COMPLICATIONS OF ACUTE CHOLECYSTITIS

Guidelines for Medical Students
Approved at the meeting of the surgical methodological commission of Danylo Halytsky Lviv National Medical University (Meeting report № 56 on May 16, 2019)

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1. **Background.** Cholelithiasis (gallstone disease) disease remains one of the most common medical problems leading to urgent and elective surgery. Cholelithiasis affects approximately 10% of the adult population in the world. It has been well demonstrated that the presence of gallstones increases with age. An estimated 20% of adults over 50 years of age and 30% of those over age 70 have biliary calculi. With every year the amount of these patients increases, and a cholecystitis with complications of cholelithiasis occupy the second place after appendicitis (some years – the first place). In the US, approximately 700,000 cholecystectomies are performed every year, including 150,000 interventions because of cholelithiasis complications. Scientific and technical progress in medicine was instrumental in the origin of new perspective directions in treatment of gallstone disease is the use of preparations for dissolution of stones, extracorporal shock wave lithotripsy, contact electrohydraulic lithotripsy, open and laparoscopic operations with bile duct explorations. Such operations, as cholecystectomy and stone extraction from the common bile duct, bilioenteric anastomoses (cholecystojejunostomy performed by N.Monastyrsky, choledochoduodenostomy performed by H.Finsterer and O.Sprengel, hepaticojejunostomy on the Roux-en-Y limb by R.Dahl), remain basic in surgical treatment of patients with gallstone disease, complicated with choledocholithiasis. However, development and perfection of endoscopic and video-laparoscopic techniques changed surgery of complicated gallbladder stone disease. Inculcated in clinical practice in 1974th the method endoscopic retrograde sphincterotomy and lithoextraction (M.Klassen & L.Demling, K.Y.Kawai) and laparoscopic bile duct exploration become the methods of choice and on this time all more often successfully used in patients with any biliary problems.

**Duration of lesson:** 4 hours

**Learning Objectives:**
To know (α = I; α = II):
- determination of concept is “gallstone disease”, “complications of cholelithiasis”;
- modern understanding of pathogenesis of cholelithiasis;
- classification of cholelithiasis and complications;
- morphologic changes are in the bile ducts and Vater’s papilla;
- frequency of complications and clinical signs;
- features of clinical symptoms depending on the different forms of complicated cholecystitis;
- diagnostic possibilities of additional methods of investigation (laboratory tests, X-ray, ultrasonography, computed tomography, magnetic resonance imaging, cholescintigraphy, endoscopic retrograde cholangiopancreatography, endosonography);
- differential diagnostics of complicated cholecystitis;
- surgical treatment patients depending on type of complications;
- principles of surgery;
- biliary drainage: indications and methods.

Able to:
- propose a diagnosis;
- define the type of complication of cholecystitis;
- diagnose the complication of cholecystitis (choledocholithiasis, jaundice, cholangitis, biliary pancreatitis, empyema, abscess, peritonitis, hepatitis);
- analyse the laboratory tests and investigations data;
- conduct the differential diagnosis;
- formulate a final diagnosis;
- appoint treatment;
- define indications to surgery;
- appoint postoperative treatment.

**Practical skills:**
• capture of anamnesis and its analysis;
• examination of patients with liver, gallbladder and bile ducts diseases;
• determination of signs, characteristic for a chronic and acute complicated cholecystitis;
• interpretation of laboratory tests data and examination;
• formulation of indication and contraindication for surgical treatment;
• choose the method of surgery or mini-invasive treatment.

4. Interdisciplinary integration

<table>
<thead>
<tr>
<th>№</th>
<th>Subject and proper department</th>
<th>To know</th>
<th>To be able</th>
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<tbody>
<tr>
<td>1</td>
<td>Anatomy, topographical anatomy (departments of Anatomy of human, Topographical anatomy and operative surgery)</td>
<td>Anatomy and topographical anatomy of liver, gallbladder and biliary system.</td>
<td>To palpate liver and gallbladder.</td>
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<td>2</td>
<td>Morphology (department of Pathoanatomy &amp; Morphology)</td>
<td>Morphological description of complications of acute cholecystitis.</td>
<td>To define morphological changes of gallbladder, bile ducts, Vater’s papilla and pancreas in the different types of complicated acute cholecystitis.</td>
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<tr>
<td>3</td>
<td>Anatomy, topographical anatomy (departments of Anatomy of human, Topographical anatomy and operative surgery)</td>
<td>Surgical approach, methods of operations</td>
<td>To choose the adequate method of surgery</td>
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<tr>
<td>4</td>
<td>Biological chemistry (department of Biological chemistry)</td>
<td>Test interpretation of surgical diseases</td>
<td>To interpretate the blood tests in patients with an complicated cholecystitis.</td>
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<tr>
<td>5</td>
<td>Internal diseases (department of Internal diseases)</td>
<td>Interpretation of examination of organs of abdominal region</td>
<td>To conduct an examination patient with complications of acute cholecystitis.</td>
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**Type clinical departments**

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<th>Subject and proper department</th>
<th>To know</th>
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<tbody>
<tr>
<td>1</td>
<td>General surgery (department of General surgery)</td>
<td>Basic principles of work of surgical department and operating block in emergency.</td>
<td>Hospitalize patients to surgical department, to prepare for the treatment and diagnostic options and surgery.</td>
</tr>
<tr>
<td>2</td>
<td>Department of Internal diseases</td>
<td>Methods of diagnostics of acute cholecystitis and its complications, pathogenesis and clinical variants.</td>
<td>To find out the complaints of patient, collect anamnesis of disease, conduct an examination of patient, ground a diagnosis, conduct a differential diagnosis, plan of additional examination.</td>
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V. Contents of the topic and its structuring

Pay a regard to etiologic factors which result in inflammation of gallbladder, bile ducts and pancreas. Acute cholecystitis with complications more frequent develops on the background of gallstone disease. The ratio of complications is 3-10 : 1; the female-to-male ratio is about 4:1, with the sex discrepancy narrowing in the older population to near equality.

CLASSIFICATION
Classification of gallbladder diseases and biliary system (WHO, ICD – 10):

Stone of gallbladder
- with an acute cholecystitis  \( \kappa 80.0 \)
- with other cholecystitis  \( \kappa 80.1 \)
- without a cholecystitis  \( \kappa 80.2 \)

Stone of bile ducts
- with a cholecystitis  \( \kappa 80.4 \)
- with a cholangitis  \( \kappa 80.3 \)
- without a cholecystitis or cholangitis \( \kappa 80.5 \)
- Other forms of cholelithiasis \( \kappa 80.8 \)
- Other forms of cholecystitis \( \kappa 81.8 \)
- Hydrops of gallbladder \( \kappa 82.1 \)
- Fistula of gallbladder \( \kappa 82.3 \)
- Cholangitis \( \kappa 83.0 \)
- The perforation of bile ducts \( \kappa 83.2 \)
- Strictures of common bile duct \( \kappa 83.8 \)
- Fistula of bile ducts \( \kappa 83.3 \)
- Postcholecystectomy syndrome \( \kappa 91.5 \)

The most often complications of acute cholecystitis:

1. Paravesical mass.
2. Paravesical abscess.
3. Empyema of gallbladder.
4. Perforation of gallbladder.
5. Peritonitis (localized, spread, total).
6. Cholecholitiasis.
7. Obstruction of common bile duct.
8. Obstructive jaundice.
10. Cholangitis.
11. Abscesses of liver.

AETIOLOGY AND PATHOGENESIS
To the causing factors take:

- cholelithiasis;
- virulent microorganisms (Table 1) which gets to the wall of gallbladder, bile duct and pancreas by a hematogenic, lymphatic ways and from duodenum;
- stagnation of bile which arises up as a result of mechanical (obstruction by a stone, narrowing of gallbladder cystic duct, innate defects) or functional disorders (spasm of sphincters, neuro-humoral dysfunction and other).

More frequent all an acute biliary problems arises up at combination of the following factors:

- violation of outflow of bile;
- presence of infection;
• damage of gallbladder, bile duct, and pancreatic duct wall (mechanical, chemical).
• sensibilisation of organism;

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<tr>
<th>Table 1 Common microorganisms isolated from bile cultures among patients with acute cholecystitis</th>
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<td>Isolated microorganisms from bile cultures</td>
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<tr>
<td>---------------------------------------------------------------</td>
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<tr>
<td>Gram-negative organisms</td>
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<tr>
<td><em>Escherichia coli</em></td>
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<td><em>Klebsiella spp.</em></td>
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<tr>
<td><em>Pseudomonas spp.</em></td>
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<td><em>Enterobacter spp.</em></td>
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<tr>
<td>Gram-positive organisms</td>
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<tr>
<td><em>Enterococcus spp.</em></td>
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<tr>
<td><em>Streptococcus spp.</em></td>
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<td><em>Staphylococcus spp.</em></td>
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<td>Anaerobes</td>
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In 90 to 95% of cases, acute cholecystitis and complications are related to gallstones. Obstruction of the bile duct or Vater’s papilla by a gallstone leads to biliary colic and is also the first event in many complications. If the bile duct remains obstructed, the biliary hypertension becomes, which is the key to understanding of other complications. Sometimes, the gallstone is dislodged, and the inflammation gradually resolves.

