LAPAROSCOPIC CHOLECYSTECTOMY CONVERSION RATE AND ASSOCIATED FACTORS

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Dr Mohd Firdauss bin Osman
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List of Abbreviations

ALP : Alkaline phosphatase

ARF : Acute renal failure

ASA : American Society of Anaesthesiologist

CBD : Common bile duct

CI : Confident interval

Cl^- : Chloride ion

CO_2 : Carbon dioxide

CT : Computed tomography

DCA : Deoxycholic acid

DM : Diabetes mellitus

DTH : Delayed-type hypersensitivity

e.g : example

ERC : Endoscopic retrograde colangiography

ERCP : Endoscopic retrograde colangiopancreaticography

gm : gram

g/dL : gram/ decilitre

HCO_3^- : Bicarbonate ion

HPE : Histopathological examination

HPT : Hypertension

HRPZ II : Hospital Raja Perempuan Zainab II

IHD : Ischaemic heart disease

K^+ : Potassium ion

LC : Laparoscopic cholecystectomy

MC2 : Mouse memory carcinoma

ml : millilitre

mmol/l : milimol/litre

MRCP : Magnetic resonance cholangiopancreatography

MRI : Magnetic resonance Imaging

N : number

Na^+ : Sodium ion

OC : Open cholecystectomy

OD : Odd ratio

PCO_2 : Partial pressure carbon dioxide

SD : Standard deviation

TWBC : Total white blood count

USA : United State of America

Abstrak

Objektif

Untuk mengenal pasti faktor-faktor risiko yang menyebabkan pembedahan laparoskopik diubah kepada pembedahan cholesistektomi terbuka.

Metodologi

Kajian berbentuk retrospektif dari rekod pesakit-pesakit yang telah menjalani pembedahan 'laparoscopic cholecystectomy' dari Januari 2008 hingga Disember 2009 di HRPZ II. Semua faktor risiko pesakit di rekodkan dan di analisis.

Keputusan

Daripada 82 rekod yang di semak, sebanyak 11 pesakit (13.4%) memerlukan penukaran prosedur. Berdasarkan analisis, didapati faktor-faktor risiko seperti umur, jantina, penyakit 'co-morbid', sejarah pembedahan di abdomen adalah tiada kaitan ketara secara statistik. Demam, radang kelenjar pancreas, dan juga keputusan ultrasound yang menunjukkan terdapat penebalan dinding pundi hempedu atau 'pericholecystic fluid' juga tidak mempunyai kaitan yang ketara dari segi statistik. Didapati, faktor risiko seperti cholesistitis akut ((p-value=0.06, adjusted OR=13.365) dan batu di dalam salur hempedu (CBD)(p-value=0.07, adjusted OR=9.278) adalah mempunyai kaitan yang ketara yang menyebabkan kepada penukaran kepada pembedahan cholesistektomi terbuka.

Kesimpulan

Cholesistitis akut dan batu di dalam salur hempedu mempunyai kaitan yang ketara dari segi statistik terhadap penukaran pembedahan 'open cholecystectomy' daripada 'laparoscopic cholecystectomy.'

Abstract

Objective:

To identify risk factor/s for conversion to open cholecystectomy.

Methodology

A retrospective records review was carried out. Records of all patients who underwent laparoscopic cholecystectomy from January 2008 till December 2009 in Hospital Raja Perempuan Zainab II (HRPZ II), Kota Bharu, Kelantan were reviewed and all risk factors were documented. Risks factors for conversion were analysed.

Results

Of 82 records reviewed, 11 (13.4%) require conversion to open cholecystectomy. Age, gender, co-morbidity and history of prior abdominal surgery were not statistically significant to the conversion rate. Fever, pancreatitis and ultrasound findings of thickened gallbladder wall or pericholecystic fluid collection were not associated with significant risk of conversion.

There were significant associations between acute cholecystitis (p-value=0.06, adjusted OR=13.365) and stone in the common bile duct (p-value=0.07, adjusted OR=9.278) with conversion to open cholecystectomy.

Conclusion

Acute cholecystitis and presence of stone in the common bile duct were associated with higher incidence of conversion to open cholecystectomy.

INTRODUCTION

Gall stones have been found in the gallbladders of Egyptian mummies dating back to 1000 b.c. The incidence of gall stones varies throughout the world as a result of ethnic, dietary and poorly understood demographic factors. In USA, autopsy studies performed suggest that gall stones are present in at least 20% of women and 8% of men over 40 years of age. In Europe, rates of cholelithiasis range from 8 to 34 % in women and 5 to 13 % in men.

Gallstones are seldom found in children less than 10 years. The prevalence of gallstones increases with age, highest at 50 to 60 years in both men and women and the male to female ratio is 1.7 to 4:1 (Jazrawi, 2002).

Since the introduction of laparoscopic cholecystectomy in late 1980's, it rapidly and almost completely replaced the open method. Advantages of laparoscopic cholecystectomy over traditional open cholecystectomy include reduced postoperative recovery time, shorter hospitalization, reduced pain, improved cosmesis, and rapid return to normal activities. However, not all cases can be completed laparoscopically and conversion to open cholecystectomy is then required. Conversion rates for both acute and chronic cholecystitis have been reported in many series and range from 2 to 20% with an average of 5%. The most common reasons of conversion are inability to delineate the anatomy secondary to adhesions or inflammation, unexpected operative findings, and iatrogenic injuries.

