
“ROLE OF PRE-OPERATIVE UPPER GASTRO-
INTESTINAL ENDOSCOPY IN SYMPTOMATIC
PATIENTS UNDERGOING ELECTIVE LAPAROSCOPIC
CHOLECYSTECTOMY FOR GALL BLADDER STONE
DISEASE:A ONE YEAR HOSPITAL BASED CROSS-
SECTIONAL STUDY”

BY

REG NO. BH0116003

Dissertation

Submitted to the

KLE Academy of Higher Education and Research, Belagavi,
Karnataka

In partial fulfillment

of the requirements for the degree of

MASTER OF SURGERY (M.S)

IN

GENERAL SURGERY

DEPARTMENT OF GENERAL SURGERY,
J. N. MEDICAL COLLEGE
BELAGAVI - 590010. KARNATAKA

APRIL - 2019

**KLE Academy of Higher Education and Research, Belagavi,
KARNATAKA**

**Endorsement by the HOD/ Principal/ Head
of the Institution**

This is to certify that the dissertation entitled “**ROLE OF PRE-OPERATIVE UPPER GASTRO-INTESTINAL ENDOSCOPY IN SYMPTOMATIC PATIENTS UNDERGOING ELECTIVE LAPAROSCOPIC CHOLECYSTECTOMY FOR GALL BLADDER STONE DISEASE: A ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY**” is a bonafide research work done by **REG NO. BH0116003.**

Dr. A.S.GOGATE M.S.
Professor and Head,
Department of General Surgery,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date:
Place: Belagavi

Dr. N.S. Mahantashetti MD
Principal,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date:
Place: Belagavi

LIST OF ABBREVIATIONS USED

PCS	:	Post cholecystectomy syndrome
RUQ	:	Right upper quadrant
UGI	:	Upper gastro intestinal
GER	:	Gastro esophageal reflux
CHA	:	Common hepatic artery
RHA	:	Right hepatic artery
CA	:	Cystic artery
CCK	:	Cholecystokinin
NaCl	:	Sodium chloride
MMC	:	Migratory motor complex
CT	:	Computed tomography
CBD	:	Common bile duct
GB	:	Gall bladder
UGE	:	Upper gastro intestinal endoscopy
MS	:	Microsofft.

ABSTRACT

INTRODUCTION:

Cholelithiasis is one of the **most common** problem encountered in surgery¹. The diagnosis of cholelithiasis has steadily increased because of the common access to ultrasound examination. The vast majority of cholecystectomies are performed for gallstone disease with cholesterol gallstones accounting for nearly 75% of all gallstones².

A proportion of patients do not get symptomatic relief after cholecystectomy for gallstone disease³. As their symptoms are not entirely due to cholelithiasis they unnecessarily undergo the risks associated with laparoscopic cholecystectomy. The ultrasound finding of gallstones may be **incidental** and peptic ulcer disease of the stomach or the duodenum, gastro-oesophageal reflux or hiatus hernia may be the true cause of pain. Symptomatology of upper GI diseases can be overlapping. Upper abdominal pain may be secondary to either cholelithiasis or gastro duodenal diseases. Differentiating between these two situations is important, because the prevalence of both conditions is common in the general population. Thus, this study tries to seek the importance of upper gastro intestinal endoscopy to reveal the association between gastrointestinal symptoms and diseases with gallstones.

OBJECTIVE:

To assess the value of preoperative upper GI endoscopy for qualifying patients with Gall bladder(GB) stone disease undergoing elective laparoscopic cholecystectomy, by determining presence of upper GI lesions.

MATERIALS AND METHODS:

This is a prospective study conducted on 66 cases of ultrasound proven gallstone disease, at the department of general surgery, **Dr. Prabhakar Kore Hospital and Medical Research Center** irrespective of age and sex. After examination all the patients were subjected to Upper GI endoscopy. The statistical analysis was conducted using SPSS version 22.

RESULTS:

Out of 66 study sample, **57.6 %** (n=38) were **female** and **42.4 %** (n=28) were **males** with the mean age of **45.8 years**. Symptomatology has shown **atypical biliary colic** accounting for **54.5%** (n=36) in comparison to **typical biliary colic** of **45.5%** (n=30). All subjects were subjected for upper GI endoscopy which detected a total number of **24** samples having **positive upper GI lesions** accounting for **36.4%** (n=24), which is statistically significant (p=0.045) and **63.6 %** (n=42) being **normal** on endoscopy.

Among the 66 study subjects 36.4% (n=24), were found to have positive UGI scopy findings, out of which 29.2% (n=7) had typical biliary colic and 70.8% (n=17) had atypical biliary symptoms.

In comparison with this 63.6% (n=42) of the study subjects had normal UGI scopy findings, out of which 54.8% (n=23) had typical biliary colic and 45.2% (n=19) had atypical biliary colic.

Inferring that the chances of yielding a positive UGI scopy is higher in patients with atypical biliary symptoms.

CONCLUSION:

The increased access to ultrasound for patients presenting with pain abdomen, has led to an increase in the diagnosis of cholelithiasis, as its symptomatology overlaps many upper GI lesions, the incidental finding of gall stones on ultrasound may mask the co-existing UGI lesions.

In this study UGI lesions were found in 36.4% of the patients. Hence treating either one of them only will lead to persistence of symptoms post treatment.

Thus we **recommend** an UGI-SCOPY pre-operatively for patients undergoing elective laparoscopic cholecystectomy for gallbladder stone disease for adequate diagnosis and treatment of the co-existing UGI lesions if present and reduce the incidence of post operative persistence of symptoms.

Key words: cholelithiasis, UGI scopy, biliary colic, laparoscopic cholecystectomy.

CONTENTS

S.NO	TOPIC	PAGE NO
1	INTRODUCTION	1
2	OBJECTIVES	4
3	REVIEW OF LITERATURE	5
4	MATERIALS AND METHODS	34
5	RESULTS	37
6	DISCUSSION	48
7	CONCLUSION	51
8	SUMMARY	52
9	BIBLIOGRAPHY	53
10	ANNEXURES	
	ANNEXURES I: CONSENT FORM	61
	ANNEXURES II: PROFORMA	65
	ANNEXURES III: PHOTOGRAPHS	67
	ANNEXURES IV: KEY TO MASTER CHART	70
	ANNEXURES V: MASTER CHART	71

LIST OF TABLES

S.NO	DESCRIPTION	PAGE NO
1	Distribution of study subjects according to age.	37
2	Distribution of study subjects according to sex.	38
3	Distribution of study subjects according to symptoms.	39
4	Distribution of study subjects according to UGI scopy findings.	40
5	Sex wise distribution of patients with typical and atypical biliary symptoms.	41
6	Age wise distribution of type of biliary symptoms.	42
7	Age wise distribution of UGI scopy findings.	43
8	Distribution of UGI scopy findings.	44
9	Relationship between type of biliary symptoms and UGI scopy findings.	46
10	Distribution of positive UGI scopy with type of biliary symptoms.	47

LIST OF GRAPHS

S.NO	DESCRIPTION	PAGE NO
1	Bar diagram showing age distribution of study subjects.	37
2	Pie chart showing sex distribution of study subjects.	38
3	Bar diagram showing the distribution of biliary colic symptoms.	39
4	Pie chart showing distribution of findings of Upper GI endoscopy.	40
5	Bar diagram shows the distribution of sex with symptoms of biliary colic.	41
6	Bar diagram shows the relationship between age and biliary colic.	42
7	Bar diagram shows the relationship between age and Upper GI Endoscopy Findings	43
8	Findings of upper GI endoscopy	45
9	Pie chart showing the positive UGI endoscopy findings in patient with Typical and Atypical biliary colic.	47

LIST OF FIGURES

S.NO	DESCRIPTION	PAGE NO
1	Gross anatomy of the gall bladder.	5
2	Deformations of gallbladder. "Phrygian cap" and Hartmann's pouch of infundibulum.	6
3	Overall arrangement of intrahepatic and extrahepatic biliary tract.	8
4	Sphincter of oddi.	8
5	Variation in cystic duct anatomy.	9
6	Arterial blood supply of extrahepatic biliary tree.	11
7	Variations of cystic artery.	11-12
8	Lymphatic drainage of gallbladder.	14
9	Hepatocystic triangle.	15
10	The triangle of solubility.	24
11	UGI- scopy finding	67

INTRODUCTION

Cholelithiasis is one of the most common problems encountered in surgery. Gallstones constitute a significant health problem in developed societies, affecting 10% to 15% of the adult population⁴. In Asia, the prevalence of gallstone disease is 5-10% of population especially among older individuals and females. The number of surgical procedures for cholelithiasis has risen markedly in developed countries since 1950. The introduction of laparoscopic cholecystectomy in 1989 further increased the cholecystectomy rate⁴. Although there is undoubtedly an element of overuse, cholecystectomy is now the most common elective abdominal surgery performed.

The diagnosis of cholelithiasis has steadily increased because of the common access to ultrasound examination. The vast majority of cholecystectomies are performed for gallstone disease with cholesterol gallstones accounting for nearly 75% of all gallstones⁵.

Gallstones are common. The majority will not develop symptoms: up to 80% will never experience biliary pain or complications such as acute cholecystitis, cholangitis, or pancreatitis⁶. Hence, most gallstones are clinically "silent," an incidental finding often uncovered during abdominal ultrasound being performed for another reason. Since most gallstones are asymptomatic, it is essential to define exactly which symptoms are caused by gallstones: true biliary pain and/or complications, versus nonspecific abdominal complaints including dyspepsia. The importance for clarifying what constitutes true biliary pain is to better predict relief following cholecystectomy.

Cholecystectomy proved successful in treating the patients of biliary colic and cholecystitis in 80-95% of patients with stones. When stones were not present, the persistence of symptoms was as high as 40%.⁷ The persistence of abdominal symptoms even after cholecystectomy is highly discouraging for surgeons. Coexistence of concurrent upper gastrointestinal problems in gallstones disease patients may have attributed to the post cholecystectomy syndrome.^{8,9}

Post-cholecystectomy syndrome (PCS) consists of a group of upper gastrointestinal symptoms that recur and/or persist after cholecystectomy. It is defined as early if occurring in the post-operative period and late if it manifests after months or years. Although this term is used widely, it is not completely accurate, as it includes a large number of disorders, both biliary and extra-biliary in origin, that may be unrelated to cholecystectomy.^{10,11.}

To identify the cause of right upper quadrant (RUQ) pain, oesophagogastroduodenoscopy. (Upper GI endoscopy) is important to identify the diseases of upper gastrointestinal tract. As it evaluates the mucosa for signs of disease from the esophagus through the duodenum & allows direct visualization of the ampulla of Vater.¹²

These upper gastrointestinal symptoms have been related to gallstones but causal relationship has not been established yet. Evaluation of gall stone disease is an immense challenge as to ascertain whether gallstones are responsible for symptoms or just an incidental finding and peptic ulcer disease of the stomach or the duodenum, gastro-oesophageal reflux or hiatus hernia may be the true cause of pain.

Thus, this study tries to seek the importance of upper gastro intestinal endoscopy to reveal the association between gastrointestinal symptoms and diseases

with gallstones to establish a plan of management which is one of the most common problems faced by the current surgical practice, for the complete cure of all patients.

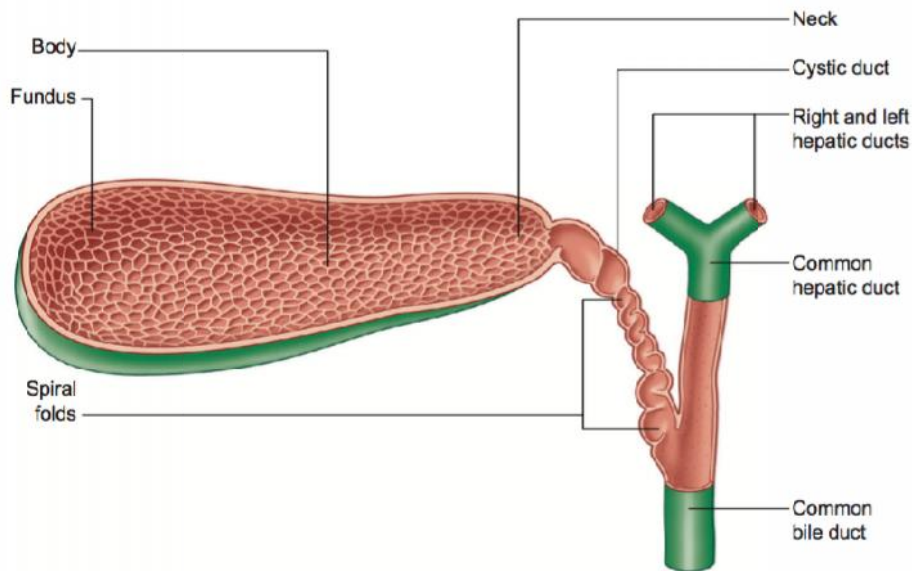
OBJECTIVE

To assess the value of preoperative upper GI endoscopy for qualifying patients with Gall bladder (GB) stone disease undergoing elective laparoscopic cholecystectomy, by determining presence of upper GI lesions.

REVIEW OF LITERATURE

Anatomy and physiology of the gall bladder:

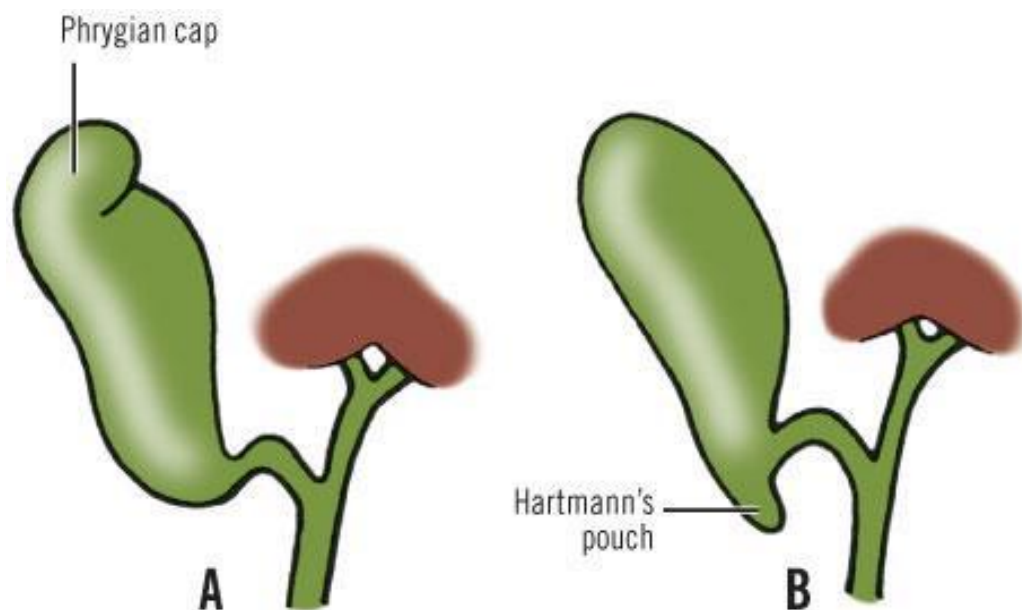
GALLBLADDER AND BILIARY TREE



The gallbladder is a flask-shaped, blind-ending diverticulum attached to the bile duct by the cystic duct. The gallbladder, is divided into the neck, infundibulum with Hartmann's pouch, body, and fundus of the gallbladder. Roughly the size and shape of a common light bulb, the gallbladder holds 25 to 50 mL of bile as an extrahepatic reservoir.¹³ It is grey-blue in colour and is usually firmly attached by connective tissue to the inferior surface of the right lobe of the liver, between segments IV and V at the lower limit of the principal plane. Although some gallbladders are almost enveloped by liver parenchyma, others hang on a mesentery, predisposing to volvulus.^{14,15} Where the gallbladder attaches to the liver, Glisson's capsule does not form, and this common surface provides the venous and lymphatic drainage of the gallbladder.

