EFSUMB – European Course Book

Editor: Christoph F. Dietrich

Ultrasound of the biliary system

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Acknowledgment: The authors thank Michael Hocke and Cui Xinwu for peer review of the manuscript.
Keywords: Gallbladder, cholelithiasis, cholecystitis, cholangiocellular carcinoma, bile duct

Content

Introduction

Biliary system diseases are common in medical practice. After obtaining the patient’s history and performing a physical examination, conventional B-mode ultrasound and colour Doppler imaging (CDI) are the imaging methods of choice in, for example, the evaluation of right upper quadrant pain and obstructive jaundice in patients with elevated cholestasis indicating enzymes and many other hepatobiliary diseases. In the hands of an experienced ultrasound practitioner ultrasound has become a diagnostic tool equal in importance to endoscopy. Primarily, ultrasound is the method of choice in the confirmation or exclusion of dilated bile ducts, cysts and calcifications.

Ultrasound is widely accepted for the diagnosis of biliary system disease. It has the greatest sensitivity for the diagnosis of cholecystolithiasis (approximately 100%) when compared with other imaging modalities. It is also of great help in the diagnosis of the spectra of appearances in acute and chronic cholecystitis and in the diagnosis of intra- and extrabiliary duct dilation. However, clarifying the aetiology of bile duct dilatation is a far more difficult question to answer, but careful evaluation of the clinical presentation together with the level and extent of obstruction can assist in determining the cause on ultrasound. Gallbladder polyps are sonographically easy to detect. Recently, ultrasound contrast agents have proven to be useful for the clarification of biliary tumours. Gallbladder carcinomas are usually detected by ultrasound at a late stage by which time the liver is already infiltrated and metastases can be detected; this because the disease presents in advanced age with few early symptoms.

Ultrasound is a routine examination in daily practice and it is the first line imaging modality of choice in many clinical presentations (e.g. abdominal pain) as well as in asymptomatic patients as a screening tool [(1)]. It is an accurate, safe, non-invasive, inexpensive, accessible, repeatable imaging modality, which is highly sensitive and specific for the detection of many biliary tree diseases and it can frequently demonstrate an alternative diagnosis as the cause of the patient’s symptoms if the biliary system is normal [(2;3)]. However, it is a highly operator dependent imaging modality and its diagnostic success is also influenced by the situation,
such as non-fasting, obesity, presence of surgical dressings and distended abdomen owing to intestinal gas.

**Topography of the gallbladder**

Topographically, the gallbladder typically lies on the inferior surface of the liver, and is commonly located in the mid-clavicular line, just below the lower costal margin on the anterior abdominal wall. Its function is to store and expel bile into the duodenum. It is situated in the gallbladder fossa of the posterior right hepatic lobe, lateral to the second part of the duodenum and anterior to the right kidney and transverse colon. It comprises a fundus, body, infundibulum (Hartman’s pouch, which is the portion of body that joins the neck) and neck including the spiral valve of Heister in the region of the neck. The cystic duct arises from the superior aspect of the gallbladder neck, goes to the main biliary duct (MBD) and is usually 2–6cm long. Its lumen contains a series of mucosal folds and the spiral valves of Heister, which prevent collapse or overdistension owing to sudden position changes. This structure is important to recognise on ultrasound as it can cause acoustic shadowing, which may sometimes be mistaken for a calculus in the neck of the gallbladder. The gallbladder often lies obliquely within the abdomen, which is important to appreciate for positioning the transducer correctly to image the long- and short-axes of the gallbladder. However, its position is dependent on the patient’s body habitus. The four typical body habitus types are hypersthenic, sthenic, hyposthenic and asthenic. The position of the gallbladder can vary, as shown in Table 1.

<table>
<thead>
<tr>
<th>Body habitus type</th>
<th>Typical gallbladder position and orientation</th>
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<tr>
<td><strong>Hypersthenic</strong></td>
<td>The diaphragm, liver and gallbladder tend to lie high in the abdomen in the right upper quadrant, under the thoracic cage and this restricts transducer access. The liver is often difficult to access; intercostal scanning, and decubitus and erect patient positions can facilitate access. The stomach is also high and this can create problems with access to deeper structures owing to overlying gas and food residue. The gallbladder is often horizontally orientated rather than in its normal oblique position.</td>
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Table 1  Variable positions of the gallbladder dependent upon body habitus type
**Sthenic**
(average build)
The liver and gallbladder tend to lie as expected in the right upper quadrant with the gallbladder fundus just visible below lower costal margin in the mid-clavicular plane and with the gallbladder lying obliquely.

**Hyposthenic**
(tall thin narrow chest, not deep in anterior-posterior diameter)
The liver and gallbladder tend to lie lower than in above 2 types, often located in the right lumbar abdominal region and the gallbladder often lies in a more vertically orientated than in hypersthenic and sthenic types.

**Asthenic**
(extreme variant of hyposthenic)
The liver and gallbladder tend to lie low down in the abdomen, sometimes as low as the right iliac fossa. The gallbladder tends to lie in a more vertically orientated than the other three types.

An understanding of these variant positions is essential for a successful ultrasound scanning technique. It is suggested that the practitioner looks at the patient as they enter the scan room and assigns them to a body habitus type. This will help the ultrasound practitioner to know where to find the gallbladder and how to align the ultrasound transducer to show the long- and short-axis of the structure.

**Anatomy**
The gallbladder is a saccular structure that has a pear or teardrop shape in cross-section. It is situated in the gallbladder fossa of the posterior right hepatic lobe, lateral to the second part of the duodenum and anterior to the right kidney and transverse colon.

Cholecystomegaly in patients with diabetes mellitus or during long-standing fasting periods may reveal gallbladder diameters of up to $15 \times 6$ cm without clinical relevance (Figure 1) in contrast to the clinically important hydrops, which sometimes shows right upper quadrant pain and occasionally fever. The normal gallbladder wall anteroposterior diameter measures 1–3mm. Many operators do not measure the normal wall thickness because it is difficult to accurately measure such a small structure. The anterior wall of the gallbladder should always be measured as it is closer to the transducer. It should be measured in line with the ultrasound beam to optimise the axial resolution of the ultrasound beam.

The gallbladder volume should be determined before and after a test meal and this can be used, to a limited degree, to assess gallbladder function. Contraction of $>60\%$ is regarded as
normal. To assess the gallbladder after the test meal, it is usual to mark an X on the abdominal wall at the site of the gallbladder when it is distended to facilitate its location after the test meal. Normal contraction is a requirement before gallstone treatment with, for example, ursodeoxycholic acid. The cystic artery, a branch of right hepatic artery, is the main arterial supply.

Figure 1  Long-axis section of the gallbladder showing a clinically insignificant large gallbladder. This is often seen in older people with a physiological atonic gallbladder. A large gallbladder (up to 15cm in length) may be found in older people, patients with diabetes and many other unspecific disorders [(1;5)].