1. LITERATURE REVIEW

1.1 Anatomy

The gallbladder is a muscular membranous sac that stores and concentrates bile (Rogers, 2010b). It is a pear shaped reservoir in a fossa on the inferior surface of the right lobe (Chaurasia, 1995). It is about 7–10 cm long with an average capacity of 30–50 mL. When obstructed, the gallbladder can distend markedly and contain up to 300 mL (Oddsdottir and Hunter, 2006).

The fossa of the gallbladder extends from the right end of the porta hepatis to the inferior border of the liver. It is divided into fundus, body and neck (Chaurasia, 1995).

The fundus projects beyond the inferior border of the liver, in the angle between the lateral border of the right rectus abdominis and the ninth costal cartilage. It is entirely surrounded by peritoneum and is related anteriorly to the anterior abdominal wall, and posteriorly to the beginning of the transverse colon (Chaurasia, 1995).

The body lies in the fossa for the gallbladder on the liver. The upper narrow end of the body is continuous with the neck at the right end of the porta hepatis. The superior surface of the body is devoid of peritoneum, and is adherent to the liver. The inferior surface is covered with the peritoneum (Chaurasia, 1995).

The neck is the narrow upper end of the gall bladder. It is situated near the right end of the porta hepatis. It first curves anterosuperiorly and then posteroinferiorly to become continuous with the cystic duct. Its junction with the cystic duct is marked by a constriction. Superiorly, the neck is attached to the liver by areolar tissue in which the cystic vessels are embedded. Inferiorly, it is related to the first part of the duodenum. The mucous membrane of the neck is

folded spirally to prevent any obstruction to the inflow or outflow of bile. The posteromedial wall or the neck is dilated outward to form a pouch known as Hartmann's pouch which is directed downward and backward. Gallstone may lodge in this pouch (Chaurasia, 1995).

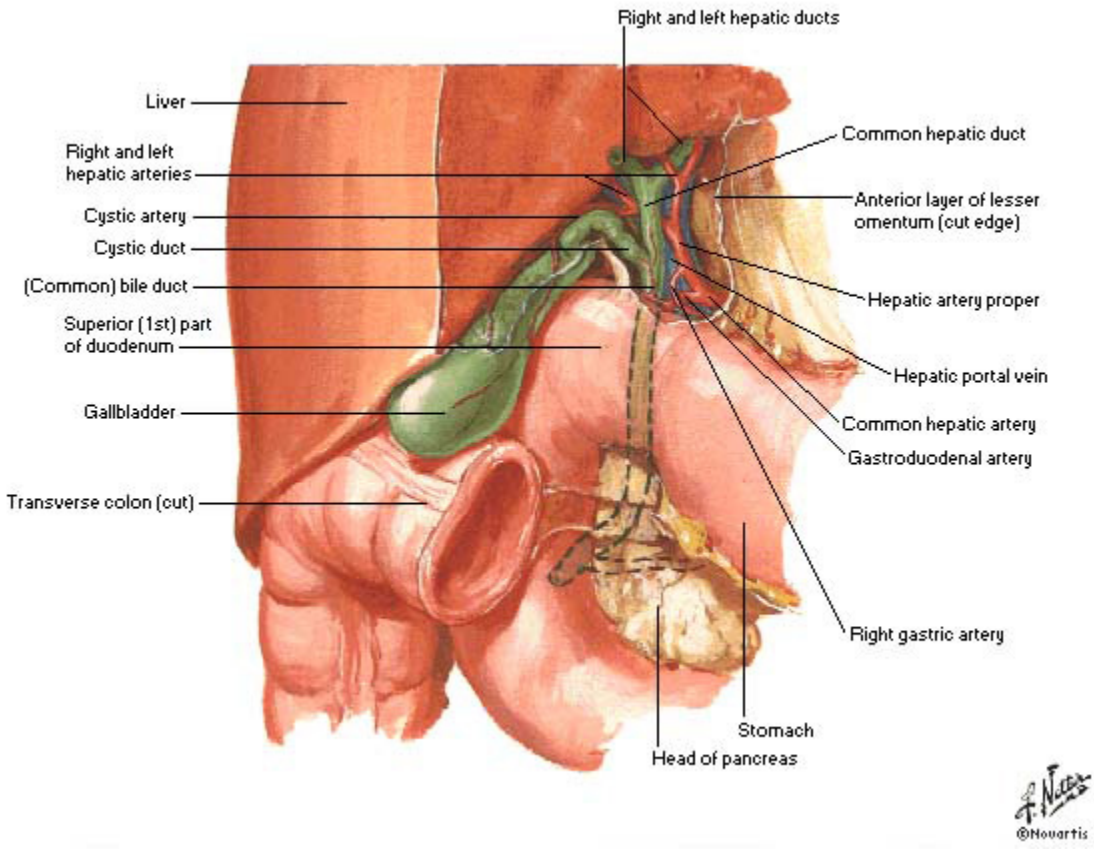


Figure 1 : Anatomy of the gallbladder

(Netter, 2006)

The cystic duct is about 3-4 cm long. It begin at the neck of the gall bladder, runs downward, backward and to the left and ends by joining the common hepatic duct at an acute angle to form the common bile duct (Chaurasia, 1995). Bile in the hepatic duct of the liver flows through the cystic duct into the gallbladder (Scanlon and Sanders, 2006).

