"A ONE YEAR CROSS-SECTIONAL STUDY TO EVALUATE THE RELATIONSHIP BETWEEN BODY MASS INDEX WITH SEVERITY OF CHOLECYSTITIS" IN KLES DR. PRABHAKAR KORE HOSPITALS BELGAUM

By

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Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

M. S.
in
GENERAL SURGERY

Under the Guidance of

Dr. M. S. SANGOLLI MS
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BELGAUM, KARNATAKA

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KLE UNIVERSITY, BELGAUM, KARNATAKA

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LIST OF ABBREVIATIONS USED

ACR	-	American College of Radiology
AIDS	-	Acquired immunodeficiency syndrome
ALT	-	Alanine transaminase
AST	-	Aspartate amino transferase
BMI	-	Body mass index
CT	-	Computed tomography
ERCP	-	Endoscopic retrograde cholangiopancreatiography
HBS	-	Hepatobiliary scintigraphy
HPR	-	Histopathology report
ICU	-	Intensive care unit
MRI	-	Magnetic resonance imaging
PTC	-	Percutaneous transhepatic cholangiography
RUQ	-	Right upper quadrant
TPN	-	Total parental nutrition

ABSTRACT

Background and Objectives

More than half a million cholecystectomies are performed per year in the United States. Risk factors for cholecystitis mirror those for cholelithiasis and include increasing age, female sex, certain ethnic groups, obesity or rapid weight loss, drugs, and pregnancy. Present study was undertaken to evaluate relationship between BMI with severity of cholecystitis based on clinical, imaging, intraoperative and HPR findings.

Methodology

This one year cross sectional study was conducted on 100 patients admitted with cholecystitis in the wards of Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2010 to December 2010. Based on the BMI, patients were divided into groups, non obese (BMI between 18 to 25 kg/m^2) and obese (>25 kg/m^2) group. The severity of disease was categorized as chronic cholecystitis and acute cholecystitis based on the clinical examination, blood investigations, ultrasonography, and histopathology. Acute cholecystitis was categorized into three categories based on Tokyo guidelines.

Results

In this study, females outnumbered males with male to female ratio of 1:1.5. Most of patients (68.3%) were aged between 30 years to 60 years and mean age was 49.31±13.57 years. Of 120 patients 62.5% patients were non-obese and 37.5% patients were obese. Mean BMI was 24.35±2.96 Kg/m^2. 70.83% patients

had chronic and 29.17% patients had acute cholecystitis. Out of 35 patients 31.4% had grade I, 62.8% grade II and 5.71% grade III severity. Of the 11, 22 and 2 patients with grade I, II and III severity 63.7% were obese in grade I and in grade II 63.6% were non obese. Male gender influenced severity of cholecystitis significantly.

Conclusion and interpretation

Overall there was no influence of BMI over severity of cholecytitis based on clinical, imaging, intraoperative and HPR findings as well as either in males or females separately.

Keywords

Body mass index; Cholecystitis; Obesity; Severity of cholecystitis;

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INTRODUCTION

Cholecystitis is defined as inflammation of the gallbladder that occurs most commonly because of an obstruction of the cystic duct from cholelithiasis. Ninety percent of cases involve stones in the cystic duct (that is, calculous cholecystitis), with the other 10% of cases representing acalculous cholecystitis.[1]

About 10% to 15% of the adult western population have gallstones. Between 1% and 4% become symptomatic in a year.[1] More than half a million cholecystectomies are performed per year in the United States alone. Regional differences exist in the cholecystectomy rates.[2]

Risk factors for cholecystitis mirror those for cholelithiasis and include increasing age, female sex, certain ethnic groups, obesity or rapid weight loss, drugs, and pregnancy.[3]

Body Mass Index is a scale to measure obesity. Increase in BMI is known to increase the prevalence of gallstone disease. On the contrary it is was observed that severity of cholecystitis was more in patients with lower BMI.[4]

Many studies have been done on risk factors for developing gall bladder disease explaining its polygenic nature.[3,5] But only one study is available on body mass index and its effect on severity cholecystitis.[4] No such study has been done in Indian setup hence depicting the need for the study.

An acute reduction of body weight also predisposes a person to cholelithiasis. But the cause of the high incidence of cholelithiasis in person who

have undergone rapid weight loss has still not been clearly elucidated. Hence body weight may influence pathogenesis of gallstones in different ways.[6]

On the other hand, male sex has recently been cited as a risk factor for severe symptomatic cholelithiasis. It has also been reported that male sex is an independent predictor for more severe acute cholecystitis. However the cause of severity of cholecystitis in males has not been revealed.[7,8]

Mean total body fat which is higher in females than males can be a possible cause for this sex difference.

A study reported increasing risk of gallstones or gall-stone disease with increasing body mass index (BMI) in women whereas this relation was much less evident in men.[9]

Hence the present study was undertaken to evaluate the relationship between body mass index with severity of cholecystitis based on clinical, imaging, intraoperative and HPR findings in order to help for better anticipation of complications during surgery and the need for conversion from laparoscopy to open cholecystectomy and in counselling the patient and relatives prior to surgery.

OBJECTIVES

Objective of the present study was to evaluate the relationship between body mass index with severity of cholecystitis based on clinical, imaging, intraoperative and HPR findings.

REVIEW OF LITERATURE

HISTORICAL ASPECTS

Archeological excavations demonstrating the presence of gall stones in young Egyptian women have confirmed that cholelithiasis has plagued mankind over 2000 years.[10]

Gall stones were described before the modern era of cholecystectomy by Langen buch in the late 19th century. He widened the understanding of gall stone pathology and performed the first successful cholecyctectomy. Numerous calculi were found in the gall bladder of the mummy of a priestess of Amenen of the 21st Egyptian dynasty [1500 B.C.]. Gallstones were first described by the Greek physician Alexander Trallianus who wrote about the calculi within the bile ducts.[7] By the 16th century, both Vesailius and Fallopius described gallstones found in the gall bladder of the dissected human bodies.[11]

Laparoscopic cholecystectomy, which was introduced in 1987, is now the preferred method of cholecystectomy.[12]

First elective cholecystectomy was done by Bobbs in 1867, First successful cholecystectomy was done by Karl Langen Buch on july 15th 1882 in Berlin on a male patient suffering from biliary colic for years, First cholecystojejunostomy for CBD obstruction by Von Winiwarter in 1882, First successful choledochotomy by Courvoisier in 1882. First successful choledochojejunostomy by Sprengel in 1891,[11] First hepaticoduodenostomy by W. J. Mayo in 1905.[12]

In 1873 Maurice Schiff proposed the ingestion of bile salts as a treatment for gall stones. 50 years later the first report of successful oral gall stones dissolution was reported by New Bridge from the university of Minesuta,[13] First time accurate diagnosis of gall bladder disease was demonstrated by Graham and Cole by oral cholecystography, First PTC is by Huard and Doxun in 1937.[11]

The endoscopic retrograde cholangiopancreatography was first performed by Mecune. In 1937, The concept of gall stones dissolution by administering bile salts was recognized by AG Rewbridge in 1937 and this was confirmed 20 years later by Johnston and Nakayama in 1957, Mirizzi introduced the operative cholangiography in 1937 in Argentina.[11]

Extra corporeal shock wave lithotripsy of gall stones was developed in the 1980 as a non invasive form of treatment in selected patients with symptomatic, uncomplicated cholelithiasis.[11]

The surgical techniques started to evolve in the late 1800, John Bobbs an Indiana surgeon and others attempted to perform cholecystolithotomy, removing the stone from the gall bladder and leaving the organ in situ, First percutaneous cholecystolithotomy by Akiyama et al in 1985, Kerlan et al in 1985.[14]

Cope et al in 1990 removed the smaller calculi by wire baskets, fragmentation of larger calculi may be done with electro hydroaulic or laser mediated intracorporal lithotripsy by Burhenne et al in 1975, Pinacus et al in 1989. Combined surgical and radiological intervention [mini cholecystotomy] was described by Burhenne et al in 1985.[14]

In 1985 first laparoscopically assisted cholecystectomy was performed by Muhe, Boblingen, Germany. In 1987, french surgeon in Lyon, Phillipe Mouret, performed the first video laparoscopic cholecystectomy.[14]

Cadiere and colleagues reported the first successful clinical implementation of telerobotics in 1998 when they accomplished a laparoscopic cholecystectomy using a prototype of the Da Vinci robotic surgical system.[14]

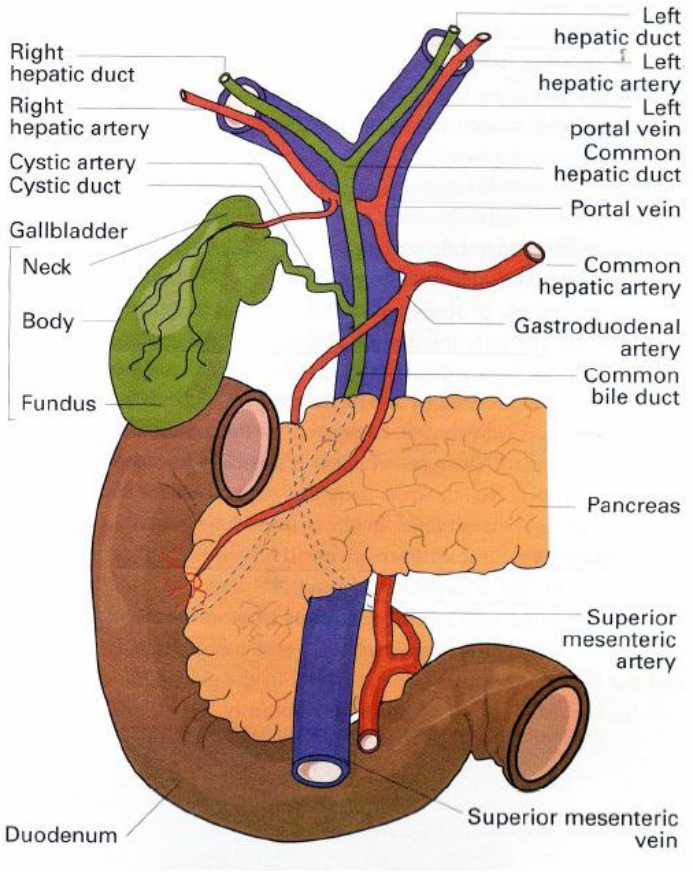
Surgical Anatomy

The gallbladder is a slate blue, piriform sac partly sunk in a fossa in the right hepatic lobe's inferior surface. It extends forward from a point near the right end of the porta hepatic to the inferior hepatic border.[15] Its upper surface is attached to the liver by the connective tissue, elsewhere it is completely covered by the peritoneum and even connected to the liver by a short mesentry.

It is 7 to 10 cm long, 3 cm broad at its widest and 30-50ml in capacity. It is described as having a fundus, body and neck.

The fundus, the expanded end projects down, forwards and to the right, extending beyond the inferior border to contact the anterior abdominal wall behind the ninth right costal cartilage, where the lateral edge of the right rectus abdominus muscle crosses the costal margin. Posteriorly it is related to transverse colon, near its commencement. The body is directed back and to the left, near the right end of the porta it is continuous with neck. It is related above to the liver, below to the transverse colon and further back to the first and upper end of second segment of duodenum.

The neck is narrow, curving up and forwards and then abruptly backwards and downwards, to become the cystic duct,at which transition there is a constriction. The neck is attached to the liver by the areolar tissue containing the cystic artery. The mucosa of the neck is obliquely rigid forming a spiral valve, when the neck is distended this gives its surface a spiral grove.



Surgical anatomy of biliary tract

From the right side of the neck a small recess may project down and back towards the duodenum, often termed Hartmann's pouch (but originally described by Broca). It has been regarded as the constant feature but Davis and Harding (1942) have shown that it is always a sequelae of pathological states especially when dilated.

The cystic duct is 3 to 4 cm long, it passes back, down and to the left from the neck of the gall bladder, joining the common hepatic duct to form the bile duct. It is adherent to the common hepatic duct for a short distance before joining it. Usually near the porta hepatis but sometimes lower, in which case the cystic duct lies along the lesser omentum's right edge. Its mucosa bears 5-10 concentric folds, they project obliquely in regular succession, like a spiral valve, which some times referred as the "valve of Heister".[15,16,17,18]

Arterial Supply of the Gallbladder:[15]

The major blood supply of the gall bladder is through the cystic artery, which is typically a branch of right hepatic artery. The gall bladder also receives many small vessels from its hepatic bed. The cystic artery usually passes behind the common hepatic and cystic ducts to the upper surface of the neck of the gall bladder, on which it runs downward forwards before dividing into superficial and deep branches. The former ramifies on the serosal surface and later on the hepatic surface of the gallbladder. The two branches must be secured during cholecystectomy. The cystic artery supplies branches to the hepatic ducts and to the upper part of the common bile duct. The lower part of the bile duct receives several branches from the posterior superior pancreatico-duodenal artery. The right hepatic artery gives branches to the middle part of the bile duct.

Variations in the artery's origin are of surgical interest. In 800 specimens Anson (1963) observed the following incidences, origin from the right hepatic artery 63.9%, the hepatic trunk 26.9%, left hepatic 5.5% gastroduodenl 2.6%,superior pancreaticoduodenal 0.3%, right gastric 0.1%, coelic trunk 0.3%,

and superior mesenteric 0.8%. An accessory cystic artery may arise from the common hepatic or one of its branches, the cystic artery supplies the the hepatic ducts and upper part of common bile duct. The cystic artery is an end artery and its occlusion is followed by the gangrene of the gall bladder.[19]

Venous Drainage of the Gallbladder:[15]

The veins draining the gall bladder vary considerably. Those from its upper surface lies in the areolar tissue between the gallbladder and liver and usually run directly into the liver through the fossa of the gallbladder to join the hepatic veins. Those from the rest of the gall bladder join to form one or more cystic veins on its neck, and these commonly enter liver, either directly or after joining with the veins draining the hepatic ducts and upper part of the bile duct. Only rarely does a single or double cystic vein drain directly in to right branch of the portal vein. They do not accompany the cystic artery.

Lymphatic Drainage[15]

The lymphatics draining the gall bladder tend to be of considerable importance for both inflammatory and malignant disease of gall bladder. The lymphatic channels from the subserosal and sub mucosal plexus drain into cystic lymph node of Lund, the sentinel lymph node, which lies in the fork created by the junction of cystic and common hepatic ducts and to a node situated at the anterior border of epiploic foramen. Efferent vessels from the nodes pass in the free edge of the lesser omentum to the celiac group of preaortic nodes.

The sentinel node can be of considerable size and may distort the normal

anatomy in patients with acute cholecystitis or carcinoma. The subserosal lymphatic vessels of the gall bladder also have connection with the subcapsular lymphatic channels of liver, and accounts for the frequent spread of carcinoma of gallbladder to the liver.

Nerve Supply[15]

The wall of the gall bladder is richly innervated with both sympathetic and parasympathetic nerve fibers, which pass along the hepatic artery and its branches. Parasympathetic fibers, mainly from the hepatic branch of anterior vagal trunk, stimulate contraction of the gall bladder and relax the ampullary sphincter. Sympathetic fibers from the cell bodies in the celiac ganglia, with the preganglionic cells in the lateral horn of the spinal cord segments, T7-T9 inhibits contraction. Autonomic plexus of the nerve exists in the muscular and sub mucous layers. Fibers from the right phrenic nerve, through communication between phrenic and celiac plexus, appear to reach the gallbladder via hepatic plexus explaining referred "shoulder pain" in the gall bladder pathology. The biliary tract pain is usually felt in the right hypochondrium and epigastrium and may radiate round to the back in the infrascapular region, in the area of distribution of spinal nerve T7-T9.

Triangle of cholecystectomy

Calots defined a triangle of anatomical area formed by the common hepatic duct medially. The cystic duct laterally and the cystic artery superiorly in 1891. The present concept is of the triangle of cholecystectomy has for its upper limit not the cystic artery but the inferior surface of the liver[20]. This triangle is of

surgical importance because a number of important structures pass through it. Therefore during cholecystectomy it is a need to identify all structure within the triangle to prevent complications.[21]

Common anomalies and variations

1. Absent gall bladder – extremely rare,autopsy incidence of 0.03% have been reported.[15]

2. Variation in size and shape of gall bladder.
    a. Bilobed gall bladder.
    b. Fundule divertculum.
    c. Phrygian cap.
    d. Hour glass gall bladder.

3. Variation in position left sided gall bladder, floating gall bladder.

4. Double gall bladder, duplication of gall bladder with two separate cavities and two separate cystic ducts has an incidence of approximately 1 in 4000. Pathological process such as cholelithiasis and cholecystitis may involve one organ while the other is spared.[22]

5. Other anamalies related to gall bladder
    a. Persistence intrahepatic gall bladder
    b. Diverticulum's of body or neck of gall bladder
    c. Accessory peritoneal fold due to congenital adhesions.

Floating gall bladder occurs when there is increase in the peritoneal investment; this condition occurs in the 5% of patients and predisposes to torsion, resulting in gangrene or perforation of the viscus.[15]

Cystic duct may be absent rarely. Two are more cystic ducts may combine, the junction of the cystic duct and common hepatic ducts may vary in its level from the porta hepatis to behind or even below the duodenum's first part, when the junction is low the two ducts may be connected by fibrous tissue. Accessory hepatic ducts may emerge, more often from the right lobe to join the main hepatic ducts rarely, the gall bladder itself.[18]

Physiology of gall bladder[17,18,23]

The primary function of gall bladder is to concentrate bile by absorption of water and sodium to acquire greater strength and digestive power. The gallbladder and bile ducts are well adopted for the function of storing and discharging bile into the duodenum during digestion. The storage in small bulk is made possible by the concentrating power of the gallbladder. The flow of bile in and out of the gallbladder is determined primarily by contraction and relaxation of the sphincter of Oddi.

The healthy gallbladder is rarely static. Continuous cycle of partial emptying and refilling is governed by the intestinal migratory myoelectric complex between meals. During relaxation and refilling it intermittently contracts and expels pulses of bile into the duodenum. This constant fluctuation prevents stone formation.

Gallbladder tone is modulated by both vagus and circulating peptides. During cephalic phase of digestion vagal stimulation is responsible for gallbladder contraction. During interdigestive period it is vagal neurons and circulating polypeptides which mediate the contraction. Vasoactive intestinal polypeptide [VIP] released by vagal neurons inhibits gallbladder contraction and mediates post prandial gallbladder filling. Gallbladder motility is inhibited by truncal vagotomy and by chronic fasting.

The gallbladder exhibits;

1. Tonic contractions which may last for 5 to 10 mins and elevates intravesical pressure to 30mm of water.

2. Rhythmic contractions [2 to 6 mins] during which pressure do not exceed 30mm of water. The maximal expulsive pressure of gallbladder is therefore less than secretory pressure of liver and explains why filling and evacuation of the gallbladder is dependent upon reciprocal contraction and relaxation of sphincter of Oddi.

Biochemistry of bile

Bile as it leaves the liver is composed of 97% of water, 1 to 2% of bile salts and 1% of bile pigments, cholesterol and fatty acids. The knowledge of the chemistry of the constituents of bile is essential as they have a great bearing in the etiology of cholelithiasis.

Bile acids and bile salts:

The bile acids of the human bile are glycocholic and taurocholic acids which are conjugated products of amino acids glycine and cystine with cholic acid respectively. Bile acids are present in bile as bile salts viz. Sodium glycocholate and sodium taurocholate. Human bile consists approximately 70 to 75% glycocholate and 25% of taurocholate.

Bile pigments

The biliary pigments are bilirubin and biliverdin. Bilirubin is the chief pigment in the human bile. Biliverdin is the exudative product of bilirubin and is present in small quantity in human bile. The pigment forms about 15 to 20% of total solids in liver bile. They are derived mainly from hemoglobin and a small amount from chromoprotiens.

