

71 The gallbladder and bile ducts

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Absence of the cystic duct

Absence of the cystic duct

This 'anomaly' is usually pathological, indicating the recent passage of a stone or, in the presence of jaundice, a stone at the lower end of the cystic duct ulcerating into the CBD (Mirizzi syndrome). The main danger at surgery is damage to the bile duct; care is essential before division of any duct.

Absence of the gallbladder

Absence of the gallbladder

Rarely, the gallbladder is absent; failure to visualise it should not be mistaken for pathology. H Joachim Burhenne, 1925–1996, radiologist, Vancouver, Canada, described this technique in 1973. (a) (b) (c) (d) -

1 2 1 2 1 1 2 Figure 71.20 The main variations in gallbladder and cystic duct anat

omy. (a) Double gallbladder. (b) Septum of the gallbladder: (1) is the most common ('Phrygian cap'). (c) Diverticulum of the gallbladder. (d) Variations in cystic duct insertion.

The Phrygian cap (Figure 71.20) is present in 5% of gallbladders and may be mistaken for a pathological deformity .

Accessory cholecystohepatic duct

Accessory cholecystohepatic duct

Ducts passing directly into the gallbladder from the liver are not uncommon. Larger ducts should be closed, but before doing so the precise anatomy should be carefully ascertained to ensure that the right hepatic duct is not being ligated (Figure 71.21

BILIARY TRACT Biliary ascariasis

BILIARY TRACT Biliary ascariasis

The roundworm *Ascaris lumbricoides* commonly infests the intestines of inhabitants in Asia, Africa and Central America. It may enter the biliary tree through the ampulla of Vater and cause biliary pain. Complications include obstruction, strictures, suppurative cholangitis, liver abscesses and empyema of the gallbladder. In uncomplicated cases, antispasmodics can be given to relax the sphincter of Oddi and the worms may return to the intestine to be dealt with by anthelmintic drugs. Worms can be extracted by ERCP. Operation may be necessary to remove the worms or deal with complications.

Bile duct injuries

Bile duct injuries

About 15% of injuries to the bile ducts are recognised at the time of operation; in the remainder, the injury declares itself postoperatively either by profuse and persistent leakage of bile (if drainage has been provided; bile peritonitis if no drainage provided) or by deepening obstructive jaundice. When the obstruction is incomplete, jaundice is delayed until subsequent fibrosis renders the lumen of the duct inadequate. Any postoperative elevation in the serum bilirubin or suggestion of duct damage requires investigation to determine the nature of the injury. Abdominal USG may show collections, dilatation of the CBD and any associated vascular lesions. Abdominal CT defines the presence of focal fluid collections, ascites, biliary obstruction with an upstream dilatation in the acute phase or long-term sequelae of longstanding bile stricture, such as hepatic atrophy or signs of secondary biliary cirrhosis. CT may identify an associated vascular injury, such as to the right hepatic artery. (c) (d) - MRCP is the 'gold standard' for complete morphological evaluation of the biliary tree as it offers detailed information about the integrity of the biliary tract. It is helpful in determining the level and degree of injury. MRCP with magnetic resonance angiography is more informative as it may identify associated vascular injuries. A HIDA scan can confirm the presence of a bile leak or biliary obstruction. If available, ERCP should be considered because this is diagnostic of a bile leak, demonstrates ductal continuity, detects the site and type of injury, identifies residual/retained CBD stones and is potentially therapeutic. The most common bile leak following cholecystectomy is from the cystic duct. This can be treated by placing a biliary endoprosthesis (stent) in the CBD across the origin of the cystic duct. Surgical repair and the subsequent outcome are related to the level and degree of injury, in conjunction with the presence or absence of concomitant vascular injury. A number of classification systems have been proposed, with the Strasberg classification being commonly used (Figure 71.33). In a debilitated patient, temporary external biliary drainage may be achieved by passing a catheter percutaneously into an intrahepatic duct. Also, stents may be passed through strictures at the time of ERCP and left to drain into the duodenum. When the general condition of the patient improves, definitive surgery can be undertaken. The principles of surgical repair are the maintenance of the duct length and the restoration of biliary drainage. For a stricture of recent onset through which a guidewire can be passed, balloon dilatation with insertion of a stent is an acceptable option, provided the services of an experienced endoscopist are available. For benign stricture or duct transection, the preferred treatment is a Roux-en-Y hepaticojejunostomy performed by an experienced hepatobiliary surgeon. Biliary reconstruction in the presence of peritonitis, combined vascular and bile duct injuries and injury at or above

E4 E5 E6 (a) A bile leak from the cystic duct stump or minor (c) A bile leak from the divided right posterior sectoral E1, transected main bile duct with a stricture more than 2 cm from E3, stricture of the hilus with the right and left ducts in E5, stricture involving the right aberrant sectoral Br J Surg 2006; 93 (2): 158-68.)

of poor surgical outcome. The long-term impact of bile duct injury is a significant decrease in the patient's quality of life and work-related limitations.

Blood supply to the bile ducts

Blood supply to the bile ducts

The supraduodenal CBD is supplied by the left and right choledochal arteries, arising from the posterior superior pancreaticoduodenal artery below and the right (RHA) and left (LHA) hepatic arteries and cystic arteries above (Figure 71.1). The choledochal arteries give small branches that form the epicholedochal plexus. The communicating arcade connects the RHA and LHA and lies cranial to the confluence of the right and left hepatic ducts. The venous drainage of the extrahepatic bile ducts consists of the epicholedochal venous plexus that drains into two marginal veins that drain into the right gastric vein, posterior superior pancreaticoduodenal vein and superior mesenteric vein and connect to the hilar plexus. Anatomical variations The right hepatic artery can be tortuous (caterpillar turn/ Moynihan's hump) and may lie very close to the gallbladder and the cystic duct before giving off a short cystic artery (Figure 71.2b,c). Biliary and ductal anomalies include double cystic duct, separate insertion into the duodenum and anomalous low insertion of a right sectional duct (usually the posterior one, which puts this sectional duct at higher risk of injury).

(c) (b) tortuous common hepatic artery; (c) tortuous right hepatic artery with a

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Figure 71.3 Rouvière's sulcus. R4U line, Rouvière's sulcus segment IV umbilical /f_i ssure.

CHOLECYSTECTOMY

Preparation for operation

CHOLECYSTECTOMY Preparation for operation

- After appropriate history taking and assessment of the patient's fitness for the procedure, routine laboratory investigations including a coagulation screen and liver function tests should be checked. The patient must sign a consent form to indicate that he or she is fully aware of the procedure being undertaken, the alternative options and the risks involved including complications that may occur. Prophylactic antibiotics should be administered at the time of induction of anaesthesia. A second-generation cephalosporin is appropriate. Subcutaneous heparin and antiembolic stockings should be prescribed (Summary box 71.4). The various factors identified as predictors of difficult cholecystectomy are listed in Table 71.3, and the risk factors for the presence of CBD stones are listed in Table 71.4. Preparation for cholecystectomy

Appropriate history taking (jaundice) and assessment Full blood count Renal and liver function tests Prothrombin time Chest radiograph and electrocardiogram (if medically indicated) Antibiotic prophylaxis, second-generation cephalosporin at the time of induction Deep vein thrombosis prophylaxis Informed consent: patient is aware of the procedure being undertaken, the alternative options and the risks involved TABLE 71.3 Risk factors of difficult cholecystectomy. History Male gender, >65 years, interval between onset and presentation (>72–96 hours) in acute cholecystitis, previous multiple attacks, previous upper abdominal surgery, prior attempt at cholecystectomy (cholecystostomy) Physical examination Morbid obesity, high ASA score Laboratory tests Abnormal liver function tests Imaging (USG/CT/MRI-MRCP) Thick-walled gallbladder (>4–5 mm) Contracted gallbladder Distended gallbladder with impacted stone in the neck Gangrenous gallbladder/gallbladder perforation Mirizzi's syndrome/cholecystoenteric fistula Cirrhosis/extrahepatic portal vein obstruction (portal cavernoma) with portal hypertension Intraoperative Shrunken gallbladder, liver edge retracted with fissure/depression/puckering near the fundus, fatty or cirrhotic liver (difficult in retraction) ASA, American Society of Anesthesiologists; CT, computed tomography; MRCP, magnetic resonance cholangiopancreatography; MRI, magnetic resonance imaging; USG, ultrasonography.

CHOLECYSTOSES (CHOLESTEROLOSIS, POL YPOSIS, ADENOM

CHOLECYSTOSES (CHOLESTEROLOSIS, POL YPOSIS, ADENOMYOMATOSIS AND CHOLECYSTITIS GLANDULARIS PROLIFERANS)

This is a relatively uncommon group of conditions affecting the gallbladder, in which there are chronic inflammatory changes with hyperplasia of all tissue elements.

CHOLEDOCHAL CYST

CHOLEDOCHAL CYST

Choledochal cysts are congenital dilatations of the intra- and/ or extrahepatic biliary system. The pathogenesis is unclear. Anomalous insertions of the biliary-pancreatic junction are frequently observed, but whether or not these play a role in the pathogenesis of the condition is unclear. Todani and colleagues proposed a classification of cystic disease of biliary tract (Figure 71.24). Patients may present at any age with jaundice, fever, abdominal pain and a right upper quadrant mass on examination; 60% of cases are diagnosed before the age of 10 years. Pancreatitis is not an infrequent presentation in adults. Patients with choledochal cysts have an increased risk of developing cholangiocarcinoma, with the risk varying directly with the age at diagnosis. Takuji Todani, b. 1931, Department of Surgery, Okayama University Medical School, Okayama, Japan, modified Alonso-Lej's classification of choledochal cysts in 1977. USG confirms the presence of an abnormal cyst and magnetic resonance imaging (MRI)/MRCP will reveal the anatomy, in particular the relationship between the lower end of the bile duct and the pancreatic duct. CT is also useful for delineating the extent of intra- or extrahepatic dilatation. Radical excision of the cyst is the treatment of choice, with reconstruction of the biliary tract using a Roux-en-Y loop of jejunum. Complete resection is important because of an association with the later development of cholangiocarcinoma. Resection and Roux-en-Y reconstruction is also associated with a reduced incidence of stricture formation and recurrent cholangitis. Type III needs endoscopic management with sphincterotomy. It should be accompanied by biopsy of the cyst epithelium to exclude dysplasia in symptomatic cases and in young patients without symptoms.

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CONGENITAL ABNORMALITIES OF THE GALLBLADDER AND BI

CONGENITAL ABNORMALITIES OF THE GALLBLADDER AND BILE DUCTS Embryology

The hepatic diverticulum arises from the ventral wall of the foregut and elongates into a stalk to form the choledochus. A lateral bud is given off, which is destined to become the gallbladder and cystic duct. The embryonic hepatic duct sends out many branches that join up with the canaliculi between the liver cells. As is usual with embryonic tubular structures, hyperplasia obliterates the lumina of this ductal system; normally recanalisation occurs subsequently and bile begins to flow. During early fetal life the gallbladder is entirely intrahepatic.

CONGENITAL DILATATION OF INTRAHEPATIC DUCTS (CAROLI

CONGENITAL DILATATION OF INTRAHEPATIC DUCTS (CAROLI'S DISEASE)

This rare congenital condition is characterised by multiple irregular saccular dilatations of the intrahepatic ducts, separated by segments of normal or stenotic ducts, with a normal extrahepatic biliary system. In Caroli's syndrome, the biliary dilatation is associated with congenital hepatic fibrosis. The presentation is varied, with most patients presenting with abdominal pain, cholangitis or end-stage liver disease. The majority of patients present before the age of 30 years. The sex distribution is equal. Malignancy is a complication of longstanding disease. Management is multidisciplinary: cholangitis or jaundice are treated with appropriate antibiotic therapy and endoscopic or interventional stenting. Hepatic resection may be considered in patients with limited disease. Patients with diffuse disease and concomitant hepatic fibrosis are candidates for liver transplantation. Recurrence is common, particularly after resection, and long-term surveillance is required.