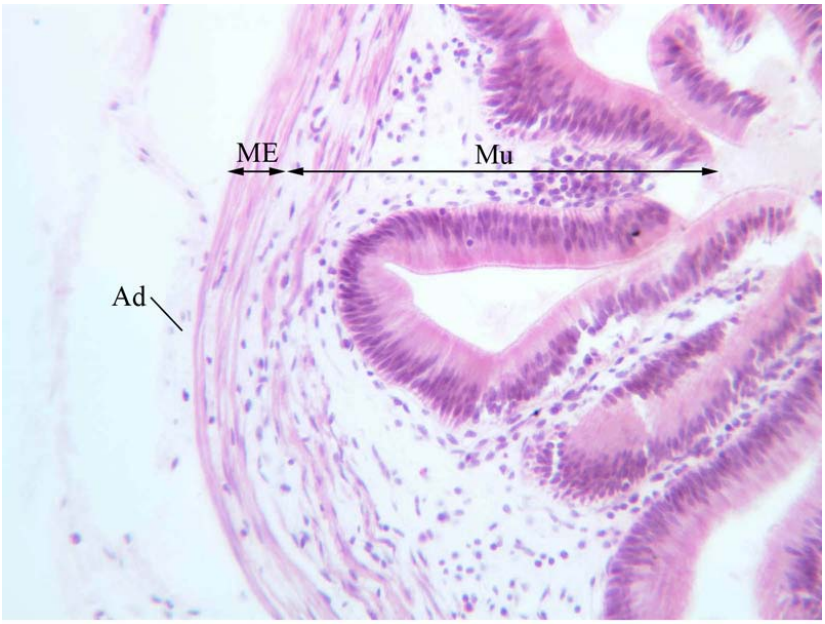
The same peritoneal lining that covers the liver covers the fundus and the inferior surface of the gallbladder (Oddsdottir and Hunter, 2006).

1.2 Histology

The gallbladder is lined by a single, highly folded, tall columnar epithelium that contains cholesterol and fat globules. The epithelial lining of the gallbladder is supported by a lamina propria (Oddsdottir and Hunter, 2006).

The muscle layer has circular longitudinal and oblique fibers, but without well-developed layers. The perimuscular subserosa contains connective tissue, nerves, vessels, lymphatics, and adipocytes (Oddsdottir and Hunter, 2006).

It is covered by the serosa except where the gallbladder is embedded in the liver. The gallbladder differs histologically from the rest of the gastrointestinal tract in that it lacks a muscularis mucosa and submucosa (Oddsdottir and Hunter, 2006).



Ad - adventitia ME - muscularis externa Mu - mucosa

Figure 2 : Histology of gall bladder

(PennState, 2008)

1.3 Physiology

1.3.1 Gall bladder function

The main functions of the gallbladder are (Oddsdottir and Hunter, 2006):

- to concentrate bile
- to store hepatic bile
- regulate the flow and deliver bile into the duodenum in response to a meal.

1.3.2 Bile

Bile, also known as gall is a greenish yellow secretion that is produced in the liver and passed to the gallbladder for concentration, storage, or transport into the first part of the small intestine, the duodenum (Rogers, 2010a).

1.3.2.1 Function of bile

Bile serves two important functions

- Role in fat digestion and absorption
    - *Bile acids* in the bile help to emulsify the large fat particles of the food into many minute particles, the surface of which can then be attacked by lipase enzymes secreted in pancreatic juice (Guyton and Hall, 2006)
    - Aid in absorption of the digested fat end products through the intestinal mucosal membrane (Guyton and Hall, 2006)

- Bile serves as a mean for excretion of several important waste products from the blood.
    - These include especially *bilirubin*, an end product of hemoglobin destruction, and excesses of *cholesterol* (Guyton and Hall, 2006).

1.3.2.2 Bile formation

Bile is secreted in two stages by the liver. The initial portion is secreted by the principal functional cells of the liver, the hepatocytes. The bile then flows in the canaliculi toward gallbladder (Guyton and Hall, 2006).

This initial secretion contains large amount of bile acids, cholesterol, and other organic constituents. It is secreted into minute bile canaliculi that originate between the hepatic cells (Guyton and Hall, 2006).

Next, the bile flows in the canaliculi towards the interlobular septa, where the canaliculi empty into terminal bile ducts and then into progressively larger ducts, finally reaching the hepatic duct and common bile duct. From these the bile either empties directly into the duodenum or is stored and concentrated in the gallbladder, shown in Figure 3 (Guyton and Hall, 2006).

In its course through the bile ducts, a second component of liver secretion is added to the initial bile. This additional secretion is a watery solution of sodium and bicarbonate ions secreted by secretory epithelial cells that line the ductules and ducts. This second type of secretion sometimes increases the total quantity of bile by as much as an additional 100 per cent. The second secretion is stimulated especially by secretin, which causes release of additional quantities of bicarbonate ions to supplement the bicarbonate ions in pancreatic secretion (to neutralize acid that empties into the duodenum from the stomach) (Guyton and Hall, 2006).

