Diseases of the biliary system: biliary dysfunction, chronic non-calculous cholecystitis, cholelithiasis.

Biliary dysfunction (biliary dyskinesia) refers to altered tonus of the sphincter of Oddi (usually increased pressure), disturbance in the coordination of contraction of the biliary ducts, and/or reduction in the speed of emptying of the biliary tree.

Relevance:

Biliary dyskinesia – is a very common pathology.

They often starting as the development of more serious biliary abnormalities (cholecystitis, cholangitis, cholelithiasis).

Manifestations of biliary dyskinesias causing major clinical features (pain and dyspeptic syndromes) cholecystitis and other organic cholepathy.

Etiopathogenesis

<table>
<thead>
<tr>
<th>Primary Causes</th>
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<tbody>
<tr>
<td>disorders of the <strong>somatic (CNS) and autonomic regulation</strong> (hypothalamic disorders vagotonia, sympathicotonia, neuroses);</td>
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<tr>
<td><em>endocrine disorders</em> (insufficient production of sex hormones, steroids, thyroid hormones);гінекологічні хвороби;</td>
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<tr>
<td>pathology of the spine;</td>
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<td>genetic disorders, eg., congenital weakness of the muscles of the gallbladder;</td>
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<tr>
<td>imbalance between the production of cholecystokinin and antiholetsystokinin.</td>
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</tbody>
</table>
Secondary

Causes:

- at organic disorders in **primary biliary system** (inflammation calculoiz, congenital anomalies of sphincteri, bend, organic stenosis of the sphincter, etc) and liver (bile chemistry violation);
- at **primary organic lesions** of other organs of the digestive system (stomach ulcer, gastritis, duodenitis, enteritis, colitis, appendicitis, pancreatitis).

*International Classification of functional disorders of the biliary system, developed by the Working Committee of functional disorders (1994)* identifies two types of biliary disorders, calling their dysfunction:

1. gallbladder dysfunction.
2. sphincter Oddi dysfunction.

Under the so-called *Rome criteria-III* (2006), sphincter Oddi dysfunction is biliary and pancreatic type.

Biliary dysfunction (BD):

- Dystonia (violation of tonus, patency)
- Dyskinesia (violation contractility, motor-evacuation function)

**Clinical classification BD**

I. Tonus:

a) hypertension;

b) hypotension.

In this case is characterized the basal tonus (tonus to stimulation) the following components of bile:

- The sphincter of Oddi and (if possible) and sphincter of the cystic bile duct or Mirizzi;
- Gallbladder, extra- and intraliver bile ducts.
II. motor-evacuation function (contractility):

a) hyperkinesia;

b) hypokinesia.

Evaluate contractility of:

- Gall bladder;
- Intraliver bile ducts.

There are also:

The same type of biliary dysfunction:

- hypertonic-hyperkinetic;
- hypotonic-hypokinetic.

-Associated biliary dysfunction (mixed character).

Diagnostic criteria of the biliary dysfunction (according to the Working Committee of functional diseases of the biliary tract, 1994):

I. Clinical data:

- recurrent episodes within 3 months of moderate to intense pain in the epigastric or right upper quadrant lasting 20-30 minutes, the pain occurs after meals, sometimes at night (especially at children’s), radiating to the back or right shoulder;

- nausea or vomiting parallel to the pain.

II. Laboratory and instrumental data:

- evidence of dysfunction of the gallbladder and sphincter Oddi;
- absence of structural changes in the gallbladder and bile ducts, which can explain the emergence of these symptoms.

A similar diagnostic criteria dysfunction of the gallbladder and sphincter of Oddi by Rome III criteria revision, 2006.

Diagnostic criteria of the biliary dysfunction

1. Clinical and medical history (various manifestations of pain and dyspeptic syndromes, depending on the nature of the BD):
- Hypotension, hypokinesia - heaviness or dull pain in the right upper quadrant, bitter taste, dry mouth, flatulence, constipations are possible;

- Hypertension, hyperkinesia - more expressed pain, nausea, vomiting, diarrhea;

- Associated BD - a combination or alternation of the above symptoms.