Gallbladder

Examination technique

Patient preparation

It is recommended that patients undergo a period of fasting prior to upper abdominal imaging of the biliary tree to maximise the distension of the gallbladder and to reduce food residue and gas in the upper gastrointestinal (GI) tract, which can reduce image quality or preclude imaging of the gallbladder and biliary tree. Typically, a patient should not eat or drink anything at least 6–8h before ultrasound examination to achieve this. However, a patient may
take small amounts of still water by mouth prior to the scan, particularly for taking medications. If the patient has not adequately fasted, the gallbladder will be partially contracted and the walls will appear thicker than normal and this can mimic pathological gallbladder wall thickening, which is a misdiagnosis. However, in emergency situations the examination can be performed if the gallbladder is partially contracted. If a diagnosis cannot be reached then a repeat scan after fasting is recommended if the clinical status of the patient permits.

There is some evidence that smoking can reduce image quality when scanning upper abdominal structures and it is good practice to encourage a patient not to smoke for 6–8h prior to an ultrasound scan. Smoking increases gas intake into upper GI tract and may reduce image quality. Some chemicals in tobacco are known to cause contraction of the smooth muscle of the GI tract and this can cause contraction of the gallbladder, even after fasting, and the gallbladder cannot be scanned.

**Patient history and physical examination**

It is recommended that a short patient history is taken and that the abdomen is examined or palpated before the ultrasound examination commences. This complements the ultrasound information with clinical data and ensures the clinical question is addressed.

**Ultrasound examination of the gallbladder**

Routinely, a convex high multifrequency (5–7MHz) transducer is used for the evaluation of the gallbladder. However, lower frequencies may be used when an increased depth of penetration is required, for example in more obese patients or when the gallbladder is deep (e.g. hypersthenic patients). In addition, in very slim patients (e.g. asthenic or hyposthenic types) where the gallbladder is very superficial, one should consider the use of a linear superficial imaging transducer to optimise image quality.

The edge of the right hepatic lobe and the liver hilum are useful landmarks for the evaluation of the gallbladder. In the right subcostal oblique section the landmark structure to be used is the interlobar fissure and the gallbladder is found by aligning the probe with the fissure and then tilting it. The gallbladder will be located inferiorly or laterally to the fissure (between liver segments IV and V).

Conventional real-time ultrasound produces images of thin slices of the biliary tree on the screen. To be entirely convinced that the entire volume of the liver tissue and structures have been imaged it is essential that the operator scans the entire organ systematically, in at least
two anatomical planes. The operator must then use this two-dimensional information to envisage a three-dimensional map of the individual patient’s biliary tree anatomy and pathology. This requires good hand-eye-brain coordination.

The gallbladder and biliary tree can be examined initially with the patient in a supine position. This is to be encouraged as a first line approach to minimise the risks of operator repetitive strain injury owing to over-reaching. Successful examination of the gallbladder and biliary tree often requires the patient to be examined in a left posterior-oblique or left-lateral decubitus position. These latter positions cause the liver/gallbladder to rotate anteromedially under the influence of gravity and this may optimise the use of the liver to image the gallbladder through an acoustic window or make the gallbladder more readily accessible below the thoracic cage.

In patients with sludge or stones, movement is essential to assess whether the pathology moves as the patient moves. Erect imaging is particularly useful to assess whether gallstones are mobile because they will descend into the dependent part of the gallbladder (fundus) when the patient erect.

In a typical sthenic patient, the transducer can be placed in the mid-clavicular line on the anterior abdominal wall and the transducer position is adjusted until the gallbladder is located. The operator should try to use the liver as an acoustic window and avoid scanning through bowel by angling cranially. The patient may be asked to take a suspended full inspiration to cause the gallbladder to descend below the lower costal margin. The transducer is then rotated over the gallbladder until the true long-axis section of the gallbladder is achieved.

It is essential to image the gallbladder in the entire long-axis section and to angle the transducer so that the ultrasound beam is swept through the structure to ensure that the whole gallbladder has been imaged in its entire long-axis as this is a three-dimensional structure.

The normal gallbladder wall measures <3 mm in the anteroposterior diameter. The anterior wall of the gallbladder should always be measured as it is closer to the transducer. It should be measured in line with the axial resolution of the machine. After scanning the gallbladder in long-axis, the transducer should be rotated over the gallbladder through 90° to image the gallbladder in its true short-axis section. Again, the transducer should be angled (cranial-caudal) to image the gallbladder in its entirety.

The demonstration of the cystic duct is easiest in deep inspiration with the patient in supine or left-lateral decubitus. It can be visualised beginning from the infundibulum of the gallbladder.
The distal segment of the cystic duct is best seen with the patient in supine, in the plane through the hepatic portal and anterior to the portal vein.

Imaging the intrahepatic biliary tree is described in the liver chapter of this course book.

An acronym has shown to be didactically helpful [“SSOTM”] when thinking about interpreting ultrasound images:

- S = size
- M = measurement
- S = shape
- O = outline
- T = texture

Size: See anatomy. The size of the gallbladder should be subjectively assessed. In a fasted state it should measure 10 × 2–4 × 2–4 cm, but this depends on the volume of bile present. Typical bile volume is normally 40–60 ml, measured by a rotating ellipsoid). However gallbladder volume estimation is highly unreliable as it shows a wide intra and inter operator variability [(4;5)].

Shape: The gallbladder is a saccular structure which has a pear or teardrop shape in long-axis cross section when distended.

Outline: The normal gallbladder wall is thin, smooth and mildly echogenic, measuring 1–3 mm in thickness in the normal state. There is no pericholic fluid around the gallbladder in the normal state.

Texture: The normal gallbladder lumen should contain bile and should not have any space occupying lesions. Normal bile appears anechoic (i.e. is completely black) and devoid of any internal echoes.

**Congenital anomalies of the gallbladder**

There are a wide variety of congenital abnormalities that are rarely encountered in adult patients on sonography and are predominantly found in the paediatric setting. If congenital abnormalities are found during routine ultrasound examination the findings are, in the clinical context, mainly without consequences to patient management.

**Agenesis, hypoplasia and microgallbladder**

Agenesisia, hypoplasia and numerical anomalies of the gallbladder have to be considered. Agenesis (absence) of the gallbladder is rare and normally without clinical significance.
Approximately 50% of gallbladder agenesis cases are discovered at autopsy and they are often associated with duodenal atresia and other congenital anomalies. Hypoplasia is associated with extrahepatic biliary atresia. A microgallbladder is defined as being less than 2–3cm long, 0.5–1.5cm wide and regarded as a typical finding of cystic fibrosis, but can also be associated with idiopathic neonatal hepatitis and alpha-1-antitrypsin disease [(6)] (Figure 2).

**Figure 2** Microgallbladder. Congenital anomaly of the gallbladder in a patient with cystic fibrosis (CF). Microgallbladder is a typical finding in CF patients. GB, gallbladder; NIERE, kidney [(6;7)].

