A DISSERTATION ON

# "A STUDY ON PREVALANCE OF SUBCLINICAL / CLINICAL HYPOTHYROIDISM IN PATIENTS WITH EXTRA HEPATIC BILIARY LITHIASIS"

Dissertation submitted to

**THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERISTY**

**CHENNAI**

with partial fulfilment of the regulations

for the Award of the degree

**M.S. (General Surgery)**

Branch – I


**INSTITUTE OF GENERAL SURGERY**,

**MADRAS MEDICAL COLLEGE ,**

**CHENNAI.**

**APRIL-2016**

# CERTIFICATE

This is to certify that the dissertation entitled "A STUDY ON PREVALANCE OF SUBCLINICAL / CLINICAL HYPOTHYROIDISM IN PATIENTS WITH EXTRA HEPATIC BILIARY LITHIASIS" is a bonafide original work of **Dr. L.ARUN KUMAR**., in partial fulfilment of the requirements for M.S.Branch– I (General Surgery) Examination of the Tamil Nadu Dr. M.G.R._Medical University to be held in APRIL 2016 under my guidance and supervision in 2014-15.

**Prof.Dr.A.RAJENDRAN, M.S**
Professor of General Surgery,
Guide and supervisor
Institute of General Surgery,
Madras Medical College,
Rajiv Gandhi Government
General Hospital
Chennai – 600003.

**Prof.Dr.P.RAGUMANI. M.S**
Director and Professor
Institute of General Surgery
Madras Medical College,
Rajiv Gandhi Government General Hospital
Chennai - 600003

**Dr. VIMALA, M.D,**
Dean
Madras Medical College &Rajiv Gandhi Government General Hospital,
Chennai-600003

# DECLARATION

I hereby solemnly declare that the dissertation titled "A STUDY ON PREVALANCE OF SUBCLINICAL / CLINICAL HYPOTHYROIDISM IN PATIENTS WITH EXTRA HEPATIC BILIARY LITHIASIS" is done by Me at Madras Medical College & Rajiv Gandhi Govt. General Hospital, Chennai during 2014-15 under the guidance and supervision of Prof.Dr.A.RAJENDRAN, M.S. The dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai towards the partial fulfillment of requirements for the award of M.S. Degree (Branch-I) in General Surgery.

Place:
Date:

DR.L. ARUN KUMAR,
Post Graduate student ,
M.S. General Surgery
Institute of General Surgery,
Madras Medical College,
Chennai-3.

# ACKNOWLEDGEMENT

I express my heartful gratitude to the Dean, **Dr.VIMALA** M.D., Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai-3 for permitting me to do this study.

I am deeply indebted to **Prof.Dr.P.RAGUMANI**,M.S., Director& Professor, Institute of General Surgery, Madras MedicalCollege & Rajiv Gandhi Government General Hospital for his support.

I am very grateful to **Prof.Dr.A.RAJENDRAN** M.S, Professor of Surgery, Institute of General Surgery, and my Assistant Professors Dr.A.SAGAYA INBA SEKAR M.S., Dr.A. RAJESWARAN M.S., and Dr.K.M.M.VISHVAK CHANTHAR M.S., Madras Medical College & Rajiv Gandhi Government General Hospital who guided and provided constant feedback for my work throughout the period of my study.

I am extremely thankful to all the Members of the INSTITUTIONAL ETHICAL COMMITTEE for giving approval for my study. I also thank all the patients who were part of the study and my Professional colleagues for their support and criticisms.

# A STUDY ON PREVALANCE OF HYPOTHYROIDISM IN PATIENTS WITH EXTRA HEPATIC BILIARY LITHIASIS

**Aim :** To show the prevalence of clinical or subclinical hypothyroidism in patients with gallstones and common bile duct stones

**Back ground :** Studies have shown an increased prevalence of previously diagnosed hypothyroidism in gallstone patient and a delayed emptying of the biliary tract in hypothyroidism, explained partly by the missing pro relaxing effect of thyroxine on the sphincter of oddi contractility. Other explanations include the known link between thyroid failure and disturbances of lipid metabolism that may consecutively lead to change of the composition of the bile and motility of biliary tract.

**Patient and method :** An observational study was done in Institute of Surgery, Rajiv Gandhi Government General Hospital, Chennai between June 2014 to June 2015. For the 195 patients with diagnosed gallstone / CBD stone, full history and clinical examination was taken and laboratory blood test for T3, T4 and TSH was done. A control group of 100 patients of similar age and sex profile admitted in same time period for other illness were also evaluated for hypothyroidism.

**Results :** Out of 195 patients with gallstone 148 (76%) were females and 47 (24%) males. Thyroid disorder in form of hypothyroidism was found in 45 (23%), from this percentage 36 (24%) were females and from this 27 (18%) were subclinical and 9 (6%) were clinical hypothyroidism and males were 9 (19%) with all subclinical cases except one. This was compared with a control group of 100 patients, 80 (80%) were females and 20 (20%) males. Hypothyroidism was found in 9 (9%) patients, all female.

**Conclusion and recommendation :** There is a significant association between hypothyroidism and gallstones / CBD stones in both genders. Gallstone and CBD stone patients, especially females should be checked for serum TSH, T3 and T4 because of high incidence of hypothyroidism among this group.

# CONTENTS

# LIST OF TABLES

## LIST OF FIGURES

# LIST OF ABBREVIATIONS

| | | |
|---|---|---|
| GB | - - | Gall Bladder |
| CBD | - - | Common Bile Duct |
| SO | - - | Sphincter of Oddi |
| CCK | - - | Cholecystokinin |
| USG | - - | Ultrasonogram |
| CECT | - - | Contrast Enhanced Computed Tomography |
| MRCP | - - | Magnetic Resonance Cholangiopancreaticography |
| ERCP | - - | Endoscopic Retrograde Cholangiopancreaticography |
| PTC | - - | Percutaneous Transhepatic Cholangiography |
| TSH | - - | Thyroid Stimulating Hormone |
| TH | - - | Thyroid Hormones |
| TR | - - | intranuclear Thyroid Receptors |
| LDL | - - | Low Density Lipoproteins |
| HDL | - - | High Density Lipoproteins |
| HIDA | - - | Dimethyl Iminodiacetic Acid |

# CHAPTER 1

# INTRODUCTION

# INTRODUCTION

## 1. BACKGROUND

Gallstones are the most common biliary pathology. Many studies were done to identify risk factor for biliary lithiasis have focused on hypersaturation of cholesterol in bile in nucleation process a critical step in the genesis of bile stone.

Thyroid disorder is a prevalent condition among adult population; however, it is frequently over looked. For decade, there has been a discussion, whether thyroid disorders could cause gallstone disease. Particularly, there are several explanations for a possible relation between hypothyroidism and gallstone disease, these explanations include the known link between thyroid failure and disturbances of lipid metabolism that may consecutively lead to change of composition of the bile. Recent studies also demonstrated low bile flow in hypothyroid subjects. Further more, the sphincter of oddi expresses thyroid hormone receptors and thyroxine has a direct prorelaxing effect on the sphincter.

The prevalence of previously undiagnosed thyroid function abnormalities has never been studied in gallstone patients before. If an increased prevalence of thyroid disorders will be found, it might have an effect on the diagnostic and therapeutic work up of patient with gallstone.

## 1.2 OBJECTIVES

The purpose of this study is

1. To study the prevalence of hypothyroidism in patients presenting with extra hepatic biliary lithiasis.

2. To determine the validity of including of thyroid profile as an essential part of diagnostic workup in patients with extra hepatic biliary lithiasis.

3. To identify undetected cases of hypothyroidism.

4. To study the extent of hypercholesterolemia in patients with extra hepatic biliary lithiasis and its correlation with hypothyroidism

# CHAPTER 2

# REVIEW OF LITERATURE

# REVIEW OF LITERATURE

## 2.1 Anatomy of the biliary system

The biliary tree consists of the system of vessels and ducts which collect and deliver bile from the liver parenchyma to the second part of the duodenum. It is conventionally divided into intrahepatic and extrahepatic biliary trees. The intrahepatic ducts are formed from the larger bile canaliculi which come together to form segmental ducts. These fuse close to the porta hepatis into right and left hepatic ducts. The extrahepatic biliary tree consists of the right and left hepatic ducts, the common hepatic duct, the cystic duct and gallbladder and the common bile duct.

### *GALLBLADDER*

The gallbladder is a flask-shaped, blind-ending diverticulum attached to the common bile duct by the cystic duct. In the adult the gallbladder is between 7 and 10 cm long with a capacity of up to 50 ml. It usually lies in a shallow fossa in the liver parenchyma covered by peritoneum continued from the liver surface. The gallbladder is described as having a fundus, body and neck. The neck lies at the medial end close to the porta hepatis, and almost always has a short peritoneal-covered attachment to the liver (mesentery); this mesentery usually contains the cystic artery. The mucosa at the medial end of the neck is obliquely ridged, forming a spiral groove continuous with the spiral valve of the cystic duct. At its

lateral end the neck widens out to form the body of the gallbladder and this widening is often referred to in clinical practice as ‘Hartmann's pouch'. The neck lies anterior to the second part of the duodenum.

The body of the gallbladder normally lies in contact with the liver surface. When the neck possesses a mesentery, this rapidly shortens along the length of the body as it comes to lie in the gallbladder fossa. It lies anterior to the second 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. This is the location where enlargement of the gallbladder is best sought on clinical examination. The fundus commonly lies adjacent to the transverse colon.

The gallbladder varies in size and shape. The fundus may be elongated and highly mobile. Rarely, the fundus is folded back upon the body of the gallbladder, the so-called Phrygian cap: on ultrasound, this may be wrongly interpreted as an apparent septum within an otherwise normal gallbladder. Again, rarely, the gallbladder may be bifid or completely duplicated, usually with a duplicated cystic duct.

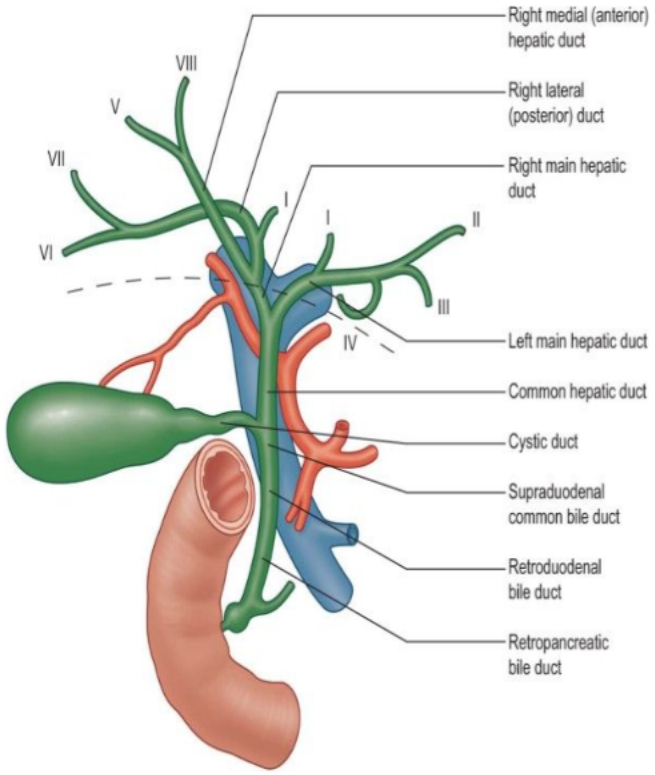



*Fig 1. Overall arrangement of the intrahepatic and extrahepatic biliary tree. The segmental ducts often branch just before, or are multiple, as they enter the main ducts, but for clarity are shown here as single ducts.*

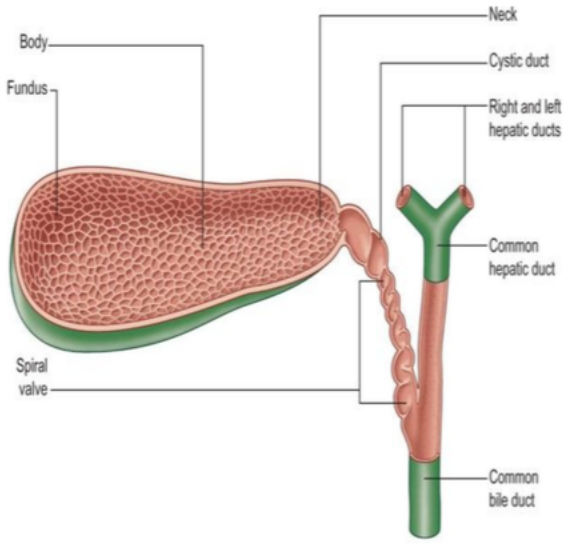



*Fig 2. Interior of the gallbladder and bile ducts.*

## *EXTRAHEPATIC BILIARY TREE*

### *CYSTIC DUCT*

The cystic duct drains the gallbladder into the common bile duct. It is between 3 and 4 cm long, passes posteriorly to the left from the neck of the gallbladder, and joins the common hepatic duct to form the common bile duct. It almost always runs parallel to, and is adherent to, the common hepatic duct for a short distance before joining it. The junction usually occurs near the porta hepatis but may be lower down in the free edge of the lesser omentum. The cystic duct

may have several important variations in its anatomy. Rarely, 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, but in these cases the cystic and common bile ducts are usually closely adherent. The cystic duct occasionally drains into the right hepatic duct, in which case it may be elongated, lie anterior or posterior to the common hepatic duct, and join the right hepatic duct on its left border. Rarely, the duct is double or even absent, in which case the gallbladder drains directly into the common bile duct. One or more accessory hepatic ducts occasionally emerge from segment V of the liver and join either the right hepatic duct, the common hepatic duct, the common bile duct, the cystic duct, or the gallbladder directly. These variations in cystic duct anatomy are of considerable importance during surgical excision of the gallbladder. Ligation or clip occlusion of the cystic duct must be performed at an adequate distance from the common bile duct to prevent angulation or damage to it. Accessory ducts must not be confused with the right hepatic or common hepatic ducts.

The mucosa of the cystic duct bears 5–12 crescentic folds, continuous with those in the neck of the gallbladder. 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.

### *HEPATIC BILE 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. The extrahepatic right duct is short and nearly vertical while the left is more horizontal and lies along the base of segment IV. The common hepatic duct lies to the right of the hepatic artery and anterior to the portal vein in the free edge of the lesser omentum.

### *COMMON BILE DUCT*

The common bile duct is formed near the porta hepatis, by the junction of the cystic and common hepatic ducts and is usually between 6 and 8 cm long. Its diameter tends to increase somewhat with age but is usually around 6 mm in adults. It descends posteriorly and slightly to the left, anterior to the epiploic foramen, in the right border of the lesser omentum, where it lies anterior and to the right of the portal vein and to the right of the hepatic artery. It passes behind the first part of the duodenum with the gastroduodenal artery on its left, and then runs in a groove on the superolateral part of the posterior surface of the head of the pancreas. The duct lies anterior to the inferior vena cava and is sometimes embedded in the pancreatic tissue.

#### *Hepatopancreatic ampulla (of Vater)*

As it lies medial to the second part of the duodenum, the common bile duct approaches the right end of the pancreatic duct. The ducts enter the duodenal wall

together, and usually unite to form the hepatopancreatic ampulla. Rarely the common bile duct and pancreatic duct drain into the duodenum separately. Circular muscle usually surrounds the lower part of the common bile duct (bile duct sphincter) and frequently also surrounds the terminal part of the main pancreatic duct (pancreatic duct sphincter) and the hepatopancreatic ampulla (sphincter of Oddi). When all elements are present, this arrangement may allow for separate control of pancreatic and common bile duct emptying. Division of the upper part of the ampulla and ampullary sphincter (sphincterotomy) may be required to allow access to the common bile duct during endoscopic retrograde cholangiography.

***Calot's triangle***

The near triangular space formed between the cystic duct, the common hepatic duct and the inferior surface of segment V of the liver, is commonly referred to as Calot's triangle. It is enclosed by the double layer of peritoneum which forms the short mesentery of the cystic duct. This space usually contains the cystic artery as it approaches the gallbladder, the cystic lymph node and lymphatics from the gallbladder. It may contain any accessory ducts which drain into the gallbladder from the liver. Appreciation of the variations in ductal and arterial anatomy are of considerable importance during excision of the gallbladder in order to avoid mistakenly ligating the common hepatic or common bile duct.

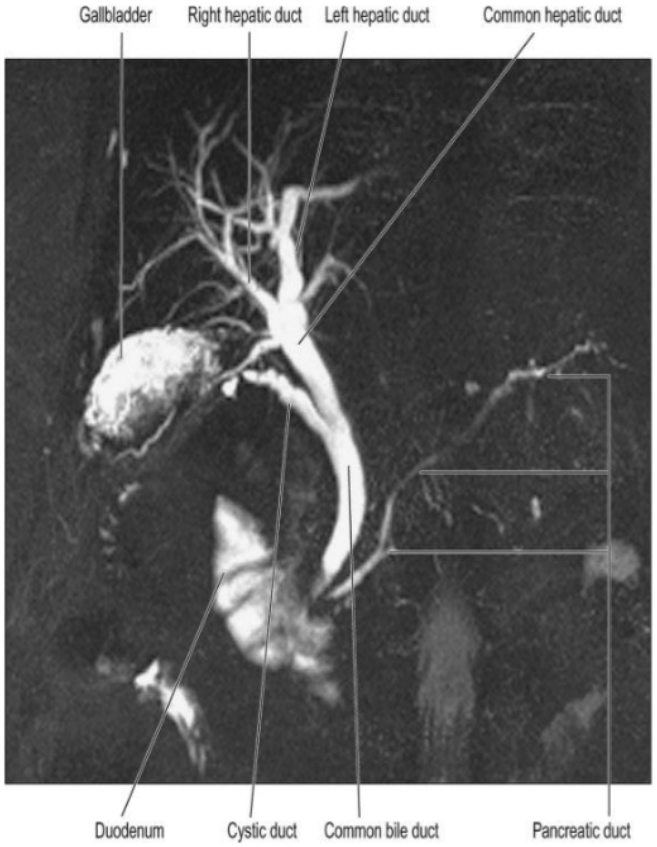



*Fig 3. Magnetic resonance cholangiopancreatogram.*

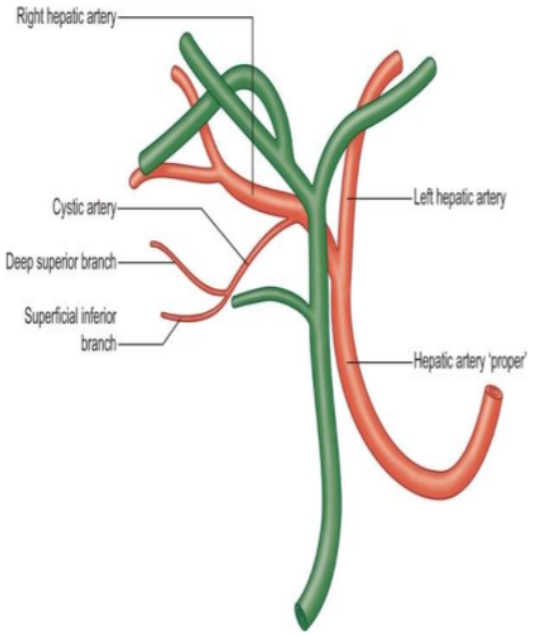



*Fig 4 Normal anatomy of the cystic artery.*

## *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 and divides into superficial and deep branches. The superficial branch ramifies on the inferior aspect of the body of the gallbladder, the deep branch on the superior aspect. These arteries anastomose over the surface of the body and fundus. The origin of the cystic artery frequently

varies. The most common variant is an origin from the common hepatic artery (occasionally low down), sometimes from the left hepatic or gastroduodenal artery, and rarely from the superior pancreaticoduodenal, coeliac, right gastric or superior mesenteric arteries. An accessory cystic artery may arise from the common hepatic artery or one of its branches and the cystic artery often bifurcates close to its origin, giving rise to two vessels which approach the gallbladder.

The cystic artery gives rise to multiple fine branches which supply the common and lobar hepatic ducts and upper part of the common bile duct. These fine branches form a network which anastomoses with the vessels ascending around the common bile duct and with the vessels from the liver parenchyma which descend with the right and left hepatic ducts.

***Ductal arteries***

The common bile duct and hepatic ducts are supplied by a fine network of vessels which usually receives contributions from several sources, and which lies in close proximity to the ducts. Disruption of the network during surgical exposure of the bile ducts over a long length frequently causes chronic ischaemia and resultant stenosis of the duct. Approaches which spare the network are necessary to avoid this complication.

***Cystic veins***

The venous drainage of the gallbladder is rarely by a single cystic vein. There are usually multiple small veins. Those arising from the superior surface of the body and neck lie in areolar tissue between the gallbladder and liver and enter the liver parenchyma to drain into the segmental portal veins. The remainder form one or two small cystic veins, which enter the liver either directly or after joining the veins that drain the hepatic ducts and upper bile duct. Only rarely does a single or double cystic vein drain into the right portal branch.

***Lymphatics***

Numerous lymphatic vessels run from the submucosal and subserosal plexuses on all aspects of the gallbladder and cystic duct. Those on the hepatic aspect of the gallbladder connect with the intrahepatic lymph vessels. 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. Hepatic nodes lying in the porta hepatis collect lymph from vessels that accompany the hepatic ducts and the upper part of the bile duct. Lymphatics from the lower part of the common bile duct drain into the inferior hepatic and upper pancreaticosplenic nodes.

***INNERVATION***

The gallbladder and the extrahepatic biliary tree are innervated by branches from the hepatic plexus. The retroduodenal part of the common bile duct and the smooth muscle of the hepatopancreatic ampulla are also innervated by twigs from the pyloric branches of the vagi.

Referred pain : In common with other structures of foregut origin, pain caused by stretch of the common bile duct or gallbladder is referred to the central epigastrium. Involvement of the overlying somatic peritoneum produces pain which is more localized to the right upper quadrant.

***MICROSTRUCTURE***

***GALLBLADDER***

The fundus of the gallbladder is completely covered by a serosa, and the inferior surfaces and sides of the body and neck of the gallbladder are usually covered by a serosa. Beneath the serosa is subserous loose connective and adipose peritoneal tissue. The gallbladder wall microstructure generally resembles that of the small intestine. The mucosa is yellowish-brown and elevated into minute rugae with a honeycomb appearance. The epithelium is a single layer of columnar cells with apical microvilli; basally, the spaces between epithelial cells are dilated. Many capillaries lie beneath the basement membrane. The epithelial cells actively absorb water and solutes from the bile and concentrate it up to ten-fold. There are no

goblet cells in the epithelium. The thin fibromuscular layer is composed of fibrous tissue mixed with smooth muscle cells arranged loosely in longitudinal, circular and oblique bundles.

***BILE DUCTS***

The walls of the large biliary ducts consist of external fibrous and internal mucosal layers. The outer layer is fibrous connective tissue containing a variable amount of longitudinal, oblique and circular smooth muscle cells. The mucosa is continuous with that in the hepatic ducts, gallbladder and duodenum. The epithelium is columnar and there are numerous tubuloalveolar mucous glands in the duct walls.

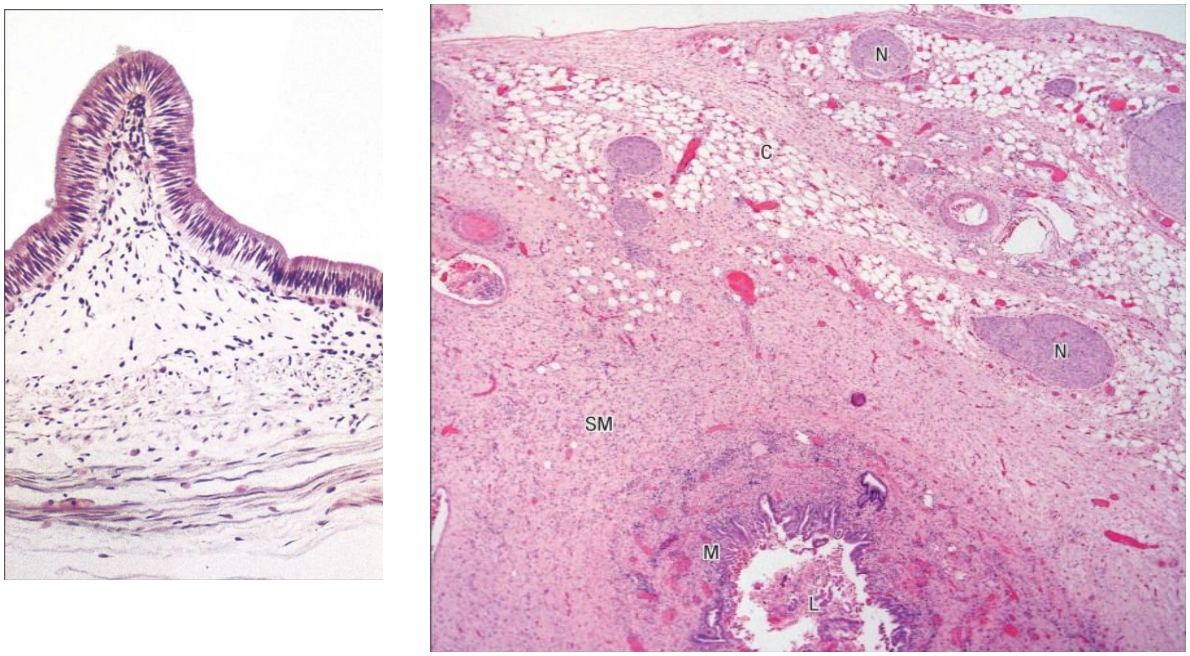



*Fig 5. Low power micrograph showing the gallbladder wall and the human common bile duct structure.*

## 2.2 Physiology

One of the many functions of the liver is to secrete bile, normally between 600 and 1000 ml/day. Bile serves two important functions: First, bile plays an important role in fat digestion and absorption, not because of any enzymes in the bile that cause fat digestion, but because bile acids in the bile do two things:

(1) they help to emulsify the large fat particles of the food into many minute particles, the surface of which can then be attacked by lipase enzymes secreted in pancreatic juice, and

(2) they aid in absorption of the digested fat end products through the intestinal mucosal membrane.

Second, bile serves as a means for excretion of several important waste roducts from the blood. These include especially bilirubin, an end product of hemoglobin destruction, and excesses of cholesterol.

***Physiologic Anatomy of Biliary Secretion :***

Bile is secreted in two stages by the liver: (1) The initial portion is secreted by the principal functional cells of the liver, the hepatocytes; this initial secretion contains large amounts of bile acids, cholesterol, and other organic constituents. It is secreted into minute bile canaliculi that originate between the hepatic cells.

(2) Next, the bile flows in the canaliculi toward the interlobular septa, where the canaliculi empty into terminal bile ducts and then into progressively larger ducts, finally reaching the hepatic duct and common bile duct. From these the bile either empties directly into the duodenum or is diverted for minutes up to several hours through the cystic duct into the gallbladder.

In its course through the bile ducts, a second portion of liver secretion is added to the initial bile. This additional secretion is a watery solution of sodium and bicarbonate ions secreted by secretory epithelial cells that line the ductules and ducts. This second secretion sometimes increases the total quantity of bile by as much as an additional 100 per cent. The second secretion is stimulated especially by secretin, which causes release of additional quantities of bicarbonate ions to supplement the bicarbonate ions in pancreatic secretion (for neutralizing acid that empties into the duodenum from the stomach).

***Storing and Concentrating Bile in the Gallbladder :***

Bile is secreted continually by the liver cells, but most of it is normally stored in the gallbladder until needed in the duodenum.he maximum volume that the gallbladder can hold is only 30 to 60 milliliters. Nevertheless, as much as 12 hours of bile secretion (usually about 450 milliliters) can be stored in the

(2) Next, the bile flows in the canaliculi toward the interlobular septa, where the canaliculi empty into terminal bile ducts and then into progressively larger ducts, finally reaching the hepatic duct and common bile duct. From these the bile either empties directly into the duodenum or is diverted for minutes up to several hours through the cystic duct into the gallbladder.

In its course through the bile ducts, a second portion of liver secretion is added to the initial bile. This additional secretion is a watery solution of sodium and bicarbonate ions secreted by secretory epithelial cells that line the ductules and ducts. This second secretion sometimes increases the total quantity of bile by as much as an additional 100 per cent. The second secretion is stimulated especially by secretin, which causes release of additional quantities of bicarbonate ions to supplement the bicarbonate ions in pancreatic secretion (for neutralizing acid that empties into the duodenum from the stomach).

***Storing and Concentrating Bile in the Gallbladder :***

Bile is secreted continually by the liver cells, but most of it is normally stored in the gallbladder until needed in the duodenum.he maximum volume that the gallbladder can hold is only 30 to 60 milliliters. Nevertheless, as much as 12 hours of bile secretion (usually about 450 milliliters) can be stored in the

gallbladder because water, sodium, chloride, and most other small electrolytes are continually absorbed through the gallbladder mucosa, concentrating the remaining bile constituents that contain the bile salts, cholesterol, lecithin, and bilirubin.

Most of this gallbladder absorption is caused by active transport of sodium through the gallbladder epithelium, and this is followed by secondary absorption of chloride ions, water, and most other diffusible constituents. Bile is normally concentrated in this way about 5-fold, but it can be concentrated up to a maximum of 20-fold.

**Composition of Bile**

| | Liver Bile | Gallbladder Bile |
|---|---|---|
| Water | 97.5 g/dl | 92 g/dl |
| Bile salts | 1.1 g/dl | 6 g/dl |
| Bilirubin | 0.04 g/dl | 0.3 g/dl |
| Cholesterol | 0.1 g/dl | 0.3 to 0.9 g/dl |
| Fatty acids | 0.12 g/dl | 0.3 to 1.2 g/dl |
| Lecithin | 0.04 g/dl | 0.3 g/dl |
| $Na^+$ | 145.04 mEq/L | 130 mEq/L |
| $K^+$ | 5 mEq/L | 12 mEq/L |
| $Ca^{++}$ | 5 mEq/L | 23 mEq/L |
| $Cl^-$ | 100 mEq/L | 25 mEq/L |
| $HCO_3^-$ | 28 mEq/L | 10 mEq/L |

This table shows that by far the most abundant substances secreted in the bile are bile salts, which account for about one half of the total solutes also in the bile. Also secreted or excreted in large concentrations are bilirubin, cholesterol, lecithin, and the usual electrolytes of plasma.

In the concentrating process in the gallbladder, water and large portions of the electrolytes (except calcium ions) are reabsorbed by the gallbladder mucosa; essentially all other constituents, especially the bile salts and the lipid substances cholesterol and lecithin, are not reabsorbed and, therefore, become highly concentrated in the gallbladder bile.