Ia Ib IVa Figure 71.24 Classification of choledochal cysts. Type Ia and Ib (80–90%): diffuse cystic dilatation; note the extension into the intrapancreatic portion in type Ib. Type II (3%): diverticulum of the common bile duct. Type III (5%): diverticulum within the intrapancreatic portion. Type IV (10%): extension into the liver; type IVa: fusiform dilatation of the entire extrahepatic bile duct with extension into the intrahepatic ducts; type IVb: multiple cystic dilatations involving only the extrahepatic bile duct. Type V: cystic dilatation only of the intrahepatic ducts.

Cancer of the gallbladder

Cancer of the gallbladder

Incidence Gallbladder cancer is extremely variable by geographical region and racial-ethnic groups; the highest incidence is among Chileans, Native Americans and residents in parts of northern India, where it accounts for as much as 9% of all biliary tract disease. Women appear to have a higher incidence across all geographical areas. In western practice, gallbladder cancer accounts for less than 1% of new cancer diagnoses. The disease usually presents in the seventh or eighth decade. The aetiology is unclear but there is a suggested association with pre-existing gallstone disease, implying that chronic inflammation may play a role. Calcification of the gallbladder wall, presumably due to chronic inflammation (porcelain gallbladder), is also associated with a small increased risk of cancer (Figure 71.5). Chronic infection may promote the development of cancer and the risk in typhoid carriers is significantly increased over that of the general population. Patients with PSC, especially with concomitant IBD, and those with an abnormal pancreatic- biliary junction are at greater risk of gallbladder cancer. In patients with gallbladder polyps (Figure 71.40) the risk of malignant transformation increases with increasing size of the polyp. Summary box 71.9 Gallbladder cancer

Pathology The majority (90%) of tumours are adenocarcinomas. Squamous carcinomas may arise from areas of mucosal squamous metaplasia. At operation, localised carcinomas are difficult to differentiate from chronic cholecystitis; the tumour most commonly is nodular and infiltrative, with thickening of the gallbladder wall, often extending to the whole gallbladder. The tumour spreads by direct extension into the liver, seeding of the peritoneal cavity and involvement of the perihilar lymphatics and neural plexuses. At the time of presentation, the majority of tumours are advanced. Clinical features Patients may be asymptomatic; symptoms, if present, are usually indistinguishable from those of benign gallbladder disease such as biliary colic or cholecystitis, particularly in older patients. Jaundice and anorexia are late features, heralding a low resectability rate and even fewer negative margins. A palpable mass is a late sign. Investigation Laboratory findings are generally non-specific but may be consistent with biliary obstruction. Non-specific findings include anaemia, leukocytosis and a mild elevation in transaminases. Serum CA19-9 and carcinoembryonic antigen may be elevated in approximately 80% of patients. The preoperative diagnosis is often made on USG and confirmed by CT thorax, abdomen and pelvis or MRI/MRCP. Preoperative staging should aim to determine the local extent of disease and exclude the presence of distant metastases. Percutaneous biopsy under radiological guidance may be considered to obtain tissue for pathological examination, but only in unresectable disease prior to palliative treatment. Laparoscopic examination is useful in staging the disease. Laparoscopy can detect peritoneal or liver metastases, which would preclude further surgical resection (Figure 71.41). PET scanning has a role in detecting metastatic disease. Treatment and prognosis The majority of patients have advanced disease at presentation and are not candidates for surgical therapy. Staging

Rare Similar presentation to benign biliary disease (gallstones) Diagnosis by USG, CT, MRI/MRCP
Most patients present with advanced disease Surgical resection in less than 10% – remainder receive palliative treatment Prognosis is poor Figure 71.40 Ultrasonography demonstrating a gallbladder polyp. Note the absence of an acoustic shadow (arrow).

laparoscopy is mandatory prior to formal laparotomy to detect occult metastases not picked up on imaging. Radical en bloc resection includes the gallbladder, wedge hepatectomy (2–5 cm of liver in the gallbladder bed or segments IVb and V if there is concomitant liver infiltration) or extended hepatectomy and bile duct resection if the bile duct is involved or the cystic duct margin is positive on intraoperative frozen section. Regional lymphadenectomy (paracholedochal portal, along the right hepatic artery and retroduodenal nodes) should be considered. The aim is to remove the tumour entirely and achieve negative histopathological margins. Some patients have the disease diagnosed following histopathological examination of the gallbladder after it has been removed. Summary box 71.10 Aims of staging gallbladder cancer (Table 71.10). In these cases, the need for further surgery is determined by the stage of disease. For early-stage disease confined to the mucosa of the gallbladder with a negative cystic duct margin and no evidence of recurrence on imaging, no further treatment is indicated. However, for transmural disease, a radical en bloc resection of the gallbladder fossa and surrounding wedge of liver along with the regional lymph nodes should be performed. If the initial procedure was performed laparoscopically, the surgeon should examine the laparoscopic port sites. Routine resection of port sites is no longer recommended. However, it is recognised that the finding of disease at the port sites is a sign of generalised peritoneal disease and carries a very poor prognosis. Adjuvant oral chemotherapy (capecitabine alone is preferred or in combination with gemcitabine/oxaliplatin) may derive survival benefit. Gallbladder cancer is a lethal disease with a grim prognosis; the median survival is less than 6 months and 5-year survival figures of 50% for localised gallbladder cancer (with chemotherapy) and 2–5% in patients with distant metastasis have been reported. For the majority of patients with advanced disease a non-operative approach to palliation is best. Obstructive jaundice can be relieved by endoscopic and/or percutaneous methods after discussion in the multidisciplinary team.

Figure 71.41 Laparoscopic staging in a patient with gallbladder cancer demonstrating gross peritoneal metastases. Assessment of local disease Detection of metastatic disease: Liver Peritoneum Lymphatics Extra-abdominal disease

Causal factors

Causal factors

Gallstones can be divided into three main types: cholesterol, pigment (brown/black) and mixed stones. In the USA and Europe, 80% of gallstones are cholesterol or mixed stones, whereas in parts of Asia 80% are pigment stones. Cholesterol or mixed stones contain 50–99% pure cholesterol plus an admixture of calcium salts, bile acids, bile pigments and phospholipids. Cholesterol is insoluble in water and is secreted from the canalicular membrane in phospholipid vesicles. Whether cholesterol remains in solution depends on the type and relative concentrations of phospholipids and bile acids in the bile. When bile is supersaturated with cholesterol and/or bile acid concentrations are low, unstable unilamellar phospholipid vesicles form, from which cholesterol crystals may nucleate. Obesity, a high-calorie diet and certain medications (e.g. oral contraceptives) can increase the secretion of cholesterol, while ileal disease or resection can deplete the bile acid pool and result in lithogenic bile (Figure 71.25). Nucleation of cholesterol monohydrate crystals from multilamellar vesicles is a crucial step in gallstone formation. Abnormal emptying of the gallbladder may aid the aggregation of nucleated cholesterol crystals; thus, removing gallstones without removing the gall bladder will inevitably lead to gallstone recurrence. Pigment stones contain <30% cholesterol. Overall, 20–30% of pigment stones are black; the incidence rises with age. For reasons that are unclear, patients with cirrhosis have a higher incidence of pigment stones. Black pigment stones are composed largely of an insoluble bilirubin pigment polymer mixed with calcium phosphate and calcium bicarbonate. These stones are associated with haemolysis, as in hereditary spherocytosis and sickle cell disease. Brown pigment stones contain calcium bilirubinate, calcium palmitate and calcium stearate, as well as cholesterol. Brown stones are more common in the bile ducts and are related to bile stasis and infection secondary to deconjugatable unconjugated bilirubinate precipitates. Brown pigment stones are also associated with the presence of foreign bodies within the bile ducts, such as endoprostheses (stents) or parasites such as *Clonorchis sinensis* and *Ascaris lumbricoides*.

Cholecystitis glandularis proliferans (adenomyomatosis)

Cholecystitis glandularis proliferans (adenomyomatosis)

Adenomyomatosis is an abnormality of the gallbladder characterized by overgrowth of the mucosa and thickening of the muscle wall, leading to cyst-like structures in the gallbladder wall or polypoid projections from the mucosa of the gallbladder and intramural diverticulae (diffuse adenomyomatosis). While generally not considered to be a premalignant condition, there is a clear association of adenomyomatosis with cholelithiasis.

in 2

These can be complicated by intramural, and later extramural, abscess and potentially fistula formation. If symptomatic, the patient is treated by cholecystectomy (Figure 71.28).

and general supportive care d Grade I (mild) c and advanced LC technique available a c e Antibiotics a d Grade II and general (moderate) b supportive care Urgent/early GB drainage No negative predictive e Antibiotics Grade III and general (severe) organ care Negative predictive factors present and/or i no FOSF Figure 71.26 Tokyo Guidelines for the management of acute cholecystitis. c general supportive care fail to control in /f_ l amination. CCI 5 or less and/or ASA-PS class II or less (low risk). e class III or greater (not low risk). Blood culture should be taken before initiation of administration of antibiotics. g formed during GB drainage. In cases of serious operative dif /f_ i culty, bail-out procedures including conversion should be considered. predictive factors: jaundice (TBil ≥ 2 mg/dL), neurological dysfunction, respiratory dysfunction. j reversible after admission and before early LC. CCI 4 or greater, ASA-PS 3 or greater are high risk. advanced laparoscopic techniques are available. ASA-PS, American Society of Anesthesiologists physical status; CCI, Charlson comorbidity index; GB, gallbladder; LC, laparoscopic cholecystectomy; PS, performance status; TBil, total bilirubin. (Reproduced with permission from Okamoto K et al . Tokyo Guidelines 2018: /f_ l owchart for the management of acute cholecystitis.

Cholecystography

Cholecystography

Oral and intravenous cholecystography have been replaced by more accurate imaging modalities.

Choledochotomy

Choledochotomy

When faced with a patient with cholangitis due to stones in the CBD, and minimally invasive techniques for stone extraction are not possible, the surgeon must undertake laparotomy , drain the CBD and remove the stones through a longitudinal incision in the duct. When the duct is clear of stones, on-table - or - - - lo Hospital, Milan, Italy .

Figure 71.34 This patient presented with jaundice 4 days after laparo

scopic cholecystectomy. The duct contained multiple stones. (b) (a) Figure 71.35 (a) Endoscopic sphincterotomy; (b) extraction of a stone from the bile duct through an ampulla (courtesy of Dr Amit Maydeo, Mumbai, India).

choledochoscopy should be performed to confirm clearance. A T-tube is inserted and the duct closed around it; the long limb is brought out on the right side and bile is allowed to drain externally . When the bile becomes clear and the patient has recovered, a cholangiogram is performed. If residual stones are found, the tube is left in place for 6 weeks so that the track is 'mature'. The radiologist can then use the track for percutaneous removal of the stones (Burhenne) (Figure 71.36). Once the radiologist has removed the tube, the track will close and the patient will recover. Such residual small stones are now usually managed with endoscopic methods.

Figure 71.36 Extraction of a stone from the common bile duct by the Burhenne technique. (b) A steerable catheter is manipulated into the duct and a basket placed around the stone. T-tube track.

Cholescintigraphy

Cholescintigraphy

^{99m}Tc Technetium-^{99m} (^{99m}Tc)-labelled derivatives of iminodiacetic acid (hepatobiliary iminodiacetic acid [HIDA]; the utility of a hepatobiliary IDA or HIDA scan is that the radiotracer follows

Figure 71.8 Endoscopic ultrasonography. CBD, common bile duct; PD, pancreatic duct.