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**Abnormal position of the gallbladder**

Abnormal positions of the gallbladder are rare. Left-sided (with or without situs inversus), intrahepatic (<5% of the population), suprahepatic, lesser sac or abdominal wall and retroperitoneal have all been described [(8)].

**Other congenital anomalies of the gallbladder**

Variations of the gallbladder’s shape are encountered far more frequently, but clinically are rarely of importance. For example Phrygian cap (Figure 3) is an inversion of the distal fundus into the body, to which it may adhere. Phrygian cap is either an anatomical variant or an acquired abnormality and is present in up to 5% of patients on sonogram. Gallbladder diverticula and volvulus are also very rare [(9)].
Figure 3  Phrygian cap (arrow): a variation in the shape of the gallbladder.

Other congenital anomalies of the gallbladder include duplication (two gallbladders), bilobed gallbladder owing to longitudinal or transverse septum (more common) and hypoplastic narrowing of biliary channels (true biliary atresia). A multiseptated gallbladder may be congenital or acquired, and reveals three or more communicating compartments lined by columnar epithelium. In adults cholecystolithiasis is often present. Heterotopia of the gallbladder, typically an incidentally finding, is also called ectopia or choristoma and is defined by normal tissue in an abnormal location. Liver parenchymal, pancreatic or gastric heterotopia has also been observed on ultrasound. An “hourglass gallbladder” is divided by a central constriction and is regarded as a variant of a transverse septated gallbladder. Pathogenetically an hourglass gallbladder is usually acquired owing to septum of inflamed fibrous tissue or adenomyomatous hyperplasia.

Aberrant bile ducts (also known as ducts of Luschka) are rarely identified by ultrasound but are present in 10% of cholecystectomy specimens. They can be buried in the gallbladder wall and may communicate with intrahepatic bile ducts or larger accessory bile ducts, or can join with the cystic duct. Rokitansky-Aschoff sinuses are outpouchings of gallbladder mucosa that penetrate into the muscle wall (“acquired herniations”). Rokitansky-Aschoff sinuses (pseudodiverticula) may show progressive occlusion of communication with the gallbladder leading to cysts. Solitary congenital diverticula have a wide size range (from 5mm up to 10cm) and can be present in all three layers of the gallbladder wall. A wandering gallbladder shows a long mesentery or no firm attachment to the liver and is regarded at risk of torsion.
The described findings are rarely encountered during routine ultrasound, but are important to know when it comes to a differential diagnosis [(10)].

**Cholelithiasis**

The term cholelithiasis describes the presence of gallstones in the biliary tract. Depending on the localisation of the concrement a further distinction between cholecystolithiasis (in the gallbladder) (Figure 4 and 5) and choledocholithiasis (in the bile duct) is used. In 5–15% of patients a combination of both conditions can be seen.

**Figure 4** Typical cholecystolithiasis: isolated calculus in the gallbladder lumen. Acoustic shadowing (SS) is typically seen posterior to a calculus as a result of reflection of ultrasound and the lack of deeper echoes reaching the transducer.
Cholecystolithiasis is the most common disease of the biliary system. It is estimated that 10% of the adult population have gallbladder stones, and that a third of people over 70 years will have gallbladder stones \((3)\). Of the patients with gallstones, 35% will, in time, become symptomatic and require surgery \((11)\).

Cholecystolithiasis can be detected by transabdominal ultrasound with a high sensitivity, whereas choledocholithiasis is more difficult to detect \((12;13)\). The literature has recently been summarised \((1)\) and transabdominal ultrasound is the first line imaging method of choice for any kind of gallbladder stones. It has the additional benefits of not using ionising radiation and is relatively cheap and safe. The accuracy of ultrasonography for the diagnosis of gallstones is up to 96% for experienced operators.

The number, size, echotexture, acoustic shadowing and mobility of gallbladder stones should be recorded and analysed. The classic ultrasound appearance of the gallbladder stone is a hyperechoic/echogenic structure located within the gallbladder lumen with posterior acoustic shadowing. Calculi typically lie on the dependent wall of the gallbladder under the influence of gravity, but this is dependent on the density of bile. To optimise acoustic shadowing and detection of small calculi, it is essential to insonate the dependent wall of the gallbladder at 90°. The gallstones should demonstrate mobility in response to movement of the patient (the “rolling stone” sign). Calculi are more difficult to see when they are small in size (2–3mm) and in number. The gallbladder filled with stones (the “shell” sign) can be easily confused with air in the digestive tract if the examiner does not have sufficient experience.
Occasionally stones become impacted in the gallbladder infundibulum, which creates a hydrops. This is not always an easy diagnosis and can sometimes be missed because this region can be difficult to visualise in some patients. The close anatomical proximity to the gas containing gastrointestinal tract can cause problems when imaging this region. A sonographically conclusive diagnosis of stone origin is not possible at present. Non-calcified cholesterol/cholesterol derived stones do not typically show shadowing and may “float” in the gallbladder lumen in contrast to calcified stones. So called “pigment stones”, which are rarely found in Europe (in less than 10% of the population), are often multiple with complete acoustic shadowing [(12)]. Very small stones can be overlooked, but a routinely performed examination of the left lateral decubitus and a standing position improves the detection rate to almost 100% for an experienced operator. “Mirizzi-syndrome” with stone impaction in the cystic duct, which can cause an obstructive jaundice, may be more difficult to visualise.

Biliary sludge

Biliary sludge is sometimes the precursor of gallstones. It represents precipitate formed in the bile and the ultrasound appearance is of homogenous echogenic material in the gallbladder lumen, with no posterior acoustic shadowing (Figures 6 a and b). It typically forms a straight horizontal line superficially, with the sludge normally collecting on the dependent wall of the gallbladder because of gravity. It moves slowly with a change in patient position and it is recommended that the patient is moved during the scan to demonstrate the motion of the sludge. If the sludge completely fills the gallbladder it can be difficult to distinguish it from the adjacent liver parenchyma. Sometimes the sludge is organised in a round shape, is hypoechoic, with no posterior acoustic shadowing and is often called “ball-like” or “tumour-like” sludge (Figures 6).

Figure 6  Biliary sludge. Variable appearances of gallbladder (GB) sludge in the same patient with two different approaches (planes).
Figure 7  Sludge. Sludge might be diffusely distributed or solitary mimicking a polyp (p) or neoplasia. Contrast-enhanced ultrasound is helpful in the differential diagnosis.
Various amounts of sludge have been observed in fasting patients with or without motility disorders of the gallbladder. Sludge is also often seen in intensive care patients (25–47%), total parenteral nutrition, stenosis in the extrahepatic bile duct and pregnancy [(1;11)]. Differential diagnosis includes neoplasia, empyema and haemorrhage. Contrast-enhanced ultrasound (CEUS) is useful for the differential diagnosis with neoplasia (Figure 7).

**Hepaticolithiasis**

Intrahepatic stones (hepaticolithiasis) are rare and are far more common in Asia than in Europe. Congenital anomalies and parasitoses are the leading causes. The ultrasound appearance is hyperechoic structures with posterior acoustic shadowing inside the intrahepatic biliary ducts (sometimes with dilated intrahepatic biliary tree above the stones) [(14;15)].

