POSTCHOLECYSTECTOMY SYNDROME: AFTER TWO YEARS AND BEYOND 127 SUCCESSFUL LAPAROSCOPIC CHOLECYSTECTOMIES IN THE HOSPITAL UNIVERSITI SAINS MALAYSIA

by

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ABSTRAK

Pembedahan kolisistektomi adalah merupakan rawatan yang standard bagi penyakit batu karang hempedu, lebih-lebih lagi dengan adanya teknik terbaru pembedahan iaitu pembedahan laparoskopi yang telah dikenalpasti mempunyai beberapa kelebihan. Walaubagaimanapun, terdapat 6 hingga 39 peratus pesakit yang telah menjalani pembedahan tersebut masih mempunyai simptom-simptom penyakit batu karang hempedu yang dialaminya sebelum pembedahan atau timbulnya simptom-simptom baru yang berkaitan. Kajian yang sama terdahulu hanya mengkaji simptom-simptom yang kekal atau timbul setahun selepas pembedahan. Tambahan pula , tiada data tempatan mengenai sindrom pascakolisistektomi ini.

Tujuan kajian ini dilakukan adalah untuk mengetahui insiden sindrom pascakolisistektomi 2 tahun atau lebih selepas pembedahan laparoskopi kolisistektomi dilakukan di Hospital Universiti Sains Malaysia di antara Mac 1995 hingga Disember 1998. Pada masa yang sama juga, penyebab sindrom pascakolisistektomi yang signifikan dapat ditentukan.

Kajian ini adalah kajian berbentuk diskriptif retrospektif yang melibatkan 127 pesakit hospital ini yang telah menjalani pembedahan elektif laparoskopi kolisistektomi yang tidak berkomplikasi. Maklumat-maklumat berkenaan dengan demografi, indikasi pembedahan, dan simptom-simptom pascakolisistektomi beserta penyiasatannya dan rawatannya di dalam rekod pesakit dianalisa.

11 (8.7%) pesakit didapati mempunyai sindrom pascakolisistektomi. 5 pesakit mempunyai simptom penyakit batu karang hempedu yang tipikal manakala 6 pesakit lagi mempunyai simptom yang tidak tipikal. Mean tempoh masa simptom yang tersebut dihidapi oleh pesakit adalah 36.2 bulan. 72.7% pesakit tersebut mempunyai

sejarah penyakit dalaman manakala 18.2% pernah menjalani prosedur pembedahan. 54.5% mengalami sindrom klinikal kolik biliari, 27.3% sindrom klinikal kolisistitis yang kronik dan 18.2% sindrom klinikal pankreatitis. Analisis univariasi hanya mendapati pesakit yang mempunyai sindrom klinikal kolisistitis yang kronik sahaja yang ada kaitan dengan sindrom pascakolisistektomi ($p<0.05$). Manakala sindrom klinikal kolik biliari dan pesakit yang mepunyai sejarah penyakit atau pernah menjalani pembedahan tidak signifikan dalam perkaitannya. Seorang pesakit mengalami penyakit barah usus besar sigmoid. Tiada pesakit yang mengalami sindrom tersebut berpunca penyakit hepatobiliari dan didapati penyiasatan rapi tidak dilakukan untuk memastikan sindrom pascakolisistektomi berpunca dari penyakit yang tersebut.

Dirumuskan bahawa kajian ini telah memberi keputusan yang lebih kurang sama dengan kajian terdahulu yang seumpamanya walaupun selepas 2 tahun atau lebih daripada tarikh pembedahan.

ABSTRACT

Cholecystectomy is considered the gold standard treatment for symptomatic gallstones disease especially with the advent of laparoscopic approach, which is known to have well documented advantages. Nevertheless, 6% to 39% of the patients continue to have either persistent symptoms or emergence of new symptoms suggestive of gallstone disease after the laparoscopic cholecystectomy. All the previous studies were looking at the postcholecystectomy symptoms or syndrome one year after the surgery. Furthermore, there is no local data regarding the postcholecystectomy syndrome as yet.

The aim of this study is to know the incidence of the postcholecystectomy syndrome after 2 years and beyond the laparoscopic cholecystectomy done in the Hospital Universiti Sains Malaysia from March 1995 until December 1998. At the same time, any significant pathology that gives rise to the syndrome is reviewed.

This is retrospective descriptive study of 127 patients who underwent uncomplicated elective laparoscopic cholecystectomy in this hospital. Patients' records were reviewed for demographic data, indications for surgery, and symptoms attributed to postcholecystectomy syndrome, their investigations and management.