LIVER GALLBLADDER SMALL BOWEL the bilirubin metabolic pathway) are excreted into the bile. This allows visualisation of the biliary tree and gallbladder. In 90% of normal individuals the gallbladder is visualised within 30 minutes following injection, with 100% being seen within 1 hour (Figure 71.9). The bowel is seen usually within an hour in the majority of patients. Non-visualisation of the gallbladder is suggestive of acute cholecystitis. If the patient has a contracted gallbladder, as often occurs in chronic cholecystitis, visualisation may be reduced or delayed. An abnormally low gallbladder ejection fraction may be suggestive of gallbladder dyskinesia; however, interpretation of cholescintigraphy in this context is controversial. Biliary scintigraphy may also be helpful in diagnosing bile leaks, biliary obstruction and in testing the patency of a bilioenteric anastomosis.

Figure 71.9 Dimethyl iminodiacetic acid (HIDA) scan before and after a meal to evaluate gallbladder function (courtesy of the Department of Nuclear Medicine, KEM Hospital, Mumbai, India).

Cholesterol polyposis of the gallbladder

Cholesterol polyposis of the gallbladder

USG may show a non-mobile defect in the gallbladder lumen with no acoustic shadow . The differential is an adenomatous polyp, and interval follow-up is indicated to ensure stability . Surgery is advised only if there is a diagnostic dilemma.

Cholesterolosis ('strawberry gallbladder')

Cholesterolosis ('strawberry gallbladder')

Cholesterolosis (cholesterosis) is characterised by the accumulation of lipids (triglycerides, cholesterol precursors and cholesterol esters) in the mucosa of the gallbladder wall. These nodules are less than 1 mm in diameter in about two-thirds of cases, which gives the mucosa a coarse and granular appearance. The nodules in the remaining one-third of cases are larger and polypoid in appearance (polypoid form). The lipid accumulation creates yellow deposits on a background of hyperaemic mucosa ('strawberry gallbladder'). It may be associated with cholesterol stones (Figure 71.27).

Clinical features

Clinical features

About one-third of patients are jaundiced at birth; in all affected babies, jaundice is present by the end of the first week and deepens progressively. The meconium may be a little bile-stained, but later the stools are pale and the urine is dark. Pruritus is severe. Clubbing and skin xanthomas, probably related to raised serum cholesterol, may be present. Prolonged steatorrhea gives rise to osteomalacia (biliary rickets). Liver function tests show an obstructive pattern with elevated bilirubin and alkaline phosphatase (ALP). Associated anomalies occur in about 20% of cases and include cardiac lesions, polysplenia, situs inversus, absent vena cava and preduodenal portal vein. Biliary atresia may be suspected prenatally, when a cystic structure is observed in the porta hepatis on fetal USG.

Patent Atretic Atretic I IIa Figure 71.23 Classification of biliary atresia. Gallbladder filling provides a clue to the type of atresia.

Clinical presentation

Clinical presentation

Gallstones are being increasingly detected incidentally during imaging for other symptoms. Prophylactic cholecystectomy is not usually indicated since the risk of developing serious complications is low. Longitudinal follow-up study of individuals with silent gallstones has shown that over 20 years only 18% developed biliary pain; the mean yearly probability was 2% during the first 5 years, 1% during the second 5 years and 0.5% during the third 5 years. If symptoms occur, patients typically complain of right upper quadrant or epigastric pain, which may radiate to the back. This may be described as colicky but the typical biliary 'colic' more often is dull, continuous and severe, lasting for several minutes or even hours, with associated nausea and vomiting (Chapter 63). Frequently, pain starts during the night and wakes the patient; minor episodes may occur intermittently during the day. In the majority of cases the process is limited by the stone slipping back into the body of the gallbladder. The development of acute cholecystitis is marked by fever. Associated symptoms that have a questionable relation to gallstones include dyspepsia, flatulence, food intolerance, particularly to fats, and some alteration in bowel frequency. As pain resolves (spontaneously or with medications) the patient improves and is able to eat and drink again, often only to suffer further episodes. A patient may have several such episodes over a few weeks and then no symptoms for some months. This may culminate in a contracted non-functioning gallbladder with the - - -

Impaired gall- Supersaturated bladder function bile • Emptying • Age • Absorption • Sex • Excretion • Genetics • Obesity • Diet Absorption/enterohepatic circulation of bile acids Cholesterol • Deoxycholate nucleating factors • Bowel transit time • Mucus • Faecal enteric flora • Glycoprotein • Ileal resection • Infection • Cholestyramine Figure 71.25 Factors associated with gallstone formation.

sis is given in Summary box 71.3. In acute cholecystitis the right upper quadrant tenderness is exacerbated during inspiration by palpation in the right sub costal region (Murphy's sign). A mass may become palpable as the omentum walls off an inflamed gallbladder. If resolution does not occur, empyema of the gallbladder may result. The wall may become necrotic and perforate, with the development of localised peritonitis (Table 71.1). Occasionally, complete obstruction of the cystic duct leads to reabsorption of bile salts by the gallbladder epithelium and secretion of uninfected mucus, resulting in a mucocele of the gallbladder. Jaundice may ensue if the gallstone migrates from the gallbladder and obstructs or compresses the CBD (Mirizzi). Rarely, a large solitary gallstone may erode the gallbladder wall, causing a cholecystoduodenal fistula and subsequent bowel obstruction, known as gallstone ileus. A palpable, non-tender gallbladder in the presence of jaundice may portend a more sinister diagnosis as a palpable gallbladder in the presence of jaundice is unlikely to be due to gallstones (Courvoisier) and usually results from a distal common duct obstruction secondary to periampullary malignancy. Summary box 71.2 Effects and complications of gallstones /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

/uni25CF /uni25CF /uni25CF /uni25CF Summary box 71.3 Differential diagnosis of acute cholecystitis /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Biliary colic Biliary obstruction (jaundice) Acute cholecystitis Acute cholangitis Chronic cholecystitis Acute pancreatitis Empyema of the gallbladder Intestinal obstruction (gallstone ileus) Mucocele of the gallbladder Perforation of the gallbladder Common Uncommon Appendicitis Acute pyelonephritis Perforated peptic ulcer Myocardial infarction Acute pancreatitis Pneumonia – right lower lobe

Clonorchiasis (Asiatic cholangiohepatitis)

Clonorchiasis (Asiatic cholangiohepatitis)

This disease is endemic in the Far East. The fluke inhabits the bile ducts, including the intrahepatic ducts. Fibrous thickening of the duct walls occurs. Many cases are asymptomatic. Complications include biliary pain, stones, cholangitis, cirrhosis and bile duct carcinoma. Because a process of recurrent stone formation is established, choledochojejunostomy with a Roux loop fixed to the adjacent abdominal wall is performed in some centres to allow easy subsequent access to the duct system.

Complications of cholecystectomy

Complications of cholecystectomy

Recovery after laparoscopic cholecystectomy is associated with less pain and faster return to normal activity than open cholecystectomy. The majority of elective patients can have this performed as a day case; however, any patient looking unwell in the postoperative period, with untoward symptoms such as fever, chills or abdominal pain, should be kept under observation. Complications can occur in 10–15% of cases. Serious complications fall into two major areas: access complications and bile duct injuries. The latter are rare, occurring in approximately 0.5% following laparoscopic cholecystectomy. In the main, biliary injury results from poor dissection and a failure to define the surgical anatomy adequately. Controversy exists as to whether operative cholangiography reduces the incidence of bile duct injury. The majority of surgeons use cholangiography only in selected cases. The operative mortality for cholecystectomy is less than 1%. Factors increasing the risk for postoperative mortality include advanced age, comorbid conditions and an acute presentation. Patients who develop jaundice in the postoperative period need urgent investigation. This is especially true if the jaundice (b) (a) is attributed to infection and cholangitis. The first step following resuscitation and administration of appropriate antibiotics is to undertake urgent USG. This will demonstrate whether there is intra- or extrahepatic ductal dilatation. The anatomy may need to be defined by MRCP or ERCP. The latter is undertaken when therapeutic manoeuvres are planned, such as the removal of an obstructing stone or the insertion of a stent across a biliary leak. If a fluid collection is present in the subhepatic space, drainage catheters may be required. These can be inserted under radiological control or, if this expertise is not available, at open operation. Small biliary leaks will usually resolve spontaneously, especially if there is no distal obstruction. If the CBD is damaged, the patient should be referred to an appropriate expert for reconstruction.

<2 cm

“ 2 cm E1 E2 E3 Figure 71.33 Schematic representation of the Strasberg classification of bile duct injuries. (a) Biliary radical in the gallbladder fossa. (b) An occluded right posterior sectoral duct. (c) A bile leak from the main bile duct without any major tissue loss. (d) A bile leak from the main bile duct at the hilus. E2, transected main bile duct with a stricture less than 2 cm from the hilus. E3, communication between the right and left hepatic ducts. E4, stricture of the hilus with separation of the right and left hepatic ducts. E5, stricture of the main bile duct. E6, complete excision of the extrahepatic ducts involving the confluence (this injury is not described in Strasberg's classification). (After Connor

S, Garden OJ. Bile duct injury in the era of laparoscopic cholecystectomy.

Computed tomography

Computed tomography

Unlike USG, computed tomography (CT) is less affected by body habitus and is not operator dependent. It allows visualisation of the liver, bile ducts, gallbladder and pancreas. CT findings in acute cholecystitis include gallbladder distension, gallbladder wall thickening, subserosal oedema, pericholecystic fat stranding and pericholecystic fluid collection. It is particularly useful in detecting hepatic and pancreatic lesions and is the modality of choice in the staging of cancers of the liver, gallbladder, bile ducts and pancreas. It can identify the extent of the primary tumour, define the relationship of the tumour to other organs and blood vessels (Figure 71.10) and detect the presence of enlarged lymph nodes or metastatic disease. However, as only 75% of gallstones are identified by CT , it is not used as a screening modality for uncomplicated gallstones.

GALLBLADDER SMALL BOWEL -

Figure 71.10 Computed tomography showing hilar mass (arrow).

Diagnosis

Diagnosis

A clinical diagnosis of acute cholecystitis must be confirmed with radiological and laboratory investigations. USG is the first choice for imaging; however, the sensitivity and specificity of diagnosing acute cholecystitis is increased when combined with positive clinical and/or laboratory findings (Table 71.1 Ludwig Georg Courvoisier , 1843–1918, surgeon, Basel, Switzerland, made his observation in 1890. No mention was made of either gallbladder tenderness or malignancy . - -

criteria for acute cholecystitis. A. Local signs of inflammation, etc. 1) Murphy's sign 2) Right upper quadrant pain/tenderness/mass B. Systemic signs of inflammation, etc. 1) Fever 2) Elevated CRP 3) Elevated WBC count C. Imaging findings Imaging findings characteristic of acute cholecystitis: Suspected diagnosis : 1 item in A + 1 item in B Definite diagnosis : 1 item in A + 1 item in B + C CRP , C-reactive protein; WBC, white blood cell. Reproduced with permission from Yokoe M et al . Tokyo Guidelines 2018: diagnostic criteria and severity grading of acute cholecystitis (with videos). J Hepatobiliary Pancreat Sci 2018; 25 (1) 41–54.

Differential diagnosis

Differential diagnosis

This includes all causes of cholestatic jaundice in a neonate; namely, α -antitrypsin deficiency, cholestasis associated with 1 intravenous feeding, choledochal cyst and inspissated bile syndrome. The most common differential diagnoses are Alagille syndrome (biliary atresia, congenital heart disease, skeletal and other abnormalities), progressive familial intra-hepatic cholestasis and cystic fibrosis. Neonatal hepatitis is the most difficult to differentiate. Liver biopsy and radionuclide excretion scans are helpful.

Diverticulosis of the gallbladder

Diverticulosis of the gallbladder

Diverticulosis of the gallbladder is usually manifest as black pigment stones impacted in the outpouchings of the lacunae of Luschka. This may be demonstrated by cholecystography , Mary E Charlson , contemporary , Clinical Epidemiologist, Weill Cornell Medical Center, New York, NY , USA. Daniel Elmer Salmon , 1850–1914, veterinary pathologist, Chief of the Bureau of Animal Industry , Washington, DC, USA. especially when the gallbladder contracts after a fatty meal. There are small dots of contrast medium within and outside the gallbladder wall (Figure 71.29). The treatment is chole - cystectomy .