Lipids

The normal bile contains cholesterol, fatty acids and phospholipids. Cholesterol normally occurs to the extent of 0.04 to 0.16% in liver bile. It is present in the free state and its concentration is more in gall bladder bile. Normally the ratio between the cholesterol and bile salts varies between 1:20 to 1:30.

Mucin

Its main constituent is mucealbumin. It increases in obstructive and inflammatory conditions of the biliary tract and it forms the cementing substance

in gall stones. The functions of bile are brought about by the bile acids and are digestion and absorption of fats and fat soluble vitamins, mild laxative effects on the intestine and also an antiputrefactive effect by their bacteriostatic property on intestinal flora and finally bile salts are the best cholerectics.

Entero Hepatic Circulation

Following normal fatty meals, emulsification of the cleavage products from triglyceride hydrolysis by pancreatic lipase results in the incorporation of fat into micelles. Absorption of fat takes primarily in the upper intestine, whereas bile acids undergo little absorption until the lower third of the small intestine is reached. In the ileum there exists specific high affinity binding sites for the active absorption of bile acids. Due to the efficiency of this absorptive process, less than 5% of the daily excreted bile reaches the colon. Upon absorption, bile acids enter the portal vein and return to liver. The efficiency of the hepatic removal of bile acids account for the extremely low peripheral blood levels normally found. This 95% return rate of bile acids to the liver has two consequences. First, most of the bile acids excreted in bile are actively recycled rather than newly synthesized. Second bile acids exert a feed back inhibition that regulates estrogens, cholesterol, and fat soluable vitamins.

Functions of the gallbladder

Reservoir of bile

During the intercibal period the sphincter of Oddi is closed and the bile excreted by the liver is directed to the gallbladder. After food, the resistance to

flow through the sphincter of Oddi is reduced, the gallbladder contracts and bile enters the duodenum. Narcotic drugs especially morphine and pethedine increases sphincter tone whereas anticholenergic and glyceryl trinitrate decrease the tone.

Concentration of bile:

By active absorption of sodium, water, chloride and bicarbonate by the mucous membrane of the gall bladder into the bloodstream and to a lesser extent into the lymphatics, the hepatic bile which enters the gallbladder becomes concentrated 5 to 10 times with a corresponding increase in the proportion of bile salts, bile pigments, cholesterol and calcium it contains. The absorptive power of the gallbladder is much varied in disease. In disease, the gallbladder instead of absorbing fluids pours fluids rapidly. The absorption of the bile salts is enhanced while more of calcium and cholesterol is seen in the lumen. Here lies the relationship between inflammation of the gall bladder and the stone formation. It is said that the desquamated epithelium forms the nucleus of the stone, the increased amount of calcium and cholesterol forms the raw material, while the increased absorption of bile salts results in the precipitation of cholesterol.

Secretion of the mucus

The gallbladder secretes 20ml of thick viscid mucus every 24 hours. Allegedly this protects the mucosa from the lytic action of the bile and facilitates the passage of thick bile through the cystic duct. The colorless fluid found in the hydrops of the gallbladder and the so called white bile found in choledochal obstruction, severe cholangitis or toxic hepatitis is not bile at all but is a mucinous secretion containing calcium carbonate with no bile salts or bile

pigments. The gall bladder also secretes calcium, especially in the presence of inflammation or cystic duct obstruction. This may result in calcium shells around preformed stones in calcification of the gall bladder wall or in the presence of the white calcium sludge.

Excretion of cholesterol

The presence of cholesterol esters in the connective tissue of the gallbladder in the pathological entity of strawberry gallbladder or cholesteroses of gallbladder led some physiologists to ascribe the function of excretion of cholesterol by gallbladder. Others say that diffuse collection of cholesterol in the wall of gallbladder is an evidence of abnormal absorption of lipid. Thus there has been much difference of opinion as to whether cholesterol is excreted or absorbed.

Pressure regulation

Gallbladder equalizes pressure within the biliary tract by virtue of its power of absorption of bile. This is brought about by the fact that the amount of bile secreted in 24hours is about 20 times or so greater than could be contained in the gallbladder. The less of its action in equalizing the pressure within the duct system is probably leading to the dilatation of the bile ducts, which so frequently follows cholecystectomy.

Change in the reaction of bile

The bile secreted by the liver is distinctly alkaline with a pH of 7.1 to 8.5 while bile that reaches the duodenum is almost neutral with a pH of 5.5 to7.7. This is due to bicarbonate being reabsorbed by gallbladder.

Epidemiology

Cholecystitis is defined as inflammation of the gallbladder that occurs most commonly because of an obstruction of the cystic duct from cholelithiasis. Ninety percent of cases involve stones in the cystic duct (ie, calculous cholecystitis), with the other 10% of cases representing acalculous cholecystitis.[1]

Risk factors for cholecystitis mirror those for cholelithiasis and include increasing age, female sex, certain ethnic groups, obesity or rapid weight loss, drugs, and pregnancy. Although bile cultures are positive for bacteria in 50-75% of cases, bacterial proliferation may be a result of cholecystitis and not the precipitating factor.[3]

Acalculous cholecystitis is related to conditions associated with biliary stasis, including debilitation, major surgery, severe trauma, sepsis, long-term total parenteral nutrition (TPN), and prolonged fasting. Other causes of acalculous cholecystitis include cardiac events; sickle cell disease; *Salmonella* infections; diabetes mellitus; and cytomegalovirus, cryptosporidiosis, or microsporidiosis infections in patients with AIDS.[24]

Uncomplicated cholecystitis has an excellent prognosis, with a very low mortality rate. Once complications such as perforation/gangrene develop, the

prognosis becomes less favorable. Some 25-30% of patients either require surgery or develop some complication.[17]

The most common presenting symptom of acute cholecystitis is upper abdominal pain. The physical examination may reveal fever, tachycardia, and tenderness in the RUQ or epigastric region, often with guarding or rebound tenderness. However, the absence of physical findings does not rule out the diagnosis of cholecystitis.[11]

Delays in making the diagnosis of acute cholecystitis result in a higher incidence of morbidity and mortality. This is especially true for ICU patients who develop acalculous cholecystitis. The diagnosis should be considered and investigated promptly in order to prevent poor outcomes.[11]

Initial treatment of acute cholecystitis includes bowel rest, intravenous hydration, correction of electrolyte abnormalities, analgesia, and intravenous antibiotics. For mild cases of acute cholecystitis, antibiotic therapy with a single broad-spectrum antibiotic is adequate. Outpatient treatment may be appropriate for cases of uncomplicated cholecystitis. If surgical treatment is indicated, laparoscopic cholecystectomy represents the standard of care.[18]

An estimated 10-20% of Americans have gallstones, and as many as one third of these people develop acute cholecystitis. Cholecystectomy for either recurrent biliary colic or acute cholecystitis is the most common major surgical procedure performed by general surgeons, resulting in approximately 500,000 operations annually.[25]

Age distribution for cholecystitis

The incidence of cholecystitis increases with age. The physiologic explanation for the increasing incidence of gallstone disease in the elderly population is unclear. The increased incidence in elderly men has been linked to changing androgen-to-estrogen ratios.

At least 10 percent of adults have gallstones. The prevalence varies with age, sex, and ethnic group. There is an increasing prevalence with age, after the age of 60 about 10 to 15% of men and 20 to 40% of women have gallstones. In a recent ultrasound survey in Denmark, a large population was reexamined at five-year intervals. In each five-year period, new gallstones formed in about three percent of the population over the age of 40.[26] There is a incidence of 25% of children with gallstones have hemolytic disease other possible predisposing factors are cystic fibrosis, liver disease, bowel resection and heart disease.[11] The overall prevalence of gallstone disease in industrialized countries appears to be between 10% to 20%.[11]

Sex distribution for cholecystitis

Gallstones are two to three times more frequent in females than in males, resulting in a higher incidence of calculous cholecystitis in females. Elevated progesterone levels during pregnancy may cause biliary stasis, resulting in higher rates of gallbladder disease in pregnant females. Acalculous cholecystitis is observed more often in elderly men.[3]

Ultrasound surveys[27] show a female:male ratio of about 2:1 in the younger age groups and The risk of gallstones is also associated with a history of childbearing, estrogen-replacement therapy, and oral-contraceptive use, but not diabetes mellitus.[28]

Prevalence of cholecystitis by race and ethnicity

Cholelithiasis the major risk factor for cholecystitis, has an increased prevalence among people of Scandinavian descent, Pima Indians, and Hispanic populations, whereas cholelithiasis is less common among individuals from sub-Saharan Africa and Asia.In the United States, white people have a higher prevalence than black people.[29,30]

The prevalence of gallstones is especially high in the Scandinavian countries and Chile. North Indians have seven times higher occurrence of gall stone as compared with south Indians and among Native Americans Mexican Americans and American Indians, especially the Pima tribe, have an increased predisposition to gallstone formation.[29]

The prevalence of gallbladder stone varies widely in different parts of the world. In India it is estimated to be around 4%. An epidemiological study restricted to rail road workers showed that north Indians have 7 times higher occurance of gall stone as compared with south Indians.[31]

There has been a marked increase in the incidence of the gall stone in the west during the past century.[32] In the united states the autopsy series have shown gall stones in atleast 20% of women and 8% of men over the age of 40 years.[33]

It is estimated that atleast 20 million persons in the United States have gall stones and that approximately 1 million new cases of cholelithiasis develop each year. Prevalence in Europe is 18.5% from the autopsy studies with the lowest prevalence from Ireland [5%] and the highest from Sweden[38%]. In Australia the prevalence rate varies from 15% to 25%. Highest prevalence in pima Indian tribe of Arizona with total and female prevalence of 49% and 73% respectively. Gall stones are rare in Africa with prevalence of less than 1% and in Japan it has been increased from 2% to 7%.[32,34]

Changing incidence in India is mainly attributed to westernization and availability of investigation that is ultrasound to urban as well as rural area and also because of increase affordability due to change in the socio-economic structure and the cost of investigations.

Etiology

Risk factors for calculous cholecystitis mirror those for cholelithiasis and include the following:[9]

- Female sex
- Certain ethnic groups
- Obesity or rapid weight loss
- Drugs (especially hormonal therapy in women)
- Pregnancy
- Increasing age

Acalculous cholecystitis is related to conditions associated with biliary stasis, to include the following:[24]

- Critical illness
- Major surgery or severe trauma/burns
- Sepsis
- Long-term total parenteral nutrition (TPN)
- Prolonged fasting

Other causes of acalculous cholecystitis include the following:

- Cardiac events, including myocardial infarction
- Sickle cell disease
- *Salmonella* infections
- Diabetes mellitus
- Patients with AIDS who have cytomegalovirus, cryptosporidiosis, or microsporidiosis

Patients who are immunocompromised are at increased risk of developing cholecystitis from a number of different infectious sources. Idiopathic cases exist.

Risk factors

Obesity

It is higher in markedly obese persons and in those who lose weight rapidly. There is little agreement about the effect of dietary components on the risk of gallstones. Fasting is normally associated with an increased biliary cholesterol saturation and this phenomenon persists or even become more

accentuated in obesity. A large clinical study showed that being even moderately overweight increases the risk for developing gallstones. Obesity also reduces gallbladder emptying.[3]

Body Mass Index

BMI, previously known as Quetelet index, was proposed by Lambert Adolphe Jacques Quetelet (1796-1874), a social scientist from Belgium, well remembered for his probability theory of social phenomena. He proposed this index in an attempt to describe the relationship between body weights in proportion to height in humans to determine the best body weight for the height. Subsequently, BMI was employed as a popular measure of nutritional status or indicator of health status/physical standards around the globe to recruit people for national services. However, at a later stage it was suggested by Garrow and Webster and a few others that BMI could be used as an indicator of body fat content. Thereafter, BMI has become one of the most common parameters often used in nutritional, metabolic and cardiovascular studies.[35]

Obesity represents a state of excess storage of body fat. Although similar, the term overweight is puristically defined as an excess body weight for height. Although men have a body fat percentage of 15-20%, women have approximately 25-30%.[35] Because differences in weight among individuals are only partly due to variations in body fat, body weight is a limited, though easily obtained index of obesity.

Although the BMI is typically closely correlated with percentage body fat in a curvilinear fashion, some important caveats to its interpretation apply. In

mesomorphic (muscular) persons, BMIs that usually indicate overweight or mild obesity may be spurious, whereas in some persons with sarcopenia (especially among persons of Asian descent), a typically normal BMI may conceal underlying excess adiposity characterized by increased percentage fat mass and reduced muscle mass.[35]

In view of these limitations, some authorities advocate a definition of obesity based on percentage body fat. For men, percentage body fat greater than 25% defines obesity, and 21-25% is borderline. For women, over 33% defines obesity, and 31-33% is borderline.[35]

Other indices used to estimate the degree and distribution of obesity include the 4 standard skin thicknesses (subscapular, triceps, biceps, suprailiac) and various anthropometric measures, of which waist and hip circumferences are the most important.

Although several classifications and definitions for degrees of obesity are accepted, the most widely accepted is the World Health Organization (WHO) criteria based on BMI.[36] Under this convention for adults, grade 1 overweight (commonly and simply called overweight) is a BMI of 25-29.9 kg/m^2. Grade 2 overweight (commonly called obesity) is a BMI of 30-39.9 kg/m^2. Grade 3 overweight (commonly called severe or morbid obesity) is a BMI greater than or equal to 40 kg/m^2.

Body mass index was classified according to Overweight and obesity by BMI in adult Asians as below.[37]

Classification	BMI (Kg/m^2)	Risk of co-morbidities
Underweight	< 18.5	Low (But increased risk of other clinical problems
Normal range	18.5 to 22.9	Average
Overweight	≥ 23	
At risk	23.0 to 24.9	Increased
Obese I	25.0 to 29.9	Moderate
Obese II	≥ 30.0	Severe

The surgical literature often uses a different classification to recognize particularly severe obesity. In this setting, a BMI greater than 40 kg/m^2 is described as severe obesity, a BMI of 40-50 kg/m^2 is termed morbid obesity, and a BMI greater than 50 kg/m^2 is termed super obese.

Gall bladder disease is higher in markedly obese persons and in those who lose weight rapidly. There is little agreement about the effect of dietary components on the risk of gallstones. Fasting is normally associated with increased biliary cholesterol saturation and this phenomenon persists or even become more accentuated in obesity. Being even moderately overweight increases the risk for developing gallstones. Obesity also reduces gallbladder emptying.

Estrogen and cholesterol lowering agents

Excess estrogen from pregnancy, hormone replacement therapy, or birth control pills appears to increase cholesterol levels in bile and decrease gallbladder

movement, both of which can lead to gallstones. Drugs that lower cholesterol levels in the blood actually increase the amount of cholesterol secreted in bile. This in turn can increase the risk of cholesterol gallstones. Clofibrate increases biliaric cholesterol and results in formation of the gall stone. Patients who are taking clofibrates are at an increase risk for cholecystectomy. As the body metabolizes fat during rapid weight loss, it causes the liver to secrete extra cholesterol into the bile, which can cause gallstones.[11]

Diabetis mellitus

Gallbladder atony consequent upon an autonomic neuropathy may favour stone formation in super saturated bile.[11] It has been stated that the diabetes patients have higher incidence of gallstone disease and are particularly prone to complications from there stone. However studies have shown that these patients do not have an increased morbidity or mortality from the stone disease. Once other comorbidities such as cardiovascular disease and renal insufficiency are taken into account. There is an increased incidence of complications.[38]

Fasting

Fasting decreases gallbladder movement causing the bile to become overconcentrated with cholesterol, which can lead to gallstones. No clear relationship has been proved between diet and gallstone formation. However, low-fiber, high-cholesterol diets, and diets high in starchy foods may also contribute to gallstone formation. Wayne et al have concluded from there study that a dietary soluble fibre psyllium inhibits cholesterol stone formation by

reducing the biliary cholesterol saturation index. Gallstones are more frequent in type 4 hyperlipidaemia.[6]

Cirrhosis of the liver

Patients with cirrhosis have 3 times greater risk for gallstones than the normal people. The stones are usually of pigment type and probably results from the chronic haemolysis. Cholecystectomy when performed in cirrhotic patients is associated with increased morbidity and mortality.[10]

Vagatomy

Early clinical study suggested that truncal vagatomy was associated with two fold increase in the incidence of gallstones, other studies have failed to confirmed this hypothesis. While ultrastonography suggested that truncal vagatomy is associated with dialated gallbladder. Nerve fibres from both vagal nerves merge to form the hepatic plexus which supplies parasympathetic motor nerves to the extra hepatic biliary system. A number of studies have investigated the effect of vagal stimulation and vagatomy on gallbladder contractability, but the results are generally has been inconclusive.[11]

Total parenteral nutrition and gallstone formation

A number of large clinical studies have confirmed the etiological relationship between TPN and gallstone formation in both children and adult. Symptomatic gallstone disease forms in approximately 45% of patients who are maintained on long term TPN. Ultrsonographic studies have helped to identify the scientific basis for gallstone formation and have our attention on the

relationship between alter gallbladder motor activity, decrease stimulation for gallbladder contraction and the formation of sludge and ultimately biliary lithiasis.[28]

Inflammatory bowel disease

Patients with ileal dysfunction which is more saturated with cholesterol and patients with jejuno-ileal operation are associated with increased risk of gallstone formation. When the ileum is diseased or removed absorption of bile salts is impaired and a significant lose of bile salts will occur, as result of lose of bile salts there will be relative increase in cholesterol leading to the gall stone formation.[33]

Miscellaneous

The prevalence of gallstones in thalassaemia is about 10%, in sickle cell disease is 10% to 40%, and in hereditary spherocytosis is 43% to 66%. Pigment gallstones are reported in 58% of patients with homozygous sickle disease and in 17% of the patients with heterozygous type.[39] Hormonal changes during pregnancy and alteration of gallbladder motility by progesterone are thought to be responsible for the development of gallstones in women. There is no increased risk of morbidity if surgical therapy for biliary disease is carried out in the second trimester.[40]

Children with cystic fibrosis have increased risk of gallbladder disease. There is a controversy over an association between the gallstone and colorectal cancer and gastric cancer.[41] There is an association between hiatal hernia and

diverticular disease of the colon and gallstone. Over 70% of patients developing gallbladder carcinoma have gallstone. A higher incidence of carcinoma have been reported in patients with having larger stones.[42]

Pathophysiology

Ninety percent of cases of cholecystitis involve stones in the cystic duct (ie, calculous cholecystitis), with the other 10% of cases representing acalculus cholecystitis.[1]

Acute calculous cholecystitis is caused by obstruction of the cystic duct, leading to distention of the gallbladder. As the gallbladder becomes distended, blood flow and lymphatic drainage are compromised, leading to mucosal ischemia and necrosis.[11]

Although the exact mechanism of acalculous cholecystitis is unclear, several theories exist. Injury may be the result of retained concentrated bile, an extremely noxious substance. In the presence of prolonged fasting, the gallbladder never receives a cholecystokinin (CCK) stimulus to empty; thus, the concentrated bile remains stagnant in the lumen.[43,44]

A study by Cullen et al demonstrated the ability of endotoxin to cause necrosis, hemorrhage, areas of fibrin deposition, and extensive mucosal loss, consistent with an acute ischemic insult.[45] Endotoxin also abolished the contractile response to CCK, leading to gallbladder stasis.