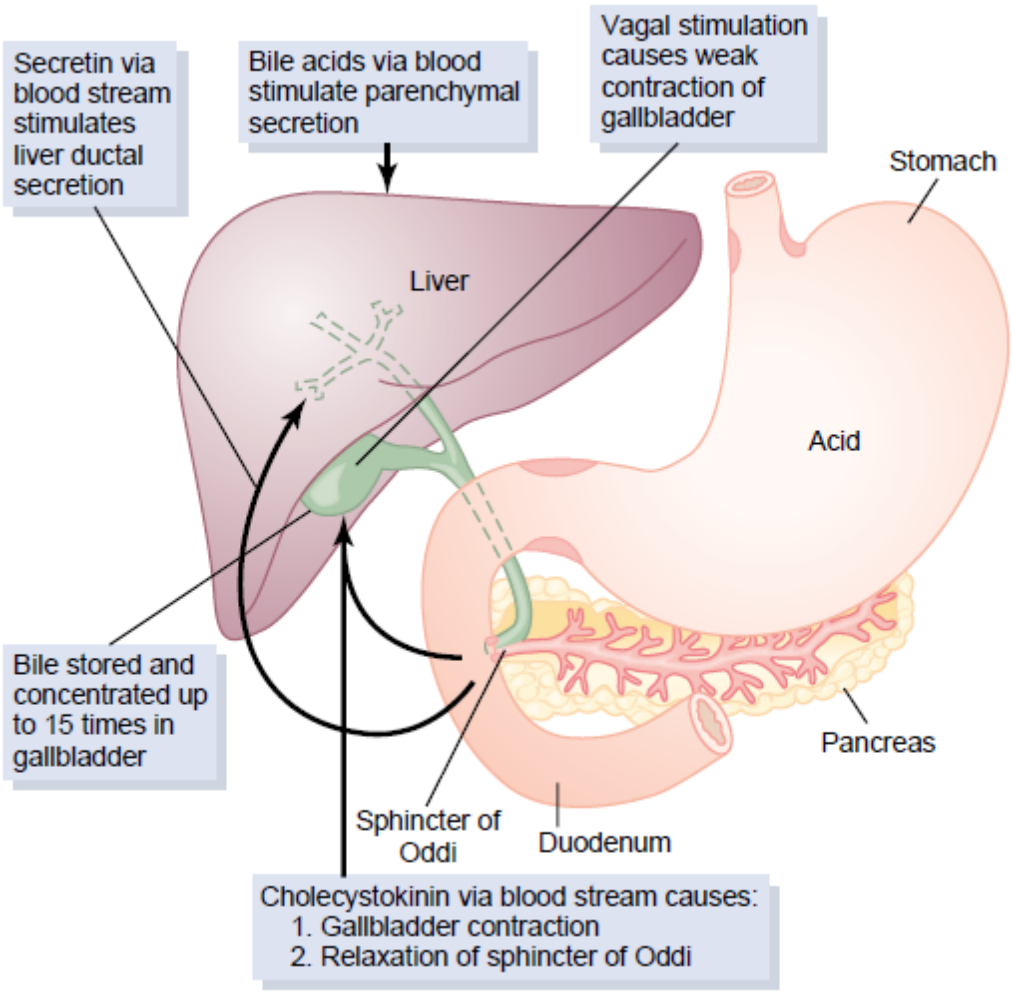


Figure 3 : Liver secretion and gallbladder emptying

(Guyton and Hall, 2006)

1.3.2.3 Storing and Concentrating of Bile in the Gallbladder.

Bile is secreted continually by the liver cells, but most of it is normally stored in the gallbladder until needed in the duodenum. As much as 12 hours of bile secretion (usually about 450 mls) can be stored in the gallbladder because water, sodium, chloride, and most other small electrolytes are continually absorbed through the gallbladder mucosa, concentrating the remaining bile constituents that contain the bile salts, cholesterol, lecithin, and bilirubin (Guyton and Hall, 2006).

Most of this gallbladder absorption is caused by active transport of sodium through the gallbladder epithelium, and this is followed by secondary absorption of chloride ions, water, and most other diffusible constituents. Bile is normally concentrated in this way about 5-fold, but it can be concentrated up to a maximum of 20-fold (Guyton and Hall, 2006).

1.3.2.4 Composition of bile

Bile is made up of the bile salts, bile pigments, and other substances dissolved in an alkaline electrolyte solution that resembles pancreatic juice (Table 1). About 500 mL is secreted per day (Ganong, 2001).

Table 1 shows that by far the most abundant substances secreted in the bile are bile salts, which account for about one half of the total solutes also in the bile. Also secreted or excreted in large concentrations are bilirubin, cholesterol, lecithin, and the usual electrolytes of plasma (Guyton and Hall, 2006).

In the concentrating process in the gallbladder, water and large portions of the electrolytes (except calcium ions) are reabsorbed by the gallbladder mucosa; essentially all other constituents, especially the bile salts and the lipid substances cholesterol and lecithin, are not reabsorbed and, therefore, become highly concentrated in the gallbladder bile (Guyton and Hall, 2006).