**Cholecystitis**

Cholecystitis is defined as inflammation of the gallbladder and is frequently classified as acute or chronic.

**Acute cholecystitis**

Acute cholecystitis is the most frequent complication of cholecystolithiasis and occurs in almost a third of patients with gallstones. Acute cholecystitis can be divided into either gallstone associated (acute calculous cholecystitis) or non-gallstone associated (acute acalculous cholecystitis). 95% of the cases are due to calculous obstruction of the gallbladder neck or cystic duct. 50% have bacterial infection (*Escherichia coli*, *Enterobacter*, *Enterococcus*, *Klebsiella*, *Clostridium*, *Peptostreptococcus*, *Bacteroides*). More than 10% perforate without a cholecystectomy.

The sonographic appearance is an enlarged distended gallbladder with a thickened wall. The thickened multiple layered wall is a constant finding and is caused by oedema, haemorrhage, ulcers and pus (Figure 8). CDI reveals hypervascularisation of the wall, which represents the typically pathoanatomically described congested vessels (“angry red colour”). Gallbladder wall thickening has also been described in acute and chronic active hepatitis and liver cirrhosis. In the latter, it represents varices. Sometimes a hypoechoic inflammatory fluid collection is seen around the gallbladder (a hypoechoic “eye-brow”). The presence of
gallstones on ultrasound examination combined with a positive ultrasound Murphy’s sign has a positive predictive value of 92% for the diagnosis of acute cholecystitis [(16)].

Figure 8  Acute cholecystitis. Cholecystitis might be confused with neoplasia using B-mode ultrasound. (a) Image demonstrates thickening of the wall of the gallbladder and presence of a calculous consistent with acute cholecystitis. Contrast-enhanced ultrasound might be helpful for this differential diagnosis. (b) Image shows better delineation of the thickened gallbladder wall.

Acute acalculous cholecystitis represents only 5–10% of cases. Patients are usually severely debilitated owing to severe trauma, sepsis, shock, burns, cancer, diabetes, multiple blood transfusions, surgery or cystic duct obstruction from various causes. Mortality is extremely
high (10–50%). A rare form of acute acalculous cholecystitis is cocaine related acute cholecystitis (Figure 9).

**Figure 9**  Acute cholecystitis. Ultrasound can differentiate between either gallstone associated (acute calculous cholecystitis) or acute acalculous cholecystitis as shown in this case. ????, free fluid; Duod, duodenum; GB, gallbladder.

A similar (transient) appearance to acute cholecystitis can be seen in approximately 50% of patients with acute hepatitis [(17)] (Figure 10).

**Figure 10**  Acute hepatitis. A similar (transient) appearance as acute cholecystitis can be seen in patients with acute hepatitis. GB, gallbladder; LN, lymph node; EDEMA: edema of the gallbladder wall.
Emphysematous cholecystitis is a rare form of acute cholecystitis associated with diabetes and peripheral atherosclerotic disease [(18)]. Vascular compromise of the cystic artery has been described as the most important pathophysiological factor. Ultrasound reveals gas bubbles inside of the thickened gallbladder wall. Perforation is a typical complication.

**Bouveret's syndrome**

Gallbladder perforation owing to a large stone (usually over 25mm) and stone passage into the bulb of the duodenum is called Bouveret syndrome, which can be easily recognised with transabdominal ultrasound identifying air bubbles from the duodenal bulb into the lumen of the gallbladder (Figure 11). It is a rare condition, with a 0.3% incidence and associated with chronic cholelithiasis in 90% of the cases [(19)]. The first report of Bouveret's syndrome (in 1896) was published by Leon Bouveret who presented two patients with this disease [(20)]. Fistula formation is thought to occur as a result of adhesions between the gallbladder and the bowel wall owing to chronic inflammation, impaired arterial blood supply and decreased venous drainage [(21)]. Secondary, the stone lodges in the digestive tube, most commonly in the distal ileum (90%), colon (3–8%) or duodenum (3%) and rarely in the proximal duodenum or pylorus, causing gastric outlet obstruction (Bouveret's syndrome) [(22)]. The patients present with nausea, vomiting, epigastric pain and abdominal distension. Less commonly they can present with haematemesis, weight loss and anorexia.

Abdominal ultrasound is a useful diagnostic tool because it will reveal aerobilia and gas bubbles in the gallbladder, and it frequently confirms the stone in the duodenum. Diagnosis is completed by endoscopy [(23)]. In a review of 128 cases of Bouveret's syndrome, endoscopy revealed gastroduodenal obstruction in nearly all cases but identified the obstructing stone in only 69% of cases [(24)].

**Figure 11** Bouveret-syndrome might be suspected in patients with unsuspected aerobilie. (a,b) Bouveret's syndrome is defined as gastric outlet obstruction caused by duodenal impaction of a large gallstone, which passes into the duodenal bulb through a cholecystogastric or cholecystoduodenal fistula. Initial attempts at endoscopic retrieval with or without mechanical or
extracorporeal lithotripsy should be performed as first-line treatment, although success rates with endoscopic treatment are variable.

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**Chronic cholecystitis**

Chronic cholecystitis is sonographically characterised by an irregular thickened gallbladder wall, mainly caused by chronic inflammation and intermittent obstruction of the gallbladder neck or cystic duct by gallstones, often causing biliary colic. The gallbladder is frequently small and contracted, and care must be taken when imaging this region so as not to confuse the bowel with a small contracted gallbladder. 95% are associated with cholecystolithiasis or at least sludge (Figure 12).
Figure 12  Chronic cholecystitis. (a) Sometimes a sludge filled gallbladder may present as a tumour like lesion using B-mode ultrasound. (b) Contrast-enhanced ultrasound is helpful for the correct diagnosis because the ventral wall thickening does not take up the contrast enhancer and can therefore be differentiated from a carcinoma.

Bacteria are present in up to a third of patients, and include similar organisms to acute cholecystitis. There are many different forms of chronic cholecystitis that cannot be differentiated by ultrasound.

Diffuse lymphoplasmacytic acalculous cholecystitis is a relatively sensitive sign for primary sclerosing cholangitis (PSC). An association with lymphoplasmacytic sclerosing pancreatitis has also been described.
Some other forms are acquired immune deficiency syndrome (AIDS) related. Acute cholecystitis can be caused by opportunistic infections such as cryptosporidia, cytomegalovirus (CMV) and microsporidia. Eosinophilic cholecystitis is seen in Churg-Strauss syndrome. In addition follicular cholecystitis (also called lymphoid polyp) can occasionally be observed [(25)(26)].