Presentation

The most common presenting symptom of acute cholecystitis is upper abdominal pain. Signs of peritoneal irritation may be present, and in some patients, the pain may radiate to the right shoulder or scapula. Frequently, the pain begins in the epigastric region and then localizes to the right upper quadrant (RUQ). Although the pain may initially be described as colicky, it becomes constant in virtually all cases. Nausea and vomiting are generally present, and patients may report fever.[46]

Most patients with acute cholecystitis describe a history of biliary pain. Some patients may have documented gallstones. Acalculous biliary colic also occurs, most commonly in young to middle-aged females. The presentation is almost identical to calculous biliary colic with the exception of reference range laboratory values and no findings of cholelithiasis on ultrasound. Cholecystitis is differentiated from biliary colic by the persistence of constant severe pain for more than 6 hours.[47]

Patients with acalculous cholecystitis may present similarly to patients with calculous cholecystitis, but acalculous cholecystitis frequently occurs suddenly in severely ill patients without a prior history of biliary colic. Often, patients with acalculous cholecystitis may present with fever and sepsis alone, without history or physical examination findings consistent with acute cholecystitis.[11]

Cholecystitis in elderly persons

Elderly patients (especially patients with diabetes) may present with vague symptoms and without many key historical and physical findings. Pain and fever may be absent, and localized tenderness may be the only presenting sign. Elderly patients may also progress to complicated cholecystitis rapidly and without warning.[3]

Cholecystitis in children

The pediatric population may also present without many of the classic findings. Children who are at higher risk for developing cholecystitis include patients with sickle cell disease, seriously ill children, those on prolonged TPN, those with hemolytic conditions, and those with congenital and biliary anomalies.[48]

Pregnant patients

Right upper quadrant pain in pregnancy can be related to a number of different diagnoses, including preeclampsia, appendicitis, and cholelithiasis. Pregnant patients must have a thorough examination because complications can arise quickly and can be life threatening to both the mother and the unborn child.[49]

Complications

Bacterial proliferation within the obstructed gallbladder results in empyema of the organ. Patients with empyema may have a toxic reaction and

may have more marked fever and leukocytosis.[50] The presence of empyema frequently requires conversion from laparoscopic to open cholecystectomy.[51]

In rare instances, a large gallstone may erode through the gallbladder wall into an adjacent viscus, usually the duodenum. Subsequently, the stone may become impacted in the terminal ileum or in the duodenal bulb and/or pylorus, causing a gallstone ileus.[11]

Emphysematous cholecystitis occurs in approximately 1% of cases and is noted by the presence of gas in the gallbladder wall from the invasion of gas-producing organisms, such as *Escherichia coli, Clostridia perfringens,* and *Klebsiella* species. This complication is more common in patients with diabetes, has a male predominance, and is acalculous in 28% of cases. Because of a high incidence of gangrene and perforation, emergency cholecystectomy is recommended. Perforation occurs in up to 15% of patients.[52]

The physical examination may reveal fever, tachycardia, and tenderness in the RUQ or epigastric region, often with guarding or rebound. The Murphy sign, which is specific but not sensitive for cholecystitis, is described as tenderness and an inspiratory pause elicited during palpation of the RUQ. A palpable gallbladder or fullness of the RUQ is present in 30-40% of cases. Jaundice may be noted in approximately 15% of patients.[17]

The absence of physical findings does not rule out the diagnosis of cholecystitis. Many patients present with diffuse epigastric pain without localization to the RUQ. Patients with chronic cholecystitis frequently do not have a palpable RUQ mass secondary to fibrosis involving the gallbladder.[17]

Elderly patients and patients with diabetes frequently have atypical presentations, including absence of fever and localized tenderness with only vague symptoms.[18]

Diagnosis

Delays in making the diagnosis of acute cholecystitis result in a higher incidence of morbidity and mortality. This is especially true for intensive care unit (ICU) patients who develop acalculous cholecystitis. The diagnosis should be considered and investigated promptly in order to prevent poor outcomes.[24]

Differential diagnosis[17,18,23]

- Abdominal Aortic Aneurysm
- Acute Mesenteric Ischemia
- Appendicitis
- Biliary Colic
- Biliary Disease
- Cholangiocarcinoma
- Cholangitis
- Choledocholithiasis
- Cholelithiasis
- Gallbladder Cancer
- Gallbladder Mucocele
- Gallbladder Tumors
- Gastric Ulcers
- Gastritis, Acute

- Pyelonephritis, Acute

Approach

The workup for cholecystitis may include laboratory tests (though these are not always reliable), radiography, ultrasonography, computed tomography (CT), magnetic resonance imaging (MRI), hepatobiliary scintigraphy (HBS), and endoscopy.

Laboratory investigations[53]

Although laboratory criteria are not reliable in identifying all patients with cholecystitis, the following findings may be useful in arriving at the diagnosis:

- Leukocytosis with a left shift may be observed in cholecystitis.
- Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels are used to evaluate the presence of hepatitis and may be elevated in cholecystitis or with common bile duct obstruction.
- Bilirubin and alkaline phosphatase assays are used to evaluate evidence of common duct obstruction.
- Amylase/lipase assays are used to evaluate the presence of pancreatitis. Amylase may also be elevated mildly in cholecystitis.
- An elevated alkaline phosphatase level is observed in 25% of patients with cholecystitis.
- Urinalysis is used to rule out pyelonephritis and renal calculi.
- All females of childbearing age should undergo pregnancy testing.

Imaging

The 2010 American College of Radiology (ACR) Appropriateness Criteria offer the following imaging recommendations:[54]

- Sonography is the preferred initial imaging test for the diagnosis of cholelithiasis acute cholecystitis, and scintigraphy is the preferred alternative.
- CT is a secondary imaging test that can identify extrabiliary disorders and complications of cholecystitis, such as gangrene, gas formation, and perforation.
- CT with intravenous contrast is useful in diagnosing cholecystitis in patients with nonspecific abdominal pain.
- MRI, often with intravenous gadolinium-based contrast medium, is also a possible secondary imaging modality useful in confirming a diagnosis of acute cholecystitis.
- MRI without contrast is useful to eliminate radiation exposure in pregnant women for whom sonograms have not indicated a clear diagnosis.

Radiography

Gallstones may be visualized on noncontrast radiography in 10-15% of cases. This finding only indicates cholelithiasis, with or without active cholecystitis.

Ultrasonography

Ultrasonography is 90-95% sensitive for cholecystitis and is 78-80% specific. It provides greater than 95% sensitivity and specificity for the diagnosis of gallstones more than 2 mm in diameter. Studies indicate that emergency clinicians require minimal training in order to use right upper quadrant ultrasonography in their practice.[55,56]

Ultrasonographic findings that are suggestive of acute cholecystitis include the following: pericholecystic fluid, gallbladder wall thickening greater than 4 mm, and sonographic Murphy sign. The presence of gallstones also helps to confirm the diagnosis.[57]

Ultrasonography is performed best following a fast of at least 8 hours because gallstones are visualized best in a distended bile-filled gallbladder.[58]

Computed tomography and magnetic resonance imaging

The sensitivity and specificity of CT scan and MRI for predicting acute cholecystitis have been reported to be greater than 95%.[59] Spiral CT scan and MRI (unlike endoscopic retrograde cholangiopancreatography [ERCP]) have the advantage of being noninvasive, but they have no therapeutic potential and are most appropriate in cases where stones are unlikely.

Findings suggestive of cholecystitis include wall thickening (>4 mm), pericholecystic fluid, subserosal edema (in the absence of ascites), intramural gas, and sloughed mucosa.

CT scan and MRI are also useful for viewing surrounding structures if the diagnosis is uncertain.

Hepatobiliary scintigraphy[53]

HBS has been found to be up to 95% accurate in diagnosing acute cholecystitis. The reported sensitivities and specificities of biliary scintigraphy are in the range of 90-100% and 85-95%.

In a typical study, the gallbladder, common bile duct, and small bowel fill within 30-45 minutes. If the gallbladder is not visualized, intravenous morphine administration can improve the accuracy of HBS by increasing resistance to flow through the sphincter of Oddi, resulting in filling of the gallbladder if the cystic duct is patent. The addition of morphine also reduces the number of false-positive scan results observed in patients who are critically ill and immobilized with viscous bile.

Endoscopic retrograde cholangiopancreatography

ERCP may be useful for visualizing the anatomy in patients at high risk for gallstones if signs of common bile duct obstruction are present. A study performed by Sahai et al found that ERCP was preferred over endoscopic ultrasonography and intraoperative cholangiography for patients at high risk for common bile duct stones undergoing laparoscopic cholecystectomy.[60]

Disadvantages of ERCP include the need for a skilled operator, high cost, and complications such as pancreatitis, which occurs in 3-5% of cases.

Histologic findings

Edema and venous congestion are early acute changes. Acute cholecystitis is usually superimposed on a histologic picture of chronic cholecystitis. Specific findings include fibrosis, flattening of the mucosa, and chronic inflammatory cells. Mucosal herniations known as Rokitansky-Aschoff sinuses are related to increased hydrostatic pressure and are present in 56% of cases. Focal necrosis and an influx of neutrophils may also be present. Advanced cases may show gangrene or perforation.[16]

Treatment

Treatment of cholecystitis depends on the severity of the condition and the presence or absence of complications. Uncomplicated cases can often be treated on an outpatient basis; complicated cases may necessitate a surgical approach. Antibiotics may be given to manage infection.

Initial Therapy and Antibiotic Treatment

For acute cholecystitis, initial treatment includes bowel rest, intravenous hydration, correction of electrolyte abnormalities, analgesia, and intravenous antibiotics. For mild cases of acute cholecystitis, antibiotic therapy with a single broad-spectrum antibiotic is adequate.

Conservative Treatment of Uncomplicated Cholecystitis[11]

Outpatient treatment may be appropriate for cases of uncomplicated cholecystitis. If a patient can be treated as an outpatient, discharge with

antibiotics, appropriate analgesics, and definitive follow-up care. Criteria for outpatient treatment include the following:

- Afebrile with stable vital signs
- No evidence of obstruction by laboratory values
- No evidence of common bile duct obstruction on ultrasonography
- No underlying medical problems, advanced age, pregnancy, or immunocompromised condition
- Adequate analgesia
- Reliable patient with transportation and easy access to a medical facility
- Prompt follow-up care

Surgical Treatment

Surgical treatment, if required, typically involves cholecystectomy, preferably laparoscopic. Percutaneous drainage may be considered in patients at high surgical risk.[61]

Cholecystectomy

Laparoscopic cholecystectomy is the standard of care for the surgical treatment of cholecystitis. Studies have indicated that early laparoscopic cholecystectomy resulted in shorter total hospital stays with no significant difference in conversion rates or complications.[62,63,64] The ACR 2010 criteria state that laparoscopic cholecystectomy is the primary mode of treatment for cholecystitis.[54]

The Society of American Gastrointestinal and Endoscopic Surgeons (SAGES) issued guidelines for the clinical application of laparoscopic biliary tract surgery in 2010. The guidelines include detailed recommendations for making the decision to operate, performing the procedure, and managing postoperative care, with the patient's safety always the primary consideration. Recommendations are as follows:[65]

- Preoperative antibiotics should be considered only to reduce the possibility of wound infection in high-risk patients, and then limited to one preoperative dose.

- Intraoperative cholangiography may improve injury recognition and decrease the risk of bile duct injury.

- If bile duct injury occurs, the patient should be referred to an experienced hepatobiliary specialist before any repair is undertaken, unless the primary surgeon has experience with biliary reconstruction.

Early operation within 72 hours of admission has both medical and socioeconomic benefits and is the preferred approach for patients treated by surgeons with adequate experience in laparoscopic cholecystectomy.[66] Immediate cholecystectomy or cholecystotomy is usually reserved for complicated cases in which the patient has gangrene or perforation.

One study suggests that when CT scanning is performed as long as 72 hours prior to surgery, it may better detect acute gangrenous cholecystitis. Acute gangrenous cholecystitis was significantly correlated with perfusion defect of the

gallbladder wall, pericholecystic stranding, and no-gallstone condition, which can be better observed through CT scanning when compared with ultrasonography.[67]

For elective laparoscopic cholecystectomy, the rate of conversion from a laparoscopic procedure to an open surgical procedure is approximately 5%. The conversion rate for emergency cholecystectomy where perforation or gangrene is present may be as high as 30%.[62]

Although laparoscopic cholecystectomy performed in pregnant women is considered safest during the second trimester, it has been performed successfully during all trimesters.

Percutaneous drainage

For patients at high surgical risk, placement of a sonographically guided, percutaneous, transhepatic cholecystostomy drainage tube coupled with the administration of antibiotics may provide definitive therapy.[68] Results of studies suggest that most patients with acute acalculous cholecystitis can be treated with percutaneous drainage alone,[69] but the SAGES guideline describes radiographically guided percutaneous cholecystostomy as a temporizing measure until the patient can undergo cholecystectomy.[65]

Endoscopic Treatment

Endoscopy may be used for therapeutic purposes, as well as for diagnosis.

Endoscopic retrograde cholangiopancreatography

Endoscopic retrograde cholangiopancreatography (ERCP) allows visualization of the anatomy and may be therapeutic by removing stones from the common bile duct. [70]

Endoscopic ultrasound-guided transmural cholecystostomy

Studies indicate that this procedure may be safe as initial, interim, or definitive treatment of patients with severe acute cholecystitis who are at high operative risk for immediate cholecystectomy.[70]

A retrospective study was performed on 910 consecutive patients who underwent laparoscopic cholecystectomy for cholecystitis. The patients were classified according to body mass index (BMI) as obese (n = 354, BMI > or =25 kg/m(2)) and nonobese (n = 556; BMI <25). In males, there was a significant negative correlation between the BMI and the severity of cholecystitis; the proportion of complicated acute cholecystitis was higher in the nonobese patients (21.5%) compared with the obese patients (8.1%) (P = .007) but not for the females (P = .80). A BMI <25 (odds ratio [OR] = 1.92, P = .01), advanced age (OR = 2.52, P < .001), male sex (OR = 1.74, P = .022), and leukocytosis (OR = 1.92, P = .024) were independent predictors for the development of complicated acute cholecystitis. There is a negative association between BMI and the inflammation severity of cholecystitis in males, which resulted in a higher incidence of severe cholecystitis in the nonobese male patients.[4] However to date this is the only study comparing body mass index and severity of cholecystitis.

Several studies have reported that male sex have severe cholecystitis compared females. The relationship between sex and outcome after laparoscopic surgery for symptomatic cholelithiasis remains unclear.[71]

A study to determine the importance of gender in the clinical presentation and subsequent clinical outcome (risk of conversion from laparoscopic to open technique and risk of postoperative mortality) for patients undergoing cholecystectomy reported that, age and clinical presentation have consistently been found to be important predictors of cholecystectomy outcomes; male gender has been cited in separate studies as possibly having prognostic significance. A statewide cholecystectomy registry (30,145 cases between 1989-1993) reported that, male gender was associated with twice the expected incidence of acute cholecystitis and pancreatitis in the elderly (≥ 65 years). Males had a significantly increased risk for conversion to open technique, but this decreased during the time frame of the study. Mortality was twice as high among males (CI, 1.4-2.9, p=0.0001). The study concluded that, males presenting for cholecystectomy are more likely to have severe disease. Independent of clinical presentation, they face increased risks of conversion to open technique and of postoperative mortality.[7]

Laparoscopic cholecystectomy has been accepted as standard procedure for the management of symptomatic cholelithiasis even when the gallbladder is acutely inflamed. With the accumulated experience in the management of acute cholecystitis, some factors including male gender were recognized to influence the clinical presentation of symptomatic cholelithiasis and increase the conversion rate during LC. A retrospective study was conducted to determine the effect of male gender on the clinical presentation of symptomatic cholelithiasis.

This study tried to clarify the correlation between male gender and the clinical presentation of symptomatic cholelithiasis. The medical records of all patients presenting with symptomatic cholelithiasis from January 1994 to August 1999 were evaluated. These cases were divided into four groups as follows: (1) elective LC group: patients with a history of biliary colic or acute attack of cholecystitis but whose LC was performed electively without any inflammatory change in the gallbladder during operation; (2) acute LC group: patients presenting with acute cholecystitis, and LC was performed successfully without conversion; (3) acute conversion group: patients who underwent LC during the course of acute cholecystitis but the procedure were disturbed by severe inflammatory change so they were converted to open surgery; (4) acute open group: patients whose acute cholecystitis was managed by direct open surgery due to the preoperative prediction that LC would not succeed. The correlation of gender, age, and operating time were assessed among these four groups. Study found that: (1) the male/female ratio increased (in the patient group sequence of simple LC, acute LC, acute open, and acute conversion group); (2) in the acute LC group male patients had significantly ($p = 0.04$, t-test) longer operating time than females; (3) although there was no significant difference between the mean age of male (55.7 +/- 13.4) and female (56.3 +/- 15.7) patients in the acute cholecystitis groups (i.e., all patients in the acute LC, acute conversion, and acute open groups), the distribution curve by age in male patients showed a significantly shift to a younger age compared with female patients ($p = 0.009$, Fisher's exact test).[8]

In a study done to determine the influence of sex on the clinical presentation of patients with symptomatic gallstone disease and the clinical

outcomes of laparoscopic cholecystectomy. It was shown that when compared with female patients, males were significantly older and more likely to have coexisting cardiovascular disease, previous upper abdominal surgery, previous hospitalization for acute cholecystitis and pancreatitis, acute cholecystitis, and suppurative cholecystitis (such as empyema), conversions, and complications. The mortality rate was nil. Analyses revealed an independent effect of sex on the prevalence of complications, even when including all of the major confounding factors in the model. In contrast, the effect of sex on conversion to open cholecystectomy was not significant when controlling for patient age. Operative time and postoperative hospital stay were significantly longer in males than in females. The tendency of male patients to have cholecystitis of greater severity should remind surgeons of the need to inform patients about the higher conversion rate among male patients, to reduce the disappointment of a large laparotomy wound or prolonged recovery period. On the other hand, there may be an increased need for surgeons to strongly advice male patients with symptomatic cholelithiasis to undergo early intervention.[71]

In a prospective study from July 2004 to December 2007, on 1059 patients who underwent laparoscopic cholecystectomy for symptomatic gallstones were recorded to assess risk factors for AC and operative outcome. Study reported that, the diagnoses of the 1059 patients who underwent laparoscopic cholecystectomy were chronic cholecystitis (n = 704 [66.5%]) and AC (n = 355; [33.5%]). An age older than 60 years (odds ratio [OR], 1.955; 95% confidence interval [CI], 1.441-2.652), male sex (OR, 1.769; 95% CI, 1.346-2.325), the presence of cardiovascular disease (OR, 1.826; 95% CI, 1.325-2.517),

the presence of diabetes mellitus (OR, 1.802; 95% CI, 1.153-2.816), and a history of cerebrovascular accident (ischemic stroke or cerebral hemorrhage) (OR, 8.107; 95% CI, 2.650-24.804) were identified as independent risk factors for AC after multivariate analysis. Approximately 85% of the patients with a history of cerebrovascular accident presented with AC (P < .001), 54.5% of whom experienced complicated AC (P < .001). Acute cholecystitis was associated with greater operative difficulty and more postoperative morbidity than chronic cholecystitis. Study concluded that, for the patients with risk factors for AC, early cholecystectomy is recommended before the disease progresses to AC.[72]

From November 1997 to November 1998, 145 cases of laparoscopic cholecystectomy (LC) were attempted at the District General Hospital of Corfu. 23 (15.8%) were obese (Group I, BMI >30) and 122 (84.2%) were nonobese patients (Group II, BMI < or =30). One-fifth of these patients suffered from acute cholecystitis. Operative time averaged 95 minutes in Group I and 78 minutes in Group II. There were no deaths. There were no significant differences between the obese and nonobese groups in conversion to open procedure (Group I: 0%, Group II: 2.4%), intraoperative and postoperative complications (Group I: 4.3%, Group II: 4.0%), operating time, and length of postoperative hospitalization. LC was a safe and effective treatment for obese patients with symptomatic cholelithiasis.[73]

Obese patients treated by laparoscopic cholecystectomy currently appear to be the largest risk subgroup amenable to consistent scientific evaluation. A study we compared the results in obese patients with those obtained in nonobese patients undergoing the laparoscopic procedure. Laparoscopic cholecystectomy

in obese patients was technically more difficult with significantly longer operating time ($p < 0.01$), but intraoperative and postoperative technical complications were not significant in the groups analyzed. Obese patients present significant anesthesiological complications ($p <$ or $= 0.001$). The results of this experience and the literature review indicate that the therapeutic advantages proved in nonobese patients can be extended to the obese population.[74]

The outcome analysis of obese patients undergoing laparoscopic cholecystectomy (LC) in Asia-Pacific countries is rarely reported. A study examined associations between body mass index (BMI) and clinical outcomes of elective LC in Taiwan. A total of 627 patients with gallbladder disease due to gallstones undergoing LC were divided into three groups based on BMI: <25.0 kg/m2 (normal, NO; n = 310), 25.0-29.9 kg/m2 (overweight, OW; n = 252), and >30 kg/m2 (obese, OB; n = 65). Both overweight and obesity were not associated with conversion and complication rates. The conversion rates of the three groups were 5.5 (NO), 6.0 (OW), and 4.6% (OB), and the complication rates were 3.2 (NO), 2.4% (OW), and 4.6% (OB), respectively. However, overweight and obesity were related to a trend toward longer operating time (NO 67.4 +/- 31.8; OW 77.8 +/- 35.6; OB 79.0 +/- 37.9 min) (P trend <0.001). One death (BMI 40.6 kg/m2) was due to septic complications. In the multivariable logistic analysis, only acute cholecystitis, but not BMI, was a predictor for conversion and complications. Based on these results, it appears that BMI was not associated with clinical outcomes and that LC is a safe procedure in obese patients with uncomplicated gallstone disease in Taiwan.[75]

METHODOLOGY

The present study was conducted in the Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2010 to December 2010.