Some of the components of the bile are reabsorbed in the intestine and then excreted again by the liver (enterohepatic circulation) (Ganong, 2001).

Composition	Liver bile	Gall bladder bile
Water (g/dL)	97.5	92
Bile salts (g/dL)	1.1	6
Bilirubin (g/dL)	0.04	0.3
Cholesterol (g/dL)	0.1	0.3-0.9
Fatty acids (g/dL)	0.12	0.3-1.2
Lecithin (g/dL)	0.04	0.3
Na^+ (mmol/L)	145.04	130
K^+ (mmol/L)	5	12
Ca^{2+} (mmol/L)	5	23
Cl^- (mmol/L)	100	25
HCO_3^- (mmol/L)	28	10

Table 1 : Composition of bile

(Guyton and Hall, 2006)

1.3.2.5 Emptying of the Gallbladder

When food begins to be digested in the upper gastrointestinal tract, the gallbladder begins to empty, especially when fatty foods reach the duodenum about 30 minutes after a meal. The mechanism of gallbladder emptying is rhythmical contractions of the wall of the gallbladder, but effective emptying also requires simultaneous relaxation of the *sphincter of Oddi*, which guards the exit of the common bile duct into the duodenum (Guyton and Hall, 2006).

Contraction of the muscle wall in the gallbladder is stimulated by the vagus nerve of the parasympathetic system and by cholecystokinin hormone , which is produced in the upper portion of the intestine (Rogers, 2010a). Substances that cause contraction of the gallbladder are called cholagogues (Ganong, 2001). The stimulus for cholecystokinin entry into the blood from the duodenal mucosa is mainly by the presence of fatty foods in the duodenum (Guyton and Hall, 2006).

In addition to cholecystokinin, the gallbladder is stimulated less strongly by acetylcholine-secreting nerve fibers from both the vagi and the intestinal enteric nervous system. They are the same nerves that promote motility and secretion in other parts of the upper gastrointestinal tract. In summary, the gallbladder empties its store of concentrated bile into the duodenum mainly in response to the cholecystokinin stimulus that itself is initiated mainly by fatty foods. When fat is not in the food, the gallbladder empties poorly, but when significant quantities of fat are present, the gallbladder normally empties completely in about 1 hour. Figure 4 summarizes the secretion of bile, its storage in the gallbladder, and its ultimate release from the bladder to the duodenum (Guyton and Hall, 2006).

1.3.3 Gall stone

1.3.3.1 Pathophysiology

Changes in bile acid metabolism and gallbladder function are critical factors in the pathogenesis of gallstones (Smelt, 2010). The combination of a diet high in saturated fats and a sedentary lifestyle in a population that is generally overweight creates an environment prone to gallstone formation (Rakel, 2007).

There are two main types of stones, cholesterol stones (80%) and pigment or calcium stones (20%) (Rakel, 2007). The estimated prevalence of gallstone disease are 10–20% in the adult population, affecting more often women than men (Smelt, 2010).

The pathogenesis of gallstones is multifactorial, based on a complex interaction of environmental and genetic factors (Marschall *et al.*, 2010).

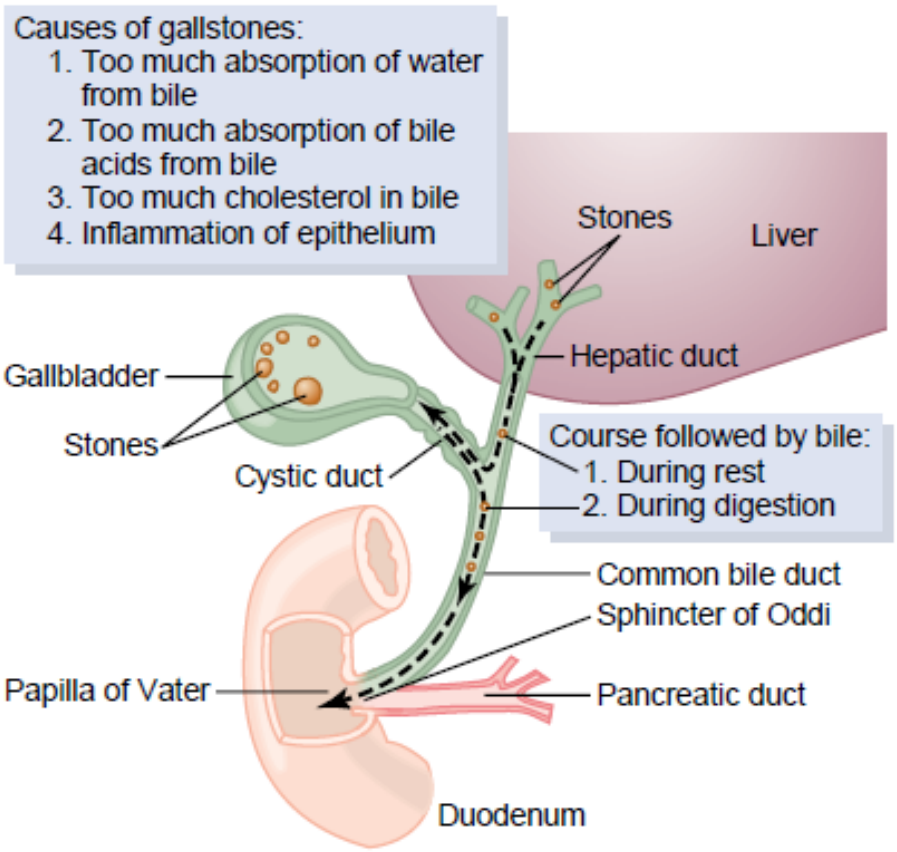


Figure 4 : Formation of gall stone

(Guyton and Hall, 2006)