**Gallstone Pathogenesis.** Bile represents the route of excretion for certain organic solids, such as bilirubin and cholesterol, the major organic solutes in bile are bilirubin, bile salts, phospholipids, and cholesterol. Bilirubin is the breakdown product of red blood cells and is conjugated with glucuronic acid before being excreted. Bile salts solubilize lipids and facilitate their absorption. Phospholipids (lecithin) are synthesized in the liver in conjunction with bile salt synthesis. The final major solute of bile is cholesterol. The normal volume of bile secreted daily by the liver is 600 to 1200 ml.

Cholesterol is highly nonpolar and insoluble in water. The key to maintaining cholesterol in solution is the formation of both micelles, a bile salt-phospholipid-cholesterol complex, and cholesterol-phospholipid vesicles. Present theory suggests that in states of excess cholesterol production, these large vesicles may also exceed their capability to transport cholesterol, and crystal precipitation may occur. Cholesterol solubility depends on the relative concentration of cholesterol, bile salts, and phospholipids.

Gallstones represent a failure to maintain certain biliary solutes, primarily cholesterol and calcium salts, in a solubilized state. Gallstones are classified by their cholesterol content as either cholesterol or pigment stones. Pigment stones are further classified as either black or brown. Pure cholesterol gallstones are uncommon (10%), with most cholesterol stones containing calcium salts in their center. In most populations, 70 to 80% of gallstones are cholesterol, and black pigment stones account for most of the remaining 20 to 30%.

Black pigment stones are usually small, brittle, black, and sometimes spiculated. They are formed by supersaturation of calcium bilirubinate, carbonate, and phosphate, most often secondary to hemolytic disorders such as hereditary spherocytosis and sickle cell disease, and in those with cirrhosis. Like cholesterol stones, they almost always form in the gallbladder. Unconjugated bilirubin is much less soluble than conjugated bilirubin in bile. Deconjugation of bilirubin occurs normally in bile at a slow rate. Excessive levels of conjugated bilirubin, as in hemolytic states, lead to an increased rate of production of unconjugated bilirubin. Cirrhosis may lead to increased secretion of unconjugated bilirubin. When altered conditions lead to increased levels of deconjugated bilirubin in bile, precipitation with calcium occurs. In Asian countries such as Japan, black stones account for a much higher percentage of gallstones than in the Western hemisphere.
An important biliary precipitate in gallstone pathogenesis is biliary "sludge," which refers to a mixture of cholesterol crystals, calcium bilirubinate granules, and a mucin gel matrix. Biliary sludge has been observed clinically in prolonged fasting states or with the use of long-term total parenteral nutrition. Both of these conditions are also associated with gallstone formation. The finding of macromolecular complexes of mucin and bilirubin, similar to biliary sludge in the central core of most cholesterol gallstones, suggests that sludge may serve as the nidus for gallstone growth.

The risk factors predisposing to gallstone formation include obesity, diabetes mellitus, estrogen and pregnancy, hemolytic diseases, and cirrhosis.

At subsequent progress of passionately destructive changes in a mucus shell which loses the protective properties, a process spreads on other layers of wall of gallbladder. There is a considerable oedema of all of layers, their infiltration by different cells, microorganisms. The paretic broaden blood vessels, there are diffuse hemorrhages. Muscular layers loosed a capacity for contraction. In a serosa there is a desquamation of mesotelium, laying of fibrin. Such changes are characterized as a phlegmonous cholecystitis. Accumulation of pus inside the gallbladder is determined as an empyema of gallbladder. Total destruction of the gallbladder wall is specific for gangrenous cholecystitis. Thus to the gall-bladder large omentum, colon or its mesentery, wall of duodenum can be fixed. The conglomerate of tissues which are saturated with an exsudate – paravesical mass (infiltrate) appears in the total.

Perforation of the gallbladder occurs in up to 10% of cases of acute cholecystitis. Perforation is a sequela of ischemia and gangrene of the gallbladder wall and occurs most commonly in the gallbladder fundus. The perforation is most frequently (50% of cases) contained within the subhepatic space by the omentum, duodenum, liver, and hepatic flexure of the colon, and a localized abscess forms. Less commonly, the gallbladder perforates into an adjacent viscus (duodenum or colon), resulting in a cholecystoenteric fistula. Rarely, the gallbladder perforates freely into the peritoneal cavity, leading to generalized peritonitis.

**CHOLEDUCOLITHIASIS**

Common bile duct stones are classified both by their point of origin and by the time at which they are discovered relative to cholecystectomy. The vast majority of common bile duct calculi initially form in the gallbladder and migrate through the cystic duct into the common bile duct. These stones are identified as secondary calculi to distinguish them from primary common bile duct calculi, which form within the biliary tract. Common duct stones are also defined as retained, if they are discovered within 2 years of cholecystectomy, or recurrent, if they are detected more than 2 years after cholecystectomy. Retained stones were most likely present at the time of the cholecystectomy.

Primary common duct stones are associated with biliary stasis and infection. Primary stones are usually of the brown pigment type, which are soft and crumble easily when manipulated. The cause of the biliary stasis, which leads to the development of primary duct stones, may include a biliary stricture, papillary stenosis, or sphincter of Oddi dysfunction. In addition, biliary cultures are positive in most patients with common duct stones. The association of stasis with bacterial glucuronidases leads to the deconjugation of bilirubin diglucuronide and the precipitation of bilirubin as its calcium salt. The underlying abnormality leading to biliary stasis usually needs to be identified and corrected to prevent the formation of recurrent stones after removal.

Up to 10% of patients undergoing cholecystectomy have common bile duct stones. Many of these patients have symptoms or laboratory abnormalities consistent with biliary obstruction. However, biliary obstruction from stones is often transient, and preoperative laboratory test results may be normal. Approximately 1 to 2% of patients treated with laparoscopic cholecystectomy without a cholangiogram for gallstones present after the cholecystectomy with a retained stone. 7-18% patients with choledocholithiasis may be asymptomatic. In other patients, clinical features suspicious for biliary obstruction due to common bile duct stones include biliary colic, jaundice, lightening of the stools, and darkening of the urine. Nausea and vomiting are common. In addition, fever and chills may be present in a patients with choledocholithiasis and cholangitis. Physical examination may be normal, but mild epigastric or right upper quadrant tenderness as well as mild
Icterus is common. The symptoms may also be intermittent, such as pain and transient jaundice caused by a stone that temporarily impacts the ampulla but subsequently moves away, acting as a ball valve. A small stone may pass through the ampulla spontaneously with resolution of symptoms. Finally, the stones may become completely impacted, causing severe progressive jaundice. Serum bilirubin (>21 mcM/l), serum aminotransferases, and alkaline phosphatase are commonly elevated in patients with biliary obstruction but are neither sensitive nor specific for the presence of common duct stones. Of these, serum bilirubin has the highest positive predictive value (28 to 50%) for the presence of choledocholithiasis. Computer models utilizing clinical variables, such as serum bilirubin, alanine aminotransferase, gamma glutamyltransferase, and patient age, have a reported sensitivity and specificity of 94 and 88%, respectively, at predicting the presence of choledocholithiasis in patients undergoing cholecystectomy for gallstones. However, laboratory values may be normal in up to one third of patients with choledocholithiasis.

Transabdominal standard ultrasound examination can provide additional information supporting the diagnosis of common duct stones. A common bile duct diameter of 9 mm or greater indicates biliary obstruction, which may be due to a calculus. A small common bile duct diameter (<3 mm) also makes choledocholithiasis unlikely. However, echogenic shadows consistent with calculi are visible in only 60 to 70% of patients with common duct stones.

Although ultrasound is clearly the first test of choice for delineation of biliary pathology, computed tomography (CT) provides superior anatomic information. Because most gallstones are radiographically isodense to bile, many will be indistinguishable from bile. However, because ultrasound is operator-dependent and provides no anatomic reconstruction of the biliary tree, CT can be used to identify the cause and site of biliary obstruction. Drip infusion cholangiography with spiral CT (DIC-CT) has also been used to detect choledocholithiasis.

Since the mid-1990s, additional noninvasive and invasive modalities have become available to image the biliary tract. Magnetic resonance imaging (MRI) uses the water in bile to delineate the biliary tree and thus provides superior anatomic definition of the intrahepatic and extrahepatic biliary tree and pancreas. Magnetic resonance cholangiography (MRCP) provides excellent anatomic detail and has a sensitivity and a specificity of 95% and 89%, respectively, at detecting choledocholithiasis.

Endoscopic ultrasound (EUS) is of limited use in the evaluation of gallbladder pathology or intrahepatic disease of the biliary tree, but it is valuable in the assessment of distal common bile duct and ampulla. With the close apposition of the distal common bile duct and pancreas to the duodenum, sound waves generated by EUS provide detailed evaluation of the bile duct and ampulla and have proven most useful in assessing small stones in distal part of common bile duct. Although less sensitive than endoscopic cholangiography, this technique does not require cannulation of the ampulla. Therefore, endoscopic ultrasound can be performed in nearly all patients and avoids the risks of pancreatitis and cholangitis. Echoendoscopes are subdivided into those that scan perpendicular to the long axis of the endoscope, known as radial echoendoscopes, and those that scan parallel, known as linear echoendoscopes.

Endoscopic retrograde cholangiopancreatography (ERCP) is an invasive test using endoscopy and fluoroscopy to inject contrast through the ampulla and image the biliary tree. ERCP has been the gold standard for diagnosing common bile duct calculi before surgery. Cannulation of the common bile duct and successful cholangiography are achieved by experienced endoscopists in more than 90% of patients. Complications of diagnostic cholangiography include pancreatitis and cholangitis and occur in up to 5% of patients. Endoscopic cholangiography has the distinct advantage of providing a therapeutic option at the time common duct stones are identified. ERCP has also proven extremely useful in the diagnosis and treatment of complications of biliary surgery.