2. Duodenal sounding.

3. Ultrasonographic tests (reading contractility of the gallbladder).

4. Cholecystography, CT

5. Tests to diagnose sphincter Oddi dysfunction.


**Fractional duodenal intubation** – informative method of assessing in vitro bile and bile formation.

*Duodenal endoscopy provides information on:*

- Tonus of the valvulars of biliary system, especially the sphincter of Oddi, in some way - tonus of the gallbladder (mainly hypotension);

- Contractile-evacuation function of the gallbladder and intraliver ducts;

- Chemical composition of bile (mixed with duodenal juice), the presence of microbial, parasitic contamination.

**Phases, portions and regulations of fractional duodenal intubation**

<table>
<thead>
<tr>
<th>Phases</th>
<th>Norm secretion of bile</th>
<th>Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>I – choledochus phase, the phase of the common bile duct</td>
<td>15–20</td>
<td>15–20</td>
</tr>
<tr>
<td>II – phase of the closed sphincter Oddi</td>
<td>–</td>
<td>3–6</td>
</tr>
<tr>
<td>III – phase cystic duct (A portion – bile, collected in phase I and III - extraliver bile)</td>
<td>3–5</td>
<td>3–5</td>
</tr>
<tr>
<td>IV – phase of the emptying of the gallbladder (portion B - gallbladder bile)</td>
<td>30–60</td>
<td>20–30</td>
</tr>
<tr>
<td>V – phase of the allotment of intraliver bile (a portion C - hepatic bile)</td>
<td>During first 15 min. – 1 ml per minute</td>
<td></td>
</tr>
</tbody>
</table>

**The main options of the biliary dysfunction according to duodenal intubation:**

Hypertension of sphincter Oddi - if the duration of Phase II more than 6 minutes.

**Gallbladder dysfunction:**

- Hypertension GB - if excreted gallbladder bile volume <30 ml;
- Hypotension GB - if excreted volume of bile > 60 ml;
- Hyperkinesia GB - when Phase IV lasts <20 minutes;
- Hypokinesia GB– when Phase IV lasts >30 minutes.

Ultrasonographic test:

I. Basic data.

Studing the degree of reduction in volume (area) of the gallbladder after 15-30 minutes. after taking cholekinetik:

- Normal - 35-50%;
- During hypokinesia GB - less than 35%;
- With hyperkinesia GB - over 50% (up to complete reduction). II. Additional data.

- if the volume of GB after 15 minutes after stimulation does not change, then it is sphincter of the cystic bile duct hypertension (in the absence of organic obstacles for outflow of bile);
- if the volume of GB after 15 minutes after stimulation increases, then it is a sphincter Oddi hypertension (in the absence of organic lesions of the sphincter).

Interpreting cholecystography

Hypertonic-hyperkinetic form of dyskinesia:
- Reduction GB;
- Well contrasted;
- Its fast emptying.

Hypotonic-hypokinetic form of dyskinesia:
- Extended, enlarged GB;
- Emptying it sharply slow or does not occur after repeated injections of stimulants.

Diagnostic tests of sphincter Oddi dysfunction – often used in postcholecystectomy syndrome to clarify the nature of patency of bloc sphincter (functional or organic):
a) ultrasound of choledoch after taking fatty foods or injection of cholecystokinin, in violation of patency of the sphincter Oddi appears pain, sonographic - choledochal diameter increases (rather than reduce it);

b) determination of hepatic and / or pancreatic enzymes during pain attack (at the sphincter obstruction - delay juice- and bilesecretion, activation of markers of cytolysis and cholestasis);

c) endoscopic retrograde cholangiopancreatography: identifying barriers in organic sphincter;

d) endoscopic manometry of sphincter Oddi - to identify lesions of ampoules of biliary or pancreatic segments;

d) quantitative scintigraphyof the liver and biliary system (slowing excretion of isotope).