Other rare forms of cholecystitis include: granulomatous cholecystitis in patients with tuberculosis and xanthogranulomatous cholecystitis (as a result of rupture of the Rokitansky-Aschoff sinuses with extravasation of bile or ulceration of gallbladder mucosa). Gangrenous cholecystitis occurs in 15% of acute cholecystitis cases with mural infarction, and with perforation in more than 25%. Typically, air is found in the gallbladder (known as pneumobilia). Clostridium perfringes appears to be of pathophysiological importance.

Porcelain gallbladder is found in 0.5% of cholecystectomies. An association (>20%) with gallbladder carcinoma is well known [1]. Cholecystectomy is therefore indicated when a porcelain gallbladder is diagnosed on ultrasound. Sonographically the calcified wall can be easily detected. It is characterised by an intramural shell-like calcification that may affect the entire wall or parts of it.

“Limy bile”, in which there is a pathological accumulation of calcium carbonate in the gallbladder, is a very rare condition. The diagnosis is made on a plain abdominal radiograph, where the gallbladder appears as an opaque pear-shaped structure.

**Miscellaneous non-neoplastic disorders**

Miscellaneous non-neoplastic disorders are various. Adenomyomatous hyperplasia is also called adenomyomatosis or diverticular disease of the gallbladder. Commonly ultrasound can only demonstrate a thickened wall. Adenomyomatous hyperplasia (generalised, segmental or localised) is relatively common (in up to 10% of cholecystectomy specimens) and it is usually asymptomatic. In the generalised form, diffuse wall thickening (>10mm) is found with intramural diverticula resembling cystic spaces within the wall. In the segmental form focal thickening of the gallbladder wall can usually be seen in the body, which gives it an hourglass configuration. In the localised form the fundus shows 0.5–3cm nodules with grey-white cut surface containing multiple cysts. The latter may cause gallbladder inversion and is also called adenomyoma.

Asymptomatic cholesterolosis is mostly characterised by cholesterol infiltration (sonographically shown as comet-tail artefacts) in an otherwise normal gallbladder wall. Cholesterolosis is present in up to 20% of cholecystectomy specimens, usually found in adult
multiparous women. Cholesterolosis is associated with bile supersaturation with cholesterol, but not with increased serum cholesterol. Cholesterol infiltration is due to an accumulation of cholesterol esters and triglycerides in subepithelial macrophages and gallbladder epithelium. Macroscopically focal or diffuse yellow flat deposits are seen on the mucosal surface, which may have speckled appearance (“strawberry gallbladder”). Association with cholesterol polyps is reported in 20%. A similar (unspecific) irregular gallbladder wall can be seen in amyloidosis (Figure 13) [(27)].

**Figure 13  Amyloidosis of the gallbladder may show irregular deposits (white arrow) [(27)].**

Gallbladder varices as a cause of gallbladder thickening can be excluded using CDI.

**Benign gallbladder tumours (polyps)**

Benign gallbladder tumours are divided into adenoma of the gallbladder (typically demonstrating central vessels penetrating the polyp using CDI) (Figure 14), adenomyosis, cholesterol polyps (containing cholesterol deposits and therefore no or few vessels), hyperplastic/metaplastic polyps, granular cell tumour (often associated with similar lesions in extrahepatic bile ducts), inflammatory polyps and villous papilloma.
Figure 14  Adenoma (7 mm) of the gallbladder typically demonstrating central vessels penetrating the polyp using colour Doppler imaging.

Adenoma of the gallbladder are mainly single (90%), rarely multiple (10%) and contain, by definition, at least low grade dysplastic epithelium. An increased prevalence is found with familial adenomatous polyposis or Peutz-Jeghers syndrome. Invasive carcinoma is rarely reported in lesions under 10mm. The treatment is total excision respective cholecystectomy. Adenomyosis is caused by hyperplasia of muscularis propria with intramural hyperplastic or cystically dilated glands mainly in the fundus. They represent 15–25% of benign polyps and appear on ultrasound as hypoechoic round structures, attached to the gallbladder wall, with no posterior acoustic shadowing. They do not move with a change in patient position, which can be used for a differential diagnosis with cholecystolithiasis. Cholesterol polyps are the most common benign polyps (50–90%). Inflammatory polyps are associated with chronic cholecystitis and are described as 3–15mm, usually sessile and single polyps, macroscopically red/grey/brown in colour. Multiple adenoma are often encountered (Figure 15).
Growing gallbladder polyps or polyps larger than 10mm should be surgically removed owing to the potential malignant transformation.

**Gallbladder carcinoma**

Gallbladder carcinoma is a rare but highly fatal malignancy. It is associated, in almost 100% of the cases, with cholecystolithiasis and occurs more frequently in patients over 60 years old. The risk of developing gallbladder cancer in a patient with gallbladder stones is 0.3% over a 30 year period and published data suggest a much higher cancer risk in stones larger than 3cm.

Carcinomas of the gallbladder can be easily recognised using transabdominal ultrasound, whereas correct staging is much more difficult and underestimation of the extent of the disease is possible. Complementary imaging such as CT and/or MRI is often required. The majority of cases are found incidentally in patients with cholelithiasis, and in 1–2% of these cases a gallbladder carcinoma can be found [(28)]. As with other tumours of the upper GI tract, a definite sonographic distinction between inflammatory and neoplastic alterations or changes in connective tissue is not possible. In some cases a sonographic diagnosis of adenomyomatosis, cholesterol polyps and other pathologies of the gallbladder can be made. The sonographic signs of a gallbladder carcinoma are irregular wall thickening, a poorly defined polypoid mass protruding in the gallbladder lumen, or, if the disease is more advanced, the replacement of the gallbladder by a solid, normally hypoechoic, mass that
completely fills the gallbladder. The presence of a gallstone in relation to this mass suggests the diagnosis of gallbladder carcinoma. The symptoms of patients with gallbladder carcinoma are often non-specific and vague; therefore, the diagnosis is made when the disease is in an advanced stage, hence the poor prognosis and the name “silent tumour”. The most common symptom of patients in the advanced stage of the disease is pain followed by anorexia. CEUS can be useful for the diagnosis of gallbladder carcinoma (Figure 16) and can help in the differentiation of normal and infiltrated areas. It can also help in the assessment of liver metastases.

**Figure 16** Gallbladder carcinoma. (a) Carcinomas of the gallbladder can usually only be recognised using conventional B-mode transabdominal ultrasound. (b) Contrast-enhanced ultrasound is helpful in delineating tumour infiltration.
Sometimes other morphology may imitate gallbladder neoplasia, e.g. hyper-regenerative nodules in liver cirrhosis (Figure 17) or even polyps.

**Figure 17** (a) Differential diagnosis of gallbladder neoplasia (between callipers (+)) may be an atypical liver appearance. (b) Demonstration in two anatomical levels is helpful for the correct diagnosis.

Cholangio(cellular) carcinoma (including Klatskin tumours)

Cholangio(cellular) carcinomas (CCCs), which are histologically mainly adenocarcinoma, are more frequently detected using transabdominal ultrasound with the newer high-resolution
ultrasound systems. They arise from the epithelial cells of the intrahepatic and extrahepatic bile ducts. Carcinomas of the bile duct are much more common extrahepatically but can also be found intrahepatically (<10%).