Observation g Early LC g

Urgent/early LC f Delayed/ elective

g LC k Advanced centre g Early LC

j and good PS h factors and i FOSF

j Poor PS k or not Delayed/ j

elective Good PS g LC Urgent/early

h f GB drainage j Observation Poor

PS a b Antibiotics and general

supportive care successful.

Antibiotics and d CCI 6 or greater
and/or ASA-PS f A bile culture
should be per

h Negative i FOSF: favourable organ system failure = rapidly k Advanced centre = intensive care
and J Hepatobiliary Pancreat Sci 2018; 25 (1): 55-72.)

EXTRAHEPATIC BILIARY ATRESIA Aetiology and physiology

EXTRAHEPATIC BILIARY ATRESIA Aetiology and physiology

Biliary atresia is present in approximately 1 in 12 000 live births and affects males and females equally. The aetiology is unclear; the extrahepatic bile ducts are progressively destroyed by an inflammatory process that starts around the time of birth. Intrahepatic changes also occur and eventually result in biliary cirrhosis and portal hypertension. Untreated, death from the consequences of liver failure occurs before the age of 3 years. The Japanese and Anglo-Saxon classification describes three main types (Kasai) (Figure 71.23): type I: atresia restricted to the CBD; type II: atresia of the common hepatic duct; type III: a patent gallbladder and a patent CBD are present; type IV: the gallbladder, cystic duct and CBD are also obliterated; type V: atresia of the right and left hepatic ducts and the entire extrahepatic biliary tree. Hats worn by the people of Phrygia, an ancient country in Asia Minor; they resemble the liberty cap of the French Revolution. Pablo Luis Mirizzi, 1893–1964, surgeon, Córdoba, Argentina. Morio Kasai, 1922–2008, Professor of Surgery, Tokyo University, Tokyo, Japan. -).

RP RP Figure 71.21 Patterns of cystic duct anatomy. Note segment VI drain

age into the cystic duct and drainage of the right posterior

sectorial duct (RP) into the neck of the gallbladder or an accessory duct (duct of Luschka). Figure 71.22 Magnetic resonance cholangiopancreatography demon

strating low insertion of the cystic duct (thick arrow) into the common bile duct (thin arrow).

Endoscopic retrograde cholangiopancreatography

Endoscopic retrograde cholangiopancreatography

This technique is now used only as a therapeutic modality in patients with obstructive jaundice; USG and MRCP have taken over the diagnostic aspect. Using a side-viewing endoscope the ampulla of Vater is identified and cannulated. Injection of water-soluble contrast into the bile duct provides excellent images of the ductal anatomy (Figure 71.13) and can identify causes of obstruction such as calculi (Figure 71.14) and malignant strictures (Figure 71.15). Bile aspirates can be obtained and sent for cytological and microbiological examination and brushings can be taken from strictures for cytology . Therapeutic interventions such as stone removal or stent placement to relieve obstruction can be performed simultaneously . Cholangioscopy is a relatively new technique in which a thin scope is inserted through the channel of an ERCP scope to visually inspect the bile duct. The main indications include indeterminate or unexplained biliary strictures , nodules or masses and crushing difficult-to-remove bile duct stones with lithotripsy . - -

Figure 71.13 Endoscopic retrograde cholangiopancreatography: normal cholangiogram.

Figure 71.14 Endoscopic retrograde cholangiopancreatography: common duct obstruction due to stone (courtesy Dr Amit Maydeo, Mumbai, India). Figure 71.15 Endoscopic retrograde cholangiopancreatography: partial occlusion of bile duct by malignant stricture (arrow).

Figure 71.16 Transhepatic cholangiogram showing stricture of common hepatic duct (courtesy of Ms Phyllis George, FRCS, London, UK).

Endoscopic ultrasonography

Endoscopic ultrasonography

Endoscopic ultrasonography (EUS) utilises an endoscope with an ultrasound transducer at its tip, which allows the endoscopist to visualise the liver and biliary tree from within the stomach and duodenum (Figure 71.8). It is accurate in detecting choledocholithiasis and in the diagnosis and staging of pancreatic and periampullary cancers. Biopsies can be taken from suspicious areas for cytological and histological analysis.

FURTHER READING

FURTHER READING

Carter DC, Russell RCG, Pitt HA, Bismuth H (eds). Rob and Smith's operative surgery: hepatobiliary and pancreatic surgery. London: Chapman & Hall, 1996. Dooley JS, Lok A, Burroughs A, Heathcote J (eds). Sherlock's diseases of the liver and biliary system, 12th edn. Oxford: Wiley-Blackwell, 2011. - Garden OJ, Parks RW . Hepatobiliary and pancreatic surgery: a companion to specialist practice, 5th edn. New York: Saunders Elsevier, 2013. Jarnagin W . Blumgart's surgery of the liver, pancreas and biliary tract, 5th edn. New York: Elsevier, 2012. Rocha FG, Matsuo K, Blumgart LH, Jarnagin WR. Hilar cholangio - carcinoma: the Memorial Sloan-Kettering Cancer Center experience. J Hepatobiliary Pancreat Sci 2010; 17 (4): 490-6. Society of American Gastrointestinal and Endoscopic Surgeons. The SAGES safe cholecystectomy program , 2015. Available from: <https://www.sages.org/safe-cholecystectomy-program> (accessed October 2020). Takada T (ed.). Tokyo Guidelines 2018: updated Tokyo Guidelines for the management of acute cholangitis/acute cholecystitis. J Hepatobiliary Pancreat Sci 2018; 25 (1): 1-114.

Floating gallbladder

Floating gallbladder

The gallbladder may hang on a mesentery , which makes it liable to undergo torsion.

Functions of the gallbladder

Functions of the gallbladder

The gallbladder is a reservoir for bile. During fasting, the resistance to flow through the sphincter of Oddi is high, and bile excreted by the liver is diverted to the gallbladder. After feeding, the resistance to flow through the sphincter is reduced, the gallbladder contracts and bile enters the duodenum. The motor responses of the biliary tract are in part affected by the hormone cholecystokinin. An additional function of the gallbladder is the concentration of bile by 5–10 times by active absorption of water, sodium chloride and bicarbonate via the mucous membrane. The gallbladder mucosa also secretes approximately 20 mL of mucus per day. If the cystic duct is completely obstructed in an otherwise healthy gallbladder, a mucocele may develop as a result of ongoing mucus secretion. Fred Bates Lund, 1865–1950, surgeon, Boston, MA, USA. The node was also named after the Italian anatomist and physician who first identified the node around 1787. The Mercedes-Benz sign takes its name from the insignia on the bonnet of a Mercedes-Benz car -

Figure 71.4 Plain radiograph showing radio-opaque stones with the centre containing radiolucent gas in a triradiate or biradiate fissure ('Mercedes-Benz' or 'seagull' sign).

GALLSTONES (CHOLELITHIASIS)

GALLSTONES (CHOLELITHIASIS)

Gallstones are the most common biliary pathology . It is estimated that gallstones affect 10–15% of the population in western societies. In the UK, the prevalence of gallstones at the time of death is estimated to be 17%. Gallstones are asymptomatic in the majority of cases (>80%). Approximately 1–2% per year will develop symptoms requiring surgery , making cholecystectomy one of the most common operations performed.

Gallbladder polyps

Gallbladder polyps

Polyps of the gallbladder are incidental findings during radiological imaging of the abdomen. The polyps are more often benign (cholesterol polyps, adenomyomas, inflammatory, adenomas or miscellaneous) but may be malignant (adenocarcinoma [80%] or squamous cell carcinoma, or cystadenomas). A majority of polyps remain stable. Cholecystectomy should be considered in symptomatic patients or as prophylaxis to prevent malignant transformation in those who also have gallstones, primary sclerosing cholangitis (PSC), biliary have Carl Freiherr von Rokitansky, 1804–1878, pathologist, Vienna, Austria. Karl Albert Ludwig Aschoff, 1866–1942, pathologist, Freiburg, Germany. colic or pancreatitis. Polyps in patients older than 50 years, sessile polyps with wall thickening greater than 4 mm and polyps larger than 10 mm merit cholecystectomy. Smaller polyps should be kept under observation and need surgery if the size is increasing.

Figure 71.29 Cholecystogram showing diverticulosis with dots of contrast medium in the gallbladder wall. Figure 71.30 Xanthogranulomatous cholecystitis. Infiltrates in the wall of the gallbladder show foamy macrophages (arrow), giant cells and lymphoplasmic cells in the background (courtesy of Dr Amita Joshi, Mumbai, India).

Gallstone ileus

Gallstone ileus

Gallstone ileus is an infrequent complication (0.4%) of cholelithiasis, occurring as a result of impaction of one or more gallstones within the gastrointestinal tract. It is seen more often than Leon Bouveret, 1850–1929, physician, Lyon, France. These three constitute Rigler's triad. Leo George Rigler, 1896–1979, Professor of Radiology, University of California, Los Angeles, CA, USA. of acute cholecystitis leads to erosion of inflamed tissues, resulting in a cholecystointestinal fistula. A majority of small gallstones pass through the intestines spontaneously. However, gallstones of size 2–5 cm get impacted, usually in the terminal ileum or at the ileocecal valve owing to the relatively narrow lumen and less active peristalsis here. Less common locations include the stomach and the duodenum (Bouveret's syndrome). Impacted stones may lead to necrosis and perforation followed by peritonitis. Clinical manifestations include acute, intermittent or chronic episodes of partial or complete gastrointestinal obstruction. Physical examination may be non-specific or may show signs of obstruction: dehydration, abdominal distension and tenderness, with high-pitched bowel sounds, and obstructive jaundice. A plain abdominal radiograph shows: a partial or complete intestinal obstruction; a pneumobilia or contrast material in the biliary tree; an aberrant rim-calcified or total-calcified gallstone; a change in the position of such a gallstone on serial films ('tumbling sign'). CT is considered superior to plain radiographs or USG, with a sensitivity of up to 93%. It additionally shows an abnormal gallbladder with air, an air-fluid level or fluid accumulation with an irregular wall. In addition to the management of intestinal obstruction, enterolithotomy has been the most common surgical procedure performed. A longitudinal incision is made on the antimesenteric border proximal to the site of gallstone impaction, and the gallstone is brought proximally to a non-oedematous segment of the bowel by gentle manipulation and extracted. A cholecystoenteric fistula should not be resected unless the patient is stable and there are residual gallstones that may cause infection or recurrent ileus (see Chapter 78).

Gallstones in pregnancy

Gallstones in pregnancy

Acute cholecystitis is the second most common non-obstetric indication for surgery in pregnant women. Hormonal (oestrogen) changes during pregnancy increase cholesterol secretion and progesterone reduces bile acid secretion, reducing the ability of bile to solubilise cholesterol; bile becomes supersaturated with cholesterol. Progesterone also slows gallbladder emptying, which further promotes the formation of stones owing to bile stasis. Prepregnancy obesity, multiparity, increasing age and genetic predisposition are risk factors. Acute right upper quadrant/epigastric pain in pregnancy may be due to severe pre-eclampsia and the HELLP syndrome (haemolysis, elevated liver enzymes, low platelet count), acute fatty liver, abruptio placentae, uterine rupture or intra-amniotic infection. USG and non-contrast MRI are acceptable diagnostic modalities. For women in their first trimester, the mainstay of treatment for mild cases is conservative. Non-steroidal anti-inflammatory drugs are effective analgesics but are generally avoided in pregnancy, especially after 32 weeks of gestation, because of potential adverse fetal effects, e.g. premature closure of the ductus arteriosus. In the second trimester, with moderate or severe disease, good surgical candidates (American Society of Anesthesiologists [ASA] I or II) should undergo cholecystectomy during their initial hospitalisation as there is a high risk of recurrence or serious complications. In the third trimester, non-operative medical management with antibiotics and fluid therapy should be initiated. The patient should be re-evaluated after delivery. Generally, a waiting period of 6 weeks following delivery is preferred to allow the mother to recover from the delivery, bond with the infant and regain her strength.

