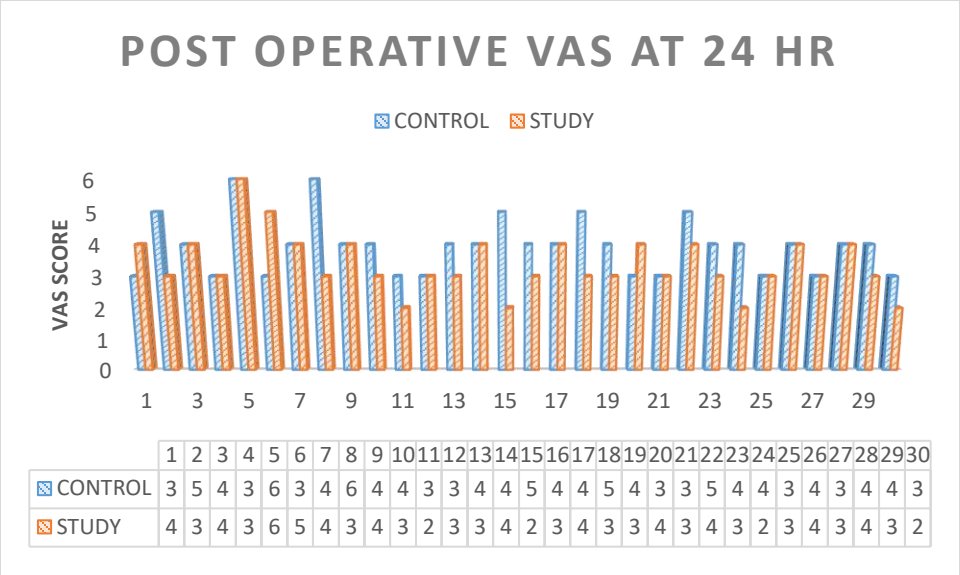
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
CONTROL	3	6	5	4	6	4	4	6	4	5	4	4	4	5	5	5	4	5	5	4	4	5	4	4	4	4	4	4	4	3
SYUDY	6	3	5	3	6	2	2	2	4	2	3	5	2	2	5	2	2	3	3	3	4	2	3	4	3	3	3	2	3	4

Fig.36: Postoperative VAS at 12hr

t-Test: Two-Sample Assuming Unequal Variances

	CONTROL	*SYUDY*
Mean	4.4	3.2
Variance	0.593103448	1.475862069
Observations	30	30
Hypothesized Mean Difference	0	
df	49	
t Stat	4.569463864	
P(T<=t) one-tail	1.6631E-05	
t Critical one-tail	1.676550893	
P(T<=t) two-tail	3.32619E-05	
t Critical two-tail	2.009575237	

The post operative pain measured by visual analog scale at 12 hr showed mean value of 4.4 (control) and 3.2 (study) ,variance of 0.59 (control) and 1.47 (study) ,p-value of <0.05 which is significant.



	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
CONTROL	3	5	4	3	6	3	4	6	4	4	3	3	4	4	5	4	4	5	4	3	3	5	4	4	3	4	3	4	4	3
STUDY	4	3	4	3	6	5	4	3	4	3	2	3	3	4	2	3	4	3	3	4	3	4	3	2	3	4	3	4	3	2

Fig.37: Postoperative VAS at 24hr

t-Test: Two-Sample Assuming Unequal Variances

	CONTROL	*STUDY*
Mean	3.933333333	3.366666667
Variance	0.754022989	0.791954023
Observations	30	30
Hypothesized Mean Difference	0	
df	58	
t Stat	2.496242529	
P(T<=t) one-tail	0.007707843	
t Critical one-tail	1.671552762	
P(T<=t) two-tail	0.015415685	
t Critical two-tail	2.001717484	

The post operative pain measured by visual analog scale at 24 hr showed mean value of 3.9 (control) and 3.3 (study) ,variance of 0.75 (control) and 0.79 (study) ,p-value of <0.05 which is significant.