**Treatment Program at patients with BD**

- **Diet.**
- **Etiotropic therapy (BD).**

- **Drug therapy pathogenetic and symptomatic:**
  
  A. The fight against pain.
  
  B. Correction of biliary excretion (choleretic rational use).
  
  C. Correction of general condition.

- **Non-drug treatment.**

**Diet.**

Diet - table №5 by Pevzner at an exacerbation or table №15 (general) outside an exacerbation (in secondary dyskinesia - a diet considering the underlying disease). Eating 5-6 times / day, in small portions; at hypertonicity, hyperkinesia or mixed biliary dyskinesia - limit spicy, fatty, fried food, extractives.

**Etiotropic therapy.**

Real secondary dyskinesia- primary treatment of digestive diseases. For example, the primary cholecystitis - anti-inflammatory therapy, with gastroduodenitis - Helicobacter treatment and so on.
Drug therapy pathogenetic and symptomatic
A. The fight against pain.

At hypertonicity, hyperkinesia:

**Choleantispasmodic**

a) Anti-Muscarinic agents (platifillini, buscopan, duspatalin, gastrozepin, riabal);
b) myotropic antispasmodics (papaverini, no-spa);
c) Ca-antagonists channels, selective to digestive tract (Pinaverium bromide);
d) at isolated hypertonicity of sphincter Oddi: aminophylline, nitrates.

At hypotonia, hypokinesia or mixed BD (hypertonicity, hypokinesis):

2.1 at Hypotonia, hypokinesia: a) prokinetic: mosapryd (Mocid MT), itopridum(hanaton, primer).

б) Bile means: cholekinetiks + choleretic at the presence of disholia.

2.2. At mixed dyskinesia– combination of prokinetic with choleantispasmodic or complex of choleretic agents.

**Classification of bile means**

1. Drugs that stimulate cholerez (choleretic).
   - Improve formation of liver bile. It is so-called true chologogue. Choleretic originally divided into four groups::
     - Products containing bile or bile acids.
     - Synthetic choleretic.
     - Herbal products.
     - Hydrocholeretic (drugs that increase the secretion of bile due to its water component).

2. Drugs that improve bile secretion.

Divided into three groups::

- Cholekinetics - drugs that increase the tone of the gallbladder while turning the reflex of opening sphincter Oddi.
- Drugs with combined (modulator) effect on biliary excretion - regulating valve tonus and improve contractility of the gallbladder and ducts.

3. Medications with complex effect on bile formation and biliary excretion (combining the characteristics of the first and second sub-groups).

**Chronic (non-calculus) cholecystitis (CNC)** - a chronic polyetiological inflammatory disease of the gallbladder that develops involving infection (bacterial, rare – viral microflora, parasitic invasion), usually accompanied by functional disorders of motility and tonus of the biliary system (dysfunction) and changes in physical and chemical properties of bile (dyscholia).

**Background and epidemiology**

- CNC - one of the most common pathologies of the digestive system;
- people are sick, starting from a young age; more women (3 times more often than men);
- disease usually recurs, resulting in temporary disability;
- appears as after acute cholecystitis, and (often) - as primary chronic process.

**Etiology**

In the development of gallbladder inflammation (inflammation of the wall of the organ) playing the role of **three factors**:

I. The main factor - infected gallbladder bile.

II. Factors causing stagnation of bile (including cystic).

III. Factors that cause irritation of the gallbladder wall.

Groups II-III are optional (contributing).

Sources of infection: foci of chronic infection in the body (tonsillitis, periodontal disease, sinusitis, pyelonephritis, adnexitis, appendicitis, cholangitis, dysbiosis, etc.).

**Pathogens of CNC:**

- More - escherichia coli (35-40% of cases);
- staphylococci, enterococci (15%);
- streptokok (10%);
- hepatitis B (10%);

- Giardia (up to 3-5% of their direct destruction of gallblader, much more % - persistence of Giardia in duodenum, promotes biliary dysfunction with spasm of the sphincter Oddi and gallbladder bile stasis);

- a third of cases - mixed flora.
Now begins to be seen the etiologic role of pyloric and nonpyloric helicobacters.