1.3.3.2 Hepatic hypersecretion and bile supersaturation with cholesterol

In patients with gallstones (particularly obese patients), the liver secretes high levels of cholesterol, probably because of increased cholesterol synthesis. This can lead to cholesterol supersaturation as the result of an increased cholesterol: bile acid ratio in the bile (Jazrawi, 2002).

Cholesterol is very insoluble in bile, and it is maintained in solution in micelles only at certain concentrations of bile salts and lecithin (Figure 5). At concentrations above line ABC in Figure 5, the bile is supersaturated and contains small crystals of cholesterol in addition to micelles (Ganong, 2001).

In bile that has a composition described by any point below line ABC (eg, point P), cholesterol is solely in micellar solution; points above line ABC describe bile in which there are cholesterol crystals as well (Small, 1968).

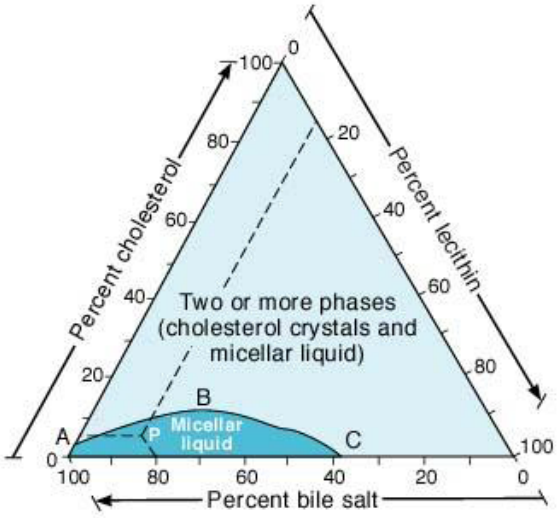


Figure 5 : Cholesterol solubility in bile as a function of the proportions of lecithin, bile salts, and cholesterol.

(Small, 1968)

1.3.3.3 Enhanced cholesterol crystal nucleation

Cholesterol crystals nucleate out of solution faster in bile from those with gallstones than in that from healthy individuals. This probably results from the overall hydrophilic/hydrophobic balance of all proteins in bile (Jazrawi, 2002).

Bile secreted by individuals with gallstones contains a higher percentage of arachidonic acid–rich phospholipids and a secondary bile acid, deoxycholic acid (DCA), both of which have an inflammatory and irritating effect on the gallbladder epithelium. This results in more mucus production, which can worsen gallbladder symptoms and stone formation (Rakel, 2007).

1.3.3.4 Gallbladder motility defects

Gallbladder motility defects have been described in several situations, including pregnancy, diabetes, patients taking female sex hormones, following parenteral nutrition, acromegalic patients taking octreotide (a synthetic somatostatin analogue) and patients undergoing rapid weight loss. In all these situations, there is an increased risk of gallstone formation (Jazrawi, 2002).

Delayed emptying prolongs the residence time of cholesterol in the gallbladder resulting in more nucleation and crystallization (Smelt, 2010). Stasis in the gallbladder leads to cholesterol precipitation and gallstone growth. Other motility defects include reduced postprandial gallbladder refilling and turnover of bile, resulting in stasis of gallbladder contents. These

motility defects have also been found in patients in whom gallstones recur following non-surgical therapy (Jazrawi, 2002).

1.3.3.5 Intestinal motility defects

Prolonged intestinal transit, longer migrating cycles and disrupted motilin release have been reported in patients with gallstones. Prolonged intestinal transit has been reported in acromegalic patients following octreotide therapy, with an increased risk of gallstone formation (Jazrawi, 2002).

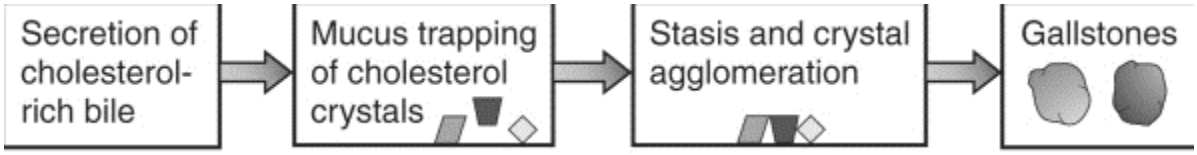


Figure 6 : Pathophysiology of gallstone formation

(Rakel, 2007)