The neck lies at the medial end, close to the portahepatis , and almost always has a short peritoneal attachment to the liver, which usually contains the cystic artery. At its lateral end, the neck widens out to form the body of the gallbladder; when this widening is clearly demarcated as a result of gallstone disease, it is referred to as '**Hartmann's pouch**'. The neck usually lies anterior to the second part of the duodenum. The body of the gallbladder normally lies in contact with the visceral surface of the liver. 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 on 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.¹⁶



Copyright ©2006 by The McGraw-Hill Companies, Inc.
All rights reserved.

Following removal of the gallbladder, there is sometimes leakage of bile from small bile ducts in the gallbladder bed. The small bile ducts may cause postoperative bile leakage if they are injured.

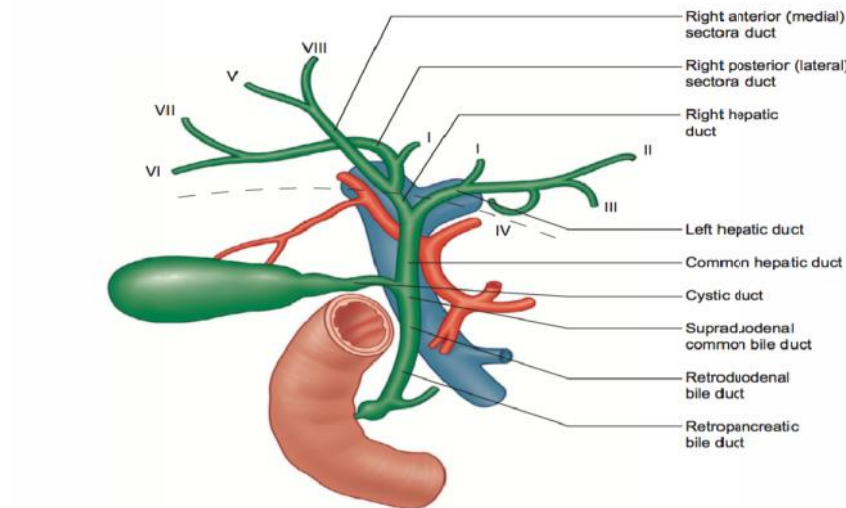
THE EXTRA HEPATIC BILIARY TREE:

The extrahepatic biliary tract consists of **the confluence of the left and right hepatic ducts, the common hepatic duct, gallbladder and the cystic duct and the common bile duct .**

The right hepatic duct is formed by the union right posterior (segments IV and VII) and right anterior (segments V and VIII) hepatic ducts at the porta hepatitis and has a shorter extrahepatic course of around 0.9cm .¹⁷ The left hepatic duct is formed by the ducts draining the segments II, III, and IV of the liver, courses horizontally along the base of segment IV, and has an extrahepatic length of about 1.7 - 2 cm.

The hepatic ducts confluence is usually extrahepatic and anterior to the portal vein bifurcation.

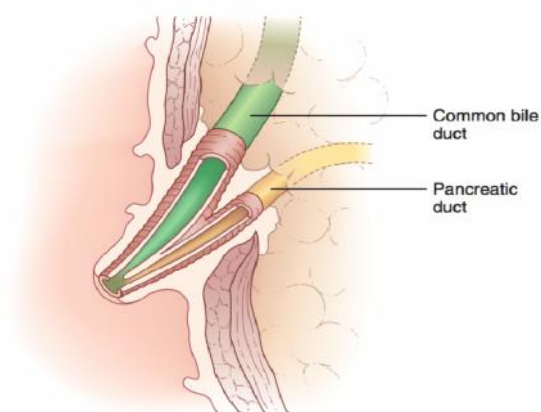
The common hepatic duct drains inferiorly. Below the takeoff the cystic duct, it is referred to as the common bile duct. The common bile duct usually measures 10 to 15 cm in length and is typically 6 mm in diameter. The common hepatic duct runs along the right side of the hepatoduodenal ligament to the right of the hepatic artery and anterior to the portal vein. The common bile duct continues inferiorly behind the first portion of the duodenum and into the head of the pancreas in an inferior and slightly rightward direction.¹⁸ The intrapancreatic distal common bile duct then joins with the main pancreatic duct (of Wirsung), with or without a common channel, and enters the second portion of the duodenum through the major papilla of Vater



Overall Arrangement of the Intrahepatic and Extrahepatic Biliary Tree.

At the choledochoduodenal junction, a complex muscular complex known as the *sphincter of Oddi* regulates bile flow and prevents reflux of duodenal contents into the biliary tree. There are three major parts to this sphincter:

- (1) the sphincter choledochus, which is a circular muscle that regulates bile flow and the filling of the gallbladder,
- (2) the pancreatic sphincter, present to variable degrees, which surrounds the intraduodenal pancreatic duct.

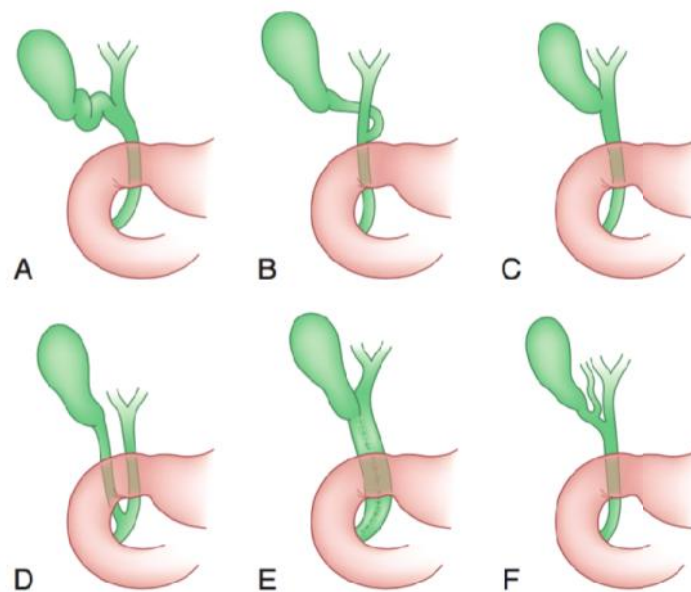


Sphincter of Oddi

- (3) The sphincter ampullae, made up of longitudinal muscle, which prevents duodenal reflux.

CYSTIC DUCT:

The cystic duct drains the gallbladder into the common bile duct. In adults, it is usually between 2 and 4 cm long and has a luminal diameter of 2–3mm .It passes posteriorly and medially from the neck of the gallbladder, often in a tortuous fashion, to unite with the common hepatic duct and form the common bile duct. The anatomy of the junction between the cystic duct and common hepatic duct is variable.



VARIABILITY IN CYSTIC DUCT ANATOMY.

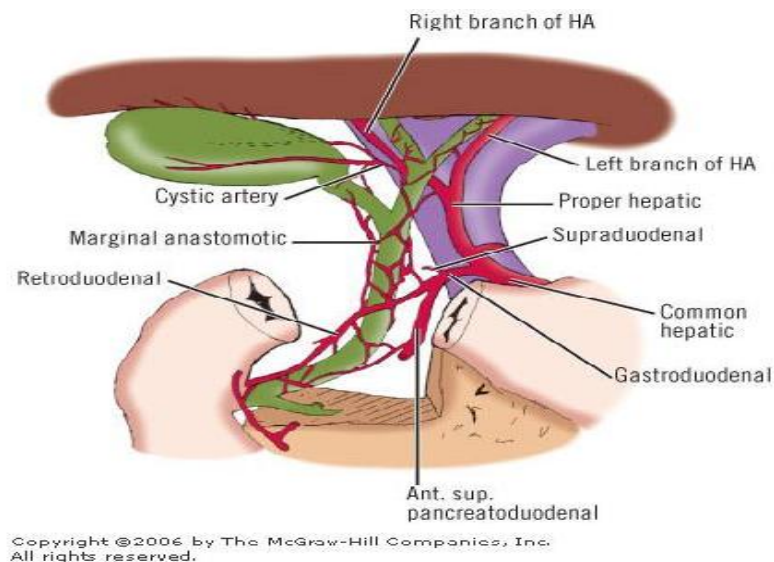
The mucosa of the cystic duct has 2–10 crescentic folds that project into the lumen and form a spiral; these are continuous with those in the neck of the gallbladder. The function of these spiral folds is unknown but they may help to preserve the patency of this narrow, tortuous duct rather than regulate the flow of bile, as is commonly stated.

VASCULAR ANATOMY:

The segmental anatomy of the liver parenchyma is based on the vascular supply and drainage, and the biliary drainage is described by the corresponding vascular segment. As opposed to the liver, where most perfusion comes from portal venous flow, the entire biliary tree is supplied solely by the arterial anatomy.

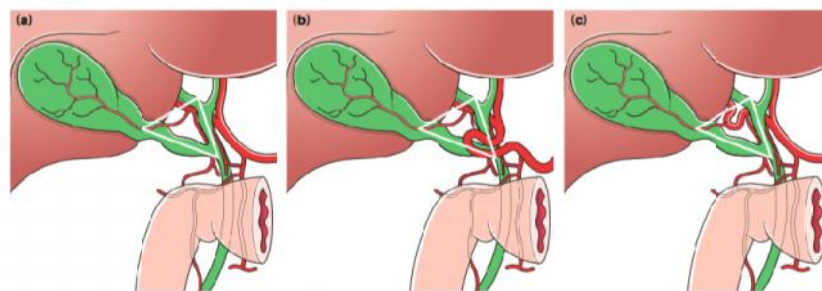
The inferior bile duct, below the level of the duodenal bulb, receives its perfusion from tributaries of the posterior superior pancreaticoduodenal and gastro duodenal arteries.

The small branches coalesce to form the two vessels that run along the common bile duct at the 3 and 9 o'clock positions. With close dissection of the areolar tissue surrounding the bile duct, these vessels can be damaged, leaving the bile duct at risk for ischemic injury. The superior common bile duct, from the duodenal bulb to the cystic duct, and common hepatic ducts receive their blood supply from the right hepatic and cystic arteries.¹⁸ As the proper hepatic artery ascends on the anterior medial side of the porta, it divides into right and left hepatic arteries. In most cases, the right hepatic artery passes posterior to the common hepatic duct to supply the right lobe of the liver. After crossing the duct, the right hepatic artery passes through the triangle of Calot, bordered by the cystic duct, common hepatic duct, and edge of liver. In this triangle, the right hepatic artery gives off the cystic artery to the gallbladder and is at risk for injury during a cholecystectomy.

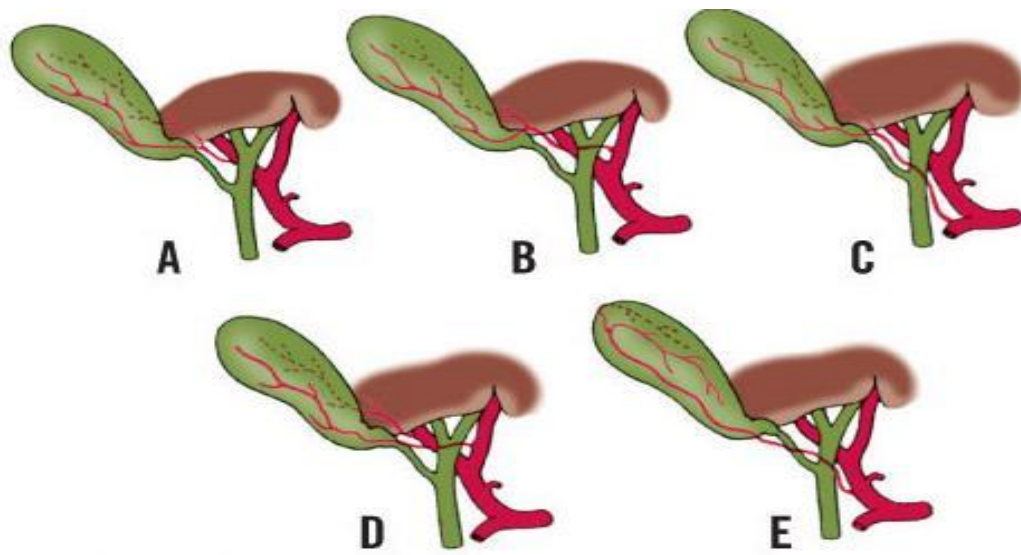


Arterial blood supply of extrahepatic biliary tract.

Normally, the cystic artery arises from the right hepatic artery, which can pass posterior or anterior to the common bile duct to supply the gallbladder. Similar to the variability of the cystic duct, the cystic artery may arise from the right hepatic, left hepatic, proper hepatic, common hepatic, gastro duodenal, or superior mesenteric artery. Although variable, the cystic artery generally lies superior to the cystic duct and is usually associated with a lymph node, known as Calot’s node.¹⁹ Because this node provides some of the lymphatic drainage of the gallbladder, it can be enlarged in the setting of gallbladder pathology, whether inflammatory or neoplastic.



A) Normal Calot’s B) Tortuous CHA C) Tortuous RHA With Short CA



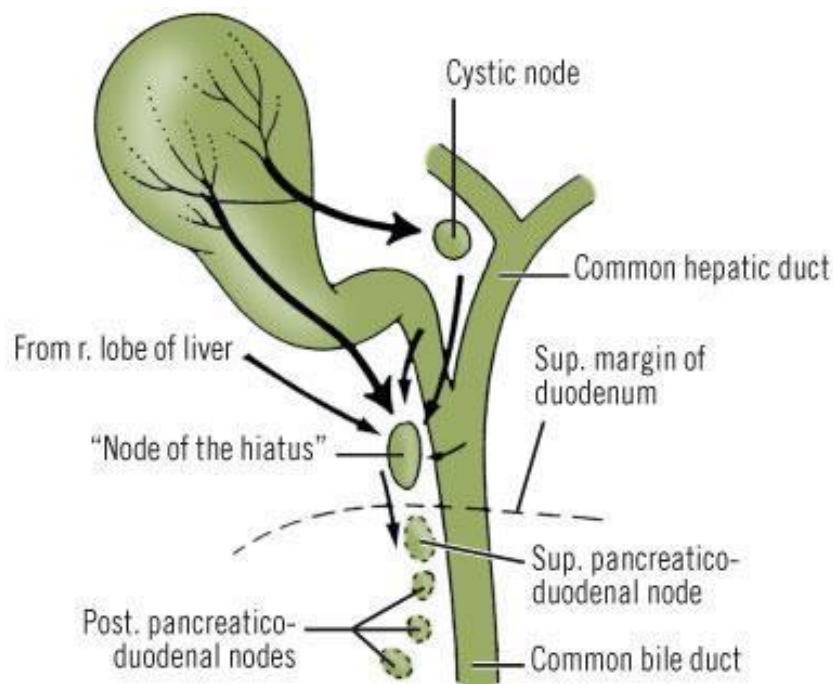
Variations of origin and course of cystic artery: A, Cystic artery arises from right hepatic artery (74.7%). B, Cystic artery arises from left hepatic artery and passes anterior to common hepatic duct (20.5%). C, Cystic artery arises from gastroduodenal artery (2.5%). D-E, Recurrent cystic arteries reach fundus of gallbladder and descend toward neck (rare). In the remainder (approximately 2.3%, not shown), cystic artery arises from a variety of other arteries.