***Sphincter of Oddi :***

The sphincter of Oddi regulates flow of bile (and pancreatic juice) into the duodenum, prevents the regurgitation of duodenal contents into the biliary tree, and diverts bile into the gallbladder. It is a complex structure that is functionally independent from the duodenal musculature and creates a high-pressure zone between the bile duct and the duodenum. The sphincter of Oddi is about 4 to 6 mm in length and has a basal resting pressure of about 13 mmHg above the duodenal pressure. On manometry, the sphincter shows phasic contractions with a frequency of about four per minute and an amplitude of 12 to 140 mmHg.

The spontaneous motility of the sphincter of Oddi is regulated by the interstitial cells of Cajal through intrinsic and extrinsic inputs from hormones and neurons acting on the smooth muscle cells. Relaxation occurs with a rise in CCK, leading to diminished amplitude of phasic contractions and reduced basal pressure, allowing increased flow of bile into the duodenum. During fasting, the sphincter of Oddi activity is coordinated with the periodic partial gallbladder emptying and an increase in bile flow that occurs during phase II of the migrating myoelectric motor

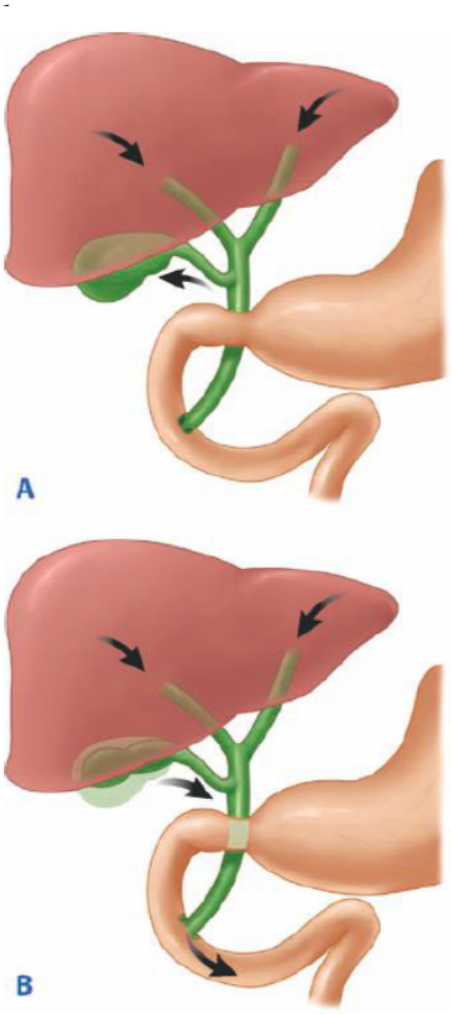



*Fig 6. The effect of cholecystokinin on the gallbladder and the sphincter of Oddi.*

*A. During fasting, with the sphincter of Oddi contracted and the gallbladder filling.*

*B. In response to a meal, the sphincter of Oddi relaxed and the gallbladder emptying.*

***Emptying of the Gallbladder—Stimulatory Role of Cholecystokinin :***

When food begins to be digested in the upper gastrointestinal tract, the gallbladder begins to empty, especially when fatty foods reach the duodenum about 30 minutes after a meal. The mechanism of gallbladder emptying is rhythmical contractions of the wall of the gallbladder, but effective emptying also requires simultaneous relaxation of the sphincter of Oddi, which guards the exit of the common bile duct into the duodenum.

By far the most potent stimulus for causing the gallbladder contractions is the hormone cholecystokinin. The stimulus for cholecystokinin entry into the blood from the duodenal mucosa is mainly the presence of fatty foods in the duodenum.

In addition to cholecystokinin, the gallbladder is stimulated less strongly by acetylcholine-secreting nerve fibers from both the vagi and the intestinal enteric nervous system. In summary, the gallbladder empties its store of concentrated bile into the duodenum mainly in response to the cholecystokinin stimulus that itself is initiated mainly by fatty foods. When fat is not in the food, the gallbladder empties poorly, but when significant quantities of fat are present, the gallbladder normally empties completely in about 1 hour.

***Function of Bile Salts in Fat Digestion and Absorption :***

The liver cells synthesize about 6 grams of bile salts daily. The precursor of the bile salts is cholesterol. The cholesterol is first converted to cholic acid or chenodeoxycholic acid in about equal quantities. These acids in turn combine principally with glycine and to a lesser extent with taurine to form glyco- and tauroconjugated bile acids. The salts of these acids, mainly sodium salts, are then secreted in the bile. Without the presence of bile salts in the intestinal tract, up to 40 per cent of the ingested fats are lost into the feces, and the person often develops a metabolic deficit because of this nutrient loss.

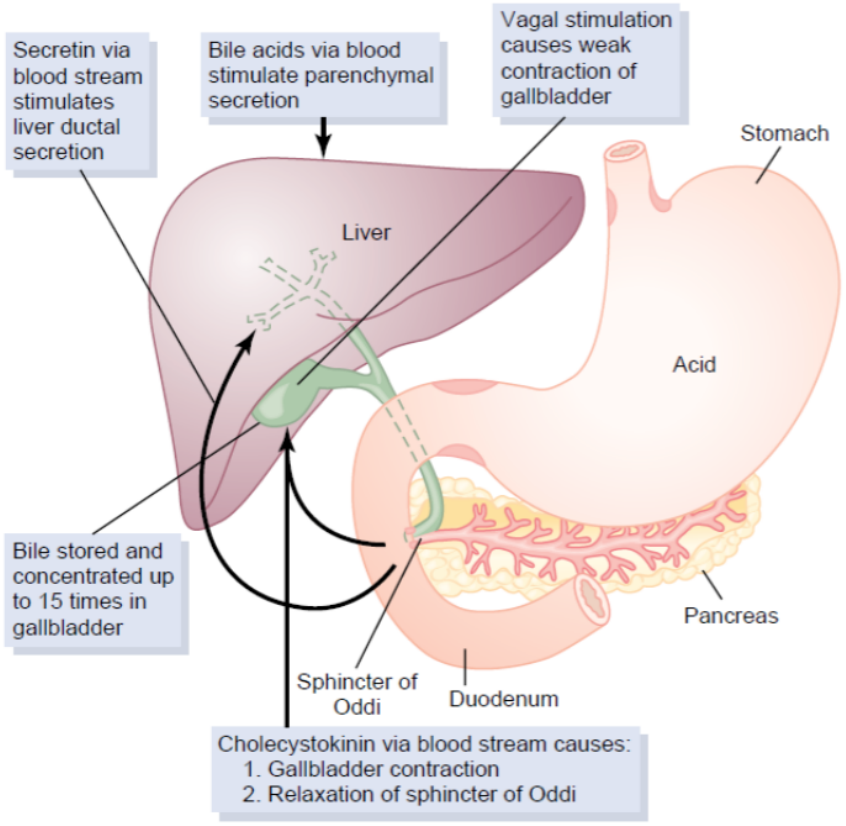



*Fig 7. Liver secretion and gallbladder emptying*

***Enterohepatic Circulation of Bile Salts :***

About 94 per cent of the bile salts are reabsorbed into the blood from the small intestine, about one half of this by diffusion through the mucosa in the early portions of the small intestine and the remainder by an active transport process through the intestinal mucosa in the distal ileum. They then enter the portal blood and pass back to the liver. On reaching the liver, on first passage through the venous sinusoids these salts are absorbed almost entirely back into the hepatic cells and then are resecreted into the bile. This recirculation of the bile salts is called the enterohepatic circulation of bile salts.

The daily rate of liver bile salt secretion is actively controlled by the availability (or lack of availability) of bile salts in the enterohepatic circulation.

***Role of Secretin in Helping to Control Bile Secretion :***

In addition to the strong stimulating effect of bile acids to cause bile secretion, the hormone secretin that also stimulates pancreatic secretion increases bile secretion, sometimes more than doubling its secretion for several hours after a meal. Thus, the secretin feedback mechanism for neutralizing duodenal acid operates not only through its effects on pancreatic secretion but also to a lesser extent through its effect on secretion by the liver ductules and ducts.

## 2.3 Pathophysiology of gallstones

Gallstones form as a result of solids settling out of solution. The major organic solutes in bile are bilirubin, bile salts, phospholipids, and cholesterol. Gallstones are classified by their cholesterol content as either cholesterol stones or pigment stones. Pigment stones can be further classified as either black or brown. In Western countries, about 80% of gallstones are cholesterol stones and about 15% to 20% are black pigment stones. Brown pigment stones account for only a small percentage. Both types of pigment stones are more common in Asia.

***Cholesterol Stones :*** Pure cholesterol stones are uncommon and account for <10% of all stones. They usually occur as single large stones with smooth surfaces. Most other cholesterol stones contain variable amounts of bile pigments and calcium, but are always >70% cholesterol by weight. These stones are usually multiple, of variable size, and may be hard and faceted or irregular, mulberry-shaped, and soft. Colors range from whitish yellow and green to black. Most cholesterol stones are radiolucent; <10% are radiopaque. Whether pure or of mixed nature, the common primary event in the formation of cholesterol stones is supersaturation of bile with cholesterol. Therefore, high bile cholesterol levels and cholesterol gallstones are considered as one disease.

***Pigment Stones :***

Pigment stones contain <20% cholesterol and are dark because of the presence of calcium bilirubinate. Otherwise, black and brown pigment stones have little in common and should be considered as separate entities.

Black pigment stones are usually small, brittle, black, and sometimes spiculated. They are formed by supersaturation of calcium bilirubinate, carbonate, and phosphate, most often secondary to hemolytic disorders such as hereditary spherocytosis and sickle cell disease, and in those with cirrhosis. In Asian countries such as Japan, black stones account for a much higher percentage of gallstones than in the Western hemisphere.

Brown stones are usually <1 cm in diameter, brownish yellow, soft, and often mushy. They may form either in the gallbladder or in the bile ducts, usually secondary to bacterial infection caused by bile stasis. Precipitated calcium bilirubinate and bacterial cell bodies compose the major part of the stone. Bacteria such as Escherichia coli secrete β-glucuronidase that enzymatically cleaves bilirubin glucuronide to produce the insoluble unconjugated bilirubin. It precipitates with calcium, and along with dead bacterial cell bodies, forms soft brown stones in the biliary tree. Brown stones are typically found in the biliary tree

of Asian populations and are associated with stasis secondary to parasite infection.

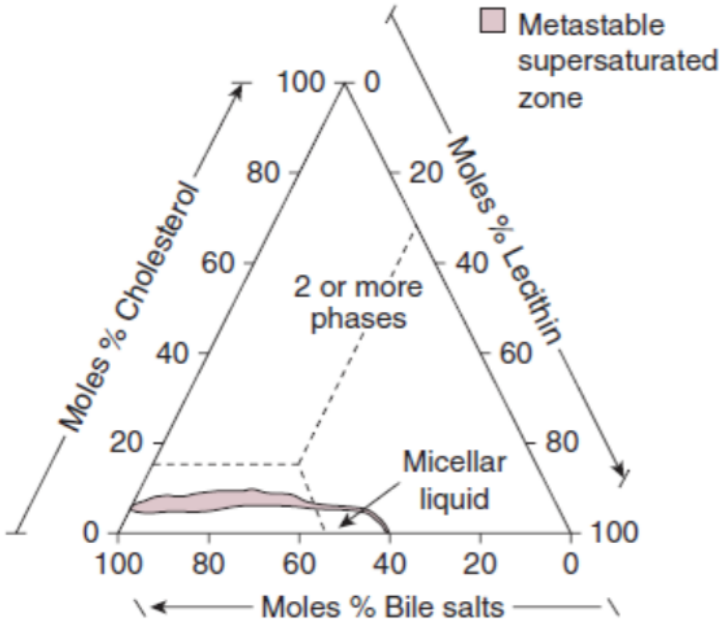



*Fig 8. Mechanism of bile stone formation*

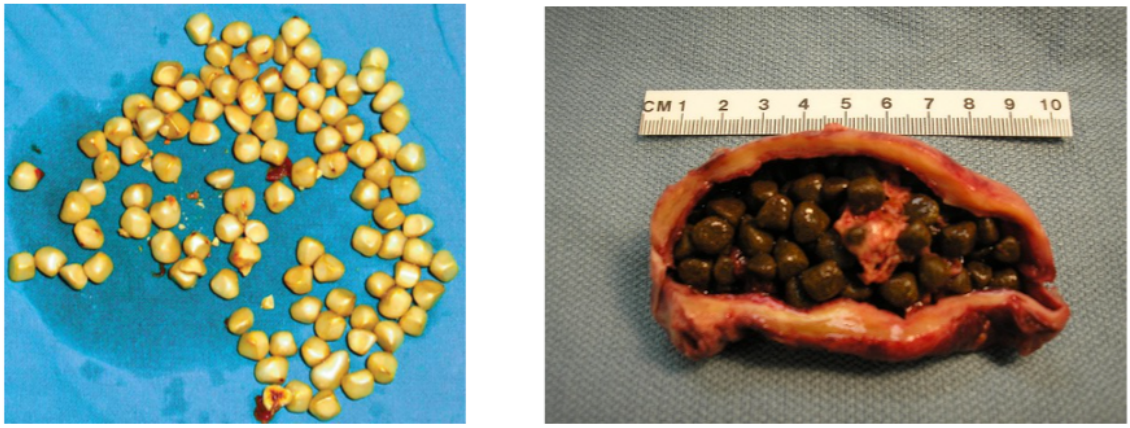


*Fig 9. Cholesterol and Pigment Stones*

***Choledocholithiasis :***

Common bile duct stones may be small or large and single or multiple, and are found in 6% to 12% of patients with stones in the gallbladder. The incidence increases with age. About 20% to 25% of patients above the age of 60 with symptomatic gallstones have stones in the common bile duct as well as in the gallbladder. The vast majority of ductal stones in Western countries are formed within the gallbladder and migrate down the cystic duct to the common bile duct. These are classified as secondary common bile duct stones, in contrast to the primary stones that form in the bile ducts. The secondary stones are usually cholesterol stones, whereas the primary stones are usually of the brown pigment type. The primary stones are associated with biliary stasis and infection and are more commonly seen in Asian populations.

The causes of biliary stasis that lead to the development of primary stones include biliary stricture, papillary stenosis, tumors, or other (secondary) stones.

***Acute Cholecystitis :*** Pathogenesis Acute cholecystitis is secondary to gallstones in 90% to 95% of cases. Acute acalculous cholecystitis is a condition that typically occurs in patients with other acute systemic diseases. In <1% of acute cholecystitis, the cause is a tumor obstructing the cystic duct. Obstruction of the cystic duct by a

gallstone is the initiating event that leads to gallbladder distention, inflammation, and edema of the gallbladder wall. Initially, acute cholecystitis is an inflammatory process, probably mediated by the mucosal toxin lysolecithin, a product of lecithin, as well as bile salts and platelet-activating factor. Increase in prostaglandin synthesis amplifies the inflammatory response. Secondary bacterial contamination is documented in 15% to 30% of patients undergoing cholecystectomy for acute uncomplicated cholecystitis. In acute cholecystitis, the gallbladder wall becomes grossly thickened and reddish with subserosal hemorrhages. Pericholecystic fluid often is present. The mucosa may show hyperemia and patchy necrosis. In severe cases, about 5% to 10%, the inflammatory process progresses and leads to ischemia and necrosis of the gallbladder wall. More frequently, the gallstone is dislodged and the inflammation resolves.

***Chronic Cholecystitis (Biliary Colic) :*** About two thirds of patients with gallstone disease present with chronic cholecystitis characterized by recurrent attacks of pain, often inaccurately labeled biliary colic. The pain develops when a stone obstructs the cystic duct, resulting in a progressive increase of tension in the gallbladder wall. The pathologic changes, which often do not correlate well with symptoms, vary from an apparently normal gallbladder with minor chronic

inflammation in the mucosa, to a shrunken, nonfunctioning gallbladder with gross transmural fibrosis and adhesions to nearby structures. The mucosa is initially normal or hypertrophied, but later becomes atrophied, with the epithelium protruding into the muscle coat, leading to the formation of the so-called Aschoff-Rokitansky sinuses.

## 2.4 Clinical Features

Gallstone disease is one of the most common problems affecting the digestive tract. Autopsy reports have shown a prevalence of gallstones from 11% to 36%.24 The prevalence of gallstones is related to many factors, including age, gender, and ethnic background. Certain conditions predispose to the development of gallstones. Obesity, pregnancy, dietary factors, Crohn's disease, terminal ileal resection, gastric surgery, hereditary spherocytosis, sickle cell disease, and thalassemia are all associated with an increased risk of developing gallstones. Women are three times more likely to develop gallstones than men, and first-degree relatives of patients with gallstones have a twofold greater prevalence.

***Natural History***

Most patients will remain asymptomatic from their gallstones throughout life. For unknown reasons, some patients progress to a symptomatic stage, with

biliary colic caused by a stone obstructing the cystic duct. Symptomatic gallstone disease may progress to complications related to the gallstones. These include acute cholecystitis, choledocholithiasis with or without cholangitis, gallstone pancreatitis, cholecystocholedochal fistula, cholecystoduodenal or cholecystoenteric fistula leading to gallstone ileus, and gallbladder carcinoma. Rarely, complication of gallstones is the presenting picture.

Gallstones in patients without biliary symptoms are commonly diagnosed incidentally on ultrasonography, CT scans, or abdominal radiography or at laparotomy. Several studies have examined the likelihood of developing biliary colic or developing significant complications of gallstone disease. Approximately 3% of asymptomatic individuals become symptomatic per year (i.e., develop biliary colic). Once symptomatic, patients tend to have recurring bouts of biliary colic. Complicated gallstone disease develops in 3% to 5% of symptomatic patients per year. Over a 20-year period, about two thirds of asymptomatic patients with gallstones remain symptom free. Because few patients develop complications without previous biliary symptoms, prophylactic cholecystectomy in asymptomatic persons with gallstones is rarely indicated. For elderly patients with diabetes, for individuals who will be isolated from medical care for extended periods of time,

and in populations with increased risk of gallbladder cancer, a prophylactic cholecystectomy may be advisable. Porcelain gallbladder, a rare premalignant condition in which the wall of the gallbladder becomes calcified, is an absolute indication for cholecystectomy.

***Acute Cholecystitis :***

About 80% of patients with acute cholecystitis give a history compatible with chronic cholecystitis. Acute cholecystitis begins as an attack of biliary colic, but in contrast to biliary colic, the pain does not subside; it is unremitting and may persist for several days. The pain is typically in the right upper quadrant or epigastrium and may radiate to the right upper part of the back or the interscapular area. It is usually more severe than the pain associated with uncomplicated biliary colic. The patient is often febrile, complains of anorexia, nausea, and vomiting, and is reluctant to move, as the inflammatory process affects the parietal peritoneum. On physical examination, focal tenderness and guarding are usually present in the right upper quadrant. A mass, the gallbladder and adherent omentum, is occasionally palpable; however, guarding may prevent this. A Murphy's sign, an inspiratory arrest with deep palpation in the right subcostal area, is characteristic of acute cholecystitis.

A mild to moderate leukocytosis (12,000–15,000 cells/mm3) is usually present. However, some patients may have a normal WBC. Serum liver chemistries are usually normal, but a mild elevation of serum bilirubin, <4 mg/mL, may be present along with mild elevation of alkaline phosphatase, transaminases, and amylase. Severe jaundice is suggestive of common bile duct stones or obstruction of the bile ducts by severe pericholecystic inflammation secondary to impaction of a stone in the infundibulum of the gallbladder that mechanically obstructs the bile duct (Mirizzi's syndrome). In elderly patients and in those with diabetes mellitus, acute cholecystitis may have a subtle presentation resulting in a delay in diagnosis. The incidence of complications is higher in these patients, who also have approximately 10-fold the mortality rate compared to that of younger and healthier patients.

The differential diagnosis for acute cholecystitis includes a peptic ulcer with or without perforation, pancreatitis, appendicitis, hepatitis, perihepatitis (Fitz-Hugh–Curtis syndrome),myocardial ischemia, pneumonia, pleuritis, and herpes zoster involving the intercostal nerve.

***Chronic Cholecystitis :***

The chief symptom associated with symptomatic gallstones is pain. The pain is constant and increases in severity over the first half hour or so and typically lasts 1 to 5 hours. It is located in the epigastrium or right upper quadrant and frequently radiates to the right upper back or between the scapulae. The pain is severe and comes on abruptly, typically during the night or after a fatty meal. It often is associated with nausea and sometimes vomiting. The pain is episodic. The patient suffers discrete attacks of pain, between which they feel well. Physical examination may reveal mild right upper quadrant tenderness during an episode of pain. If the patient is pain free, the physical examination is usually unremarkable. Laboratory values, such as WBC count and liver function tests, are usually normal in patients with uncomplicated gallstones.

Atypical presentation of gallstone disease is common. Association with meals is present in only about 50% of patients. Some patients report milder attacks of pain, but relate it to meals. The pain may be located primarily in the back or the left upper or lower right quadrant. Bloating and belching may be present and associated with the attacks of pain. In patients with atypical presentation, other conditions with upper abdominal pain should be sought out, even in the presence

of gallstones. These include peptic ulcer disease, gastroesophageal reflux disease, abdominal wall hernias, irritable bowel disease, diverticular disease, liver diseases, renal calculi, pleuritic pain, and myocardial pain. Many patients with other conditions have gallstones. When the pain lasts >24 hours, an impacted stone in the cystic duct or acute cholecystitis should be suspected. An impacted stone without cholecystitis will result in what is called hydrops of the gallbladder.

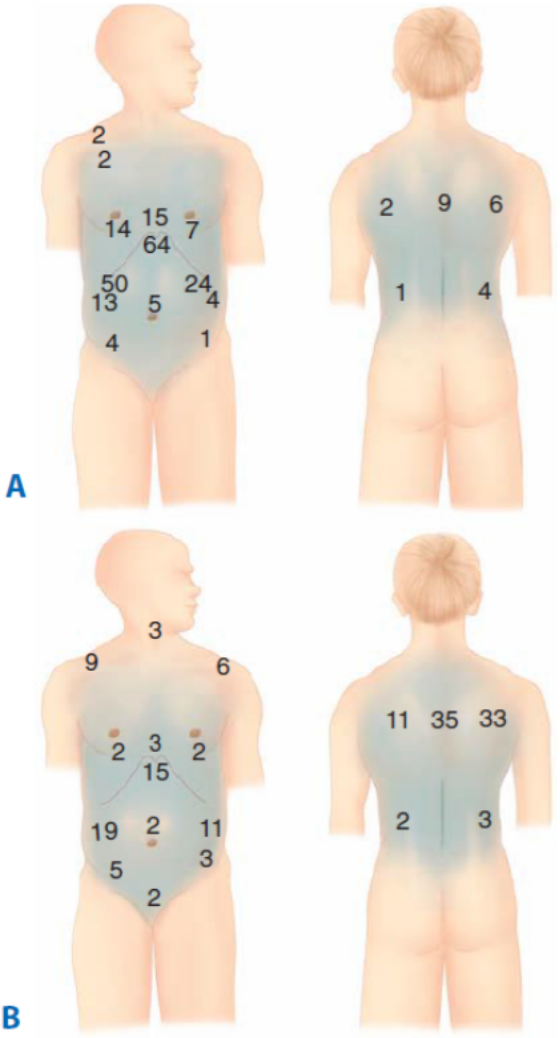



Fig 10.

A. Sites of the most severe pain during an episode of biliary pain in 107 patients with gallstones (% values add up to >100% because of multiple responses). The subxiphoid and right subcostal areas were the most common sites; note that the left subcostal area was not an unusual site of pain.

B. Sites of pain radiation (%) during an episode of biliary pain in the same group of patients.

Essentials of Diagnosis :

✓ Episodic abdominal pain.

✓ Dyspepsia

✓ Gallstones on cholecystography or ultrasound scan.

***Choledocholithiasis :*** Choledochal stones may be silent and often are discovered incidentally. They may cause obstruction, complete or incomplete, or they may manifest with cholangitis or gallstone pancreatitis. The pain caused by a stone in the bile duct is very similar to that of biliary colic caused by impaction of a stone in the cystic duct. Nausea and vomiting are common.

Physical examination may be normal, but mild epigastric or right upper quadrant tenderness as well as mild icterus are common. The symptoms may also be intermittent. A small stone may pass through the ampulla spontaneously with resolution of symptoms. Finally, the stones may become completely impacted, causing severe progressive jaundice. Elevation of serum bilirubin, alkaline phosphatase, and transaminases are commonly seen in patients with bile duct stones. However, in about one third of patients with common bile duct stones, the liver chemistries are normal.

in the gallbladder (if still present), as well as determining the size of the common bile duct. As stones in the bile ducts tend to move down to the distal part of the common duct, bowel gas can preclude their demonstration on ultrasonography. A dilated common bile duct (>8 mm in diameter) on ultrasonography in a patient with gallstones, jaundice, and biliary pain is highly suggestive of common bile duct stones. Magnetic resonance cholangiography (MRC) provides excellent anatomic detail and has a sensitivity and specificity of 95% and 89%, respectively, at detecting choledocholithiasis >5 mm in diameter. Endoscopic cholangiography is the gold standard for diagnosing common bile duct stones. It has the distinct advantage of providing a therapeutic option at the time of diagnosis. In experienced hands, cannulation of the ampulla of Vater and diagnostic cholangiography are achieved in >90% of cases, with associated morbidity of <5% (mainly cholangitis and pancreatitis).

## 2.5 Diagnostic Studies [28-32]

***Baseline Blood Investigations :***

- Complete hemogram
- Differential Count
- Renal Function Test
- Liver Function Test including liver enzymes
- Fasting Lipid Profile

When patients with suspected diseases of the gallbladder or the extrahepatic biliary tree are evaluated, 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

A variety of diagnostic modalities are available for the patient with suspected disease of the gallbladder and the bile ducts. In 1924, the diagnosis of gallstones was improved significantly by the introduction of oral cholecystography by Graham and Cole. For decades, it was the mainstay of investigation for

gallstones. In the 1950s, biliary scintigraphy was developed, as well as intrahepatic and endoscopic retrograde cholangiography (ERC), allowing imaging of the biliary tract. Later ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI) vastly improved the ability to image the biliary tract.

***Plain Xray Abdomen :***

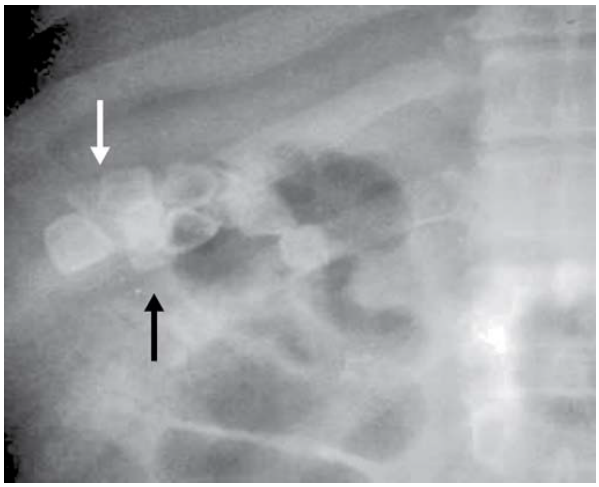


*Fig 11. Multiple gallstones in a plain X-ray. Only 10% gallstones are radio-opaque. Often they are faceted because of compactness and equal pressure exerted by each stone. Centre of the gallstone is often found radiolucent and is called as Mercedes Benz sign/Seagull sign*

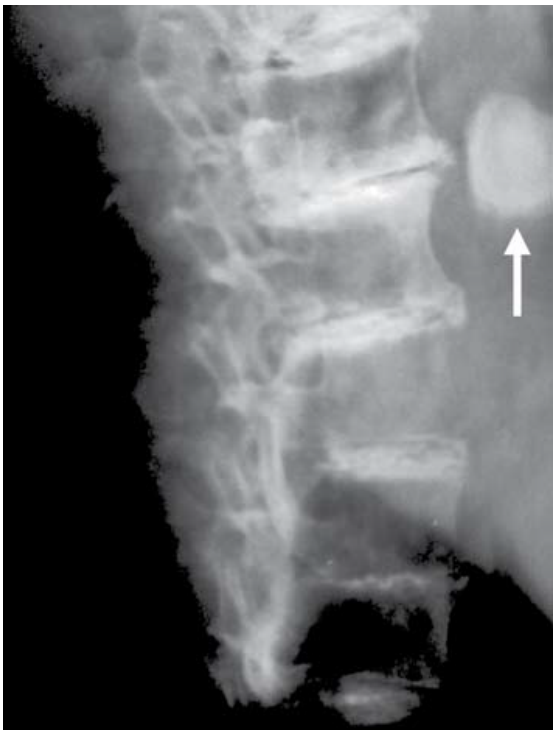


*Fig 12. Lateral X-ray of spine showing radio-opaque shadow infront of the vertebrae—could be radio-opaque gallstone*

***Ultrasonography :***

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. 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. A thickened gallbladder wall and local tenderness indicate cholecystitis. The patient has acute cholecystitis if a layer of edema is seen within the wall of the gallbladder or between the gallbladder and the liver in association with localized tenderness. When a stone obstructs the neck of the gallbladder, the gallbladder may become very large, but thin walled. A contracted, thick-walled gallbladder is indicative of chronic cholecystitis.

The extrahepatic bile ducts are also well visualized by ultrasound, except for the retroduodenal portion. Dilation of the ducts in a patient with jaundice establishes an extrahepatic obstruction as a cause for the jaundice. Frequently, the site and, sometimes, the cause of obstruction can be determined by ultrasound. Small stones in the common bile duct frequently get lodged at the distal end of it, behind the duodenum, and are, therefore, difficult to detect.