11 (8.7%) patients were noted to have postcholecystectomy syndrome. 5 (45.5%) patients had typical symptoms of gallstone disease and 6 (54.5%) had atypical symptoms. Mean time interval the symptoms manifested for the first time was 36.2 months (24 to 55 months). 8 (72.7%) of the patients had medical illness and 2 (18.2%) had history of past surgical procedures. Majority of them (n = 6, 54.5%) had clinical syndrome of biliary colic, 27.3% had chronic cholecystitis and 18.2% had pancreatitis. Univariate analysis revealed significant association with

postcholecystectomy syndrome only in patients with chronic cholecystitis ($p<0.05$) and not in biliary colic group ($p=0.07$), patients with history of medical illness ($p=0.098$) or surgical operations ($p=0.181$). One patient was noted to have sigmoid colon cancer 3 years after cholecystectomy. No patient with postcholecystectomy syndrome directly related to hepatobiliary pathology since it was not fully investigated.

In conclusion, the incidence of postcholecystectomy syndrome in this retrospective study of local population was comparable with other previous studies even after 2 years and beyond of the surgery.

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INTRODUCTION

1.0: INTRODUCTION

Cholecystectomy is still the gold standard for the treatment of symptomatic gallstones. Since the introduction of laparoscopic cholecystectomy by Mouret in 1987, it has rapidly gained popularity and is now considered the gold standard treatment for symptomatic gallstone disease (1,2). Its advantages over open cholecystectomy include reduced postoperative hospitalisation, reduced pain and morbidity, better cosmesis, early return to work and considerable financial savings (3,4,5,6). Gallstones are associated with a wide variety of symptoms. These include abdominal pain, pain related to fatty foods, nausea and vomiting as well as dyspepsia, abdominal bloating and increased flatulence. While cholecystectomy is the best treatment for symptomatic gallstones, recent studies show that up to 34 percent of patients still have symptoms after laparoscopic cholecystectomy(7). These symptoms either because of lack of improvement or relapse of symptoms initially attributed to the presence of gallstones or because of the emergence of new gastrointestinal complaints presumably related to the operation. The term postcholecystectomy syndrome(8) has been used to describe this condition which range from mild ill defined digestive symptoms to severe attacks of abdominal pain and jaundice. We therefore reviewed our local patients who continue to have significant postcholecystectomy syndrome after two years and beyond the laparoscopic cholecystectomy, which causing them to seek treatment since the advent of laparoscopic cholecystectomy practices in Hospital Universiti Sains Malaysia.

LITERATURE REVIEW

2.0: LITERATURE REVIEW

2.1: ANATOMY OF THE BILIARY SYSTEM

2.1.1: ANATOMY OF THE EXTRAHEPATIC BILIARY TREE

The biliary tree consists of fine intrahepatic biliary radicles, the right and left hepatic ducts, the common hepatic duct and the common bile duct. The left hepatic duct is formed within the umbilical fissure from the union of the three segmental ducts draining the left liver (segments II, III, and IV). The right hepatic duct drains segments V, VI, VII, and VIII. The biliary drainage of the caudate lobe (segment I) is variable (9). In 80% of the individuals, the caudate lobe drains into both the right and left hepatic ducts; in 15% of patients, the caudate lobe drains only into the left hepatic duct; and in the remaining 5% of cases, the lobe is drained exclusively by the right hepatic duct.

The right and left hepatic ducts converge to form the common hepatic duct occurs in an extrahepatic location anterior to the portal venous bifurcation. The extrahepatic portion of the right duct is short whereas the left hepatic duct has an extrahepatic length of 2 cm or more (9).

The common hepatic duct descends in the hepatoduodenal ligament and is joined at a variable position by the cystic duct to form the common bile duct, which ends at the papilla of Vater, usually in the second part of the duodenum. The common bile duct is approximately 8 cm in length and up to 10 mm in diameter (10). The upper third, or supraduodenal portion, of the common bile duct courses downward in the free edge of the lesser omentum, anterior to the portal vein and to the right of the hepatic artery. The middle third, or retroduodenal portion, of the common bile duct passes behind the first portion of the duodenum, lateral to the portal vein and anterior to the inferior vena cava.

The lower third, or intrapancreatic portion, of the common bile duct traverses the posterior aspect of the pancreas in a tunnel or groove to enter the second portion of the duodenum, where it is accompanied by the pancreatic duct of Wirsung. The intramural, or intraduodenal, portion of the common bile ducts passes obliquely through the duodenal wall to enter the duodenal lumen at the papilla of vater.

The biliary ducts from intrahepatic biliary ductules till ampulla of vater may become pathologically dilated, usually congenitally, either the whole length or certain portion of the duct. This dilatation when present is called choledochal cyst. There is one reported case of postcholecystectomy symptom found to have solitary stone in the choledochal cyst (11).