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 drain into segmental portal veins within the liver. The remainder of the organ drains by one or two small cystic veins into either portal vein branches within the liver or portal venous tributaries draining the hepatic ducts and upper bile duct. Only rarely does a single or double cystic vein drain directly into the right branch of the portal vein.²⁰

Lymphatics of the Biliary Tract:

Long collecting trunks drain the lymphatic plexus of the fundus and body of the gallbladder. The trunks are on the right and left borders (lateral and medial borders of the gallbladder wall) and are connected by an oblique trunk to form a large "N" on the surface. The trunks on the left drain into the cystic node, which lies in the angle formed by the cystic and common hepatic ducts. The trunks on the right follow the cystic duct, passing without entering the cystic node. These vessels and the efferent vessels of the cystic node drain to the node of the anterior border of the epiploic foramen, called the "hiatal node" and to the superior pancreatic duodenal nodes on the common bile duct.²¹ For all practical purposes, lymphatics of the gallbladder and hepatic ducts have the same lymphatic drainage. Enlarged metastatic lymph nodes may cause jaundice by obstructing the common bile duct.^{22,23.}



Copyright ©2006 by The McGraw-Hill Companies, Inc.
All rights reserved.

Lymphatic drainage of the gallbladder.

INNERVATION:

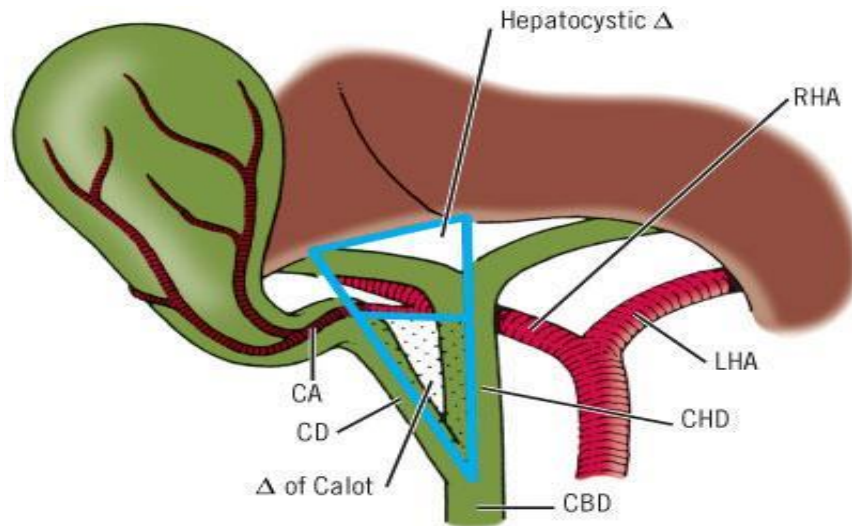
The gallbladder and the extrahepatic biliary tree are innervated by branches from the hepatic plexus. Gallbladder contraction occurs in response to cholecystokinin (CCK) and parasympathetic (vagal) stimulation. Postganglionic sympathetic nerve fibres from the coeliac and superior mesenteric ganglia are inhibitory to gallbladder smooth muscle.²⁴ Sympathetic afferents from the gallbladder convey pain sensation; they travel with the greater and lesser splanchnic nerves and have their cell bodies in the T7–9 spinal cord segments. Visceral pain from the gallbladder is referred to the right hypochondrium and epigastrium and may radiate around the back below the right scapula. Inflammation of the parietal peritoneum overlying the gallbladder produces localized right upper quadrant pain. A diverse range of neurotransmitters have been identified within intrinsic neurones of the gallbladder.

The common bile duct and smooth muscle of the hepatopancreatic ampulla are also innervated by the vagi, either directly or via the hepatic plexus, and by sympathetic nerves.

CALOT'S TRIANGLE:

The triangular region formed between the cystic duct, the common hepatic duct and the inferior surface of the liver is the hepatobiliary triangle. It is often mistakenly referred to as Calot's triangle, which is an isosceles triangle based on the common hepatic duct, with the cystic artery and cystic duct forming its sides.²⁵ The hepatobiliary triangle is bridged by the double layer of peritoneum that forms the short and variable mesentery of the cystic duct. Between these two layers there is a variable amount of fatty connective tissue, lymphatics, the cystic lymph node, autonomic nerves, and usually the cystic artery as it runs from the right hepatic artery

to the gallbladder, occasionally, there may also be an accessory bile duct. Understanding the variations in biliary and arterial anatomy as they relate to the triangle is of considerable importance during excision of the gallbladder in order to avoid injury to the common hepatic or common bile duct or right hepatic artery.²⁶



Triangle of Calot

PHYSIOLOGY:

Bile secretion is from the hepatocytes, Bile is secreted into bile canaliculi, which encircle each hepatocyte. Within the hepatic lobule, these canaliculi coalesce to form small bile ducts, eventually entering a portal triad. Four to six portal triads combine to create a hepatic lobule, the smallest **functional unit** of the liver, identified by its central terminal hepatic venule. On the opposite aspect from the canalicular surface of the hepatocyte lies the sinusoidal surface, which contacts the space of Disse. In this contact area, the hepatocyte is responsible for the absorption of circulating components of bile, an important step in the enterohepatic circulation of bile. Once absorbed and secreted into the bile canaliculi, the tight junctions in the biliary tree keep these components within the bile secretory pathway. The secretion of bile components into the biliary tree form a major stimulus to bile flow, and the

volume of bile flow is an osmotic process. Because bile salts combine to form spherical pockets, known as micelles, the salts themselves provide no osmotic activity. Instead, the cations that are secreted into the biliary tree along with the bile salt anion provide the osmotic load to draw water into the duct and increase flow to keep bile electrochemically neutral. For this reason, bile maintains an osmolality approximately comparable to that of plasma.

Bile, as it leaves the liver, is composed of 97% water, 1–2% bile salts and 1% bile pigments, cholesterol and fatty acids. The liver excretes bile at a rate estimated to be approximately 40ml/h. The rate of bile secretion is controlled by cholecystokinin (CCK), which is released from the duodenal mucosa.²⁷ With feeding, there is increased production of bile. The primary bile salts, cholate and chenodeoxycholate, are synthesized in the liver by cholesterol. They are conjugated there with taurine and glycine, and act within the bile as anions (bile acids) that are balanced by sodium. Bile salts are excreted into the bile by the hepatocyte and aid in the digestion and absorption of fats in the intestines. About 95% of the bile acid pool is reabsorbed and returned through the portal venous system to the liver, also known as the enterohepatic circulation. The remaining 5% is excreted in the stool.^{28,29} Cholesterol and phospholipids synthesized in the liver are the principal lipids found in bile. The synthesis of phospholipid and cholesterol by the liver is regulated in part by bile acids. The color of bile is due to the presence of the pigment bilirubin diglucuronide, which is the metabolic product from the breakdown of haemoglobin, and is present in bile in concentrations 100 times greater than plasma. Once in the intestine, bacteria convert it into urobilinogen, a small fraction of which is absorbed and secreted into the bile.

GALLBLADDER:

In the fasting state, secreted bile will pass through the biliary tree into the intestine directly and be reabsorbed. Additionally, bile will collect in the gallbladder, which serves as a storage site of secreted bile. To store bile secretions, the gallbladder is extremely efficient in water absorption and thus concentration of bile components. This absorption is an osmotic process performed via the active NaCl transport. With the absorption of NaCl and water across the gallbladder epithelium, the chemical composition of bile changes in the gallbladder lumen. Increases in cholesterol concentration, in addition to calcium, which is not as efficiently absorbed, then lead to decreased stability of phospholipid cholesterol vesicles. The reduced vesicle stability predisposes to nucleation of this stagnant pool of cholesterol and thus to cholesterol stone formation.^{30,31} The gallbladder neck and cystic duct also secrete glycoproteins to help protect the gallbladder from the detergent activity of bile. These glycoproteins also promote cholesterol crystallization.

The gallbladder fills through a retrograde mechanism. With an increase in the tonic activity of the sphincter of Oddi in the fasting state, pressure increases in the common bile duct. This increased pressure allows filling of the lower intraluminal pressure gallbladder, which is capable of storing up to 600 mL of the daily production of bile. The passage of fat, protein, and acid into the duodenum induces CCK secretion from duodenal epithelial cells. Cholecystokinin, as its name suggests, then causes gallbladder contraction, with intraluminal pressures up to 300 mm Hg. Vagal activity also induces gallbladder emptying, but is a less powerful stimulus to gallbladder contraction than CCK.

Sphincter of Oddi :

The sphincter of Oddi is a complex structure that is functionally independent from the duodenal musculature. It creates a high-pressure zone between the bile duct and the duodenum. The sphincter regulates the flow of bile and pancreatic juice into the duodenum, prevents the regurgitation of duodenal contents into the biliary tract, and also diverts bile into the gallbladder. The sphincter of Oddi also has very high-pressure phasic contractions, which play a role in preventing the regurgitation of duodenal contents into the biliary tract. Both neural and hormonal factors influence the sphincter of Oddi. In response to CCK, both sphincter of Oddi pressure and phasic wave activity diminish. After a meal, sphincter pressure relaxes in coordination with gallbladder contraction, thereby allowing the passive flow of bile into the duodenum. During fasting, high-pressure phasic contractions of the sphincter of Oddi persist through all phases of the MMC.^{32,33} Sphincter of Oddi activity appears to be coordinated with the partial gallbladder emptying and increases in the bile flow that occur during phase III of the MMC . This activity may be a preventive mechanism against the accumulation of biliary crystals during fasting.

OPERATIVE PROCEDURE:

LAPAROSCOPIC CHOLECYSTECTOMY:

The anaesthetized patient is placed supine on the operating table. The pneumoperitoneum may be achieved with an open (Hasson) or a closed (Verres needle) method, via an infra-umbilical longitudinal or transverse incision. The central part of the umbilicus is elevated with a towel clip, and a 10-mm longitudinal infra-umbilical skin incision is made. The umbilical ligament is then identified, grasped, and a 0.5cm incision made along its length. The abdominal cavity is then entered using artery forceps and the first port introduced over a graduated bougie. In patients

who have had multiple previous operations, the peritoneal cavity may be entered under direct vision by a cut-down in an area not associated with a previous incision. Once the first port has been placed, the peritoneal cavity is carefully insufflated with warmed CO₂ to a pressure of 12mmHg. A 30-degree laparoscope is introduced via the umbilical port and the peritoneal cavity is inspected. The second 10-mm port is inserted under direct vision in the midline in the epigastrium, passing just to the right of the falciform ligament, towards the gallbladder. Two 5- mm ports are introduced, one in the right mid-clavicular and one in the right mid-axillary line, angled towards the gallbladder.

Placing the patient in a steep reverse Trendelenberg position with a left down tilt and aspirating a distended stomach, aids in vision and access. Any adhesions between the gallbladder and omentum or duodenum are divided, and the gallbladder fundus grasped and retracted towards the patient's right shoulder. A 5-mm grasper is then placed on Hartmann's pouch and using the operator's left hand, is retracted to the patient's right, opening up the portahepatis. The anterior and posterior peritoneum over the neck of the gallbladder is then divided with a diathermy hook and Calot's triangle carefully dissected. Intraoperative cholangiogram can be performed in patients with a high risk of common bile duct stones, or to define the biliary anatomy. The cystic duct is clipped proximal and distal to the opening and then divided. If the cystic duct is wide, a ligature (e.g. Endoloop) may be used to secure the cystic duct stump. The cystic artery is then clipped and divided. The gallbladder is carefully dissected of the gallbladder bed.

Prior to the final disconnection, and using the gallbladder as a retractor, haemostasis of the gallbladder bed is secured and the positions of the clips placed on

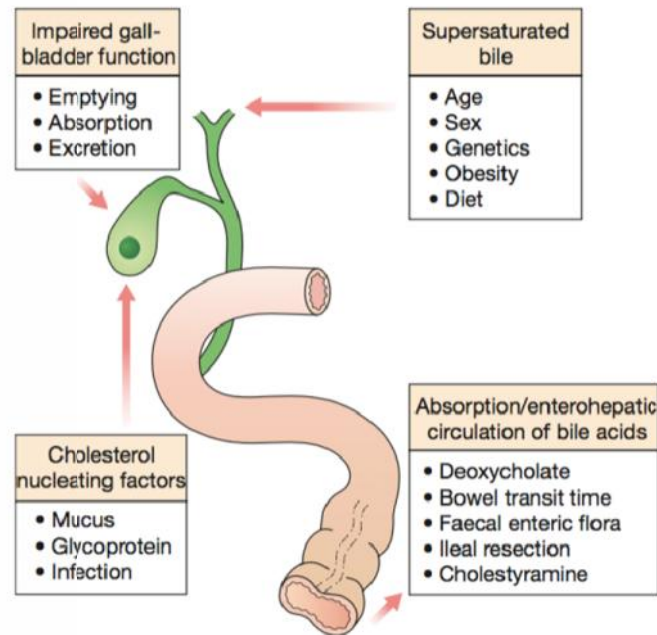
the cystic duct and the cystic artery are checked. The dissection is then completed and the gallbladder is retrieved via the epigastric or umbilical ports. If the gallbladder has been punctured, it should be retrieved in a bag, with every effort being made to aspirate the bile and recover any spilt stones. The pneumoperitoneum is then released and the ports are removed. The wounds are infiltrated with local anaesthetic and closed with absorbable sutures.

GALLBLADDER STONE DISEASE:

Gallstones are the most common biliary pathology. They are asymptomatic in the majority of cases (>80%). Approximately 1–2% of asymptomatic patients will develop symptoms requiring surgery per year, making cholecystectomy one of the most common operations performed by general surgeons.³⁴

FACTORS INVOLVED IN GALLSTONE FORMATION:

The process of gallstone formation is complex, and many areas remain unclear. **Obesity, high-caloric diets and certain medications (e.g. oral contraceptives)** can increase secretion of cholesterol and supersaturate the bile, increasing the lithogenicity of bile. **Resection of the terminal** ileum, which diminishes the enterohepatic circulation, will deplete the bile acid pool and result in cholesterol supersaturation.³⁵ Nucleation of cholesterol monohydrate crystals from multilamellar vesicles is a crucial step in gallstone formation.³⁶ **Abnormal emptying of the gallbladder** may aid the aggregation of nucleated cholesterol crystals, hence, removing gallstones without removing the gallbladder inevitably leads to gallstone recurrence.



Factors involved in gallstone formation.