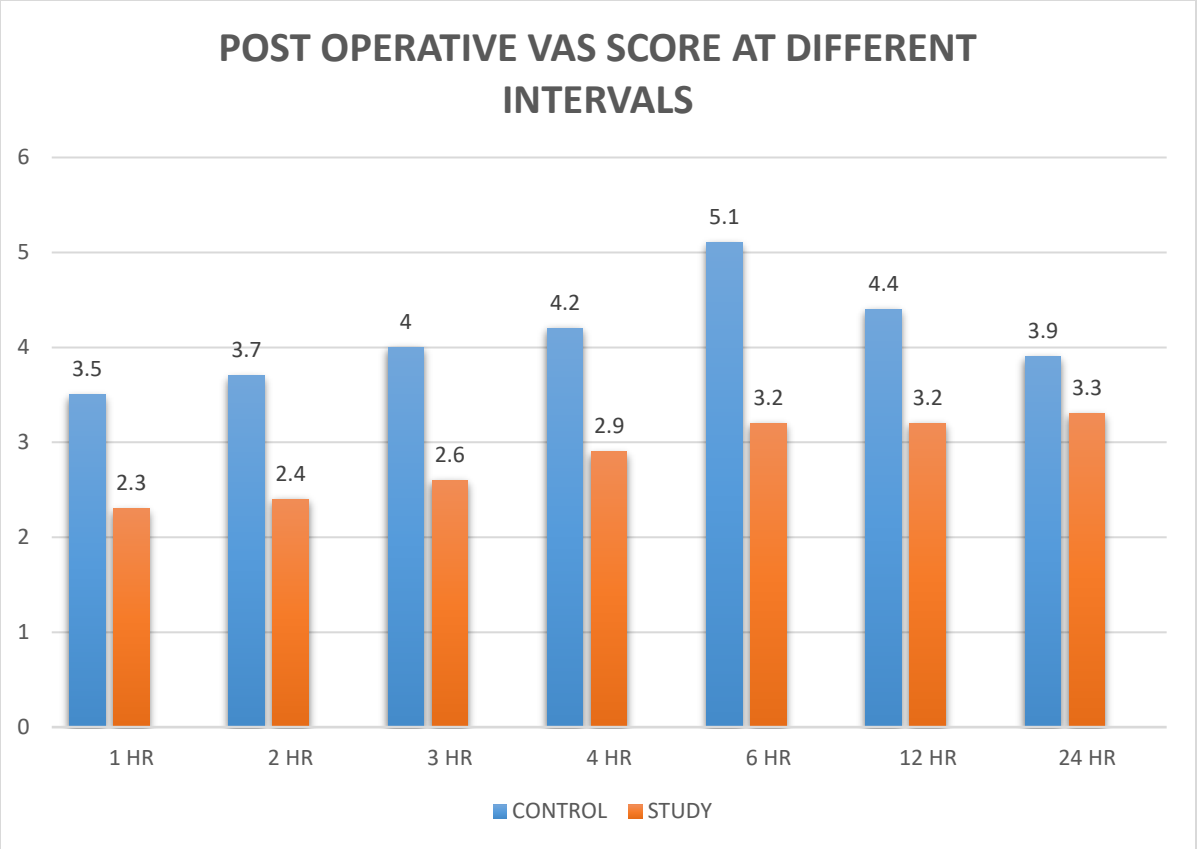


Fig.38: Postoperative VAS at different intervals

MEAN	**1 HR**	**2 HR**	**3 HR**	**4 HR**	**6 HR**	**12 HR**	**24 HR**
CONTROL	3.5	3.7	4	4.2	5.1	4.4	3.9
STUDY	2.3	2.4	2.6	2.9	3.2	3.2	3.3
VARIANCE							
CONTROL	0.32	0.40	0.44	0.57	0.71	0.59	0.75
STUDY	0.35	0.31	0.36	1.06	2.18	1.47	0.79
P-VALUE	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	0.015
REMARKS	*SIGNIFICANT*	*SIGNIFICANT*	*SIGNIFICANT*	*SIGNIFICANT*	*SIGNIFICANT*	*SIGNIFICANT*	*SIGNIFICANT*

From the above table it is clear that in our study group patients has decreased postoperative pain when compared to control group which is statistically significant.