The gallbladder is a pear shaped; distensible reservoir lies in a bed on the undersurface of the liver between the right and left lobes. The gallbladder has a capacity of 30 to 50 ml. It is a muscular structure with a fundus, body and neck. The fundus is the rounded portion of the gallbladder that usually extends beyond the edge of the liver. Hartmann's pouch is a dilatation of the gallbladder outlet adjacent to the origin of the cystic duct in which gallstones frequently become impacted. It may obscure the junction of the cystic duct with the common hepatic duct. The gallbladder may occasionally be partially or completely embedded within the liver parenchyma (intrahepatic gallbladder) or abnormally positioned beneath the left lobe of the liver. The gallbladder may also be suspended from the cystic fossa by a complete mesenteric attachment that may predispose to torsion of the gallbladder.

The cystic duct arises from the neck of the gallbladder and passes downward in the hepatoduodenal ligament to join the common hepatic duct at an acute angle. The length of the cystic duct is variable, ranging from 0.5 to 4 cm, depending upon the location of its union with the common hepatic duct.

2.1.2: ANOMALIES OF THE EXTRAHEPATIC BILIARY TREE

Knowledge of the variable anatomy of the extrahepatic biliary tree is important for the general surgeon, because the failure to recognize anatomic variations may result in a significant ductal injury.

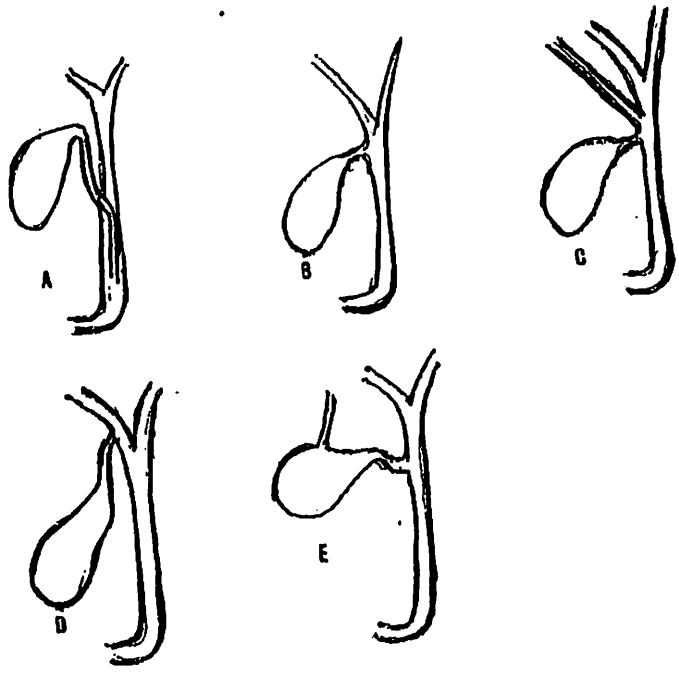
There are five ductal anomalies of significance to the surgeon during performance of a cholecystectomy (Figure 1) (12). These anomalies should be easily recognized at the time of operation by careful and meticulous dissections. One of the most common ductal variations is the presence of a long cystic duct with a low union with the common hepatic duct. In this case, the cystic duct is usually closely adherent to the hepatic duct for a variable length. Efforts to display the entire length of the cystic duct and its union with the common hepatic duct may result in a ductal injury and should be avoided (12).

Accessory hepatic ducts, usually from the right lobe of the liver, may join the common hepatic duct or cystic duct. These accessory ducts often course through the Callot's triangle and may be injured during dissection in this area. Segmental hepatic ducts may also directly enter the common hepatic duct, cystic duct, and, occasionally, the gallbladder. Small cholecystohepatic ducts from the liver may enter the gallbladder directly. If it is discovered during dissection of the gallbladder from the liver bed, it should be ligated to avoid a postoperative biliary leak.

Figure 1: Duct anomalies.

A, Long cystic duct with low fusion with common hepatic duct.
B, Abnormally high fusion of cystic duct with common hepatic duct (trifurcation).
C, Accessory hepatic duct.
D, Cystic duct entering right hepatic duct.
E, Cholecystohepatic duct.

(Adapted from Laparoscopic anatomy of the biliary tree; In Surgical Anatomy And Embryology, *Surgical Clinic of North America.*



2.2: PHYSIOLOGY

Bile, which is formed in the liver, is made up of bile salts, bile pigments, and other substances namely cholesterol and alkaline phosphatase dissolved in an alkaline electrolyte solution. About 500 ml of the bile is secreted per day. Some of the components of the bile are reabsorbed in the intestine and then excreted again by the liver.

The glucuronides of the bile pigments, biliverdin and bilirubin, which are also breakdown products of the haemoglobin, are responsible for the golden yellow colour of bile.

Bile acids are sterols synthesized by the liver from cholesterol. The two primary bile acids are chenodeoxycholic and cholic acid; these are conjugated with glycine or taurine, derivative of cystine, to form sodium and potassium salts of bile acids, glycocholic and taurocholic acid, in the alkaline hepatic bile. The conjugation will increase their solubility in water. In the colon, bacteria convert cholic acid to deoxycholic acid and lithocholic acid, secondary bile acids.