II. Factors contributing to the stagnation of bile:

- violations of the diet;
- psycho-emotional stress;
- hypodynamia;
- violation of the innervation and endocrine regulation bladder (congenital and gained character);
- violation of the metabolism, contributing to the development of dyscholia (obesity, diabetes, atherosclerosis, etc.).
- regular use of a number of drugs (fibrate derivatives, estrohenotherapy of menopause, progestins, octreotide, some cephalosporins, H2histaminoblocators etc.);
- parasitic infestation and dysbiosis in the small intestine, contributing to impaired patency of the biliary system due to reflex (protective) spasm of the sphincter Oddi;
- structural changes along the the biliary system, that violating flow of bile (membrane, bend, stricture, external union, etc.).

III. Factors contributing irritation (damage) of the gallbladder wall:

- irritation wall changed due dyscholia components of bile (microliths, sludge, stones);
- wall irritation by activated pancreatic and intestinal enzymes due to pancreatic-biliary duodeno-reflux, while developing the so-called enzymatic (chemical) cholecystitis;
- toxic factors (alcohol, heavy metals, pesticides);
- allergic, including autoimmunity (autoimmune cholecystitis);
- gallbladder injury.

**Classification**

There is no generally accepted clinical classification of chronic cholecystitis. After A.M. Nohallerom, J. S. Zimmerman, V.O. Galkin (1986), with complement:

I. The presence of calculous:

1. Chronic non-calculous (noncalculous) cholecystitis.
2. Chronic calculous cholecystitis.

II. The nature of the course:

1. Frequently relapsing.
2. Recurrent.
3. Latent (monotonous, flabby, monotonous flow).

III. Phase of the disease (for relapsing course options):

1. Aggravation (decompensation).
2. fading exacerbation (subcompensation).
3. Remission (compensation).

IV. The degree of severity:

1. Mild - unexpressed pain, no exacerbation (latent) or rare (1-2 per year), short (2-3 weeks).
2. Moderate - the pain is more pronounced, expressed dyspeptic symptoms, exacerbation are protracted, 5-6 per year.
3. Severe - pronounced pain, diarrhea, inflammatory syndromes, monthly - signs of exacerbation as long biliary colic, frequent complications.

V. Functional status:

- the presence of secondary dyskinesia disturbances in the biliary system and, in particular in gallbladder, indicate the nature of the gallbladder dyskinesia (dysfunction).

VI. Complications (if any):

- pericholecistitis;
- Cholangitis, pancreatitis, hepatitis (the primary cholecystitis);
- hydrops of the gallbladder;
- empyema of the gallbladder;
- gallbladder perforation with peritonitis;
- Formation of stones (at primary noncalculous cholecystitis).
- cancer of the gallbladder.

**Diagnostic criteria CNC**

- Clinic - medical history, various (depending on the nature dyskinesia) pain in the right upper quadrant after diet disorders, nervous stress, alcohol intake; biliary dyspepsia (bitter taste, dry mouth, nausea after eating, bloating, etc.) intoxication syndrome of varying severity (weakness, fever, tachycardia, etc.).
- Evidence: positive bladder symptoms (Kera, Murphy, Ortner, George Musso, etc.).
- Duodenal intubation: white blood cells in cystic portion of bile; evaluation of related disorders, identifying microlite.
- Detection of pathogenic microorganisms in bacteriological sowing of the bile.
- Ultrasound: the gallbladder wall at CNC - thicker than 4 mm (at least in the neck organ), dual circuit peryprotes; body size is usually increased, content stagnant.
- For more information on the structural changes of the gallbladder may give oral cholecystography, computed tomography.
- At the exacerbation in blood - leukocytosis, leukocyte formula shift, increased ESR.

**Hospitalization**

Applies during exacerbation of CNC with moderate and severe recurrent course.

I. Regime in hospital – in bed.

II. Diet - table number 5 or 5a.

III. Drug therapy.

1. Anti-inflammatory anti-infective therapy (preferably with regard cultured from bile pathogens):
- Semisynthetic penicillins (ampioks, oxacillin by 0.25-0.5 g 4 g. daily, amoxicillin 0.5 -1.0 2.3 g. per day);

- Macrolides (with staphylococcus infections), erythromycin inside of 200-400 mg 4 g. per day (starting dose of 400-600 mg), clarithromycin (klatsyd) 0.25 - 0.5 g, 2 g. per day, roxithromycin, azithromycin;