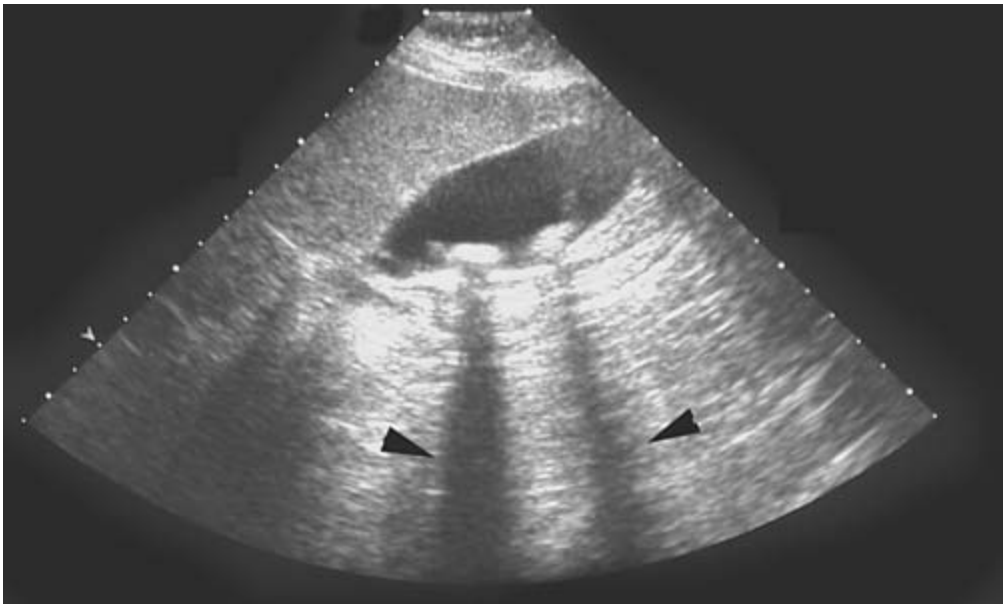


*Fig 13. An ultrasonography of the gallbladder. Arrows indicate the acoustic shadows from stones in the gallbladder.*

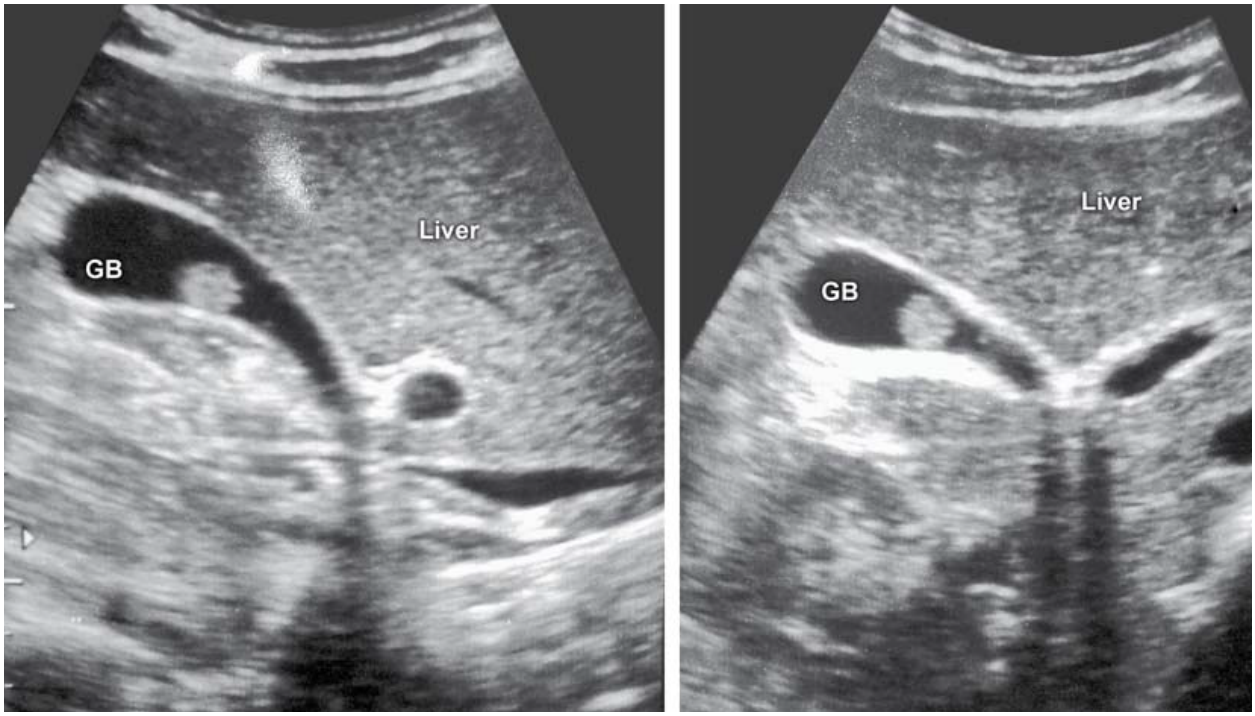



*Fig 14. Ultrasound gallbladder showing echogenic lesion. US should be done with change of position to find out movement of thelesion with posterior acoustic shadow to say it as gallstone. Otherwise it will be gallbladder polyp or sludge ball.*

***Oral Cholecystography***

Once considered the diagnostic procedure of choice for gallstones, oral cholecystography has largely been replaced by ultrasonography. It involves oral administration of a radiopaque compound that is absorbed, excreted by the liver, and passed into the gallbladder. Stones are noted on a film as filling defects in a visualized, opacified gallbladder. Oral cholecystography is of no value in patients with intestinal malabsorption, vomiting, obstructive jaundice, and hepatic failure.

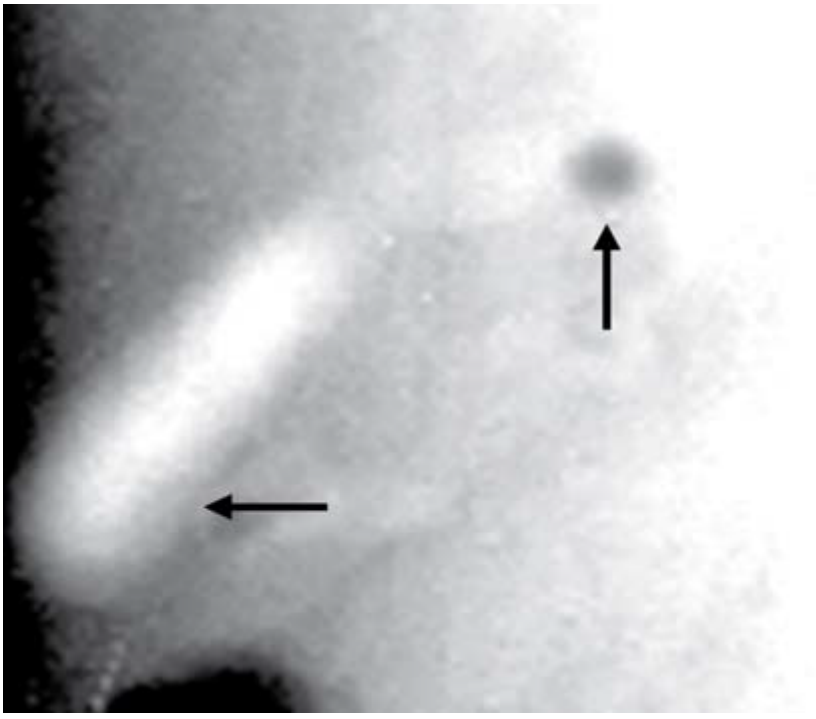


*Fig 15. Oral cholecystogram with smooth filling defect (Cystic duct stone).*

***Biliary Radionuclide Scanning (HIDA Scan)***

Biliary scintigraphy provides a noninvasive evaluation of the liver, gallbladder, bile ducts, and duodenum with both anatomic and functional information. 99mTechnetium-labeled derivatives of dimethyl iminodiacetic acid (HIDA) are injected intravenously, cleared by the Kupffer cells in the liver, and excreted in the bile. Uptake by the liver is detected within 10 minutes, and the gallbladder, the bile ducts, and the duodenum are visualized within 60 minutes in fasting subjects. The primary use of biliary scintigraphy is in the diagnosis of acute cholecystitis, which appears as a nonvisualized gallbladder, with prompt filling of the common bile duct and duodenum. Evidence of cystic duct obstruction on biliary scintigraphy is highly diagnostic for acute cholecystitis. The sensitivity and specificity for the diagnosis are about 95% each. False-positive results are increased in patients with gallbladder stasis, as in critically ill patients and in patients receiving parenteral nutrition.

***Computed Tomography :***

Abdominal CT scans are inferior to ultrasonography in diagnosing gallstones. The major application of CT scans is to define the course and status of the extrahepatic biliary tree and adjacent structures. It is the test of choice in evaluating the patient with suspected malignancy of the gallbladder, the extrahepatic biliary system, or nearby organs, in particular, the head of the pancreas. Use of CT scan is an integral

part of the differential diagnosis of obstructive jaundice. Spiral CT scanning provides additional staging information, including vascular involvement in patients with periampullary tumors.

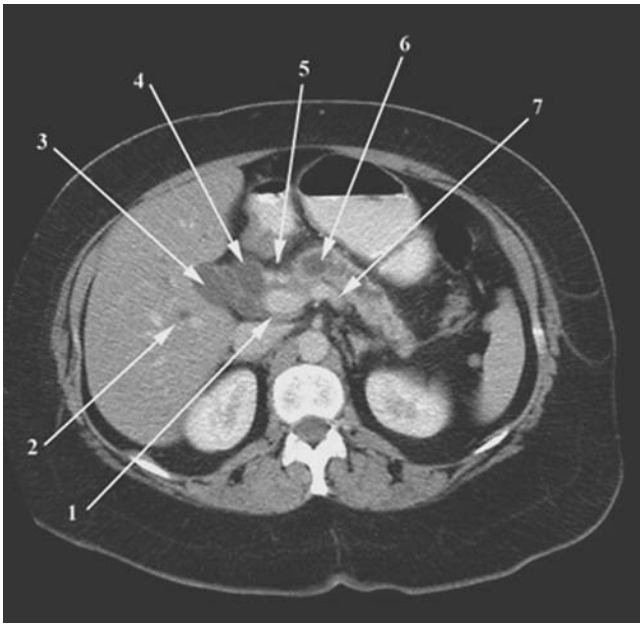



*Fig 16. Computed tomography scan of the upper abdomen from a patient with cancer of the distal common bile duct. The cancer obstructs the common bile duct as well as the pancreatic duct.*

*1 = the portal vein;*

*2 = a dilated intrahepatic bile duct*

*3 = dilated cystic duct and the neck of the gallbladder*

*4 = dilated common hepatic duct*

*5 = the bifurcation of the common hepatic artery into the gastroduodenal artery and the proper hepatic artery*

*6 = dilated pancreatic duct*

*7 = the splenic vein*

***Percutaneous Transhepatic Cholangiography***

Intrahepatic bile ducts are accessed percutaneously with a small needle under fluoroscopic guidance. Once the position in a bile duct has been confirmed, a guidewire is passed, and subsequently, a catheter is passed over the wire Through the catheter, a cholangiogram can be performed and therapeutic interventions done, such as biliary drain insertions and stent placements. Percutaneous transhepatic

cholangiography (PTC) has little role in the management of patients with uncomplicated gallstone disease but is particularly useful in patients with bile duct strictures and tumors, as it defines the anatomy of the biliary tree proximal to the affected segment. As with any invasive procedure, there are potential risks. For PTC, these are mainly bleeding, cholangitis, bile leak, and other catheter-related problems.

***Magnetic Resonance Imaging***

Available since the mid-1990s, MRI provides anatomic details of the liver, gallbladder, and pancreas similar to those obtained from CT. Many MRI techniques (i.e., heavily T2-weighted sequences, pulse sequences with or without contrast materials) can generate high-resolution anatomic images of the biliary tree and the pancreatic duct. It has a sensitivity and specificity of 95% and 89%, respectively, at detecting choledocholithiasis. MRI with magnetic resonance cholangiopancreatography (MRCP) offers a single noninvasive test for the diagnosis of biliary tract and pancreatic disease. In many centers, MRCP is first performed for diagnosis of biliary and pancreatic duct pathology, reserving endoscopic retrograde cholangiopancreatography (ERCP) for therapeutic purposes only.

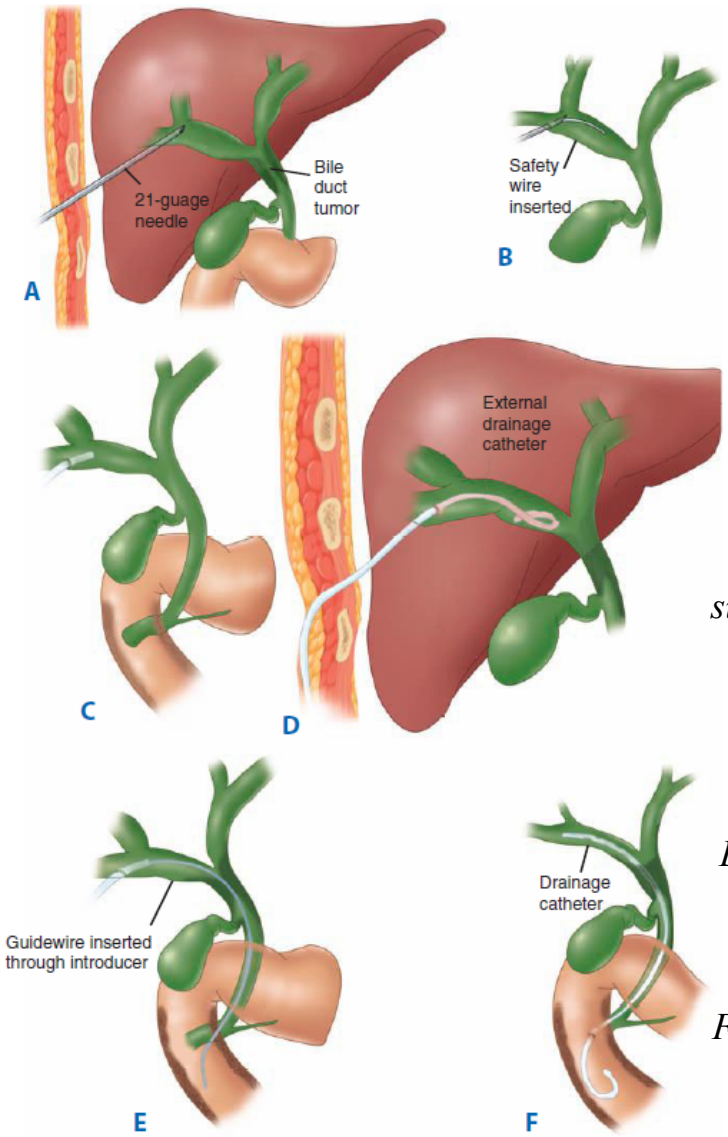



*Fig 17. Schematic diagram of percutaneous transhepatic cholangiogram and drainage for obstructing proximal cholangiocarcinoma.*

*A. Dilated intrahepatic bile duct is entered percutaneously with a fine needle.*

*B. Small guidewire is passed through theneedle into the duct*

*C. A plastic catheter has been passed over the wire, and the wire is subsequently removed. A cholangiogram is performed through the catheter.*

*D. An external drainage catheter in place.*

*E. Long wire placedvia the catheter and advanced past the tumorand into the duodenum.*

*F. Internal stent has been placed through the tumor.*

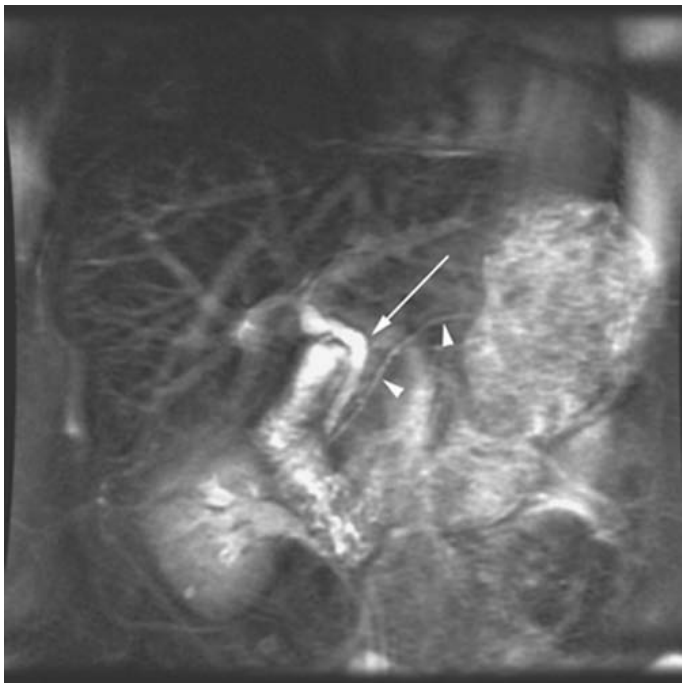
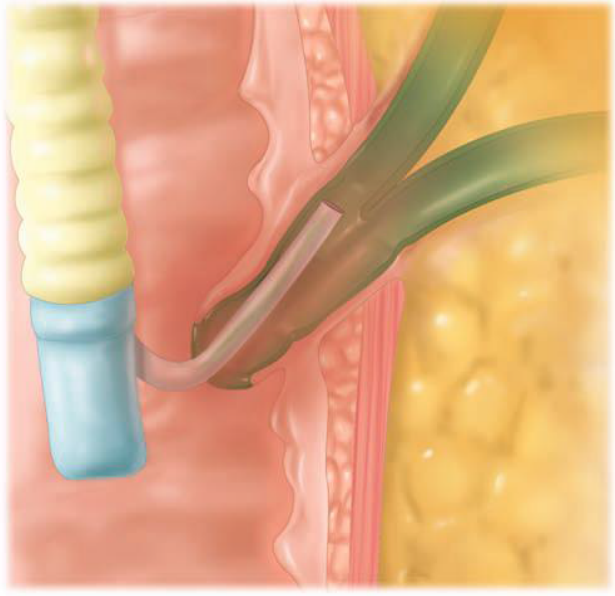


*Fig 18. Magnetic resonance cholangiopancreatography.*

*This view shows the course of theextrahepatic bile ducts (arrow) and the pancreatic duct (arrowheads).*



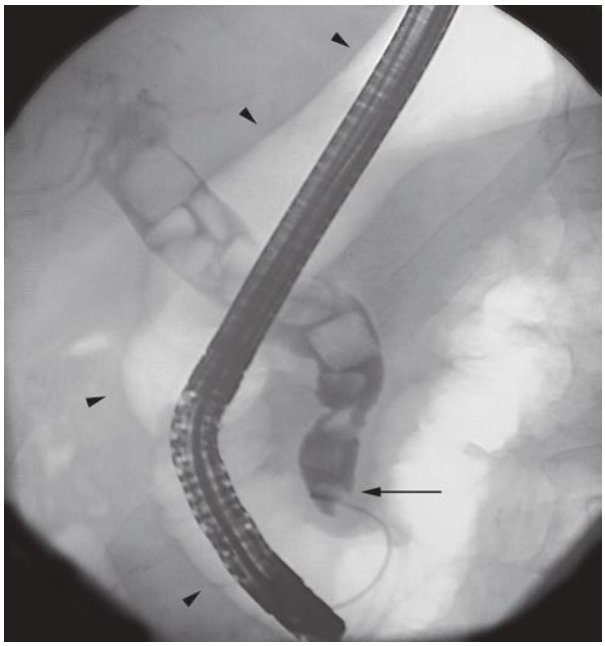


*Fig 19. Endoscopic retrograde cholangiography. A schematic picture showing the side-viewing endoscope in the duodenum and a catheter in the common bile duct. An endoscopic cholangiography showing stones in the common bile duct. The catheter has been placed in the ampulla of Vater (arrow). Note the duodenal shadow indicated with arrowheads.*

***Endoscopic Retrograde Cholangiopancreatography :***

Using a side-viewing endoscope, the common bile duct can be cannulated and a cholangiogram performed using fluoroscopy. The procedure requires intravenous (IV) sedation for the patient. The advantages of ERC include direct visualization of the ampullary region and direct access to the distal common bile duct, with the possibility of therapeutic intervention. The test is rarely needed for uncomplicated gallstone disease, but for stones in the common bile duct, in particular, when associated with obstructive jaundice, cholangitis, or gallstone pancreatitis, ERC is the diagnostic and often therapeutic procedure of choice. Once the endoscopic cholangiogram has shown ductal stones, sphincterotomy and stone extraction can be performed, and the common bile duct cleared of stones. In the hands of experts, the success rate of common bile duct cannulation and cholangiography is >90%. Complications of diagnostic ERC include pancreatitis and cholangitis and occur in up to 5% of patients. The development of small fiber-optic cameras that can be threaded through endoscopes used for endoscopic retrograde cholangiopancreatography (ERCP) has facilitated the development of intraductal endoscopy. By providing direct visualization of the biliary and pancreatic ducts, this technology has been shown to increase the effectiveness of ERCP in the diagnosis of certain biliary and pancreatic diseases. Intraductal endoscopy has been shown to have therapeutic applications that include biliary

stone lithotripsy and extraction in high-risk surgical patients. As with most endoscopic procedures, intraductal endoscopy generally is considered safe, but there are no large trials that specifically address this issue. Typical complications such as bile duct perforation, minor bleeding from sphincterotomy or lithotripsy, and cholangitis have been described. Further refinement of this technology will enhance ERCP as a diagnostic and therapeutic tool.

***Endoscopic Ultrasound***

Endoscopic ultrasound requires a special endoscope with an ultrasound transducer at its tip. The results are operator dependent, but offer noninvasive imaging of the bile ducts and adjacent structures. It is of particular value in the evaluation of tumors and their resectability. The ultrasound endoscope has a biopsy channel, allowing needle biopsies of a tumor under ultrasonic guidance. Endoscopic ultrasound also has been used to identify bile duct stones, and although it is less sensitive than ERC, the technique is less invasive as cannulation of the sphincter of Oddi is not necessary for diagnosis of choledocholithiasis.

## 2.6 Management [33-45]

### *Symptomatic Gallstones*

Patients with symptomatic gallstones should be advised to have elective laparoscopic cholecystectomy. While waiting for surgery, or if surgery has to be postponed, the patient should be advised to avoid dietary fats and large meals. Diabetic patients with symptomatic gallstones should have a cholecystectomy promptly, as they are more prone to develop acute cholecystitis that is often severe. Pregnant women with symptomatic gallstones who cannot be managed expectantly with diet modifications can safely undergo laparoscopic cholecystectomy during the second trimester. Laparoscopic cholecystectomy is safe and effective in children as well as in the elderly. Cholecystectomy, open or laparoscopic, for patients with symptomatic gallstones offers excellent long-term results. About 90% of patients with typical biliary symptoms and stones are rendered symptom free after cholecystectomy. For patients with atypical symptoms or dyspepsia (flatulence, belching, bloating, and dietary fat intolerance), the results are not as favorable.

***Acute Cholecystitis***

Patients who present with acute cholecystitis will need IV fluids, antibiotics, and analgesia. The antibiotics should cover gram-negative aerobes as well as anaerobes. A third generation cephalosporin with good anaerobic coverage or a second-generation cephalosporin combined with metronidazole is a typical regimen.

Cholecystectomy is the definitive treatment for acute cholecystitis. In the past, the timing of cholecystectomy has been a matter of debate. Early cholecystectomy performed within 2 to 3 days of the illness is preferred over interval or delayed cholecystectomy that is performed 6 to 10 weeks after initial medical treatment and recuperation. Several studies have shown that unless the patient is unfit for surgery, early cholecystectomy should be recommended, as it offers the patient a definitive solution in one hospital admission, quicker recovery times, and an earlier return to work.

Laparoscopic cholecystectomy is the procedure of choice for acute cholecystitis. The conversion rate to an open cholecystectomy is higher (10%–15%) in the setting of acute cholecystitis than with chronic cholecystitis. When patients present late, after 3 to 4 days of illness, or if they are unfit for surgery, they

can be treated with antibiotics with laparoscopic cholecystectomy scheduled for approximately 2 months later. Approximately 20% of patients will fail to respond to initial medical therapy and require an intervention.

For those unfit for surgery, a percutaneous cholecystostomy or an open cholecystostomy under local analgesia can be performed. Failure to improve after cholecystostomy usually is due to gangrene of the gallbladder or perforation. For these patients, surgery is unavoidable. For those who respond after cholecystostomy, the tube can be removed once cholangiography through it shows a patent ductus cysticus. Laparoscopic cholecystectomy may then be scheduled in the near future. For the rare patients who can't tolerate surgery, the stones can be extracted via the cholecystostomy tube before its removal.

***Choledocholithiasis***

For patients with symptomatic gallstones and suspected common bile duct stones, either preoperative endoscopic cholangiography or an intraoperative cholangiogram will document the bile duct stones. If an endoscopic cholangiogram reveals stones, sphincterotomy and ductal clearance of the stones is appropriate, followed by a laparoscopic cholecystectomy. An intraoperative cholangiogram at the time of cholecystectomy will also document the presence or absence of bile

duct stones. Laparoscopic common bile duct exploration via the cystic duct or with formal choledochotomy allows the stones to be retrieved in the same setting. An open common bile duct exploration is an option if the endoscopic method has already been tried or is, for some reason, not feasible. If a choledochotomy is performed, a T tube is left in place. Patients >70 years old presenting with bile duct stones should have their ductal stones cleared endoscopically. Studies comparing surgery to endoscopic treatment have documented less morbidity and mortality for endoscopic treatment in this group of patients. They do not need to be submitted for a cholecystectomy, as only about 15% will become symptomatic from their gallbladder stones, and such patients can be treated as the need arises by a cholecystectomy.

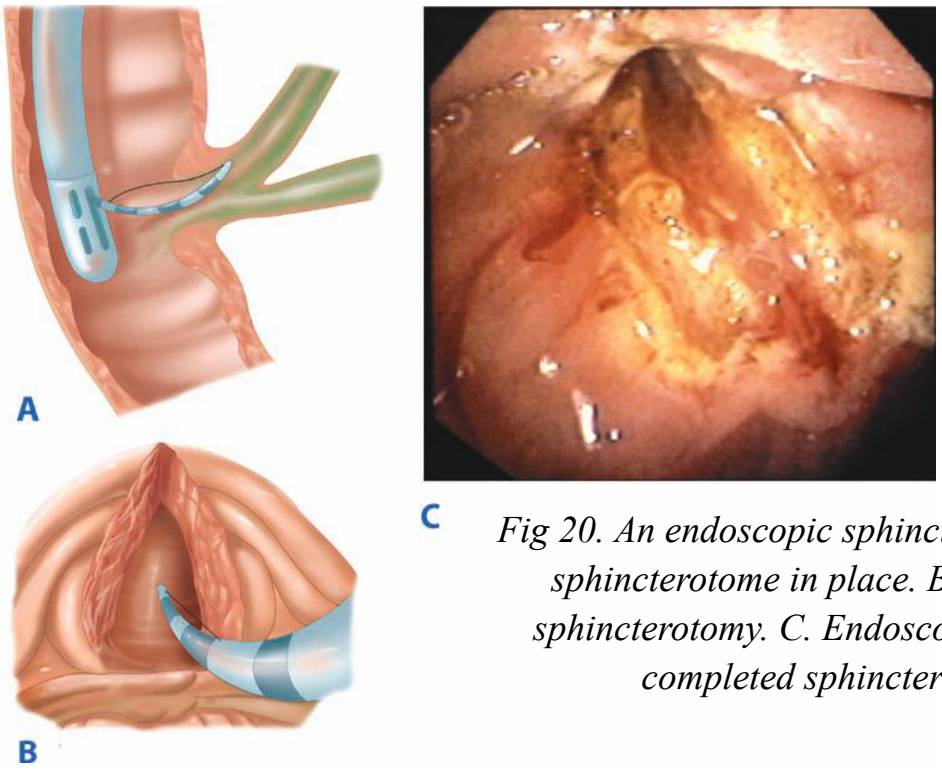



*Fig 20. An endoscopic sphincterotomy. A. The sphincterotome in place. B. Completed sphincterotomy. C. Endoscopic picture of completed sphincterotomy.*

## *Operative interventions for gallstone disease*

### *Cholecystostomy*

A cholecystostomy decompresses and drains the distended, inflamed, hydropic, or purulent gallbladder. It is applicable if the patient is not fit to tolerate an abdominal operation. Ultrasound-guided percutaneous drainage with a pigtail catheter is the procedure of choice

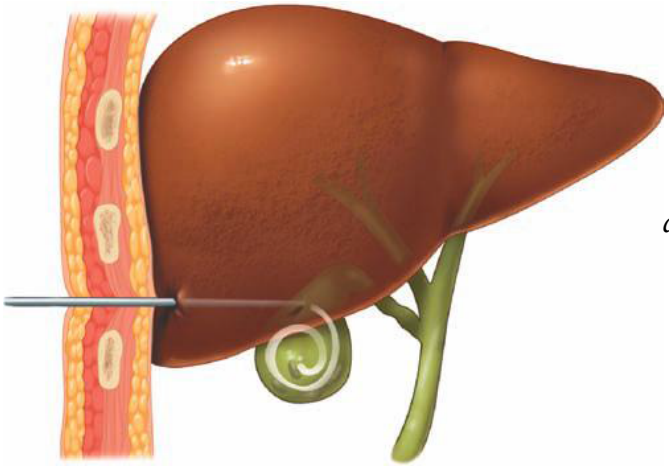


*Fig 21. Percutaneous cholecystostomy. A pigtail catheterhas been placed through the abdominal wall, the right lobe of theliver, and into the gallbladder*

## *Cholecystectomy*

Carl Langenbuch performed the first successful cholecystectomy in 1882, and for >100 years, it was the standard treatment for symptomatic gallbladder stones. Open cholecystectomy was a safe and effective treatment for both acute and chronic cholecystitis. In 1987, laparoscopic cholecystectomy was introduced by Philippe Mouret in France and quickly revolutionized the treatment of gallstones. It not only

supplanted open cholecystectomy, but also more or less ended attempts for noninvasive management of gallstones, such as extracorporeal shock wave and bile salt therapy. Laparoscopic cholecystectomy offers a cure for gallstones with a minimally invasive procedure, minor pain and scarring, and early return to full activity. Today, laparoscopic cholecystectomy is the treatment of choice for symptomatic gallstones. Absolute contraindications for the procedure are uncontrolled coagulopathy and end-stage liver disease.

***Open Cholecystectomy* :**

The same surgical principles apply for laparoscopic and open cholecystectomies. Open cholecystectomy has become an uncommon procedure, usually performed either as a conversion from laparoscopic cholecystectomy or as a second procedure in patients who require laparotomy for another reason. After the cystic artery and cystic duct have been identified, the gallbladder is dissected free from the liver bed, starting at the fundus. The dissection is carried proximally toward the cystic artery and the cystic duct, which are then ligated and divided.

***Intraoperative Cholangiogram or Ultrasound***

The bile ducts are visualized under fluoroscopy by injecting contrast through a catheter placed in the cystic duct. Their size can then be evaluated, the presence or

absence of common bile duct stones assessed, and filling defects confirmed, as the dye passes into the duodenum. Routine intraoperative cholangiography will detect stones in approximately 7% of patients, as well as outlining the anatomy and detecting injury. A selective intraoperative cholangiogram can be performed when the patient has a history of abnormal liver function tests, pancreatitis, jaundice, a large duct and small stones, a dilated duct on preoperative ultrasonography, and if preoperative endoscopic cholangiography for the above reasons was unsuccessful. Laparoscopic ultrasonography is as accurate as intraopera tive cholangiography in detecting common bile duct stones, and it is less invasive; however, it requires more skill to perform and interpret.