Bile salts have a number of important actions. The bile salts can combine with lipids to form micelles, water-soluble complexes from which the lipids can be more easily absorbed. They are also detergents and reduction in surface tension allows fat to be emulsified in the intestine, thus facilitating its digestion and absorption.

On reaching the distal ileum, 90 to 95% of the bile salts are reabsorbed back by mostly active transport process and then transported back to the liver and passed once again into the biliary system(13). The remaining 5% enter the colon and are converted to the salts of deoxycholic acid and lithocholic acid. Lithocolate is relatiovely insoluble and is mostly excreted in the stools; only 1% is absorbed. However deoxycholate is absorbed.

The absorbed bile salts are transported back to the liver via the portal vein and reexcreted in the bile. This is called enterohepatic circulation allows a relatively small bile salt pool (2-4 g) to circulate some 6-8 times per day through the intestine(13). The daily faecal loss equals that of hepatic synthesis (0.2-0.6 g/24 h) (10). When the bile is excluded from the intestine, up to 50% of ingested fat appears in the faeces (13).

Bile flows into the gallbladder when the sphincter of Oddi is closed. The gallbladder has a capacity of 50 ml. In the gallbladder, the bile is concentrated by absorption of water. It contracts in response to cholecystokinin (CCK), which is released from the duodenal mucosa by the presence of food, notably fatty acids. Gallbladder contraction is accompanied by reciprocal relaxation of the sphincter of Oddi. The production and secretion of the bile is increased by the hormone secretin and by stimulation of the vagus nerves.

2.3: PATHOGENESIS OF GALLSTONES

Gallstones are formed from the constituents of bile. The great majority of stones result from failure to keep cholesterol in micellar form in the gallbladder, and pigment stones (composed of calcium bilirubinate) are rare in the western countries but common in the tropical countries. Most cholesterol stones become mixed with bile pigments as they increase in size; such "mixed" stones are much more common than pure cholesterol stones.

Gallstones are common in Europe and North America and less common in Asia and Africa (14). Their incidence increases with age. In developed countries they occur in at least 20% of women over the age of 40; the incidence in males is about one-third of

that in females (15). The disease has increased markedly in frequency and cholecystectomy is the commonest major elective abdominal operation in many Western countries (16).

Cholesterol Stones

Cholesterol stones may occur in both sexes from the late teens onwards but are particularly common in middle aged, obese, multiparous females. Stone formation is encouraged if bile becomes supersaturated with cholesterol (i.e. lithogenic) either by excessive cholesterol excretion or by reduction in the amount of bile salt and lecithin available for micelle formation (14). Supersaturation is likely to occur during the concentration of bile while it is within gallbladder, and is favoured by stasis or decreased gallbladder contractility. The formation of cholesterol crystals is the key event, and this nucleation may be due to coalescence of cholesterol molecule around particles of mucus, bacteria, calcium bilirubinate or mucosal cells. Not all individual with supersaturated bile develop gallstones so that other factors must be implicated. Pure cholesterol stone are yellowish-green with a regular shape but rough surface. They are usually solitary. In contrast, mixed stones are darker and are usually multiple.

Cholesterol stones are particularly common in some tribes of north American Indians, where more than 75% of women over 40 are affected (14). Such individual have a small bile salt pool. Conversely, the high incidence of stones in Chilean women reflects high levels of excretion (15).

Obesity and high-calorie or high-cholesterol diets favour cholesterol stone formation by producingly high supersaturated gallbladder bile, Drastic weight reduction

and diets designed to lower serum cholesterol levels may also promote stone formation by mobilising cholesterol and increasing its excretion.

Disease or resection of the terminal ileum and drugs such as cholestyramine may favour cholesterol nucleation by reducing bile salt pool. Hormonal influences are reflected in an increased of stone formation in women taking oral contraceptives or postmenopausal oestrogen replacement. Pregnancy may also have an effect by increasing stasis within the gallbladder. Similarly, after vagotomy the gallbladder become flaccid and increases in volume. Hypercholesterolaemia as such is not associated with stone formation.

Pigment stones

Pigment stones consist of calcium bilirubinate and are usually multiple, small and amorphous. Stones found in occidental patients are usually composed of black pigments, whereas brown pigments stones are common in orientals. Pigment stones account for 25% of all gallstones in Western patients but for 60% of those in some oriental countries such as Japan (15).

Chronic haermolysis favours pigment stone formation by increasing pigment excretion, and stone formation is common in congenital spherocytosis, heamoglobinopathy and malaria. Cirrhosis and biliary stasis are also important associations. Some patients with brown pigment stones have increased concentrations of unconjugated bilirubin in the bile. In oriental patients this may be due to the action of beta-glucuronidase, which is produced by E. coli and which invades ductal systems infested with the parasites Clonorchis sinensis or Ascaris lumbricoides (15).