Study design

A one year cross sectional study.

Place

The present study was conducted in the Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum attached to Jawaharlal Nehru Medical College, Belgaum.

Study period

One year from January 2010 to December 2010.

Source of data

Patients admitted with cholecystitis in the wards of Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the study period.

Sample size and method

Due to the scarcity of the literature based on the 80% of average of last three year cholecystitis admissions, a minimum of 120 patients was planned.

Selection criteria

Inclusion

- All patients admitted with cholecystitis irrespective of age and gender.

Exclusion

- Patient refusal
- Immunocompromised patients(HIV infected patients, Chronic steroid therapy)
- Hepatitis B patients

Procedure

Ethical clearance for the study was obtained from Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belgaum. Based on the selection criteria patient admitted with cholecystitis at Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were screened for eligibility. The eligible patients were briefed about the nature of the study and a written informed consent (Annexure I) was obtained from the selected patients. Thorough history was taken and clinical examination was done for all patients and findings were recorded on predesigned and pretested proforma (Annexure II).

Patients were examined for height and weight and their Body Mass Index was calculated by using the formula as below.

$$\text{Body Mass Index} = \frac{\text{Weight (Kg)}}{\text{Height}^2 \text{ (m)}}$$

Based on the BMI patients were divided into groups that is, patients with BMI between 18 to 25 kg/m^2 as non-obese group and more than 25 kg/m^2 as obese group.[37]

Further patient were examined clinically and investigations were done. Ultrasonography findings like sonographic murphy sign, thickened gallbladder wall more than four mm, pericholecystic fluid collection were noted. After cholecystectomy following intra-operative findings were also noted to assess severity.

- Pericholecystic collection
- Empyema of Gall bladder
- Gangrene of Gall bladder
- Perforation of Gall bladder
- Severe adhesion to an adjacent organ

Gall bladder specimen was sent for histopathological study and the finding was noted. Demographic characteristics like age, sex were matched in both the groups.

Based on the clinical examination, blood investigations, ultrasonography, and histopathology the severity of disease was categorized chronic cholecystitis and acute cholecystitis.

Acute cholecystitis was further categorized into three categories based on Tokyo guidelines on acute cholecystitis as mentioned below.[75]

Mild or grade I

Cholecystitis in a healthy patient with no organ dysfunction and only mild inflammatory changes in gallbladder or who does not meet the criteria in grade II or grade III.

Moderate or grade II

Presence of any one of the following conditions was graded as moderate to grade II.

1. Elevated WBC count (>18 000 /mm^3).
2. Palpable tender mass in the right upper abdominal quadrant.
3. Duration of complaints more than 72 hours.
4. Marked local inflammation (biliary peritonitis, pericholecystic abscess, hepatic abscess, gangrenous cholecystitis, emphysematous cholecystitis)

Severe or Grade III

Dysfunction of any one of the following organs/systems

1. Cardiovascular dysfunction (hypotension requiring treatment with dopamine 5 μg/kg per min, or any dose of dobutamine)
2. Neurological dysfunction (decreased level of consciousness)
3. Respiratory dysfunction (oxygen saturation/chest X-ray)
4. Renal dysfunction (creatinine more than 2.0 mg/dL)
5. Hepatic dysfunction (PT-INR more than 1.5)
6. Hematological dysfunction (platelet count more than 100,000 /mm^3)

Statistical Analysis:

Data obtained was tabulated and expressed as rates, ratios and percentages. Association between BMI and severity was calculated by chi-square test and Fisher exact test. A probability value ('p' value) of less 0.05 was considered as statistically significant.

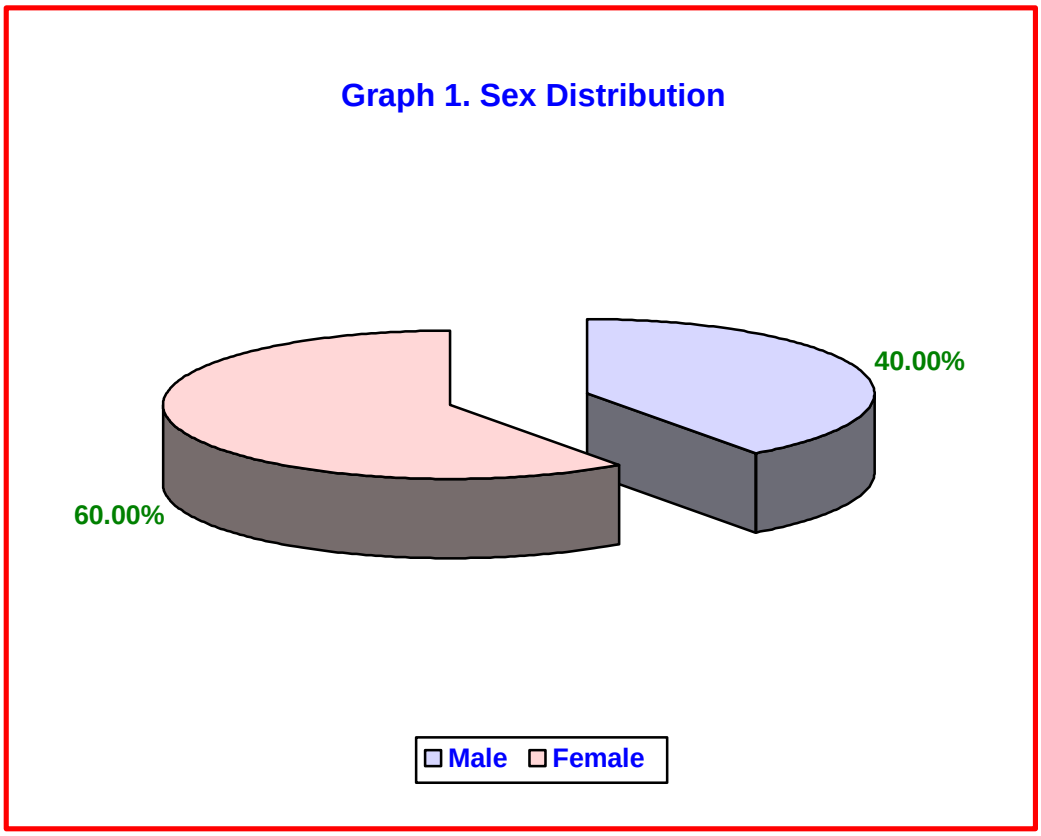
RESULTS

The present one year cross sectional study was conducted on 100 patients admitted with cholecystitis in the wards of Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2010 to December 2010. Patients were examined for height and weight and their Body Mass Index. Based on the BMI, patients were divided into groups that is, patients with BMI between 18 to 25 kg/m^2 as non-obese group and more than 25 kg/m^2 as obese group.

Data obtained was tabulated and expressed as rates, ratios and percentages. Association between BMI and severity was calculated by odds ratio (OR) and statistical significance was tested by applying chi-square test. The data was analysed as below.

Table 1. Sex distribution

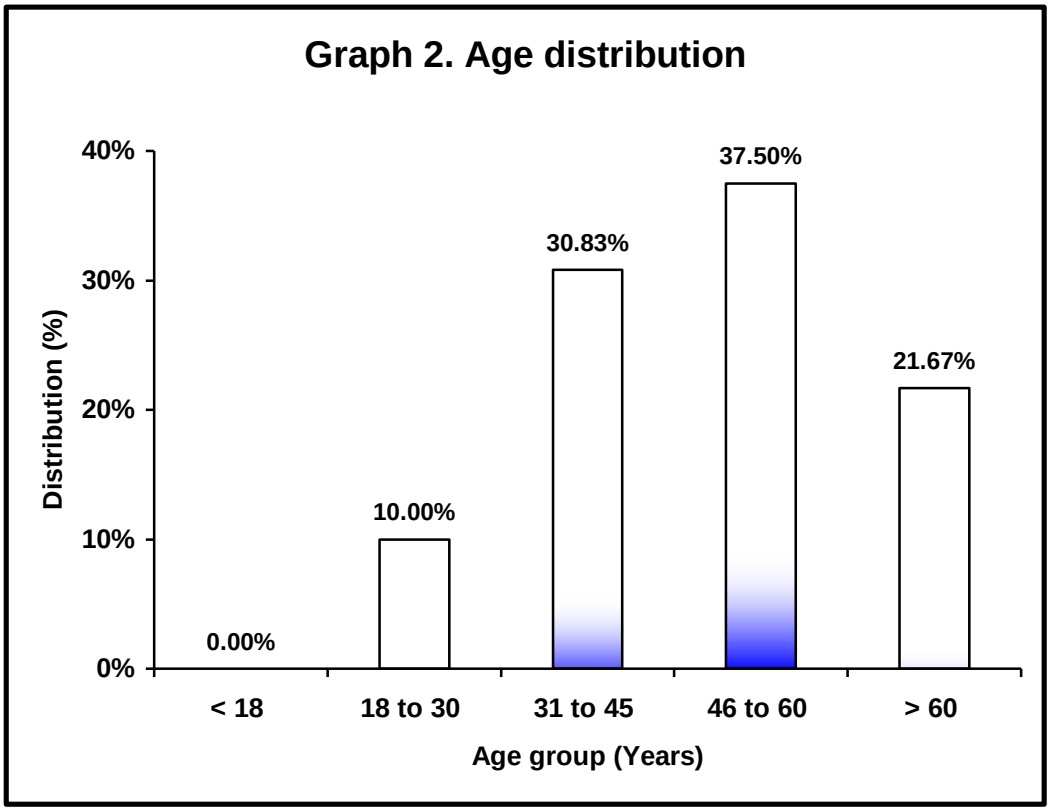
Sex	Distribution (n=120)	
	Number	**Percentage**
Male	48	40.00
Female	72	60.00
Total	**120**	**100.00**



In this study out of 120 cases 48 were male 72 were female i.e 40% and 60% respectively. Male to female ratio was 1:1.5. Sex distribution was comparable in both the groups

Table 2. Age distribution

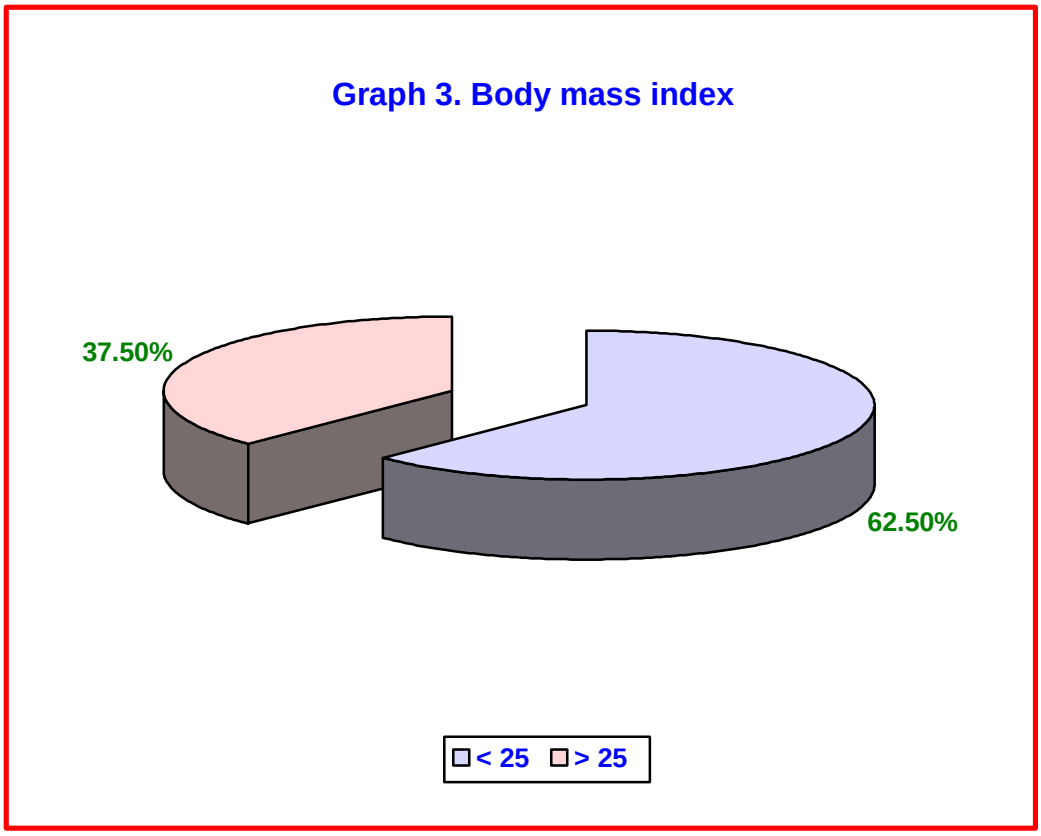
Age group (Years)	Distribution (n=120)	
	Number	**Percentage**
< 18	0	0.00
18 to 30	12	10.00
31 to 45	37	30.83
46 to 60	45	37.50
> 60	26	21.67
Total	**120**	**100.00**



This graph shows age distribution of patients in our study. 68.3% of patients were aged between 30 years to 60 years, 10% were between 18 to 30 years and 21.67% were aged more than 60 years. The mean age in was 49.31 ± 13.57 years with range being 18 to 78 years.

Table 3. Body mass index

Body mass index (Kg/m^2)	Distribution (n=120)	
	Number	**Percentage**
< 25	75	62.50
> 25	45	37.50
Total	**120**	**100.00**

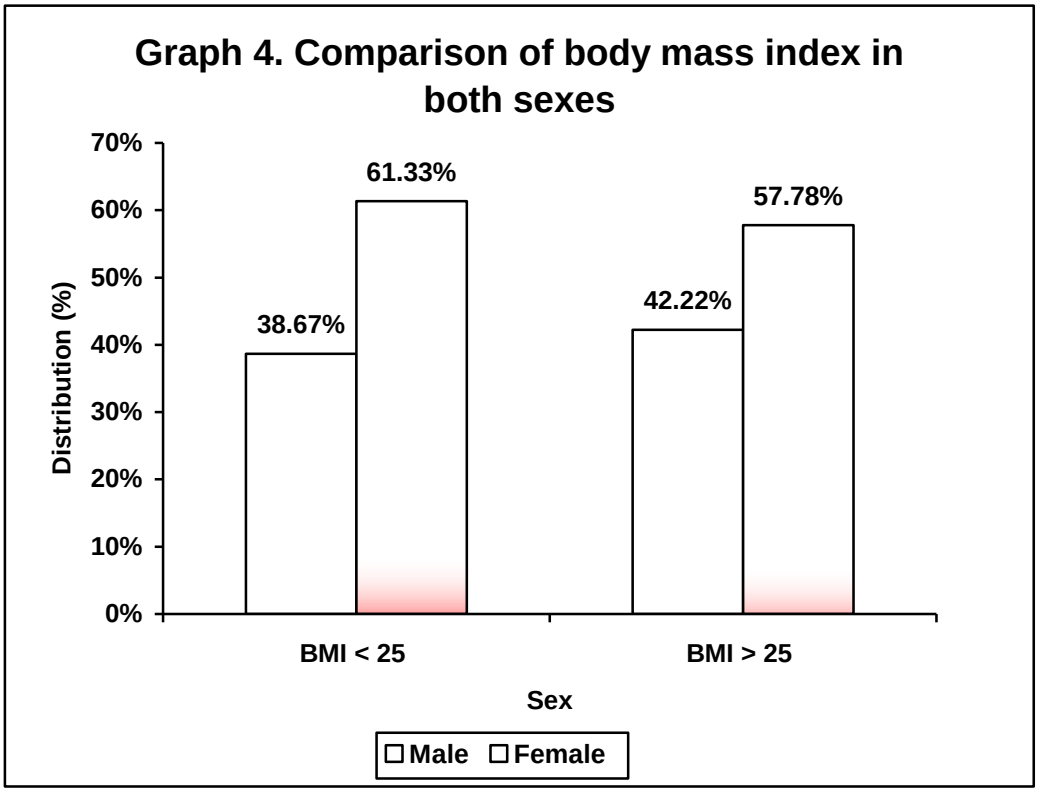


Of 120 enrolled patients 62.5% patients were in non obese group and 37.5% patients in obese group. The mean BMI was 24.35 ± 2.96 Kg/m2 with range being 19.33 to 33.53 years.