Perihilar CCCs of the bile ducts are called Klatskin tumours and are classified by Bismuth-Corlette I-IV depending on the localisation of the tumour and involvement of the hepatic ducts (Table 2).

**Table 2** Bismuth-Corlette classification.

<table>
<thead>
<tr>
<th>Bismuth-Corlette classification</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Tumours below the confluence of the left and right hepatic ducts.</td>
</tr>
<tr>
<td>II</td>
<td>Tumours reaching the confluence</td>
</tr>
<tr>
<td>III</td>
<td>Tumours occluding the common hepatic duct and either the right or left hepatic duct (Type a or b).</td>
</tr>
<tr>
<td>IV</td>
<td>Tumours that are multicentric or involve the confluence and both the right or left hepatic duct</td>
</tr>
</tbody>
</table>

Bismuth-Corelette Type IV can be easily confused on sonography with hepatocellular carcinoma. Mixed forms have been observed. Sonographically dilated bile ducts proximal of the stenosis is a typical finding. Delineation of the tumour is more difficult. Intrahepatic CCCs can often be found subcapsular, polygonal and diffusely delineated. Risk factors for the development of a CCC are PSC, infection of the hepatobiliary tract with liver fluke (*e.g.* *Clonorchis sinensis*), congenital abnormalities of the hepatobiliary tract (*e.g.* biliary atresia, choledochal cysts). Bile duct adenoma are rarely encountered ([29]).

CEUS has improved the detection and characterisation of CCC but prospective studies are lacking. Peritoneal metastases are common but are difficult to visualise reliably using imaging methods without laparoscopy.

CCC can occur along the bile duct as Klatskin tumours (hilar CCC are more common), but they may also appear as primary solid tumours in the liver (peripheral CCC). For the peripheral type there are no typical sonographic characteristics and the diagnosis is usually made incidentally within the framework of a biopsy of an unclear liver lesion. Ultrasound examination shows a solid mass that can have any echogenicity and exhibits signs of a malignant growth. Typically the liver metastases of a peripheral CCC are situated like satellites around the primary focus.
For reasons that are still unclear, the incidence of intrahepatic cholangiocarcinoma has been rising over the past decade all over the world, while rates of extrahepatic cholangiocarcinoma are declining [(30;31)].

**Extrahepatic bile duct**

The extrahepatic bile duct system can be easily displayed using transabdominal ultrasound with a patient in a left-posterior oblique or left-lateral decubitus position (Figures 18 and 19). This position causes the liver to rotate anteromedially, and thus the liver can be used as an acoustic window for imaging the extrahepatic biliary tree. The main extrahepatic bile duct often lies obliquely and it is superiorly more lateral so it can usually be imaged by placing the transducer below the right costal margin in the region of the mid-clavicular line; an oblique position is required to align the transducer parallel to the long-axis of the bile duct. The MBD appears as a tube situated in front of the portal vein. When there is a good acoustic window, the MBD can be followed into the retropancreatic portion to the papilla of Vateri. Often, the bile duct is imaged with the transducer parallel to the midline. In this section the hepatic artery will normally appear as a round structure between the MBD and the portal vein. Frequently the duct is measured at this point; however, a single measurement of the bile duct at this level can be misleading as the duct may be normal at this point, but distended lower down in early obstructive jaundice and it is therefore recommended that the duct be imaged along the entire length and measured at several points, including near to the head of pancreas. The duct should be evaluated for size (normal is up to 6mm), wall thickness and content. After colecystectomy the normal size of the MBD may increase. In some patients the papilla of Vateri at the end of the CBD can be displayed using transabdominal ultrasound.

**Figure 18** Papilla of Vateri. (a) The papilla of Vateri at the distal end of the common bile duct. (b) The main pancreatic duct is displayed next to the papilla. DW, main pancreatic duct; DHC, common bile duct.
Figure 19  Slightly dilated main bile duct (10mm) next to the papilla. DP, pancreatic duct; DHC, common bile duct
**Patients with jaundice**

Patients with jaundice should be examined sonographically as early as possible, ideally as soon as they present. In some cases it is better to scan the patient without fasting to assess the case and then repeat after fasting if necessary. For example, dilated intrahepatic bile ducts can be seen without fasting and may account for the jaundice. Ultrasound can differentiate jaundice caused by obstructive and hepatocellular origin in almost all patients by focusing and analysing the diameter of the main bile duct. Figure 19 shows a mildly distended main bile duct, but microlithiasis might be overlooked. The left lateral decubitus position is helpful for adequate visualisation of the liver hilum [(32-34)] and should be performed consistently in all patients. The papilla Vateri is less reliably displayed using the transabdominal approach.

**Congenital disorders**

**Choledochal cysts**

In infants choledochal cysts are the most common cause of obstructive jaundice, but they may be found at any age. They are not actually cysts but a dilatation of the CBD, which may secondarily obstruct other biliary ducts or the duodenum (Figure 20). Choledochal cysts (classification of Todani Type 1–5 (Table 3)) are often associated with other hepatobiliary tract abnormalities, e.g. anomalous pancreaticobiliary duct junction [(35)]. Choledochal cysts may rupture spontaneously [(36–38)].

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>Characterised by a segmental or diffuse fusiform dilation of CBD (50–90% of all cases).</td>
</tr>
<tr>
<td>Type 2</td>
<td>Diverticulum of CBD</td>
</tr>
<tr>
<td>Type 3</td>
<td>Dilation of intraduodenal CBD (choledochocele)</td>
</tr>
<tr>
<td>Type 4</td>
<td>Multiple cysts of extrahepatic bile ducts with (4a) or without (4b) cysts of intrahepatic ducts</td>
</tr>
<tr>
<td>Type 5</td>
<td>One or more cysts of intrahepatic ducts (Caroli’s disease)</td>
</tr>
</tbody>
</table>

CBD, common bile duct
It is importance to note that the mean age to develop biliary tract carcinoma for 2–10% of patients is 35 years. Carcinomas may develop within the wall of the cyst, the gallbladder or bile ducts. Therapeutically complete cyst removal with biliary reconstruction, usually with Roux-en-Y hepaticojejunostomy should be performed when a carcinoma is diagnosed in these patients.

The role of ultrasound techniques is not yet fully determined because of a low incidence of this disease in adults and limited clinical experience. MRI and endoscopic retrograde cholangiography (ERC) are the diagnostic methods of choice. Endoscopic ultrasound (EUS) should be considered because of the excellent resolution of the CBD.

**Figure 20** Choledochal cysts are the most common cause of obstructive jaundice in infants and beyond infancy, but can be found at any age. This image shows tortuous distension of the main bile duct (D). Typically gallstones and sludge are displayed (S).