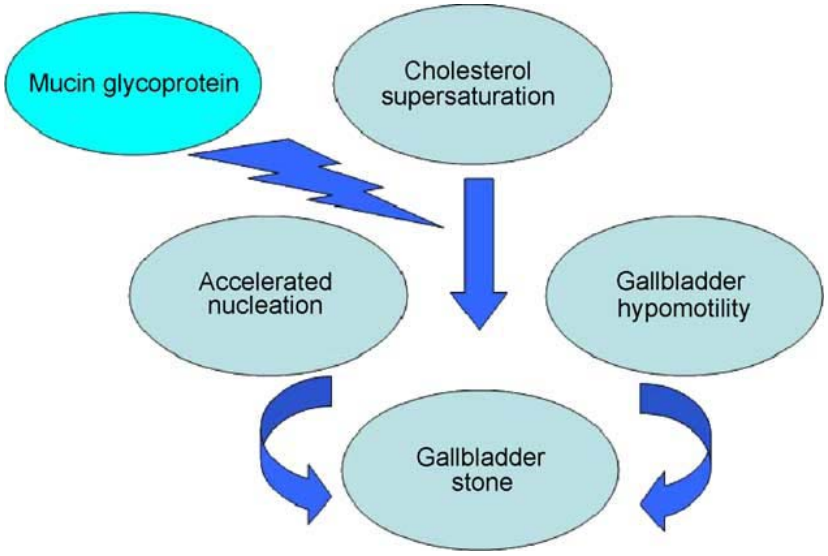


Figure 7 : Pathogenesis of gallstone formation.

(Dayan *et al.*, 2004)

Cholesterol stones (Walton and Lobo, 2008, Jazrawi, 2002)

- Greater age
- Female gender
- Family history and genetic factors (increased risk in first-degree relatives)
- Obesity, weight reduction
- Residence in a developed country (incidence also high in North American Indians)
- Clofibrate (increases cholesterol secretion by the liver)
- Ceftriaxone (forms complexes with calcium in gallbladder bile that precipitate out of solution)
- Octreotide (a somatostatin analogue used in acromegaly and gastroenteropancreatic neuroendocrine tumours)
- Gastrointestinal motility disorders
- Parenteral nutrition (reduces gallbladder motility)
- Pregnancy, oral contraceptives and hormone replacement therapy
- Diabetes mellitus, cystic fibrosis, ileal disease and loss
- Surgery (e.g. vagotomy, biliopancreatic bypass)

Pigment stones (Jazrawi, 2002)

- Chronic haemolysis (haemoglobinopathies)
- Biliary infection, inflammation and infestation
- Cirrhosis
- Residence in the Far East (cirrhosis caused by viral hepatitis is common, and bile duct infestation by the liver fluke *Clonorchis sinensis* causes stones that may contain dead parasites)
- Total parenteral nutrition

Table 2 : Risk factors for gallstone formation

1.3.3.6 Sign and symptoms

Gallstone disease is one of the most common diseases involving the hepatobiliary systems. It carries a substantial burden to health care system. The incidence of gallstone is increasing due to the increase number of aging population (Marschall *et al.*, 2010). Gallstones may be symptomatic or found incidentally. Symptoms arise due to stones in the gallbladder, in the bile duct, or both (Walton and Lobo, 2008).

Most patients with gallstones (up to 70%) are asymptomatic or have nonspecific dyspeptic symptoms (Jazrawi, 2002). Gallstones are often found incidentally in patients undergoing investigation for other reasons (Walton and Lobo, 2008).

The asymptomatic gallstone carries low risk of developing complications. Recent studies indicate that the yearly incidence of complications is 0.3%, while the risk of gallbladder cancer is as low as 0.02% (Portincasa *et al.*, 2006). For these reasons, silent stone are usually managed conservatively with no medical or surgical intervention (Howard and Fromm, 1999).

For unknown reasons some patients progress to a symptomatic stage. Most symptoms and signs are caused by complications of gallstone disease (Table 2) (Jazrawi, 2002).

Symptoms attributable to gallbladder stones manifest in a number of ways. In uncomplicated cases, the main sign is tenderness in the right upper quadrant during episodes of biliary colic (Jazrawi, 2002). Approximately 3 percent of asymptomatic individuals develop biliary colic each year (Oddsdottir and Hunter, 2006). Once symptomatic, patients tend to have recurring bouts of biliary colic. The probability of recurrence of symptom after 1 year is about 50% and as high as 70% after 2 years (Howard and Fromm, 1999).

Biliary colic is the only specific symptom of uncomplicated gallstone disease. It occurs in less than 10% of individuals with gallstones. It comprises sudden-onset, severe epigastric or right upper abdominal pain that lasts for several hours, usually radiates to the back and is sometimes accompanied by nausea and vomiting. The intensity rises to a crescendo; however, patients often believe it to be constant, and thus fluctuations may not be apparent (Jazrawi, 2002). This biliary pain produce when gallstone block or obstruct the cystic or common bile duct (Howard and Fromm, 1999).

Symptomatic gallstone disease may progress to complications related to the gallstones. In this symptomatic gallstone, they have spectrum of presentation from acute or chronic cholecystitis, biliary colic, pancreatitis or obstructive jaundice. In severe form of acute cholecystitis, 10% - 30% of patient will develop life threatening complication such as empyema, gangrene or perforation of gallbladder (Hadad *et al.*, 2007b). Rarely, complication of gallstones is the presenting picture (Oddsdottir and Hunter, 2006).