Table 4. Comparison of body mass index in both sexes

Sex	BMI < 25 (n=75)		BMI > 25 (n=45)	
	Number	**Percentage**	**Number**	**Percentage**
Male	29	38.67	19	42.22
Female	46	61.33	26	57.78
Total	**75**	**100.00**	**45**	**100.00**

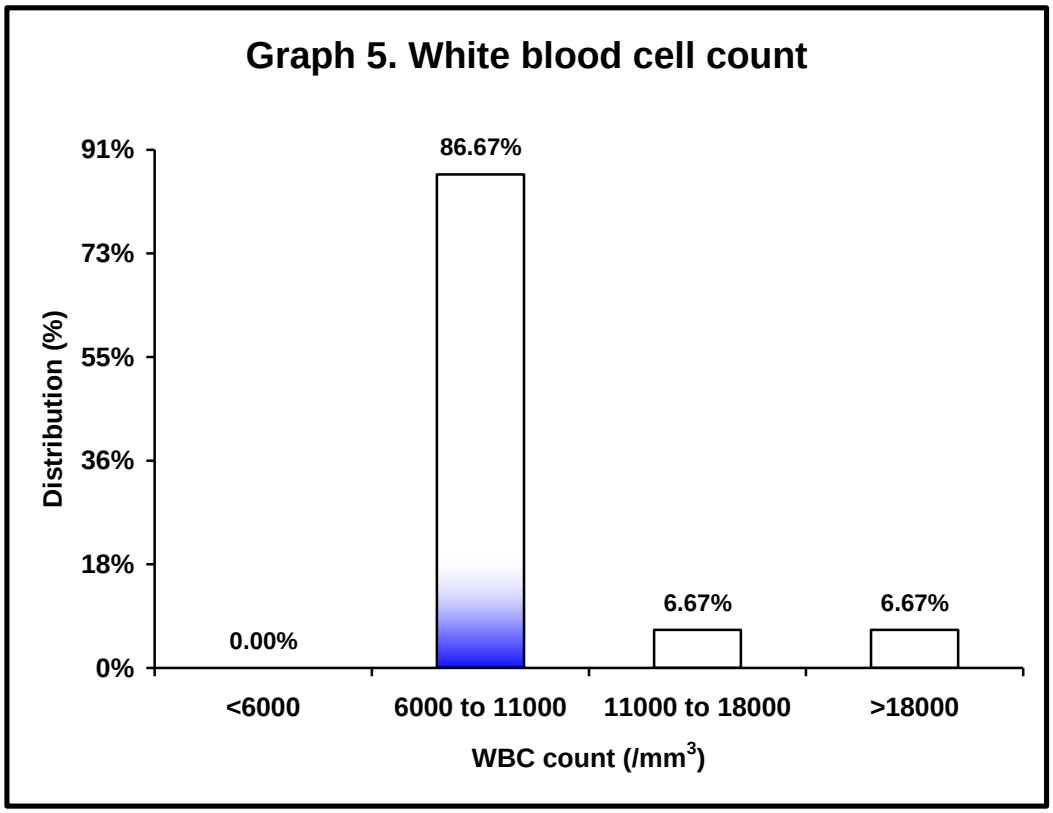
x^2_1=0.148 p=0.700



Of 75 nonobese patients 38.67% were males and 61.33% were females and of 45 obese group 42.22% were males and 57.78% were females. The male to female ratio in non obese group was 1:1.58 and in obese group it was 1:1.36. The difference between two group was not significant (p=0.700)

Table 5. White blood cells count

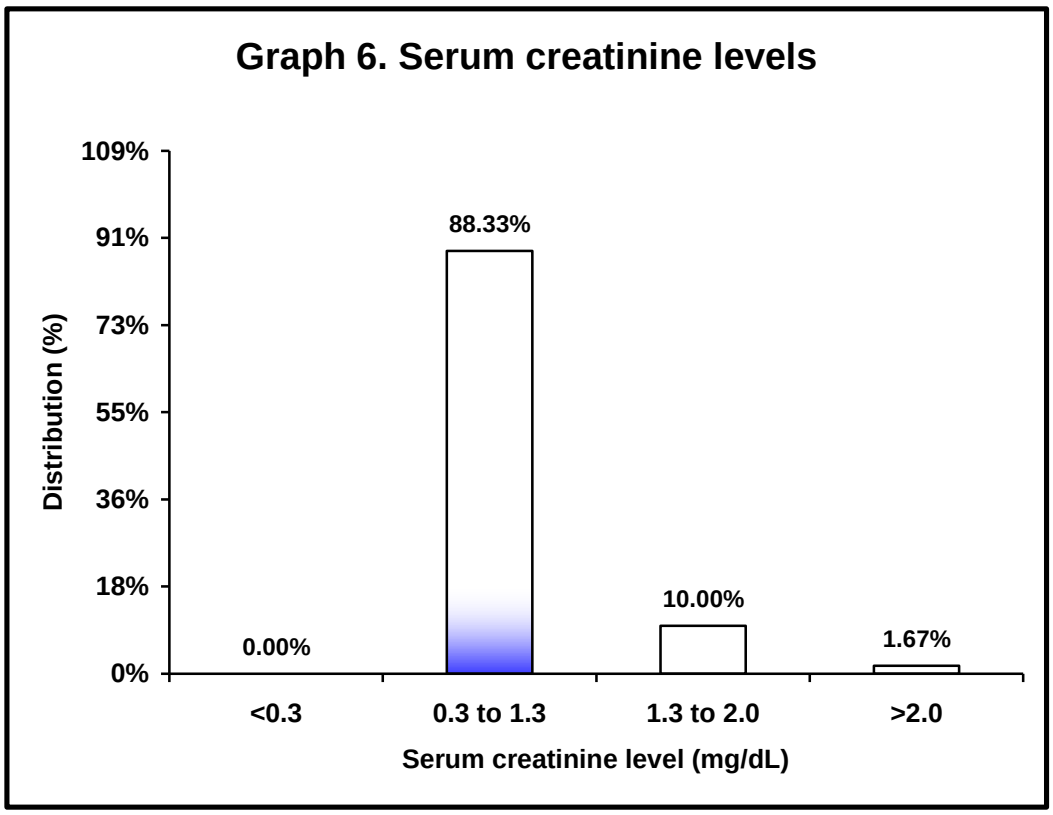
WBC count (/mm^3)	Distribution (n=120)	
	Number	**Percentage**
< 6000	0	0.00
6000 to 11000	104	86.67
11000 to 18000	8	6.67
> 18000	8	6.67
Total	**120**	**100.00**



This table shows distribution of white blood cell count. In this study most of the white blood cell count distribution was between 6000-11000 cells/ml. In 8 (6.67%) patients white blood cell count was more than 18000cells/ml

Table 6. Serum creatinine levels

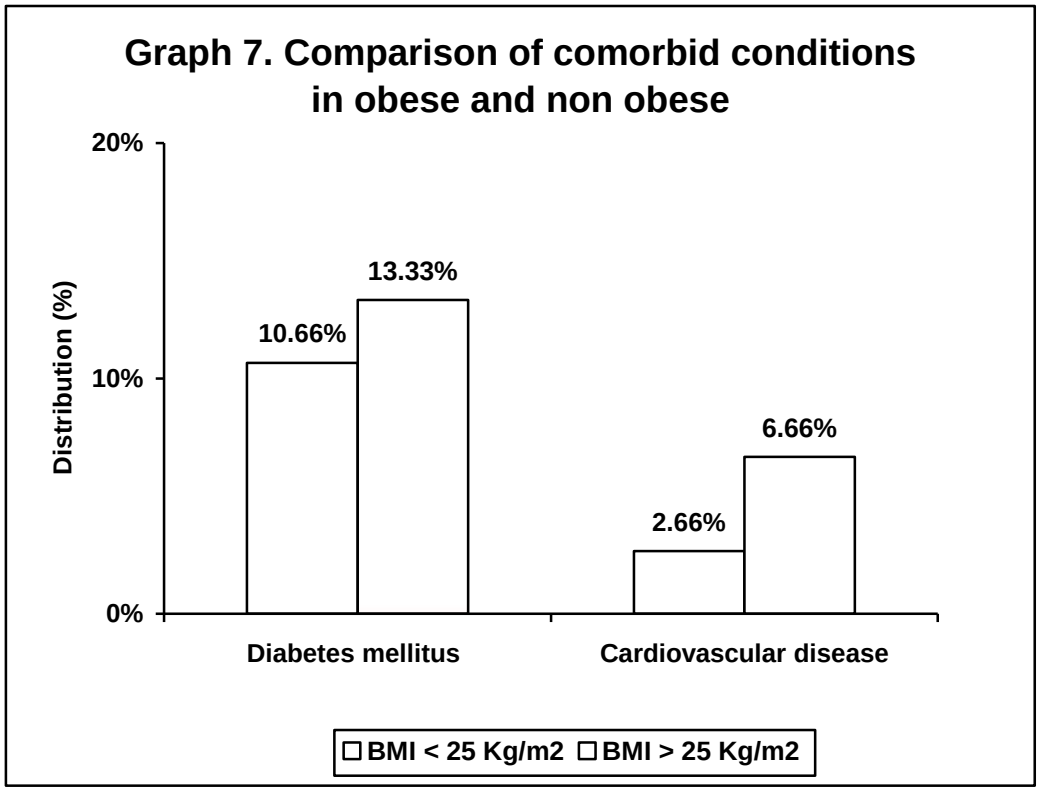
Serum creatinine levels (mg/dL)	Distribution (n=120)	
	Number	Percentage
< 0.3	0	0.00
0.3 to 1.30	106	88.33
1.30 to 2.0	12	10.00
> 2.0	2	1.67



Majority of the patients enrolled in this study had a normal creatinine level except for 2 patients who had a creatinine level of more than 2mg/dl

Table 7. Comparison of comorbid conditions in obese and non obese

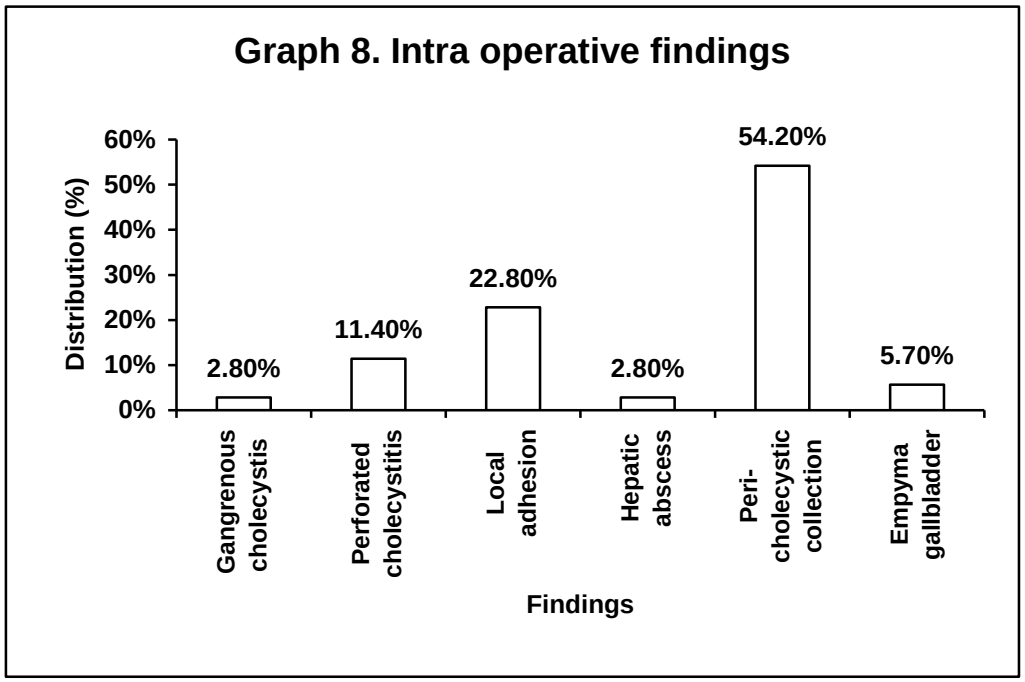
Condition	BMI < 25 (n=75)		BMI > 25 (n=45)	
	Number	Percentage	Number	Percentage
Diabetes mellitus	8	10.66	6	13.33
Cardiovascular disease	2	2.66	3	6.66



In this study of 75 non-obese patients 10.66% had history of diabetes mellitus and 2.66% had cardiovascular disease. Of the 45 obese patients, 13.33% had diabetes mellitus and 6.66% had cardiovascular disease.

Table 8. Intra operative findings

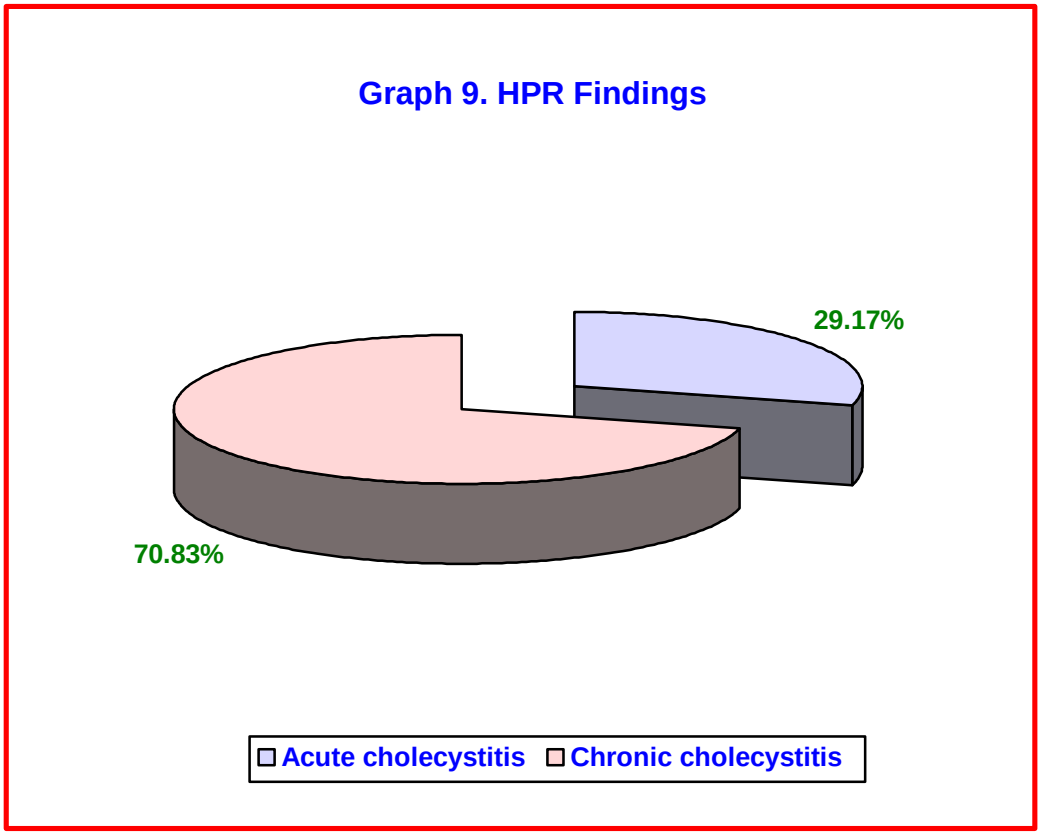
Age group (Years)	Distribution (n=35)	
	Number	**Percentage**
Gangrenous cholecystitis	1	2.8
Perforated cholecystitis	4	11.4
Local adhesion	8	22.8
Hepatic abscess	1	2.8
Peri-cholecystic collection	19	54.2
Empyema gallbladder	2	5.7
Total	**35**	**100**



In this study in cases of acute cholecystitis almost half the cases had pericholecystic collection(54.2%), other 22.8% had local adhesion between gallbladder and adjacent organs, 11.4% i.e 4 cases had perforated gall bladder, 5.7% had empyema gall bladder and 2.8% had gangrenous cholecystitis and 2.8% had hepatic abscess.

Table 9. HPR Findings

Findings	Distribution (n=120)	
	Number	**Percentage**
Acute cholecystitis	35	29.17
Chronic cholecystitis	85	70.83
Total	**120**	**100.00**

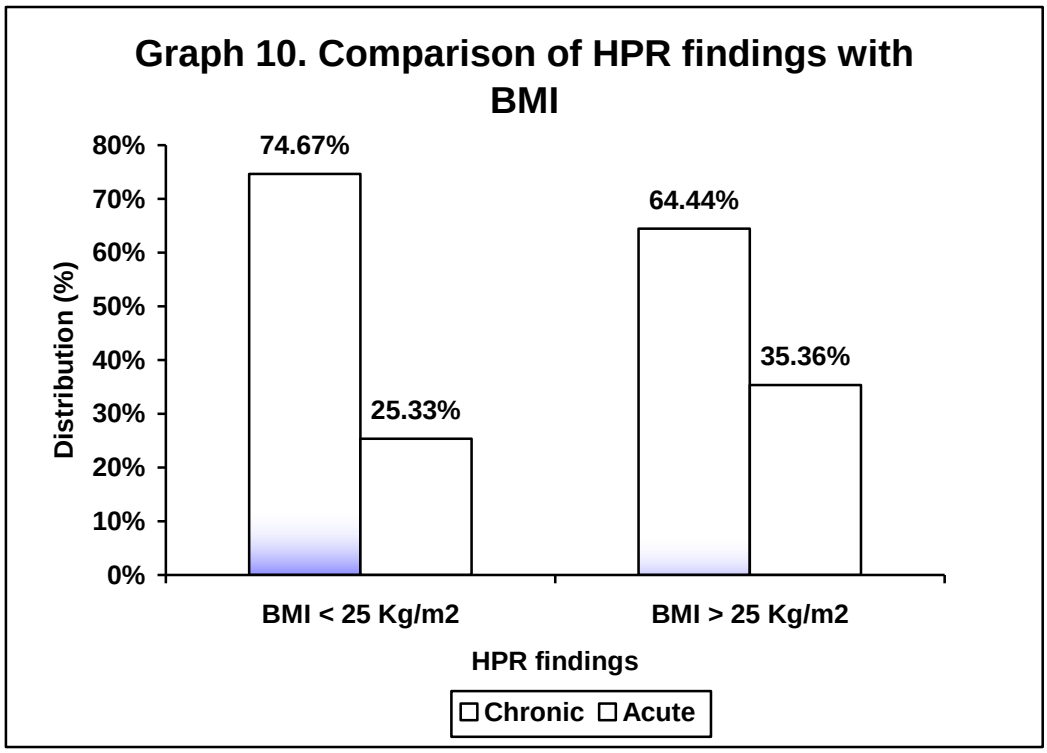


In this study 70.83% patients had chronic cholecystitis and 29.17% patients had acute cholecystitis. Type of cholecystitis was confirmed by histopathological findings

Table 10. Comparison of HPR findings with BMI

HPR findings	BMI < 25 (n=75)		BMI > 25 (n=45)	
	Number	**Percentage**	**Number**	**Percentage**
Chronic	56	74.67	29	64.44
Acute	19	25.33	16	35.56
Total	**75**	**100.00**	**45**	**100.00**

x^2_1=2.689 p=0.101

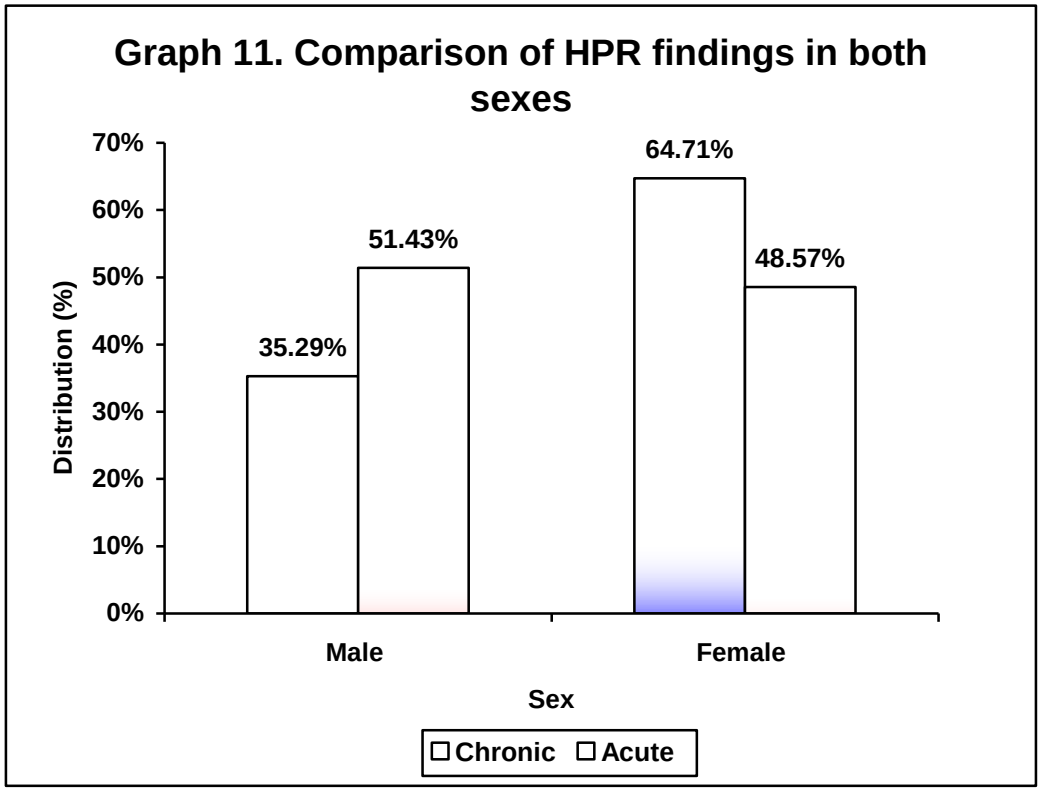


In this study of 75 non-obese patients 74.6% had chronic cholecystitis and 25.3% had acute cholecystitis and of 45 obese patients 64.4% had chronic cholecystitis and 35.5% had acute cholecystitis. There was no significant difference between obese and non-obese patients suffering from acute or chronic cholecystitis