TYPES OF GALLSTONES:

Current research suggests that different types of gallstones have different pathogenesis.^{37,38,39} 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.⁴⁰ Pigment stones can be further classified as either black or brown. In Asia, 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.^{41,42} 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.⁴³

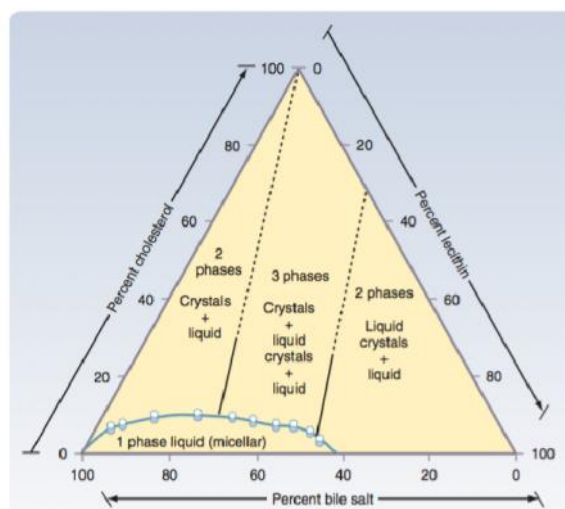
Whether pure or of mixed nature, the common primary event in the formation of cholesterol stones is super saturation of bile with cholesterol. Therefore, high bile cholesterol levels and cholesterol gallstones are considered as one disease. Cholesterol is highly nonpolar and insoluble in water and bile. Cholesterol solubility depends on the relative concentration of cholesterol, bile salts, and lecithin (the main phospholipid in bile). Super saturation almost always is caused by cholesterol hyper secretion rather than by a reduced secretion of phospholipid or bile salts.⁴⁴ Cholesterol is secreted into bile as cholesterol-phospholipid vesicles. Cholesterol is held in solution by micelles, a conjugated bile salt-phospholipid-cholesterol complex, as well as by the cholesterol-phospholipid vesicles. The presence of vesicles and micelles in the same aqueous compartment allows the movement of lipids between the two. Vesicular maturation occurs when vesicular lipids are incorporated into micelles. Vesicular phospholipids are incorporated into micelles more readily than vesicular cholesterol. Therefore, vesicles may become enriched in cholesterol, become unstable, and then nucleate cholesterol crystals.

In unsaturated bile, cholesterol enrichment of vesicles is in consequential. In the supersaturated bile, cholesterol-dense zones develop on the surface of the cholesterol-enriched vesicles, leading to the appearance of cholesterol crystals. About one third of biliary cholesterol is transported in micelles, but the cholesterol-phospholipid vesicles carry the majority of biliary cholesterol.⁴⁵

PIGMENT STONES:

Pigment stone is the name used for stones containing <25% cholesterol.⁴⁶ There are two types: black and brown. Black stones are largely composed of an insoluble bilirubin pigment polymer mixed with calcium phosphate and calcium bicarbonate. Overall, 20–30% of stones are black. The incidence rises with age. Black stones are associated with haemolysis, usually hereditary spherocytosis or sickle cell disease. For reasons that are unclear, patients with cirrhosis have a higher instance of pigmented stones.

Brown pigment stones contain calcium bilirubinate, calcium palmitate and calcium stearate, as well as cholesterol. Brown stones are rare in the gallbladder. They form in the bile duct and are related to bile stasis and infected bile. Stone formation is related to the deconjugation of bilirubin deglucuronide by bacterial - glucuronidase. Insoluble unconjugated bilirubinate precipitates. Brown pigment stones are also associated with the presence of foreign bodies within the bile ducts such as end prostheses (stents) or parasites such as *Clonorchis sinensis* and *Ascarislumbricoides*.⁴⁷



THE TRIANGLE OF SOLUBILITY

CLINICAL PRESENTATION:

Pain, gallstone colic or the **BILIARY COLIC** 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, gastro esophageal reflux disease, abdominal wall hernias, irritable bowel disease, diverticular disease, liver diseases, renal calculi, pleuritic pain, and myocardial pain.

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 hydrops of the gallbladder. The bile gets absorbed, but the gallbladder epithelium

continues to secrete mucus, and the gallbladder becomes distended with mucinous material. The gallbladder may be palpable but usually is not tender. Although hydrops may persist with few consequences, early cholecystectomy is generally indicated to avoid complications.

COMPLICATIONS OF GALLBLADDER STONE:

Biliary colic

Acute cholecystitis

Chronic cholecystitis

Empyema of the gallbladder

Mucocoele

Perforation

Biliary obstruction

Acute cholangitis

Acute pancreatitis

Intestinal obstruction (gallstone ileus)

MANAGEMENT:

The diagnosis of symptomatic gallstones or chronic calculous cholecystitis depends on the presence of typical symptoms and the demonstration of stones on diagnostic imaging. An abdominal ultrasound is the standard diagnostic test for gallstones.⁴⁸ Gallstones are occasionally identified on abdominal radiographs or CT scans. In these cases, if the patient has typical symptoms, an ultrasound of the gallbladder and the biliary tree should be added before surgical intervention. Stones diagnosed incidentally in patients without symptoms should be left in place. Occasionally, patients with typical attacks of biliary pain have no evidence of stones

on ultrasonography. Sometimes only sludge in the gallbladder is demonstrated on ultrasonography. If the patient has recurrent attacks of typical biliary pain and sludge is detected on two or more occasions, cholecystectomy is warranted. In addition to sludge and stones, cholesterosis and adenomyomatosis of the gallbladder may cause typical biliary symptoms and may be detected on ultrasonography. Cholesterosis is caused by the accumulation of cholesterol in macrophages in the gallbladder mucosa, either locally or as polyps. It produces the classic macroscopic appearance of a “strawberry gallbladder.” Adenomyomatosis or cholecystitis glandularis proliferans is characterized on microscopy by hypertrophic smooth muscle bundles and by the ingrowths of mucosal glands into the muscle layer (epithelial sinus formation) Granulomatous polyps develop in the lumen at the fundus, and the gallbladder wall is thickened and septae or strictures may be seen in the gallbladder. In symptomatic patients, cholecystectomy is the treatment of choice for patients with these conditions.⁴⁹

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.

INDICATIONS FOR CHOLECYSTECTOMY:

- Symptomatic cholelithiasis :
 - Biliary colic
 - Acute cholecystitis
 - Choledocholithiasis
 - Gallstone pancreatitis
 - Cholangitis or obstructive jaundice
- Asymptomatic cholelithiasis :
 - Sickle cell disease
 - Total parenteral nutrition
 - Chronic immunosuppression
 - No immediate access to health care facilities (eg, missionaries, military personnel, peace corps workers, relief workers)
 - Incidental cholecystectomy for patients undergoing procedure for other indications
- Acalculous cholecystitis :
 - Gallbladder dyskinesia
 - Gallbladder polyps >10 mm in diameter
 - Porcelain gallbladder

REVIEW OF LITERATURE:

Cholelithiasis is one of the commonest problem encountered in surgery . The pain due to the obstructing stone that causes sudden expansion of the gall bladder is called “Biliary Colic”⁵⁰. This typical pattern of pain occurs at right upper quadrant or epigastric region and lasts between 15mins to several hours commonly after a fatty meal. When pain gradually disappears it leaves behind a dull ache usually with nausea and vomiting. As the supply is splanchnic nerve, pain radiates to back, right scapula or shoulder tip and occasionally to back. Any other spectrum of symptoms that does not fit typical pain criteria is considered atypical and includes any abdominal discomfort, dyspepsia, nausea, belching, heart burn, food intolerance, flatulence, vomiting, and loss of appetite.⁵¹

The diagnosis of cholelithiasis has steadily increased because of the common access to ultrasound examination.^{52,53} The ultrasound finding of gallstones may be incidental and peptic ulcer disease of the stomach or the duodenum, gastro-oesophageal reflux or hiatus hernia may be the true cause of pain. Symptomatology of upper GI diseases can be overlapping. Upper abdominal pain may be secondary to either cholelithiasis or gastroduodenal diseases.

The majority of patients presenting to general practitioners with chronic or colicky upper abdominal pain undergo ultrasound examination. Ultrasound is non-invasive, readily available and inexpensive mode of investigation. After ultrasound detection of gallstones the main focus of the attending clinician revolves around treating the gallstones and further investigations to rule out other pathologies that may produce similar symptoms are seldom considered.

Almost all of the patients with proven gallstones are referred to surgeons with

a view to performing laparoscopic cholecystectomy. With the advancement in laparoscopic skills, laparoscopic cholecystectomy has become a very common and safe operation. As a result patients consent to surgery without as much consideration as they had done in the past, when it was performed with a much more invasive, open technique. A proportion of patients experience similar pain after laparoscopic cholecystectomy. The cause of this pain may be peptic ulcer disease, hiatus hernia or other causes.

These patients should first have been investigated to rule out gastroduodenal pathology before undergoing operation to remove gallstones. This approach will not only decrease persistence of symptoms but can also be helpful in detecting gastroduodenal pathologies at an early stage. Persistent post cholecystectomy pain has been reported in 20 -30% of patients .The presence of such persistent pain is also called “post cholecystectomy syndrome”.The relationship between such persistent pain and cholecystolithiasis is often unclear. Coexistence of concurrent upper gastrointestinal problems with gallstones may have attributed to the post cholecystectomy syndrome. There is always an immense challenge in the evaluation of patients with upper gastrointestinal symptoms, who have gallstones to decide whether the stones in gallbladder are the source of the symptoms or an incidental finding. Differentiating between these two situations is important, because the prevalence of both conditions is common in the general population.

1) Schwenket al.⁵⁴ suggested that because of the high incidence of simultaneous disease in the upper GI tract, preoperative OGD should be performed before elective surgical therapy of symptomatic cholelithiasis. In his study, 1064/ 1143(93.1%) patients underwent OGD and 30.2 %(345patients) had pathological findings. Of

these, 68.3% were inflammatory in nature. 28 patients (2.5%) underwent additional GI surgical procedures along with cholecystectomy and bile duct exploration. 227 (19.8%) underwent pharmacological treatment of the GI disease after their biliary surgery.

2) Diettrich et al,⁵⁵ 1990 studied on 100 patients, out of which 31 had abnormal OGD findings, 18% of patients had their cholecystectomy differed for 4 to 8 weeks, after additional medical treatment, 7 patients were discharged on only conservative medical treatment.

3) Rassek et al.⁵⁶ showed that out of 960 patients for elective cholecystectomy, 589 underwent gastroscopy, of them 56% had normal gastroscopy, and 11.3%(113 patients) underwent a change in plan of therapy because of the OGD findings.

4) Thybusch et al.⁵⁷ discuss the value and therapeutic implications of routine OGD before cholecystectomy. In his study 160/338 (47.3%) patients undergoing cholecystectomy also had OGD. Among those he observed peptic ulcer disease (in 6.8%), gastric erosions (1.8%), gastritis (25.7%), polyps (3.2%), hiatus hernia (4.7%) oesophagitis (3%) and gastric cancer (0.6%). Findings on OGD did not necessarily correlate with clinical symptoms. In 8.3% of patients the OGD findings influenced management and surgery was postponed awaiting medical treatment. Further, two patients with gastric cancer underwent gastrectomy.

5) Rashid et al.³ in his study, the routine use of UGI endoscopy resulted in detection of other co-existing pathologies in about one third (33%) of patients, which lead to a change in the management plan for these patients.

6) Sosada et al.⁵⁸ recommend routine pan-endoscopy for each patient who qualifies to

undergo laparoscopic cholecystectomy. He suggested that in asymptomatic cholelithiasis, pain is because of a peptic ulcer. Out of 2800 treated for cholelithiasis, OGD which was performed 1–4 days prior to surgery, showing pathological changes in the stomach or duodenum in 1187(42%) patients; gastric ulcer in 179 (6.4%), duodenal ulcer in 127 (4.5%), gastritis in 375 (26.3%), polyps in 143(5.1%) and cancer in (3.1%) patients. The surgery was delayed for patients with ulcers and they were treated appropriately. 16 patients were asymptomatic after healing of the ulcer, thus they were proven to have asymptomatic cholelithiasis and a cholecystectomy was not performed.

7) Similar studies by Venkatesh Kolla et al.⁵⁹ 74 showed a predominance of positive findings on UGE, in the group with atypical symptoms (p=0.004). Therapeutic approach was changed in a total of 10 (4.6%), who were diagnosed with ulcer, malignancy and celiac disease.

8) Similarly Karmacharya A et al.⁶⁰ 75 showed that patients with typical pattern of pain had normal endoscopic findings and those with atypical pain had pathology in upper gastroendoscopy (p<0.001). Serious pathology resulting to change of the planned treatment was found in three cases (3.12%). Among them two had gastric carcinoma and one had active peptic ulcer disease.

Literature review regarding the role of upper gastrointestinal endoscopy for gall stone disease patients reveals:

Gall stones do not cause any symptoms in 80% of carriers. Within 5 years only 10 to 15 % become symptomatic. The risk of further symptoms is approximately 2% per year with an overall risk of biliary complications (acute pancreatitis and acute choledocholithiasis) in asymptomatic patients of 0.3% per year; the risk of gall

bladder cancer is 0.02% per year.

Half of the patients with symptoms develop an attack within the year, but in 30% of all the cases, there is only one symptomatic attack. The approximate rates at which subjects **develop** biliary pain is 2% and with subsequent decrease over time. Although biliary colic was specific for gallstones, 80 % of the referred patients with gallstones presented with other abdominal symptoms. There is no current evidence that justifies the use of single abdominal symptoms, other than biliary colic, in the diagnosis of symptomatic gallstones. Further research should focus on the prognosis of patients with non-specific abdominal symptoms and gallstones. About two thirds of patients with gallstones, develop symptoms infrequently and complications at an even lower rate. In most cases, gallstones with an atypical presentation should have other causes of right upper quadrant pain ruled out such as peptic ulcer disease, pneumonia, renal calculi, liver disease, hernia, reflux, or angina.⁶¹

An innocent condition such as asymptomatic gallstones, one need to know the specific symptoms of gallstone disease. In total, 80% of the patients with gallstones were referred with other abdominal symptoms.⁶² The discussion remains whether these latter patients had asymptomatic gallstones or their gallstones caused symptoms other than biliary colic. The benefit of cholecystectomy for gallstone patients with dyspepsia has remained debatable. Only about one half of patients were symptom free after cholecystectomy. About 20-30% of cholecystectomised patients still complained of dyspepsia, which might have been associated with preoperatively undiagnosed functional gut diseases unrelated to gallstones.⁶³ Because gallstone dyspepsia and functional dyspepsia had coexisting symptoms, it is difficult to differentiate them based on dyspeptic symptoms. It is

therefore important and necessary to identify and treat these co-existing upper gastrointestinal conditions, so that the patient is completely relieved of his symptoms after surgery and subsequent treatment.

METHODOLOGY

This is a hospital based one year cross-sectional study conducted in KLES DR PRABHAKAR KORE HOSPITAL.

STUDY DESIGN: Observational study.

STUDY PLACE: The study was conducted in, DEPARTMENT OF GENERAL SURGERY, KLES DR PRABHAKAR KORE HOSPITAL.

STUDY PERIOD: ONE YEAR PERIOD (JANUARY 2017-DECEMBER 2017).

SOURCE OF DATA: All symptomatic patients getting admitted to the surgery wards, in KLES DR PRABHAKAR KORE HOSPITAL with ultrasonographically proven GALL BLADDER STONE DISEASE, who merit surgery.