- Tetracyclines (tetracycline inside of 0.1-0.25 g 4 g. per day, orally or doxycycline / drip of 200 mg per day);

- Biseptol (septryn, Bactria) by 480-960mh (Table 1-2). 2 g. per day;

- Cephalosporins (cephalexin by 0.25-0.5 4 g. per day; tsefobid 1 g 2 g. per day / m; zinnat by 0.25-0.5 g 2 g. per day after meals, etc.);

- Fluoroquinolones (500-750 mg ciprofloxacin for 2 years. a day abaktal 0.4 g 2 g. per day, taryvid 0.2 g 2 g. per day);

- Metronidazole (flahil, Metrogyl et al.) to 0.4-0.5 g 3 g. per day, tinidazol 0.5-1 g 1 g. per day;

- At parasitic lesions – taking metronidazole, nitrofurans (Nifuroxazide, furadonin, furazolidone in medium doses); vermoks 1 table. 2 g. Per day, vormil etc..

The course of anti-infective therapy – 7 – 14 days.

2. Detoxification intravenous therapy (Neogemodez 200-400 ml of 5% glucose 200-400 ml, etc.).

3. Effective pain relief and correction of dyspepsia (especially nausea and vomiting):

   - Parenteral antispasmodic, spazmoanalhetics, no –shpa 2% - 2.0 , atropine sulfate 0.5 – 1.0 0.1% solution platifillin 1-2 ml of 0.2% solution baralgin in 5 ml, fenikaberan, duspatalin, riabal etc.;

   - Prokinetic action: metoclopramide (tservukal) 2,0 0,05% solution, chlorpromazine 1.0 2.5% solution (vomiting); domperidone or mosapryd 10-20 mg (1-2 table.) 3 g. per day for food and others.

4. Vitamin therapy (ascorbic acid, vitamin B, folic acid, etc.).

5. Sedatives, herbal preparations (persen, fitosed, tincture hawthorn et al.), the lack of effectiveness – elenium, tazepam more.
6. In the second half of inpatient treatment, subsiding exacerbation phase, after the end of anti-infective drugs prescribed:

a) cholagogue depending on the type of secondary biliary dyskinesia;

6) started a course of physiotherapy inhibitory or tonic type (depending on the type of secondary dyskinesia), which ends with the end of treatment in hospital.

7. To correct concomitant maldyhestia prescribed enzymes (festal, dyhestal, mezim, creon, panzinorm etc.) after meals; sorbents (smectite, espumizan) rates to 7 days;

8. To prevent dysbiosis after antibiotic prescribed probiotics (Hilak forte, Symbiter Bifi-forms, laktyv-ratiopharm).

**Gallstone disease (GSD)** – disease caused by violation of metabolic cholesterol and/or bilirubin, which is characterized by the formation of gallstones in allbladder and / or bile ducts.

**Background and epidemiology:**

- quite common (sick one in four women and one in ten men);

- a significant negative impact on the performance and quality of life;

- often develop serious acute and chronic complications.

More common in women under 40, especially those that many birth, overweight, natural blonde, with a probable genetic predisposition (GSD mother or other relationships). After '50 GSD frequency in men and women is almost the same. After 70 years in biliary system calculos can be found in almost 2/3 of people (usually oligosymptomatic or asymptomatic).

**Gallstones** – crystal structure, resulting in bile with altered physic-chemical properties (bile with symptoms of dysholia).

**Lithogenesis process goes through the following stages:**

- Physico-chemical stage – dysholiya phenomenon, ie the presence of insoluble crystals of cholesterol and / or bilirubinatu Ca, revealed by microscopy and bile are not detected during ultrasound biliary system;

- Pre-stones stage - accumulation of crystals of up to 1.2 mm (microlite) that displays ultrasound, including sludge syndrome (lithogenesis for cholesterol stones) - a condition where accumulation of cholesterol crystals begin to appear
with ultrasound as hyperechoic sediment on the bottom contour of the gallbladder, but still does not characteristic of stones hipoechoic shade;

- Calculosis - stage of formed concrements.

There are cholesterol, pigment (bilirubin) and mixed calculus in structure.