Hydatid disease

Hydatid disease

A large hydatid cyst may obstruct the hepatic ducts. Sometimes, - a cyst will rupture into the biliary tree and its contents cause obstructive jaundice or cholangitis, requiring appropriate surgery (see Chapter 6).

IMAGING Plain radiographs

IMAGING Plain radiographs

A plain radiograph of the gallbladder will show radio-opaque stones in 10% (Figure 71.4). It may also show calcification of the gallbladder – the rare ‘porcelain’ gallbladder (Figure 71.5). Paolo Mascagni , 1752–1815,

Figure 71.5 Porcelain gallbladder.

atous cholecystitis) (Figure 71.6). Gas in the biliary tree may also be seen after endoscopic sphincterotomy or following a surgical anastomosis.

Imaging studies and biopsy

Imaging studies and biopsy

Fasting USG is the gold standard when biliary atresia is suspected. A shrunken gallbladder, a hyperechogenic liver hilum ('triangular cord sign') or a cyst at the liver hilum with out bile duct dilatation with associated anomalies support the diagnosis. Hepatobiliary scintigraphy may reveal the diagnosis but MR CP is highly sensitive and specific in the diagnosis. Inflammatory cells, a fibrotic liver parenchyma exhibiting signs of cholestasis and biliary neoductal structures establishes the definite diagnosis on liver biopsy . Cholangiography is required to define the surgical anatomy .

Immunoglobulin G4-related cholangitis

Immunoglobulin G4-related cholangitis

This recently recognised entity presents with diffuse or segmental narrowing of the intra- or extrahepatic bile ducts. Its features may make differentiation from PSC, cholangiocarcinoma and pancreatic cancer difficult. However, patients often have elevated serum IgG4 levels and concomitant autoimmune pancreatitis, IgG4-related sialadenitis or retroperitoneal fibrosis. Biliary biopsies show lymphoplasmacytic sclerosing cholangitis. Treatment is with systemic steroids. Failure to respond to steroid therapy should make one reconsider the diagnosis and exclude an underlying malignancy.

Introduction

Introduction

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Laparoscopic cholecystectomy

Laparoscopic cholecystectomy

Laparoscopic cholecystectomy is the procedure of choice for the majority of patients. The indications and preparation for cholecystectomy are the same whether it is performed by laparoscopy or by open technique. The patient is placed supine on the operating table. Following induction and maintenance of general anaesthesia, the abdomen is prepared in a standard fashion. Pneumoperitoneum is established. The authors' preference is to use an open subumbilical cut down with direct visualisation of the peritoneum to place the initial port. This port will function as the camera port. An angled telescope (30°) is preferred. Many surgeons prefer a 'closed' technique using a Janos Verres, 1903–1979, chest physician and chief of the Department of Internal Medicine, The Regional Hospital, Kapuvár, Hungary. Verres needle to establish pneumoperitoneum (see Chapter 7). Recently, single-port laparoscopic cholecystectomy has been described. Proponents report decreased postoperative pain and improved cosmesis. However, systematic reviews have reported a higher failure rate, longer operative time and increased blood loss without any substantive benefits with the technique. Additional operating ports are inserted in the subxiphoid area and in the right subcostal area. The patient is placed in a reverse Trendelenburg position slightly rotated to the left. This exposes the fundus of the gallbladder, which is retracted towards the diaphragm. The neck of the gallbladder is then retracted towards the right iliac fossa, exposing Calot's triangle. The key, as in open surgery, is the identification and safe dissection of Calot's triangle (Table 71.5). This area is laid wide open by dividing the peritoneum on the posterior and anterior aspects. The cystic duct is carefully defined, as is the cystic artery. The gallbladder is separated from the liver bed for about 2 cm to allow confirmation of the anatomy. Unless there are specific indications, routine cholangiogram is not performed. However, if doubt exists regarding the anatomy, cholangiogram is warranted. Real-time intraoperative imaging using indocyanine green (ICG) fluorescence cholangiography (with special scopes and imaging system) improves visualisation of the biliary tree during laparoscopic cholecystectomy and enables better visualisation and identification of the biliary tree. It can be considered a means of increasing the safety of laparoscopic cholecystectomy. This is likely to reduce risk of biliary duct injury. Once the anatomy is clearly defined and the triangle of Calot has been laid wide open, the cystic duct and artery are clipped and divided. The gallbladder is then removed from its bed by sharp or cautery dissection and, once free, removed via the umbilicus in a retrieval bag.

stones. Further Abdominal Liver History of Risk of evaluation USG: CBD function cholangitis or CBD required diameter tests pancreatitis stones Low, Absent Normal ≤ 6 mm None 2–3% Medium, Present 2×8 – 10 mm MRCP +/- 20–40% normal ERCP stone extraction High, Present, with $2 \times \geq 10$ mm MRCP +/- 50–80% jaundice normal ERCP stone extraction ERCP, endoscopic retrograde cholangiopancreatography; MRCP, magnetic resonance

cholangiopancreatography; USG, ultrasonography.

Late symptoms after cholecystectomy

Late symptoms after cholecystectomy

In up to 15% of patients, cholecystectomy fails to relieve the symptoms for which the operation was performed. Such patients may be considered to have a 'postcholecystectomy' syndrome. In most such cases, this is merely a continuation of earlier symptoms. A detailed history with full investigation should be undertaken to confirm the diagnosis and exclude the presence of a stone in the bile duct, a stone in the cystic duct stump or operative damage to the biliary tree. This is best performed by MRCP or ERCP, the latter having the advantage that a stone in the CBD can be removed.

Learning objectives

Learning objectives

To understand the surgical anatomy and physiology of the • gallbladder and bile ducts To be familiar with the pathophysiology and management bile ducts • of gallstones

Low insertion of the cystic duct

Low insertion of the cystic duct

The operating surgeon must identify variations in the anatomy (Figure 71.21) to avoid inadvertent damage to the common hepatic duct or CBD. Complete dissection of the cystic duct (Figure 71.22) should be avoided because there is a potential to devascularise the CBD, which could result in stricture formation.

Lymphatics

Lymphatics

The subserosal and submucosal lymphatic vessels of the gall bladder drain into the cystic lymph node of Lund (the sentinel lymph node), which lies in the fork created by the junction of the cystic and common hepatic ducts. Efferent vessels from this lymph node go to the hilum of the liver and to the coeliac lymph nodes. The subserosal lymphatics also connect with the subcapsular lymph channels of the liver; this accounts for the frequent spread of carcinoma of the gallbladder to the liver.

Magnetic resonance cholangiopancreatography

Magnetic resonance cholangiopancreatography

Magnetic resonance cholangiopancreatography (MRCP) is a non-invasive modality that provides excellent images of the gallbladder and biliary system (Figures 71.11 and 71.12). These images are comparable to those obtained at endoscopic retrograde cholangiopancreatography (ERCP) or percutaneous transhepatic cholangiography (PTC) (see Percutaneous transhepatic cholangiography and Endoscopic retrograde cholangiopancreatography) without the potential complications of the latter; they can demonstrate ductal abnormalities, including obstruction/stricture, stones and tumours.

Figure 71.11 Magnetic resonance cholangiopancreatography: cross-sectional image demonstrating hilar mass (thick arrow) and gallstones (thin arrow). Figure 71.12 Magnetic resonance cholangiopancreatography: projectional images demonstrating stones and hilar obstruction (arrow).

Malignant tumours of the bile duct

Malignant tumours of the bile duct

Summary box 71.7 Bile duct cancer (cholangiocarcinoma)

Cholangiocarcinoma Incidence Cholangiocarcinoma is an uncommon malignancy. The overall annual incidence is 1–1.5 per 100 000 with the peak incidence in the eighth decade. The male-to-female ratio is approximately 1.5:1. Anatomically, tumours involving the biliary confluence (hilar cholangiocarcinoma or Klatskin tumours) account for 60% of cases, with the remainder involving the distal bile (20–30%) or intrahepatic ducts (10–20%).

Risk factors A minority of patients have a known risk factor; the major risk factor in western practice is PSC. It is estimated that a longstanding history of PSC increases the risk of developing biliary tract cancer 20-fold compared with the normal population; those with concomitant IBD are at significantly higher risk. Cholangiocarcinoma appears to occur at an earlier age in patients with PSC (30–50 years of age) than in the general population. In addition, disease is usually multifocal and detected at an advanced stage with a resultant poor prognosis compared with the general population. Congenital cystic disease, hepatolithiasis, oriental cholangiohepatitis, hepatitis C virus infection and infestation with liver flukes have also been associated with an increased risk of cholangiocarcinoma. While the pathophysiology is unclear, it is thought that these parasites cause chronic inflammation that leads to DNA mutations through production of carcinogens and free radicals; the latter can stimulate cellular proliferation in the intrahepatic bile ducts and ultimately lead to invasive cancer (Summary box 71.8).

Gerald Klatskin, 1910–1986, hepatologist, VA Hospital, Newington, CT, USA. Early symptoms are often non-specific, with abdominal pain, early satiety, anorexia and weight loss commonly seen. Symptoms associated with biliary obstruction (pruritus and jaundice) may be present in a minority of patients. In these patients, examination often demonstrates clinical signs of jaundice, cachexia is often noticeable and the gallbladder is palpable if the obstruction is in the distal CBD (Courvoisier's sign).

Summary box 71.8 Risk factors for cholangiocarcinoma

Investigations Biochemical investigations (elevated bilirubin, ALP and GGTP) will confirm obstructive jaundice. The tumour marker carbohydrate 19-9 (CA19-9) may also be elevated. Imaging studies such as USG, CT and MRI/MRCP are essential for duct diagnosis, staging and assessing the anatomical relationship between the tumour and the major perihilar vascular structures (Figure 71.38). These studies allow the level of biliary obstruction to be defined and determine the locoregional extent of disease and the presence of metastases. Direct cholangiography using ERCP or PTC is also used following non-invasive studies. Both can define the level of obstruction and allow biopsy (non-diagnostic in 40–80% of patients) and placement of endobiliary stents for biliary drainage. The choice between the modalities depends on local availability and the anatomical site of the tumour, with PTC preferred for more proximal lesions and ERCP favoured for distal tumours. Despite a higher incidence of postoperative infection

with stenting, patients undergoing anticipated major liver resection - need preoperative biliary drainage as hyperbilirubinaemia (bilirubin level >6 mg/dL) may impair hepatic regeneration and hypertrophy. Cholangioscopy is an adjunct to ERCP, with diagnostic accuracy increased from 78% to 93% and sensitivity from 58% to 100%. Positron emission tomography (PET) is useful in detecting lymph node and distant metastases but has limited value in the assessment of local resectability.

Malignancy arising from the biliary epithelium; histologically of three types: mass forming, intraductal growing and periductal. Incidence increasing. Most patients present with abnormal liver function tests or frank jaundice. Diagnosis by USG, CT or MRI. Majority of patients receive palliative care only. Complete surgical excision possible in $<10\%$. Prognosis poor: 90% die within 1 year from liver failure or biliary sepsis. Adjuvant chemoradiation therapy has a limited role. Chronic inflammatory conditions: Thorium dioxide (Thorotrast), PSC, Vinyl chloride, Oriental cholangiohepatitis, Dioxin, Hepatitis C infection, Asbestos, Parasitic infections, Post surgical Opisthorchis viverrini, Biliary-enteric anastomosis, C. sinensis, Non-alcoholic fatty liver disease, Congenital Choledochal cysts, Caroli's disease.