Inclusion criteria:

All symptomatic patients having GB stone disease proven by ultrasonography who merit surgery.

Exclusion criteria:

- Acute abdomen
- CBD stones
- Obstructive jaundice
- Cholangitis
- GB stone pancreatitis
- GB neoplasm
- Previous GB or pancreatic surgery.

SAMPLE SIZE: 66.

Rationale for Sample Size :

A study carried out by Mohamed Toum Musa M Ibrahim et al⁶⁴ has observed that the pathological findings with upper GI endoscopy for all those diagnosed with Gall bladder stone disease was 60%.

In our study expecting similar results with 95% confidence levels and 20% of relative precision in the result, the study requires a minimum of 66 subjects.

$$n=(Za)^2[pXq]/d^2$$

$$n=2^2[60X40]/12^2$$

$$n=9600/144$$

$$n=66.$$

METHOD: This prospective study was conducted on the patients meeting the inclusion criteria, for patients attending the **GENERAL SURGERY OPD** at KLES DR PRABHAKAR KORE HOSPITAL.

After clinical assessment, and ultrasonography, patients were admitted and subjected to UGI scopy and a biopsy would be taken if necessary.

The statistical analysis was be performed using SPSS version 22.

Institutional approval was taken for the study. Verbal and written informed consent was taken from each participant. The data collected from the patients includes personal information, presenting symptoms – typical (biliary colic) / atypical (vauge upper abdominal symptoms), investigations including ultrasonography, Upper GI endoscopy findings, biopsy reports if present.

Typical biliary colic is defined as the pain which 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.

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.

UGE was performed in the endoscopy room 1-2 days prior to operation. The presence of ulcer and inflammation of gastric mucous membrane was subjected to histopathological examination. The clinical symptoms were categorized as typical or atypical based on the characters mentioned above. The endoscopic findings were divided as normal, inflammatory, erosions, ulcers and others including carcinomas. Similar categorizations were applied for histopathological findings as well.

STATISTICAL ANALYSIS OF DATA:

Descriptive statistics of the pathological UGE findings (gastritis, hiatus hernia, peptic ulcer disease, polyps, malignancy etc.) were analysed in terms of percentage and its 95% confidence levels. All the data was entered in MS EXCEL and analysed using SPSS 22. Chi square test will be used to find the association between pain type, gender and endoscopic findings.

RESULT

TABLE 1: DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO AGE.

AGE IN YEARS	FREQUENCY	PERCENT
16-30	15	22.7%
31-45	15	22.7%
46-60	22	33.3%
61-75	13	19.7%
76-90	1	1.6%
TOTAL	66	100%

	MEAN	STANDARD DEVIATION
AGE	45.80	16.56

In the study a total of 66 patients were included, age ranges from **16-90** years, for both male and female with mean age of **45.80**.

In the present study 33.3% (n=22) of them were found in the age group ranging from 46-60. Youngest patient was **16** years of age and oldest patient was **81** years of age.

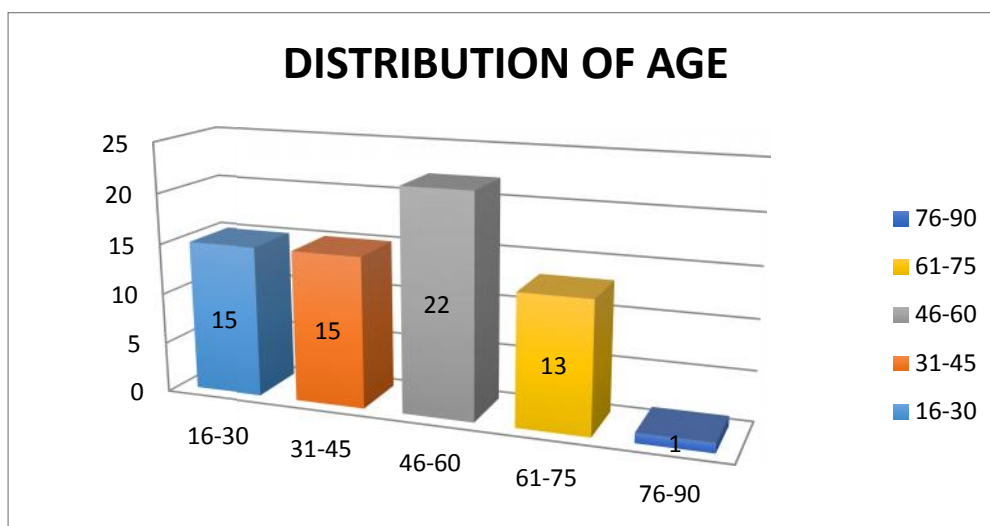


FIGURE 1: BAR DIAGRAM SHOWING AGE DISTRIBUTION OF STUDY SUBJECTS

TABLE 2: DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO SEX.

SEX	FREQUENCY	PERCENTAGE
MALE	28	42.4%
FEMALE	38	57.6%
TOTAL	66	100%

The sex distribution of the study subjects showed a **female preponderance**, with **38** of the 66 study subjects, i.e. 57.6% were females, whereas **28** of the 66 study subjects, i.e. 42.4% were males, with a male to female ratio of **1:1.3**.

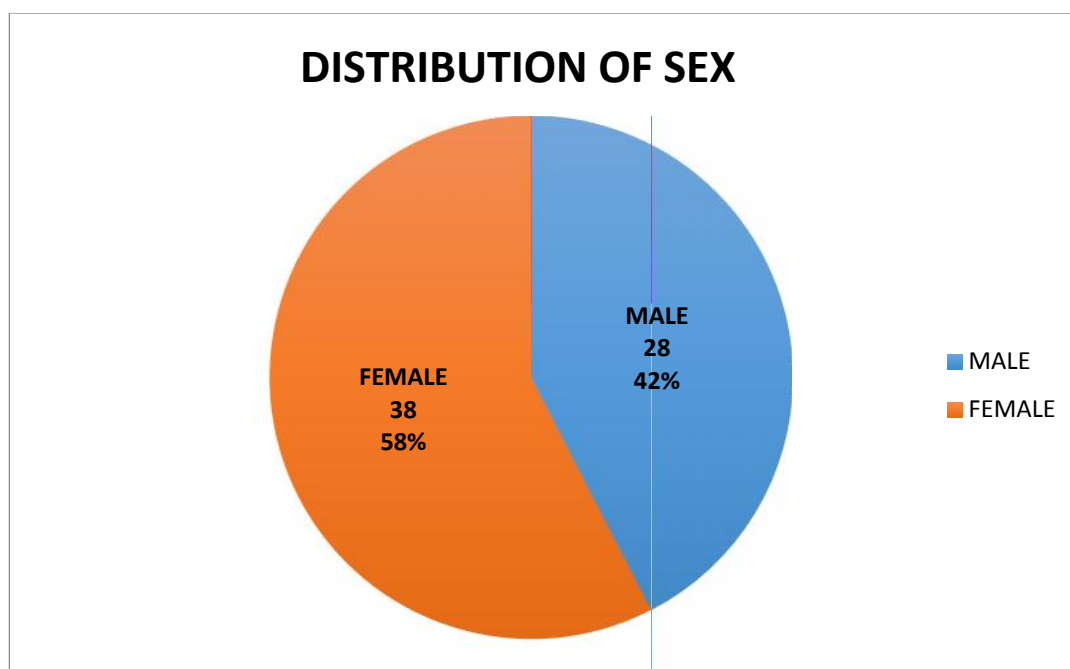


Figure 2: PIE CHART SHOWING SEX DISTRIBUTION OF STUDY SUBJECTS

TABLE 3: DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO SYMPTOMS.

TYPE OF GALLBLADDER SYMPTOMS	FREQUENCY	PERCENTAGE
TYPICAL SYMPTOMS	30	45.5%
ATYPICAL SYMPTOMS	36	54.5%
TOTAL	66	100%

Analysis of study subjects with cholelithiasis revealed that **majority** of patients had **atypical symptoms**, i.e. **36** of 66 study subjects (54.5%), and **30** of the 66 patients had **typical symptoms**, i.e. 45.5%.

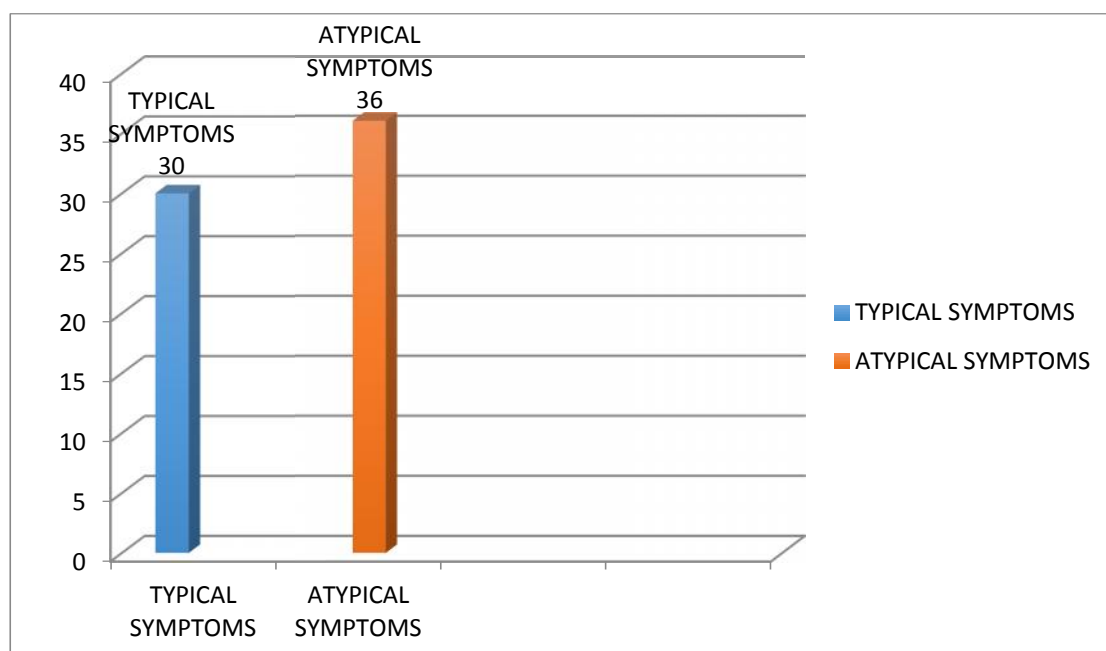


Figure 3: BAR DIAGRAM SHOWING DISTRIBUTION OF TYPE OF SYMPTOMS AMONG STUDY SUBJECTS.

TABLE 4: DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO UGI SCOPY FINDINGS.

UGI ENDOSCOPY FINDINGS	FREQUENCY	PERCENTAGE
NORMAL UGI SCOPY	42	63.6%
POSITIVE UGI SCOPY	24	36.4%
TOTAL	66	100%

The study subjects, were subjected to UGI scopy 1-2 days before their planned laparoscopic cholecystectomy, out of the 66 subjects, **42** i.e.63.6% of the patients were found to have **normal findings**, and **24** i.e.36.4% were found to have **positive UGI findings**.

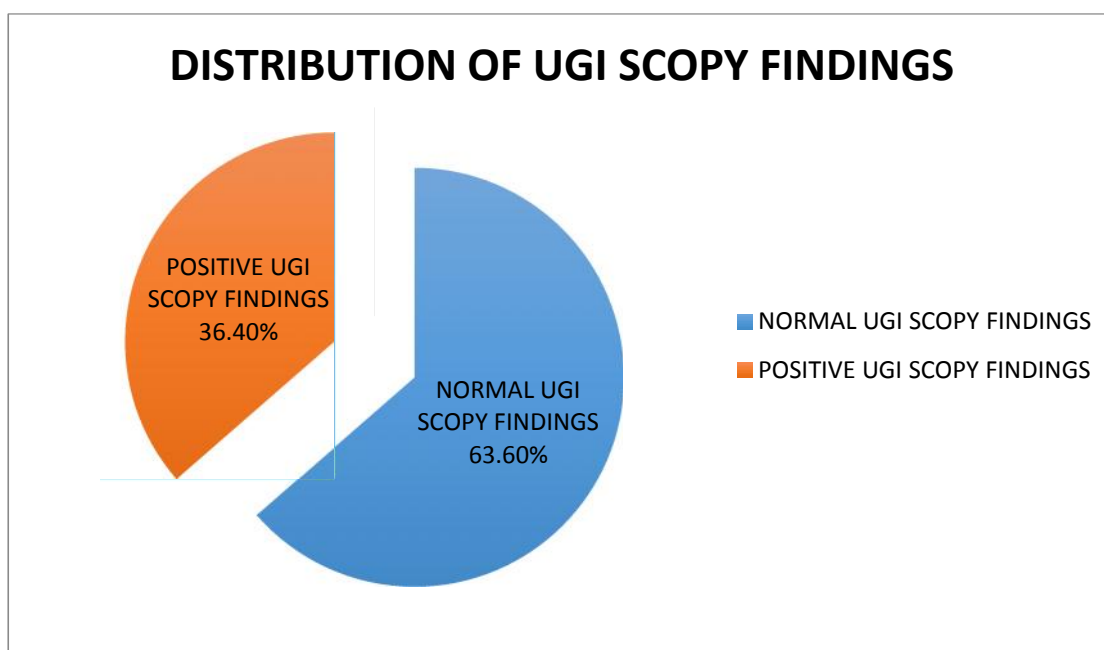


FIGURE 4: PIE CHART SHOWING THE DISTRIBUTION OF UGI SCOPY FINDINGS AMONG STUDY SUBJECTS.

TABLE 5: SEX WISE DISTRIBUTION OF PATIENTS WITH TYPICAL AND ATYPICAL BILIARY SYMPTOMS.

SEX	TYPICAL BILIARY SYMPTOMS	ATYPICAL BILIARY SYMPTOMS	TOTAL	p-VALUE
MALE	15(53.5%)	13(46.5%)	28(100%)	0.256
FEMALE	15(39.5%)	23(60.5%)	38(100%)	
TOTAL	30(45.5%)	36(54.5%)	66(100%)	

Among the 66 subjects included in the study, 53.5% (n=15) and 39.5% (n=15) of males and females respectively had typical biliary symptoms, whereas 46.5% (n=13) and 60.5% (n=23) of males and females respectively had atypical biliary symptoms. However the sex wise distribution of type of biliary symptoms was statistically not significant ($p=0.256$)

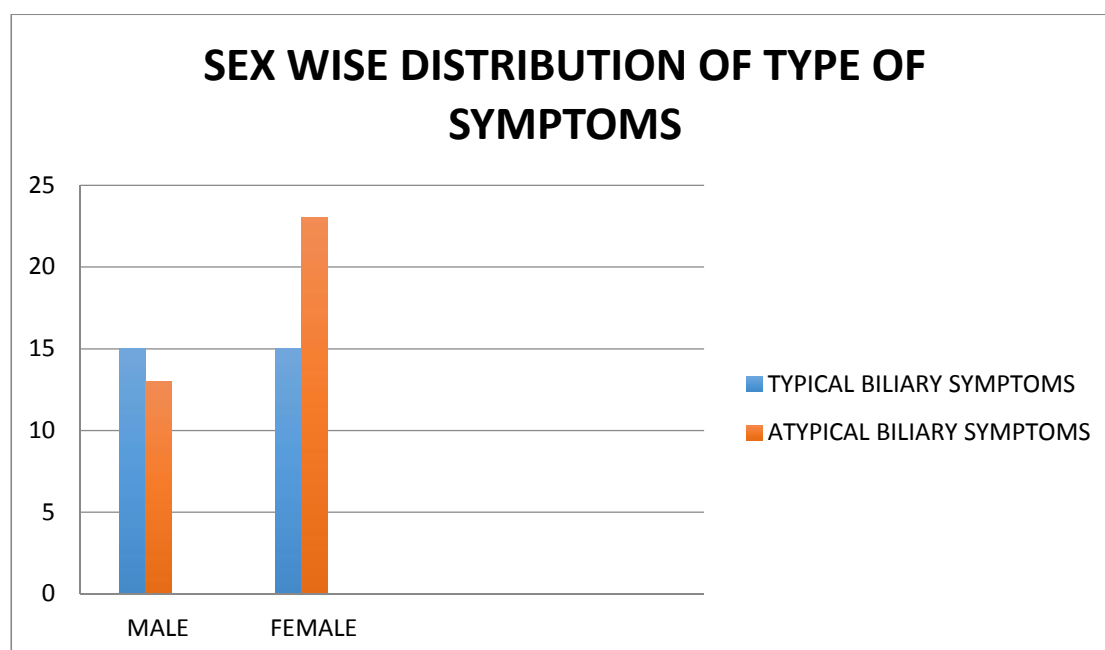


Figure 5: BAR DIAGRAM SHOWING SEX WISE DISTRIBUTION OF TYPE OF BILIARY SYMPTOMS.