Table 11. Comparison of HPR findings in both sexes

Sex	Chronic (n=85)		Acute (n=35)	
	Number	**Percentage**	**Number**	**Percentage**
Male	30	35.29	18	51.43
Female	55	64.71	17	48.57
Total	**85**	**100.00**	**35**	**100.00**

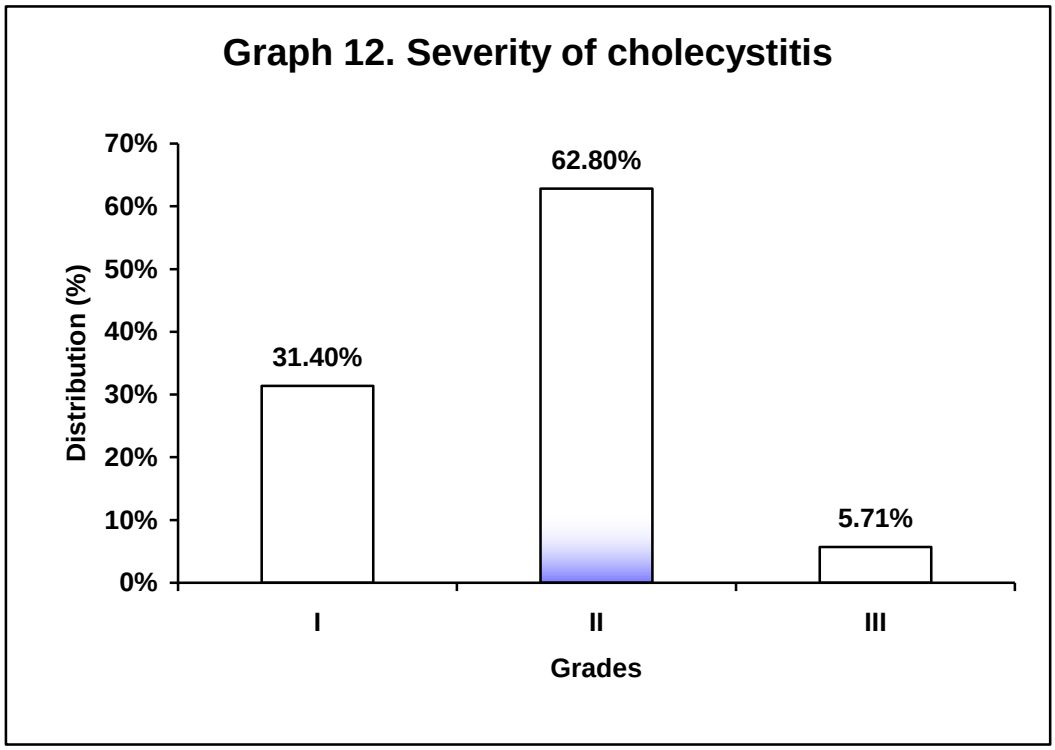
x^2_1=2.689 p=0.101



Of the 85 chronic cholecystitis 35.29% were and 64.71% were females. Among those with acute cholecystitis 51.43% were male and 48.57% were females and there was no statistically significant difference (p=0.101).

Table 12. Severity of cholecystitis

Grades	Distribution (n=35)	
	Number	**Percentage**
I	11	31.4
II	22	62.8
III	2	5.71
Total	**35**	**100**

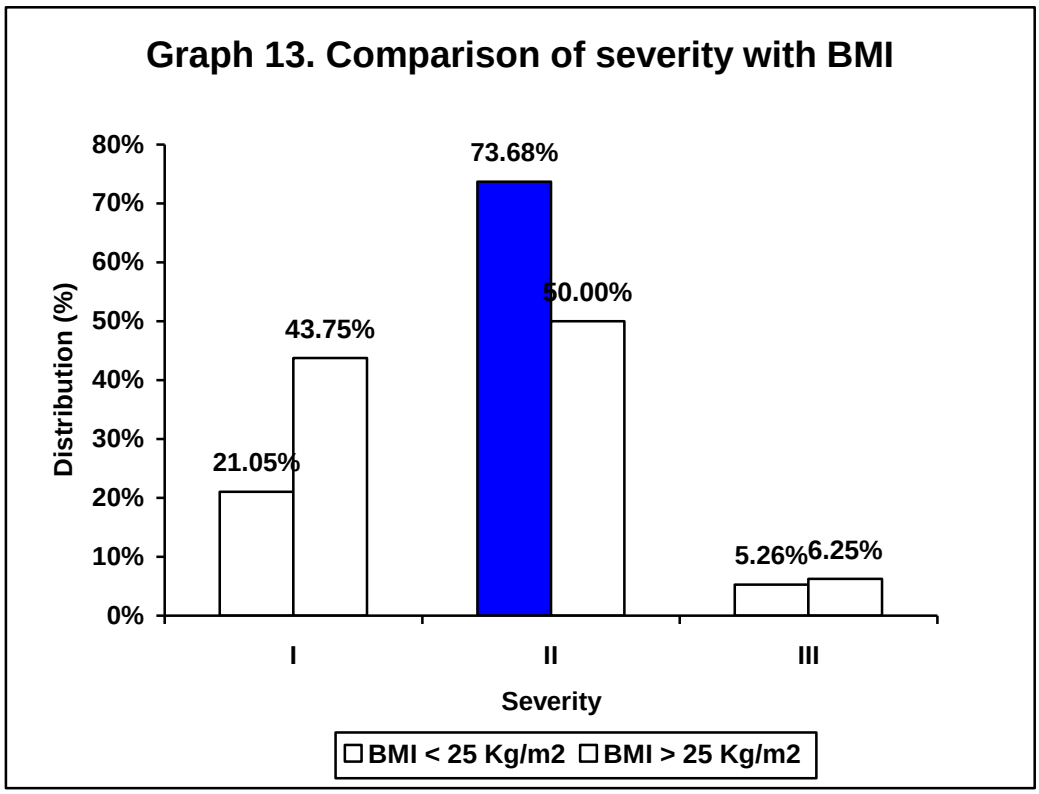


In our study out of 35 patients 31.4% had grade I, 62.8% grade II and 5.71% grade III severity of acute cholecystitis.

Table 13. Comparison of severity with BMI

Severity	BMI < 25 (n=19)		BMI > 25 (n=16)	
	Number	Percentage	Number	Percentage
I	4	21.05	7	43.75
II	14	73.68	8	50.00
III	1	5.26	1	6.25
Total	**19**	**100.00**	**16**	**100.00**

x^2_1=2.076 p=0.150

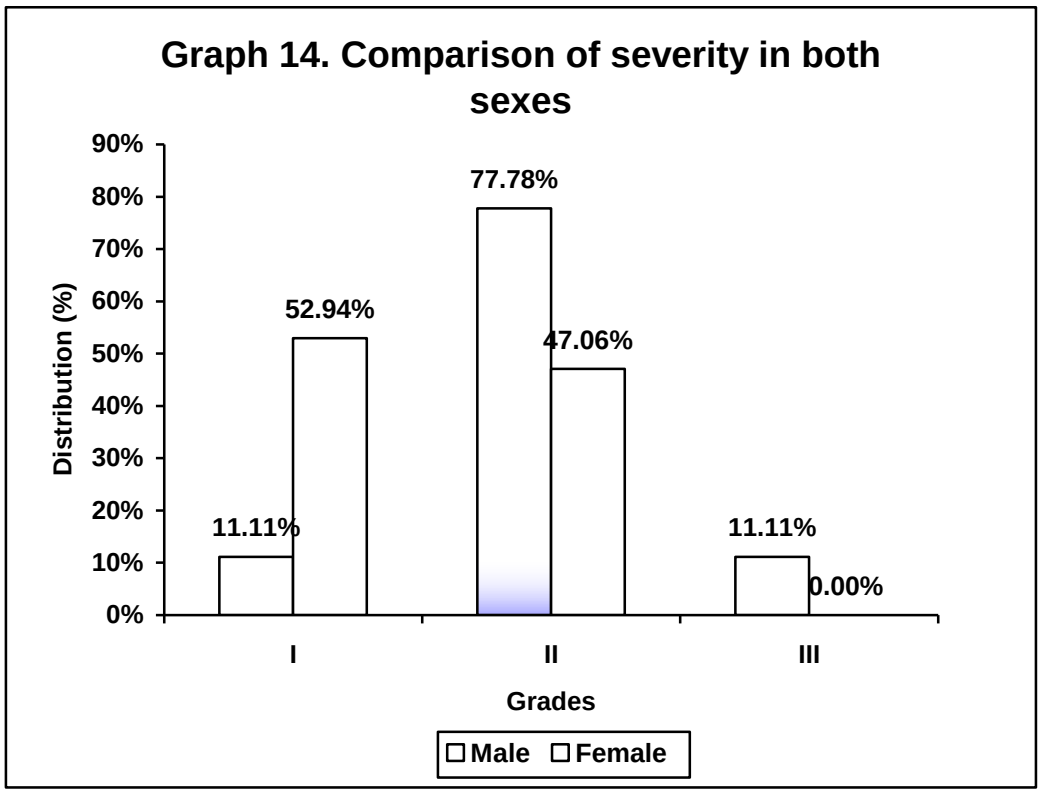


It was observed that out of 19 non obese and 16 obese patients most of the patients (73.68% non obese and 50% obese) had grade II severity. However this difference was statistically not significant (p=0.150).

Table 14. Comparison of severity in both sexes

Severity	Male (n=18)		Female (n=17)	
	Number	**Percentage**	**Number**	**Percentage**
I	2	11.11	9	52.94
II	14	77.78	8	47.06
III	2	11.11	0	0.00
Total	**18**	**100.00**	**17**	**100.00**

x^2_1=7.148 p=0.008



It was seen that, out of 18 males 77.78% had grade II severity and of the 17 females 52.94% had grade I severity suggesting male gender significantly influences severity of the cholecystitis (p=0.008).

Table 15. Comparison of severity with BMI and sex

Grades	Male (n=18)				Female (n=17)			
	BMI<25 (n=12)		BMI>25 (n=6)		BMI<25 (n=7)		BMI>24 (n=10)	
	No	**%**	**No**	**%**	**No**	**%**	**No**	**%**
I	1	8.33	1	16.6	3	42.8	6	60.00
II	10	83.33	4	66.6	4	57.1	4	40.00
III	1	8.33	1	16.6	0	16.67	0	0.00
Total	**12**	**100.00**	**6**	**100.00**	**7**	**100.00**	**10**	**100.00**
	p=1.000 (Fisher's exact test)				p=0.637 (Fisher's exact test)			

Among 12 non obese male patients, majority (83.33%) had grade II severity followed by 8.33% each with grade I and III. Among 6 obese male patients 66.67% had grade II severity whereas 16.67% each had grade I and III severity respectively. This difference between obese and non-obese males was statistically not significant (p=1.000).

In 7 non obese female patients most (57.14%) of them had grade II severity and 42.87% had grade I severity. In 10 female obese patients 60% had grade I and 40% had grade II. No female patients had grade III. This difference between obese and non-obese females was not significant (p=0.637).

DISCUSSION

Cholecystitis is one of the most common reasons for hospital admission with abdominal pain. Approximately 90–95% of acute cholecystitis is related to gallstones, with 5 to 10% of cases due to acalculous disease.

About 10% to 15% of the adult western population has gallstones. Between 1% and 4% become symptomatic in a year. More than half a million cholecystectomies are performed per year in the United States alone. Regional differences exist in the cholecystectomy rates. Laparoscopic cholecystectomy, which was introduced in 1987, is now the preferred method of cholecystectomy.

Risk factors for cholecystitis mirror those for cholelithiasis and include increasing age, female sex, certain ethnic groups, obesity or rapid weight loss, drugs, and pregnancy.

Obesity is an established risk factor for gallstones. The positive relation between body mass index (BMI) and risk of gallstones have been shown in several epidemiologic studies.[3,9]

An acute reduction of body weight also predisposes a person to cholelithiasis. But the cause of the high incidence of cholelithiasis in person who have undergone rapid weight loss has still not been clearly elucidated. Hence body weight may influence pathogenesis of gallstones in different ways.[5,6]

On the other hand, male sex has recently been cited as a risk factor for severe symptomatic cholelithiasis. It has also been reported that male sex is an

independent predictor for more severe acute cholecystitis. However the cause of severity of cholecystitis in males has not been revealed.[7,8]

There may be many possible causes of this sexual difference with reference to severity of cholecystitis in male but these causes have not been revealed yet. Mean total body fat which is higher in females than males can be a possible cause for this sex difference.

Numerous studies have shown that laparoscopic cholecystectomy in obese patients was not significantly more difficult and it was not associated with increased morbidity.[74,75]

Hence the present study was undertaken to evaluate the relationship between body mass index with severity of cholecystitis based on clinical, imaging, intraoperative and HPR findings in order to help for better anticipation of complications during surgery and the need for conversion from laparoscopy to open cholecystectomy and in counselling the patient and relatives prior to surgery.

The present one year cross sectional study was conducted on 120 patients admitted with cholecystitis in the wards of Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2010 to December 2010. Patients were examined for height and weight and their Body Mass Index was calculated. Based on the BMI, patients were divided into two groups, patients with BMI between 18 to 25 kg/m^2 as non-obese group and more than 25 kg/m^2 as obese group.

In the present study females outnumbered males (60% vs 40%) with male to female ratio of 1:1.5. The most common age group was 30 to 60 years (68.3%) with mean age of 49.31 ± 13.57 years with range being 18 to 78 years.

Similar observations were reported in a study from Seoul, Korea. The study reported male to female ratio as 1:1.4 and mean age as 52±16.1 years.[4] This could be probably due to the fact that, female patients have higher incidence of cholelithiasis.[3]

Of the 120 enrolled patients, 62.5% patients were in non obese group and 37.5% patients in obese group. The number of patients in non-obese group were higher than in the obese group. There was no statistical difference in age and sex distribution between obese and non-obese group.

Similar findings were observed in a study with 61.1% in non-obese group and 38.9% in obese group.[4]

Of 48 males 60.41% were in non-obese group and 39.5% were in obese group and of 72 females 63.8% were non obese and 36.11% were obese. The male to female ratio in non obese group was 1:1.58 and in obese group it was 1:1.36. However there was no difference between the obese and non-obese group in terms of male to female ratio.

In this study most of the patients had white blood cell count distribution between 6000-11000 cells/mm^3 (86.67%). In 8 (6.67%) patients white blood cell count was more than 18000 cells/mm^3. These patients also had complicated acute

cholecystitis and were categorized into grade II severity as per Tokyo guidelines.[75]

Majority of the patients enrolled in this study had a normal creatinine level except for two patients who had a creatinine level of more than 2mg/dl.This was due to renal dysfunction secondary to complicated acute cholecystitis and were included in grade III severity.

In this study of 75 non-obese patients 10.66% had history of diabetes mellitus and 2.66% had cardiovascular disease. Of the 45 obese patients, 13.33% had diabetes mellitus and 6.66% had cardiovascular disease. In a similar study, among non obese, 7.9% had diabetes mellitus and 14.9% had cardiovascular disease and in obese patients 8.8% had diabetes mellitus and 24% had cardiovascular disease.[4]

Of 120 enrolled patients in this study 70.83% patients had chronic cholecystitis and 29.17% patients had acute cholecystitis. Type of cholecystitis was confirmed by histopathological findings.

In a similar study 54.8% had chronic cholecystitis and 45.2% acute cholecystitis.[4]

In this study, of the 75 non-obese patients, 74.6% had chronic cholecystitis and 25.3% had acute cholecystitis and of the 45 obese patients 64.4% had chronic cholecystitis and 35.5% had acute cholecystitis. However there was no statistical significance in distribution acute and chronic cholecystitis in obese and non-obese patients (p=0.101).

In a similar study where in non-obese patients 53.9% had chronic cholecystitis 46% had acute cholecystitis and in obese patients 56.2% had chronic cholecystitis and 43.8% had acute cholecystitis.[4]

In this study, of the 48 males, 30 (62.5%) had chronic cholecystitis and 18 (37.5%) had acute cholecystitis and of 72 females 55 (76.3%) had chronic cholecystitis and 17 (23.6%) had acute cholecystitis. Even though proportion of male patients having acute cholecystitis was higher when compared with females this was not statistically significant (p=0.101). These findings were contradictory to a study which reported proportion of male patients having acute cholecystitis was 52.8%, 47.2% had chronic cholecystitis and in females 39% had acute cholecystitis and 61% had chronic cholecystitis.[4]

In this study based on the Diagnostic criteria and severity assessment of acute cholecystitis: Tokyo Guidelines[75] acute cholecystitis was graded into three grades. Out of 35 patients 31.4% had grade I, 62.8% grade II and 5.71% grade III severity of acute cholecystitis. In a similar study complicated acute cholecystitis was seen in 26% and uncomplicated acute cholecystitis was seen in 73%, however there no grading system used and acute cholecystitis was graded as chronic cholecystitis, complicated and uncomplicated acute cholecystitis.[4]

As per Tokyo guidelines[75] we can infer that grade I indicates an uncomplicated acute cholecystitis where as grade II indicates a complicated acute cholecytitis and grade III indicates a complicated acute cholecystitis with any organ dysfunction. In this study more patients had grade II cholecystitis i.e about 62.8%, where as it was 31.4% in grade I, grade III accounted for 5.71%. The

increased number of complicated cholecystitis than uncomplicated cholecystitis in our study may be due the patients referred to our hospital as a tertiary care centre. Also in our study we only included patients who underwent surgery for cholecystitis, this was because type of cholecystitis whether acute or chronic was finally confirmed by histopathology. Hence this might be the other reason for more patients of complicated cholecystitis than uncomplicated as they are more likely to undergo surgery.

In this study 54.2% cases had pericholecystic collection, 22.8% had local adhesion between gallbladder and adjacent organs, 11.4% cases had perforated gall bladder, 5.7% had empyema gall bladder and 2.8% each had gangrenous cholecystitis and hepatic abscess. Other cases had a contracted gallbladder with cholelitheasis. These findings were confirmed by imaging and intraoperative findings. Other studies have not mentioned about the imaging and intraoperative findings.[4]

In this study severity of cholecystitis was compared between obese and non-obese group. It was observed that of 11 patients with grade I severity 36.3% were non obese and 63.7% were obese. In 22 patients with grade II, 63.6% were non-obese and 36.4% were obese. In grade III, of the 2 patients 50% each were in obese and non obese group. This difference of severity of cholecystitis between obese and non obese group was statistically not significant (p=0.150).

Further evaluation of difference of severity of cholecystitis in male and females based on body mass index was done. Among 12 non obese male patients, majority (83.33%) had grade II severity followed by 8.33% each with grade I and

III. Among 6 obese male patients 66.67% had grade II severity whereas 16.67% each had grade I and III severity respectively. This difference between obese and non-obese males was statistically not significant (p=1.000).

In 7 non obese female patients most (57.14%) of them had grade II severity and 42.87% had grade I severity. In 10 female obese patients 60% had grade I and 40% had grade II. No female patients had grade III. This difference between obese and non-obese females was statistically not significant (p=0.637).

A similar study in males also showed, there was significant negative correlation between the BMI and severity of cholecystitis, the proportion of complicated acute cholecystitis was higher in the non-obese male patients compared to obese male patients but this was not significant in female patients.[4] However in this study there was no significant correlation found between BMI and severity of cholecystitis.