The anatomical extent of the disease is classified according to either the Bismuth-Corlette (Figure 71.39) or the Memorial Sloan Kettering Cancer Center (MSKCC) classification. The MSKCC classification T-stage criteria for hilar cholangiocarcinoma are as follows:

- T1 tumour involving the biliary confluence without extension to second-order biliary radicles.
- T2 tumour involving the biliary confluence with unilateral extension to second-order biliary radicles and ipsilateral portal vein involvement or ipsilateral hepatic atrophy.
- T3 tumour involving the biliary confluence with bilateral extension to second-order biliary radicles; or unilateral portal vein involvement; or unilateral extension to second-order biliary radicles with contralateral hepatic lobar atrophy; or main or bilateral portal venous involvement.

Treatment: A multidisciplinary approach is required. The choice of treatment depends on the site and extent of the disease. Unfortunately, the majority of patients present with advanced disease. However, 10–15% are suitable for surgical resection, which offers the only hope for long-term survival. The aim of surgical resection is to achieve complete resection with negative pathological margins (R0 resection) and safely restore biliary-enteric continuity. Whether or not the disease is resectable depends on patient factors (comorbidities, presence or absence of chronic liver disease) and tumour factors (extent of disease within the biliary tree, vascular involvement, remnant liver volume, increase in the remnant after portal vein embolisation, presence or absence of metastatic disease). Depending on the site of disease, surgery involves either a standard or extended hepatic resection with caudate lobe excision with en bloc lymphadenectomy and reconstruction of the biliary tree. Distal common duct tumours may require pancreaticoduodenectomy (Whipple procedure). Local resection should be avoided. In selected patients, liver transplantation has been recommended for those with locally unresectable disease without evidence of distant metastases. Transplantation is often preceded by neoadjuvant chemoradiation therapy and staging laparoscopy. While emerging data are encouraging, this aggressive approach remains controversial and is reserved for selected patients.

in specialised centres (Mayo protocol). Marvin Corlette described the classification of cholangiocarcinoma in 1975.

Figure 71.38 Magnetic resonance imaging scan showing a hilar cholangiocarcinoma with dilatation of the intrahepatic biliary tree. Right hepatic duct Common hepatic duct

Figure 71.39 Bismuth–Corlette classification of cholangiocarcinoma.

Left hepatic duct Type I Type II Type IIIA Type IIIB Type IV

the T-stage, margin status, metastatic lymph node spread, perineural and perivascular invasion, non-papillary tumour subtypes and poor tumour differentiation. Of these, the only variable in which the surgeon plays a major role is margin-negative resection, and emphasis needs to be placed on achieving R0 resection. Approximately 35% of patients will survive 5 years after surgery. Adjuvant chemotherapy and radiotherapy have a limited role and have not been demonstrated to add survival benefit following surgical resection. However, patients at high risk for recurrence (positive surgical margins or node positive) may benefit from adjuvant therapy and should be referred for medical or radiation oncology opinion. The majority of patients who present with unresectable disease are candidates for palliative chemotherapy – gemcitabine with cisplatin. The aim is to maintain or improve quality of life by relieving symptoms and preventing cholestatic liver failure. Biliary obstruction can be relieved by endoscopic (ERCP) or percutaneous (PTC) methods. Surgical bypass rarely has a role apart from in patients with a distal bile duct lesion found to have unresectable disease at operation.

Open cholecystectomy

Open cholecystectomy

For patients in whom a laparoscopic approach is not indicated or in whom conversion from a laparoscopic approach is required, open cholecystectomy is performed. An upper midline, short subcostal (Kocher) or right upper transverse incision is made, centred over the lateral border of the rectus muscle. The gallbladder is appropriately exposed and packs are placed on the hepatic flexure of the colon, the duodenum and the lesser omentum to ensure a clear view of the anatomy of the porta hepatis. These packs may be retracted by the assistant's hand ('It is the left hand of the assistant that does all the work' – Moynihan). An artery or Duval forceps is placed on the infundibulum of the gallbladder and the peritoneum overlying Calot's triangle is placed on a stretch. The peritoneum is then divided close to the wall of the gallbladder and the fat in the triangle of Calot carefully dissected away to expose the cystic artery and the cystic duct. The cystic duct is cleaned down to the CBD, whose position is clearly ascertained. The cystic artery is tied and divided. The whole of the triangle of Calot is displayed to ensure that the anatomy of the ducts is clear and the cystic duct is then divided between ligatures (Figure 71.32). The gall bladder is then dissected away from its bed. Emil Theodor Kocher , 1841–1917, Professor of Surgery , Bern, Switzerland, first surgeon to win the Nobel Prize in Physiology or Medicine (1909) for his work on the physiology and surgery of the thyroid gland. Pierre Alfred Duval , 1874–1941, Professor of Surgery , Paris, France -

cholecystectomy. Operative steps
Purpose Retraction (Figure 71.31a) Opens the hepatocystic triangle
Proper retraction in the Increases the angle between correct direction: the fundus the cystic duct and the CBD is retracted

towards the Limits the dissection
above patient's right shoulder and
Rouvière's sulcus the infundibulum
is retracted Mental and spatial
orientation inferolaterally towards
the of the anatomy, variation and
patient's right side landmarks Look
out for red /f_l ag signs Time out
The surgeon should recognise
Failure of timely progression these
clues, stop dissection and of the
dissection decide on the strategy
for safe Anatomical disorientation
operation before proceeding Dif
/f_i culty in visualisation of Do not
hesitate to seek a the operative

/f_i eld second opinion Dif /f_i culty
achieving CVS is a Achieve CVS (
Figure 71.31b) warning Clearance
of the hepatocystic Further
dissection may be triangle of all
/f_i brofatty and hazardous, with
an increased soft areolar tissue to
see risk of biliary and/or vascular
only 2 structures enter the injury
gallbladder (cystic artery and Stop
and recon /f_i rm (with the duct)
team/second surgeon) that CVS
has been achieved Exposure of the
cystic plate May be documented
by (Figure 71.31c) photographs
and/or video This is done by

separating the recordings
gallbladder from its liver bed to
expose at least the medial third of
the cystic plate To avoid bleeding
from the liver Separate the
gallbladder from sinuses and bile
leak the fossa This is done by
leaving the cystic plate attached to
liver CBD, common bile duct; CVS,
critical view of safety. (b) (c)
Figure 71.31 (a–c) Operative
images of laparoscopic cholecys

tectomy. See Table 71.5 for the important steps during operation (courtesy of Dr Sameer Rege, Mumbai, India).

PRIMARY SCLEROSING CHOLANGITIS

PRIMARY SCLEROSING CHOLANGITIS

PSC is a rare idiopathic and progressive biliary tract disease characterised by inflammation and destruction of the intrahepatic and extrahepatic bile ducts that can lead to liver fibrosis and cirrhosis. Association with hypergammaglobulinaemia and markers such as anti-smooth muscle antibodies and anti-nuclear factor suggest an immunological basis; cystic fibrosis transmembrane conductance regulator (CFTR) gene mutations have been associated with the development of PSC. The majority of patients are between 30 and 60 years of age. There appears to be a male predominance and a strong association with inflammatory bowel disease (IBD), especially ulcerative colitis (IBD in PSC, 80%; PSC in IBD, 5%). Patients may be asymptomatic, but common symptoms include pruritus, fever, fatigue, right upper quadrant discomfort, jaundice and weight loss. Liver function tests reveal a Summary box 71.5 Causes of benign biliary stricture /uni25CF /uni25CF - /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Summary box 71.6 Radiological investigation of biliary strictures /uni25CF /uni25CF /uni25CF - /uni25CF /uni25CF - cholestatic pattern, elevated serum ALP and gamma-glutamyl transpeptidase (GGT) and smaller rises in the aminotransferases; bilirubin values can be variable. MRCP (or ERCP) may demonstrate stricturing and beading of the bile ducts (Figure 71.37). Liver biopsy is helpful to confirm the diagnosis (concentric periductal 'onion skinning') and may help guide therapy by excluding cirrhosis. Important differential diagnoses are - secondary sclerosing cholangitis, immunoglobulin G4 (IgG4) cholangitis, autoimmune hepatitis, human immunodeficiency

(a) A T-tube in situ with a stone in the bile duct. (c) The stone is extracted from the duct along the Congenital Inflammatory Biliary atresia Stones Cholangitis Bile duct injury at Parasitic surgery Pancreatitis Cholecystectomy Sclerosing cholangitis Choledochotomy Radiotherapy Gastrectomy Hepatic resection Trauma Transplantation Idiopathic USG MRCP ERCP (with brush cytology in cases of a dominant stricture) PTC CT

virus (HIV) cholangiopathy and cholangiocarcinoma. The last may arise in patients with PSC and is difficult to diagnose; a high index of suspicion is required, especially in the setting of unexplained clinical deterioration. Patients with PSC are at increased risk for cholangiocarcinoma and gallbladder cancer, as well as colon cancer in those with concurrent IBD. Medical management with antibiotics, vitamin K, cholestyramine, steroids and immunosuppressant drugs may not relieve symptoms. Endoscopic stenting of dominant strictures and, in selected patients with predominantly extrahepatic disease, operative resection may be worthwhile. For patients with cirrhosis, liver transplantation is the best option; 5-year survival following transplantation in high-volume centres is in excess of 80%. Screening for malignancies involving the gallbladder (polyp)

and bile ducts and colonoscopy for IBD or malignancy are therefore critical, and bone densitometry for bone density is mandatory .

Figure 71.37 Sclerosing cholangitis in a patient with ulcerative colitis, visualised by endoscopic retrograde cholangiopancreatography.

Percutaneous transhepatic cholangiography

Percutaneous transhepatic cholangiography

This is an invasive technique in which the bile ducts are cannulated percutaneously. The main indication is to drain intra-hepatic ducts when strictures cannot be accessed at ERCP. The procedure is undertaken after confirming normal coagulation parameters; antibiotics should be given prior to the procedure. Under fluoroscopic or sonographic control, a slender (Chiba or Okuda) needle is introduced percutaneously into the liver substance. Successful entry into the bile duct is confirmed by contrast injection or aspiration of bile. Water-soluble contrast medium is injected to visualise the biliary system and images are taken to demonstrate strictures or obstruction (Figure 71.16). Bile can be sent for cytology. This technique enables placement of a catheter into the bile ducts to provide external or internal biliary drainage and insertion of indwelling stents in situ for a number of days. The drainage catheter can be left and the track dilated sufficiently for the introduction of a fine flexible choledochoscope to diagnose strictures, take biopsies and remove stones.

Summary box 71.1 Radiological investigation of the biliary tree

/uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Kunio Okuda, 1921–2003, Professor of Medicine, Chiba University, Chiba, Japan.

Peroperative cholangiography During open or laparoscopic cholecystectomy, a catheter can be placed in the cystic duct and contrast injected directly into the biliary tree. The technique defines the anatomy and is used mainly to exclude the presence of stones within the bile ducts (Figures 71.17–71.19). A radiographic plate or image intensifier can be used to obtain and review the images intra-operatively. The operating table should be tilted head-down by approximately 20° to facilitate filling of the intrahepatic ducts. Care should be taken when injecting contrast not to introduce air bubbles into the system as these may mimic the appearance of stones.

Operative biliary endoscopy (choledochoscopy) At operation, a flexible fiberoptic endoscope can be passed either via the cystic duct or directly via a choledochotomy (open or laparoscopic) into the CBD, enabling stone identification and removal under direct vision. After exploration of the CBD, the CBD is closed with a clip.