TABLE 6: AGE WISE DISTRIBUTION OF TYPE OF BILIARY SYMPTOMS.

AGE	TYPICAL BILIARY SYMPTOMS	ATYPICAL BILIARY SYMPTOMS	TOTAL	p-VALUE
16-30	11(73.3%)	4(26.7%)	15(100%)	0.357
31-45	8(53.3%)	7(46.7%)	15(100%)	
46-60	7(31.8%)	15(68.2%)	22(100%)	
61-75	4(30.8%)	9(69.2%)	13(100%)	
76-90	0(0%)	1(100%)	1(100%)	
TOTAL	30(45.5%)	36(54.5%)	66(100%)	

This table shows the age wise distribution of type of biliary symptoms, the incidence of typical biliary symptoms was higher in the 16-30 age group, whereas atypical biliary symptoms were higher in the 61-75 age group. However the relationship between age and type of biliary symptoms is not statistically significant ($p=0.357$).

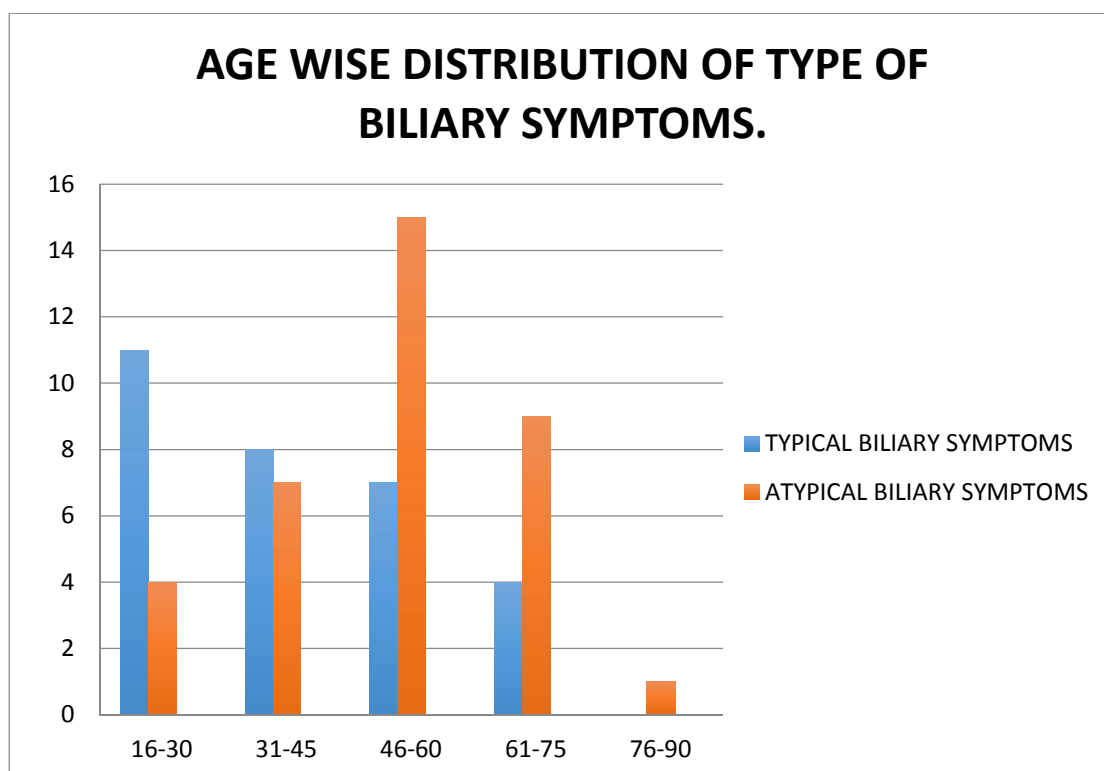


Figure 6: BAR DIAGRAM SHOWING AGE WISE DISTRIBUTION OF TYPE OF BILIARY SYMPTOMS.

TABLE 7: AGE WISE DISTRIBUTION OF UGI SCOPEY FINDINGS.

AGE	NORMAL UGI SCOPEY	POSITIVE UGI SCOPEY	TOTAL	p-VALUE
16-30	12(80%)	3(20%)	15(100%)	0.486
31-45	9(60%)	6(40%)	15(100%)	
46-60	14(63.6%)	8(36.4%)	22(100%)	
61-75	7(53.8%)	6(46.2%)	13(100%)	
71-90	0(0%)	1(100%)	1(100%)	
TOTAL	42(63.6%)	24(36.4%)	66(100%)	

Among the age distribution of study subjects the incidence of normal UGI scopy findings was higher in the 16-30 years age group, where as incidence of positive UGI scopy findings was higher in the 61-75 years age group. However the relationship between age and UGI scopy findings was not significant ($p=0.486$).

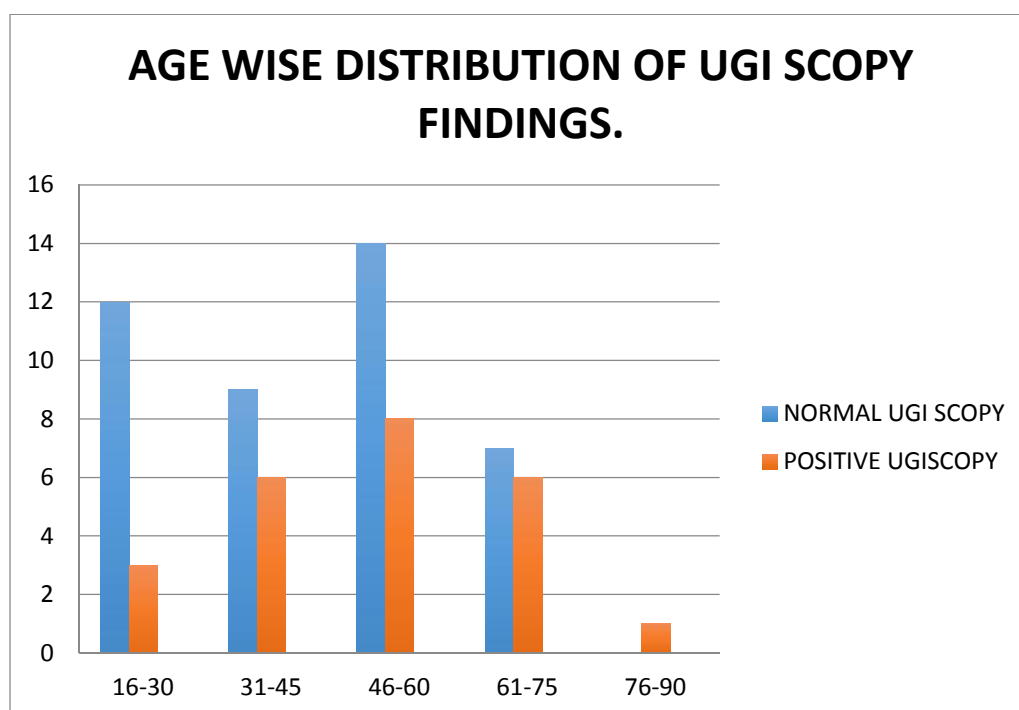
**Figure 7: BAR DIAGRAM SHOWING AGE WISE DISTRIBUTION OF UGISCOPY FINDINGS.**

TABLE 8: DISTRIBUTION OF UGI SCOPY FINDINGS.

UGI SCOPY FINDINGS	FREQUENCY	PERCENT
LAX CARDIA, FUNDAL GASTRITIS	7	10.6%
CHRONIC DIFFUSE EROSIVE GASTRITIS	1	1.5%
LAX CARDIA, GRADE B ESOPHAGITIS	5	7.8%
SCATTERED GASTRIC EROSIONS IN FUNDUS AND ANTRUM	2	3.0%
PERIAMPULLARY DIVERTICULUM IN SECOND PART OF DUODENUM	1	1.5%
LAX CARDIA, FUNDAL GASTRITIS, GRADE A ESOPHAGITIS	3	4.5%
DIFFUSE CHRONIC GASTRITIS	2	3.0%
LAX CARDIA, BILE REFLUX ESOPHAGITIS, DUODENITIS.	1	1.5%
LAX CARDIA, HIATUS HERNIA	1	1.5%
BILE REFLUX GASTRITIS	42	63.6%
NORMAL		
TOTAL	66	100%

The above table shows the break up of the different UGI scopy findings, with majority of patients, 63.6% having normal findings, majority of the positive findings in UGI scopy were fundal gastritis and esophagitis.

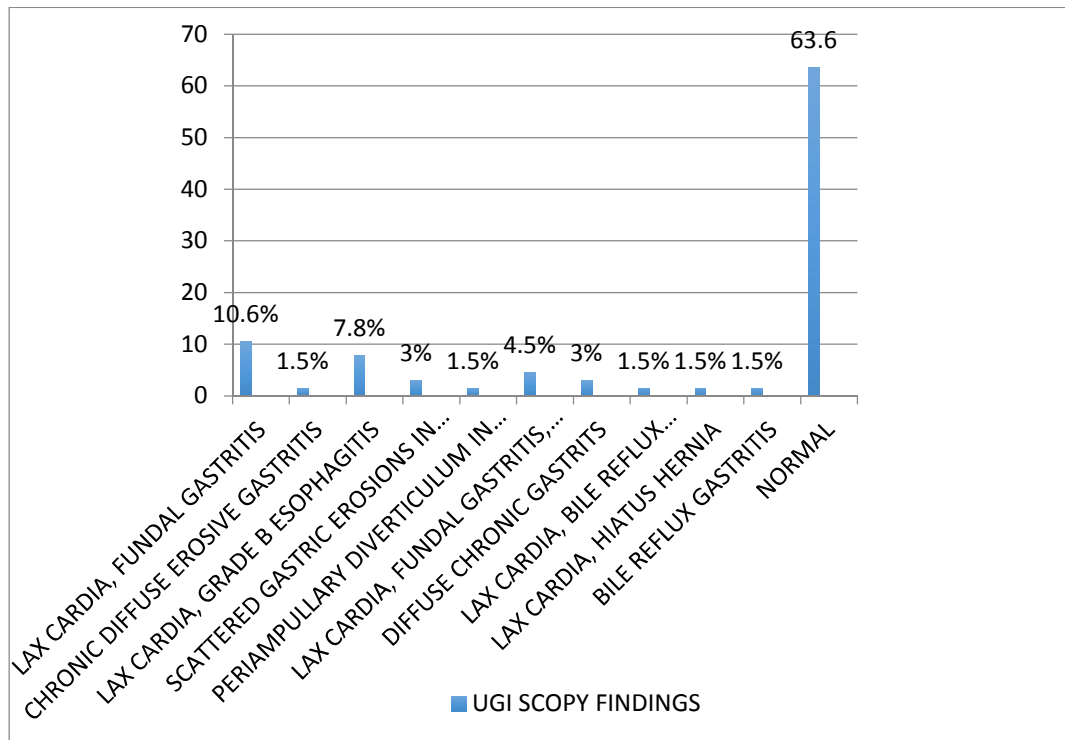


FIGURE 8: BAR DIAGRAM SHOWING THE FREQUENCY OF DIFFERENT UGI SCOPY FINDINGS

TABLE 9: RELATIONSHIP BETWEEN TYPE OF BILIARY SYMPTOMS AND UGI SCOPY FINDINGS.

TYPE OF BILIARY SYMPTOMS	NORMAL UGI SCOPY	POSITIVE UGI SCOPY	TOTAL	p-VALUE
TYPICAL BILIARY COLIC	23(76.7%)	7(23.3%)	30(100%)	0.045
ATYPICAL BILIARY COLIC	19(52.8%)	17(47.2%)	36(100%)	
TOTAL	42(63.6%)	24(36.4%)	66(100%)	

$\chi^2 = 4.036$, $df=1$, $p=0.045$.

Among the 66 study subjects 36.4% (n=24), were found to have positive UGI scopy findings, out of which 29.2% (n=7) had typical biliary colic and 70.8% (n=17) had atypical biliary symptoms.

In comparison with this 63.6% (n=42) of the study subjects had normal UGI scopy findings, out of which 54.8% (n=23) had typical biliary colic and 45.2% (n=19) had atypical biliary colic.

Inferring that the chances of yielding a positive UGI scopy is higher in patients with atypical biliary symptoms.

TABLE 10: DISTRIBUTION OF POSITIVE UGI SCOPY WITH TYPE OF BILIARY SYMPTOMS.

TYPE OF BILIARY SYMPTOMS	POSITIVE UGI SCOPY
TYPICAL BILIARY COLIC	7(29.2%)
ATYPICAL BILIARY SYMPTOMS	17(70.8%)
TOTAL	24(100%)

Among the 24 subjects in the study who had positive UGI scopy findings, 7 (29.2%) had typical biliary symptoms and 17 (70.8%) had atypical biliary symptoms.