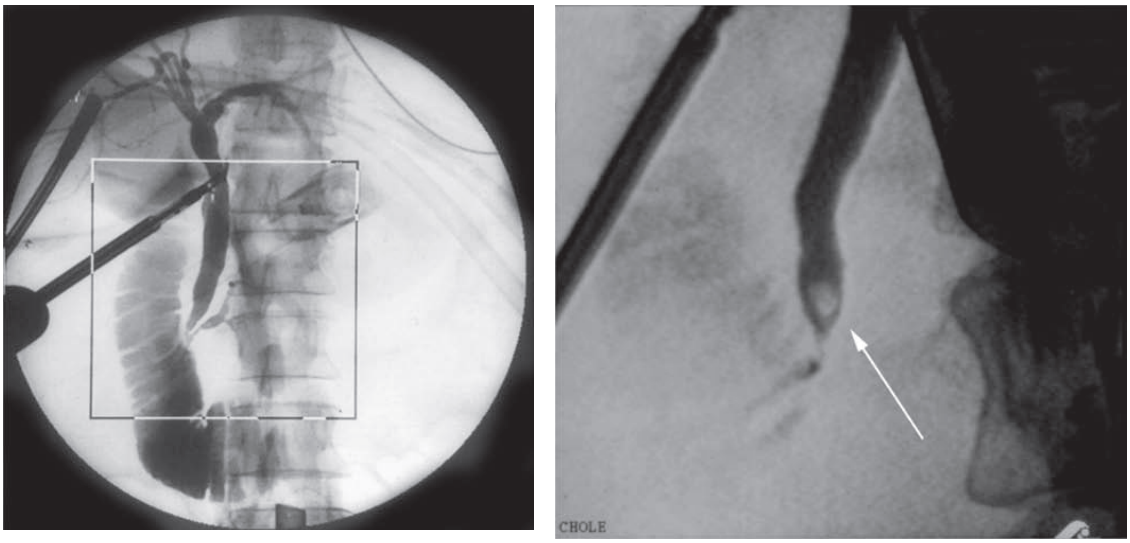
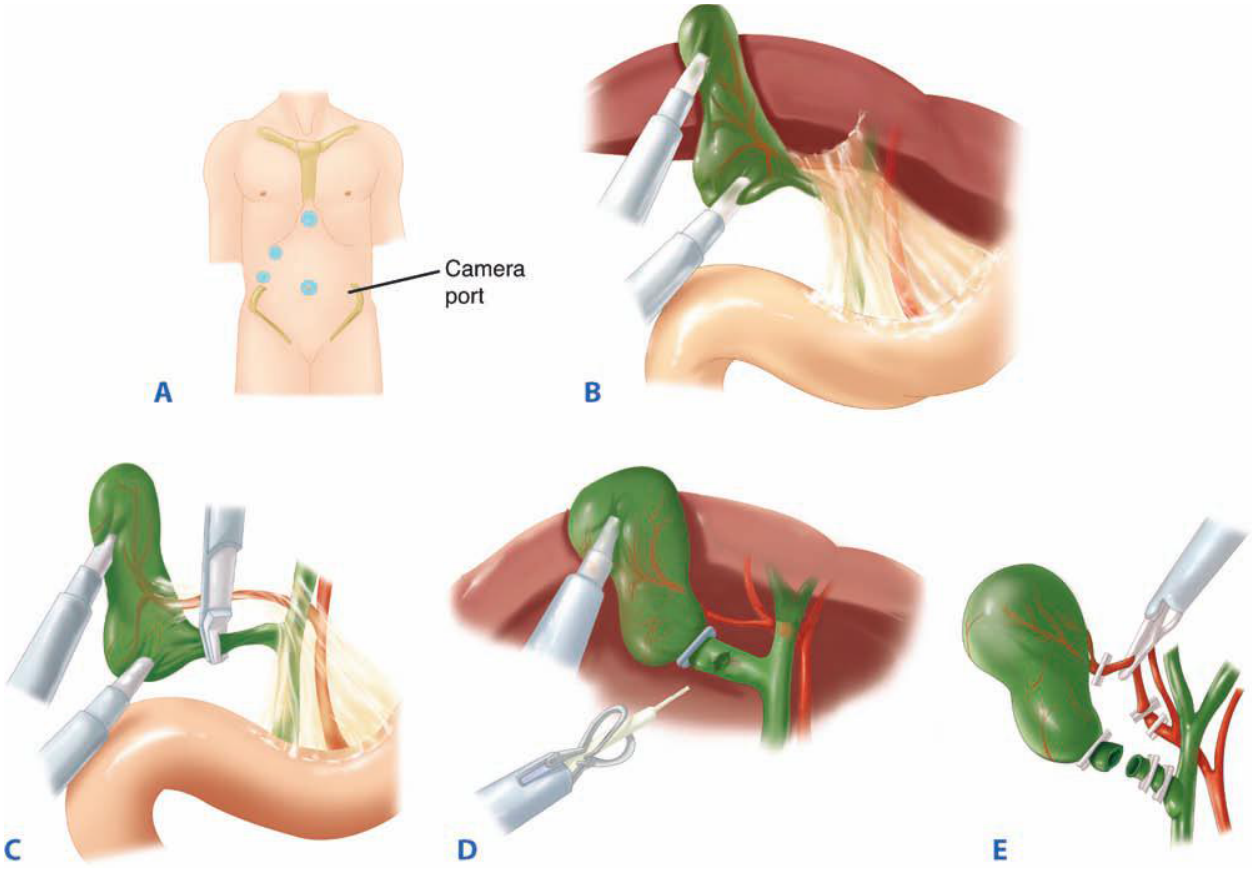



*Fig 22. A. An intraoperative cholangiogram. The bile ducts are of normal size, with no intraluminal filling defects. Cholangiography grasper that holds the catheter and the cystic duct stump partly projects over the common hepatic duct. B. An intraoperative cholangiogram showing common bile duct stone (arrow). A small amount of contrast has passed into the duodenum..*

***Common Bile Duct Exploration***

Common bile duct stones that are detected intraoperatively on intraoperative cholangiography or ultrasonography may be managed with laparoscopic choledochal exploration as a part of the laparoscopic cholecystectomy procedure. Patients with common bile duct stones detected preoperatively, but in whom endoscopic clearance was either not available or unsuccessful, should also have their ductal stones managed during the cholecystectomy. If the stones in the duct are small, they may sometimes be flushed into the duodenum with saline irrigation via the cholangiography catheter after the sphincter of Oddi has been relaxed with glucagon. When the duct has been cleared, the cystic duct is ligated and cut and the cholecystectomy completed. Occasionally, a choledochotomy, an incision into the common bile duct itself, is necessary. The flexible choledochoscope is then passed into the duct for visualization and clearance of stones. The choledochotomy is sutured with a T tube left in the common bile duct with one end taken out through the abdominal wall for decompression of the bile ducts. By managing common bile duct stones at the time of the cholecystectomy, the patients can have all of their gallstone disease treated with one invasive procedure. It does, however,

depend on the available surgical expertise.



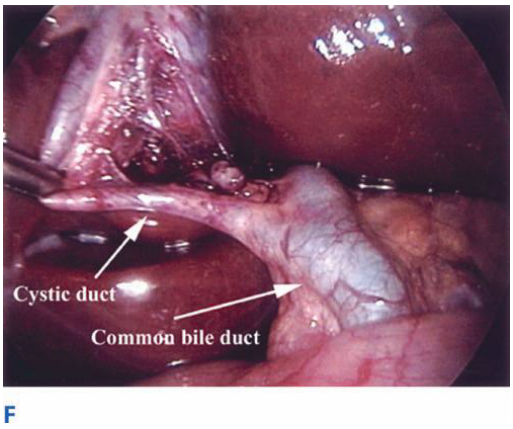




*Fig 23. Laparoscopic cholecystectomy.*

*A. The trocar placement.*

*B. The fundus has been grasped and retracted cephalad to expose the proximal gallbladder and the hepatoduodenal ligament. Another grasper retracts the gallbladder infundibulum posterolaterally to better expose the triangle of*

*C. The triangle of Calot has been opened and the neck of the gallbladder and part of the cystic duct dissected free. A clip is being placed on the cystic duct–gallbladder junction.*

*D. A small opening has been made into the cystic duct, and a cholangiogram catheter is to be inserted.*

*E. The cystic duct has been divided, and the cystic artery is being divided.*

*F. An intraoperative picture showing a grasper pulling the infundibulum of the gallbladderlaterally, exposing the triangle of Calot that has been dissected. The cystic artery can*

## 2.7 Thyroid Function

***Hyperthyroidism :***

Hyperthyroidism is a common disorder, especially in women, the Incidence being about 80/100 000 women yearly. In young patients its cause is usually Graves disease, whereas in elderly patients toxic nodular goitre is also a common cause. In this disorder the basal metabolism is elevated, and synthesis and breakdown of proteins, carbohydrates and fat are accelerated. Its clinical symptoms include nervousness, heat intolerance and sweating, palpitations, tremulousness, weight loss with good appetite, muscle weakness, emotional lability, and hyperdefecation. Possible clinical signs of hyperthyroidism are thyroid enlargement, eye stare and lid lag, warm and smooth skin, fine tremor, brisk reflexes, onycholysis, tachycardia or atrial fibrillation, and in Gravesí disease proptosis of the eyes and ophthalmoplegia, and pretibial myxoedema. Measurement of serum thyroid-stimulating hormone, i.e. thyrotropin (TSH), is the most sensitive test in screening for hyperthyroidism, an undetectable value being a hallmark. Confirmation can be made by measuring serum free T4 (FT4). In a patient without ophthalmopathy, measurement of thyroid radioiodine (131I) uptake is performed to establish the cause of thyrotoxicosis. Hyperthyroidism may be treated with antithyroid drugs, 131I, or subtotal thyroidectomy.

***Hypothyroidism :***

Hypothyroidism is likewise a common disorder, again especially in women, the incidence being about 350/100 000 women yearly. The prevalence of subclinical hypothyroidism among women over 60 years of age is as high as 20%. Hypothyroidism is defined as in any state which results in a deficiency of TH, including hypothalamic or pituitary diseases, generalized tissue resistance to TH, and disorders directly affecting the thyroid gland.

Thyroid deficiency in adults is characterized by a slowing of all metabolic processes. The possible clinical symptoms of the condition include weakness, lethargy and fatigue, memory impairment, dementia, cold intolerance, weight gain, constipation, loss of hair, hoarseness, deafness, dyspnea, myalgia and arthralgia, paraesthesias, precordial pain and menstrual irregularity.

Clinical signs of hypothyroidism may be dry, coarse and cold skin, periorbital and peripheral oedema, coarse and thin hair, pallor of skin, thick tongue, slow speech, decreased reflexes, hypertension, bradycardia, pleural and pericardial effusions, ascites and vitiligo. In laboratory tests, e.g. hypercholesterolaemia and anaemia are associated findings.

The laboratory hallmark of primary hypothyroidism and the most sensitive test for detecting early thyroid failure is an increased serum TSH concentration. The serum FT4 level is decreased in clinical hypothyroidism. In the subclinical form an increased serum TSH level is accompanied by a normal serum FT4 level,

and the patient is asymptomatic. The presence of thyroperoxidase antibody confirms chronic autoimmune thyroiditis as the cause of hypothyroidism. The treatment of hypothyroidism with levothyroxine is usually lifelong.

***The effects of thyroid hormones :***

The thyroid gland secretes both T4 and triiodothyronine (T3) into the circulation. In extrathyroidal tissues, T4 is converted to T3, assumed to be the major active TH. Characteristic of THs is the multiplicity of the cellular functions they regulate in virtually every type of vertebrate tissue. The diverse responses to TH can be divided into two major categories: (1) regulation of metabolic activity, energy consumption and muscular activity in adult mammals, and (2) regulation of postembryonic or perinatal growth and development. Most actions of THs can be explained by their interaction with nuclear receptors. THs bind to specific intranuclear TH receptors (TR), TR1, TR1 or TR2. This ligand-receptor complex binds to TH response elements in the target genes to regulate the rate of synthesis of specific messenger RNAs. This results in a change in the amount or activity of the cognate proteins, which in turn alter the rate of the metabolic process. The TRs are expressed in a tissue- and development-stage-specific fashion. For example, TR1 is known to be highly expressed in brain, muscle and fat, liver and kidney. TR2 is highly expressed in the pituitary. The expression of TRs in the SO has not been studied. The genomic effects of THs necessarily require a finite period of time for protein synthesis and for the biological response. Extranuclear sites of TH

action include the cell membrane, the cytoskeleton, the sarcoplasmic reticulum, the cytoplasm, the mitochondria, and in vascular smooth-muscle cells presumably the contractile elements. For example, THs mediate sugar uptake, adenylate cyclase, and $Ca^{2+}$- ATPase activity directly at the level of the plasma membrane of various tissues.

***Thyroid hormones and cholesterol metabolism :***

The elevation of serum cholesterol levels is a clinically important accompaniment of hypothyroidism. Patients with overt hypothyroidism have approximately 50% higher serum cholesterol levels than euthyroid patients. Treatment of hypothyroid patients who also have hyperlipidaemia will have beneficial effects on serum cholesterol levels.

THs have been shown to have a number of effects on cholesterol metabolism. LDL receptor activity is increased (Ness et al. 1990) because of increased expression of the LDL receptor gene, and the expression may be decreased in hypothyroidism, leading to reduced removal of cholesterol from the serum. THs increase the synthesis of cholesterol by regulating the expression of HMG-CoA reductase, and the regulation is reduced in hypothyroid patients, leading to decreased cholesterol synthesis. Also the synthesis of bile salts is increased by THs by the effect on cholesterol-7-hydroxylase, and a decrease in biliary bile salt concentration in hypothyroidism has been reported. Absorbtion of cholesterol is decreased by THs. Biliary secretion of cholesterol is reduced in

hypothyroidism compared to euthyroidism. However, when serum cholesterol values rise, bile may also become supersaturated with cholesterol and thus result in gallstone formation. An association has been reported between cholesterol gallstones and treated hypothyroidism in women. It has also been reported that gallbladder stones may have been dissolved after T4 treatment.

## 2.8 Prevalence of Clinical and Subclinical Hypothyroidism in Gall Stone Patients

Several recent studies report an association between hypothyroidism, or subclinical hypothyroidism, and gall stones. In a retrospective study on patients over 60 years of age, it was noted for the first time that CBD stone patients have significantly more diagnosed hypothyroidism (11%), not only when compared to control patients from whom gallstones had been excluded (2%), but also when compared to gallbladder stone patients without CBD stones (6%). In this study, there was no difference between groups in the frequency of other diseases. This finding suggested that factors other than merely those affecting cholesterol metabolism, for example, specific effects on bile flow, might be behind the association between CBD stones and hypothyroidism.

A prospective study showed that even subclinical hypothyroidism is more common among gall stone patients. This study investigated the prevalence of previously undiagnosed subclinical hypothyroidism in clinically euthyreotic CBD

stone patients compared to non gallstone controls. It was found that 5.3% of the gall stone patients had subclinical hypothyroidism, defined as serum thyrotropin above the normal upper limit (6.0 mU/L), compared to only 1.4% in the control group. In women over 60 years, the prevalence of subclinical hypothyroidism was as high as 11.4% in the gall stone group compared to 1.8% among the control patients.

Finally in 2010, a large, medical registry-based study from Finland confirmed that hypothyroid patients did indeed seem to have a higher likelihood for gall stone treatment. In this study, the prevalence of gall stone treatments was investigated in patients with diagnosed hypothyroidism and compared to age, sex, and area of residence adjusted glaucoma (control) patients. Patients with other diseases were excluded to create a "purely" hypothyroid (or glaucoma) cohort of patients. Out of 14,334 patients in each group who met the inclusion criteria, 0.23% in the hypothyroid cohort and 0.16% in the control cohort had been treated for gall stones. The groups did not differ in the number of gall stone treatments before the diagnosis of hypothyroidism or glaucoma, but after these diagnoses there were 56% more gall stone treated individuals in the hypothyroid cohort than in the control cohort. This may suggest that the higher risk for gall stones in hypothyroid patients may increase after taking medication for hypothyroidism. As the process of bile stone formation takes time, stone formation may have started during the untreated period of hypothyroidism and have been completed regardless

of thyroxine replacement therapy. This hypothesis is supported by the findings that both subclinical and clinical hypothyroidism are more common in gall stone patients. However, the question remains whether thyroxine replacement therapy is sufficient to cause the physiological effects of thyroxine, as it seems that even though thyroxine replacement therapy has been initiated, the gall stones do indeed form or continue to grow. It has also been reported that gallstones have dissolved after initiation of thyroxine therapy. It is possible that thyroxine replacement therapy is not sufficient, or not sufficient at all times of the day, in all patients to maintain normal sphincter of Oddi function, causing the formation of gall stones. These interesting findings raise a question about the mechanisms underlying the association, which is stronger between hypothyroidism and CBD stones than between hypothyroidism and gallbladder stones.

| ıthor | Year | Journal | Study type | Patient population | Patient number | Gender | Age (years) | Main finding | Risk factors other than hypothyroidism contributing to development of CBD stones |
|---|---|---|---|---|---|---|---|---|---|
| kinen al. [8] | 2001 | Hepato-gastroenterol | Retrospective | CBD stone patients, age and sex matched GB stone patients and controls | 168 | 65% F | >60 | Hypothyroidism: CBD 11%, GB 6%, controls 2% | Groups did not differ in any other diagnosed diseases |
| ukkarinen al. [4] | 2007 | J. Clin. Endocrinol. Metab. | Prospective, multicenter | Clinically euthyreotic CBD stone patients and nongallstone controls | 445 | 61% F | median 67 (range 18–98) | Subclinical hypothyroidism (TSH > 6.0 mU/L): CBD 5.3%, control 1.4%. | Groups did not differ in any other diagnosed diseases |
| | | | | | 161 | F | >60 | Subclinical hypothyroidism: CBD 11.4%, control 1.8% | Groups did not differ in any other diagnosed diseases |
| ukkarinen al. [5] | 2010 | Scand. J. Gastroenterol. | Medical registry-based cohort | Hypothyroid patients and age, sex, and area of residence adjusted glaucoma (control) patients | 28,668 | 68% F | median 62 (range 18–88) | CBD stone treatments: hypothyroid cohort 0.23%, control cohort 0.16%. After diagnosing hypothyroidism 56% more CBD stone treatments than after diagnosing glaucoma. | Patients with other diseases were excluded from the cohort to create a "purely" hypothyroid (or glaucoma) cohort of patients. Also no difference in any other medications between the groups. |

***How Hypothyroidism May Affect the Formation of Gall Stones?***

In general, the pathogenesis of gallstones is a complex process involving mechanisms affecting bile content and bile flow. There are several factors that may contribute to the formation of gall stones in hypothyroid patients. Based on the investigations currently available, it cannot be concluded whether hypothyroid individuals develop isolated gall stones or present with CBD stones in addition to gallbladder stones. *However, based on what is known about the effects of hypothyroidism on the formation of gallbladder and CBD stones, it seems likely that in hypothyroidism both the risk for gallbladder-originated as well as for de novo CBD stones is increased.*

In hypothyroidism, the lack of thyroxine

- ➢ decreases liver cholesterol metabolism resulting in bile cholesterol supersaturation, which in turn impairs the motility, contractility, and filling of the gallbladder, contributing to the retention of cholesterol crystals and to the nucleation and growth of gallstones
- ➢ diminishes bile secretion from hepatocytes resulting in impaired clearance of precipitates from the bile ducts
- ➢ reduces SO relaxation resulting in delayed bile flow and thus the formation and accumulation of CBD stones.

***Hypothyroidism May Reduce Hepatic Bile Secretion :***

In a prospective study in humans, the dynamic Tc99m HIDA biligraphy performed in the acute hypothyroid stage after thyroidectomy showed that the hepatic maximal uptake and appearance of radioactivity in the large bile ducts at the hepatic hilum was similar to the euthyreotic stage in the same patients. This suggested that hepatocytic bile secretion may not be significantly reduced in humans in the early phase of hypothyroidism. Thus decreased bile hepatic secretion may have at least some impact on the delayed bile flow in prolonged hypothyroidism.

***Hypothyroidism Reduces Bile Flow into the Duodenum :***

In a prospective human study, hepatic clearance was significantly decreased and the hilum-duodenum transit time had a tendency to increase in the hypothyroid stage after thyroidectomy, when compared to the euthyreotic stage in the same patients. As the hepatic maximal uptake and the appearance of radioactivity in the large bile ducts at the hepatic hilum were similar in the hypothyroid and euthyreotic stages of this study, the findings are hardly attributable to different hepatic secretion but strongly suggest that bile flow into the duodenum is reduced in the hypothyroid stage. This could be due to changes in bile composition and gallbladder motility, and because of changes in the resistance to flow, that is, in the SO motility.

***Hypothyroidism Leads to Impaired SO Relaxation :***

The existence of gastrointestinal hypoactivity in hypothyroidism has been well known for decades. For example, the effect of thyroxine has been documented in anal canal pressure and in lower esophageal sphincter pressure. The effect of THs on smooth muscle contraction depends on the smooth muscle type and the species studied. THs have a direct, relaxing effect on vascular smooth muscle contractility. This effect is mediated by intranuclear binding of TH to the TR, and partly by nongenomic mechanisms involving extranuclear sites of action. The potassium (K+) channel blocker glibenclamide attenuates triiodothyronine-induced vasodilatation in rat skeletal muscle arteries, and triiodothyronine-induced vasodilatation may thus be mediated by ATP-sensitive $K^+$- channels.

Since Sandblom et al. first demonstrated the hormonal action of cholecystokinin (CCK) on the SO in 1935, several other hormones have been shown to affect SO activity. *In 2001, it was shown for the first time that thyroxine has a direct effect on SO contractility in physiological concentrations in pig experiments.* Triiodothyronine had a similar effect on thyroxine, whereas cortisone, estrogen and testosterone had no effect. Thus the effect of THs is not an unspecific effect of any hormone. Since the effect of thyroxine on the precontracted SO is relaxing, the absence/insufficient concentration of thyroxine may result in increased tension of the SO in hypothyroidism.

***Mechanisms by Which Thyroxine Mediates SO Relaxation :***

Several examinations were performed to determine how the relaxant effect of thyroxine on SO is mediated. The experiments with α- and β-adrenoceptor antagonists, NO synthesis inhibitor, and the elimination of nerve function with tetrodotoxin showed that the thyroxine-induced relaxation of SO is not mediated via neural effects. Human SO was shown to express TR β1 and β2. The presence of TRs in the SO is necessary but not sufficient evidence that thyroxine exerts its prorelaxant effect via a hormone-receptor complex action. The prorelaxant effect of thyroxine is probably partly mediated via transporter proteins, and partly via binding to nuclear receptors, subsequently leading to the activation of $K^+$ channels. The opening of $K^+$ channels is followed by hyperpolarisation, which closes cell membrane $Ca^{2+}$ channels, reduces $Ca^{2+}$ influx, and results in reduced contraction of the SO smooth-muscle cell in response to any specific stimulus.

***Conclusions and Clinical Implications :***

In summary, several recent studies report an association between hypothyroidism, or subclinical hypothyroidism, and gall stones. The higher prevalence of hypothyroidism in gall stone patients compared to gallbladder stone patients suggests that not only changes in the cholesterol metabolism, or bile excretion rate, but particularly changes in the function of the SO that may underline the association between gall stones and hypothyroidism. It remains to be

removed are at an increased risk to develop CBD stones when compared to euthyroid individuals in the same situation.

It seems likely that the lack of thyroxine in hypothyroidism gives rise to a reduction in bile flow in many ways. In addition to the increased cholesterol load in bile and the reduced bile secretion rate, the deficiency of the pro relaxant effect of thyroxine on the SO appears to be a crucial factor leading to the reduced bile flow in hypothyroidism.

The initial formation of bile cholesterol crystals may begin during the untreated period of hypothyroidism, and the stones may continue to develop or mature even after the thyroxine replacement therapy has begun. It is possible that thyroxine replacement therapy is not sufficient in all patients to maintain normal SO function, causing increased risk of CBD stone formation. Studies with subclinical hypothyroid patients have demonstrated that a positive effect on the changes in the serum cholesterol level, on cardiovascular effects, or on neuromuscular symptoms may be achieved with early replacement treatment with thyroxine, and it can be assumed that patients at risk of forming gall stones due to subclinical hypothyroidism may also benefit from such early treatment. *Most importantly, when treating patients with gall stones or microlithiasis, clinicians should be aware of the possible hypothyroid background and consider examining the thyroid function, at least in female patients over 40 years of age, in which group the prevalence of clinical and subclinical hypothyroidism is the highest.*

# Chapter 3

# MATERIALS AND METHODS

# MATERIALS AND METHODS

**3.1 Type of study** : Prospective and Observational Study

**3.2 Study approval :** Prior to commencement of this study - Thesis & Ethical Committee of Madras Medical College and Rajiv Gandhi Government General Hospital, chennai had approved the thesis protocol.

**3.3 Place of study :** Rajiv Gandhi Government General Hospital

**3.4 Period of study :** Duration starting from 01 July 2014 to 30 June 2015

**3.5 Sample size :** 195 cases with comparative control group of 100 cases

**3.6 Selection of patients:**

a) **Sampling method-** Purposive**.**

b) **Inclusion criteria-** Patients with symptomatic or asymptomatic cholelithiasis or choledocholithiasis

c) **Exclusion criteria - -** Acalculous Cholecystitis

**3.7 Study procedure:**

Method of sampling was non-random, purposive. After admission short history was taken and physical examination was conducted on each patient admitted in surgery department with features suggestive of extrahepatic biliary lithiasis. Baseline investigations, as routinely required, were done, followed by imaging studies. Patients were then explained about their disease process and the possible line of management. All the necessary information regarding the study was explained to the patients or their valid guardian. Informed written consent was taken from the patients or their guardian willing to participate in the study. Detailed history was taken from the study group to establish proper diagnosis. Thorough physical examination was done in each case. Data collection sheets were filled in by the investigator himself. All of the preoperative factors related to the patient were noted down in the data sheet. After proper evaluation and preparation, patients who required surgical management were taken up for surgery. Strict aseptic precautions were followed during the operation. Meticulous techniques were practiced as far as possible. The operation procedure and related peroperative factors were observed directly and recorded in the data collection sheet instantly. After completing the collection of data it was compiled in a systematic way.

### 3.8 Operational definitions:

**Cholelithiasis :** a condition marked by presence of calculi in the gallbladder

**Choledocholithiasis :** a condition marked by presence of calculi in the common bile duct

**Hypothyroidism :** abnormally low activity of the thyroid gland, develops when the thyroid gland fails to produce or secrete as much thyroxine as the body needs

**Jaundice :** Those with **S**. bilirubin **>** 1.2 mg/ dl were recorded as jaundiced.

**Hypercholestrolemia :** an excess of cholesterol in the bloodstream.

**Diabetes :** Those known as diabetic from history and those with RBS more than 11 m mol/ l were included as diabetic.

**ERCP :** (short for endoscopic retrograde cholangiopancreatography) is a procedure used to diagnose and treat diseases of the gallbladder, biliary system, pancreas, and liver.

**Types of operations:** were recorded during each operation.

### 3.9 Variables studied:

**Dependent variable:** Hypothyroidism (TFT).

**Independent variables:**

i. Age

ii. Sex

iii. Co-morbidities: COPD, jaundice, diabetes, obesity and malnutrition

iv. Lipid Profile

v. Ultrasonogram and CECT findings

vi. Types of operations

vii. Bile stone analysis

### 3.10 Ethical consideration

All the patients/ legal guardians were given an explanation of the study and about the investigative and operative procedures with their merits and demerits, expected results, and possible complications. If he/she agreed then the case had been selected for this study. The study did not involve any additional investigation or any significant risk. It did not cause economic burden to the patients. The study was approved by the institutional review board prior to commencement of data

collection. Informed consent was taken from each patient/guardian. Data were collected by approved data collection form.

### 3.11 Data collection

Data were collected by pre-tested structured questionnaire. Data were collected from all the respondents by direct interview after getting informed written consent from them or from their legal guardian.

### 3.12 Data analysis

Data analysis was done both manually and by using computer. Calculated data were arranged in systemic manner, presented in various table and figures and statistical analysis was made to evaluate the objectives of this study with the help of Statistical Package for Social Science (SPSS).

# CHAPTER 4

# RESULTS

# RESULTS

This prospective and observational study was carried out to determine the prevalence of hypothyroidism among patients admitted with cholelithiasis and choledocholithiasis. One hundred and ninety five patients fulfilling the inclusion criteria from Surgery department of Madras Medical College and Rajiv Gandhi Government General Hospital during the period of 1 July 2014 to 30 June 2015. All cases were evaluated clinically. Only essential investigations necessary for diagnosis and preoperative assessment were carried out before operations. All patients underwent thyroid function test in addition. The patients of both sexes and different ages were included in the study. A comparative control consisting of hundred patients of similar age and sex profile admitted for conditions other than gall stone disease were also evaluated for hypothyroidism. The results obtained are as follows.