Plain radiograph: calcification,
air within the biliary system
USG: stones and biliary dilatation
MRCP:

anatomy and stones CT scan:
anatomy, and liver, biliary and
pancreatic cancer Radioisotope
scanning (HIDA scan): function
ERCP: anatomy, stones and biliary
strictures, with or without
cholangioscopy PTC: anatomy and
biliary strictures EUS: anatomy,
stones

Angle to 20° Radiographic /f_i lm Figure 71.17 Peroperative cholangiography using a radiolucent table- top. Figure 71.18 Peroperative cholangiography. Technique of introducing contrast.

the bile duct, a tube can be left in the cystic duct remnant or in the CBD (T tube) and drainage of the biliary tree established. After 7-10 days, a track will be established. This track can be used subsequently for the passage of a choledochoscope or radiologically guided stone retrieval catheter (Burhenne technique) to remove residual stones. Laparoscopic ultrasonography At laparoscopy , a laparoscopic ultrasound probe can be used to closely image the extrahepatic biliary system. This technique is useful in biliary and pancreatic tumour staging as it can determine the relationship of the tumour to major vessels such as the hepatic artery , superior mesenteric artery , portal vein and superior mesenteric vein.

Figure 71.19 Peroperative
cholangiography. (a) Gentle

infusion of contrast, passing with

out hindrance into the duodenum. A normal duct. (b) Dilated duct containing multiple stones; there is a delay in contrast passing into the duodenum.

Postcholecystectomy choledocholithiasis

Postcholecystectomy choledocholithiasis

Any obstruction to the flow of bile can give rise to stasis, with the formation of stones within the duct. Duct stones may be detected many years after cholecystectomy and may also be related to the development of new pathology, such as infection of the biliary tree or infestation by *Ascaris lumbricoides* *Clonorchis sinensis*. The consequence of duct stones is either obstruction to bile flow or infection. Stones in the bile ducts are more often (80%) associated with infected bile than with stones in the gallbladder. Symptoms The individual may be asymptomatic; symptomatic patients with cholangitis have bouts of pain, jaundice and fever ('Charcot's triad'). Signs Febrile, icteric tenderness may be elicited in the epigastrium and right hypochondrium. Management It is essential to confirm that the jaundice is due to duct obstruction. Liver function tests and USG are the initial tests, and MRCP will identify the nature of the obstruction. Pus may be present within the biliary tree and liver abscesses may develop. Measures required include rehydration, attention to clotting, exclusion of diabetes and the administration of appropriate broad-spectrum antibiotics. Once resuscitation has taken place, relief of the obstruction is essential. Endoscopic papillotomy/sphincterotomy is the preferred technique, followed by removal of the stones using a Dormia basket or placement of a stent or a nasobiliary drain for flushing if stone removal is not possible (Figures 71.34 and 71.35). If this fails, PTC drainage can be done, with subsequent percutaneous choledochoscopy . Surgery , in the form of choledochotomy , is Jean Martin Charcot , 1825–1893, physician, La Salpêtrière, Paris, France. Enrico Dormia , 1928–2009, Professor of Urology , University of Milan and Chief of the Department of Urology , S Car aged by minimally invasive techniques.

SURGICAL ANATOMY AND PHYSIOLOGY

SURGICAL ANATOMY AND PHYSIOLOGY

The gallbladder is a pear-shaped structure, 7.5–12 cm long, with a normal capacity of about 25–30 mL. Its anatomical divisions are fundus, body and neck, which terminates in a narrow infundibulum. The gallbladder lies on the underside of the liver in the main liver scissura at the junction of the right and left lobes. Its relationship to the liver varies from being embedded within the liver substance to being suspended by a mesentery. The muscle fibres in the wall of the gallbladder are arranged in a criss-cross manner, being particularly well developed in its neck. The mucous membrane contains indentations (crypts of Luschka) that sink into the muscle coat. The cystic duct is about 3 cm in length, but this is variable. Its lumen is 1–3 mm in diameter; its mucosa is arranged in spiral folds (valves of Heister); and the wall is surrounded by the sphincter of Lütken. The cystic duct joins the supraduodenal segment of the common hepatic duct in 80% of cases; however, the junction may be much lower in the retroduodenal or even retropancreatic part of the bile duct. Occasionally, the cystic duct may join the right hepatic duct or even a right hepatic sectorial duct (see Low insertion of the cystic duct). The common hepatic duct is usually less than 2.5 cm long and is formed by the union of the right and left hepatic ducts. The common bile duct (CBD) is about 7.5 cm long and is formed by the junction of the cystic and common hepatic ducts. It is divided into four parts: Hubert Luschka, 1820–1875, Professor of Anatomy, Tübingen, Germany. Lorenz Heister, 1683–1758, Professor of Surgery and Botany, Helmstädt, Germany. Ulrich Lütken, b. 1894, surgeon, University Clinic, Berlin, Germany, published a monograph on the structure and function of the extrahepatic biliary tract in 1926. Ruggero Oddi, 1845–1906, physiologist, Perugia, Italy. Abraham Vater, 1684–1751, Professor of Anatomy and Botany, Wittenberg, Germany. Jean François Calot, 1861–1944, surgeon, Paris, France. 1 supraduodenal portion, about 2.5 cm long, runs in the free edge of the lesser omentum; 2 retroduodenal portion; 3 infraduodenal portion, lies in a groove, at times in a tunnel, on the posterior surface of the pancreas; 4 intraduodenal portion, passes obliquely through the wall of the second part of the duodenum, where it is surrounded by the sphincter of Oddi and terminates by opening on the summit of the ampulla of Vater. The cystic artery, a branch of the right hepatic artery, usually arises behind the common hepatic duct (Figure 71.1). Occasionally, an accessory cystic artery arises from the gastroduodenal artery. In 15% of cases the right hepatic artery and/or cystic artery cross in front of the common hepatic duct and cystic duct. Calot's triangle, or the hepatobiliary triangle, was initially described by Calot as the space bordered by the cystic duct inferiorly, the common hepatic duct medially and the superior border of the cystic artery. This has been modified in contemporary literature as the area bounded superiorly by the inferior surface of the liver, laterally by the cystic duct and the medial border of the gallbladder and medially by the common hepatic duct ('hepatocystic triangle'). It is an important surgical landmark as the cystic artery usually can be found within its boundaries (Figure

71.2a). The cystic lymph node often lies superficial to the cystic artery and acts as a landmark to locate this artery in difficult cases. The cystic plate is a flat ovoid fibrous sheet continuous with the liver capsule of segments IV (medially) and V

To be aware of unusual disorders of the biliary tree • To be aware of malignant disease of the gallbladder and •

(laterally). It is located in the gallbladder bed and needs to be exposed to achieve the critical view of safety (CVS) during cholecystectomy . Rouvière's sulcus on the undersurface of the right lobe of the liver running to the right of the hepatic hilum marks the position of the right posterior sectoral pedicle. The advantage of identifying Rouvière's sulcus and the line joining M. Henri Rouvière , 1876–1952, Professor of Anatomy , LeBleymard, France. Berkeley George Andrew Moynihan (Lord Moynihan), 1865–1936, Professor of Clinical Surgery , Leeds, UK. (Rouvière's sulcus /umbilical fissure) is that the cystic duct and the cystic artery lie ventral (anterosuperior) to the line and the CBD lies below the line. CBD injury can be minimised by maintaining the dissection ventral to the line during cholecystectomy . In the case of difficulty , all dissection during laparoscopic cholecystectomy should be performed ventral to the R4U line (Figure 71.3).

artery artery Hepatic artery Left choledochal artery Common hepatic artery Cystic artery Retroduodenal artery Right choledochal artery Gastro duodenal artery Figure 71.1 Anatomy of the gallbladder and bile ducts. Note the arrangement of the arterial tree. (a) (b) Figure 71.2 (a) The usual anatomy of the 'hepatocystic triangle'; short cystic artery. (b) and (c) are examples of the 'caterpillar turn' or 'Moynihan's hump', which can lead to inadvertent arterial injury or bleeding during cholecystectomy.

Stricture of the bile duct

Stricture of the bile duct

The causes of benign biliary stricture are given in Summary box 71.5 . Bile duct strictures may be investigated radiologically as described in Summary box 71.6 .

Surgical physiology

Surgical physiology

Bile is produced by the liver and stored in the gallbladder before being released into the duodenum. The liver excretes bile at approximately 40 /uni00A0 mL/h. As it leaves the liver its composition is 97% water; the remaining 3% consists of bile salts (cholic and chenodeoxycholic acids, deoxycholic and lithocholic acids), phospholipids, cholesterol and bilirubin. About 95% of bile salts are reabsorbed in the terminal ileum and returned to the liver (enterohepatic circulation).

TORSION OF THE GALLBLADDER

TORSION OF THE GALLBLADDER

This is a very rare complication, requires a long mesentery and therefore occurs most often in an older patient with a mucocele of the gallbladder. Presentation is with extreme right upper quadrant abdominal pain. Urgent exploration is indicated, with cholecystectomy as the only treatment.

TRAUMA

TRAUMA

- Injury to the gallbladder and extrahepatic biliary tree is rare and may occur as a result of blunt or penetrating abdominal trauma. Iatrogenic injury is perhaps more frequent than external trauma. Physical signs are those of an acute abdomen. Management depends on the location and extent of biliary and associated injury . In a stable patient, a transected bile duct to the gallbladder can be dealt with by cholecystectomy .

TUMOURS OF THE BILE DUCT

Benign tumours of the bile duct

TUMOURS OF THE BILE DUCT Benign tumours of the bile duct

Benign neoplasms such as papilloma, adenoma, papilloma - tosis, leiomyoma and neural and endocrine tumours causing biliary obstruction are uncommon and may be an incidental finding. For symptomatic patients, the duration of symptoms may vary from a few days to months. Intraductal papillary neoplasm of the bile duct Intraductal papillary neoplasm of the bile duct (IPNB) is a rare variant of bile duct tumours characterised by papillary growth within the bile duct lumen with ductal dilatation. It is regarded as the biliary counterpart of an intraductal papillary - mucinous neoplasm of the pancreas. IPNBs display a spectrum from premalignant lesions to invasive cholangiocarcinoma. The most common radiological findings are bile duct dilata - o - tion and intraductal masses. USG, CT and MRI are usually - performed to assess tumour location and extension. Cholan - gioscopy can confirm the histology and assess the extent of the tumour, including superficial spr ead along the biliary for surgical resection in a manner similar to that for other types of intrahepatic cholangiocarcinomas and extrahepatic bile duct carcinomas, i.e. major hepatectomy with or without extrahepatic bile duct resection.

Tenets for safe cholecystectomy (Table 71.5)

Tenets for safe cholecystectomy (Table 71.5)

Safe zone of dissection The safe zone of dissection lies cephalad to a line extending from the roof of Rouvière's sulcus to the umbilical fissure across the base of segment IV (R4U line). The operating - surgeon tends to zoom the laparoscope closer to the surgical field to get a better view during difficulty, but this results in non-visualisation of the normal clues/landmarks necessary for correct orientation. Dissection on the posterior aspect of the hepatocystic triangle can be safely started immediately ventral and cephalad to the sulcus. The B-SAFE method uses five anatomical landmarks (B, bile duct; S, sulcus of Rouvière; A, hepatic artery; F, umbilical fissure; E, enteric/duodenum) to correctly place a cognitive map during dissection. Concept of 'time out' During difficult gallbladder surgery, the surgeon may become disoriented and enter the zone of danger. To avoid this, the concept of time out has been introduced as it serves as a procedural cognitive aid to recall and apply essential safety measures. Judicious use of energy sources With a monopolar energy device (mostly hook cautery), it is important to: /uni25CF keep a low setting (approximately 30 /uni00A0 W) to avoid arcing of the current to the bile duct; /uni25CF divide small amounts of the tissue at a time after a gentle pull to avoid injury to the deeper structures by the heel of the cautery hook; /uni25CF use intermittent short bursts of current at intervals to avoid thermal lateral spread; /uni25CF avoid blind use of cautery in brisk bleeding. Lateral thermal spread occurs less with an ultrasonic energy source, but it may be cumbersome to use the long and straight jaws to dissect in the hepatocystic triangle. Concept of the critical view of safety The aim of the CVS is the conclusive identification of the cystic duct and cystic artery to avoid misidentification injury. 'Stopping rules' With the help of red flag signs (severe adhesions, severe acute inflammation, large impacted stone in the neck of the gall bladder, Mirizzi syndrome, chronic inflammation with fibrosis Frederic Eugene Basil Foley, 1891-1965, urologist, The Miller and Anker Hospitals, St. Paul, MN, USA. or pre-empt difficult situations that increase the risk of biliary/vascular injury and to stop in time. Call for help/second opinion The operating surgeon should not hesitate to seek a second opinion whenever needed, and this should be considered a sign of good clinical practice rather than of surgical ineptitude. Bailout techniques/strategies The primary aim is the safety of the patient from biliary/vascular injury. It is important to perform an alternative procedure (bailout technique) that allows the surgeon to complete the operation in a safe manner. There are five bailout strategies: 1 abort the procedure altogether; 2 convert to an open procedure; 3 carry out a tube cholecystostomy using a 14 /uni00A0 Fr Foley catheter (a simple procedure to provide symptomatic relief until a definitive procedure can be performed); 4 carry out a subtotal

cholecystectomy (open/laparoscopic): leaving behind a part of the gallbladder is safer than a difficult dissection in the hepatocystic triangle with a potential for bile duct injury in an attempt to remove the entire gallbladder; 5 fundus-first approach. The choice of bailout procedure depends on the clinical situation and the experience/expertise of the surgeon. Conversion to open cholecystectomy should be 'by choice' at an early stage in a difficult cholecystectomy, e.g. the anatomy is not clear, the pathology is too difficult or no progress is being made, rather than the surgeon having to convert because of a complication, e.g. bleeding or bile duct injury.