RESCUE ANALGESIA NEEDED

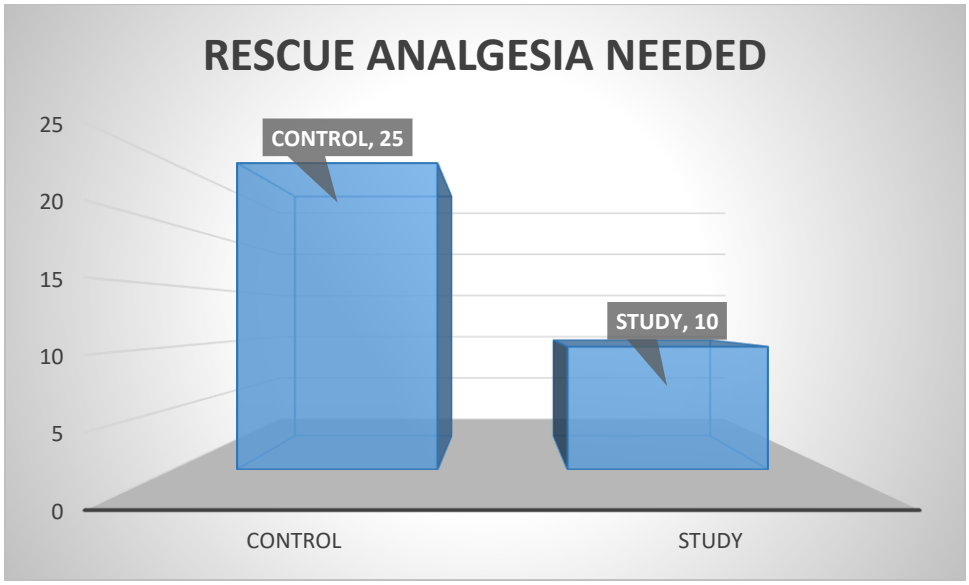


Fig.39: Rescue analgesia needed

Out of 60 patients,35 patients needed rescue analgesia ,of them 25 were control group and 10 were study group.

DISCUSSION

Laparoscopic cholecystectomy is a part of day case surgery hence adequate analgesia and early recovery is of utmost importance. Postoperative pain is the most common complications of laparoscopic surgery. The pain is to the maximum within 6 hour of the procedure then it gradually decreases over a couple of days, but it varies considerably between patients.(12)

The peritoneum is the serous membrane that covers the abdominal cavity and most of the intra-abdominal organs. It is a very thin layer highly vulnerable to damage and it is not designed to cope with variable conditions such as the dry and cold carbon dioxide. During laparoscopic surgery, postoperative pain is multifactorial in origin,can be somatic pain at port site (port site pain) and visceral pain which is caused by residual carbon dioxide in the peritoneal cavity. Visceral pain is mainly due to stretching of the visceral peritoneum and peritoneal inflammation and phrenic nerve irritation .(13)

The accurate pain assessment is difficult because of its individual threshold, subjectivity, and difficulty in measurement. In this study we compared the post-operative pain relief in laparoscopic cholecystectomy cases using intra peritoneal Bupivacaine 0.5% and saline and found that IP administration of Inj. bupivacaine in LC is a *safe* method and offers significant pain relief and significant decrease in requirement of analgesics. (14)

The main advantage of using local anaesthetics is that they do not have the adverse effects of opioids, which may delay recovery and discharge from hospital. These effects include post- operative nausea, sedation, impairment of return of gastrointestinal motility, and pruritus. In addition, time to return of bowel function in thepostoperative period may be reduced when the use of opioids is obviated by administering local anaesthetics.