**Etiopathogenesis**

In the etiopathogenesis of the disease playes role the same three groups of factors (stagnation of bile, bile infection, damage to the walls of the bladder and ducts) that are characteristic’s for chronic cholecystitis (details - see. CNC) and heredity.

For cholesterol stones leading role played by stagnation of bile from cholesterol and its glut and to some extent genetics.

For bilirubin (pigment) stones essential role played by structural irregularities in the hepatobiliary system and the presence of infection, contributing, including infringement conjugation of bilirubin and its formation of insoluble salts.

**Classification of GSD (cholelithiasis)**

I. Localization and presence of inflammatory changes::

1. Cholecystolithiasis (calculosis gallbladder):
   - acute or chronic cholecystitis (acute or chronic calculous cholecystitis);
   - without cholecystitis.

2. Cholanhiolithiasis - choledocholithiasis, intraliver cholelithiasis (calculosis in choledoh or intraliver bile ducts):
   - with cholangitis (angiocholitis - inflammation of the large bile ducts, including choledochitis or cholanhiolit - inflammation of the small bile ducts);
   - with non-calculous cholecystitis;
   - without cholangitis, cholecystitis.

3. GSD localized stones in the bladder and ducts (with signs of inflammation of the relevant parts of the biliary system or not).

II. Severity of the disease:
A. Chronic calculous cholecystitis classify as CNC, the degree of severity (mild, moderate, severe), the nature of the course (often recurrent, relapsing, latent) on similar criteria;

B. GSD without cholecystitis share the progress to:

- latent form (oligosymptomatic or asymptomatic course with no episodes of pain, including availability of stones);

- with occasional attacks (biliary colic 1-2 g. per year, with no complications);

- with frequent attacks (3 or more times a year, likely complications).

C. GSD with recurrent flow allocate phase of the disease:

- aggravation;

- remission.

III. Additional characteristics of concrements:

A. Building - cholesterol, pigment, mixed.

At formulating the diagnosis division stones in structure, usually not performed.

B. By number - single (1-2), multiple.

C. Size - small (3-5 mm), medium (6-15 mm), large (over 15mm).

Crystals of cholesterol and / or Ca bilirubinatu size 1-2 mm, do not give hipoechoic shadow ultrasound, called microliths.

IV. Complications (if any):

-acute (including abscess or gangrenous) cholecystitis, cholangitis;

-perycholecystitis;

-cholemia;

-chronic hepatitis, secondary biliary cirrhosis;

-acute or chronic pancreatitis;

-so-called "Reactive pancreatitis" (an edema of the pancreas and moderate violation of its functions completely disappear after exacerbations GSD or removal of concrements); in fact, it is - version mild acute pancreatitis severity;
-so-called "Disabled" (full bladder filling concrements while maintaining or increasing its size) or "puckered" (substantial reduction in the size gallbladder practically to the value of existing of concrements) gallbladder;

-perforation of the wall of the biliary system with peritonitis.

**Clinic of GSD**

In 60% (over the age of '70 - 80%) patients with GSD the flow is mild (heaviness in the right upper quadrant, E. dispersion) or even asymptomatic. In other patients with gallstones, and the vast majority of patients with clinical manifestations of GSD bright enough.

A typical manifestation of relapse (acute) GSD is bile (biliary, hepatic) colic -acute attack of abdominal pain visceral nature, the most common cause of which is a temporary obstruction of the biliary ductal system with calculous often - in the cystic duct (in the beginning).

In any form of flow (e.g.latent) can be observed dyspeptic complaints, bitterness, dry mouth, regurgitation of food, bloating, stool multidirectional disorders.

When connecting an acute inflammation (or exacerbation of chronic) - fever (from long subfebrile - to 38-39 °C).

A steady obstruction of the biliary system be calculosis (especially - at choledoch, sphincter of Oddi) accompanied by jaundice mechanical genesis of varying degrees of severity and duration.

**Diagnostic criteria GSD**

1. Clinical and medical history - a common complaint at this time in history, a strong pain on palpation in the right upper quadrant, positive bladder symptoms. However, the lack of a typical clinic does not exclude the probability of latent GSD.