In this study severity of cholecystitis was also compared between male and female patients. It was seen that, of the 18 male patients, 77.78% had grade II severity and 11.11% each had grade I and III. Among the 17 females, 52.94% had grade I severity and 47.06% had grade II. However no female patient was seen with grade III. This difference between severity of cholecystitis among male and female was statistically significant (p=0.008) suggesting males had a more severe cholecystitis when compared to females. These findings were similar to other studies were it was seen that male presenting with cholecystitis were more likely to have a severe disease.[7,8]

Limitations of the present study were smaller sample size and conversion rate of laparoscopy to open cholecystectomy could not be assessed.

Further studies with larger sample size would help in better understanding of pathogensis of cholecystitis and role of body fat in inflammation in cholecystitis and its influence on severity.

CONCLUSION

The findings of the present study showed that, overall there was no influence of body mass index over severity of cholecytitis based on clinical, imaging, intraoperative and HPR findings as well as either in males or females separately. The result of this study also showed that males had severe form of cholecystitis when compared to females.

SUMMARY

More than half a million cholecystectomies are performed per year in the United States alone. Regional differences exist in the cholecystectomy rates. Risk factors for cholecystitis mirror those for cholelithiasis and include increasing age, female sex, certain ethnic groups, obesity or rapid weight loss, drugs, and pregnancy. The present study was undertaken to evaluate the relationship between body mass index with severity of cholecystitis based on clinical, imaging, intraoperative and HPR findings.

The present one year cross sectional study was conducted on 100 patients admitted with cholecystitis in the wards of Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2010 to December 2010. Patients were examined for height and weight and their Body Mass Index. Based on the BMI, patients were divided into groups that is, patients with BMI between 18 to 25 kg/m^2 as non-obese group and more than 25 kg/m^2 as obese group. Based on the clinical examination, blood investigations, ultrasonography, and histopathology the severity of disease was categorized chronic cholecystitis and acute cholecystitis. Acute cholecystitis was categorized into three categories based on Tokyo guidelines on acute cholecystitis.

In this study females outnumbered males with male to female ratio of 1:1.5. Most of the patients (68.3%) were aged between 30 years to 60 years and the mean age was 49.31 ± 13.57 years. Of 120 enrolled patients 62.5% patients were in non obese group and 37.5% patients in obese group. The mean BMI was

24.35 ± 2.96 Kg/m^2. In this study 70.83% patients had chronic cholecystitis and 29.17% patients had acute cholecystitis. Of 75 non-obese patients 74.6% had chronic cholecystitis and 25.3% had acute cholecystitis and of 45 obese patients 64.4% had chronic cholecystitis and 35.5% had acute cholecystitis. In our study out of 35 patients 31.4% had grade I, 62.8% grade II and 5.71% grade III severity of aute cholecystitis.

It was observed that out of 11 grade I severity patients 36.3% were in non obese and 63.7% were in obese group and of 22 patients in grade II, 63.6% were non-obese and 36.4% in obese group. In grade III of 2 patients 50% patient were in each group showing no statistically significant difference in severity of cholecystitis between obese and non-obese patients.

It was seen that of 11 patients with grade I of 2 were males and 9 females and of 22 patients in grade II 14 were males and 8 females. In grade III of 2 patients both were males showing statistically significant influence of male gender with severity of cholecystitis.

Overall, there was no influence of body mass index over severity of cholecytitis based on clinical, imaging, intraoperative and HPR findings as well as either in males or females separately. The result of this study also showed that males had severe form of cholecystitis when compared to females.

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ANNEXURE I – CONSENT FORM

Title of Research Study: "A ONE YEAR CROSS-SECTIONAL STUDY TO EVALUATE THE RELATIONSHIP BETWEEN BODY MASS INDEX AND SEVERITY OF CHOLECYSTITIS"

Principal Investigator

Dr. CHETAN HOSKATTI
Post Graduate Student,
Department Of Surgery,
J.N.Medical College, Belgaum.

Introduction and purpose

A One Year Cross-Sectional Study To Evaluate The Relationship Between Body Mass Index And Severity Of Cholecystitis is being conducted by Dr. Chetan Hoskatti, post graduate student in surgery under the guidance of Dr. M. S. Sangolli, Professor of Surgery, J. N. Medical College, Belgaum, under KLE University, Belgaum.

Respected we request u to participate in our study as you are eligible to be included. Your participation in this study is voluntary. Your decision whether or not to participate in the study will not affect your relationship with J.N.M.C. If you decide to participate you are free to withdraw at any point of time. The purpose of my study is evaluate the relationship between body mass index and severity of cholecystitis

Procedure

If you agree to enroll yourself in my study you will be measured for height and weight and then Body Mass Index will be calculated by using the formula Weight in kilograms divided by square of Height in meters. You will be asked for relevant history and examination findings noted. Investigations done will be noted. After surgery intra-operative and Histopathological study of gall bladder specimen will be noted. Based on these severity of the disease will be graded.

Benefits and risks

There are no risks or benefits by taking part in the study except for helping us in doing research.

Voluntary participation/withdrawal

I Mr./Ms._____ have been explained about the research study, the need of the study, the diagnostic intervention, their risks, benefits and alternatives available in my own vernacular language.

Taking part in this study is voluntary. I may choose not to take part in the study, or withdraw from the study anytime later. My decisions will not change the present or future health care or any service I receive. The study doctor or sponsor may stop my participation in the study without any consent. While taking part in the study I will be told of any important new findings that may change my willingness to continue or take part. If I choose not to take part in the study I will receive the standard treatment for patients with my conditions.

Cost

Nil

Compensation

In the event that I become injured as a result of taking part in this study, treatment will be offered to me or I will be given information about where to receive medical care: but my insurance company or I will be responsible for the costs. No reimbursement, compensation or free medical care will be given.

Confidentiality

All information collected about me during the course of the study will be kept confidential to the extent, permitted by law. I will be identified in research records by a code number. Information of the study may be published but my identity will be kept confidential in any publication.

Consent to participate in the study

I voluntarily agree to participate in this study by signing up this form below. I may withdraw at any time from this study. I am not giving any of my legal rights by signing up this form. My signature / thumb impression below indicates that I have read or information in the consent been read to me including the risks and benefits and have cleared my doubts. I will be given a copy of this consent form.

In case of any queries, you can contact the following:

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J. N. Medical College,
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(9620153262)

Signature of the study patient:

Name of the study patient:

Signature of the legally authorized representative:

Relationship to the patient:

Signature of the witness:

Signature of the investigator:

Date:

ANNEXURE II – PROFORMA

Title: "A ONE YEAR CROSS-SECTIONAL STUDY TO EVALUATE THE RELATIONSHIP BETWEEN BODY MASS INDEX AND SEVERITY OF CHOLECYSTITIS"

Name : Age:

Sex : IP No.:

Education : Religion:

Marital Status : Occupation:

Date of admission: Date of discharge:

HISTORY :

Complaints : < 72 hours / > 72 hours

Comorbid disease : Cardiovascular disease ( )

Diabetes ( )

GENERAL PHYSICAL EXAMINATION:

Built and Nourishment :

Weight : _____ kilograms

Hieght : _____ meters

BMI : _____ kg/mt^2

Pallor / Icterus / Cyanosis / Clubbing / Edema / Lymphadenopathy :

Vital Signs : PR:

BP:

RR:

Temp:

SYSTEMIC EXAMINATION:

Per Abdomen:

Respiratory System:

Central Nervous System:

Cardio-Vascular System

INVESTIGATION:

Hb: USG:

WBC:

Bl. Urea

Sr. Creatinine:

INTRA- OPERATIVE FINDINGS :

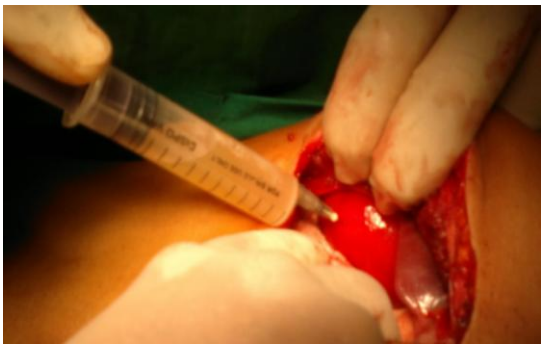
Type of operation: open/ laparoscopic Cholecystectomy

- Pericholecystic collection ( )
- Empyema of Gall bladder ( )
- Gangrene of Gall bladder ( )
- Perforation of Gall bladder ( )
- Severe adhesion to an adjacent organ ( )

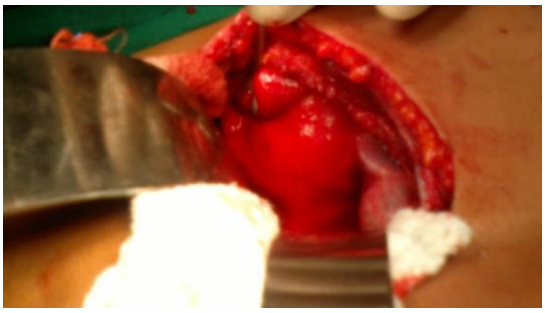
GRADE OF SEVERITY:

HPR FINDINGS OF GALLBLADDER SPECIMEN:

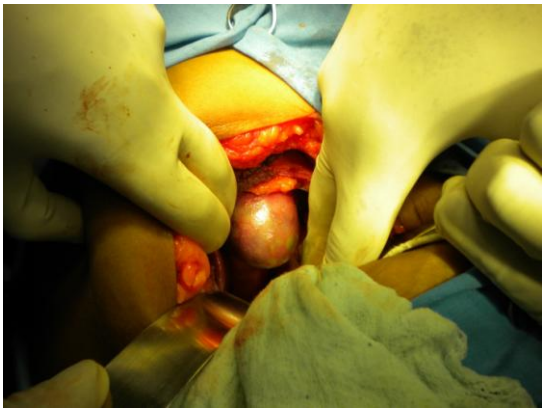
ANNEXURE III – PHOTOGRAPHS



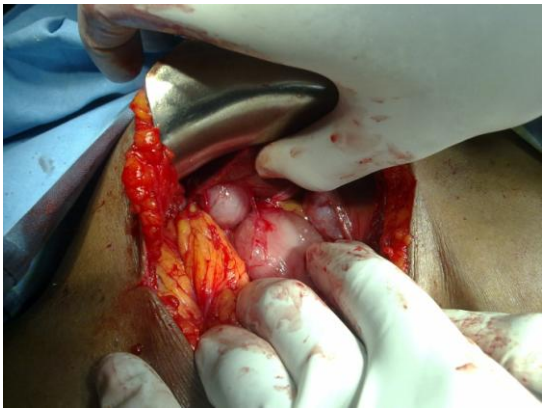
Photograph 1. Empyema gallbladder



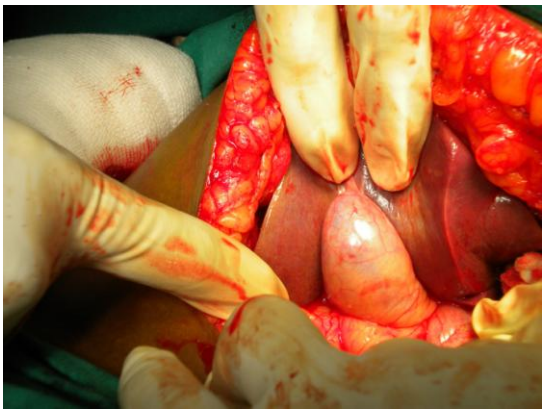
Photograph 2. Acute cholecytitis



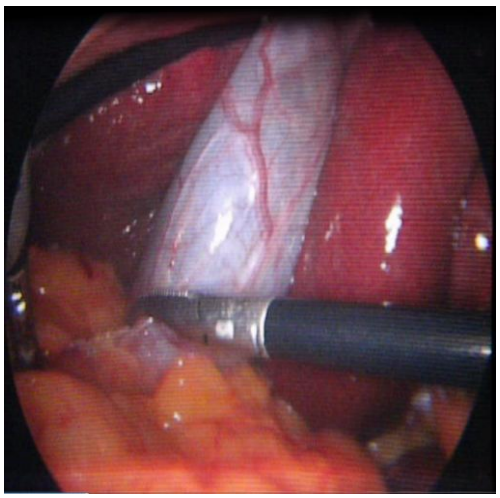
Photograph 3. Gangrenous cholecystitis



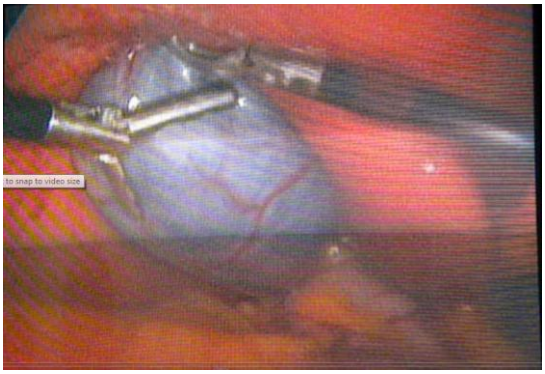
Photograph 4. Acute cholecystitis with severe adhesion to adjacent organs



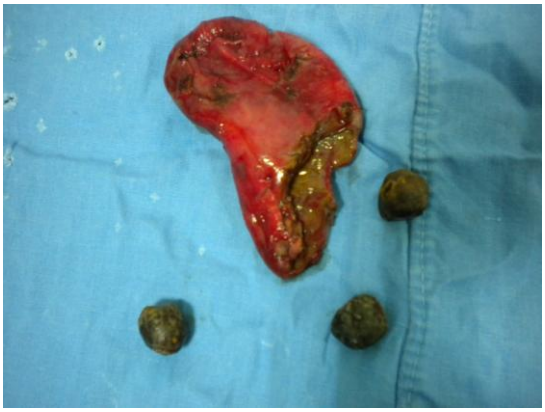
Photograph 5. Acute cholecystitis with pericholecystitic collection



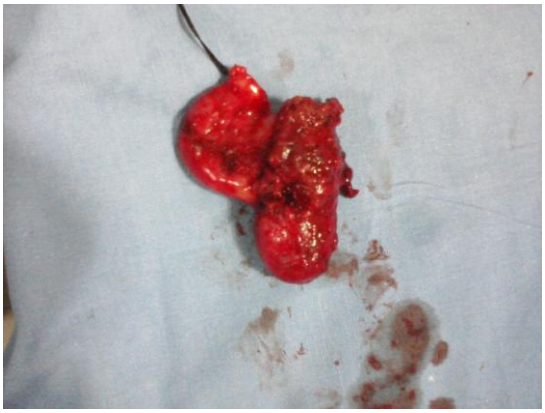
Photograph 6. Chronic cholecystitis



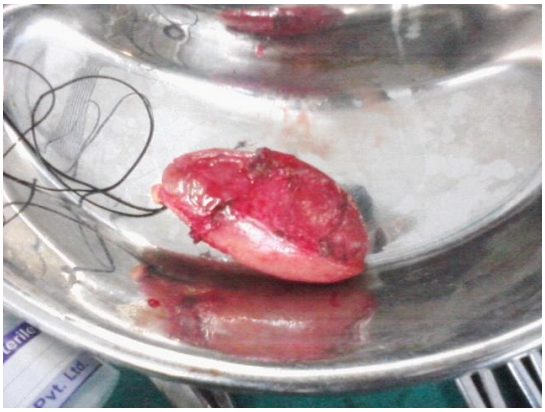
Photograph 7. Chronic cholecystitis



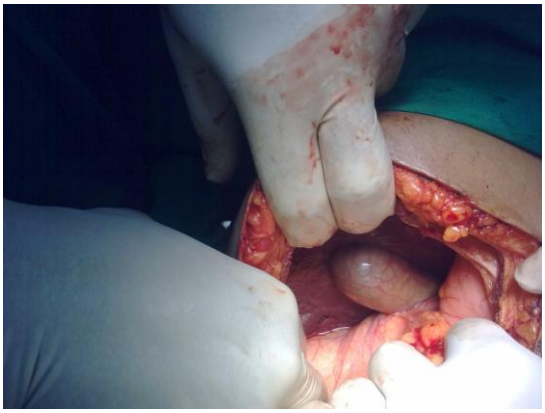
Photograph 8. Specimen of perforated gall bladder



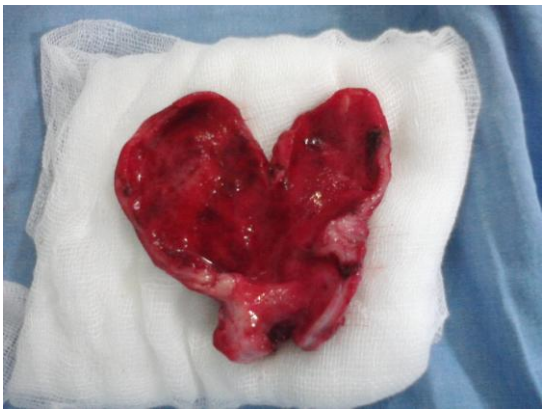
Photograph 9. Perforated gall bladder specimen



Photograph 10. Empyema gallbladder



Photograph 11. Chronic calculous cholecystitis



Photograph 12. Specimen of acute cholecystitis with thickened wall

ANNEXURE IV - MASTER CHART

Serial Number	In Patient Number	Sex	Age (Years)	Duration of symptoms (> 72 hrs	History	General Physical Examination						Systemic examination				Investigatons				Imaging findings	Severity	Intra operative findings	HPR findings
									Vitals														
										BP (mm Hg)													
						Weight (Kg)	Height (Cms)	BMI (Kg//m2)	Pulse rate	SBP	DBP	Per abdomen	Respiratory System	Central nervous system	Cardiovascular system	WBC (/mm3)	Blood urea (mg/dL)	Sr. Creat (mg/dL)	PT/INR				
1	348757	f	61	NA	P+D	68	178	21.46	74	130	80	C	N	N	N	8000	32	0.80	1.20	CCC	-	C	CC
2	349317	M	59	+	P+DM	66	174	21.80	82	150	70	A	N	N	N	18500	40	0.70	0.90	AC	II	LA	AC
3	355638	f	47	-	DM, CVD	69	150	30.67	85	140	80	A	N	N	N	12000	30	0.60	0.80	AC	I	PC	AC
4	353860	M	40	NA	P	67	167	24.02	76	130	90	C	N	N	N	10500	40	0.50	0.80	CCC	-	C	CC
5	35505	f	46	NA	DM	69	171	23.60	79	130	90	C	N	N	N	10500	42	0.80	0.50	CCC	-	C	CC
6	352727	f	52	NA	P+F	51	156	20.96	92	160	100	A	N	N	N	12000	45	1.30	0.60	AC	I	PC	AC
7	345555	f	70	NA	P	51	154	21.50	82	140	90	C	N	N	N	9800	40	1.10	0.80	CCC	-	C	CC
8	346967	M	50	NA	DM	67	170	23.18	86	140	90	C	N	N	N	10200	48	0.70	1.10	CCC	-	C	CC
9	355809	f	37	NA	DM	70	160	27.34	78	130	90	C	N	N	N	10300	46	0.60	0.90	CCC	-	C	CC
10	316148	M	52	+	P+F	85	175	27.76	89	150	95	A	N	N	N	19000	32	1.10	0.50	AC	II	LA	AC
11	355613	f	60	NA	P	60	166	21.77	95	160	100	C	N	N	N	9900	39	0.40	0.80	CCC	-	C	CC
12	353070	f	48	NA	P	61	168	21.61	76	130	90	C	N	N	N	10400	41	1.20	0.75	CCC	-	C	CC
13	355886	M	43	NA	P	71	170	24.57	94	160	100	C	N	N	N	8500	46	1.30	0.60	CCC	-	C	CC
14	352293	f	39	NA	P+DM	56	159	22.15	90	145	95	C	N	N	N	9600	45	1.10	0.95	CCC	-	C	CC
15	352281	f	42	-	P+F+V	70	163	26.35	100	130	90	A	N	N	N	19000	52	1.00	1.10	AC	II	GP	AC
16	350774	f	78	NA	P	72	164	26.77	82	160	100	C	N	N	N	8900	40	1.20	0.90	CCC	-	C	CC
17	361841	M	45	NA	P	75	168	26.57	85	135	90	C	N	N	N	10500	43	1.40	0.65	CCC	-	C	CC
18	361234	M	48	NA	P	68	170	23.53	76	140	90	C	N	N	N	9200	42	0.80	1.20	CCC	-	C	CC
19	350588	f	57	NA	P	66	170	22.84	79	145	95	C	N	N	N	8800	48	0.90	1.00	CCC	-	C	CC
20	349590	f	55	NA	P	74	166	26.85	92	130	90	C	N	N	N	9200	39	1.20	0.85	CCC	-	C	CC