In the present study majority of the patients were in the middle age group. The mean age in our study was 36.7 years(control) and 41.1 years(study) because of the prevalence of the disease in middle age group.

The number of females in the study was more compared to that of males in both groups; overall this may be explained by the fact that the disease has a female preponderance Cholelithiasis is characteristically a disease of middle-aged women.In a study by Novacek showed that female gender is the most important risk factors and the rates of gallstones are two to three times higher among women than men which correlates with the present study.(17)

The duration of surgery in both groups is comparable, control(94.46mins) and study(93.6mins) indicates that there is no delay in surgery because of instillation of bupivacaine intraperitoneally.

In our study there was significant pain relief in the first 24 hours postoperatively by intraperitoneal bupivacaine after taking care of the port site pain by injection bupivacaine with no significant toxicity.

Boddy AP et al., conducted a meta analysis on the efficacy of intraperitoneal local anaesthetics in relieving post-laparoscopic cholecystectomy pain, and found intraperitoneal local anaesthetics to be efficient in relieving postoperative pain without side effect of analgesic toxicity.

Yari M et al., in their study demonstrated that intraperitoneal administration of bupivacaine has reduced both the visceral and shoulder pains at 4^{th} postoperative hour but had no effect on the reduction of rescue analgesic (opioids) . In contrast, our study showed a significant decrease in visceral pain relief after LC in the first 24 hours postoperatively and also there was a significant reduction in the requirement of rescue analgesia.

Our study yielded results comparable to a similar study done by Yadava A et al., where they had found significant decrease in mean pain VAS score by using intraperitoneal bupivacaine and tramadol after LC. In their study they had also added IP magnesium sulphate (MgSO4) to bupivacaine and had concluded that addition of MgSO4, by antagonising N-methyl-D-aspartate (NMDA) receptors and thereby reducing neuronal signalling as well as pain processing in the central nervous system, resulted in longer duration of pain-free period and less consumption of rescue analgesics in postoperative period compared to intraperitoneal instillation of tramadol with bupivacaine.(16)

On the contrary Shukla U et al., found that addition of IP instillation of dexmedetomidine 1 microgm/kg; which blocks the release of substance *P* in the nociceptive pathway at dorsal root neuron level; to bupivacaine 0.25% in elective LC is superior to bupivacaine alone and maybe better than bupivacaine and tramadol for postoperative pain relief.

Govil N et al., conducted a study designed to study the effect of intraperitoneal instillation of levobupivacaine along with clonidine for pain relief after LC . They concluded that intraperitoneal levobupivacaine produces postoperative analgesia better than what was obtained with intraperitoneal placebo and the combination of intraperitoneal levobupivacaine and clonidine is superior to the plain levobupivacaine for the management of postoperative pain in patients undergoing laparoscopic cholecystectomy. These findings corroborated with our study of intraperitoneal bupivacaine after LC had shown to reduce

postoperative pain significantly, however we did not add intraperitoneal clonidine to bupivacaine.

Narchi et al in his study found intraperitoneal local anaesthetics to be more effective in reducing pain upto 48 hours postoperatively in patients undergoing diagnostic laparoscopy.(19)

Utilizing 20 ml of either 0.25% bupivacaine or 0.5% lignocaine, Rademaker et al failed to demonstrate any reduction in postoperative pain. A
possible explanation of the failed effect given by them was the instillation of local anaesthetics in the supine position prevented its flow over the coeliac plexus and phernic nerve endings.(20)

Keeping in view that the importance of positioning during instilling the local anaesthetic Scheinin et al administered 100 ml of either 0.15% plain bupivacaine or with adrenaline in head down tilt maintained for 20minutes.They found no relief in pain after laparoscopic cholecystectomy. The lack of analgesic efficacy can be attributed to the lower concentration of bupivacaine used and more extensive and longer duration of surgery compared to gynaecological laparoscopies.(15)

The good results of our study may be related to the use of higher concentration of bupivacaine compared to other studies because it is the concentration which may be important in laparoscopic cholecystectomy rather than volume. Also the drug was instilled in trendlenberg position so as to encourage its accumulation in gall bladder bed.