2.4: COMMON CLINICAL SYNDROMES ASSOCIATED WITH GALLSTONES

The majority of individuals with gallstones is asymptomatic or has only vague symptoms of distension and flatulence. Half of such patients will develop some symptoms or complications due to gallstones within 10 years. Gallstones may give rise to symptoms in several ways.

Biliary colic

Biliary colic is due to transient obstruction of the gallbladder from an impacted stone. There is severe gripping pain, often developing in the evening, and maximal in the evening, and maximal in the epigastrium and right hypochondrium with radiation to the back. Though continuous, the pain may wax and wane in intensity over several hours. Vomiting and retching occur. Resolution follows when the stone falls back into the common gallbladder lumen or passes onwards into the common bile duct. The patient recovers rapidly but repeated bouts of colic are common.

Acute cholecystitis

Acute cholecystitis results in a more prolonged and severe illness. It usually begins with an attack of biliary colic, though its onset may be more gradual. There is severe right hypochondrial pain radiating to the right subscapular region, and occasionally to the right shoulder (from diaphragmatic irritation), together with tachycardia, pyrexia, nausea, vomiting and leucocytosis. Abdominal tenderness and

rigidity may be generalized but are most marked over the gallbladder. Murphy's sign (a catching of the breath at the height of inspiration while the gallbladder is palpated) is usually present. A right hypochondrial mass may be felt. This is due to omentum "wrapped" around the inflamed gallbladder.

In 85-90% of cases the attack settles within 4-5 days (15). In the remainder, tenderness may spread and pyrexia signals empyema formation. The gallbladder may become gangrenous and perforate, giving rise to biliary peritonitis.

Jaundice can develop during the acute attack. Usually this is associated with stones in the common bile duct but compression of the bile ducts by the gallbladder may be responsible.

Acute cholecystitis must be differentiated from perforated peptic ulcer, high retrocaecal appendicitis, acute pancreatitis, myocardial infarction and basal pneumonia. Acute cholecystitis can develop in the absence of gallstones (acalculous cholecystitis). This is rare.

Chronic cholecystitis

Chronic cholecystitis is the common form of symptomatic gallbladder disease. It is almost invariably associated with gallstones. Recurrent flatulence, right upper quadrant pain and fatty food intolerance are common. The pain is worse after meals and is often associated with a feeling of distension and heartburn.

The differential diagnosis includes duodenal ulcer, hiatus hernia, myocardial ischaemia, chronic pancreatitis and gastrointestinal neoplasia.

Mucocele

A piriform swelling is palpable in the right hypochondrium. It is not tender and there is no pyrexia.

2.5: CHOLECYSTECTOMY

For over 100 years cholecystectomy has been the standard treatment for symptomatic cholelithiasis. Cholecystectomy, first performed in 1882 by Langenbeck in Berlin. It is one of the most common surgical procedures done in western industrialized societies, is widely acknowledged to be safe, simple operation which definitely eradicates gallstones in the vast majority of patients (16). Cholecystectomy is considered the gold standard for the treatment of symptomatic gallstones, especially after the introduction of the laparoscopic technique (1,2). The overall morbidity ranges from 4 to 6 percent and operative mortality, from 0.5 to 3.4 percent with increased mortality of 9 percent for patients older than 65 years old and of 10 to 67 percent for patients with complications of gangrene and perforation (17). Alternative treatments for gallstones have been advocated recently including stone dissolution, lithotripsy and percutaneous stone removal. All of these approaches, however, leave the gallbladder in situ with the possibility of stone recurrence. The development of minimally invasive techniques in urology and gastroenterology has stimulated general surgeons to explore the therapeutic potential of laparoscopy in the management of gallbladder disease.

2.5.1: LAPAROSCOPIC CHOLECYSTECTOMY

Mouret from France performed the first laparoscopic cholecystectomy in 1987(13). Since its introduction, laparoscopic cholecystectomy has rapidly gained in popularity and is now considered the standard treatment for symptomatic gall stone disease (10,18). With experience and training, it is applicable to over 95 percent of patients (19). The procedure reproduces all the steps of standard laparotomy cholecystectomy. Its advantages over open surgery are well documented (4,5,6). These include reduced postoperative hospitalisation, reduced pain and morbidity, better cosmesis and considerable financial savings (4,5). The complication (morbidity rate between 2.00 to 6.87%) and death (mortality rate between 0.04 to 0.23%) rates are low for laparoscopic cholecystectomy (19).