ANNEXURE IV - MASTER CHART

Serial Number	In Patient Number	Sex	Age (Years)	Duration of symptoms (> 72 hrs	History	General Physical Examination						Systemic examination				Investigatons				Imaging findings	Severity	Intra operative findings	HPR findings
						Weight (Kg)	Height (Cms)	BMI (Kg//m2)	Vitals			Per abdomen	Respiratory System	Central nervous system	Cardiovascular system	WBC (/mm3)	Blood urea (mg/dL)	Sr. Creat (mg/dL)	PT/INR				
									Pulse rate	BP (mm Hg)													
										SBP	DBP												
21	342045	M	65	+	P	75	165	27.55	82	160	100	A	N	N	N	17000	32	0.80	0.90	AC	I	PC	AC
22	361674	f	60	NA	P	59	164	21.94	86	150	95	C	N	N	N	10500	40	0.40	0.65	CCC	-	C	CC
23	341898	f	62	NA	P+D	54	160	21.09	78	130	90	C	N	N	N	9800	30	0.80	1.20	CCC	-	C	CC
24	342919	M	37	-	P+F+DM+CVD	72	175	23.51	89	150	95	A	N	N	N	19400	40	1.30	0.90	AC	II	PC	AC
25	340956	f	70	NA	P+DM	56	165	20.57	95	140	95	C	N	N	N	9900	42	0.90	0.80	CCC	-	C	CC
26	355583	M	64	NA	P	60	168	21.26	76	130	90	C	N	N	N	10650	45	0.70	0.80	CCC	-	C	CC
27	366617	f	59	NA	P	70	161	27.01	82	130	90	C	N	N	N	9850	40	0.60	0.50	CCC	-	C	CC
28	379184	f	40	-	P+F	61	164	22.68	85	160	100	A	N	N	N	19200	48	1.20	0.60	AC+PC	II	EG	AC
29	381717	M	75	NA	P	75	170	25.95	76	140	90	C	N	N	N	10200	46	1.10	0.80	CCC	-	C	CC
30	382518	M	48	-	P+F+V	65	173	21.72	79	140	90	A	N	N	N	12000	32	1.40	1.10	AC	I	PC	AC
31	355370	f	19	NA	P	54	166	19.60	92	130	90	C	N	N	N	8500	39	1.30	0.90	CCC	-	C	CC
32	358925	f	44	-	P+F	74	165	27.18	82	150	95	A	N	N	N	13000	41	0.40	0.50	AC	I	PC	AC
33	364028	M	58	NA	P	66	165	24.24	86	160	100	C	N	N	N	10200	46	1.20	0.80	CCC	-	C	CC
34	363290	f	55	NA	P	56	163	21.08	78	130	90	C	N	N	N	10800	45	1.00	0.75	CCC	-	C	CC
35	356820	M	30	NA	P	80	172	27.04	89	160	100	C	N	N	N	9600	52	0.80	0.60	CCC	-	C	CC
36	363888	f	66	NA	P	65	158	26.04	95	145	90	C	N	N	N	9300	40	1.50	0.95	CCC	-	C	CC
37	359422	M	55	NA	P	65	175	21.22	76	130	90	C	N	N	N	10200	43	1.40	1.10	CCC	-	C	CC
38	377700	f	18	NA	P	61	168	21.61	94	160	95	C	N	N	N	9100	42	1.20	0.90	CCC	-	C	CC
39	379908	f	36	+	P+V+DM	63	145	29.96	90	135	90	A	N	N	N	20000	48	0.70	0.65	AC	II	PC	AC
40	380965	f	36	NA	CVD	56	160	21.88	85	140	90	C	N	N	N	9600	39	0.60	1.20	CCC	-	C	CC

ANNEXURE IV - MASTER CHART

Serial Number	In Patient Number	Sex	Age (Years)	Duration of symptoms (> 72 hrs	History	General Physical Examination						Systemic examination				Investigatons				Imaging findings	Severity	Intra operative findings	HPR findings
						Weight (Kg)	Height (Cms)	BMI (Kg//m2)	Vitals			Per abdomen	Respiratory System	Central nervous system	Cardiovascular system	WBC (/mm3)	Blood urea (mg/dL)	Sr. Creat (mg/dL)	PT/INR				
									Pulse rate	BP (mm Hg)													
										SBP	DBP												
41	360512	M	18	-	P	66	169	23.11	82	145	95	A	N	N	N	21000	32	1.30	1.00	AC	II	LA	AC
42	379029	f	35	NA	P+F	58	162	22.10	85	130	90	C	N	N	N	10500	40	0.40	0.85	CCC	-	C	CC
43	382425	f	26	NA	P	56	160	21.88	76	160	95	C	N	N	N	8900	30	1.20	0.90	CCC	-	C	CC
44	264103	M	52	-	P+F+DM	95	176	30.67	79	150	95	A	N	N	N	20100	40	1.40	0.65	AC	II	LA	AC
45	397741	f	22	NA	P	54	160	21.09	92	130	90	C	N	N	N	10600	42	1.10	1.20	CCC	-	C	CC
46	367650	M	29	NA	P	85	176	27.44	82	150	95	C	N	N	N	8900	45	1.00	0.90	CCC	-	C	CC
47	372940	f	40	NA	P	56	160	21.88	86	140	90	C	N	N	N	10200	40	0.80	0.80	CCC	-	C	CC
48	376678	f	37	-	P+F	74	160	28.91	78	130	90	A	N	N	N	19500	48	1.30	0.80	AC	II	LA	AC
49	358131	f	54	NA	P	61	161	23.53	89	130	90	C	N	N	N	10600	46	0.70	0.50	CCC	-	C	CC
50	372659	M	29	NA	P	86	174	28.41	95	160	100	C	N	N	N	9800	32	1.20	0.60	CCC	-	C	CC
51	358897	f	62	NA	P	66	168	23.38	76	140	90	C	N	N	N	10200	39	0.40	0.80	CCC	-	C	CC
52	360605	M	55	+	P+F	65	169	22.76	94	140	90	MA	N	N	N	10900	41	0.60	1.10	AC	II	LA	AC
53	367283	f	65	NA	DM	65	170	22.49	90	130	90	C	N	N	N	10600	46	0.80	0.90	CCC	-	C	CC
54	353769	M	50	NA	P	66	173	22.05	88	150	95	C	N	N	N	10100	45	1.10	0.50	CCC	-	C	CC
55	367650	f	51	NA	P+F	68	159	26.90	83	160	95	A	N	N	N	9900	52	1.20	0.80	AC	I	PC	AC
56	356767	M	61	-	P+F+V	56	168	19.84	120	92	58	A	N	N	N	19500	40	3.30	1.70	AC+empyema	III	EG	AC
57	368299	f	55	NA	P	65	164	24.17	82	160	100	C	N	N	N	10500	43	1.30	0.60	CCC	-	C	CC
58	368121	M	36	NA	P	69	174	22.79	85	145	95	C	N	N	N	8500	42	1.20	0.95	CCC	-	C	CC
59	369528	f	73	+	P+F	59	162	22.48	76	130	90	A	N	N	N	13800	48	0.80	1.10	AC	I	PC	AC
60	366452	M	63	-	P	75	168	26.57	79	160	100	A	N	N	N	19000	39	0.90	0.90	AC	II	LA	AC

ANNEXURE IV - MASTER CHART

Serial Number	In Patient Number	Sex	Age (Years)	Duration of symptoms (> 72 hrs	History	General Physical Examination						Systemic examination				Investigatons				Imaging findings	Severity	Intra operative findings	HPR findings
						Weight (Kg)	Height (Cms)	BMI (Kg//m2)	Vitals			Per abdomen	Respiratory System	Central nervous system	Cardiovascular system	WBC (/mm3)	Blood urea (mg/dL)	Sr. Creat (mg/dL)	PT/INR				
									Pulse rate	BP (mm Hg)													
										SBP	DBP												
61	369224	f	56	NA	P+DM	56	159	22.15	92	135	90	C	N	N	N	8900	32	1.30	0.65	CCC	-	C	CC
62	368512	M	24	NA	P	65	170	22.49	82	140	90	C	N	N	N	10900	40	0.90	1.20	CCC	-	C	CC
63	371412	M	59	+	P+F	61	164	22.68	86	145	95	A	N	N	N	9100	30	1.30	1.00	AC+HA	II	HA	AC
64	369215	f	46	NA	P	75	166	27.22	78	130	90	C	N	N	N	10300	40	1.20	0.85	CCC	-	C	CC
65	369992	f	40	+	P+F	68	161	26.23	89	160	95	A	N	N	N	16000	42	1.10	0.90	AC	I	PC	AC
66	371452	M	26	NA	P	96	176	30.99	95	150	95	C	N	N	N	10100	45	1.00	0.65	CCC	-	C	CC
67	370114	f	28	NA	P	61	166	22.14	76	130	90	C	N	N	N	10200	40	1.20	1.20	CCC	-	C	CC
68	371421	M	55	NA	P	75	166	27.22	94	150	95	C	N	N	N	9800	48	1.00	0.90	CCC	-	C	CC
69	374606	f	41	NA	P	80	165	29.38	90	140	90	C	N	N	N	8600	46	0.80	0.80	CCC	-	C	CC
70	368422	M	55	+	P+F	63	168	22.32	81	130	90	MA	N	N	N	14400	32	1.50	0.80	AC	II	PC	AC
71	276508	f	45	-	P	64	163	24.09	85	130	90	A	N	N	N	10600	39	1.40	0.50	AC	I	PC	AC
72	374553	f	40	NA	P	62	168	21.97	84	160	100	C	N	N	N	8200	41	1.20	0.60	CCC	-	C	CC
73	365421	M	55	NA	P	64	170	22.15	89	140	90	C	N	N	N	10100	46	1.30	0.80	CCC	-	C	CC
74	364428	M	60	NA	P	61	164	22.68	82	140	90	C	N	N	N	10900	45	1.20	1.10	CCC	-	C	CC
75	370794	f	71	NA	P	62	155	25.81	85	130	90	C	N	N	N	10500	52	0.90	0.90	CCC	-	C	CC
76	371452	M	55	-	P+F	98	176	31.64	76	90	60	A	N	N	N	18000	40	2.30	1.80	AC+ perforation	III	GP	AC
77	377954	f	60	NA	P	56	159	22.15	79	160	100	C	N	N	N	10100	43	1.20	0.80	CCC	-	C	CC
78	369452	M	54	-	P	74	176	23.89	92	130	90	A	N	N	N	8900	42	1.00	0.75	AC+collection	II	GP	AC
79	360011	f	32	NA	P	52	164	19.33	82	160	100	C	N	N	N	10800	48	0.80	0.60	CCC	-	C	CC
80	344879	f	35	NA	P+DM	70	160	27.34	86	145	95	C	N	N	N	8200	39	1.50	0.95	CCC	-	C	CC

ANNEXURE IV - MASTER CHART

Serial Number	In Patient Number	Sex	Age (Years)	Duration of symptoms (> 72 hrs	History	General Physical Examination						Systemic examination				Investigatons				Imaging findings	Severity	Intra operative findings	HPR findings
									Vitals														
										BP (mm Hg)													
						Weight (Kg)	Height (Cms)	BMI (Kg//m2)	Pulse rate	SBP	DBP	Per abdomen	Respiratory System	Central nervous system	Cardiovascular system	WBC (/mm3)	Blood urea (mg/dL)	Sr. Creat (mg/dL)	PT/INR				
81	368452	M	42	NA	P	70	164	26.03	78	130	90	C	N	N	N	9600	32	1.40	1.10	CCC	-	C	CC
82	356653	f	48	+	P+F	64	156	26.30	89	160	95	A	N	N	N	13000	40	1.20	0.90	AC	I	PC	AC
83	363534	f	45	NA	P	51	152	22.07	95	135	90	C	N	N	N	10200	30	1.50	0.65	CCC	-	C	CC
84	370794	M	38	NA	P	76	178	23.99	76	140	90	C	N	N	N	10500	40	1.40	1.20	CCC	-	C	CC
85	377954	f	41	NA	P	72	162	27.43	82	145	95	C	N	N	N	8800	42	1.20	1.00	CCC	-	C	CC
86	369415	M	46	NA	P	74	168	26.22	85	130	90	C	N	N	N	10900	45	1.30	0.85	CCC	-	C	CC
87	360014	f	40	NA	P	56	162	21.34	76	160	100	C	N	N	N	9100	40	1.20	0.90	CCC	-	C	CC
88	374600	f	38	-	P+F+CVD	62	155	25.81	79	150	95	MA	N	N	N	13000	48	0.90	0.65	AC	II	PC	AC
89	376508	f	68	NA	P	75	163	28.23	92	145	95	C	N	N	N	10100	46	1.00	1.20	CCC	-	C	CC
90	374553	f	70	NA	P	58	165	21.30	82	130	90	C	N	N	N	9200	32	1.10	0.90	CCC	-	C	CC
91	370794	M	71	NA	P	70	164	26.03	86	160	100	C	N	N	N	10800	39	1.20	0.80	CCC	-	C	CC
92	370786	M	68	NA	P+F	74	176	23.89	78	135	90	MA	N	N	N	12800	41	1.00	0.80	AC	II	PC	AC
93	364018	M	35	NA	P	74	167	26.53	89	140	90	C	N	N	N	10600	46	0.90	0.50	CCC	-	C	CC
94	377954	f	40	NA	P	58	168	20.55	95	145	95	C	N	N	N	10300	45	0.70	0.60	CCC	-	C	CC
95	360011	f	45	NA	P	69	163	25.97	76	130	90	A	N	N	N	14500	52	1.30	0.80	AC	I	PC	AC
96	344874	f	43	NA	P	88	162	33.53	94	160	95	C	N	N	N	10100	40	1.20	1.10	CCC	-	C	CC
97	356635	f	38	NA	P	60	166	21.77	90	150	95	C	N	N	N	10500	43	0.80	0.90	CCC	-	C	CC
98	365421	M	36	NA	P	89	171	30.44	88	150	95	C	N	N	N	10800	42	1.20	0.50	CCC	-	C	CC
99	363564	f	29	-	P+F	59	160	23.05	83	130	90	A	N	N	N	10100	48	0.40	0.80	AC+gangrene	II	GC	AC
100	363280	f	38	NA	P	65	165	23.88	86	160	95	C	N	N	N	9800	39	1.30	0.75	CCC	-	C	CC

ANNEXURE IV - MASTER CHART

Serial Number	In Patient Number	Sex	Age (Years)	Duration of symptoms (> 72 hrs	History	General Physical Examination						Systemic examination				Investigatons				Imaging findings	Severity	Intra operative findings	HPR findings
						Weight (Kg)	Height (Cms)	BMI (Kg//m2)	Vitals			Per abdomen	Respiratory System	Central nervous system	Cardiovascular system	WBC (/mm3)	Blood urea (mg/dL)	Sr. Creat (mg/dL)	PT/INR				
									Pulse rate	BP (mm Hg)													
										SBP	DBP												
101	365257	M	48	NA	P	66	170	22.84	86	130	90	A	N	N	N	19600	32	0.80	1.20	AC	II	PC	AC
102	364295	f	41	NA	P	61	168	21.61	90	150	95	C	N	N	N	10200	40	1.70	0.90	CCC	-	C	CC
103	364528	f	46	NA	P	80	170	27.68	82	140	90	C	N	N	N	10600	30	0.70	0.80	CCC	-	C	CC
104	364259	f	49	NA	P	68	172	22.99	85	130	90	C	N	N	N	10800	40	0.60	0.80	CCC	-	C	CC
105	364258	M	58	NA	P	66	168	23.38	76	130	90	C	N	N	N	9100	42	0.80	0.50	CCC	-	C	CC
106	370425	f	51	NA	DM	78	166	28.31	79	160	100	C	N	N	N	9700	45	1.10	0.60	CCC	-	C	CC
107	360492	f	60	NA	P	55	159	21.76	92	140	90	A	N	N	N	18650	40	1.30	0.80	AC	II	PC	AC
108	371422	M	65	-	P	85	175	27.76	82	140	95	MA	N	N	N	15000	48	1.10	1.10	AC	II	PC	AC
109	374248	f	68	NA	P	66	169	23.11	86	130	90	C	N	N	N	10300	46	1.00	0.90	CCC	-	C	CC
110	360448	M	56	NA	P	69	171	23.60	78	150	90	C	N	N	N	9900	32	1.00	0.50	CCC	-	C	CC
111	360498	f	65	+	P+F	58	162	22.10	89	160	95	A	N	N	N	17000	39	0.90	0.80	AC+perforation	II	GP	AC
112	371422	M	45	-	P+F	67	170	23.18	95	130	90	A	N	N	N	22000	41	1.10	0.75	AC	II	EG	AC
113	360452	f	68	NA	P	64	170	22.15	76	160	100	C	N	N	N	10800	46	0.90	1.20	CCC	-	C	CC
114	362422	f	61	NA	P	66	159	26.11	94	145	95	C	N	N	N	9100	45	0.80	0.90	CCC	-	C	CC
115	360454	M	44	NA	P	76	166	27.58	90	160	95	C	N	N	N	10200	52	1.10	0.80	CCC	-	C	CC
116	365421	f	52	NA	P+CVD	73	162	27.82	86	130	90	C	N	N	N	8900	40	0.80	0.80	CCC	-	C	CC
117	366421	M	54	NA	P	71	170	24.57	81	160	100	C	N	N	N	10100	43	0.90	0.50	CCC	-	C	CC
118	367452	f	62	NA	P	53	165	19.47	90	145	95	C	N	N	N	10800	42	1.10	0.60	CCC	-	C	CC
119	371421	M	78	NA	P	72	175	23.51	90	145	95	C	N	N	N	10600	48	1.00	0.80	CCC	-	C	CC
120	370491	f	51	NA	P	68	172	22.99	83	160	100	C	N	N	N	10500	39	0.90	1.10	CCC	-	C	CC

ANNEXURE IV – KEY TO MASTER CHART

-	-	Negative
+	-	Positive
A	-	Features of acute cholecystitis
AC	-	Acute cholecystitis findings
C	-	Chronic cholecystitis findings
CC	-	Chronic cholecystitis
CCC	-	Chronic calculous cholecystitis
Cms	-	Centimeter
CVD	-	Cardiovascular disease
D	-	Dyspepsia
DBP	-	Diastolic blood pressure
dL	-	Deci Liter
DM	-	Diabetes mellitus
EG	-	Empyema gallbladder
f	-	Female
F	-	Fever
GC	-	Gangrenenous cholecystitis
GP	-	Gallbladder perforation
HA	-	Hepatic abscess
HPR	-	Histopathology report
hrs	-	Hours
INR	-	International normalized ratio

Kg	-	Kilogram
LA	-	Local adhesions
M	-	Male
m	-	Meter
MA	-	Mass in right upper quadrant
mg	-	Milligram
mm	-	Millimeter
mm Hg	-	Millimeters of mercury
NA	-	Not applicable
P	-	Pain in right upper quadrant
PC	-	Pericholecystic collection
PT	-	Prothrombin time
SBP	-	Systolic blood pressure
V	-	Vomiting
WBC	-	White blood cell