**Choledocholithiasis**

Cholecystolithiasis can be detected by transabdominal ultrasound with high sensitivity (Table 4), whereas choledocholithiasis is more difficult to detect (Table 5). Dietrich et al have summarised the literature [(39)]. In contrast to the very helpful shadowing exhibited by gallbladder stones, primary choledocholithiasis does not often show shadowing, especially when the stones are very small. Even with the most modern ultrasound equipment the
sensitivity for choledocholithiasis is still largely dependent on the expertise of the ultrasound practitioner with a large difference of between 25% and 100%.

In contrast to the transabdominal approach EUS and miniprobe endosonography (extraductal ultrasound (EDUS) or EUS) are more efficient. EUS shows a detection rate of 94–100% while EDUS diagnosis of choledocholithiasis was confirmed to be correct in 33 out of 34 patients (97%). As expected, EDUS failed to detect peripheral lesions [(40)]. Parasites also have to be considered, e.g. *Ascarias* particularly in certain geographical regions [(14;41)].

**Table 4** Detection of cholecystolithiasis by transabdominal ultrasound – a review of the literature [(39)]

<table>
<thead>
<tr>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>98</td>
<td>94–98</td>
<td>(12)</td>
</tr>
<tr>
<td>70</td>
<td>100</td>
<td>(42)</td>
</tr>
<tr>
<td>97</td>
<td>92</td>
<td>(43)</td>
</tr>
<tr>
<td>91</td>
<td>99</td>
<td>(44)</td>
</tr>
<tr>
<td>98</td>
<td>nm</td>
<td>(45)</td>
</tr>
<tr>
<td>91</td>
<td>100</td>
<td>(46)</td>
</tr>
<tr>
<td>87</td>
<td>93</td>
<td>(47)</td>
</tr>
</tbody>
</table>

nm, not mentioned

**Table 5** Detection of choledocholithiasis by transabdominal ultrasound – review of the literature [(39)].

<table>
<thead>
<tr>
<th>Patients (n)</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>NPV (%)</th>
<th>PPV (%)</th>
<th>Gold standard</th>
</tr>
</thead>
<tbody>
<tr>
<td>62</td>
<td>25</td>
<td>100</td>
<td>56</td>
<td>100</td>
<td>ERCP with or without EST or IOC</td>
</tr>
<tr>
<td>52</td>
<td>80</td>
<td>94</td>
<td>nm</td>
<td>nm</td>
<td>ERCP/EST or surg. expl.</td>
</tr>
<tr>
<td>nm</td>
<td>38</td>
<td>100</td>
<td>nm</td>
<td>nm</td>
<td>No results</td>
</tr>
<tr>
<td>35</td>
<td>47</td>
<td>90</td>
<td>nm</td>
<td>nm</td>
<td>ERCP/EST</td>
</tr>
<tr>
<td>142</td>
<td>63</td>
<td>95</td>
<td>nm</td>
<td>nm</td>
<td>ERCP with or without EST or surg. expl.</td>
</tr>
<tr>
<td>36</td>
<td>50</td>
<td>100</td>
<td>74</td>
<td>100</td>
<td>ERCP/EST</td>
</tr>
<tr>
<td>50</td>
<td>100</td>
<td>97</td>
<td>92</td>
<td>100</td>
<td>ERCP/PTC</td>
</tr>
<tr>
<td>132</td>
<td>68</td>
<td>nm</td>
<td>nm</td>
<td>nm</td>
<td>ERCP/EST</td>
</tr>
<tr>
<td>29</td>
<td>38</td>
<td>100</td>
<td>nm</td>
<td>nm</td>
<td>ERCP</td>
</tr>
</tbody>
</table>
Endoscopic retrograde cholangiopancreatography (ERCP) was considered the diagnostic gold standard with a reported success rate of 90–96%. However, the value of diagnostic ERCP might be grossly overestimated, because the rate of correctly diagnosed choledocholithiasis seems to be much lower, especially because small gallstones (<3mm) with normal or even dilated bile ducts are easily overlooked with all diagnostic techniques, even ERCP. The combination of ERCP with endoscopic sphincterotomy (EST), including stone extraction using the dormia basket or balloon, is now the therapeutic method of choice in patients with choledocholithiasis, but it is an invasive technique with a significant risk of complication for the patient [(40)]. The reported results using magnetic resonance cholangiopancreaticography (MRCP) and CT are less convincing, especially in small stones without MBD dilatation. However, these imaging techniques are improving and it is quite likely that these methods will become more important in the next few years, particularly because they are non-invasive. The method of choice to exclude choledocholithiasis without sphincterotomy is EUS.

Figure 21 Choledocholithiasis (between callipers (×)). In contrast to the shadowing exhibited by gallbladder stones (seen in this image) primary choledocholithiasis does not often show shadowing and is, therefore, more difficult to diagnose.
Cholangitis

Sonographic features of cholangitis (Figure 22) are enlarged extrahepatic bile ducts with more or less symmetrical thickening of the wall in contrast to asymmetric thickening in PSC.

Figure 22  Cholangitis. Image shows a thickened bile duct wall (between callipers (×)).


Primary sclerosing cholangitis

PSC is a chronic inflammatory liver disease characterised by progressive fibrosis and destruction of the intra- and extrahepatic biliary tree leading to stricturing of the intrahepatic and/or extrahepatic bile ducts. The aetiology and pathogenicity of PSC are not well understood. It has a clear association with inflammatory bowel disease and is often progressive, leading to liver cirrhosis and end-stage liver failure. PSC is a pre-cancerosis condition and the differentiation to cholangiocarcinoma is very difficult. Up to 20% of patients with PSC develop cholangiocarcinoma. On ultrasound examination, enlarged perihepatic lymphnodes can be visualised in >90% of cases [(48;49)] and asymmetric thickening of the bile duct walls with benign strictures and alternating dilatations can be found in 70% (Figure 23) [(48)].

Cholangiocarcinoma in PSC is often detected at an advanced stage. Patients present with jaundice, weight loss and abdominal pain. Screening strategies used include regularly performed transabdominal ultrasound to detect the malignant tumour at an early stage with a treatment option.
Figure 23  Primary sclerosing cholangitis. Asymmetric thickening of the bile duct walls (in between markers) with benign strictures and alternating dilatations (to the left of the arrow) are typical sonographic findings in patients with primary sclerosing cholangitis and are found in more than 70% of cases.

Secondary sclerosing cholangitis

Secondary sclerosing cholangitis used to be much more common than PSC. Typical causes were biliary obstruction caused by choledocholithiasis, post-operative, chronic pancreatitis, choledochal cyst or extrahepatic biliary atresia. Infections in immunodeficient patients have also been encountered, as well as toxins, ischaemia and malignancy. The most significant complications of secondary sclerosing cholangitis, e.g. hepatic lobar atrophy, can be reduced and are rarely seen today because of improved diagnostics and therapy. Typical sonographic findings in these patients are enlarged extrahepatic bile ducts with more or less symmetrical thickening of the wall in contrast to asymmetric thickening in PSC. However, the diagnosis of secondary sclerosing cholangitis is not made by ultrasound; the gold standard is ERC in combination with patient history.
Parasitic infections

In Asia the risk of suffering a cholangiocarcinoma of the intrahepatic bile ducts is associated with infection with liver flukes such as *Clonorchis* and *Opisthorchis*. Patients are infected by eating raw or undercooked fish with the adult worms inhabiting and laying eggs in the biliary tree. This leads to a chronic inflammation with a malignant transformation of the epithelium.