Patient selection

The indications for laparoscopic cholecystectomy are the same as standard cholecystectomy(19) and these include:

Cholelithiasis & biliary colic or cholecystitis

Symptomatic gallbladder polyp

Gallstone pancreatitis

Symptomatic biliary dyskinesia

Calcified gallbladder wall

Large gallstone >2 cm

Nonfunctioning gallbladder

Chronic typhoid carrier

Morbidly obese patients and prior upper abdominal surgery are no longer considered a contraindication (19). In fact, morbidly obese patients benefit greatly from a laparoscopic approach and are discharged the same or next day with little or no morbidity.

Patient evaluation

Patients with symptoms compatible with gallstone disease usually have diagnosis confirmed on ultrasonography, which provides information about the gallbladder wall thickness (chronic cholecystitis), pericholecystic fluid (acute cholecystitis), and the size of the common bile duct. Endoscopic retrograde cholangiopancreatography is indicated for patients with abnormal liver function tests suggesting common bile duct stones and for patients with gallstone pancreatitis who are not improving.

Equipment

The equipment required for laparoscopic cholecystectomy includes a high flow insufflator (carbon dioxide), light source (xenon), high resolution camera (which will be attached to the endoscope), high resolution video monitor, irrigation device and electrocautery unit or ultrasonic scalpel.

Technique

Preoperative antibiotics can be used routinely or reserved for patients with medical risk factors and for patients with evidence of recent episodes of cholecystitis. In Hospital Universiti Sains Malaysia, antibiotic prophylaxis was given routinely though

there was no standardisation (19).The procedure is performed with the patient under general anaesthesia (20). The stomach is decompressed with a nasogastric tube to facilitate exposure but if an open technique (Hasson cannula) is used, it is unnecessary.

With the patient in supine position, a 2 or 3 cm incision is made superior or inferior to the umbilicus. The Hasson cannula is inserted. Insufflating CO_2 into the peritoneal cavity creates pneumoperitoneum. A telescope with camera attached is inserted through the cannula and the abdominal and pelvic cavities are inspected. The other one 10 mm incision at the epigastric region and 2 incisions of 5 mm in size at the right subcostal margin are made under direct vision. Through the most lateral right subcostal cannula, a grasper retracts the fundus of the gallbladder over the liver toward the right hemidiaphragm. Through the other subcostal cannula, a grasper retracts the Hartmann's pouch laterally and anteriorly. The dissection is begun at the neck of the gallbladder and proceeds along the cystic duct. After the cystic duct and artery have been identified, a titanium clip is placed at the junction of the neck and cystic duct before it is transected. The gallbladder is dissected from its bed, and before the last attachments at the fundus are divided, the gallbladder bed is irrigated and is inspected for bleeding and bile leaks. The stumps of the cystic duct and artery are inspected for bleeding and bile leaks. Under direct vision the gallbladder is removed through the epigastric incision, most of the time together with the port.

The subcostal cannulas are also removed under direct visualization. Small drainage tube is inserted under direct vision if there is considerable bile leakage or when it is considered necessary to the operating surgeon. Rectus sheath openings of the epigastric and umbilical incisions were closed with J needle vicryl suture. This is followed by skin closure with absorbable suture or adhesive tapes. The other two 5 mm incisions, only the skin is closed in a similar manner.

Complications

There are six common surgical complications directly related to laparoscopic cholecystectomy namely:

Bile duct injury (21)

Bleeding (22)

Bile spillage

Stones in the peritoneal cavity (23)

Wound infection

Injury to another organ (24) or vessel (22)

Other types of complications have been reported and are uncommon than these six. Duct injury is a severe injury, and its prevalence decreases with experience (21) and time (22). Bleeding can occur from avulsion of the cystic artery, from injury to the hepatic injury, or the liver bed. Major vascular injuries to the portal vein, vena cava, or aorta are rare (prevalence, 0.11%) and require prompt exploratory laparotomy (22). Bile spillage may occur if the gallbladder is injured with the cautery unit or a grasper. Stones may escape from the gallbladder if the gallbladder is injured and opened. The stones should be retrieved because they may cause abscesses (23). Migration and development of gallstones in wounds and other places have been reported but are uncommon. Wound infection is a rare and minor problem. Because the incisions are small, infections are insignificant and typically resolve with dressings changes and antibiotics. Injury to the intestine (24) or another organ can occur with introduction of a cannula or insertion of an instrument without visualisation, when an electrocautery unit is on a high-power setting or inadvertently makes contact with an organ, or with a loop of bowel directly under the initial point of entry with the Hasson cannula.

Conversion to open cholecystectomy

Conversion to standard open cholecystectomy is indicated if a safe dissection of the structure in the triangle of Calot cannot be performed, because of dense adhesions/fibrosis from previous surgery, the presence of *Mirizzi* syndrome and severe acute disease with gross inflammatory oedema. Conversion to open cholecystectomy may be necessary because of the onset of above complications which cannot be readily and safely dealt with by laparoscopic approach.