The percutaneous route is also available for access to the biliary tract and for treatment of obstructing jaundice. This approach is favored in patients with more proximal bile duct obstruction involving, or proximal to, the hepatic duct bifurcation. Percutaneously placed polyurethane stent can usually be passed across an obstructing biliary lesion into the duodenum to permit internal biliary drainage. Serial dilatation of the stent tract can also facilitate passage of a flexible choledochoscope into the biliary tree for direct visualization, biopsy, or management of stones or any obstructing lesions.
Another imaging tool for the diagnosis of biliary tract abnormalities is the use of intraoperative cholangiography. With the injection catheter inserted via the cystic duct during a cholecystectomy or through another point in the biliary tree, intraoperative cholangiography can help delineate anomalous biliary anatomy, identify choledocholithiasis, or guide biliary reconstruction. Some surgeons advocate routine cholangiography during cholecystectomy. Advocates for routine cholangiography note that common duct injuries are less frequent when cholangiography is used routinely. However, because it adds operative time and fluoroscopic exposure to the operation, many surgeons use intraoperative cholangiography selectively during the performance of a cholecystectomy. Indications for the selective use of cholangiography include pain at the time of operative intervention, abnormal hepatic function panel, anomalous or confusing biliary anatomy, or inability to perform ERCP following cholecystectomy, dilated biliary tree, or any preoperative suspicion of choledocholithiasis.

**OBSTRUCTIVE JAUNDICE**

Jaundice (the yellowish staining of the skin, sclera, and mucous membranes with the pigment bilirubin) is a frequent manifestation of choledocholithiasis and other biliary tract disorders, and the evaluation and management of the jaundiced patient is a common problem facing the general surgeon. Normal serum bilirubin ranges from 0.5 to 1.3 mg/dl, and when levels exceed 2.0-2.5 mg/dl, the bilirubin staining of the tissues becomes clinically apparent as jaundice. In addition, the presence of conjugated bilirubin in the urine is one of the first changes noted by patients.

**Diagnostic Evaluation**

The differential diagnosis of jaundice parallels the metabolism of bilirubin. Disorders resulting in jaundice can be divided into those causing "medical" jaundice, such as increased production of bilirubin, decreased hepatocyte transport or conjugation, or impaired excretion of bilirubin, or into those causing "surgical" jaundice through impaired delivery of bilirubin into the intestine.

The patterns of elevation of the different fractions of bilirubin provide important diagnostic clues as to the cause of cholestasis. In general, an elevated indirect bilirubin level suggests intrahepatic cholestasis and an elevated direct bilirubin level suggests extrahepatic obstruction.

Alkaline phosphatase is an enzyme with a wide tissue distribution but is found primarily in the liver and bones. In the liver, it is expressed by the bile duct epithelium. In conditions of biliary obstruction, levels rise as a result of increased synthesis and release into the serum.

GGTP is another enzyme found in hepatocytes and released from the bile duct epithelium. Elevation of GGTP is an early marker and also a sensitive test for hepatobiliary disease. Like AP elevation, however, it is nonspecific and can be produced by a variety of disorders in the absence of liver disease.

**Management**

A choice of rational method of treatment at choledocholithiasis and obstructive jaundice is important for the increase of efficiency of treatment of such patients.

*Conservative therapy* consists of complex measures which influence on the different links of disease.

For the improvement of outflow of bile spasmylic preparations are propose: No-Spanum 2.0, Papaverinum 2% - 2.0 (i/v, i/m), Platypthilinum 0.2% - 1.0, Atropinum 0.1% - 1.0 subcutaneously.

In case of pain non-narcotic analgesics should be widely used (Diclophenac, Keterolac, Ketoprofen), and also in combination with spasmylics (Baralginum, Spasganum, Spasmalgonum, Baralgitax). Narcotic analgetics appointing is not desirable as a result of their the spastic effect on sphincter of Oddi. However, at a necessity introduction, they are combined with spasmylics.

Antibacterial therapy is recommended. It is expedient to use antibiotics which must ability be concentrated in a bile: Cefoperazone, Ceftazidime, Doxycycline - concentrated in a bile even in the conditions of obstructive jaundice. If it necessary, the antibacterial preparations of other groups (Ciprofloxacyn, Metronidazole, and other) should be used.
With the purpose of support of general homoeostasis infusion therapy is applied. At presence of severe obstructive jaundice the volume of infusion must be increased with diuretic preparations (Furosemid) and hepatoprotectors (Essenciale, Lypoic acid, Thiotriazolin, Lipamid, and other).

**Endoscopic Management.** Endoscopic sphincterotomy and stone extraction was introduced more than 40 years ago (M. Classen and L. Demling in Germany and K.Y. Kawai in Japan) and permits common bile duct stones to be removed without the need for conventional surgery. The endoscopic approach is particularly useful for patients before cholecystectomy in whom a high suspicion exists for common bile duct calculi, particularly if laparoscopic common bile duct exploration is not available. Endoscopic clearance of stones from the common bile duct before cholecystectomy can avoid the need for an open operation. Furthermore, if endoscopic stone extraction is not possible (e.g., multiple gallstones, intrahepatic stones, large gallstones, impacted stones, duodenal diverticula, prior gastrectomy bile duct stricture), this information is known before cholecystectomy, and an open common bile duct exploration or drainage procedure can be performed.

Common bile duct calculi not suspected before surgery but detected on intraoperative cholangiography may also be suitable for endoscopic management if expertise using endoscopic techniques is available and the equipment and/or ability to perform laparoscopic common bile duct exploration is not. The cholecystectomy is completed laparoscopically, and the endoscopic procedure is scheduled for soon thereafter. Endoscopic sphincterotomy with stone extraction is also the procedure of choice for patients with retained common bile duct stones after cholecystectomy. The presence of jaundice, biliary-type pain, and biliary dilatation on imaging studies should raise the suspicion of either a ductal injury or retained stones.

Endoscopic sphincterotomy and stone extraction is well tolerated in most patients. Complications occur in 5 to 8% of patients and include cholangitis, pancreatitis, perforation, and bleeding. The overall mortality rate is 0.2 to 0.5%. Complete clearance of all common duct stones is achieved endoscopically in 71 to 75% of patients at the first procedure and in 84 to 93% of patients after multiple endoscopic procedures.

**Laparoscopic Common Bile Duct Exploration.** Laparoscopic exploration of the common bile duct for choledocholithiasis enables appropriate patients to undergo complete management of their calculous biliary tract disease with one invasive procedure. The laparoscopic approach is ideal for patients with common bile duct stones identified during intraoperative cholangiography or ultrasound or in patients with suspected choledocholithiasis treated at centers where laparoscopic common bile duct exploration is routinely performed. Intraoperative cholangiography is accomplished via the cystic duct before duct exploration. Once the presence of stones is confirmed, balloon-tipped Fogarty catheters are inserted through the cystic ductotomy into the duodenum and gently withdrawn with the balloon inflated. If this maneuver fails to remove the stone, a wire basket can be inserted under fluoroscopic guidance into the common bile duct to retrieve the stones. A small flexible choledochoscope is next introduced through one of the 5-mm cannulas and directed into the distal common bile duct. The scope can be used to push stones into the duodenum or to remove stones by use of the basket under direct vision. Occasionally, the cystic duct needs to be dilated to accept the choledochoscope or to remove larger stones. If the duct can be cleared via the cystic duct, a postoperative T tube is not necessary. If the cystic duct cannot be dilated, an anterior choledochotomy can be used. Postoperative biliary drainage using a T tube is then necessary.

Clearance of all common bile duct stones is achieved in 75 to 95% of patients with laparoscopic common bile duct exploration. The morbidity and mortality of laparoscopic common bile duct exploration are similar to those of laparoscopic cholecystectomy alone. In a prospective randomized trial comparing laparoscopic common bile duct exploration at the time of laparoscopic cholecystectomy with postoperative endoscopic stone extraction after laparoscopic cholecystectomy, the complication rate and retained stone rate were similar among the two groups. The median hospital stay was significantly shorter for patients managed with the single invasive procedure (1 versus 3.5 days).

**Open Common Bile Duct Exploration.** Open common bile duct exploration is performed much less frequently now than 15 years ago with the increased use of endoscopic, percutaneous, and laparoscopic techniques to remove common bile duct stones. Occasionally, when these methods fail,
are not available, or are not possible because of prior surgery, or when open operation is otherwise necessary, open common bile duct exploration becomes necessary. After the duodenum is mobilized, a longitudinal choledochotomy is made. Techniques including irrigation via soft rubber catheters, passing and retracting of balloon-tipped catheters, and use of Dormia stone baskets are all used to remove stones from the bile duct. The flexible and rigid choledoco-chroscope are both useful for identifying additional stones. At the completion of the exploration, a T tube is placed in the common bile duct via the choledochotomy, and the choledochotomy is closed around the T tube. A completion cholangiogram is performed to be certain that all the stones have been removed.