**Table 1 : Age and Sex Distribution of patients with gall stone**

| Age / Sex | Male | Female | Total |
|---|---|---|---|
| 20 - 29 | 6 (3.1) | 16 (8.2) | 22 (11.3) |
| 30 - 39 | 6 (3.1) | 47 (24.1) | 53 (27.2) |
| 40 - 49 | 14 (7.18) | 40 (20.5) | 54 (27.6) |
| 50 - 59 | 9 (4.62) | 25 (12.82) | 34 (17.44) |
| > 60 | 12 (6.2) | 20 (10.25) | 32 (16.45) |
| Total | 47 (24.1) | 148 (75.9) | 195 (100) |

* Figures in parentheses indicates percentages

The mean age of patients was 44.75 years with more than half of the patients (55%) belonging to the 30 - 50 age group. The male to female ratio was ~ 1 : 3

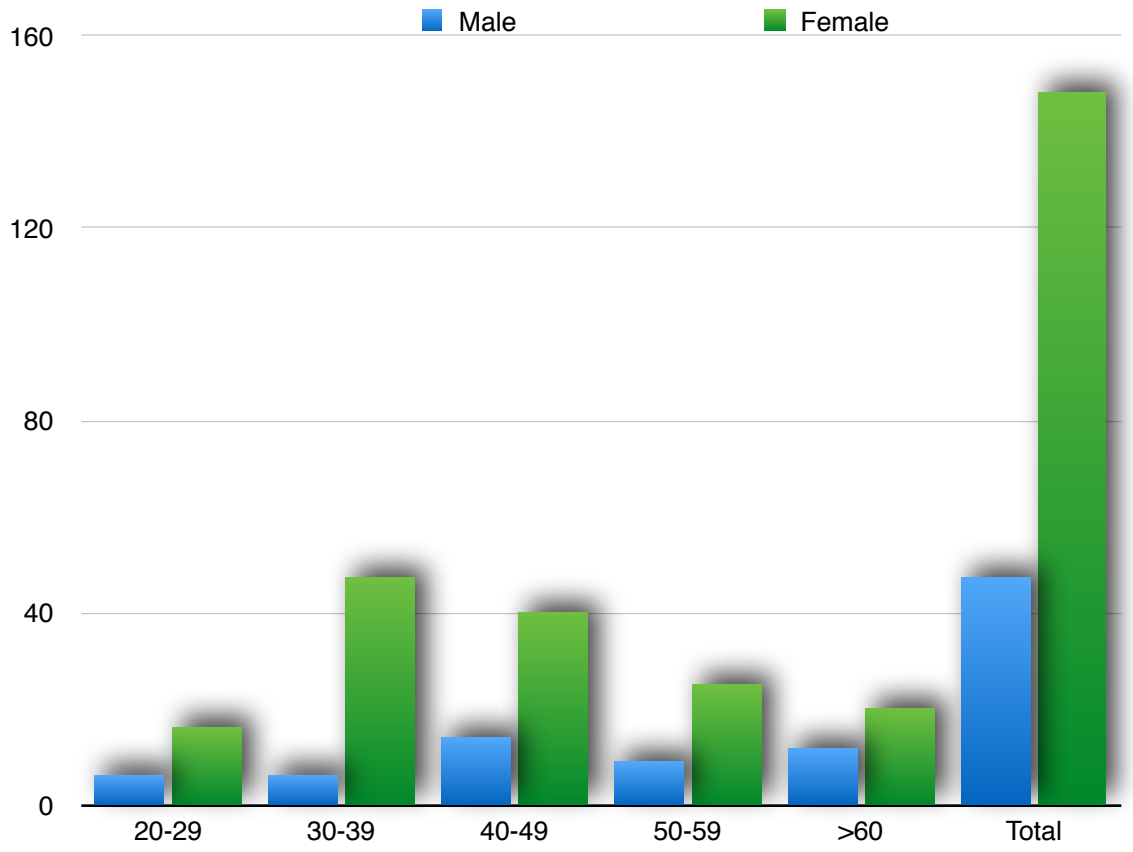


**Table 2 : Prevalance of Comorbid Factors in patient group**

| Co Morbid Factor | Number | Percentage |
|---|---|---|
| Diabetes Mellitus | 29 | 14.88 |
| Hypertension | 10 | 5.13 |
| DM & HTN | 5 | 2.56 |
| Bronchial Asthma | 3 | 1.54 |
| Others | 8 | 4.10 |
| No Comorbidity | 140 | 71.80 |
| Total | 195 | 100 |

Diabetes Mellitus was the most predominant comorbid factor seen in around 15 % of the patients. The other comorbid factors included systemic hypertension, bronchial asthma, TB etc.

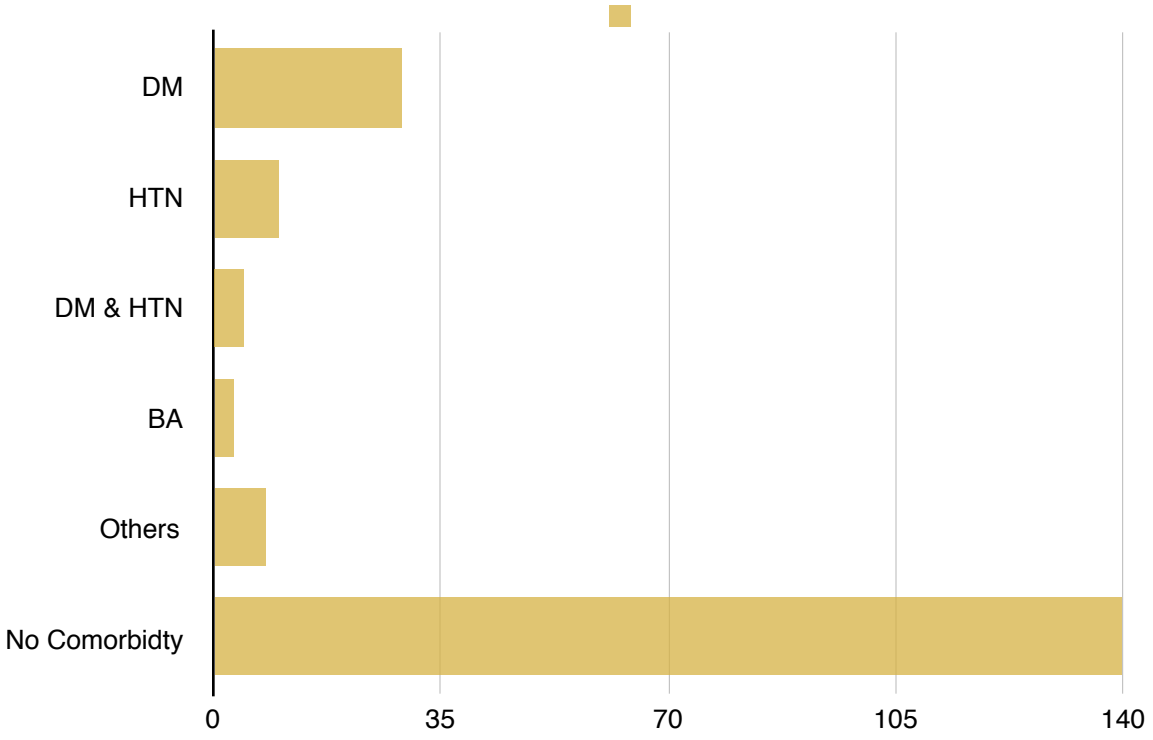


**Table 3 : Ultrasonogram / CECT features of patients in gallstone group**

| | Numbers | Percentage |
|---|---|---|
| Gallbladder stones - Single | 83 | 42.56 |
| Gallbladder stones - Multiple | 94 | 48.20 |
| Common bile duct stones | 8 | 4.10 |
| Both GB and CBD stone | 10 | 5.14 |
| **Total** | **195** | **100** |
| F/s/o Acute Cholelithiasis | 17 | 8.71 |

Around ninety percent of the patients presented with gallbladder stones, either single or multiple, with eight patients presenting with isolated CBD stones and ten patients with both gallbladder and CBD stones. 9% of the patients had features of acute cholecystitis.

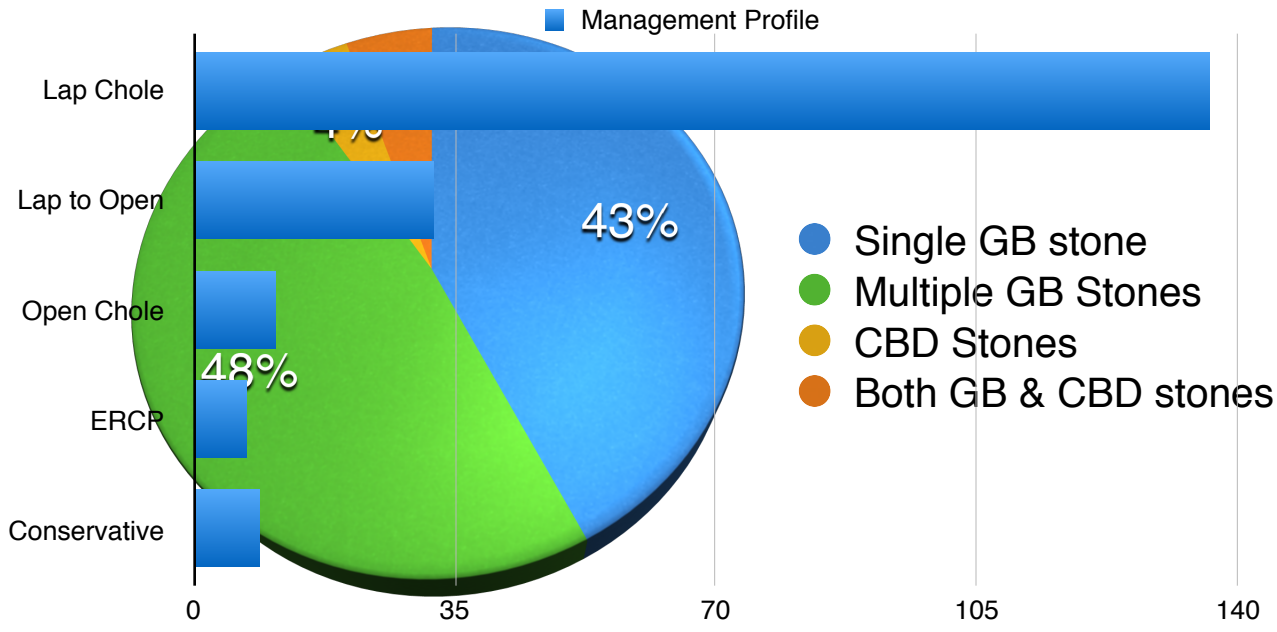


**Table 4 : Management profile of gallstone patients**

| | Numbers | Percentage |
|---|---|---|
| Laparoscopic Cholecystectomy | 136 | 69.74 |
| Lap converted to open | 32 | 16.40 |
| Open Cholecystectomy | 11 | 5.64 |
| ERCP | 7 | 3.60 |
| Conservative | 9 | 4.62 |
| **Total** | **195** | **100** |

85% of the patients underwent laparoscopic cholecystectomy, out of which nearly 16% had to be converted to open surgery. Eleven patients underwent elective open cholecystectomy while sixteen patients had non surgical treatment (ERCP/ Conservative) has the primary modality of treatment.

**Table 5 : Prevalance of hypothyroidism in patients with gallstones**

| | Numbers | Percentage |
|---|---|---|
| Clinical Hypothyroidism | 10 | 5.13 |
| Subclinical Hypothyroidism | 35 | 17.95 |
| Euthyorid | 150 | 76.92 |
| **Total** | **195** | **100** |

Fischers exact probability = 0.49 P - Value = 0.012 (Significant)

23.08 % of the patients with cholelithiasis / choledocholithiasis had thyroid function tests showing hypothyroidism, out of which nearly 18 % was subclinical hypothyroidism.

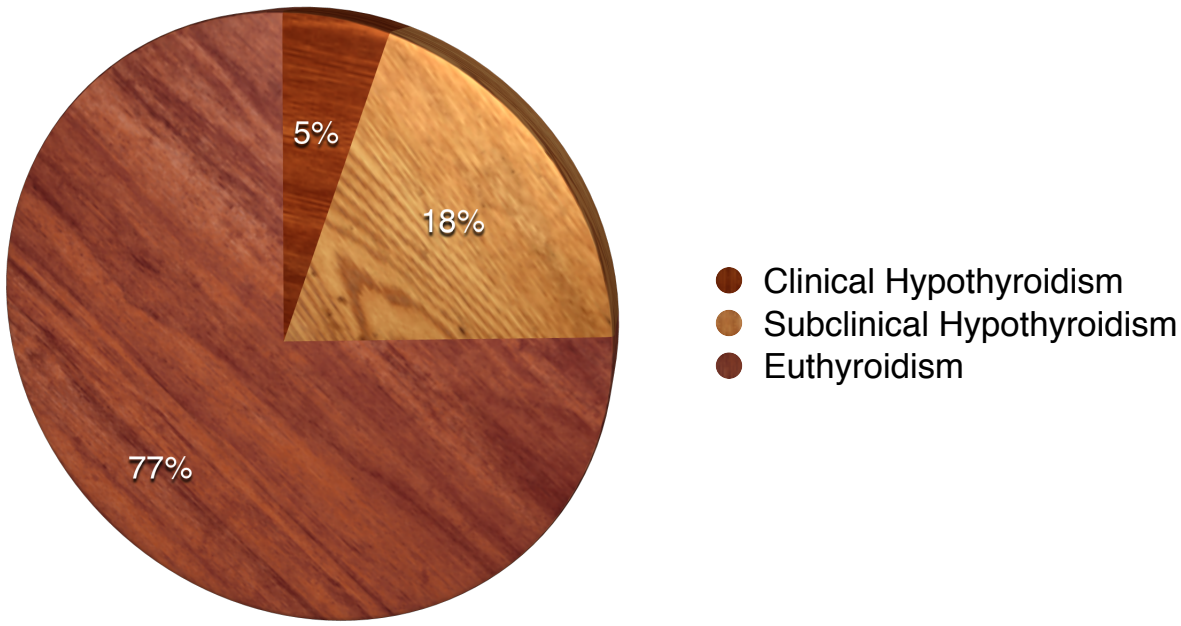
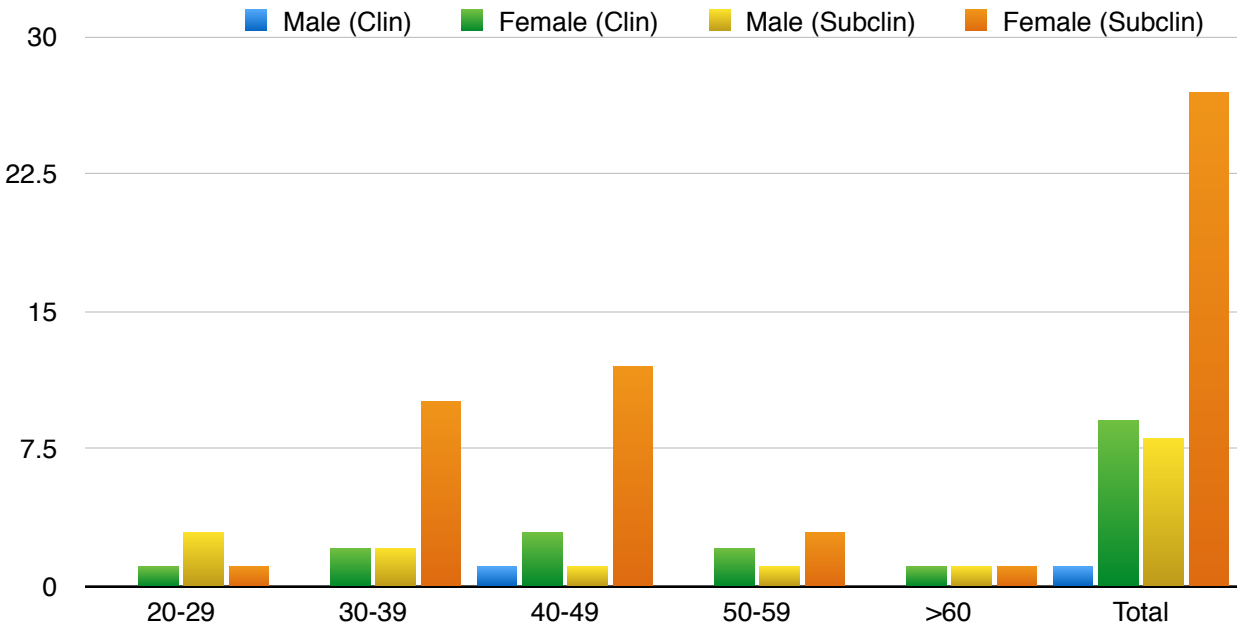


**Table 6 : Age and Sex distribution of Hypothyroidism in gallstone patients**

| Age / Sex | Clinical Hypothyroidism | | Subclinical Hypothyroidism | | Total |
|---|---|---|---|---|---|
| | Male | Female | Male | Female | |
| 20 - 29 | 0 | 1 | 3 | 1 | 5 |
| 30 - 39 | 0 | 2 | 2 | 10 | 14 |
| 40 - 49 | 1 | 3 | 1 | 12 | 17 |
| 50 - 59 | 0 | 2 | 1 | 3 | 6 |
| > 60 | 0 | 1 | 1 | 1 | 3 |
| **Total** | **1 (0.05)** | **9 (4.6)** | **8 (4.1)** | **27 (13.85)** | **45 (23.08)** |
| **Total** | **Male - - 9 (4.6)** | | **Female - - 36 (18.46)** | | **45 (23.08)** |

$X^2 = 6.28$ df = 4 P = 0.12 (NS)

* Figures in parentheses indicates percentages




As noted, female sex and 30 - 50 age group was the predominant group, with more than 75 % of the hypothyroid pateints belonging to this group.

**Table 7 : Prevalence of hyperbilirubinemia and hypercholestrolemia in gallstone patients**

| | Hypothyroid | Euthyroid | Total |
|---|---|---|---|
| **Hyperbilirubinemia** | 7/45 (15.56) | 8/150 (5.33) | 15/195 (7.7) |
| **Hypercholestrolemia** | 4/45 (8.9) | 16/150 (10.67) | 20/195 (10.26) |

* Figures in parentheses indicates percentages

Hyperbilirubinemia was found in fifteen patients overall, but around 15% of hypothyroid patients had features of hyperbilirubinemia while only 5% of the euthyroid patients had hyperbilirubinemia. Hypercholestrolemia was seen in 10% of all patients which was equally distributed between both groups.

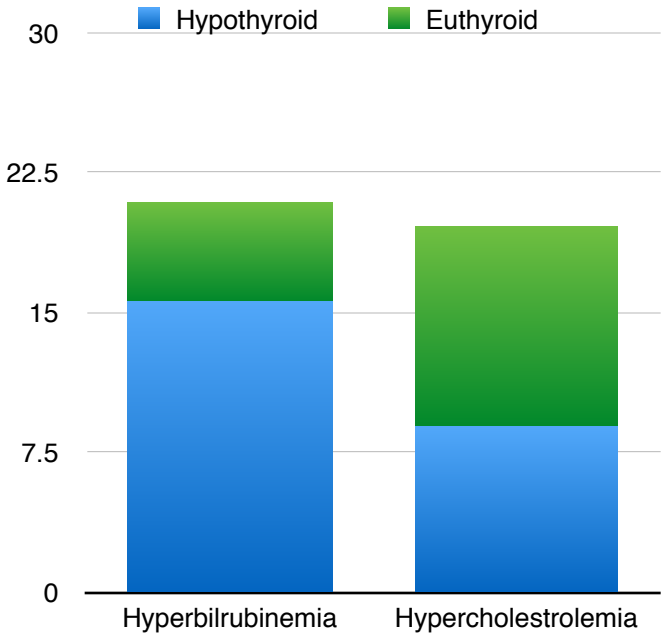


**Table 8 : Imaging findings in gallstone patients with hypothyroidism**

| | Numbers | Percentage |
|---|---|---|
| Gallbladder stones - Single | 15/83 | 33.33 |
| Gallbladder stones - Multiple | 22/94 | 48.89 |
| Common bile duct stones | 3/8 | 6.67 |
| Both GB and CBD stone | 5/10 | 11.11 |
| **Total** | **45/195** | **100** |
| F/s/o Acute Cholelithiasis | 6/17 | 13.33 |

* Figures in parentheses indicates percentages

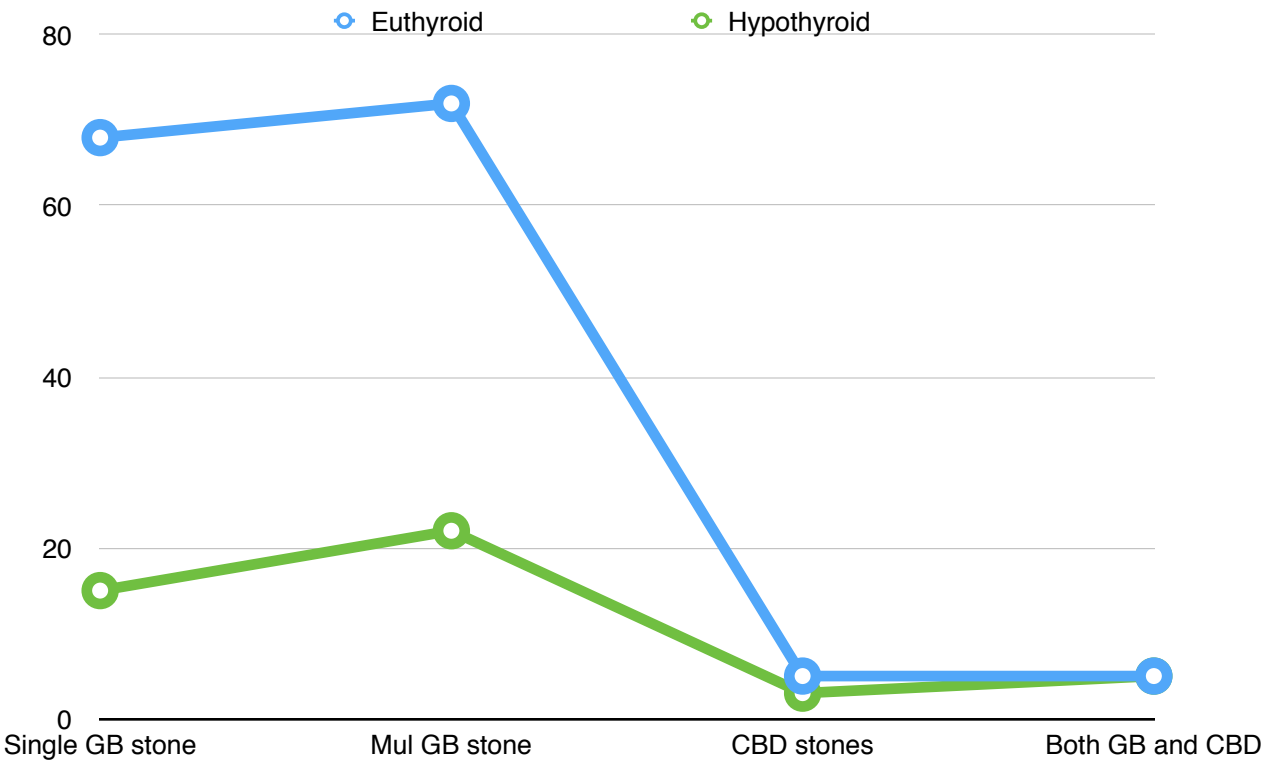


**Table 9 : Management profile in gallstone patients with hypothyroidism**

| | Numbers | Percentage |
|---|---|---|
| Laparoscopic Cholecystectomy | 23/136 | 51.11 |
| Lap converted to open | 13/32 | 28.89 |
| Open Cholecystectomy | 5/11 | 11.11 |
| ERCP | 2/7 | 4.44 |
| Conservative | 2/9 | 4.44 |
| **Total** | **45** | **100** |

The prevalence of laparoscopic converted to open and primary open surgeries was more in hypothyroid patients.

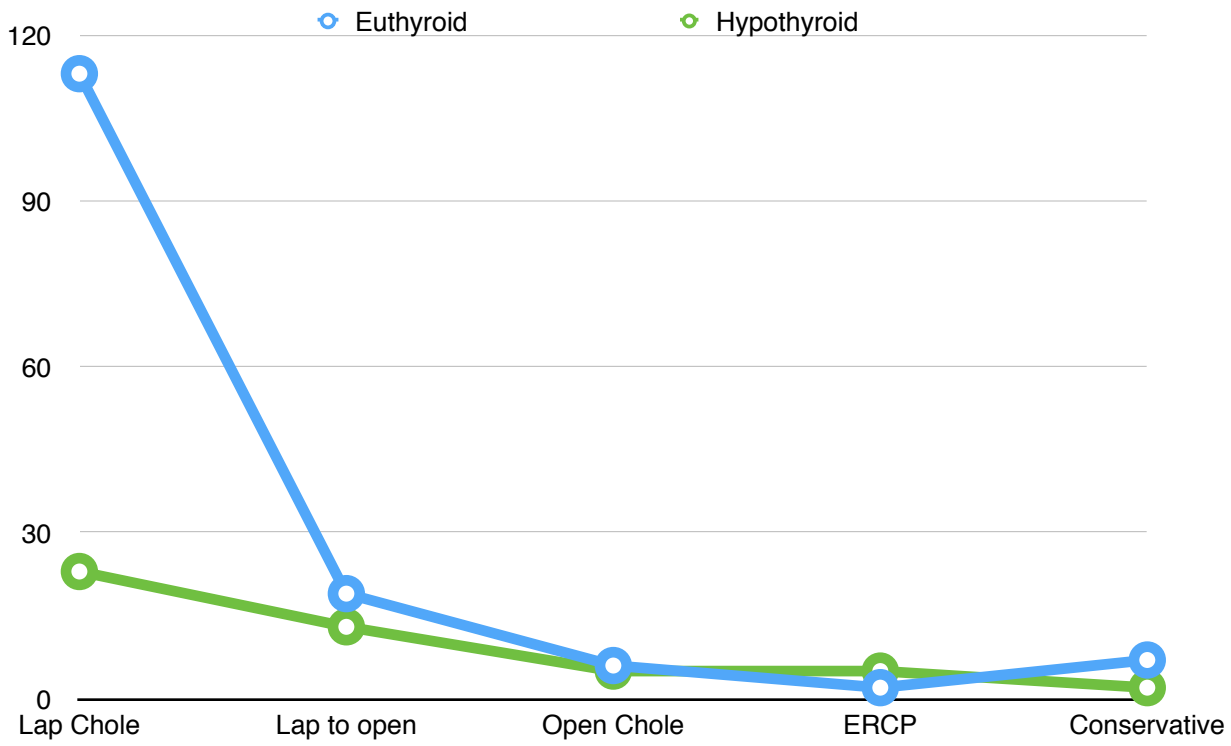
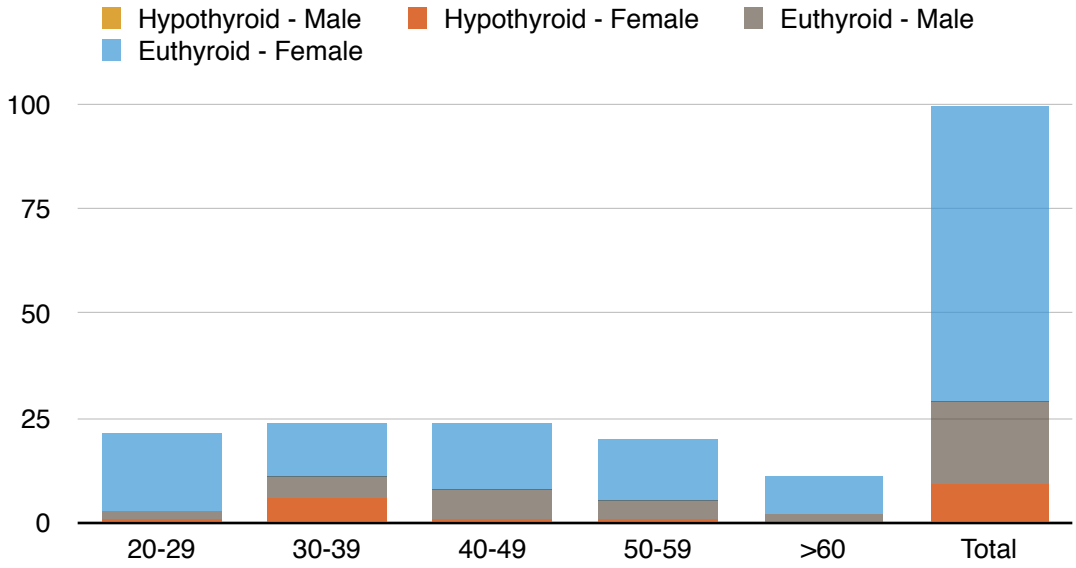


**Table 10 : Age and Sex distribution with relation to thyroid profile in the control group**

| Age / Sex | Hypothyroidism | | Euthyroid | | Total |
|---|---|---|---|---|---|
| | Male | Female | Male | Female | |
| 20 - 29 | 0 | 1 | 2 | 18 | 21 |
| 30 - 39 | 0 | 6 | 5 | 13 | 24 |
| 40 - 49 | 0 | 1 | 7 | 16 | 24 |
| 50 - 59 | 0 | 1 | 4 | 15 | 20 |
| > 60 | 0 | 0 | 2 | 9 | 11 |
| **Total** | **0** | **9** | **20** | **71** | **100** |
| **Total** | **Male - - 20** | | **Female - - 80** | | |
| **Total** | **Hypothyroidism - - 9** | | **Euthyroid - - 91** | | |

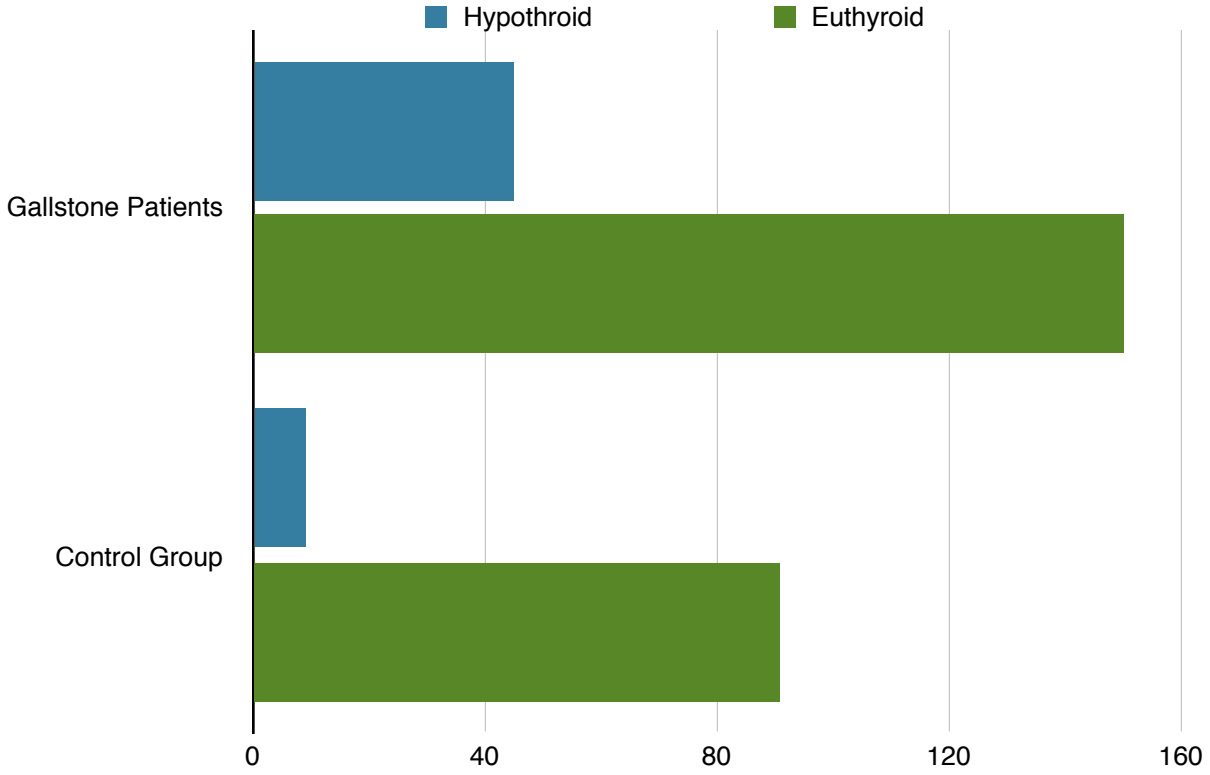



Out of the control group of hundred patients, only nine patients had hypothyroidism in their thyroid function patients. All of them were females with all except one in the 30 - 60 age group.