2.6: POSTCHOLECYSTECTOMY SYNDROME

Introduction

Postcholecystectomy syndrome is defined as relapse of symptoms initially attributed to the presence of gallstones or emergence of new gastrointestinal complaints presumably related to the operation (8). Luman *et al* preferred to use the term postcholecystectomy symptoms rather than postcholecystectomy syndrome because it gives more accurate description (25). It ranges from mild ill-defined digestive symptoms to severe attacks of abdominal pain and jaundice. Other symptoms include postprandial abdominal pain, heartburn, nausea, vomiting, dyspepsia, abdominal bloating and flatulence.

Epidemiology

The reported incidence of persistent symptoms after cholecystectomy varies widely (Table 1). Studies that have assessed symptomatic outcome after open cholecystectomy have given varying results. Ros and Zambon prospectively evaluated 93 patients two years after cholecystectomy (16). Only 53 patients were completely free of symptoms. Konsten *et al* followed up over 300 Dutch patients through postal questionnaires to their general practitioners at median interval of 10 years after cholecystectomy and reported symptoms in 18%(26). Gilliland and Traverso followed up over 600 patients for a mean of 45 months and found that 88% of patients had complete symptomatic relief after cholecystectomy (27).

There were also several studies (Table 1) that have examined symptoms after laparoscopic cholecystectomy. In a three month follow up of 52 patients, Peters *et al* reported that 77% of patients considered their symptoms to have been cured by the procedure (28). In a comparative study of open and laparoscopic cholecystectomy, Velpen *et al* reported that 95% of patients considered that they had obtained overall symptomatic improvement and 93% of the patients were pleased with the end result regardless of the access used (3). Other studies by Mc Mahon *et al* (29), Wilson *et al* (16) and Peterli *et al* (30) support the findings of no difference in surgical approach by Velpen *et al.*

Qureshi *et al* analysed patients perceptions of postoperative symptoms and global satisfaction (31). They noted that 25% of patients complained of more than two symptoms postoperatively but 84% considered the procedure to be a complete success. Study by Wilson and Macintyre, evaluated symptomatic outcome of 115 patients a year after laparoscopic cholecystectomy and compared the outcome with 200 patients who

had undergone the open procedure. Over 90% of patients in both groups considered the procedures to have been successful in achieving symptomatic relief (32). McMahon *et al* also reported that over 90% of patients were improved by the operation (29). None of these studies assessed patients' symptoms in a prospective manner; only patients' postoperative symptoms were evaluated and patients were either expected to list preoperative symptoms (31), or their case notes were reviewed. Luman *et al* prospectively analysed symptoms before and after laparoscopic cholecystectomy (25). Patients undergoing laparoscopic cholecystectomy were given standard questionnaires. The same questionnaires were administered again six months after the operation. They found that 13% had persistent pain and 3% developed diarrhea at follow up. They also concluded that abdominal bloating and psychiatric medications were predictive for persistence of pain after laparoscopic cholecystectomy.

Recently published article by Peterli *et al* (33) in the World Journal of Surgery, December 2000 also analysed symptoms of postcholecystectomy syndrome after the surgery using standard questionnaire. They compared the prevalence of postcholecystectomy symptoms after open and laparoscopic cholecystectomy. There was no significant difference of the symptoms: 90% of patients after open and 94% after laparoscopic cholecystectomy had no or only minor symptoms.

Table 1: SEVERAL STUDIES ON POSTCHOLECYSTECTOMY SYNDROME IN THE ENGLISH LITERATURE

AUTHOR	n	DESIGN	CHOLECYSTECTOMY	INCIDENCE
Luman *et al* 1996(25)	100	Prospective (questionnaire)	laparoscopic	13 %
Peterli *et al* 2000(33)	397	Prospective (questionnaire)	Open (n=163) Laparoscopic (n=234)	10 % 6 % (excluding minor symptoms)
Wilson *et al* 1993(32)	315	Prospective (questionnaire)	Open (n=200) Laparoscopic (n=115)	6.6 % 6.0 %
Mc Mahon *et al* 1995(29)	299	Prospective (questionnaire)	Minilaparotomy (n=148) Laparoscopic (n=151)	Both 10 %
Qureshi *et al* 1993(31)	100	Prospective (questionnaire)	laparoscopic	39 %
Konsten *et al* 1993(26)	325	Prospective (questionnaire)	Open	18.5 %
Velpen *et al* 1993(3)	160	Prospective (questionnaire)	Open (n=80) Laparoscopic (n=80)	Both 18 %
Ros *et al* 1987(16)	124	Prospective (questionnaire)	Open	47 %
Gilliland *et al* 1990(27)	671	Retrospective	Open	18%