2. In protracted bilious colic, inflammation - a change in laboratory parameters, leukocytosis, increased ESR, levels of total and conjugated bilirubin, transaminases, alkaline phosphatase; without exacerbation GSD come into these figures to be within normal limits.

3. Instrumental methods of diagnosis:

   A. Ultrasonography (US) - the most effective method for diagnosing gallstones.

       Ultrasound signs:
- Echopositive (hyperechoic) floating or fixed to the neck of the bladder (an exacerbation) lesions of various sizes, giving down from themselves echonegative (hypoechoic) track-shadow;

- in "disconnected" by calculosis bladder - organ itself is not visible, but the place it begins placing hipoechoic clear path;

- ultrasound signs of exacerbation (visual) cholecystitis - thickening of the walls of the bladder walls outline dual walllls, edema;

- the presence of stones in the ducts - extension of ducts, revealing a large concretions (clearly not always visible).

B. Plain radiographs of the abdomen - to differentiate visible calcified or pigment stones that are redipositive and visible on the radiograph (redionegative) cholesterol biliary stones (required for choice of treatment).

C. Contrast cholecystography - used rare due to the smaller of ultrasound informative and a number of contraindications, while allowing the diagnosis of "disconnected" gallbladder and refine the degree of calcification of cholesterol stones.

D. Endoscopic retrograde cholangiopancreatography- can more accurately assess the condition and patency of the bile ducts, particularly choledochal simultaneously carry through sphincter of Oddi choledochal stone extraction intraendoskopichnymy tweezers. However, the method has a high incidence of local complications (up to 15-18% of cases, which limits its use in therapeutic clinic).

E. Computer tomography (CT) and magnetic resonance cholangiopancreatography (MRCPG) - have advantages over ultrasound in the diagnosis of primary cholecystolitiasis.

However, CT allows you to clearly assess the degree of calcification of calculus (weakening coefficient CT 70 Hounsfield units corresponds the degree of calcification of concrements, not suitable for drug dissolution or shock-wave lithotripsy) and MRCPG most clearly appreciate the state of extra- and intraliver bile duct, showing them calculus and other pathological changes.

**Treatment methods** include active fight against calculosis and more conservative (symptomatic, supportive) therapy.

I. Apply 3 methods of active treatment GSD (liquidation calculosis):

- Apply 3 methods of active treatment GSD (liquidation calculosis):
Non-invasive conservative methods of oral drug dissolution of stones: ursodeoxycholic acid

Minimally invasive techniques:

a) fragmentation stones by extracorporeal shock wave lithotripsy;

b) direct contact litoliz (by transcutaneous injected under ultrasound in gallbladder of alcohol-esters);

c) transpapillary extraction of large stones from choledochal sphincter Oddi or intra endoscopic forceps during upper endoscopy.

Invasive surgical techniques:

a) Laparoscopic cholecystectomy ("gold standard" treatment GSD);

b) classic cholecystectomy through a wide access (especially in complicated stones and bile duct);

c) cholecystectomy through a small access (rarely used).

Litolythic oral therapy (LOT) is performed by using medications containing derivative of oxycholic acid- ursodeoxycholic acid UDCA (ursofalk, ursohol, ursosan).

LOT is shown for 15-20% of patients with cholelithiasis. Indications for LOT:

- The early stage of the disease in the presence of pure cholesterol noncalcified stones;

- The sizes of stones - to 10 mm;

- Single calculus (dissolve and more small stones, if they take up 1/3 of bladder volume);

- Uncomplicated GSD, with relatively rare attacks of biliary colic moderate intensity;

- Preserved contractile function of the gallbladder.

II. Additional symptomatic therapy is made on a background of drug litoliz, as a preparation for lithotripsy or surgery and in postoperative period, or at the rejection of operation and dynamic observation of patients.

1. Diet: №5 - out of exacerbations and in latent GSD, in exacerbations, complicated GSD - a more strict version of it (№5a), and preoperative period
against the background of acute complications - even short-term hunger with elements of parenteral nutrition.

2. The main directions of therapy "on demand":

- Effective pain relief.
- If available - fight against inflammation.
- Correction of maldigestion phenomena and stool disorders.