Figure 71.32 Ligatures are passed and tied around the cystic artery and cystic duct. The grey shaded area represents Calot's triangle.

Treatment

Treatment

For breast-fed infants, introducing supplemental formula feeds using a medium-chain triglyceride-based feed and fat-soluble vitamin supplementation (titrated according to growth) is a priority . Patent segments of proximal bile duct are found in 10% of type I lesions. A direct Roux-en-Y hepaticojejunostomy Daniel Alagille , 1925–2005, paediatric hepatologist, Hôpital Bicêtre, Paris, France. César Roux , 1857–1934, Professor of Surgery and Gynaecology , Lausanne, Switzerland, described the Roux-en-Y loop in 1908. Jacques Caroli , 1902–1979, gastroenterologist, Hôpital St Antoine, Paris, France, described cavernous ectasia in the biliary tree in 1958. will achieve bile flow in 75%, but progressive fibrosis results in disappointing long-term results. A simple biliary-enteric anastomosis is not possible in the majority of cases in which the proximal hepatic ducts are either very small (type II) or atretic (type III). These are treated by the Kasai procedure , in which radical excision of all bile duct tissue up to the liver capsule is performed. A Roux-en-Y loop of jejunum is anastomosed to the exposed area of liver capsule above the bifurcation of the portal vein, creating a portoenterostomy . The chances of achieving effective bile drainage after portoenterostomy are maximal when the operation is performed before the age of 8 weeks, and approximately 90% of children whose bilirubin falls to normal can be expected to survive for 10 years or more. Early referral for surgery is critical. Postoperative complications include bacterial cholangitis, which occurs in 40% of patients. Repeated attacks lead to hepatic fibrosis, and 50% of long-term survivors develop portal hypertension, with one-third having variceal bleeding. Liver transplantation should be considered in children in whom a portoenterostomy is unsuccessful. Results are improving, with 70–80% alive 2–5 years following transplant.

Atretic Patent Atretic IIb III

Treatment

Asymptomatic gallstones do not need intervention, however prophylactic cholecystectomy may be performed for asymptomatic cholelithiasis in the following situations: /uni25CF large (>3 /uni00A0 cm) gallstones; /uni25CF choledocholithiasis; /uni25CF chronic haemolytic conditions (sickle cell disease, hereditary spherocytosis); /uni25CF gallbladder polyps >1 /uni00A0 cm in diameter; /uni25CF suspicion/risk of malignancy (anomalous pancreatic ductal drainage); /uni25CF calcification of the wall (porcelain gallbladder); /uni25CF some ethnic groups or subjects living in areas with a high prevalence of gallbladder cancer associated with gallstones (some parts of northern India, Native Americans, Mexican Americans, Colombia, Chile, Bolivia); /uni25CF transplant patients (during transplantation); /uni25CF bariatric surgery . For patients with symptomatic gallstones, cholecystectomy is the treatment of choice if there are no medical contraindications. The initial non-operative treatment is based on four steps: 1 Nil by mouth and intravenous fluid administration until the pain resolves. 2 Analgesics. 3 Antibiotics. As the cystic duct is blocked in most instances, the concentration of antibiotic in the serum is more important

than the concentration in the bile. A broad-spectrum antibiotic effective against Gram-negative aerobes is most appropriate (e.g. cefazolin, cefuroxime or ciprofloxacin). 4 Subsequent management. When the temperature, pulse and other physical signs show that the inflammation is subsiding, oral fluids are reinstated, followed by a regular diet. If jaundice with deranged ALP and enzyme levels is present, MRCP should be performed to exclude choledocholithiasis. If there is any concern regarding the diagnosis or the presence of complications such as perforation, CT should also be performed. The timing of surgery in acute cholecystitis remains controversial. Early cholecystectomy, undertaken by an experienced surgeon with excellent operating facilities within 5–7 days of the onset of the attack, is safe and shortens total hospital stay. Nevertheless, the conversion rate in laparoscopic cholecystectomy is higher in acute than in elective surgery. If early operation is not indicated, one should wait approximately 6 weeks for the inflammation to subside before operating. The Tokyo Guidelines (2013/2018) allow the assessment of severity and grading of acute cholecystitis (Table 71.2) and provide a consensus-derived treatment algorithm based on grading, patient comorbidity and the facilities and expertise available (Figure 71.26). et al 25 Acute and chronic inflammation of the gallbladder can occur in the absence of stones and give rise to a clinical picture similar to that of calculous cholecystitis. Some patients have non-specific inflammation of the gallbladder, whereas others have one of the cholecystoses. Acute acalculous cholecystitis is particularly seen in critically ill patients and those recovering from major surgery, trauma and burns. The diagnosis is often missed and the mortality rate is high. The treatment is cholecystectomy for patients who are able to tolerate surgery. In selected patients, non-surgical treatment (such as antibiotics or percutaneous cholecystostomy) may be an effective alternative to surgery.

TABLE 71.2 Tokyo Consensus Guidelines for severity grading of acute cholecystitis. Grade III (severe) acute cholecystitis Associated with dysfunction of any one of the following organs/systems: 1 Cardiovascular dysfunction Hypotension requiring treatment with dopamine ≥ 5 g/kg/min, or any dose of epinephrine 2 Neurological dysfunction Decreased level of consciousness 3 Respiratory dysfunction $\text{PaO}_2/\text{FIO}_2$ ratio <300 4 Renal dysfunction Oliguria; creatinine >2.0 mg/dL 5 Hepatic dysfunction Prothrombin time (PT-INR) >1.5 6 Haematological dysfunction Platelet count $<100\,000/\text{mm}^3$ Grade II (moderate) acute cholecystitis Associated with any one of the following conditions: 1 Elevated white cell count ($>18\,000/\text{mm}^3$) 2 Palpable tender mass in the right upper abdominal quadrant 3 Duration of complaint >72 hours 4 Marked local inflammation (gangrenous cholecystitis, pericholecystic abscess, hepatic abscess, biliary peritonitis, emphysematous cholecystitis) Grade I (mild) acute cholecystitis Does not meet the criteria of grade II or grade III acute cholecystitis. Grade I can also be defined as acute cholecystitis in a healthy person with no organ dysfunction and mild inflammatory changes in the gallbladder, making cholecystectomy a safe and low-risk operative procedure $\text{PaO}_2/\text{FIO}_2$ ratio is the ratio of arterial oxygen partial pressure (PaO_2 mmHg) to fractional inspired oxygen (FIO_2) expressed as a fraction (not a percentage) at sea level, the normal $\text{PaO}_2/\text{FIO}_2$ ratio is $\sim 400\text{--}500$ mmHg ($\sim 55\text{--}65$ kPa); PT-INR, prothrombin time–international normalised ratio. Reproduced with permission from Yokoe M. Tokyo Guidelines 2018: diagnostic criteria and severity grading of acute cholecystitis (with videos). J Hepatobiliary Pancreat Sci 2018; (1) 41–54.

Typhoid infection of the gallbladder

Typhoid infection of the gallbladder

Salmonella Typhi or Salmonella Typhimurium can infect the gallbladder. Acute or, more frequently, chronic cholecystitis occurs, with the patient becoming a typhoid carrier excreting the bacteria in the bile. Gallstones may be present (surgeons should not give patients their stones after their operation if there is any suspicion of typhoid). It is debatable whether the stones are secondary to the Salmonella cholecystitis or whether pre-existing stones predispose the gallbladder to chronic infection. Treatment with ampicillin and cholecystectomy are indicated. In the case of penicillin allergy, a quinolone antibiotic can be used.

Figure 71.27 The interior of a strawberry gallbladder (cholesterosis) (courtesy of Dr Sanjay P Thakur, Patna, India).

Figure 71.28 Types of cholecystitis glandularis proliferans (polyps, intra

mural or diverticular stones and /f_i stula).

Ultrasonography

Ultrasonography

Transabdominal ultrasonography (USG) (Figure 71.7 initial imaging modality of choice as it is accurate, readily John Benjamin Murphy , 1857–1916, surgeon, Mercy Hospital, Chicago, IL, USA. operator dependent and may be compromised by excessive body fat and intraluminal bowel gas. The size of the gall - bladder and presence of stones or polyps can be determined. Acute calculous cholecystitis is diagnosed radiologically (sensi - tivity 90–95%) by thickening of the gallbladder wall (>3 /uni00A0 mm), presence of pericholecystic fluid or direct tenderness when the probe is pushed against the gallbladder (ultrasonog raphic Murphy's sign). Additionally , the presence of inflammation around the gallbladder, the size of the CBD and, occasionally , the presence of stones within the extrahepatic biliary tree can be determined.) is the In a patient with obstructive jaundice, USG can iden - tify intra- and extrahepatic biliary dilatation, the level of the obstruction and the cause of the obstruction - stones, com - mon hepatic duct or CBD, lesions within the wall of the CBD suggestive of cholangiocarcinoma, gallbladder cancer or mass lesions in the pancreatic head.

Figure 71.6 Gas in gallbladder and gallbladder wall (Clostridium perfringens infection). Emergency cholecystectomy is indicated. Figure 71.7 Ultrasound examination. Gallstones in neck of gallbladder with acoustic shadowing.

Xanthogranulomatous cholecystitis

Xanthogranulomatous cholecystitis

Xanthogranulomatous cholecystitis (Figure 71.30) is an uncommon inflammation of the gallbladder, more frequently seen in India and Japan. It is more common in females. - It is caused by extravasation of bile into the gallbladder wall from rupture of the Rokitansky-Aschoff sinuses or by mucosal ulceration as a result of a focal or diffuse destructive - inflammatory process, with accumulation of lipid-laden macrophages (xanthoma cells), fibrous tissue and acute and chronic inflammatory cells. USG shows gallbladder wall intramural hypoechoic nodules or bands and often the presence of gallstones. CT shows 5- to 20-mm intramural hypoattenuating nodules and poor/heterogeneous contrast enhancement. As with acute cholecystitis, early enhancement of the adjacent liver parenchyma may occur. Extension into the liver along with enlarged hepatoduodenal lymph nodes closely mimics gallbladder carcinoma. Diagnosis is difficult and depends on pathological examination. Intraoperatively , frozen-section examination should be carried out to differentiate xanthogranulomatous cholecystitis from carcinoma of the gallbladder (coexistence of gallbladder cancer 2.3–13.3%). Because of diagnostic difficulties if there is preoperative suspicion of xanthogranulomatous cholecystitis open cholecystectomy should be considered.