Aetiology

Removal of the gallbladder is associated with several physiological changes in the upper gastrointestinal tract which may account for the persistence of symptoms or the development of new symptoms after cholecystectomy (34). The cholecystosphincter of Oddi reflex (35), cholecysto-antral reflex (36) and cholecysto-oesophageal reflexes (37) are all disrupted, and a number of local upper gastrointestinal hormonal changes also occur after cholecystectomy (38,39). Thus, there is an increased incidence of gastritis, alkaline duodenogastric reflux and gastro-oesophageal reflux after cholecystectomy, all

of which may be the basis for postcholecystectomy syndrome (34). But, some authors(40) did not consider organic diseases outwith the biliary tract, such as hiatus hernia, duodenal ulcer, pancreatitis, duodenal diverticulitis and irritable bowel syndrome, as causes of postcholecystectomy syndrome.

Generally, causes of symptoms after cholecystectomy(29) may be divided into four categories:-

1. Common bile duct stones may have been overlooked.
2. Other gastrointestinal conditions such as gastro-oesophageal reflux, peptic ulcer disease, and functional motility disorder may have coexisted and their symptoms erroneously attributed to gallstones.
3. Loss of the reservoir function of the gallbladder has been shown to result in a number of adverse changes:-
    a. Impairment of the antropyloric motor unit.
    b. Increased duodenogastric reflux.
    c. Increased gastro-oesophageal reflux.
    d. Reduced bile salt pool which may result in subclinical fat malabsorption and diarrhea.
4. The abdominal wound may result in pain from nerve damage or the development of an incisional hernia.

Clinical manifestations

Clinical features of the syndrome consist of symptoms similar to those symptoms prior to cholecystectomy or symptoms referable to the biliary tract after the surgery. It ranges from mild ill-defined digestive symptoms to severe attacks of abdominal pain and

jaundice. Other symptoms include postprandial abdominal pain, heartburn, nausea, vomiting, dyspepsia, abdominal bloating and flatulence.

Bodvall(8) regards dyspepsia as the mildest form of the postcholecystectomy syndrome and this seems to be an integral part of gallstone disease, both before and after operation. Ros and Zambon(16) found that 22 of the 46 patients (almost 50%) had relapse of dyspepsia. Velpen *et al*(3) reported 18% of his study patients had diarrhea. Velpen *et al*(3), Luman *et al*(24) did not detect symptomatic relief from heartburn or de novo development of this symptom after laparoscopic cholecystectomy. The cure rate for nausea, vomiting, and early satiety was excellent as also reported by Velpen *et al.*

Luman *et al* found that preoperative abdominal bloating, constipation, and the use of psychotropic drugs were significantly more common in the symptomatic group(25). Patients with persistent pain after laparoscopic cholecystectomy had a higher prevalence of normal or only minimally inflamed gall bladder histology than the asymptomatic group. These findings suggest that persistent abdominal pain laparoscopic cholecystectomy may have been due to underlying irritable bowel syndrome and were unrelated to gall stones. Indeed, McMahon *et al* showed that patients with the postcholecystectomy syndrome tended to be anxious and depressed (29) and it is well known that patients with irritable bowel syndrome and "functional dyspepsia" tend to be neurotic, anxious, and depressed(14).

Possibility of scar–related pain could not be ruled out in those patients with normal investigation findings. In the era of open cholecystectomy high proportion of patients may attribute continued symptoms to the scar. Ros and Zambon(16) found that in 22% of their patients dull upper abdominal pain is attributed to the wound whereas Velpin *et al*(3) reported 9% of scar-related pain.

Management

Investigations are aiming towards finding the above causes of postcholecystectomy syndrome, which includes all modality of investigations of the hepatobiliary system namely liver function test (LFT), ultrasonography of the hepatobiliary system and endoscopic retrograde cholangiopancreatography (ERCP) (11).

The advent of ERCP has revolutionized the management of this syndrome. This investigation improved the assessment of patients as well as providing an opportunity for treatment at the same time especially if common duct stones are found.

Goenka *et al*(41) found that 117 (75%) out of 156 postcholecystectomy syndrome patients with successful ERCP had one or more abnormalities seen at ERCP. Bile duct stones (55 patients), benign strictures of bile duct (27 patients) were the common lesions detected. A significantly higher ($p<0.001$) positive diagnosis was possible at ERCP in patients presenting with jaundice (100%) as compared to those without jaundice (61.8%).

Only if the hepatobiliary causes have been excluded then other modality such as upper gastrointestinal endoscopy is considered.

OBJECTIVES

3.0: AIMS

1. To know the clinical epidemiology of the postcholecystectomy syndrome after two years and beyond of the laparoscopic cholecystectomy.

2. To find out the incidence of postcholecystectomy syndrome in patients underwent laparoscopic cholecystectomy in Hospital Universiti Sains Malaysia.

3. To study the approach and management of specific causes of postcholecystectomy syndrome.