**Table 11 : Comparison of prevalence of hypothyroidism in control and patient groups**

| | Hypothyroidism | Euthyroidism | Total |
|---|---|---|---|
| Gallstone Patients | 45 (23.08) | 150 (76.92) | 195 |
| Control Group | 9 (9) | 91 (91) | 100 |

P - Value < 0.001 - - Highly Significant




As clearly evident, the prevalence of hypothyroidism was significantly more in the the gall patient group compared to the control group of patients.

# CHAPTER 5

# DISCUSSION

# DISCUSSION OF RESULTS

This prospective, observational and comparative study was conducted among 195 purposively selected patients with evidence of cholelithiasis or choledocholithiasis in Institute of General Surgery, Madras Medical College and Rajiv Gandhi Government General Hospital. The study was carried out with a view to determine the prevalence of hypothyroidism in patients with extra hepatic biliary lithiasis in view of determining its importance as a causative factor and to include thyroid function tests as part of routine workup in gallstone patients.

Age of 195 patients ranged from 22-80 years. Most of the patients (107, 55.80 %) were in between 30-49 years; with mean age 44.75 years and standard deviation 1.83 years. 24.1 % of the patients were males while 75.9% of the patients were females. The male to female ratio was ~ 1 : 3. So, it can be assumed that females are the predominantly involved group.

On analysing the comorbid factors, as expected, Diabetes Mellitus was the predominant comorbid factor, seen in 29 patients (14.88%), while ten patients (5.13%) had systemic hypertension, five patients (2.56%) had both, three patients (1.54%) had bronchial asthma, while eight patients had other factors like Tuberculosis, Epilepsy, Retroviral disease etc.

On evaluation of patients with ultra sonogram of the abdomen and Contrast Enhanced Computed Tomography (when indicated), it was seen that 90.76% patients had isolated gall bladder stones, either single or multiple. Single gall bladder stone was seen in eighty-three patients (42.56%) while multiple stones was seen in ninety four patients (48.20%). The size of the largest stone ranged from 4mm to 22 mm, with the average size being 10mm. Isolated common bile duct stones was seen in eight patients (4.1%). Of these eight patients, one patient had underwent cholecystectomy earlier and had presented with common bile duct stones at present. Both gall bladder and common bile duct stones was seen in ten patients (5.14%).

Acute cholecystitis, evident by clinical features of fever, jaundice & right hypochondrial pain and imaging findings of pericholecystic fluid collection and thickened gall bladder wall was seen in seventeen patients (8.71%).

Proceeding to management, 168 patients underwent primary laparoscopic cholecystectomy has the initial line of management. Out of these 168 patients, 136 patients (69.74%) had successful laparoscopic surgery while in 32 patients (16.4%) it had to be converted to open surgery. The reasons for conversion to open surgery included dense adhesions, bleeding and technical reasons. Eleven patients had

open cholecystectomy as the primary modality of treatment, the most common indication being acute cholecystitis. Nine patients (4.62%) , all of them cases of acute cholecystitis, were managed conservatively followed by interval cholecystectomy in five patients. One patient was lost in follow up while three patients were managed conservatively alone. Seven patients (3.6%), all patients with common bile duct stones, underwent ERCP with/without stenting, followed by either laparoscopic or open cholecystectomy with common bile duct exploration.

Thyroid function tests showed out of the total 195 patients, forty five patients (23.08%) patients had evidence of hypothyroidism. Out of these 45 patients, ten patients (5.13%) had clinically evident hypothyroidism, in the form of goitre or clinical symptoms, while thirty five patients (17.95%) had subclinical hypothyroidism, evident by raised TSH values alone. Out of these forty five patients, only five patients were previously known cases of hypothyroidism, which essentially meant that ninety percent of the hypothyroidism were in the previously undiagnosed group. P - Value of the prevalence was found to be 0.012 which denotes a significant level of prevalence.

Out of the ten patients with clinical hypothyroidism, nine patients were females (4.6%) and one male patient. In the subclinical hypothyroidism group, twenty seven patients (13.85%) were females and eight patients (4.1%) were males. Thirty one patients (16%, 70%) belonged to the 30 - 49 age group. This table shows us that female patients especially middle aged females (30 - 50 years), were the predominantly involved group, with a significant number of these patients having undetected hypothyroidism. Age and Sex based distribution analysed by Chi Square test showed no significance with p value of 0.12.

Blood investigations showed features of hyperbilirubinemia in fifteen patients (7.7%), out of which seven patients belonged to the hypothyroid group while eight patients belonged to the euthyroid group. Significantly 15.56 % of hypothyroid patients had hyperbilirubinemia while only 5.33% of the euthyroid patients had hyperbilirubinemia.

Hypercholestrolemia, indicated by elevated cholesterol and LDL levels, was seen in twenty patients (10.26%). 9% of patients with hypothyroidism had hypercholestrolemia while 10.7% of euthyroid patients had hypercholestrolemia, showing no significant difference between both the groups.

Analysis of the diagnostic studies in gallstone patients with hypothyroidism showed fifteen patients had single gall bladder stone, twenty two patients had multiple gallstones, three patients had common bile duct stones and five patients had both gall bladder and common bile duct stones. In total eighteen patients had common bile duct stones, either isolated or along with gallbladder stones. Of these eighteen patients, ten patients had hypothyroidism, indicating patients with common bile duct stones had more chances of being hypothyroid. Importantly, one patient had underwent laparoscopic cholecystectomy for gallbladder stones three years ago, who was not tested for thyroid function at that time, had presented with common bile duct stones at present and was found to be hypothyroid. Six patients had features suggestive of acute cholecystitis.

Of the 45 gallstone patients with hypothyroidism, twenty three (51.11%) patients underwent successful laparoscopic cholecystectomy. Thirteen patients (28.9%) were attempted for laparoscopic cholecystectomy but had to be converted to open surgery, with the predominant reason being adhesions and bleeding. Five patients (11.11%) underwent elective open cholecystectomies. Two patients underwent ERCP and two other patients were managed conservatively. The conversion rate was found to be higher in the hypothyroid group compared to the

euthyroid group (29% vs 13%).

A control group of 100 patients admitted in Institute of General Surgery, MMC for diseases other than biliary pathology were purposively selected and thyroid function tests was done. The group had twenty male and eighty female patients, evenly distributed across all age groups. Thyroid function tests showed nine patients (9%) had hypothyroidism. All the nine patients were females. Six of these nine patients belonged to the 30 - 39 age bracket. There was one patient each in the 20-29, 40-49 and 50-59 age brackets.

Comparing the control group of patients with the gallstone patients group showed that while only 9% of patients in control group had hypothyroidism, more than 23% of gallstone patients had subclinical/clinical hypothyroidism. This indicates the significantly increased ($p$ value $< 0.001$) prevalence of hypothyroidism among the extra hepatic biliary lithiasis patients.

## LIMITATIONS OF THE STUDY

As this study has been carried out over a limited period of time with a limited number of patients and there was lack of financial and infrastructural support, it could not have been large enough to be of reasonable precision. The follow up period was not long enough to comment about recurrence in patients with hypothyroidism and the response of the patients to thyroid medications. More number of patients with common bile duct stones, especially those recurring following cholecystectomy has to be analysed to underline the importance of hypothyroidism as one of the causative factors of gallstones. All the facts and figures mentioned here may considerably vary from those of large series covering wide range of time, but still then, as the cases of this study were collected from a tertiary level hospital in our country, this study has some credentials in reflecting the facts regarding prevalence of hypothyroidism in gallstone patients and its possible correlation with the natural progression of the disease process.

## SUMMARY

Cholelithiasis and Choledocholithiasis are one of the commonly encountered diseases in a general surgery department. Multiple risk factors have been attributed to the development of gallstones which include age, female gender, obesity, high fat diet, family history etc. Patients usually present with right hypochondrial pain, dyspepsia, vomiting and occasionally fever or jaundice. Management includes diagnostic imaging studies followed by surgery, if indicated. Hypothyroidism is not an accepted or proven risk factor for gallstones as of yet. Several studies have postulated the possible pathophysiology behind the role of thyroxine in the normal physiology of the biliary system. No concurrence has been established

**Age and Sex Distribution :**

The most commonly involved age group in patients with gall stones was between 30 - 60, with incidence increasing with age. This correlates with the general literature that gallstones is a disease of the elderly. Female sex, is in itself a risk factor for developing gallstones with more than seventy five percent of the involved patients belonging to the female gender.

**Co Morbid Factors :**

Diabetes Mellitus is the prevalent comorbid factor, as cholelithiasis like previously told is a disease of the elderly. Other comorbid factors include hypertension, bronchial asthma etc. None of these seemed to have a significant

correlation with the disease process.

**Diagnostic Studies :**

In our study, common bile duct stones was found in upto ten percent of the patients. There were equal amounts of single and multiple gall bladder stones patients. Nine percent of patients presented as acute cholelithiasis. USG was sensitive in detecting stones in nearly all patients with CECT abdomen and MRCP required only in few cases

**Management :**

Laparoscopic Cholecystectomy was the mainstay of management with primary open cholecystectomy done in only eleven patients. Interval cholecystectomy, which was also done laparoscopically, was done in sixteen patients. Conversion to open was seen in significant amount of patients. This can be attributed to technical problems and as the study was done in a teaching institute and good percentage of surgeries was done by trainees.

**Hyperbilirubinemia :**

Deranged liver function test with raised bilirubin and liver enzymes was found in a minor number of patients with hypothyroid patients having increased risk of presenting with hyperbilirubinemia.

**Hypercholestrolemia :**

Lipid profile showed hypercholestrolemia and hypertriglyceridemia was seen in 20 patients with equal distribution among both the hypothyroid and

euthyroid groups. The concept of cholesterol metabolism derangement expected in hypothyroid patients which was postulated as a cause of gallstones in these patients, was not found to be exclusive for these patients. Moreover, among the Indian population, pure cholesterol stones are extremely rare, with pigment stones found in all our patients. Pigment stones is caused by both stasis and infection than by cholesterol metabolism dysfunction.

**Hypothyroidism and gallstones :**

Forty five patients in our study had gallstones showing a prevalence of nearly one fourth of the patients being affected. When compared to a control group, there was a highly significant level of prevalence of hypothyroidism in gallstone patients. The prevalence of hypothyroidism in common bile duct stones was even more significant with more than fifty percent of those patients having hypothyroidism. Significantly a post cholecystectomy patient presenting with CBD stone was hypothyroid. The incidence of complications was also more in the hypothyroid group.

All these indicated a pathophysiological role of thyroxine in gallstone formation, apart from the supposed lipid metabolism abnormalities. The increased prevalence in CBD stones in these cases and incidence of recurrent stones indicates thyroxine plays a vital role in bile flow and gallbladder emptying. This has been substantiated by physiological studies in animal models by Johanna Laukkarinen et al.

# CONCLUSION

This prospective ocservational type of study was conducted in Institute of General Surgery, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, from 1 July 2014 to 30 June 2015. It can be concluded from the findings of the study that hypothyroidism is a highly probable risk factor for development of hypothyroidism especially for middle aged females. Undetected and untreated hypothyroidism in such patients will result in persistence of the basic pathophysiology responsible for the initial disease process resulting in recurrence and complications. So, it can be assumed that patients at risk of forming gall stones due to hypothyroidism will benefit from early treatment. Most importantly, when treating patients with cholelithiasis or choledocholithiasis, clinicians should be aware of the possible hypothyroid background and consider examining the thyroid function, at least in female patients over 40 years of age, in which group the prevalence of clinical and subclinical hypothyroidism is the highest.

## RECOMMENDATIONS

On the basis of the findings of the study, the following recommendations can be made:

1. Evaluation of thyroid profile should be a part of general workup in patients with both cholelithiasis and choledocholithiasis.
2. Proper evaluation and preoperative preparation in patients with hypothyroidism, anticipating complications.
3. The patients with subclinical / clinical hypothyroidism should be started with appropriate thyroid medications.
4. Further research is necessary in large scale for guidance regarding management profile for patients with coexistent hypothyroidism and gallstones.
5. Physiological and pharmacological studies necessary to determine the exact pathophysiology.

## BIBLIOGRAPHY


1. Healey J E, Schroy P C 1953 Anatomy of biliary ducts within the human liver; analysis of prevailing pattern of branchings and major variations of biliary ducts. Arch Surg 66: 599–616
2. Bioulac-Sage P, Le Bail B, Balabaud C 1991 Liver and biliary tract histology. In: McIntyre N, Benhamou J-P, Bircher J, Rizzetto M, Rodes J (eds) Oxford textbook of clinical hepatology Vol 1.Oxford University Press: Oxford: pp 12–20
3. Suzuki et al., 2000. Suzuki M, Akaishi S, Rikiyama T, Naitoh T, Rahman MM, Matsuno S: Laparoscopic cholecystectomy, Calot's triangle, and variations in cystic arterial supply. *Surgical Endoscopy* 2000; 14:141-144.
4. IHPBA Brisbane, 2000. IHPBA Brisbane 2000 Terminology of Liver Anatomy and Resections
5. Marve GM: Nerves and hormones interact to control gallbladder function. News Physiol Sci 13:64, 1998.
6. Portincasa P, Di Ciaula A, vanBerge-Henegouwen GP:Smooth muscle function and dysfunction in gallbladder disease. Curr Gastroenterol Rep 6:151, 2004.
7. Russell DW: The enzymes, regulation, and genetics of bile acid synthesis. Annu Rev Biochem 72:137, 2003.
8. Johnson LR: Gastrointestinal Physiology, 6th ed. St. Louis: Mosby, 2001.
9. Kidd JF, Thorn P: Intracellular Ca2+ and Cl- channel activation in secretory cells. Annu Rev Physiol 62:493, 2000.
10. Fuchs M: Bile acid regulation of hepatic physiology: III. Regulation of bile acid synthesis: past progress and future challenges. Am J Physiol Gastrointest Liver Physiol 284:G551, 2003.
11. Chiang JY: Bile acid regulation of hepatic physiology: III. Bile acids and nuclear receptors. Am J Physiol Gastrointest Liver Physiol 284:G349, 2003.

12. Brett M, Barker DJ. The world distribution of gallstones. Int J Epidemiol. 1976;5:335.
13. Nakeeb A, Comuzzie AG, Martin L, et al. Gallstones: genetics versus environment. Ann Surg. 2002;235:842.
14. Brasca A, Berli D, Pezzotto SM, et al. Morphological and demographic associations of biliary symptoms in subjects with gallstones: findings from a population-based survey in Rosario, Argentina. Dig Liver Dis. 2002;34:577.
15. Attili AF, De Santis A, Capri R, et al. The natural history of gallstones: the GREPCO experience. The GREPCO Group. Hepatology. 1995;21:655.
16. Bellows CF, Berger DH, Crass RA. Management of gallstones. Am Fam Physician. 2005;72:637.
17. Strasberg SM. The pathogenesis of cholesterol gallstones a review. J Gastrointest Surg. 1998;2:109.
18. Stewart L, Oesterle AL, Erdan I, et al. Pathogenesis of pigment gallstones in Western societies: the central role of bacteria. J Gastrointest Surg. 2002;6:891.
19. Trowbridge RL, Rutkowski NK, Shojania KG. Does this patient have acute cholecystitis? JAMA. 2003;289:80.
20. Fletcher DR. Gallstones. Modern management. Aust Fam Physician. 2001;30:441.
21. Ko C, Lee S. Epidemiology and natural history of common bile duct stones and prediction of disease. Gastrointest Endosc. 2002;56:S165.
22. Lilly MC, Arregui ME. A balanced approach to choledocholithiasis. Surg Endosc. 2001;15:467.
23. Ross SO, Forsmark CE. Pancreatic and biliary disorders in the elderly. Gastroenterol Clin North Am. 2000;30:531.
24. Lilly MC, Arregui ME. A balanced approach to choledocholithiasis. Surg Endosc. 2001;15:467.

25. Ross SO, Forsmark CE. Pancreatic and biliary disorders in the elderly. Gastroenterol Clin North Am. 2000;30:531.
26. Hunter JG. Acute cholecystitis revisited: get it while it's hot. Ann Surg. 1998;227:468.
27. Lee HJ, Choi BI, Han JK, et al. Three-dimensional ultrasonography using the minimum transparent mode in obstructive biliary diseases: early experience. J Ultrasound Med. 2002;21:443.
28. Ralls PW, Jeffrey RB Jr, Kane RA, et al. Ultrasonography. Gastroenterol Clin North Am. 2002;31:801.
29. Wexler RS, Greene GS, Scott M. Left hepatic and common hepatic ductal bile leaks demonstrated by Tc-99m HIDA scan and percutaneous transhepatic cholangiogram. Clin Nucl Med. 1994;19:59.
30. Breen DJ, Nicholson AA. The clinical utility of spiral CT cholangiography. Clin Radiol. 2000;55:733.
31. Liu TH, Consorti ET, Kawashima A, et al. Patient evaluation and management with selective use of magnetic resonance cholangiography and endoscopic retrograde cholangiopancreatography before laparoscopic cholecystectomy. Ann Surg. 2001;234:33.
32. Magnuson TH, Bender JS, Duncan MD, et al. Utility of magnetic resonance cholangiography in the evaluation of biliary obstruction. J Am Coll Surg. 1999;189:63.
33. Kiviluoto T, Siren J, Luukkonen P, et al. Randomised trial of laparoscopic versus open cholecystectomy for acute and gangrenous cholecystitis. Lancet. 1998;351:321.
34. Lo CM, Liu CL, Fan ST, et al. Prospective randomized study of early versus delayed laparoscopic cholecystectomy for acute cholecystitis. Ann Surg. 1998;227:461.

35. Chikamori F, Kuniyoshi N, Shibuya S, et al. Early scheduled laparoscopic cholecystectomy following percutaneous transhepatic gallbladder drainage for patients with acute cholecystitis. Surg Endosc. 2002;16:1704.

36. Patel M, Miedema BW, James MA, et al. Percutaneous cholecystostomy is an effective treatment for high-risk patients with acute cholecystitis. Am Surg. 2000;66:33.

37. Tranter S, Thompson M. Comparison of endoscopic sphincterotomy and laparoscopic exploration of the common bile duct.Br J Surg. 2002;89:1495

38. Rhodes M, Sussman L, Cohen L, et al. Randomised trial of laparoscopic exploration of common bile duct versus postoperative endoscopic retrograde cholangiography for common bile duct stones. Lancet. 1998;351:159.

39. Byrne MF, Suhocki P, Mitchell RM, et al. Percutaneous cholecystostomy in patients with acute cholecystitis: experience of 45 patients at a US referral center. J Am Coll Surg. 2003;197:206.

40. Akhan O, Akinci D, Ozmen MN. Percutaneous cholecystostomy. Eur J Radiol. 2002;43:229.

41. Richards C, Edwards J, Culver D, et al. Does using a laparoscopic approach to cholecystectomy decrease the risk of surgical site infection? Ann Surg. 2003;237:358.

42. Flum DR, Dellinger EP, Cheadle A, et al. Intraoperative cholangiography and risk of common bile duct injury during cholecystectomy. JAMA. 2003;289:1639.

43. Biffl W, Moore E, Offner P, et al. Routine intraoperative ultrasonography with selective cholangiography reduces bile duct complications during laparoscopic cholecystectomy. J Am Coll Surg. 2001;193:272.

44. Halpin VJ, Dunnegan D, Soper NJ. Laparoscopic intracorporeal ultrasound versus fluoroscopic intraoperative cholangiography: after the learning curve. Surg Endosc. 2002;16:336.

45. Barwood NT, Valinsky LJ, Hobbs MS, et al. Changing methods of imaging the common bile duct in the laparoscopic cholecystectomy era in Western Australia: Implications for surgical practice. Ann Surg. 2002;235:41.

46. Feingold KR, Gavin LA, Schambelan M, Schriock E, Sebastian A and Stern JI (1993):The thyroid. In: Cecil essentials of medicine, pp 470-481. Eds. TE Andreoli, JC Bennett,CCJ Carpenter, F Plum and LH Jr Smith, W.B. Saunders Company, Philadelphia, PA.

47. Woeber KA (1997): Subclinical thyroid dysfunction. Arch Intern Med 157:1065-1068.

48. Woeber KA (2000): Update on the management of hyperthyroidism and hypothyroidism.Arch Intern Med 160:1067-1071.

49. Wuttke W (1989): The thyroid system. In: Human physiology, pp. 383-386. Eds. RF Schmidt and G Thews, Springer-Verlag, Berlin, Germany.

50. Vanderpump MP, Tunbridge WM, French JM, Appleton D, Bates D, Clark F, Grimley Evans J, Hasan DM, Rodgers H and Tunbridge F (1995): The incidence of thyroid disorders in the community: a twenty-year follow-up of the Whickham Survey. Clin Endocrinol 43:55-68.

51. J. Laukkarinen, J. Sand, S. Aittom¨aki et al., “Mechanism of the prorelaxing effect of thyroxine on the sphincter of Oddi,” Scandinavian Journal of Gastroenterology, vol. 37, no. 6, pp. 667–673, 2002.

52. J. M. Donovan, “Physical and metabolic factors in gallstonepathogenesis,” Gastroenterology Clinics of North America, vol. 28, no. 1, pp. 75–97, 1999.

53. L. Behar, K. Y. Lee, W. R. Thompson, and P. Biancani, “Gallbladder contraction in patients with pigment and cholesterol stones,” Gastroenterology, vol. 97, no. 6, pp. 1479–1484, 1989.

54. R. P. Jazrawi, P. Pazzi, M. L. Petroni et al., “Postprandial gallbladder motor function: refilling and turnover of bile in health and in cholelithiasis,” Gastroenterology, vol. 109, no. 2, pp. 582–591, 1995.

55. J. Laukkarinen, P. K¨o¨obi, J. Kalliovalkama et al., “Bile flow to the duodenum is reduced in hypothyreosis and enhanced in hyperthyreosis,” Neurogastroenterology and Motility, vol. 14, no. 2, pp. 183–188, 2002.

56. J. Laukkarinen, J. Sand, R. Saaristo et al., “Is bile flow reduced in patients with hypothyroidism?” Surgery, vol. 133, no. 3, pp.288–293, 2003.

57. J. Sand, H. Tainio, and I. Nordback, “Peptidergic innervation of human sphincter of Oddi,” Digestive Diseases and Sciences, vol. 39, no. 2, pp. 293–300, 1994.

58. J. Sand, P. Arvola, V. J¨antti et al., “The inhibitory role of nitric oxide in the control of porcine and human sphincter of Oddi activity,” Gut, vol. 41, no. 3, pp. 375–380, 1997.

59. J. Sand, I. Nordback, P. Arvola, I. P¨orsti, A. Kalloo, and P. Pasricha, “Effects of botulinum toxin A on the sphincter of Oddi: an in vivo and in vitro study,” Gut, vol. 42, no. 4, pp. 507–510, 1998.

60. J. Sand, P. Arvola, I. P¨orsti et al., “Histamine in the control of porcine and human sphincter of Oddi activity,” Neurogastroenterology and Motility, vol. 12, no. 6, pp. 573–579, 2000.

61. J. Sand, P. Arvola, and I. Nordback, “Calcium channel antagonists and inhibition of human sphincter ofOddi contractions,” Scandinavian Journal of Gastroenterology, vol. 40, no. 12, pp. 1394–1397, 2005.

# APPENDIX - I : ETHICAL COMMITTEE CLEAREANCE

## INSTITUTIONAL ETHICS COMMITTEE
### MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax: 044 25363970

**CERTIFICATE OF APPROVAL**

To

**Dr.L.Arun Kumar,**
Post Graduate,
Institute of General Surgery,
Madras Medical College,
Chennai – 600 003.

Dear **Dr.L.Arun Kumar,**

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"A study on prevalence of subclinical or clinical hypothyroidism in patients with extrahepatic biliary lithiasis"** No.33062014.

The following members of Ethics Committee were present in the meeting held on 03.06.2014 conducted at Madras Medical College, Chennai-3.

| | | |
|---|---|---|
| 1. | Dr. C.Rajendran, M.D, | -- Chairperson |
| 2. | Prof. Kalaiselvi, M.D,<br>Vice Principal, MMC, Ch-3 | -- Member Secretary |
| 3. | Prof. Nandhini, M.D,<br>Inst. of Pharmacology, MMC, Ch-3 | -- Member |
| 4. | Prof.G.Muralidharan, M.S,<br>Prof & HOD General Surgery, MMC, Ch-3 | -- Member |
| 5. | Prof.V.Padmavathi, M.D,<br>I/c. Director of Pathology, MMC, Ch-3 | -- Member |
| 6. | Thiru. S. Govindasamy, BA., BL | -- Lawyer |
| 7. | Tmt.Arnold Saulina, MA MSW | -- Social Scientist |
| 8. | Thiru.S.Ramesh Kumar,<br>Administrative Officer, MMC, Ch-3. | -- Lay Person |

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

Sd/Chairman & Other Members

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

5/6/14

Member Secretary, Ethics Committee

MEMBER SECRETARY.
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

# Appendix-II

**DATA COLLECTION SHEET**

**Title*: "A study on prevalence of clinical or subclinical hypothyroidism in patients with extrahepatic biliary lithiasis"***

**QUESTIONAIRE**

**PATIENT DETAILS:**

Name: Age: Sex:

IP No. :

**ON ADMISSION:**

Main Complaints :

Symptoms of Gallstone/CBD stones

H/o right upper quadrant pain :

H/o vomiting :

H/o dyspepsia :

H/o fever :

H/o jaundice :

Symptoms of Hypothyroidism

H/o weight gain :

H/o weakness/lethargy :

H/o intolerance to cold :

H/o constipation :

H/o swelling of face or limbs :

H/o loss of hair / dry skin :

Co – Morbid Illness :

Significant Past History :

# Appendix – III

# Statistical formula

## A. Sample size:

To determine the sample size, this formula was used; $n = \frac{z^2 pq}{d^2}$

Where,

$n$ = the desired sample size,

$z$ = the standard normal deviate, usually set at 1.96 at 5% level,

which corresponds to 95% confidence level,

p = proportion of population, q

= 1- p

d = the degree of accuracy level considered as 5.0 %,

which assumes 0.05

If population size, N < 10,000 than the required sample size is very much smaller which was calculated by the following formula –

$$n_f = \frac{n}{1 + \frac{n}{N}}$$

Where,

$n_f$ = the desired sample size, when population size, N < 10,000

n = the desired sample size, when population size, N > 10,000 N

= the roughly estimated population size.

**CLINICAL EXAMINATION:**

Pulse : BP :

RR : Temp :

Pallor : Icterus :

CVS : RS :

P/A :

Thyroid examination :

**INVESTIGATIONS :**

Hemogram : Renal Function Test :

Liver Function Test :

BT/CT : Blood Grouping : Fasting Lipid Profile :

ECG : CXR :

USG Abdomen :

Thyroid profile : T3 -- T4 -- TSH —

Operative Procedure :

**FOLLOW UP :**

B. Arrithmatic mean, $X = \frac{\sum fx}{N}$ (for grouped data)

C. Standard deviation , $SD = \sqrt{\overline{\sum (X- X)^2}}$

('O' indicates observed value and 'E' indicates expected value)

D. $$Z = \frac{P_1 - P_2}{\sqrt{\left[\frac{P_1 Q_1}{N_1} + \frac{P_2 Q_2}{N_2}\right]}}$$

$P_1$ indicates proportion in first group

$P_2$ indicates proportion in second group

$Q_1 = 100 - P_1$

$Q_2 = 100 - P_2$

$N_1$ indicates sample size of first group

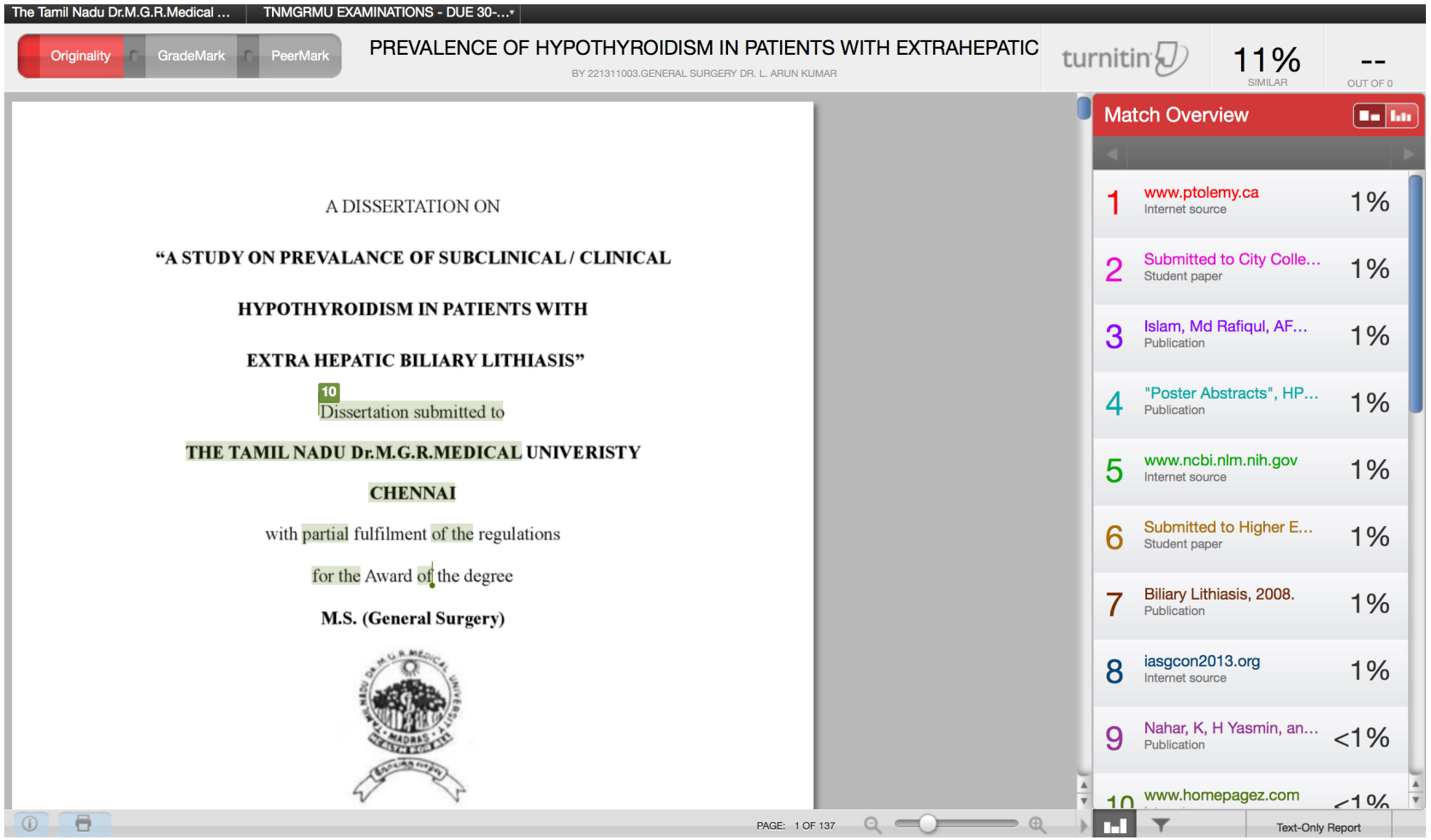
$N_2$ indicates sample size of second group.