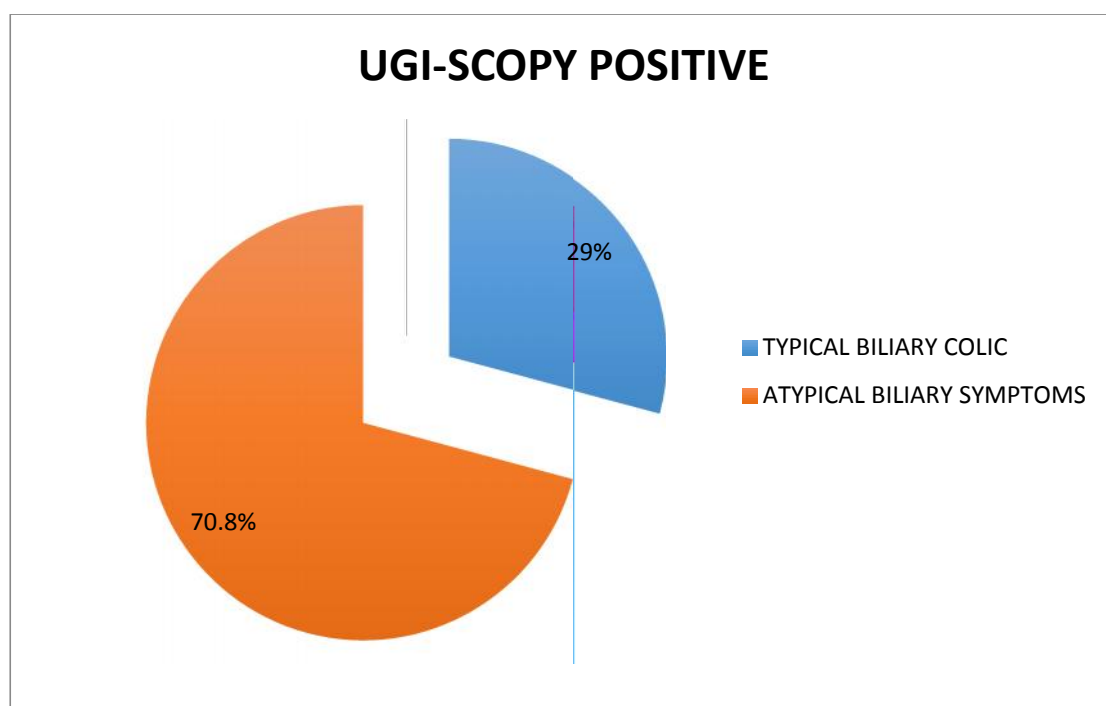


FIGURE 10: PIE CHART SHOWING THE DISTRIBUTION OF POSITIVE UGI SCOPY FINDINGS AMONG PATIENTS WITH TYPICAL AND ATYPICAL BILIARY SYMPTOMS.

DISCUSSION

Upper gastro intestinal endoscopy has been recommended pre-operatively in patients undergoing elective laparoscopic cholecystectomy for gall bladder stone disease by Thybusch et al.,⁵⁷ Sosada et al.⁵⁸ and many others to diagnose the co-existent UGI lesions in these patients, which otherwise went unnoticed and led to persistence of the patients symptoms post surgery.

A total of 66 patients were included in the study, the age of the patients ranged from 16-81 years, and most of the patients belonged to the 46-60 age group, the mean age of the patients was **45.8 years**, which is comparable to other studies (40.6).^{65,66,67}

Female preponderance was noted in the study with 57.6% (n=38) females and 42.4% (n=28) males, with a male to female ratio of **1:1.3**, which is strongly supported by many studies. Rashid et al.³ also published similar results with male to female ratio 1:4.

This study has shown that majority of the patients **54.5%** (n=36) presented with **atypical biliary symptoms**, as compared to patients who presented with typical **biliary colic**, (**45.5%**, n=30).

This study found that **36.4%** (n=24) of the study subjects had positive UGI-scopy findings, which is statistically significant with a p value of **0.045**. This result is supported by

- Study performed by Diettrich et al.⁵⁵ also suggested 31/100 patients had abnormalities on UGI-SCOPY.
- Schwenk et al.⁵⁴ suggested that 30.2% (345 patients) had pathological findings.
- Rassek et al.⁵⁶ recommends that investigation of the upper gastrointestinal tract must precede an elective cholecystectomy, as his study showed that 44% of patients had positive UGI-SCOPY findings.

It has been observed from this study that the prevalence of **typical biliary colic** is higher among the **young** (16-30 years) as compared to atypical symptoms i.e. **73.3%** and 26.7% respectively, while on the other hand **atypical biliary symptoms** is more common in the **older** age group (61-75 years) than typical biliary colic i.e. **69.2%** and 30.8% respectively.

Among the 66 study subjects, 39.5% and **60.5%** of the **females** had typical and **atypical** biliary symptoms respectively, whereas **53.5%** and 46.5% of males had **typical** and atypical symptoms respectively. But the sex wise distribution of symptoms was almost equal and not statistically significant with a p value of **0.256**.

Among the 66 study subjects **36.4%** (n=24), were found to have **positive** UGI scopy findings, out of which 29.2% (n=7) had typical biliary colic and 70.8% (n=17) had atypical biliary symptoms. In comparison with this **63.6%** (n=42) of the study subjects had **normal** UGI scopy findings, out of which 54.8% (n=23) had typical biliary colic and 45.2% (n=19) had atypical biliary colic. Inferring that the chances of yielding a positive UGI scopy is higher in patients with atypical biliary symptoms.

In a similar study by Mohamad Mozafar et al.⁶⁸ they found that, among the 178 patients with atypical pain, 148 (83%) had abnormal findings in UGI-SCOPY. In another study FAISAL et.al.⁶⁹ concluded that 77.2% of their patients with a typical symptoms had positive UGI-SCOPY findings.

Karmacharya A et al⁶⁰ have observed that normal UGI findings are seen in patients with typical pain reinforcing the fact that patients with typical abdominal pain has less likelihood of presenting with coexisting upper gastrointestinal lesion as evidenced in other studies.

Sosada et al.⁵⁸ observed in his study gastric ulcer in 179 (6.4%), duodenal ulcer in 127 (4.5%), gastritis in 375 (26.3%), polyps in 143 (5.1%) and cancer in 3

(0.1%) patients. Similarly in this study UGI-SCOPY showed lax cardia and fundal gastritis in 7 (10.6%), chronic diffuse erosive gastritis in 1 (1.5%), lax cardia and grade b esophagitis in 5 (7.8%), scattered gastric erosions in fundus and antrum in 2 (3.0%), periampullary diverticulum in second part of duodenum in 1 (1.5%), lax cardia with fundal gastritis and grade a esophagitis in 3 (4.5%), diffuse chronic gastritis in 2 (3.0%), lax cardia with bile reflux esophagitis and duodenitis in 1 (1.5%), lax cardia and hiatus hernia in 1 (1.5%), bile reflux gastritis in 1 (1.5%).

CONCLUSION

The increased access to ultrasound for patients presenting with pain abdomen, has led to an increase in the diagnosis of cholelithiasis, as its symptomatology overlaps many upper GI lesions, the incidental finding of gall stones on ultrasound may mask the co-existing UGI lesions.

In this study UGI lesions were found in 36.4% of the patients. Hence treating either one of them only will lead to persistence of symptoms post treatment.

Thus we **recommend an UGI-SCOPY** pre-operatively for patients undergoing elective laparoscopic cholecystectomy for gallbladder stone disease for adequate diagnosis and treatment of the co-existing UGI lesions if present and reduce the incidence of post operative persistence of symptoms.

SUMMARY

In summary, this study included **66 study subjects**, who met the inclusion criteria. The mean age of the subjects was **45.80 years**, with a male: female ratio of **1:1.3**. Of the 66 patients, 24 (36.4%) patients had positive findings on UGI scopy which was statistically significant with a p-value of 0.045.

The study also inferred that the chances of positive UGI-SCOPY findings were higher in patients presenting with atypical biliary symptoms i.e. 70.8%, as compared to 29.2% of patients who had typical biliary colic.

BIBLIOGRAPHY

- 1) Wang JK, Foster SM, Wolff BG. Incidental Gallstones. *The Permanente Journal*. 2009;13(2):50-54.
- 2) Nakeeb A, Comuzzie AG, Martin L, et al. Gallstones: Genetics Versus Environment. *Annals of Surgery*. 2002;235 (6):842-849.
- 3) Rashid F, Rashid N, Waraich N, Ahmed J, Iftikhar SY. Role of routine oesophagogastroduodenoscopy before cholecystectomy. *Int J Surg*. 2010;8[(3)]:236-8.
- 4) Epidemiology of Gallbladder Disease: Cholelithiasis and Cancer Laura M. Stinton and Eldon A. Shaffer, *Gut Liver*. 2012 Apr; 6(2): 172–187.
- 5) Glambek I., Arnesjo B. and Soreide O.: Correlation between gallstones and abdominal symptoms in a random population. Results from a screening study. *Scand J. Gasstroentrol. Ap.*, , 1989;24 (3):277-81.
- 6) Gibney EJ. Asymptomatic gallstones. *Br J Surg*. 1990;77:368–372. [PubMed]. Sakorafas GH, Milingos D, Peros G. Asymptomatic cholelithiasis: is cholecystectomy really needed? A critical reappraisal 15 years after the introduction of laparoscopic cholecystectomy. *Dig Dis Sci*. 2007;52:1313–1325.
- 7) Janowitz P, Kratzer W, Zemmler T, et al.: Gallbladdersludge: Spontaneous course and incidence of complications in without stones. *Hepatology*. Aug, 1994;20 (2):291-4,
- 8) Kraag N, Thijs C, Knipschild P. Dyspepsia - how noisy are gallstones? A meta-analysis of epidemiologic studies of biliary pain, dyspeptic symptoms, and food intolerance. *Scand J Gastroenterol*. 1995;30(5):411-21.
- 9) Schirmer B D, Winters K L, Edlich R F. Cholelithiasis and cholecystitis. *J Long*

- Term Eff Med Implants. 2005;15(3):329-38.
- 10) Murshid K R. The post cholecystectomy syndrome, a review. SGA. 1996;2(3):124-37.
- 11) Schofer J.M.: Biliary causes of postcholecystectomy syndrome. J. Emerg. Med. Aug, 22 [Epub ahead of print], 2008.
- 12) Talley N.J: Gallstones and upper abdominal discomfort, Innocent bystander or a cause of dyspepsia? J. Clin. Gastroenterol. 1995; 20 (3): 182-3.
- 13) Di Ciaula A, Wang DQ, Portincasa P 2012 Gallbladder and gastric motility in obese newborns, pre-adolescents and adults. J Gastroenterol Hepatol 27:1298–305.
- 14) Guiteau JJ, Fisher M, Cotton RT et al 2009 Intrahepatic gallbladder. J Am Coll Surg 209:672.
- 15) Gupta V, Singh V, Sewkani A et al 2009 Torsion of gall bladder, a rare entity: a case report and review article. Cases J 2:19
- 16) Bartel J. Cholelithiasis und Körperkonstitution cholelithotripsie. Frankfurt Z Pathol 1916;19:206.
- 17) Lindner HH, Green RB. Embryology and surgical anatomy of the extrahepatic biliary tract. Surg Clin North Am 1964;44:1273.
- 18) Dowdy GS Jr, Waldron GW, Brown WG. Surgical anatomy of the pancreatobiliary ductal system. Arch Surg 1962; 84:93.
- 19) Balijs M, Huis M, Nikoli V, Štulhofer M. Laparoscopic visualization of the cystic artery anatomy. World J Surg 1999;23: 703-707. [PubMed: 10390590]
- 20) Sugita M, Ryu M, Satake M, Kinoshita T, Konishi M, Inoue K, Shimada H.

- Intrahepatic inflow areas of the drainage vein of the gallbladder: analysis by
angio-CT. *Surgery* 2000;128:417-421. [PubMed: 10965313]
- 21) Fahim RB, McDonald JR, Richards JC, Ferns DO. Carcinoma of the gallbladder:
a study of its modes of spread. *Ann Surg* 1962; 156:114. [PubMed: 13891308]
- 22) Clermont D. Lymphatiques des voies biliares. *Toulouse Med* 1909; 814:114.
- 23) Rouviere H. Anatomy of the Human Lymphatic System. Tobias MJ (transl). Ann
Arbor MI: Edwards Brothers, 1938.
- 24) Burnett W, Cairns FW, Bacsich P. Innervation of the extrahepatic biliary system.
Ann Surg 1964;159:8. [PubMed: 14103280]
- 25) Calot JF. De la cholécystectomie. (Thesis) Paris, 1890, No. 25, p. 50.
- 26) Moosman DA, Collier FA. Prevention of traumatic injury to the bile ducts: a study
of the structures of the cystohepatic angle encountered in cholecystectomy and
supraduodenal choledochostomy. *Am J Surg* 1951;32:132.
- 27) Guyton AC. Textbook of Medical Physiology (7th ed). Philadelphia: WB
Saunders, 1986.
- 28) Paigen B, Carey M C. Gallstones. In: The Genetic Basis of Common Diseases,
King, Rotter, Motulsky, eds. 2nd Edition. 2001.
- 29) Everhart JE, Khare M, Hill M, et al. Prevalence and ethnic differences in
gallbladder disease in the United States. *Gastroenterology* 1999; 117: 632–39.
- 30) Lammert F, Carey MC, Paigen B. Chromosomal organization of candidate genes
involved in cholesterol gallstone formation: a murine gallstone map.
-

- Gastroenterology 2001; 120: 221–38.
- 31) Sarin SK, Negi VS, Dewan R, et al. High familial prevalence of gallstones in the first-degree relatives of gallstone patients. *Hepatology* 1995; 22: 138–141
- 32) Almasy L, Blangero J. Multipoint quantitative-trait linkage analysis in general pedigrees. *Am J Hum Genet* 1998; 62: 1198–1211.
- 33) Shiffman ML, Sugerman HJ, Kellum JM, et al. Gallstone formation after rapid weight loss: a prospective study in patients undergoing gastric bypass surgery for treatment of morbid obesity. *Am J Gastroenterol* 1991; 86: 1000–1005 .
- 34) Brett M, Barker DJ. The world distribution of gallstones. *Int J Epidemiol.* 1976;5:335.
- 35) Di Ciaula A, Garruti G, Lunardi Baccetto R, Molina-Molina E, Bonfrate L, Wang DQ, Portincasa P. Bile Acid Physiology. *Ann Hepatol.* 2017 Nov;16(Suppl. 1: s3-105.):s4-s14. [PubMed]
- 36) Jones MW, Deppen JG. Physiology, Gallbladder. [Updated 2018 Jan 8]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2018 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK482488/>
- 37) Stolk MF, Van Erpecum KJ, Peeters TL, Samsom M, Smout AJ (2001) Interdigestive gallbladder emptying, antroduodenal motility, and motilin release patterns are altered in cholesterol gallstone patients. *Dig Dis Sci* 46 (6): 1328–34. [PubMed]
- 38) Maki T (1966) Pathogenesis of calcium bilirubinate gallstone. *Ann Surg* 164

- (1):90–100.
- 39) Elek G, Rockenbauer A (1982) The free radical signal of pigment gallstone. *Klin Wochenschr* 60 (1): 33–5. [PubMed]
- 40) Ravnborg L, Teilum D, Pedersen LR, Gallbladder stones classified by chemical analysis of cholesterol content. Frederiksberg, 1987-1988. *Scand J Gastroenterol.* 1990 Jul; 25(7):720-4
- 41) Arora, Bhavinder. (2017). EVALUATION OF THYROID PROFILE IN PATIENTS WITH GALLSTONE DISEASE.. *Global Journal for Research Analysis.* 6. 723-726.
- 42) Dr. Rahmath Unissa et al, *International Journal of Pharmaceutical Sciences and Business Management*, Vol.1 Issue. 1, September- 2013, pg. 32-34
- 43) Bell GD, Dowling RH, Whitney B, Sutor DJ. The value of radiology in predicting gallstone type when selecting patients for medical treatment. *Gut.* 1975;16(5):359-364.
- 44) Dijkers A, Tietge UJ. Biliary cholesterol secretion: More than a simple ABC. *World Journal of Gastroenterology: WJG.* 2010;16(47):5936-5945. doi:10.3748/wjg.v16.i47.5936.
- 45) Small DM. Role of ABC transporters in secretion of cholesterol from liver into bile. *Proceedings of the National Academy of Sciences of the United States of America.* 2003;100(1):4-6. doi:10.1073/pnas.0237205100.
- 46) R. D. SOLOWAY, B. W. TROTMAN, et.al. PIGMENT GALLSTONES. *GASTROENTEROLOGY* , 72:167-182, 1977

- 47) Qiao T, Ma RH, Luo XB, Luo ZL, Zheng PM, Cholecystolithiasis is associated with *Clonorchis sinensis* infection, *PLoS One*. 2012; 7(8):e42471
- 48) Trowbridge RL, Rutkowski NK, Shojanian KG. Does this patient have acute cholecystitis? *JAMA*. 2003;289:80.
- 49) Fletcher DR. Gallstones. Modern management. *Aust Fam Physician*. 2001;30:441.
- 50) Basselink MGM, Erpecum KJ. Biliary Colic is a valuable clinical descriptor for Biliary pain due to “ uncomplicated” gallstone disease. *J Gastrointest Surg*. 2009;13(9):1745-6.
- 51) Berger MY, Hartman TC, Vander VJ, Bohnen A. Is biliary pain exclusively related to gall bladder stone ? A controlled prospective study. *Br J Gen Pract*. 2004;54(303): 574-9.
- 52) Hahn U, Mossner J. Planned cholecystectomy: preoperative endoscopy of the upper gastrointestinal tract. *Internist (Berl)* 1999;40(11):12-25.
- 53) Croce E, Azzola M, Golia M, Russo R, Pompa C. Laparocholecystectomy. 6,865 cases from Italian institutions. *Surg Endosc* 1994;8(9):1088–91.
- 54) Schwenk W, Bohm B, Badke A, Zarras K, Stock W. [Preoperative esophagogastroduodenoscopy before elective surgical therapy of symptomatic cholelithiasis]. *Leber Magen Darm* 1992;22(6):225–9.
- 55) Diettrich H, Wundrich B, Kobe E, Noack S, Weber K. Gastroscopy before cholecystectomy. *Gastroenterol J*. 1990;50[(4)]:173-4.