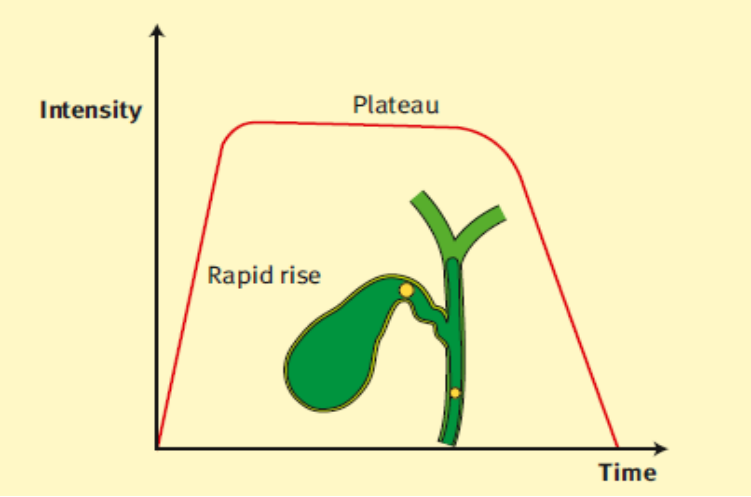


Figure 8 : Pain typically experienced during an attack of biliary colic

(Moser and Roslyn, 2001).

Size of the gallstone is also very important which has been shown to influence the type of complication. There is four fold greater risk of developing acute pancreatitis with gallstone smaller than 5 mm diameter (Howard and Fromm, 1999).

On the other hand, the prolonged presence of gallbladder stone of more than 3 cm may be a risk in the development of gallbladder carcinoma. The calcification (porcelain) gallbladder is also a risk factor for gallbladder cancer (Howard and Fromm, 1999).

1.3.3.7 Complication

Complication	**Symptoms**	**Signs**
Stone impaction in common bile duct	Biliary colic	Jaundice
Acute cholecystitis	Fever, abdominal pain, nausea, vomiting	Murphy's sign (localized tenderness over gallbladder)
Chronic cholecystitis	Nonspecific	Nonspecific abdominal pain
Acute pancreatitis	Abdominal and back pain, vomiting, collapse	Hypotension, tenderness with rebound
Acute cholangitis	Pain, fever, rigors	Jaundice

Table 3 : Common complications of gallstone disease

(Jazrawi, 2002)

1.3.3.8 Investigations

1.3.3.8.1 Blood Tests

- **Full blood count**
  - An elevated white blood cell (WBC) count may indicate or raise suspicion of cholecystitis (Oddsdottir and Hunter, 2006).

- **Liver function test**
  - If associated with an elevation of conjugated bilirubin, alkaline phosphatase, and aminotransferase, cholangitis should be suspected.
  - Cholestasis, an obstruction to bile flow, is characterized by an elevation of bilirubin and a rise in alkaline phosphatase. Serum aminotransferases may be normal or mildly elevated (Oddsdottir and Hunter, 2006).
  - In patients with biliary colic, blood tests typically will be normal.

1.3.3.8.2 Ultrasonography

- An ultrasound will show stones in the gallbladder with sensitivity and specificity of over 90 percent (Oddsdottir and Hunter, 2006).
- Abdominal ultrasonography is more sensitive than plain x-ray in detecting small stones and biliary sludge, and patients are not exposed to radiation. Plain abdominal radiography detects only about 20% of stones (those that are calcified) (Jazrawi, 2002).

- In acute cholecystitis, a layer of edema is seen within the wall of the gallbladder or between the gallbladder and the liver.
- A contracted, thick-walled gallbladder indicates chronic cholecystitis.

1.3.3.8.3 Oral Cholecystography

- Oral Cholecystography involves oral administration of a radiopaque compound that is absorbed, excreted by the liver, and passed into the gallbladder (Oddsdottir and Hunter, 2006). It is insensitive in detecting small stones (Jazrawi, 2002).
- Oral cholecystography is of no value in patients with intestinal malabsorption, vomiting, obstructive jaundice, and hepatic failure (Oddsdottir and Hunter, 2006).

1.3.3.8.4 Biliary Radionuclide Scanning (HIDA Scan)

- Biliary scintigraphy provides a noninvasive evaluation of the liver, gallbladder, bile ducts, and duodenum with both anatomic and functional information (Oddsdottir and Hunter, 2006).
- The primary use of biliary scintigraphy is in the diagnosis of acute cholecystitis, which appears as a nonvisualized gallbladder, with prompt filling of the common bile duct and duodenum (Oddsdottir and Hunter, 2006).

1.3.3.8.5 Computed Tomography

- Abdominal computed tomography (CT) scans are inferior to ultrasonography in diagnosing gallstones (Oddsdottir and Hunter, 2006).
- CT scan is the test of choice in evaluating the patient with suspected malignancy of the gallbladder, the extrahepatic biliary system, or nearby organs, in particular the head of the pancreas (Oddsdottir and Hunter, 2006).

1.3.3.8.6 Percutaneous Transhepatic Cholangiography

- An intrahepatic bile duct is accessed percutaneously with a small needle under fluoroscopic guidance (Oddsdottir and Hunter, 2006).

- Percutaneous transhepatic cholangiography is particularly useful in patients with bile duct strictures and tumors, as it defines the anatomy of the biliary tree proximal to the affected segment (Oddsdottir and Hunter, 2006).