The standard technique for open common bile duct exploration may not be appropriate for all patients. The common bile duct may be explored through a large cystic duct, avoiding the need for a postoperative T tube. In patients with definite sphincter stenosis, multiple common bile duct stones, primary common bile duct stones, or intrahepatic stones, a drainage procedure (Roux-en-Y choledochojjunostomy, or choledochoduodenostomy) should be performed. Anastomosis to the duodenum can be performed rapidly with a single anastomosis. This anatomic arrangement continues to allow endoscopic access to the entire biliary tree. The downside of this approach is that the bile duct distal to the anastomosis does not drain well and may collect debris that obstructs the anastomosis or the pancreatic duct, a process known as sump syndrome. Anastomosis to the jejunum in a Roux-en-Y arrangement provides excellent drainage of the biliary tree without a risk of sump syndrome, but does not allow future endoscopic evaluation of the biliary tree.

Intrahepatic stones, which are almost uniformly brown pigment stones, represent a different management challenge than secondary bile duct stones. Relatively uncommon in Western compared with Asian populations, these stones tend to occur specifically in patients with stasis of the biliary tree, such as those with strictures, parasites, choledochal cysts, or sclerosing cholangitis. Because these stones collect at sites above obstructions, the transhepatic approach to cholangiography is generally more successful. Percutaneous drainage catheters are left in place and upsized to perform percutaneous stone extraction. Long-term management of intrahepatic stones must be carefully tailored to the disease but frequently requires hepaticojjunostomy for better biliary drainage. Liberal use of choledochoscopy at the time of drainage procedure ensures removal of all current stones. This approach allows a stone clearance rate of more than 90%.

Open common bile duct exploration is associated with low operative mortality (0 to 2%) and operative morbidity (8 to 16%). With the use of intraoperative choledochoscopy, the rate of retained common bile duct stones is less than 5%.

**CHOLANGITIS**

Cholangitis is one of the two main severe complications of CBD stones, the other being gallstone pancreatitis. Acute cholangitis is an ascending bacterial infection in association with partial or complete obstruction of the bile ducts. Prior to the 1970s the mortality rate of patients with acute cholangitis was reported to be over 50%, but advances in intensive care, new antibiotics, and biliary drainage dramatically reduced the mortality rate to less than 7% by the 1980s. However, even in the 2000s the reported mortality rates in severe cases still ranged from 11% to 27%, and even now the severe form of acute cholangitis remains a fatal disease unless appropriate management is instituted.

Hepatic bile is sterile, and bile in the bile ducts is kept sterile by continuous bile flow and by the presence of antibacterial substances in bile, such as immunoglobulin. Mechanical hindrance to bile flow facilitates bacterial contamination. Positive bile cultures are common in the presence of bile duct stones as well as with other causes of obstruction. Biliary bacterial contamination alone does not lead to clinical cholangitis. The onset of acute cholangitis involves two factors: (1) increased bacteria in the bile duct, and (2) elevated intraductal pressure in the bile duct allowing translocation of bacteria or endotoxin into the vascular and lymphatic system (cholangio-venous/lymphatic reflux). Because of its anatomical characteristics, the biliary system is likely to be affected by the elevated intraductal pressure. In acute cholangitis, bile ductules tend to become more permeable to the translocation of bacteria and toxins with the elevated intraductal biliary pressure. This process results in serious and fatal infections such as hepatic abscess and sepsis. Gallstones are the most common cause of
obstruction in cholangitis. The most common organisms cultured from bile in patients with cholangitis include \textit{E. coli}, \textit{Klebsiella pneumoniae}, \textit{Streptococcus faecalis}, \textit{Enterobacter}, and \textit{Bacteroides fragilis}.

\textbf{Clinical manifestations.} Cholangitis may present as anything from a mild, intermittent, and self-limited disease to a fulminant, potentially life-threatening septicemia. The patient with gallstone-induced cholangitis is typically older and female. The most common presentation is fever, epigastric or right upper quadrant pain, and jaundice. These classic symptoms, well known as Charcot's triad (1877), are present in about two thirds of patients. The illness may progress rapidly with septicemia and disorientation, known as Reynolds pentad (e.g., fever, jaundice, right upper quadrant pain, septic shock, and mental status changes) (Reynolds and Dragan, 1959). Fever and abdominal pain are the most frequently observed clinical manifestations in acute cholangitis, with an incidence of each of up to 80\% or more, whereas jaundice is observed in 60\%–70\% of cases. The incidence of Charcot’s triad is reported in not more than 72\% (range, 15.4\% to 72\%) of patients with acute cholangitis, and Reynolds’ pentad is extremely rare, reported in only 3.5\%–7.7\% of the patients. However, the presentation may be atypical, with little if any fever, jaundice, or pain. This occurs most commonly in the elderly, who may have unremarkable symptoms until they collapse with septicemia. Patients with indwelling stents rarely become jaundiced. On abdominal examination, the findings are indistinguishable from those of acute cholecystitis.

The clinical information used to establish the diagnosis of acute cholangitis includes a history of biliary disease, symptoms and signs, laboratory data, and imaging findings.

\textbf{Laboratory data.} Laboratory data indicative of inflammation (e.g., leukocytosis and an elevated C-reactive protein [CRP] level), and evidence of biliary stasis (e.g., hyperbilirubinemia, elevation of biliary enzymes and liver enzymes) are frequently seen in patients with acute cholangitis, and such laboratory findings support the diagnosis. Table 2 summarizes the positive rate for various blood tests in patients with acute cholangitis reported in the literature.

Table 2. Positive rates for blood tests in acute cholangitis

<table>
<thead>
<tr>
<th>Item</th>
<th>Positive rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC &gt;10 000/mm$^3$</td>
<td>63–82</td>
</tr>
<tr>
<td>Total bilirubin ↑</td>
<td>78–91</td>
</tr>
<tr>
<td>ALP ↑</td>
<td>74–93</td>
</tr>
<tr>
<td>AST ↑</td>
<td>93</td>
</tr>
<tr>
<td>ALT ↑</td>
<td>97</td>
</tr>
<tr>
<td>AST or ALT ↑</td>
<td>57</td>
</tr>
<tr>
<td>Prolonged prothrombin time</td>
<td>26</td>
</tr>
<tr>
<td>Amylase ↑</td>
<td>7–35</td>
</tr>
<tr>
<td>Creatinine ≥1.5 mg/d</td>
<td>16</td>
</tr>
<tr>
<td>CA19-9 ↑</td>
<td>28–100</td>
</tr>
<tr>
<td>Endotoxin ↑</td>
<td>36</td>
</tr>
</tbody>
</table>

WBC, white blood cells; ALP, alkaline phosphatase; AST, aspartate aminotransferase; ALT, alanine aminotransferase; CA19–9, carbohydrate antigen 19–9

\textbf{Imaging findings.} Abdominal ultrasound (US) and abdominal computerized tomography (CT) with intravenous contrast are very helpful studies in evaluating patients with acute biliary tract disease. US should be performed in all patients suspected of having acute biliary inflammation/infection. It is usually impossible to identify evidence of bile infection itself by imaging modalities. The role of diagnostic imaging in acute cholangitis is to determine the presence/absence of biliary obstruction, the level of the obstruction, and the cause of the obstruction, such as gallstones and/or biliary strictures. Assessment should include both US and CT. These studies complement each other and CT may better demonstrate dilatation of the bile duct and pneumobilia. Imaging evidence of biliary dilatation (evidence of biliary obstruction) and/or the etiology of the underlying disease (tumor, gallstones, stent-related, etc.) can support the clinical diagnosis of cholangitis.
Diagnostic criteria for acute cholangitis. Table 3 shows the diagnostic criteria for acute cholangitis that were finally adopted by the Organizing Committee of TG-13. The basic concepts of the criteria are as follows: (1) Charcot’s triad is a definite diagnostic criterion for acute cholangitis, (2) if a patient does not have all the components of Charcot’s triad (acute cholangitis is suspected), then definite diagnosis can be achieved if both an “inflammatory response” and “biliary obstruction” are demonstrated by the laboratory data (blood tests) and imaging findings.

Table 3 Diagnostic criteria for acute cholangitis

<table>
<thead>
<tr>
<th>A. Clinical context and clinical manifestations</th>
<th>1. History of biliary disease</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2. Fever and/or chills</td>
</tr>
<tr>
<td></td>
<td>3. Jaundice</td>
</tr>
<tr>
<td></td>
<td>4. Abdominal pain (RUQ or upper abdominal)</td>
</tr>
<tr>
<td>B. Laboratory data</td>
<td>5. Evidence of inflammatory response(^a)</td>
</tr>
<tr>
<td></td>
<td>6. Abnormal liver function tests(^b)</td>
</tr>
<tr>
<td>C. Imaging findings</td>
<td>7. Biliary dilatation, or evidence of an etiology (stricture, stone, stent etc)</td>
</tr>
<tr>
<td>Suspected diagnosis</td>
<td>Two or more items in A</td>
</tr>
<tr>
<td>Definite diagnosis</td>
<td>(1) Charcot’s triad (2 + 3 + 4)</td>
</tr>
<tr>
<td></td>
<td>(2) Two or more items in A + both items in B and item C</td>
</tr>
</tbody>
</table>

\(^a\) Abnormal WBC count, increase of serum CRP level, and other changes indicating inflammation

\(^b\) Increased serum ALP, r-GTP (GGT), AST, and ALT levels

Severity assessment of acute cholangitis. Patients with acute cholangitis may present with anything from a mild, self-limited illness to a severe, potentially life-threatening illness. Most cases respond to initial medical treatment consisting of general supportive therapy and intravenous antibiotics, but some cases do not respond to medical treatment, and the clinical manifestations and laboratory data do not improve. Such cases may progress to sepsis, with or without organ dysfunction, requiring appropriate management that includes intensive care, organ-supportive care, and urgent biliary drainage, in addition to medical treatment.