- 56) Rassek D, Osswald J, Stock W. Routine gastroscopy before cholecystectomy. *Chir-urg.* 1988 May;59[(5)]:335-7.
- 57) Thybusch A, Schaube H, Schweizer E, Gollnick D, Grimm H. [Significant value and therapeutic implications of routine gastroscopy before cholecystectomy]. *J Chir (Paris)* 1996; 133(4):171–4.
- 58) Sosada K, Zurawinski W, Piecuch J, Stepien T, Makarska J. Gastroduodenoscopy: a routine examination of 2,800 patients before laparoscopic cholecystectomy. *Surg Endosc* 2005;19(8):1103–8.
- 59) Venkatesh Kolla, Neelam Charles, Sanjay Datey, Devendra Mahor, Anand Gupta, Sanjeev Malhotra. Upper gastrointestinal endoscopy prior to laparoscopic cholecystectomy: a clinical study at a tertiary care centre in central India. *Int Surg J.* 2016 May;3(2):637-642
- 60) Karmacharya A, Malla BR, Joshi HN, Gurung RB, Rajbhandari M. The Predictive Value of Pre-Operative Symptoms Including Upper Gastrointestinal Endoscopy before Laparoscopic Cholecystectomy for Elective Symptomatic Cholecystolithiasis. *Kathmandu Univ Med J* 2013;44(4):300-304.
- 61) Saldinger PF. *Blumgart Surgery of the Liver, Biliary Tract and Pancreas.* 4th ed. Philadelphia: Saunders; 2006. Chapter 31, The Natural History of Gallstones and Asymptomatic Gallstones; p.477-478.
- 62) Berger MY, van der Velden JJ, Lijmer JG, de Kort H, Prins A, Bohnen AM. Abdominal symptoms do they predict gallstones? A systematic review. *Scand J Gastroenterol.* 2000; 70-6.

- 63) Kraagg N, Thijs C, Knipschild P. Dyspepsia - how noisy are gallstones? A meta-analysis of epidemiologic studies of biliary pain, dyspeptic symptoms, and food intolerance. *Scand J Gastroenterol.* 1995; 30:411-421.
- 64) Mohamed Toum Musa M Ibrahim, Randa Zaki, A M Khair et al. Upper gastrointestinal endoscopic findings in patients with gallstone disease, Khartoum Medical Journal (2012) vol.05.no.2.pg:749-752.
- 65) GREPCO. The epidemiology of gallstone disease in Rome, Italy. Part I Prevalence data in men. Part II The epidemiology of gallstone disease in Rome, Italy. *Hepatology* 1988; 8:904–13.
- 66) Healey J E Jr, Schroy P C. Anatomy of the biliary ducts within the human liver: analysis of the prevailing pattern of branchings and the major variations of the biliary ducts. *Arch Surg* 1953;66:599-600. [PubMed: 13039731]
- 67) Newman H F, Northrup J D. Extrahepatic biliary tract anatomy. *West J Surg Obstet Gynecol* 1963;71:59. [PubMed: 13938264]
- 68) MOHAMAD MOZAFAR, MOHAMADREZA SOB HIYEH and MOTAHARE HEIBATOLLAHI: Is esophago-gastroduodenoscopy essential prior to the elective surgical therapy of symptomatic cholelithiasis? *Gastroenterology and Hepatology From Bed to Bench*, 3 (2): 77- 82, 2010.
- 69) Faisal A, Gadallah AN, Omar SA, Nagy MAM The role of upper gastrointestinal endoscopy in prevention of post-cholecystectomy pain prior the elective surgical therapy of chronic cholecystitis. *Med J Cairo Univ.* 2013;81(1):289-93.

ANNEXURES I: CONSENT FORM

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Mr/Mrs/Miss. _____ we are requesting you to enroll yourself in study titled “ Role of pre operative upper gastro intestinal endoscopy in symptomatic patients undergoing elective laproscopic cholecystectomy for gall bladder stone disease” in KLES HOSPITAL & MRC,BELAGAVI conducted by Dr.***** **, Post Graduate in M.S. General Surgery under the guidance of Dr.** *****. Professor, Department of General Surgery, J.N. Medical College, Belagavi under KAHER, Belagavi.

Respected Sir/Madam, We request you to enroll yourself to participate in our study as you are eligible for participating in the study. During the study your clinical presentation, need for pre operative investigations and operative outcome will be accessed by some questions which will be answered by your operating surgeon.

Purpose of the study:

In this study we are assessing the role of **pre operative upper gastro intestinal endoscopy in patients undergoing elective laproscopic cholecystectomy for ultrasonographically proven gall bladder stone disease by determining upper gi lesions.**

Procedure Involved:

If you agree to enroll your-self in my study, I will ask your present & past history. You will undergo step wise physical examination and your routine blood investigation will be carried out, and an upper gi endoscopy will be performed 1-2 days prior to your elective laproscopic cholecystectomy.

Risks and Benefits:

Risk-There is no risk involved. Benefits- Any co existing upper gi pathologies which could be the true of of your symptoms will be identified and treated accordingly, thus reducing the incidence of post operative persistence of symptoms.

Alternatives:

Even if you decline the participation in the study, your operative outcomes will not be documented. Your participation in this research is voluntary. You may choose not to enrol yourself in this study. If you decide to withdraw from the study at any time, you will still receive the standard health care by the treating doctors. If you decide to participate you are free to withdraw at any time.

Privacy and Confidentiality:

The only people to know that you are a research subject are members of the research team. No information about you or information provided by you during the research will be disclosed to other without your written permission except:

1. In emergency to protect your rights and welfare.
2. If required by law.

Institutional/sponsors policy:

There is no possibility of any harm or injury during your participation in this study.

Authorization to Publish Results:

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with your identity remaining confidential.

Questions:

In case you have any questions related to the study, in future or in case of study related injury or illness, you can contact Dr.***** **, Department of General Surgery, KLES Hospital and MRC, Belagavi, phone number: *****. or Dr. ** *****, Dept Of General Surgery, KLES Hospital and MRC, Belagavi.

If you have any queries about your rights as a study subject, you may call Dr. ***** **, Professor, Department of Pathology and Chairman, J.N. Medical College Institutional Ethical Committee for Human Subjects Research, Phone number- ***** , or extension ***** at J.N. Medical College, Belagavi.

Consent for participation in prospective study

I, Mr/Ms/Mrs. _____ voluntarily agree for the participation as a subject of study. By signing this consent form I am not giving up any of my legal rights, I may withdraw from the study anytime. I am signing the consent form after having read or been read for me in vernacular language, including the risks and the benefits and having all my questions answered.

Subject Name :

Signature or the Left Thumb Print of Subject :

Date:

Witness Name: _____

Signature: _____

Date:

Investigators Name: _____

Signature: _____

Date :

Place : _____

ANNEXURES II: PROFORMA

PROFORMA

**ROLE OF PRE-OPERATIVE UPPER GASTRO-INTESTINAL ENDOSCOPY
IN PATIENTS UNDERGOING ELECTIVE LAPAROSCOPIC
CHOLECYSTECTOMY FOR ULTRASONOGRAPHICALLY PROVEN
GALL BLADDER STONE DISEASE:A ONE YEAR HOSPITAL BASED
CROSS-SECTIONAL STUDY**

Name & Address of the patient:

Age of the Patient: _____ IP. No. _____

Weight of Patient: _____ Sex. _____

Operating Surgeon: _____

HISTORY:

Chief Complaints:

Differentiation between typical pain and atypical pain:

Site of pain-

Duration of pain-

Association of pain with food intake-

Intermittent or continuous-

Association with nausea or vomiting-

Any radiation of pain-

Other atypical symptoms-Dyspepsia

Abdominal distension

Belching

Heart burn

Food intolerance

Flatulence

Loss of appetite

Past History:

- History of Diabetes Mellitus/Hypertension/Asthma/Tuberculosis
- Previous surgeries:
- Other co-morbidities:

Family History

General Physical Examination:

Pulse : B.P:

Temperature:

Systemic examination:

Cardiovascular System:

Respiratory System:

Central Nervous system:

Per abdomen:

Investigations:

Upper GI scopy findings:

Diagnosis:

Proposed Surgery: laproscopic cholecystectomy

Operative outcome –


Duration of surgery - _____ minutes.

Any intra operative complications:

Signature of operating surgeon:



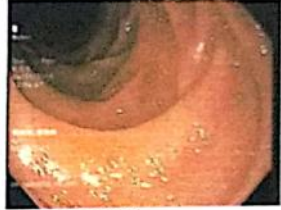
ANNEXURES III: PHOTOGRAPHS

UGI SCOPY DONE AT OUR HOSPITAL SHOWING PERI PYLORIC EROSIONS AND DIFFUSE GASTRIC EROSIONS.

 **KLES Dr.PRABHAKAR KORE HOSPITAL and MRC**
DEPARTMENT OF GASTROENTEROLOGY
Belagavi - 590010
Phone No: (0831)2473777, (1165)

Study: UPPER GI ENDOSCOPY

Sex: M

OESOPHAGUS STOMACH DUODENUM

GASTRO-DUODENOSCOPY REPORT


Oesophagus
Normal mucosa and lumen
No ulcer /stricture /growth /varices
O.g jt at 38 cms.

Stomach
E/O Diffuse erosions in the body and antrum
No ulcer /stricture /growth /varices
Fundal view-no varices /hiatus hernia

Duodenum part I
Normal
No ulcer /deformity/ duodenitis

Duodenum part II
Normal

Endoscopy impression
DIFFUSE GASTRIC EROSIONS


DR. SANTOSH HAJARE MD,DM,DNB (Gastro)

Study Doc: 91-00-25442912



KLES Dr.PRABHAKAR KORE HOSPITAL and MRC
DEPARTMENT OF GASTROENTEROLOGY
Belagavi - 590010
Phone No: (0831)2473777, (1165)

Sex: M

Study: UPPER GI ENDOSCOPY



OESOPHAGUS



STOMACH



DUODENUM

GASTRO-DUODENOSCOPY REPORT

Oesophagus

E/O LA Grade B Oesophagitis
No ulcer /stricture /growth /varices
O.g.jt at 38 cms.

Stomach

E/O Peri-Pyloric erosions
No ulcer /stricture /growth /varices
Fundal view-no varices /hiatus hernia

Duodenum part I

Normal
No ulcer /deformity/ duodenitis

Duodenum part II

Normal

Endoscopy impression

LA GRADE B OESOPHAGITIS
PERI-PYLORIC EROSIONS

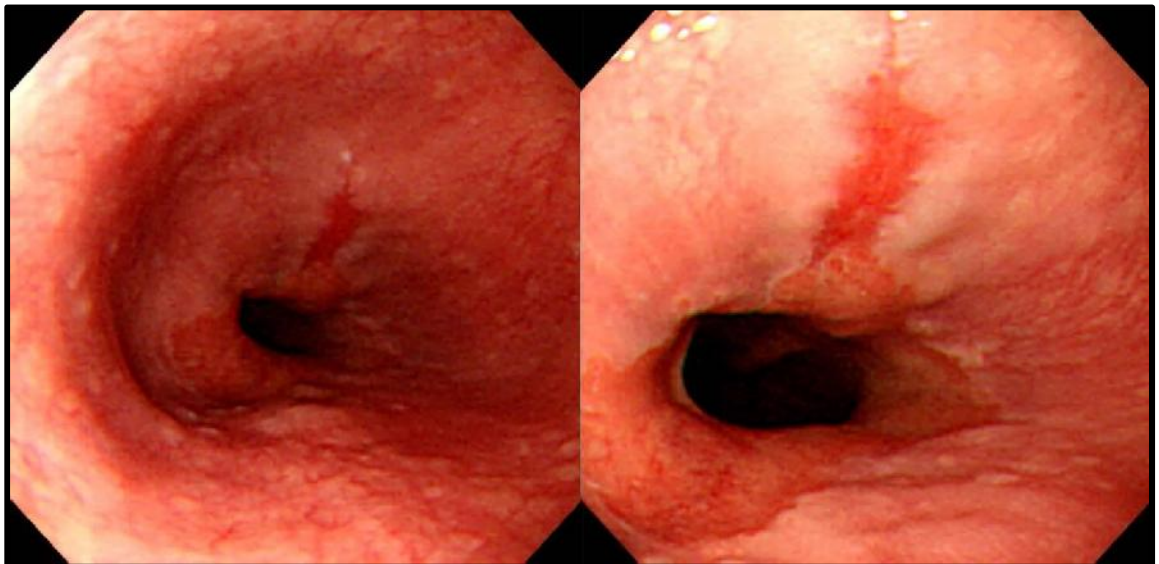
DR. SANTOSH HAJARE MD,DM,DNB (Gastro)

ScopyDoc 91-20-2

OTHER UGI FINDINGS:



GASTRIC EROSIONS



REFLUX ESOPHAGITIS

KEY TO MASTERCHART

Sex-

- Male- M
- Female- F

Gallbladder symptoms

- Typical biliary colic-1
- Atypical biliary colic-2

UGI Endoscopy findings-

- UGI Endoscopy normal - 0
- UGI Endoscopy positive - 1