In the present study found that there was a significant difference in the study groups with respect to the time for intake of rescue analgesic consumption. Similar results were obtained by Chundrigar et al in their study. Chundrigar et al noted pain relief upto 2 hours post op with the intraperitoneal administration of 0.25% Bupivacaine, although in the present study we could note pain relief upto 24 hours post op. This may be due to the fact that we instilled the local anaesthetic in the trendelenburg position at the end of surgery which may have resulted in better dispersion of the drug and hence the beneficial effect upto 24 hour post op. (18)

Pasqualucci et al also noted significant difference in analgesic consumption between the groups up to 24 hrs.(21)

The results of the present study demonstrate that intraperitoneal instillation of bupivacaine significantly lower the intensity of postoperative pain and produces lower VAS upto 24 hours postoperatively. The
postoperative analgesic requirements are also less.

CONCLUSION

In conclusion, irrigation with bupivacaine in the surgical bed in laparoscopic cholecystectomy will significantly lowers the intensity of postoperative visceral pain, as well as the analgesic consumption in the initial postsurgical hours, thereby decreasing the burden both to the patient as well as to the hospital.Therefore, we can establish this protocol for use in laparoscopic cholecystectomies with the purpose of faster return of the patient to their normal life, and thus, a
shorter hospital stay. Finally, bupivacaine at the dosage used was very safe and had no significant side effects. Therefore,we can reduce the pain in patients who undergo laparoscopic cholecystectomy in the ambulatory centers.

MASTER CHART

S.NO	NAME	AGE/SEX	IP.NO	DURATION OF SURGERY	VAS (PAIN AFTER SURGERY AT VARIOUS INTERVAL	1 HR	2 HR	3 HR	4 HR	6 HR	12 HR	24 HR	RESCUE ANALGESIA NEEDED
1	SANGEETHA	33/F	1878382	89		3	3	3	3	4	3	3	NO
2	PUNITHA	45/F	1886448	120		4	3	3	4	6	6	5	YES
3	SNEHA	25/F	1886786	93		4	4	4	4	5	5	4	YES
4	MEGHALA	37/F	1885543	97		3	4	4	4	5	4	3	YES
5	MASTHAN BEE	58/F	1904779	105		4	5	6	6	6	6	6	YES
6	LATHA	37/F	1916930	119		3	4	5	5	5	4	3	YES
7	PRAMILA	38/F	1916930	84		3	3	4	3	4	4	4	NO
8	ROJA	52/F	1916125	110		4	3	4	4	6	6	6	YES
9	VIJAYAKUMARI	31/F	1917830	75		4	5	5	5	5	4	4	YES
10	KAMSALA	58/F	1916094	104		3	4	4	6	6	5	4	YES
11	BHUVANESWARI	27/F	1929635	85		4	3	4	4	5	4	3	YES
12	THIYAGARAJAN	36/M	1930556	79		3	3	3	4	5	4	3	YES
13	RAHMATH BEEVI	50/F	1925489	93		4	4	4	3	5	4	4	YES
14	PARAMESHWARI	50/F	1932718	98		4	3	4	4	6	5	4	YES
15	SUMITHRA	26/F	1903810	89	CONTROL GROUP	3	4	4	4	5	5	5	YES
16	DHANASEKAR	28/M	1935670	77		4	5	5	5	6	5	4	YES