Severity assessment criteria. Table 4 summarizes the risk factors reported in the literature for poor outcome in patients with acute cholangitis. Organ dysfunction is the most common predictor of a poor outcome. On the other hand, based on the pathophysiology, “severe” acute cholangitis can also be defined as that which accompanies organ dysfunction caused by sepsis. Thus, “the onset of organ dysfunction” is an important factor in the definition of severe (grade III) acute cholangitis.

Table 4 Prognostic factors in acute cholangitis

<table>
<thead>
<tr>
<th>Prognostic factor</th>
<th>Positive value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Related to organ dysfunction</td>
<td></td>
</tr>
<tr>
<td>Shock</td>
<td></td>
</tr>
<tr>
<td>Mental confusion</td>
<td></td>
</tr>
<tr>
<td>Elevated serum creatinine</td>
<td>&gt;1.5–&gt;2.0 mg/dl</td>
</tr>
<tr>
<td>Elevated BUN</td>
<td>&gt;20–&gt;64 mg/dl</td>
</tr>
<tr>
<td>Prolonged prothrombin time</td>
<td>&gt;1.5–&gt;2.0 s</td>
</tr>
<tr>
<td>Hyperbilirubinemia</td>
<td>&gt;2.2–&gt;10 mg/dl</td>
</tr>
<tr>
<td>Reduced platelet count</td>
<td>&lt;10 × 10⁴–&lt;15 × 10⁴/mm³</td>
</tr>
<tr>
<td>Unrelated to organ dysfunction</td>
<td></td>
</tr>
<tr>
<td>High fever</td>
<td>&gt;39°C–&gt;40°C</td>
</tr>
<tr>
<td>Leukocytosis</td>
<td>&gt;20 000 /mm³</td>
</tr>
</tbody>
</table>
Another factor for the severity assessment of acute cholangitis is “response to initial medical treatment”; treatment consisting of general supportive care and antibiotics should be instituted as soon as possible for all patients who are diagnosed with acute cholangitis. Patients diagnosed with acute cholangitis that is not complicated by organ dysfunction, who did not respond to medical treatment and who continue to have SIRS and/or sepsis require additional treatment that includes either a change of antibiotic or biliary drainage. The severity of such cases is classified as moderate (grade II). Patients who respond to medical treatment and whose clinical manifestations and laboratory data improve are classified as having mild (grade I) disease. Table 5 and Table 6 show the concepts and criteria for the severity assessment of acute cholangitis.

Table 5  Criteria for severity assessment of acute cholangitis

<table>
<thead>
<tr>
<th>Severity of acute cholangitis</th>
<th>Mild (grade I)</th>
<th>Moderate (grade II)</th>
<th>Severe (grade III)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of organ dysfunction</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Response to initial medical treatment&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Consisting of general supportive care and antibiotics

Table 6  Definitions of severity assessment criteria for acute cholangitis

Mild (grade I) acute cholangitis

“Mild (grade I)” acute cholangitis is defined as acute cholangitis which responds to the initial medical treatment<sup>a</sup> Moderate (grade II) acute cholangitis “Moderate (grade II)” acute cholangitis is defined as acute cholangitis that does not respond to the initial medical treatment<sup>a</sup>

Moderate (grade II) acute cholangitis

“Moderate (grade II)” acute cholangitis is defined as acute cholangitis that does not respond to the initial medical treatment and is not accompanied by organ dysfunction

Severe (grade III) acute cholangitis

“Severe (grade III)” acute cholangitis is defined as acute cholangitis that is associated with the onset of dysfunction at least in any one of the following organs/systems:

1. Cardiovascular system Hypotension requiring dopamine ≥5 μg/kg per min, or any dose of dobutamine
2. Nervous system Disturbance of consciousness
3. Respiratory system PaO2/FiO2 ratio < 300
4. Kidney Serum creatinine > 2.0 mg/dl
5. Liver PT-INR > 1.5
6. Hematological system Platelet count < 100 000 /μl

Note: compromised patients, e.g., elderly (>75 years old) and patients with medical comorbidities, should be monitored closely

<sup>a</sup> General supportive care and antibiotics
**Differential diagnosis.**
Diseases which should be differentiated from acute cholangitis are acute cholecystitis, gastric and duodenal ulcer, acute nonbiliary pancreatitis, acute hepatitis, hepatic abscess and septicemia of other origins, AIDS, fever.

**Management**
A choice of rational method of treatment at an acute cholangitis is important for the increase of efficiency of treatment of such patients. Most widespread is active tactic. It foresees hospitalization of all patients with an acute cholangitis in surgical department, urgent examination and lead through at first hour all of patient of conservative measures which have also on a purpose preparation to the possible operation. Flowcharts for the management of acute cholangitis according the Tokyo Consensus Meeting (2007) and Guideline (2013) is presented at the Fig.1.

![](image)

**Fig. 1.** Flowcharts for the management of acute cholangitis.

The treatment of acute cholangitis should be guided by the grade of severity of the disease. Biliary drainage and antibiotics are the two most important elements of treatment. When a diagnosis of acute cholangitis is suspected, medical treatment, including nil per os (NPO) and the use of intravenous fluids, antibiotics, diuretics, hepatoprotectors, and analgesia, together with close monitoring of blood pressure, pulse, and urinary output should be initiated. Antibacterial therapy is recommended (Table 7). It is expedient to use antibiotics which must ability be concentrated in bile: Cefoperazone, Ceftazidime, Doxycycline - concentrated in a bile even in the conditions of obstructive jaundice. If it necessary, the antibacterial preparations of other groups (Ciprofloxacin, Metronidazole, and other) should be used. Simultaneously, a severity assessment of the cholangitis should be documented, even if it is mild.

Identifying the causative organism(s) is an essential step in the management of acute biliary infections. Positive rates of bile cultures range from 59 to 93 % for acute. Common duct bile should be sent for culture in all cases of suspected cholangitis.

On the other hand, positive rates of blood cultures among patients with acute cholangitis ranged from 21 to 71 %. Most of the bacteremic isolates are organisms that do not form vegetations on normal cardiac valves nor miliary abscesses. Their intravascular presence does not lead to an extension of therapy or selection of multidrug regimens. Therefore, there are recommendation to take such cultures only in high-severity infections when such results might mandate changes in therapy.
Before dosing antimicrobial agents, renal function should be estimated with the commonly used equation: Serum creatinine = [(140 – age) [optimum body weight (kg)] / 72 x serum creatinine (mg/dl). Regarding the timing of therapy, it should be initiated as soon as the diagnosis of biliary infection is suspected. For patients in septic shock, antimicrobials should be administered within 1 h of recognition. For other patients, as long as 4 h may be spent obtaining definitive diagnostic studies prior to beginning antimicrobial therapy. Antimicrobial therapy should definitely be started before any procedure, either percutaneous, endoscopic, or operative, is performed.

Frequent reassessment is important, and patients may need to be reclassified as having mild (grade I), moderate (grade II), or severe (grade III) disease, based on the response to medical treatment. Appropriate treatment should be performed in accordance with the severity grade. Patients with concomitant diseases such as acute pancreatitis or malignant tumor, and elderly patients are likely to progress to a severe level; therefore, such patients should be monitored frequently.

Mild (grade I) acute cholangitis

Medical treatment may be sufficient. Biliary drainage is not required in most cases. However, for nonresponders to medical treatment, the necessity of biliary drainage should be considered. Treatment options such as endoscopic, percutaneous, or operative intervention may be required, depending on the etiology. Some patients, such as those who develop postoperative cholangitis, may only require antibiotics and generally do not require intervention.

Moderate (grade II) acute cholangitis

Patients with acute cholangitis who do not respond to medical treatment have moderate (grade II) acute cholangitis. In these patients, early endoscopic or percutaneous drainage or even emergent operative drainage with a T-tube should be performed. A definitive procedure should be performed to remove the cause of the obstruction once the patient is in a stable condition.

Severe (grade III) acute cholangitis

Patients with acute cholangitis and organ failure are classified as having severe (grade III) acute cholangitis. These patients require organ support, such as ventilatory/circulatory management (e.g., endotracheal intubation, artificial respiration management, and the use of vasopressin), and treatment for disseminated intravascular coagulation (DIC) in addition to the general medical management. Urgent biliary drainage must be anticipated. When the patient is stabilized, urgent (ASAP) endoscopic or percutaneous transhepatic biliary drainage or an emergent operation with decompression of the bile duct with a T-tube should be performed. Definitive treatment of the cause of the obstruction, including endoscopic, percutaneous, or operative intervention, should be considered once the acute illness has resolved.

PYOGENIC LIVER ABSCESS

Pyogenic liver abscesses are the most common liver abscesses. Previously they were felt to be due to portal infection, often occurring in young patients secondary to acute appendicitis. However, with earlier diagnosis this cause of abscesses has decreased. Pyogenic liver abscesses occur as a result of impaired biliary drainage and biliary hypertension. Pyogenic hepatic abscesses may be single or multiple and are more frequently found in the right lobe of the liver. The abscess cavities are variable in size and, when multiple, may coalesce to give a honeycomb appearance. Approximately 40% of abscesses are monomicrobial, an additional 40% are polymicrobial, and 20% are culture negative. The most common infecting agents are gram-negative organisms. *Escherichia coli* is found in two thirds, and *Streptococcus faecalis*, *Klebsiella*, and *Proteus vulgaris* are also common. Anaerobic organisms such as *Bacteroides fragilis* are also seen frequently.