E. $$SD = \sqrt{\frac{\sum (X - \bar{X})}{(N-1)}}$$

Here, $\bar{X}$ indicates mean value

$X$ indicates individual value

N indicates sample


The Tamil Nadu Dr.M.G.R.Medical ...
TNMGRMU EXAMINATIONS - DUE 30-...
Originality
GradeMark
PeerMark
PREVALENCE OF HYPOTHYROIDISM IN PATIENTS WITH EXTRAHEPATIC
BY 221311003.GENERAL SURGERY DR. L. ARUN KUMAR
turnitin
11%
SIMILAR
--
OUT OF 0
A DISSERTATION ON
"A STUDY ON PREVALANCE OF SUBCLINICAL / CLINICAL
HYPOTHYROIDISM IN PATIENTS WITH
EXTRA HEPATIC BILIARY LITHIASIS"
Dissertation submitted to
THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERISTY
CHENNAI
with partial fulfilment of the regulations
for the Award of the degree
M.S. (General Surgery)
PAGE: 1 OF 137
Match Overview
1 www.ptolemy.ca Internet source 1%
2 Submitted to City Colle... Student paper 1%
3 Islam, Md Rafiqul, AF... Publication 1%
4 "Poster Abstracts", HP... Publication 1%
5 www.ncbi.nlm.nih.gov Internet source 1%
6 Submitted to Higher E... Student paper 1%
7 Biliary Lithiasis, 2008. Publication 1%
8 iasgcon2013.org Internet source 1%
9 Nahar, K, H Yasmin, an... Publication <1%
10 www.homepagez.com
Text-Only Report

# APPENDIX V — MASTER CHART

| S.No | Name | Age | Sex | Ip No. | Symptoms of cholelithiasis | | | | | Symptoms of hypothyroidism | Co Morbidity | P/A (Tenderness) | Thyroid Examination | LFT | Lipid Profile | TFT | | | USG Abdomen | | | | Procedure/ Complications | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Dyspepsia | Right Hypo Pain | Vomiting | Fever | Jaundice | | | | | T.Bil/Dir.Bil/ SGOT/SGPT | Chol/TGL/ HDL | TSH | T3 (total /free) | T4 (Total /free) | No. of calculi | Size (largest) | Acute | CBD | | |
| 1 | Murugan | 48 | M | 102711 | + | + | - | - | - | - | - | - | N | 1.1/-/29/22/37 | 125/55/77 | 3.63 | 0.99 | 2.2 | Mul | 4mm | - | N | Lap to open, Bile leak, ERCP | |
| 2 | Kalaivani | 33 | F | 110237 | + | + | - | - | - | - | - | - | N | 0.5 | 224/139/40 | 4.42 | 2.3 | 1.45 | Mul | 6mm | - | N | Lap Chole | |
| 3 | Jeya | 34 | F | 112346 | + | + | + | - | - | - | - | - | N | 1.0 | 164/56/65 | 1.93 | 2.6 | 1.4 | Mul | 8mm | - | N | Lap to open | |
| 4 | Mangalakshmi | 58 | F | 115187 | + | - | - | - | - | - | BA | - | N | 1.2 | 155/81/63 | 0.60 | 3.1 | 1.38 | Sin | 9mm | + | N | Lap Chole | |
| 5 | Kala | 43 | F | 115702 | + | + | + | + | + | + | - | + | Goitre | 1.1 | 135/46/16 | >150 | 1.7 | 0.4 | Mul | - | - | N | Lap to open | Hypothyroid |
| 6 | Shakunthalamma | 40 | F | 109684 | - | + | + | + | - | - | - | - | N | 2.7/1.0/46/29 | 350/201/57 | 10.37 | 1.9 | 1.15 | Mul | 5mm | - | Dil | Open Chole | Hypothyroid |
| 7 | Pitchammal | 60 | F | 114654 | + | - | - | - | + | - | DM | + | N | 1.4/0.6/30/28 | 162/77/64 | 72.07 | 1.2 | 0.5 | Mul | 9mm | - | N | Lap Chole | Hypothyroid |
| 8 | Ambujam | 52 | F | 110548 | - | + | - | - | - | - | DM | - | N | 1.2/.7/18/27/34 | 128/77/67 | 4.3 | 1.9 | 1.12 | Mul | 10mm | - | N | Lap Chole | |
| 9 | Meena | 32 | F | 113762 | - | + | - | - | - | - | Post Chol | - | N | 4.5/1.9/60/22/226 | 139/86/84 | 7.58 | 3 | 1.12 | - | - | - | Dil with 8mm | Open CBD exploration | Hypothyroid |
| 10 | Kanniyammal | 35 | F | 110784 | + | + | + | - | - | - | - | - | N | .3/18/22/44 | 168/104/60 | 4.56 | 1.7 | 1.15 | Mul | 8mm | - | N | Lap Chole | |
| 11 | Mohana | 47 | F | 113222 | + | + | - | - | - | - | DM | - | N | 0.5 | 147/83/69 | 3.95 | 2.5 | 1.26 | Mul | 5mm | - | N | Lap Chole | |
| 12 | Vellangani | 48 | F | 113384 | + | + | + | + | + | - | DM/SHT | - | N | 1.2 | 121/100/56 | 5.98 | 2.5 | 1.01 | Sin | 5mm | - | N | Lap Chole | Hypothyroid |
| 13 | Vasantha | 54 | F | 111873 | - | + | - | - | - | - | DM/SHT | - | N | 1.1 | 139/94/56 | 4.78 | 2.3 | 1.33 | Mul | - | - | N | Lap Chole | |
| 14 | Sumathy | 36 | F | 117736 | + | + | - | - | - | - | - | - | Goitre | 1.4/0.6/38/46/40 | 158/81/56 | 3.6 | 3.22 | 1.67 | Mul | 5mm | - | N | Lap Chole | |
| 15 | Gowri | 50 | F | 117711 | + | + | + | + | - | - | DM | - | N | 1.0 | 133/66/50 | 2.04 | 2.6 | 0.87 | Sin | 10mm | - | N | Lap Chole | |
| 16 | Eswaran | 46 | M | 120134 | + | + | - | + | - | - | DM | - | N | 103/64/72 | 3.25 | 2.7 | 0.93 | Mul | 5mm | + | N | Lap to open | |
| 17 | Vivek | 24 | M | 126472 | + | + | - | + | - | - | - | + | N | 1.8 | 193/144/50 | 2.87 | 3.1 | 1.1 | Mul | 10mm | + | N | Lap Chole | |
| 18 | Ramesh Kumar | 43 | M | 122572 | + | - | - | - | - | - | - | - | N | 1.0 | 145/96/58 | 3.2 | 2.1 | 1.32 | Mul | 5mm | - | N | Lap to open | |
| 19 | Govindan | 25 | M | 126226 | - | + | - | + | - | - | - | + | N | 2.1/.9/23/81/110 | 245/187/40 | 7.6 | 2.52 | 0.76 | Mul | 7mm | + | Dil with 11mm | Open CBD exploration | Hypothyroid |
| 20 | Subramani | 55 | M | 121372 | + | + | - | - | - | - | SHT | - | N | 0.9 | 132/62/70 | 1.11 | 3.0 | 1.23 | Mul | 13mm | - | N | Lap Chole | |
| 21 | Gomathy | 34 | F | 126352 | + | - | - | - | - | - | - | - | N | 1.2 | 164/81/65 | 3.2 | 2.8 | 0.98 | Mul | - | - | N | Lap Chole | |
| 22 | Vanaja | 55 | F | 126931 | + | + | - | - | - | - | DM | - | N | 1.1 | 175/99/58 | 2.33 | 1.9 | 1.1 | Mul | 5mm | - | N | Lap to open | |
| 23 | Panjali | 45 | F | 125449 | + | - | - | - | - | - | - | - | N | 0.8 | 128/76/70 | 5.02 | 3.8 | 1.4 | Mul | 5mm | - | N | Lap Chole | |
| 24 | Chellammal | 65 | F | 127430 | + | + | - | + | + | + | DM | + | Goitre | 3.0/1.9/80/38/44 | 147/113/42 | 25.48 | 1.4 | 0.82 | - | - | + | Dil with 10mm | ERCP foll by open chole | Hypothyroid |
| 25 | Saroja | 70 | F | 119085 | + | + | - | - | + | - | - | + | N | 1.8/1.1/120 | 134/101/61 | 1.2 | 2.1 | 0.88 | Mul | 4mm | - | Dil | Open Chole | |
| 26 | Shakunthalamma | 35 | F | 113949 | + | + | - | - | - | - | DM | - | N | 1.1 | 168/108/44 | 17.22 | 2.3 | 1.1 | Mul | 7mm | - | N | Lap Chole | Hypothyroid |
| 27 | Aravathi | 50 | F | 125042 | - | + | - | - | - | - | - | - | N | 1.2/30/32/40 | 173/78/62 | 4.8 | 3.1 | 1.3 | Mul | 6mm | - | N | Lap Chole | |
| 28 | Sova Biswas | 28 | F | 71833 | + | + | + | - | - | - | - | - | N | 0.8/36/43/72 | 168/136/45 | 5.24 | 2.9 | 1.06 | Sin | 15mm | + | N | Lap to open | |
| 29 | Rajeshwari | 35 | F | 71818 | - | + | - | - | - | - | - | + | N | 0.8/22/24/42 | 252/171/35 | 1.08 | 2.8 | 1.16 | Mul | 10mm | + | 7.6mm | Lap Chole | |
| 30 | Abraham | 65 | M | 67697 | + | + | - | - | - | - | DM/SHT | + | N | 1.8/0.6/43/22/24 | 167/100/56 | 3.28 | 3.2 | 1.54 | Sin | 7mm | - | N | Lap to open | |
| 31 | Ranjitham | 48 | F | 76882 | + | + | + | - | - | - | BA | + | N | 0.5/20/18/97/76 | 220/121/43 | 4.01 | 4.2 | 1.38 | Mul | 5mm | - | N | Lap Chole | |
| 32 | Anjali Devi | 48 | F | 76734 | + | + | - | - | - | - | - | + | N | 1.4/1/36/48/46 | 167/96/48 | 4.97 | 2.9 | 0.93 | Sin | 6mm | - | N | Conservative | |
| 33 | Anjalai | 55 | F | 76888 | + | - | - | - | - | - | - | - | N | 1.1 | 106/59/75 | 6.78 | 2.7 | 0.97 | Sin | 9mm | - | N | Lap Chole | Hypothyroid |

| | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 34 | Mallika | 65 | F | 71851 | + | - | - | - | - | - | - | - | N | 1.3/38/20/30 | 171/89/50 | 4.36 | 102 | 10.3 | Sin | 18mm | + | N | Lap Chole | |
| 35 | Savithri | 39 | F | 74476 | + | + | - | - | - | - | - | - | N | 1.8/30/48/32 | 131/64/64 | 8.96 | 3.60 | 1.11 | Mul | 10mm | - | N | Lap to open | Hypothyroid |
| 36 | Azhagammai | 75 | F | 77152 | + | + | - | - | - | - | DM | + | N | 1.0 | 142/73/56 | 1.86 | 2.4 | 1.18 | Mul | 8mm | + | N | Open Chole | |
| 37 | Kasturi | 54 | F | 82089 | + | - | - | - | - | - | - | - | N | 1.2 | 159/84/52 | 2.72 | 2.2 | 1.23 | Mul | 6mm | - | N | Lap Chole | |
| 38 | Kasturi | 45 | F | 79354 | + | - | - | - | - | - | - | - | N | 0.9 | 133/102/58 | 1.92 | 110 | 9.1 | Sin | 6mm | - | N | Lap Chole | |
| 39 | Subathra | 65 | F | 81065 | + | + | - | - | - | - | HTN | - | N | 1.6/40/28/59 | 240/146/42 | 4.87 | 2.7 | 0.86 | Mul | 10mm | - | N | Lap Chole | |
| 40 | Raja | 58 | M | 40702 | + | + | + | - | - | - | DM | - | N | 0.5/59/29/20 | 134/76/67 | 2.62 | 4.0 | 1.4 | Sin | 8mm | - | N | Lap Chole | |
| 41 | Dilli | 28 | F | 1052 | + | + | - | - | - | - | - | + | N | 1.2/45/36/38 | 222/130/53 | 1.98 | 1.13 | 0.7 | Mul | 8mm | - | N | Lap Chole | |
| 42 | Jayanthi | 28 | F | 8417 | + | + | + | - | - | - | - | + | N | 1.1/39/25/30 | 137/86/55 | 2.20 | 2.7 | 1.17 | Sin | 11mm | - | N | Lap Chole | |
| 43 | Sakunthala | 60 | F | 10504 | - | + | - | - | - | - | DM/BA | + | N | 0.8/0.5/77/19/15 | 122/75/65 | 3.24 | 2.01 | 1.4 | Sin | 8mm | - | N | Lap Chole | |
| 44 | Savithri | 35 | F | 15755 | + | + | - | - | - | - | - | + | N | 0.4/0.3/55/16/12 | 165/86/66 | 4.22 | 1.98 | 1.2 | Sin | 18mm | + | N | Lap Chole | |
| 45 | Kurshith | 39 | F | 13013 | + | + | + | - | - | - | DM | - | N | 0.6/140/20/14 | 158/81/59 | 3.1 | 2.8 | 1.1 | Sin | 6mm | - | N | Open Chole | |
| 46 | Revathy | 32 | F | 13056 | + | - | - | - | - | - | - | - | N | 1.0/0.3/41/26/23 | 144/97/63 | 2.34 | 3.12 | 0.78 | Mul | 12mm | - | N | Lap Chole | |
| 47 | Komala | 35 | F | 10379 | + | + | - | - | - | - | - | - | N | 1.1 | 132/75/76 | 3.21 | 3.2 | 0.98 | Sin | 7mm | - | N | Lap Chole | |
| 48 | Amutha | 45 | F | 9839 | + | + | + | - | - | - | - | - | N | 0.4 | 238/183/32 | 2.13 | 2.6 | 1.8 | Mul | 4mm | - | N | Lap Chole | |
| 49 | Saradha | 40 | F | 12553 | + | + | - | - | - | - | - | - | N | 0.7 | 101/77/53 | 6.2 | 120 | 10.1 | Sin | 12mm | - | N | Lap Chole | Hypothyroid |
| 50 | Shantha devi | 33 | F | 11429 | + | + | - | + | - | - | - | + | N | 1.2 | 225/109/54 | 6.1 | 95 | 5.2 | Mul | 4mm | - | N | Lap to open | Hypothyroid |
| 51 | Mallika | 50 | F | 14114 | + | + | + | + | + | - | HTN | + | N | 9.5/4.6/568/71/75 | 177/145/75 | 3.12 | 2.13 | 1.3 | Contracted GB with 12mm CBD with distal calculi | | | | ERCP | |
| 52 | Pramila | 32 | F | 11468 | - | + | - | - | - | - | - | - | N | 0.6/0.2/39/15/10 | 136/75/64 | 4.26 | 1.23 | 1.0 | Sin | 7mm | - | N | Lap Chole | |
| 53 | Jagadeeswari | 32 | F | 16103 | + | + | + | - | - | - | - | - | N | 0.5/0.2/64/13/27 | 156/76/67 | 1.11 | 1.92 | 3.2 | Sin | 4mm | - | N | Lap Chole | |
| 54 | Chithra | 35 | F | 16174 | + | + | - | - | - | - | - | - | N | 0.6/0.1/54/15/17 | 158/103/66 | 2.10 | 3.9 | 1.4 | Sin | 5mm | - | N | Lap Chole | |
| 55 | Manickam | 30 | M | 13912 | - | + | - | - | - | - | - | - | N | 1.0\0.3 | 164/99/52 | 3.12 | 3.2 | 0.9 | Sin | 12mm | - | N | Lap Chole | |
| 56 | Ganapathy | 28 | M | 15289 | + | + | - | - | - | - | - | - | N | 2.1/0.6/79/24/31 | 224/146/43 | 1.33 | 2.9 | 1.2 | Mul | 7mm | - | N | Lap Chole | |
| 57 | Shankar | 45 | M | 40489 | + | + | - | - | - | - | TB | + | N | 0.6/59/27/17 | 218/165/48 | 4.34 | 1.7 | 1.07 | Mul | 12mm | - | N | Lap Chole | |
| 58 | Saraswathy | 35 | F | 33518 | + | + | + | + | - | - | - | - | N | 0.7/26/36/46 | 102/75/63 | 1.53 | 2.72 | 1.37 | Sin | 6mm | - | N | Lap Chole | |
| 59 | Sasikala | 32 | F | 26245 | + | - | + | - | - | - | - | + | N | 1.2\0.4 | 112/56/46 | 1.22 | 2.0 | 1.38 | Mul | 8mm | - | N | Lap to open | |
| 60 | Srimathi | 36 | F | 26189 | + | + | - | - | - | - | - | - | N | 1.1\0.6 | 122/62/61 | 6.11 | 2.9 | 1.0 | Sin | 10mm | - | N | Open Chole | Hypothyroid |
| 61 | Elizabeth | 50 | F | 26614 | + | + | - | - | + | - | DM/SHT | + | N | 1.0/0.4/11/37/51 | 168/76/60 | 2.29 | 2.7 | 1.18 | Sin | 7mm | - | N | Lap Chole | |
| 62 | Umarani | 63 | F | 26800 | + | + | - | - | + | - | - | + | N | 1.0/0.4/34/50/342 | 138/88/60 | 0.71 | 2.9 | 1.06 | Distal CBD calcluli with dil CBD | | | | ERCP | |
| 63 | Jaya | 54 | F | 10257 | + | + | + | - | + | - | - | + | N | 3.4/1.7/267/97/479 | 143/84/55 | 8.12 | 3.6 | 1.18 | Distal CBD calcluli with dil CBD | | | | ERCP foll by open chole | Hypothyroid |
| 64 | Elsi | 40 | F | 28882 | + | - | - | - | - | - | - | - | SNT | 0.6/0.2/16/55/54 | 123/75/59 | 3.27 | 2 | 1.19 | Sin | 15mm | - | N | Lap Chole | |
| 65 | Shanthi | 46 | F | 28893 | + | - | - | - | - | - | - | - | N | 0.8/0.4/14/36/58 | 129/78/55 | 1.12 | 3.44 | 1.14 | Mul | 8mm | - | N | Lap Chole | |
| 66 | Mumtaz | 29 | F | 26792 | + | + | + | - | - | - | - | + | N | 1.6\0.6 | 159/80/54 | 4.42 | 2.6 | 1.4 | Mul | 6mm | + | N | Conservative | |
| 67 | Amutha | 45 | F | 27528 | + | + | - | - | - | - | DM | - | N | 0.5/13/50/33 | 175/100/49 | 3.8 | 2.22 | 1.87 | Sin | 12mm | - | N | Lap Chole | |
| 68 | Kuppu | 46 | F | 27101 | + | + | + | - | - | - | - | + | N | 0.8/0.2/20/44/73 | 136/86/54 | 2.56 | 2.89 | 1.1 | Sin | 12mm | - | N | Lap Chole | |
| 69 | Raji | 67 | M | 20728 | - | + | + | - | - | - | - | + | N | 0.9/0.2/18/15/62 | 142/100/50 | 11.22 | 1.1 | 0.8 | Mul | 14mm | - | N | Lap to open | Hypothyroid |
| 70 | Meenakshi | 57 | F | 30977 | + | + | - | - | - | + | Hypothy | - | Goitre | 0.9 | 137/55/67 | 22.1 | 0.5 | 0.67 | Mul | 10mm | + | N | Lap to open | Hypothyroid |
| 71 | Kamatchi | 67 | F | 30561 | + | + | + | - | - | - | - | + | N | 1.5/0.5/20/38/85 | 166/99/42 | 17.7 | 3 | 1 | Mul | 8mm | - | Dil with 13mm | Lap to open | Hypothyroid |
| 72 | Revathy | 33 | F | 31470 | + | + | - | - | - | - | - | - | N | 0.6/52/31/68 | 166/91/63 | 1.43 | 4.1 | 0.9 | Mul | 9mm | - | N | Lap Chole | |
| 73 | Aminammal | 47 | F | 27924 | - | + | - | - | - | - | DM | - | N | 0.6/0.2/32/30/61 | 123/88/67 | 2.8 | 3.96 | 1.56 | Sin | 10mm | - | N | Lap Chole | |
| 74 | Indira Gandhi | 45 | F | 18822 | + | + | + | - | + | - | - | + | N | 0.7/46/52/37 | 157/64/57 | 0.78 | 2.99 | 1.32 | Sin | 11mm | - | Dil | Open CBD | |

| | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 75 | Gayathribai | 60 | F | 27851 | + | + | + | + | - | - | - | + | N | 1.3/0.6/54/36/158 | 112/80/78 | 1.6 | 3.1 | 0.91 | CBD cal 11mm with dil CBD 16mm | | | | ERCP | |
| 76 | Krishnan | 70 | M | 25891 | - | + | - | - | - | - | - | + | N | 3.01/1.3/36/46/386 | 163/97/54 | 1.77 | 4 | 1.38 | Post chole status | | | | ERCP | |
| 77 | Krishnamoorthy | 52 | M | 30922 | + | + | - | - | - | - | DM | + | N | 0.8/34/28/62 | 130/88/69 | 1.3 | 2.4 | 1.8 | Sin | 15mm | - | N | Lap to open | |
| 78 | Praveena | 33 | F | 36957 | + | + | - | - | + | - | - | + | N | 5.9/2.7/132/189/235 | 158/81/56 | 9.71 | 2.6 | 0.75 | Sin | 12mm | - | Dil with 6mm | Open CBD exploration | Hypothyroid |
| 79 | Tamilselvi | 30 | F | 31495 | + | - | + | - | - | - | - | - | N | 0.9/0.2/27/20/48 | 106/56/73 | 3.82 | 2.6 | 0.97 | Mul | 5mm | - | N | Lap Chole | |
| 80 | Sivagami | 56 | F | 37150 | + | + | + | - | - | - | DM | - | N | 1.0/0.6/38/24/55 | 111/63/59 | 5.84 | 2.1 | 0.67 | Mul | 11mm | - | N | Lap Chole | Hypothyroid |
| 81 | Kala | 65 | F | 37227 | + | - | - | - | - | - | SHT | - | N | 0.9/0.3/32/28/36 | 157/89/46 | 2.33 | 2.1 | 0.98 | Sin | 10mm | - | N | Lap Chole | |
| 82 | Chandrammal | 77 | F | 36935 | + | + | + | - | - | - | DM | + | N | 0.6/46/70/81 | 132/76/56 | 0.99 | 2.56 | | Distal CBD calcluli with dil CBD | | | | ERCP | |
| 83 | Shankar | 65 | M | 34184 | + | + | - | - | - | - | DM | + | N | 0.6/0.3/24/32/75 | 110/85/69 | 2.82 | 3.88 | 0.86 | Mul | 10mm | - | N | Lap Chole | |
| 84 | Jayakar | 49 | M | 38439 | + | + | + | - | - | + | - | + | Goitre | 0.7/0.3/12/16/41 | 134/98/56 | 52.01 | 1.4 | 0.5 | Mul | 14mm | + | N | Lap to open | Hypothyroid |
| 85 | Pushpa | 40 | F | 42089 | + | + | - | - | - | - | RVD | + | N | 0.4/0.1/24/17/51 | 154/65/64 | 2.58 | 2.8 | 1.29 | Mul | 6mm | - | N | Open Chole | |
| 86 | Parvathy | 44 | F | 41974 | + | - | - | - | - | - | - | - | N | 1.3/0.1/84/52/75 | 167/74/64 | 2.44 | 2.6 | 1.18 | Sin | 10mm | - | N | Lap Chole | |
| 87 | Mahalakshmi | 26 | F | 41386 | + | + | - | - | - | - | - | + | N | 3.0/1.4/457/453/179 | 133/77/52 | 1.7 | 3.7 | 1.45 | Mul | 7mm | + | N | Interval Chole | |
| 88 | Mumtaz | 29 | F | 39872 | + | - | - | - | - | - | - | - | N | 0.8/0.3/28/22/21 | 175/98/42 | 1.87 | 3.5 | 1.2 | Mul | 5mm | - | N | Lap Chole | |
| 89 | Umavathy | 56 | F | 42927 | + | + | - | - | - | - | DM | + | N | 0.7/0.2/14/19/48 | 105/68/56 | 1.1 | 2.86 | 0.8 | Mul | 8mm | - | N | Lap Chole | |
| 90 | Sarala | 31 | F | 41431 | + | - | - | - | - | - | - | - | N | 0.8/0.3/17/11/23 | 113/66/76 | 0.98 | 3.2 | 1.11 | Mul | 8mm | - | N | Lap Chole | |
| 91 | Pushpa | 45 | F | 41968 | + | - | - | - | - | - | - | - | N | 0.8 | 129/60/77 | 1.52 | 2.87 | 1.37 | Sin | 8mm | - | N | Lap Chole | |
| 92 | Ushapriya | 24 | F | 39992 | + | + | + | - | - | + | - | + | Goitre | 1.0/0.4/184/43/76 | 150/66/48 | 24.12 | 1.34 | 0.55 | Mul | 5mm | - | Dil | Lap Chole | Hypothyroid |
| 93 | Saraswathy | 35 | F | 43107 | + | + | - | - | - | - | - | - | N | 0.4/0.3/22/20/63 | 125/73/55 | 2.73 | 4.12 | 0.97 | Mul | 4mm | - | N | Lap Chole | |
| 94 | Gayatri Devi | 42 | F | 40640 | + | - | - | - | - | - | - | - | N | 0.4/10/11/49 | 164/99/49 | 2.11 | 2.89 | 0.89 | Sin | 20mm | - | N | Lap Chole | |
| 95 | Shantha | 66 | F | 42742 | + | + | - | - | - | - | HTN | - | N | 0.6/0.5/21/20/63 | 155/66/56 | 2.54 | 2.2 | 1.52 | Mul | 5mm | - | N | Lap Chole | |
| 96 | Gangadevi | 35 | F | 43866 | + | + | + | - | - | - | - | + | N | 1.0/22/39/54 | 103/51/79 | 0.94 | 3.7 | 1.3 | Mul | 4mm | - | N | Lap Chole | |
| 97 | Kalavathi Devi | 50 | F | 41078 | + | + | - | - | - | - | HTN | + | N | 0.6/0.1/185/238/120 | 116/63/74 | 1.9 | 3.65 | 1.69 | Mul | 13mm | - | N | Lap to open | |
| 98 | Saraswathy | | F | | + | + | - | - | - | - | Hypothy | + | N | 1.0/0.6/24/30/54 | 116/55/69 | 0.67 | 1.6 | 0.9 | Sin | 12mm | - | N | Lap Chole | Hypothyroid |
| 99 | Vasuki | 51 | F | 45397 | + | - | - | - | - | - | - | - | N | 5.5/1.6/19/14/159 | 119/93/66 | 2.3 | 2.78 | 1.77 | Mul | 10mm | - | N | Lap Chole | |
| 100 | Shabeena | 32 | F | 49451 | + | + | - | - | - | - | Hypothy | + | N | 0.9/0.1/32/21/38 | 138/103/45 | 0.55 | 2.7 | 1.57 | Mul | 10mm | - | N | Lap to open | Hypothyroid |
| 101 | Jayakaran | 40 | M | 44853 | + | + | + | - | - | - | - | + | N | 1.4/0.5/30/40/58 | 108/64/73 | 2.62 | 2.2 | 0.78 | Mul | 8mm | - | N | Lap to open | |
| 102 | Jada Subramaniam | 72 | M | 43065 | + | + | + | + | + | - | DM | + | N | 16/11/87/52/263 | 145/76/69 | 3.1 | 2.1 | 1.1 | Mul | 10mm | CBD cal 10mm | | AMA | |
| 103 | Malliga | 51 | F | 50038 | + | + | - | - | - | - | DM | - | N | 1.1/0.5/39/56/100 | 137/82/59 | 1.67 | 2.8 | 1.4 | Mul | 4mm | - | N | Lap Chole | |
| 104 | Malini | 25 | F | 47396 | + | + | - | - | - | - | - | + | N | 1.1./0.3/26/24/40 | 164/75/64 | 2.44 | 2.61 | 0.84 | Sin | 12mm | - | N | Lap Chole | |
| 105 | Vasanthi | 30 | F | 50420 | + | + | + | - | - | - | - | + | N | 1.8/0.5/42/29/38 | 158/103/49 | 2.31 | 3.15 | 0.99 | Mul | 8mm | - | N | Lap Chole | |
| 106 | Sangeethavani | 38 | F | 47440 | + | + | + | + | - | - | - | + | N | 2.6/1.2/50/34/124 | 122/76/60 | 1.28 | 3.9 | 1.23 | Mul | 10mm | + | Dil | Interval Chole | |
| 107 | Banu | 45 | F | 50858 | + | + | + | - | - | - | - | + | N | 1.1/0.4/15/18/90 | 120/77/52 | 9.08 | 2 | 1.1 | Sin | 12mm | + | N | Interval Chole | Hypothyroid |
| 108 | Sumathy | 30 | F | 45998 | + | + | - | - | - | + | Hypothy | - | N | 1.2/0.5/30/32/56 | 165/123/42 | 0.3 | 1.9 | 1.4 | Sin | 8mm | - | N | Lap Chole | Hypothyroid |
| 109 | Feroza Begam | 25 | F | 51127 | + | - | - | - | - | - | - | - | N | 0.4/0.2/24/35 | 174/118/60 | 2.02 | 4 | 1.67 | Sin | 8mm | - | N | Lap Chole | |
| 110 | Amudha | 31 | F | 49811 | + | + | - | - | - | - | - | - | N | 1.0/0.5/35/20/58 | 134/94/58 | | 2.79 | 1.73 | Mul | 10mm | - | N | Lap Chole | |
| 111 | Ponpirai | 35 | F | 51090 | + | - | - | - | - | - | DM | - | N | 1.2/0.4/24/15/52 | 106/64/71 | 0.8 | 2.68 | 1.2 | Sin | 6mm | - | N | Lap to open | |
| 112 | Mary | 49 | F | 51314 | + | + | + | - | - | - | DM | + | N | 1.1/0.6/30/36/48 | 236/178/40 | 8.18 | 1.88 | 1 | Mul | 6mm | - | N | Conservative | Hypothyroid |
| 113 | Thangavelu | 55 | M | 54564 | + | + | - | + | - | - | - | + | N | 0.5/0.1/33/65/125 | 157/76/56 | 3.12 | 2.91 | 0.85 | Sin | 20mm | - | N | Lap to open | |
| 114 | Kalyani | 45 | F | 58840 | - | + | - | - | - | - | DM | - | N | 0.5/0.2/22/18/103 | 181/133/46 | 7.08 | 1.5 | 0.77 | Sin | 10mm | - | N | Lap Chole | Hypothyroid |
| 115 | Fazilath | 28 | F | 2313 | + | + | - | - | - | - | - | + | N | 0.6/0.2/76/20/82 | 121/77/60 | 1.56 | 3.2 | 0.83 | Sin | 22mm | - | N | Lap Chole | |
| 116 | Krishnakumari | 45 | F | 58432 | + | - | - | - | - | - | - | - | N | 0.6 | 136/75/61 | 2.6 | 0.85 | 2.61 | Mul | 8mm | - | N | Lap Chole | |

| | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 117 | Veerasamy | 57 | M | 1510 | + | + | - | - | - | - | - | - | N | 0.6/0.2/58/18/120 | 114/56/79 | 2.65 | 3.72 | 0.95 | Mul | 8mm | - | N | Lap Chole | |
| 118 | Dhayalan | 45 | M | 133320 | + | + | - | - | - | - | - | - | N | 3.0/1.6/61/72/168 | 130/64/67 | 2.9 | 3.6 | 1.68 | Sin | 10mm | - | N | Lap Chole | |
| 119 | Sabutha | 39 | F | 356 | + | + | - | - | - | - | - | - | N | 1.1/0.4/44/46/76 | 103/50/83 | 2.43 | 4.2 | 1.4 | Mul | 13mm | - | N | Lap Chole | |
| 120 | Rathinavathy | 68 | F | 951 | + | + | + | - | - | - | - | + | N | 0.8/0.3/42/35/86 | 234/176/45 | 0.76 | 3.91 | 1.81 | Sin | 14mm | - | N | Lap to open | |
| 121 | Malarvizhi | 45 | F | 51156 | + | - | - | - | - | + | - | - | Goitre | 0.6/0.2/32/30/61 | 108/64/76 | 12.01 | 2.5 | 0.92 | Sin | 7mm | - | N | Lap Chole | Hypothyroid |
| 122 | Muthulakshmi | 44 | F | 6139 | + | - | - | - | - | - | - | - | N | 1.4/0.4/34/46/66 | 212/192/36 | 1.21 | 2.56 | 1.38 | Mul | 4mm | - | N | Lap Chole | |
| 123 | Munusamy | 50 | M | 3851 | + | + | - | - | - | - | - | - | N | 0.8/0.3/46/30/80 | 107/46/79 | 1.56 | 4.03 | 1.7 | Mul | 8mm | - | N | Lap to open | |
| 124 | Noori | 30 | F | 452 | + | + | + | - | - | - | - | + | N | 0.8/0.5/77/19/15 | 106/56/77 | 32.1 | 0.31 | 0.1 | Sin | 4mm | - | N | Lap to open | Hypothyroid |
| 125 | Swetha | 25 | F | 8775 | + | + | - | - | - | - | - | - | N | 1.2/0.5/32/30/46 | 176/67/60 | 2.56 | 2.9 | 0.95 | Mul | 6mm | - | N | Lap Chole | |
| 126 | Samundeshwari | 58 | F | 3936 | - | + | - | - | - | - | - | - | N | 1.1/-/29/18/90 | 146/94/58 | 1.86 | 3.07 | 0.8 | Sin | 7mm | - | N | Lap Chole | |
| 127 | Manohari | 22 | F | 58508 | + | + | - | - | - | - | - | - | N | 0.8/0.2/40/28/53 | 175/118/64 | 1.7 | 3.6 | 1.4 | Sin | 12mm | - | N | Lap Chole | |
| 128 | Chithra | 45 | F | 10923 | + | + | - | - | - | - | - | - | N | 1.3/0.4/43/33/72 | 222/138/56 | 2.3 | 2.93 | 1.17 | Sin | 7mm | - | N | Lap Chole | |
| 129 | Baizanth | 29 | F | 9266 | - | - | - | - | - | - | - | - | N | 1.1 | 142/75/64 | 3.1 | 2.5 | 1.82 | Sin | 9mm | - | N | Lap Chole | |
| 130 | Vani | 37 | F | 9736 | + | + | + | - | - | - | - | + | N | 0.9/0.2/30/32/54 | 164/56/65 | 2.98 | 2.99 | 1.44 | Mul | 10mm | - | N | Lap Chole | |
| 131 | Amudha | 40 | F | 7124 | + | + | - | - | - | - | - | - | N | 1.1/0.5/34/45/30 | 163/99/56 | 7.98 | 1.8 | 0.92 | Mul | 11mm | - | N | Lap Chole | Hypothyroid |
| 132 | Savithri | 55 | F | 58557 | + | - | + | - | - | + | Hypothy | - | N | 0.9/0.4/32/30/30 | 129/75/66 | 4.14 | 2.8 | 0.98 | Mul | 12mm | - | N | Lap Chole | Hypothyroid |
| 133 | Shanthi | 78 | F | 9001 | + | + | - | - | - | - | - | - | N | 1.0/0.3/45/24/35 | 132/86/59 | 1.55 | 3.17 | 0.96 | Mul | 7mm | - | N | Lap Chole | |
| 134 | Vijaya | 45 | F | 1595 | + | + | + | - | - | - | DM | + | N | 0.9/40/42/30 | 144/76/51 | 1.73 | 2.9 | 0.89 | Mul | - | - | N | Lap Chole | |
| 135 | Aadhi Gownder | 45 | M | 9037 | + | + | - | - | - | - | - | - | N | 0.9/0.4/30/26/44 | 164/96/68 | 1.23 | 3.02 | 0.94 | Mul | 8mm | - | N | Lap Chole | |
| 136 | Punitha | 37 | F | 13179 | - | + | - | - | - | - | - | - | N | 1.3/0.3/40/46/68 | 175/134/50 | 0.91 | 3 | 1.11 | Sin | 7mm | - | N | Lap Chole | |
| 137 | Nandhini | 25 | F | 15081 | + | - | - | - | - | - | - | - | N | 1.1/-/30/25/60 | 164/97/46 | 0.7 | 4.02 | 1.5 | Sin | 5mm | - | N | Lap Chole | |
| 138 | Kanniga | 32 | F | 14108 | + | + | - | + | - | - | - | + | N | 1.0/0.5/28/35/50 | 175/79/64 | 1.77 | 3.67 | 1.38 | Sin | 8mm | - | N | Lap Chole | |
| 139 | Jothi | 38 | F | 14382 | + | - | - | - | - | - | - | - | N | 0.7/0.3/12/16/41 | 230/186/56 | 2.1 | 2.93 | 1.32 | Sin | 8mm | - | N | Lap Chole | |
| 140 | Venugopal | 62 | M | 16356 | + | + | - | - | - | - | DM/SHT | - | N | 1.8/0.5/42/29/38 | 147/82/70 | 2.34 | 3.7 | 1.57 | Mul | 10mm | - | N | Lap Chole | |
| 141 | Shanmugha Priya | 35 | F | 21446 | + | - | - | - | - | - | - | - | N | 0.4/0.3/22/20/63 | 210/98/55 | 2.42 | 2.74 | 1.7 | Sin | 8mm | - | N | Lap Chole | |
| 142 | Neelakandan | 40 | M | 18393 | + | + | - | - | - | - | HTN | - | N | 0.9/0.2/28/32/54 | 164/79/69 | 0.76 | 2.94 | 1.6 | Mul | 6mm | - | N | Lap Chole | |
| 143 | Malliga | 45 | F | 29900 | + | - | - | - | - | - | HTN | - | N | 1.3/0.4/30/36/65 | 125/86/59 | 2.33 | 3.08 | 0.9 | Mul | 5mm | - | N | Lap Chole | |
| 144 | Ramesh Babu | 40 | M | 22581 | + | + | - | - | - | - | DM | - | N | 1.5/0.6/20/38/55 | 187/76/58 | 0.91 | 2.8 | 1.38 | Mul | 8mm | - | N | Lap Chole | |
| 145 | Boopathy | 48 | M | 19762 | + | + | - | - | - | - | - | - | N | 1.1/0.3/24/26/46 | 149/86/64 | 1.79 | 3.7 | 1.72 | Sin | 7mm | - | N | Lap Chole | |
| 146 | Vishwanathan | 58 | M | 19735 | + | - | + | - | - | - | - | - | N | 1.6 | 125/53/78 | 1.36 | 4.2 | 1.44 | Sin | 18mm | - | N | Lap to open | |
| 147 | Kalavathy | 33 | F | 24158 | + | - | - | - | - | - | - | - | N | 0.7/-/20/24/32 | 165/76/59 | 0.89 | 4 | 0.87 | Sin | 5mm | - | N | Lap Chole | |
| 148 | Gunasekar | 24 | M | 29131 | - | + | - | - | - | - | - | - | N | 1.2/0.4/36/34/68 | 147/68/59 | 1.7 | 3.77 | 1.62 | Sin | 7mm | - | N | Lap Chole | |
| 149 | Kuttiyammal | 50 | F | 32167 | + | + | - | - | - | - | - | - | N | 1.2/0.5/42/30/54 | 114/91/76 | 1.48 | 2.69 | 1.66 | Sin | 8mm | - | N | Lap Chole | |
| 150 | Tamil Kudiarasan | 25 | M | 39700 | - | + | - | - | - | - | - | - | N | 1.1/0.4/20/28/38 | 135/64/55 | 8.22 | 1.8 | 0.96 | Sin | 8mm | - | N | Lap Chole | Hypothyroid |
| 151 | Vasuki | 51 | F | 45357 | + | + | - | - | - | - | - | - | N | 0.8/0.2/30/20/44 | 138/86/55 | 2.43 | 2.85 | 1.42 | Sin | 6mm | - | N | Lap Chole | |
| 152 | Shantha rubi | 32 | F | 43331 | + | + | - | - | - | + | - | - | Goitre | 1.8/1.0/46/52/70 | 135/58/49 | 26.1 | 0.68 | 0.44 | Mul | 15mm | - | N | Lap Chole | Hypothyroid |
| 153 | Kamatchi | 30 | F | 46251 | + | - | - | - | - | - | - | - | N | 1.2/0.3/40/42/54 | 156/79/61 | 2.11 | 3.89 | 1.7 | Sin | 6mm | - | N | Lap Chole | |
| 154 | Sridhar | 62 | M | 45990 | + | + | - | - | - | - | - | - | N | 1.1/-/32/30/26 | 168/100/48 | 2.33 | 2.99 | 1.53 | Sin | 8mm | - | N | Lap Chole | |
| 155 | Marimuthu | 45 | F | 48847 | + | + | - | - | - | - | DM | + | N | 1.4/0.6/32/42/44 | 146/66/51 | 12.32 | 1.4 | 0.7 | Mul | 6mm | - | N | Lap Chole | Hypothyroid |
| 156 | Neelaveni | 60 | F | 44350 | + | + | - | - | - | - | - | - | N | 2.1/0.9/50/34/82 | 169/109/67 | 0.79 | 3.71 | 0.94 | Mul | 12mm | - | N | Lap Chole | |
| 157 | Amudha | 39 | F | 48636 | + | + | - | - | - | - | - | - | N | 1.3/0.4/30/28/40 | 176/136/64 | 0.9 | 2.94 | 0.91 | Sin | 6mm | - | N | Lap Chole | |
| 158 | Arumugam | 35 | M | 49079 | + | + | + | - | - | - | - | + | N | 1.1/0.6/40/36/70 | 107/55/88 | 7.12 | 1.8 | 0.8 | Mul | - | - | N | Lap Chole | Hypothyroid |

| | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 159 | Malliga | 58 | F | 48175 | + | + | - | - | - | - | - | - | N | 1.8/0.6/36/50/86 | 144/111/59 | 1.9 | 4.07 | 1.1 | Mul | 14mm | - | N | Lap Chole | |
| 160 | Pampirai | 35 | F | 53425 | + | + | - | - | - | - | - | - | N | 1.8/1.0/60/54/98 | 103/53/70 | 9.11 | 1.9 | 1.2 | Sin | 14mm | - | N | Lap to open | Hypothyroid |
| 161 | Sachin | 34 | M | 53530 | + | + | - | - | - | - | - | + | N | 1.1/0.3/20/18/30 | 154/73/69 | 2.9 | 3.21 | 1.73 | Mul | 7mm | - | N | Lap Chole | |
| 162 | Umavathy | 53 | F | 53834 | + | + | + | - | - | - | - | - | N | 1.4/0.3/40/32/60 | 103/55/79 | 2.66 | 4.15 | 1 | Mul | 12mm | - | N | Lap to open | |
| 163 | Thamotharan | 58 | M | 54064 | + | + | + | - | - | - | - | - | N | 1.1/0.5/23/30/50 | 106/60/72 | 8.34 | 2.6 | 1 | Sin | 6mm | - | N | Lap to open | Hypothyroid |
| 164 | Sumathy | 30 | F | 54341 | + | - | - | - | - | - | - | - | N | 1.0/0.3/20/18/40 | 122/79/64 | 2.47 | 3.59 | 1.55 | Mul | 10mm | - | N | Lap Chole | |
| 165 | Thiyagarajan | 31 | M | 56659 | + | - | - | - | - | - | - | - | N | 0.9/-/20/24/38 | 174/94/43 | 0.79 | 2.8 | 0.88 | Mul | 8mm | - | N | Lap Chole | |
| 166 | Gunasekar | 57 | M | 57037 | - | + | + | - | - | - | - | + | N | 1.0/0.6/20/30/40 | 173/79/54 | 1.73 | 3 | 0.9 | Sin | 5mm | - | N | Lap Chole | |
| 167 | Ashif Hussain | 42 | M | 53140 | + | + | - | - | - | - | - | + | N | 1.5/0.5/46/40/90 | 130/82/63 | 1.32 | 3.56 | 1.63 | Sin | 11mm | - | N | Lap Chole | |
| 168 | Manjula | 40 | F | 57796 | + | + | - | - | - | - | - | + | N | 1.2/0.3/40/42/54 | 211/165/59 | 1.84 | 3.7 | 1.82 | Mul | 6mm | - | N | Lap Chole | |
| 169 | Mahalakshmi | 41 | F | 125228 | + | - | - | - | - | - | - | - | N | 1.4/0.6/34/20/40 | 134/91/58 | 0.78 | 2.9 | 1.77 | Sin | 7mm | - | N | Lap Chole | |
| 170 | Manikavasagam | 62 | M | 128880 | + | + | - | - | - | - | - | - | N | 1.2/0.3/40/34/46 | 138/66/70 | 2.9 | 2.9 | 1.8 | Mul | - | - | N | Lap Chole | |
| 171 | Girirajan | 63 | M | 130240 | + | + | + | - | - | - | - | + | N | 0.8/0.2/34/36/38 | 184/84/49 | 2.67 | 2.94 | 1.47 | Mul | 7mm | - | N | Lap to open | |
| 172 | Saraswathy | 47 | F | 127830 | + | + | - | - | - | - | - | - | N | 0.6/-/18/26/46 | 115/75/69 | 6.14 | 2.1 | 0.88 | Mul | 6mm | - | N | Lap Chole | Hypothyroid |
| 173 | Mohan | 43 | M | 129613 | + | + | + | - | - | - | - | - | N | 1.0/0.4/20/34/68 | 204/150/50 | 1.52 | 3.25 | 1.63 | Sin | 7mm | - | N | Lap Chole | |
| 174 | Jayalakshmi | 70 | F | 119716 | - | + | + | - | - | - | DM | - | N | 0.7/-/18/20/34 | 176/125/55 | 1.8 | 2.81 | 1.3 | Sin | 18mm | - | N | Lap Chole | |
| 175 | Devi | 52 | F | 121862 | + | + | - | - | - | - | - | + | N | 1.0/-/20/28/52 | 155/93/49 | 1.12 | 3.48 | 0.89 | Sin | 16mm | - | N | Lap Chole | |
| 176 | Muniammal | 40 | F | 118754 | + | - | - | - | - | - | - | - | N | 1.1/0.5/38/42/54 | 164/110/60 | 2.47 | 2.86 | 0.94 | Sin | 15mm | - | N | Lap Chole | |
| 177 | Velamani | 48 | M | 113384 | + | - | - | - | - | - | - | - | N | 1.2/0.6/32/30/64 | 156/103/46 | 6.1 | 3.2 | 1.3 | Mul | 9mm | - | N | Lap Chole | Hypothyroid |
| 178 | Vaideki | 25 | F | 117391 | + | + | + | - | - | - | - | + | N | 1.1/0.4/20/24/40 | 166/85/42 | 18.1 | 1.1 | 0.64 | Mul | 10mm | - | N | Lap Chole | Hypothyroid |
| 179 | Natarajan | 64 | M | 116702 | + | + | - | - | - | - | - | - | N | 1.0/0.4/18/24/30 | 192/138/50 | 0.94 | 3.18 | 1.3 | Sin | 9mm | - | N | Lap to open | |
| 180 | Dhanalakshmi | 80 | F | 100768 | + | + | - | - | - | - | - | - | N | 1.4/0.4/24/40/42 | 117/86/61 | 1.38 | 3.2 | 1.45 | Sin | 10mm | - | N | Lap Chole | |
| 181 | Thirunavukarasu | 67 | M | 99503 | + | + | - | - | - | - | HTN | - | N | 1.2/0.6/30/38/40 | 127/86/67 | 0.64 | 4.2 | 0.94 | Sin | 8mm | - | N | Lap Chole | |
| 182 | Suganthi | 31 | F | 97151 | + | + | - | - | - | - | - | - | N | 1.1/0.4/40/38/60 | 158/91/53 | 5.88 | 3.1 | 1.14 | Sin | 7mm | - | N | Lap Chole | Hypothyroid |
| 183 | Vegavalli | 55 | F | 96434 | + | + | - | - | - | - | - | - | N | 2.0/1.0/60/54/98 | 188/129/48 | 1.76 | 3.67 | 1.69 | Sin | 20mm | - | N | Lap Chole | |
| 184 | Narayanan | 65 | M | 101708 | + | - | - | - | - | - | DM | - | N | 0.7/-/18/24/28 | 128/76/62 | 2.8 | 4.09 | 1.28 | Mul | 9mm | - | N | Lap Chole | |
| 185 | Lalitha | 62 | F | 99055 | + | + | - | - | - | - | HTN | + | N | 1.0/0.4/20/26/42 | 166/65/66 | 0.81 | 4.04 | 1.5 | Sin | 14mm | - | N | Lap Chole | |
| 186 | Reehana | 42 | F | 103077 | + | + | + | - | - | - | - | + | N | 1.4/0.5/30/34/54 | 139/85/64 | 8.12 | 2.9 | 1.2 | Mul | 5mm | - | N | Lap Chole | Hypothyroid |
| 187 | Babu | 35 | M | 101372 | + | + | - | - | - | - | - | - | N | 1.0/0.5/45/24/35 | 196/150/54 | 1.39 | 3.62 | 1.47 | Mul | 8mm | - | N | Lap Chole | |
| 188 | Kokila | 45 | F | 103347 | + | + | - | - | - | - | - | - | N | 1.3/0.6/32/24/40 | 157/76/56 | 0.65 | 3.6 | 1.34 | Sin | 14mm | - | N | Lap Chole | |
| 189 | Siva | 29 | M | 103899 | + | + | - | - | - | - | - | - | N | 1.1/0.2/34/30/40 | 147/80/61 | 14.1 | 2.6 | 1 | Sin | 10mm | + | N | Lap to open | Hypothyroid |
| 190 | Rani | 29 | F | 110080 | - | - | - | - | - | - | - | - | N | 1.4/0.6/40/24/38 | 183/149/43 | 2.5 | 2.89 | 0.9 | Mul | 8mm | - | N | Lap Chole | |
| 191 | Ayisha | 34 | F | 110602 | + | + | - | - | - | - | - | + | N | 1.1/0.5/20/30/32 | 158/66/49 | 1.3 | 3.21 | 0.93 | Sin | 13mm | - | N | Lap Chole | |
| 192 | Rubavathy | 70 | F | 112550 | + | + | - | - | - | - | DM | - | N | 0.9/0.4/20/28/40 | 164/119/49 | 1.65 | 2.76 | 0.9 | Mul | 10mm | - | N | Lap Chole | |
| 193 | Jagan | 30 | M | 112010 | + | + | + | - | - | - | - | + | N | 0.9/-/40/43/36 | 149/59/55 | 12.02 | 1.6 | 0.92 | Mul | 6mm | - | N | Lap Chole | Hypothyroid |
| 194 | Mala | 40 | F | 114798 | - | + | - | - | - | - | - | - | N | 1.2/0.5/30/36/50 | 147/94/59 | 1.98 | 3.6 | 1.3 | Sin | 8mm | - | N | Lap Chole | |
| 195 | Kasthuriammal | 47 | F | 114373 | + | + | + | + | - | - | - | + | N | 1.7/0.9/39/42/60 | 186/134/47 | 13.2 | 1.4 | 0.62 | Mul | 7mm | - | N | Lap to open | Hypothyroid |

| S.No | Name | Age | Sex | Ip No | Diagnosis | TFT |
|---|---|---|---|---|---|---|
| 1 | Vanitha | 38 | F | 47209 | Right Varicose Veins | |
| 2 | Shanthi | 26 | F | 47210 | Left Fibroadenoma | |
| 3 | Gopi | 43 | M | 61127 | Bilateral Ing Hernia | |
| 4 | Geetha | 32 | F | 47419 | Subacute Appendicitis | |
| 5 | Vijayarani | 36 | F | 47466 | SNT Right Lobe | |
| 6 | Sekar | 49 | M | 61173 | Ca Stomach | |
| 7 | Selvam | 26 | M | 61101 | Anal Stenosis | |
| 8 | Valliyammal | 56 | F | 79649 | Carcinoma Cervix | |
| 9 | Neela | 50 | F | 79442 | Ventral Hernia | Hypothyroid |
| 10 | Meenatchi | 39 | F | 79444 | Incisional Hernia | |
| 11 | Shanthi | 50 | F | 9575 | Right Fibroadenoma | |
| 12 | Susila | 55 | F | 9763 | Carcinoma Rt Breast | |
| 13 | Rajathi | 44 | F | 9698 | Fissure in Ano | |
| 14 | Rani | 58 | F | 74139 | Left Varicose Veins | |
| 15 | Savithri | 62 | F | 74187 | Incisional Hernia | |
| 16 | Kaveri | 60 | F | 74199 | Ca Stomach | |
| 17 | Lakshmi | 46 | F | 74212 | Right Ca Breast | |
| 18 | Nivedha | 21 | F | 74241 | Subacute Appendicitis | |
| 19 | Krishnamoorthy | 45 | M | 42032 | Ca. Esophagus | |
| 20 | Sasikala | 34 | F | 74383 | Fissure in Ano | |
| 21 | Ravanaiah | 55 | M | 42056 | Right Ing Hernia | |
| 22 | Vasugi | 48 | F | 76686 | Rec. Incisional Hernia | |
| 23 | Hemalatha | 21 | F | 76703 | Right Fibroadenoma | |
| 24 | Sulochana | 40 | F | 76683 | Secondaries Neck | |
| 25 | Rajeshwari | 40 | F | 76655 | Lymph Cyst Neck | |
| 26 | Pushpa | 35 | F | 76796 | Fibroid Uterus | Hypothyroid |
| 27 | Kuppu | 54 | F | 76905 | Ca. Buccal Mucosa | |
| 28 | Anjalai | 55 | F | 76888 | Incisional Hernia | |
| 29 | Ranjitham | 48 | F | 76882 | Gastritis | Hypothyroid |
| 30 | Jodhy | 45 | F | 76933 | MNG | |
| 31 | Balu | 51 | M | 42140 | Left Hydrocele | |
| 32 | Amulu | 34 | F | 73681 | UV Prolapse | |
| 33 | Divya | 23 | F | 73688 | Cervical Lymphadenitis | |
| 34 | Priya Samraj | 31 | F | 73748 | Paraumblical Hernia | |
| 35 | Lakshmi | 45 | F | 73762 | Left Fibroadenoma | |
| 36 | Geetha | 38 | F | 73774 | SNT Left lobe | |
| 37 | Punithavathi | 25 | F | 73851 | Left Varicose Veins | |
| 38 | Palanivel | 35 | M | 42049 | Multiple Lipoma | |
| 39 | Alamelu | 28 | F | 73862 | Fistula in Ano | |
| 40 | Saranya | 20 | F | 73808 | Right Fibroadenoma | |
| 41 | Lakshmi | 45 | F | 73896 | Hemorrhoids | |
| 42 | Anushya | 35 | F | 71697 | Hemorrhoids | |
| 43 | Muthu | 42 | M | 41992 | Grade III Hemorrhoids | |
| 44 | Devagi | 60 | F | 71358 | Gastritis | |
| 45 | Revathi | 24 | F | 71229 | Femoral Hernia | |
| 46 | Thaiba | 40 | F | 68575 | Paraumblical Hernia | |
| 47 | Ruckmani | 48 | F | 71566 | Incisional Hernia | |
| 48 | Eswari | 50 | F | 69457 | Acute Pancreatitis | |
| 49 | Vijaya | 41 | F | 71853 | Fem Pop Occlusion Left | |
| 50 | Manju | 38 | F | 71977 | Fissure in Ano | Hypothyroid |
| 51 | Chokkalingam | 71 | M | 47247 | Adhesive Colic | |
| 52 | Nithyakalyani | 60 | F | 74032 | Right Ca Breast | |
| 53 | Bamavathy | 57 | F | 73910 | Ca Ascending Colon | |
| 54 | Bheer Mohammed | 38 | M | 47313 | Fistula in Ano | |
| 55 | Alamelu | 28 | F | 73862 | MNG | |
| 56 | Nagarathinam | 70 | F | 74274 | Ca Breast | |
| 57 | Suguna | 44 | F | 78998 | Obstructive Jaundice | |
| 58 | Kumar | 45 | M | 47314 | Paraumblical Hernia | |
| 59 | Shanmugham | 39 | M | 47478 | Right Varicose Veins | |
| 60 | Pavunamma | 60 | F | 78871 | Incisional Hernia | |
| 61 | Maheshwari | 50 | F | 79017 | Rt Tubo Ovarian Mass | |
| 62 | Thulasi | 51 | F | 78991 | Incisional Hernia | |
| 63 | Rossy | 37 | F | 79742 | Incisional Hernia | |
| 64 | Perumalsamy | 65 | M | 47486 | Bilateral Hydrocoele | |
| 65 | Nivetha | 21 | F | 79012 | Left Fibroadenoma | |
| 66 | Kamaraj | 40 | M | 47516 | Right Renal Calculi | |
| 67 | Kamatchi | 30 | F | 9586 | Fissure in Ano | Hypothyroid |
| 68 | Srinivasalu | 35 | M | 47484 | Gastritis | |
| 69 | Anjali | 55 | F | 76888 | Cystitis | |
| 70 | Prema | 57 | F | 78546 | Rec. Ca. Breast | |
| 71 | Uma | 20 | F | 78792 | Right Fibroadenoma | |
| 72 | Premavathy | 57 | F | 77254 | Fibroid Uterus | |
| 73 | Poonguzhali | 18 | F | 77856 | Subacute Appendicitis | |
| 74 | Bindhu | 33 | F | 76360 | Incisional Hernia | Hypothyroid |
| 75 | Adhilakshmi | 26 | F | 76340 | Paraumblical Hernia | Hypothyroid |
| 76 | Anitha | 52 | F | 76870 | Incisional Hernia | |
| 77 | Saroja | 47 | F | 79121 | Grade III Hemorrhoids | |
| 78 | Durga | 20 | F | 76655 | Left Ureteric Colic | |
| 79 | Sundaram | 50 | M | 64305 | Cervical Lymphadenitis | |
| 80 | Iruthaiyanathan | 56 | M | 64258 | Fissure in Ano | |
| 81 | Kumari | 40 | F | 79877 | Obstructive Jaundice | |
| 82 | Kasiammal | 60 | F | 79584 | Prolene Sinus | |
| 83 | Vasantha | 42 | F | 79608 | Subacute Appendicitis | |
| 84 | Munisundaram | 48 | M | 66991 | Bilateral Hydrocoele | |
| 85 | Amsa | 60 | F | 3118 | Incisional Hernia | |
| 86 | Sarasamma | 40 | F | 3282 | Paraumblical Hernia | |
| 87 | Radha | 19 | F | 3670 | Right Fibroadenoma | |
| 88 | Sureka | 27 | F | 3135 | Grade III Hemorrhoids | |
| 89 | Samundeshwari | 58 | F | 3136 | Ca cervix | |
| 90 | Annammal | 34 | F | 2766 | Fissure in Ano | |
| 91 | Rajeshwari | 30 | F | 1811 | Incisional Hernia | |
| 92 | Duraiarasan | 27 | M | 66855 | Branchial Sinus | |
| 93 | Iyyanar | 34 | M | 67059 | Gynaecomastia | |
| 94 | Leelambal | 36 | F | 4823 | Acute Appendicitis | |
| 95 | Noori | 35 | F | 4763 | Left Fibroadenoma | Hypothyroid |
| 96 | Fathima | 25 | F | 4878 | MNG | |
| 97 | Renuga | 39 | F | 2940 | MNG | Hypothyroid |
| 98 | Saradha | 60 | F | 2757 | Ca Stomach | |
| 99 | Stella Mary | 27 | F | 7576 | Hemorrhoids | |
| 100 | Parimala | 26 | F | 7626 | Incisional Hernia | |

**KEY :**

BA - - Bronchial Asthma
DM - - Diabetes Mellitus
SHT / HTN - - Systemic Hypertension
Hypothy - - Hypothyroidism
CBD - - Common Bile Duct
ERCP - - Endoscopic Retrograde Cholangiopancreaticography
Sin - - Single
Mul - - Multiple

Normal Range :

Total Bilirubin - 0.2 to 1.0
Direct Bilirubin - 0 to 0.5
SGOT - < 40
SGPT - < 42
ALP - 30 to 90
Sr. Cholesterol - < 200
Triglyceride - 40 - 160
HDL - M > 35, F >42
TSH - 0.30 to 5.5
T3 - 1.7 to 4.2
T4 - 0.7. to 1.80