Cholangiocellular carcinomas (extrahepatic)

CCCs are more common extrahepatically (Figure 24) but can also be found intrahepatically (so called Klatskin tumours). Sonographically dilated bile ducts proximal of the stenosis are the typical finding. Delineation of the, often small, initial tumour is more difficult. CEUS has improved the detection and characterisation of CCC.

Figure 24 Cholangiocellular carcinoma. Adenoma of the papilla and extrahepatic cholangiocellular carcinoma are drained by stents that can be easily displayed by ultrasound. In this patient with severe deficits after stroke, who had refused operation, an adenoma of the papilla was treated and followed up for more than 8 years when a carcinoma finally developed.

Colour Doppler imaging

The majority of circumscribed CCCs are slightly hyperperfused in the native colour Doppler but CDI findings vary widely.
Contrast-enhanced ultrasound

CEUS can be very helpful and displays a variable, but mainly hyperperfused, perfusion picture in the arterial phase. In the late portal venous phase CCCs are contrasted as punched-out defects. This behaviour is not always easy to demonstrate in the case of the Klatskin tumours, which often exhibit an appreciable pericholangitic component. As far as differential diagnoses are concerned, in the case of the hilar type of CCC, inflammatory bile duct alterations should be considered, e.g. cholangitis. However, stratification of the bile ducts can be preserved and may actually be accentuated in the sonographic image. For the detection of CCCs the examination technique in the liver specific late phase has proved to be diagnostically useful in patients showing normal CT, MRI and MRCP results, but so far there have been no conclusive studies on differential diagnosis of PSC and CCCs.

Other tumours of extrahepatic bile ducts

Adenomas represent only 10% of the incidence of carcinoma and are much more common in the gallbladder than in the extrahepatic biliary tree (Figure 25). Other forms of benign tumours of extrahepatic bile ducts are rare, e.g. carcinoid, and show no pathognomonic ultrasound patterns. Even rarer is the so called benign hepatobiliary papillomatosis now described as intraductal papillary mucinous neoplasia of the bile ducts.

Figure 25 Adenomas of the papilla Vateri (between markers) are rarely encountered on ultrasound. Sometimes it is difficult to differentiate sludge from neoplasia. Contrast-enhanced ultrasound techniques and elastography are helpful in delineating vascularised and depth infiltration. (a) This image sequence shows an adenoma shown by transabdominal B-mode ultrasound, (b) transabdominal contrast-enhanced ultrasound, (c) endoscopic elastography [(50)] and (d) contrast enhanced endoscopic ultrasound using low mechanical index technique (CELMI EUS) [(51;52)]. (e) In contrast to adenoma in (harder) carcinoma infiltration of deeper layers can be delineated using elastography, shown in a patient with malignant infiltration.
Carcinomas of extrahepatic bile ducts account for 95% of adenocarcinoma of all extrahepatic bile duct malignancies (bile duct carcinoma and cholangiocarcinoma). Klatskin (hilar) tumours comprise 70% of tumours and arise at the confluence of right and left hepatic ducts at liver hilus. Mostly they are slow growing with infrequent distant metastases.

Tumour, node, metastasis (TNM) staging for extrahepatic bile duct carcinoma applies to carcinomas arising above ampulla of Vater, including carcinomas in congenital choledochal cysts and intrapancreatic portion of the CBD. The classification excludes sarcomas and carcinoid tumours.

Features to report from a surgical point of view include obstruction, bile duct wall thickness (as a sign of infection), stones, tumour location and size, depth of invasion, tumour extension to adjacent structures and regional lymph nodes.

After endoscopic therapy of a biliary obstruction with papillotomy and stent placement, the position of the stent can be easily controlled by ultrasound (Figure 26).
Figure 26  Bile duct carcinoma drained by stents can be easily displayed on ultrasound.
(a) A stent is shown using panoramic imaging. (b) Stents can sometimes be better delineated using low mechanical index harmonic imaging techniques.

Pneumobilia (also known as aerobilia)

Pneumobilia can be caused by a variety of diseases, e.g. perforation of stones of the biliary duct system into the GI tract. However, the most common reason of pneumobilia can be found after papillotomy with installation of a biliary stent. A very rare, but alarming, cause for pneumobilia is a fulminant cholangitis with aerogenic bacteria. Pneumobilia is normally present after endoscopic sphincterotomy and surgical biliodigestive anastomosis.
Characteristic signs that can be found on ultrasound examination are linear jerks with typical reverberations ("ring-down-artefact") and in contrast to sessile calcifications air is movable in the bile ducts if the patient position is changed (Figure 27).

**Figure 27** (a) Pneumobilia of peripheral bile ducts after papillotomy. Pneumobilia can be helpful in the differentiation of (b) sufficiently and (c) insufficiently drained liver lobes.
Other forms of drainage also show a typical appearance, *e.g.* metal stents (Figure 28) and surgical drainage (Figure 29).

**Figure 28** Metal stent (between markers).
New techniques

CEUS has been introduced into daily clinical procedure for many presentations when imaging the liver, pancreas and kidneys and for monitoring local ablative treatment, mainly of the liver, but also of other organs. New applications have also been introduced, e.g. the application of BR1 (SonoVue®, Bracco, Italy) into the bile ducts via a conventional ERC. Percutaneous transhepatic cholangiography and drainage (PTCD) is a procedure for diagnosis and treatment of dilated bile ducts in both malignant and benign biliary obstruction. PTCD has some limitations owing to the blind puncture technique of peripherally located intrahepatic bile ducts. Severe (major) complications have been described in approximately 2% of cholangiography and up to 10% of transcutaneous interventions, e.g. sepsis, haemorrhage, abscesses, peritonitis and haematobilia the latter especially true when puncturing close to the liver hilum. Nevertheless, the complication rate varies with patients co-morbidity and investigators experience. Ultrasound-guided PTCD has been described for dilated and non-dilated bile ducts and several studies have shown a reduction in complications and a faster access to the biliary ducts. CEUS-PTCD has recently been described. Future studies in a larger numbers of patients are necessary to evaluate this new technique concerning the optimal dosage, the limitations and additional indications [(52)]. Other hepatobiliary interventions have been recently summarised [(54;55)].
Figure 30  Choledocholithiasis (arrow) diagnosed by contrast enhanced ultrasound and contrast-enhanced percutaneous cholangiography and treated by contrast-enhanced ultrasound guided cholangiodrainage [(52)]. (a) Choledocholithiasis shown by B-mode ultrasound may be confused with tumour like lesions but intravascular (b) and extra vascular contrast-enhanced ultrasound can confirm this differential diagnosis.
References