Patients usually are symptomatic with right upper quadrant pain and fever. Jaundice occurs in up to one third of affected patients. A thorough history and physical examination are necessary to attempt to localize the primary causative site. Leucocytosis, an elevated sedimentation rate, and an elevated ALT, AST, AP, CRP levels are the most common laboratory findings. Significant abnormalities in the results of the remaining liver function tests are unusual. Blood cultures reveal the causative organism in approximately 50% of cases.
<table>
<thead>
<tr>
<th>Severity</th>
<th>Community-acquired biliary infections</th>
<th>Healthcare-associated biliary infections</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Grade I</td>
<td>Grade II</td>
</tr>
<tr>
<td></td>
<td>Cholangitis</td>
<td>Cholangitis and cholecystitis</td>
</tr>
<tr>
<td></td>
<td>Cholecystitis</td>
<td></td>
</tr>
<tr>
<td>Antimicrobial agents</td>
<td>Penicillin-based therapy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ampicillin/sulbactam&lt;sup&gt;b&lt;/sup&gt; is not recommended without an aminoglycoside</td>
<td>Pipingillin/tazobactam</td>
</tr>
<tr>
<td></td>
<td>Cefazolin&lt;sup&gt;c&lt;/sup&gt;, or cefotiam&lt;sup&gt;c&lt;/sup&gt;, or cefuroxime&lt;sup&gt;c&lt;/sup&gt;, or ceftriaxone, or cefotaxime ± metronidazole&lt;sup&gt;d&lt;/sup&gt;</td>
<td>Ceftriaxone, or cefotaxime, or cefepime, or ceftazidime, or ceftazidime ± metronidazole&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Cefmetazole&lt;sup&gt;a&lt;/sup&gt;, Cefoxitin&lt;sup&gt;a&lt;/sup&gt;, Flomoxef&lt;sup&gt;a&lt;/sup&gt;, Cefoperazone/sulbactam</td>
<td>Cefoperazone/sulbactam</td>
</tr>
<tr>
<td></td>
<td>Ertapenem</td>
<td>Ertapenem</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Ciprofloxacin, or levofloxacin, or p pazufloxacin ± metronidazole&lt;sup&gt;d&lt;/sup&gt;</td>
<td>Ciprofloxacin, or levofloxacin, or p pazufloxacin ± metronidazole&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Moxifloxacin</td>
<td>Moxifloxacin</td>
</tr>
</tbody>
</table>

<sup>a</sup> Local antimicrobial susceptibility patterns (antibiogram) should be considered for use
<sup>b</sup> Ampicillin/sulbactam has little activity left against *Escherichia coli*. It is removed from the North American guidelines
<sup>c</sup> Fluoroquinolone use is recommended if the susceptibility of cultured isolates is known or for patients with β-lactam allergies. Many extended-spectrum β-lactamase (ESBL)-producing Gram-negative isolates are fluoroquinolone-resistant
<sup>d</sup> Anti-anaerobic therapy, including use of metronidazole, tinidazole, or clindamycin, is warranted if a biliary-enteric anastomosis is present. The carbapenems, piperacillin/tazobactam, ampicillin/sulbactam, cefmetazole, cefoxitin, flomoxef, and cefoperazone/sulbactam have sufficient anti-anaerobic activity for this situation
<sup>e</sup> Vancomycin is recommended to cover *Enterococcus* spp. for grade III community-acquired acute cholangitis and cholecystitis, and healthcare-associated acute biliary infections. Linezolid or daptomycin is recommended if vancomycin-resistant *Enterococcus* (VRE) is known to be colonizing the patient, if previous treatment included vancomycin, and/or if the organism is common in the community
Ultrasound examination of the liver reveals pyogenic abscesses as round or oval hypoechoic lesions with well-defined borders and a variable number of internal echoes. CT scan is highly sensitive in the localization of pyogenic liver abscesses. The abscesses are hypodense and may contain air-fluid levels indicating a gas-producing infectious organism as well as peripheral enhancement. MRI of the abdomen also can detect pyogenic abscesses with a high level of sensitivity but plays a limited role because of its inability to be used for image-guided diagnosis and therapy.

The current cornerstones of treatment include correction of the underlying cause (biliary drainage), needle aspiration, and IV antibiotic therapy. On presentation, percutaneous aspiration and culture of the aspirate may be beneficial to guide subsequent antibiotic therapy. Initial antibiotic therapy needs to cover gram-negative as well as anaerobic organisms. Aspiration and placement of a drainage catheter is beneficial for only a minority of pyogenic abscesses, because most are quite viscous and drainage is ineffective. Antibiotic therapy must be continued for at least 8 weeks. Aspiration and IV antibiotic therapy can be expected to be effective in 80 to 90% of patients. If this initial mode of therapy fails, the patients should undergo surgical therapy, including laparoscopic or open drainage. Anatomic surgical resection can be performed in patients with recalcitrant abscesses.

BILIARY PANCREATITIS

Acute pancreatitis is an inflammatory disease of the pancreas that is associated with little or no fibrosis of the gland. Although acute pancreatitis is documented in association with acalculous biliary tract disease, bile duct stones (choledocholithiasis) represent the most common form of associated biliary abnormality. The mechanism by which a gallstone may cause pancreatitis is not entirely clear, although gallstones have been implicated ever since E.Opie made the seminal observation in 1901 of two patients who died of acute pancreatitis with stones impacted in their ampulla of Vater. This led him to propose the "common-channel hypothesis," in which a blockage below the junction of the biliary and pancreatic ducts would cause bile to flow into the pancreas, which could then be damaged by the detergent action of bile salts.

Important objections to this theory include the anatomic reality that the majority of individuals have such a short common channel that a stone located there would block both the pancreatic and biliary ducts, effectively isolating the two systems. Furthermore, hydrostatic pressure in the biliary tract is lower than in the pancreas, a condition that would favour abnormal flow of pancreatic juice into the bile duct rather than in the opposite direction. These reservations are bolstered by the observation that, in experimental animals, the flow of normal bile through an unobstructed pancreatic duct does not result in acute pancreatitis (although forceful retrograde injection of bile into the pancreas causes injury similar to that of acute pancreatitis).

Another proposed mechanism of causation postulates that passage of a gallstone through the sphincter of Oddi renders it momentarily incompetent permitting the reflux of duodenal juice containing activated digestive enzymes into the pancreatic ductal system. However, it is questionable whether the transit time through the sphincter of Oddi is long enough to cause sufficient incompetence. Finally, the observation remains that procedures designed to render the sphincter incompetent, such as sphincterotomy, do not routinely cause pancreatitis.

Therefore, although it is reasonable to dismiss an incompetent sphincter of Oddi as an etiologic factor in acute pancreatitis, it is not as simple to dismiss the role of gallstones. A clinical study showed that 88% of patients with acute pancreatitis passed gallstones in their feces within 10 days the attack. This is in contrast to only 11% of gallstone patients who did not have pancreatitis, suggesting that the process of passing a gallstone may be linked to the development of acute pancreatitis.

This information justifies searching for a more likely causative factor than abnormal bile or duodenal juice backflow into the pancreas. A common phenomenon shared by gallstone disease and other conditions causing acute pancreatitis, such as helminthic infestation of the pancreatic duct or its blockage by tumours, is ductal hypertension resulting from ongoing exocrine secretion into an obstructed pancreatic duct. A simple mechanical explanation has been proposed whereby elevated intraductal pressure causes rupture of the smaller ductules and leakage of pancreatic juice into the
parenchyma. Although the pancreatic duct fluid pH is maintained in the range of 8 to 9 by the secretion of bicarbonate, the interstitial pH of 7 within the pancreatic tissue favours activation of proteases when transductal extravasation of fluid occurs. Although pancreatic ductal obstruction and hypertension are likely initiating factors in the etiology of acute pancreatitis, the mechanism by which ductal hypertension initiates pancreatic injury remains under investigation. Although the mechanical factors that initiate the process are unclear, the colocalization theory of Steer and Saluja has gained acceptance as the cellular mechanism of acute pancreatitis. In the normal pancreas, the inactive digestive zymogens and the lysosomal hydrolases are found separately in discrete organelles. However, in response to ductal obstruction, hypersecretion, or a cellular insult, these two classes of substances become improperly colocalized in a vacuolar structure within the pancreatic acinar cell. A cascade has been postulated in which trypsinogen colocalizes with cathepsin B to produce activated trypsin, which, in turn, activates the other digestive zymogens. These active digestive enzymes then begin autodigestion within the pancreatic acinar cells, leading to pancreatitis.

**Factors determining the severity of acute pancreatitis.** Clinically, the severity of acute pancreatitis varies significantly. Some patients experience a mild form of the disease that is self-limiting, while others suffer a more severe and sometimes lethal attack. The factors determining the severity of pancreatitis are multifactorial, but their identification is of considerable therapeutic importance, because their manipulation may decrease the morbidity and mortality associated with the disease. It generally is believed that pancreatitis begins with the intrapancreatic activation of digestive enzyme zymogens, acinar cell injury, and activation of transcription factors such as nuclear factor- B (NF- B) and activator protein 1. This, in turn, leads to the production of proinflammatory factors, acinar cell necrosis, systemic inflammatory response syndrome, and distant organ dysfunction, including lung injury that frequently manifests as the acute respiratory distress syndrome (ARDS). The ultimate severity of acute pancreatitis depends on the extent of the systemic inflammatory response, as well as several cytokines and chemokines and their receptors that play a critical role in the activation and migration of inflammatory cells to the affected site.