17	JAWAHAR SINHA	30/M	1936929	98		4	4	4	4	5	4	4	YES
18	CHANDRALEKHA	27/F	1944695	103		3	4	4	4	5	5	5	YES
19	SURIYAGANDHI	50/F	1949089	113		4	3	5	5	6	5	4	YES
20	MONISHA	19/F	1955093	75		2	4	4	4	3	4	3	NO
21	GUNASEKAR	30/M	1952331	84		4	3	4	4	6	4	3	YES
22	UMAMAHESWARI	40/F	1951953	96		3	4	4	4	5	5	5	YES
23	WILSON	16/M	1940241	84		4	4	4	4	3	4	4	NO
24	VIJAYALAKHSMI	36/F	1956045	99		4	4	4	5	6	4	4	YES
25	SARANYA	25/F	1945573	83		3	3	3	4	5	4	3	YES
26	ARUMUGAM	52/M	1940188	98		4	4	4	3	4	4	4	NO
27	SAMEENA	29/F	1945565	96		4	4	4	4	5	4	3	YES
28	ANANDHAN	50/M	1941248	108		3	4	4	5	6	4	4	YES
29	VALARMATHI	35/F	1947284	101		4	4	4	4	5	4	4	YES
30	JAHANGIR	33/M	1941806	79		3	3	3	4	5	3	3	YES

S.NO	NAME	AGE/ SEX	IP.NO	DURATION OF SURGERY(MINS)	VAS (PAIN AFTER SURGERY AT VARIOUS INTERVAL	1 HR	2 HR	3 HR	4 HR	6 HR	12 HR	24 HR	RESCUE ANALGESIA NEEDED
1	BOOBALAN	42/M	1878392	88		4	4	3	5	6	6	4	YES
2	JOYCE SULOCHANA	33/F	1885783	98		3	3	3	4	5	3	3	YES

3	FAIZEN BEE	55/F	1886548	105		2	2	3	4	4	5	4	YES
4	INDIRANI	45/F	1886996	78		3	3	2	4	5	3	3	YES
5	KAMATCHI	50/F	1900834	107		4	3	4	5	6	6	6	YES
6	GOVINDHASAMY	56/M	1906531	68		2	3	3	3	4	2	5	YES
7	KALYANI	50/F	1906745	75		2	2	2	4	5	2	4	YES
8	RAJALAXMI	18/F	1909693	94		2	2	3	4	4	2	3	NO
9	NANDHINI	24/F	1911191	113		2	3	2	3	3	4	4	NO
10	RAZIYA BANU	41/F	1913350	84		2	2	2	2	3	2	3	NO
11	PREMA	41/F	1919764	97		2	2	2	2	2	3	2	NO
12	BABY	55/F	1922801	93		2	2	4	5	6	5	3	YES
13	MANJULA	48/F	1925805	102		3	3	2	3	3	2	3	NO
14	MUTHULAKSHMI	45/F	1926228	88		2	2	3	3	3	2	4	NO
15	SAIYATH SULAIMAN	29/M	1933116	85		2	2	3	3	3	5	2	YES
16	ABDUL RAZAK	40/M	1933087	95	STUDY GROUP	2	3	2	3	3	2	3	NO
17	VENKATESAN	51/M	1934679	108		2	2	3	2	2	2	4	NO
18	DEVAKUMAR	49/M	1936006	89		2	2	2	2	2	3	3	NO
19	PADMINI	50/F	1938136	75		2	2	2	2	2	3	3	NO
20	RAMANI	26/F	1936641	95		2	2	2	2	2	3	4	NO
21	KAMALA	57/F	1938605	107		2	2	3	2	2	4	3	NO
22	KALAISELVI	38/F	1937929	89		2	2	2	4	6	2	4	YES
23	SUMATHI	30/F	1937367	89		2	2	3	2	2	3	3	NO
24	MEGHABURNISHA	50/F	1943730	94		2	3	3	3	2	4	2	NO
25	SABURNISHA	26/F	1943282	118		2	2	2	2	2	3	3	NO
26	JAISHEELI	36/F	1945253	78		2	2	3	2	2	3	4	NO
27	LAKSHMI	55/F	1946249	104		2	2	3	3	2	3	3	NO
28	ARUNA	35/F	1947833	87		3	3	3	2	2	2	4	NO

29	KAVITHA	29/F	1949724	95		2	2	3	2	2	3	3	NO
30	SASIKALA	29/F	1954033	110		3	3	3	2	2	4	2	NO

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