In addition to the cells of the immune system such as the neutrophils, the pancreatic acinar cells are also a source of inflammatory mediators during pancreatitis. Over the past few years, the list of factors associated with pancreatitis and associated lung injury has grown rapidly to include tumor necrosis factor alpha, monocyte chemotactic protein-1, Mob1, interleukin-1 (IL-1 ), platelet activating factor (PAF), substance P, adhesion molecules [intercellular adhesion molecule-1 (ICAM-1) and selectins], IL-6, IL-8, IL-10, C5a, the CCR1 receptor and its ligands, granulocyte-macrophage colony-stimulating factor, macrophage migration inhibitory factor, COX-2, prostaglandin E1, nitric oxide, and reactive oxygen species. In addition, several studies have focused on the protective role played by heat shock proteins in pancreatitis. The ultimate severity of pancreatitis and associated lung injury depends on the balance between the pro- and anti-inflammatory factors. Several therapeutic regimens aimed at reducing the inflammatory response have been tested and include anti–tumor necrosis factor alpha antibody, IL-1 receptor antagonist, anti–ICAM-1 and anti-CD3 antibodies, IL-10, recombinant PAF acetylhydrolase, and the calcineurin antagonist FK506. Recent studies also indicate that Toll-like receptor 4 (TLR4) is significant in determining the severity of acute pancreatitis. The TLR4 initiates a complex signaling pathway when it interacts with lipopolysaccharides that result in a proinflammatory response. Mice in which TLR4 is genetically deleted have significantly reduced pancreatitis; this suggests that TLR4 is a significant promoter of proinflammation. However, this effect appears independently of lipopolysaccharides and is probably mediated by a hitherto unknown TLR4 agonist. It is likely that TLR4 antagonists would be a good therapy against pancreatitis.

**Diagnosis**

The clinical diagnosis of pancreatitis is one of exclusion. The other upper abdominal conditions that can be confused with acute pancreatitis include perforated peptic ulcer, a gangrenous small bowel obstruction, acute appendicitis, and acute cholecystitis. Because these conditions often have a fatal outcome without surgery, urgent intervention is indicated in the small number of cases in which doubt persists.
All episodes of acute pancreatitis begin with severe pain, generally following a substantial meal. The pain is usually epigastric, but can occur anywhere in the abdomen or right lower chest. It has been described as "knifing" or "boring through" to the back, and may be relieved by the patient leaning forward. It precedes the onset of nausea and vomiting, with retching often continuing after the stomach has emptied. Vomiting does not relieve the pain, which is more intense in necrotizing than in edematous pancreatitis. In case of acute gallbladder inflammation, patient feels acute pain in RUA.

On examination, the patient may show tachycardia, tachypnea, hypotension, and hyperthermia. The temperature is usually only mildly elevated in uncomplicated pancreatitis. Voluntary and involuntary guarding can be seen over the epigastric region. The bowel sounds are decreased or absent. Enlarged painful gallbladder can be palpable. The abdomen may be distended with intraperitoneal fluid. There may be pleural effusion, particularly on the left side.

**Serum markers.** The levels of pancreatic enzymes are elevated in the serum of most pancreatitis patients. Because of the ease of measurement, serum amylase levels are measured most often. It remains elevated for 3 to 5 days before returning to normal. There is no significant correlation between the magnitude of serum amylase elevation and severity of pancreatitis. Because hyperamylasemia can be observed in many extrapancreatic diseases, measuring pancreatic-specific amylase (p-amylase) rather than total amylase, which also includes salivary amylase, makes the diagnosis more specific (88 to 93%).

Other pancreatic enzymes also have been evaluated to improve the diagnostic accuracy of serum measurements. Specificity of these markers ranges from 77 to 96%, the highest being for lipase. Measurements of many digestive enzymes also have methodologic limitations and cannot be easily adapted for quantitation in emergency labs. Because serum levels of lipase remain elevated for a longer time than total or p-amylase, it is the serum indicator of highest probability of the disease.

**Ultrasound.** US examination is the best way to confirm the presence of gallstones in suspected biliary pancreatitis. It also can detect extrapancreatic ductal dilations and reveal pancreatic edema, swelling, and peripancreatic fluid collections (PFCs). However, in about 20% of patients, the US examination does not provide satisfactory results because of the presence of bowel gas, which may obscure sonographic imaging of the pancreas.

**Computed tomography.** A CT scan of the pancreas is more commonly used to diagnose pancreatitis. CT scanning is used to distinguish milder (nonnecrotic) forms of the disease from more severe necrotizing or infected pancreatitis in patients whose clinical presentation raises the suspicion of advanced disease. CT scanning with bolus IV contrast has become the gold standard for detecting and assessing the severity of pancreatitis. Although clinically mild pancreatitis is usually associated with interstitial edema, severe pancreatitis is associated with necrosis. In interstitial pancreatitis, the microcirculation of the pancreas remains intact, and the gland shows uniform enhancement on IV contrast-enhanced CT scan. In necrotizing pancreatitis, however, the microcirculation is disrupted; therefore, the enhancement of the gland on contrast-enhanced CT scan is considerably decreased. The presence of air bubbles on a CT scan is an indication of infected necrosis or pancreatic abscess. Currently, IV (bolus) contrast-enhanced CT scanning is routinely performed on patients who are suspected of harboring severe pancreatitis, regardless of their Ranson's or APACHE scores.

**Treatment of Biliary Pancreatitis.** Most patients pass the offending gallstone(s) during the early hours of acute pancreatitis, but have additional stones capable of inducing future episodes. This raises the question of the timing of surgical or endoscopic clearance of gallstones. The issue of when to intervene is controversial. Several studies have been aimed at resolving this controversy, but the issue is clouded by the fact that each position is open to some theoretical objection. Additional points of contention include varying inclusion criteria, years of observation of the studied groups, and a lack of uniformity regarding definitions. General consensus favors either urgent intervention (cholecystectomy) within the first 48 to 72 hours of admission, or briefly delayed intervention (after 72 hours, but during the initial hospitalization) to give an inflamed pancreas time to recover. Cholecystectomy and operative common duct clearance is probably the best treatment for otherwise healthy patients with obstructive pancreatitis. However, patients who are at high risk for surgical intervention are best treated by endoscopic sphincterotomy, with clearance of stones by ERCP.
In the case of acute biliary pancreatitis in which chemical studies suggest that the obstruction persists after 24 hours of observation, emergency endoscopic sphincterotomy and stone extraction is indicated. Routine ERCP for examination of the bile duct is discouraged in cases of biliary pancreatitis, as the probability of finding residual stones is low, and the risk of ERCP-induced pancreatitis is significant. Patients who are suspected of harboring a persistent impacted stone in the distal common bile duct or ampulla should have confirmation by radiologic imaging (CT, magnetic resonance cholangiopancreatography, or endoscopic ultrasonography) before intervention.

**Questions (α =І, α =ІІ):**
1. Anatomy and physiology of gallbladder and extrahepatic bile ducts.
2. Aetiology and pathogenesis of acute cholelithiasis.
3. Methods of examination of the patients with biliary pathology.
5. Surgical treatment of choledocholithiasis.
6. Minimally invasive treatment in choledocholithiasis.
7. Clinical picture of acute cholangitis in young and elderly patients.
8. Differential diagnostics of acute cholangitis.
9. Medical program of acute cholangitis.
15. Intraoperative complications at biliary surgery and their treatments.
17. Postoperative treatment of patients, operated concerning complicated acute cholecystitis.

**Practical task (α =І, α =ІІІ):**
1) To collect anamnesis, conduct palpation, percussion, auscultation of patient with a complicated acute cholecystitis;
2) To choose the most often signs of acute cholecystitis from information of anamnesis;
3) To discover and estimate protective tenderness of muscles in the right subcostal area, an acute gallbladder or mass, presence of fluid collections in abdominal cavity;
4) To demonstrate the presence of symptoms and their degree;
5) To conduct differential diagnostics of acute cholangitis, acute biliary pancreatitis;
6) To determine a indications for operations in acute complicated cholecystitis;
7) To ground and formulate the previous diagnosis of basic disease and complications;
8) To use deontology principles.

**Typical tasks (α =ІІ):**
1. Male, 50 y.o., complains of temperature to 37,8°C, icteric colouring of skin, which it noticed two days ago. During two years marks the origin of pain in the Rt subcostal area with an irradiation in the back at the reception of spicy or fried food. Five days ago after the use of such meal there was similar pain. At objective examination: skin and mucous are the yellow colouring, a tongue is moist, with a brown raid, by auscultation in lungs - vesicular breathing; pulse -90 b/min. BP-140/90 mm Hg. Abdomen is soft, moderate painfulness in the right subcostal area, signs of peritoneum irritation are absent. Laboratory tests - CBC: Hb - 122 g/l, leucocytes - 12.2 x10⁹/l, bilirubin - 110 mcmol/l, 75mcmol/l is direct. Most reliable preliminary diagnosis?
A. Cholecystitis, complicated with obstructive jaundice.
B. Acute pancreatitis.
C. Acute hepatitis.
2. Female, 46 y.o., complains of colicky pains in right subcostal area, the yellow colouring of skin, darkening of urine, light excrement, itch of skin appeared on height of attack two days ago. At palpation pain is expressed in right subcostal area, enlarged painful gallbladder. Preliminary diagnosis?
A. Cholecystitis, complicated with obstructive jaundice.
B. Pancreatic head cancer.
C. Leptospirosis.
D. Acute hepatitis.
E. Acute attack of chronic hepatitis.

3. Male, 50 y.o., complains with dull pain in right subcostal area, nausea, vomiting, sudden increase of temperature – 38.0°C. He is ill for 2 days. In anamnesis – choledocholithiasis. Skin is moderately icteric. A gall-bladder is enlarged, painless. An abdomen is moderate tense in right subcostal area. Signs of irritation peritoneum are absent. Lower margin of liver - +2 cm from costal arc, painful. Preliminary diagnosis?
A. Purulent cholangitis
B. Haemolytic jaundice
C. Parenchimatous icterus
D. Karoli Syndrome
E. Acute cholecystitis, complicated with obstructive jaundice.

4. Female, 48 y.o., suffering from cholelithiasis for 10 years. The last attack 7 day ago has begun. The sclera and skin turned yellow; itch. Stool is acholic. Temperature is 37.6°C. Laboratory tests: Hb – 107 g/l, leucocytes – 11.0x10⁹/l, banding – 8%, ESR – 16mm/h; total bilirubin – 320 µmol/l, protein – 75 g/l, glucose – 4.7 mmol/l, AsAT – 0.3, AIAT – 0.95, alkaline phosphatase - 910 EU, amylase – 28 g/h/l. Ultrasound: liver of normal size with dilated intrahepatic ducts; large gallbladder with thick (7mm) wall and many stones from 2 to 9 mm in size; CBD dilated till 18mm contains 14mm stone in distal part.
Formulate the diagnosis.
A. Chronic calculous cholecystitis, choledocholithiasis, obstructive jaundice.
B. Acute pancreatitis.
C. Cancer of head of pancreas.
D. Acute calculous cholecystitis, choledocholithiasis, obstructive jaundice.
E. Haemolytic jaundice.

5. In a 42-year-old women, after consumption of fatty food, developed an acute pain in the right upper quadrant, sclera icterus. Body temperature – 37.2°C. On examination: slight muscle rigidity in the right upper quadrant, signs of peritoneum irritation are negative. Biochemical test: bilirubin – 81 µmol/l (mostly due to direct), amylase – 25 g/h/l. US: enlarged gallbladder with stones from 3 to 9 mm in diameter, CBD – 12 mm.
Formulate the diagnosis.
A. Chronic calculous cholecystitis, choledocholithiasis, obstructive jaundice.
B. Acute biliary pancreatitis, choledocholithiasis, obstructive jaundice.
C. Cancer of head of pancreas.
D. Acute calculous cholecystitis, choledocholithiasis, obstructive jaundice.
E. Cholelithiasis, acalculous cholecystitis, choledocholithiasis, obstructive jaundice.

6. A 48-year-old patient complains of epigastric pain with irradiation to the back, nausea, vomiting, abdominal swelling. Described complaints developed during last 24 hours after alcohol consumption. CBC: erythrocytes – 4.2 x 10¹²/l, Hb – 135 g/l, leucocytes – 15.2 x 10⁹/l, banding
neutrophils – 10% segmented neutrophils – 77%, ESR – 18 mm/h. Biochemical test: bilirubin – 19.6 µmol/l, protein – 72 g/l, glucose – 5.2 mmol/l, amylase – 220 g/h/l, AsAT – 0.4 mmol/l, AIAT – 0.6 mmol/l, Ca²⁺ - 1.9 mmol/l.

Formulate the diagnosis.
A. Chronic calculous cholecystitis, choledocholithiasis, obstructive jaundice.
B. Acute biliary pancreatitis.
C. Acute pancreatitis, severe course.
D. Acute calculous cholecystitis, acute biliary pancreatitis, mild course.
E. Acalculous cholecystitis, acute biliary pancreatitis.

7. Male 47 y.o., complains with pain at the right subcostal area irradiated to the back, fever till 38.7°C. He is ill during 3th days. Skin is of normal colour; HR - 88 b/min., BP - 120/80 mm Hg. A tongue is dry. An abdomen is symmetric, muscular defence in the right subcostal area is present, where the enlarged painful gallbladder is palpable. Ortner’s sign is positive. Leucocytes – 14.2 x 10⁹/l.

What complication of acute cholecystitis das patient have?
A. Empyema of gallbladder
B. Hydrops of gallbladder
C. Cholangitis
D. Acute biliary pancreatitis
E. Cholelithiasis.

Atypical tasks (α =III):
1. Female, 53 y.o., during operation because of acute calculus phlegmonous cholecystitis, common bile duct is dilated to 13 mm. At cholangiography two stones 1x1cm and bile sludge were found. How to finish the operation?
A. Insert the drainage tube by Kehr’s maneure.
B. To put drainage by Halsted maneure.
C. To perform sphincterotony.
D. To perform choledochotomy, stone extraction and drainage.
E. To perform choledochotomy, stone extraction and bilioigestive anastomosis.

2. Female, 48 y.o., suffering from cholelithiasis for 10 years. The last attack 7 day ago has begun. The sclera and skin turned yellow; itch. Stool is acholic. Temperature is 37.6°C. Laboratory tests: Hb – 107 g/l, leucocytes – 11.0x10⁹/l, banding – 8%, ESR – 16mm/h; total bilirubin – 320 µmol/l, protein – 75 g/l, glucose – 4.7 mmol/l, AsAT – 0.3, AIAT – 0.95, alkaline phosphatase - 910 EU, amylase – 28 g/h/l. Ultrasound: liver of normal size with dilated intrahepatic ducts; large gallbladder with thick (7mm) wall and many stones from 2 to 9 mm in size; CBD dilated till 18mm contains 14mm stone in distal part.

What the tactics of treatment?
A. Open cholecystectomy, choledocholithotomy, external biliary drainage.
B. Laparoscopic cholecystectomy, choledocholithotomy, external biliary drainage.
C. Open cholecystectomy.
D. Therapeutic ERCP, laparoscopic cholecystectomy.
E. Laparoscopic cholecystectomy, rendezvous procedure, external biliary drainage.

3. In a 42-year-old women, after consumption of fatty food, developed an acute pain in the right upper quadrant, sclera icterus. Body temperature – 37.2°C. On examination: slight muscle rigidity in the right upper quadrant, signs of peritoneum irritation are negative. Biochemical test: bilirubin – 81 µmol/l (mostly due to direct), amylase – 25 g/h/l. US: enlarged gallbladder with stones from 3 to 9 mm in diameter, CBD – 12 mm.

What the tactics of treatment?
A. Open cholecystectomy, choledocholithotomy, external biliary drainage.
B. Laparoscopic cholecystectomy, choledocholithotomy, external biliary drainage.
C. Open cholecystectomy.
D. Therapeutic ERCP, laparoscopic cholecystectomy.
E. Laparoscopic cholecystectomy, rendezvous procedure, external biliary drainage.

4. During the operation at plump female 68 y.o. because of acute destructive cholecystitis the purulent cholangitis was found. There were no stones in CBD, but stenosis of Vaters’ papilla was confirmed. Cholecystectomy was performed. How is it necessary to finish the operation?
A. Perform external draining of CBD
B. Perform supraduodenal choledocho-duodenoanastomosis
C. Perform transduodenal sphincteroplasty
D. Perform choledochojejunostomy
E. To drain an abdominal region

**MCQs (α =І, α =ІІ)**

1. Indications for a intraoperative common bile ducts investigation are:
   A. The diameter of CBD more than 11 mm
   B. Empyema of the gallbladder
   C. History of pancreatitis
   D. Liver Cirrhosis
   E. Calculous cholecystitis

2. Specify complications which are proper in acute calculous cholecystitis:
   A. Cholangitis
   B. Liver cirrhosis
   C. Cicatrical stricture of terminal part of CBD and Vater’s papilla
   D. Haematemesis
   E. Melena

3. The preparations which in use for an intravenous cholangiography:
   A. Bilignost
   B. Yodlipole
   C. Bilitraste
   D. Cholevidum
   E. Barium sulphate

4. Contraindications for the intravenous cholangiography is:
   A. Advanced age
   B. Hyperbilirubinemia >200 mcM/l
   C. Discinesiya of gallbladder
   D. Individual unbearableness of iodine preparations

5. Urgent operation is indicated at:
   A. Perforation of gallbladder, peritonitis
   B. Biliary pancreatitis
   C. Biliary colic
   D. Obstruction of the cystic duct
   E. Stricture of CBD

6. Patient with a “destructive” cholecystitis, complicated with widespread peritonitis, needs:
   A. Urgent operation
   B. Operation after stomach and colon barium investigation
C. Percutaneous cholecystostomy
D. Conservative treatment
E. Surgical treatment on the vital indication

7. Normal level of bilirubin in serum blood is:
   A. 8.5-20.5 mcmol/l
   B. 27.6-32.8 mcmol/l
   C. 22.5-28.3 mcmol/l
   D. 40.1-40.6 mcmol/l
   E. 33.6-36.7 mcmol/l

8. What does not complication of an acute calculous cholecystitis:
   A. Subhepatic abscess
   B. Cholangitis
   C. Obstructive jaundice
   D. Varices of oesophagus
   E. Peritonitis

12. An increase in which of the following enzyme levels does NOT imply cholestasis?
   A. Alanine aminotransferase (ALT)
   B. Alkaline phosphatase
   C. Gamma glutamyltransferase (GGT)
   D. 51-nucleotidase (51 -N)

Literature