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Departments of Pediatrics №2

CONFIRMED by

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METHODOLOGICAL RECOMMENDATIONS ON PRACTICAL CLASSES FOR STUDENTS

International Medical Faculty, course 6

Educational discipline "**PEDIATRICS**"

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1. **Topic № 21**

Differential diagnosis of diseases of the hepatobiliary system and pancreas in children. Leading clinical symptoms, syndromes and variants of the course in functional and organic diseases of the hepatobiliary system and pancreas in children (dysfunction of the gallbladder and sphincter of Oddi, acute and chronic cholecystitis, acute and chronic pancreatitis and chronic hepatitis). Differential diagnosis of diseases of the hepatobiliary system and pancreas in children. Data of laboratory and instrumental studies in diseases of the hepatobiliary system and pancreas in children. Differential diagnosis of diseases accompanied by exocrine insufficiency of the pancreas and functional and organic diseases of the hepatobiliary system in children. Differential diagnosis of diseases of the hepatobiliary system and pancreas in children. Treatment of children with functional and organic diseases of the hepatobiliary system and pancreas, correction of exocrine pancreatic insufficiency in children. Providing emergency care for acute liver failure and complications of portal hypertension syndrome. Prevention and dispensary monitoring of diseases of the hepatobiliary system and pancreas in children.

2. **Relevance of the topic.**

Digestive diseases are the second most common diseases in childhood after diseases of the bronchopulmonary system. Diseases of the biliary system in school-age children account for 80% of all chronic diseases of the digestive system, and functional disorders of the biliary tract (FDBT) are the most common among them. In turn, the pathology of the pancreas in children remains one of the most difficult sections of pediatric gastroenterology, as the symptoms of this pathology are similar to the symptoms of other diseases, and detailed verification is complicated by limited diagnostic capabilities due to difficult access to both the organ and its secretion. A feature of chronic diseases of the digestive system in children and adolescents is the defeat of adjacent organs due to their anatomical and functional relationship, general blood and lymph circulation, neurohumoral and autonomic regulation. The urgency of the problem is also due to the tendency to recurrence and insufficient effectiveness of modern therapies.

3. Objectives of the lesson

3 .1. General and goals

- Improve knowledge of the anatomical and physiological characteristics of the digestive system
- Learn to differentiate between organic and functional diseases
- To improve the understanding of the mechanisms of different types of digestion and the pathogenesis of their disorders
- Know the classification and clinical syndromes of organic and functional diseases of the gastrointestinal tract
- Know modern methods of diagnosis of organic and functional diseases of the biliary tract and pancreas
- To improve the ability of differential diagnosis of the above diseases
- Be able to prescribe differentiated treatment depending on the type of digestive disorders in children

3 .2. *Educational and goals:*

Get acquainted with modern recommendations and protocols for the treatment of diseases of the pancreas and biliary tract, reduce mortality, disability, frequency and severity of these diseases, as well as contribute to improving the physical development of the child.

3 .3. *Specific goals:*

- to acquaint students with modern ideas about the prevalence of functional disorders of the biliary tract, chronic pancreatitis in children;

- discuss the etiopathogenesis, classification, clinical course, additional diagnostic methods, differential diagnosis, treatment and prevention of FDBT, chronic cholecystitis, chronic pancreatitis;
- to work out the anamnesis of factors that contribute to the pathology of the digestive system, analysis of anamnestic data, diagnosis of FDBT, chronic cholecystitis and chronic pancreatitis on the basis of clinical and instrumental methods of examination, differential diagnosis with diseases with similar syndromes according to the form with classification, drawing up a plan of regime, treatment and prevention measures.

3 .4.On the basis of theoretical knowledge on the topic:

Be able to:

- to conduct a quality history in the case of organic and functional diseases of the biliary tract and pancreas
- physical examination followed by interpretation of the obtained data
- prescribe diagnostic procedures
- make diagnoses according to modern classifications
- prescribe modern therapy and further prevention of organic and functional diseases of the biliary tract and pancreas
- in case of complicated course of organic diseases of the biliary tract and pancreas, be able to recognize them in a timely manner and provide quality care.
- to develop preventive measures, to determine the principles of dispensary observation.

Master practical skills:

- physical examination of the pancreas and biliary tract in children of different ages;
- *to* identify characteristic of diseases of the pancreas and biliary tract changes during ultrasound examination;
- evaluate the results of biochemical blood tests.

4 . Materials of pre-classroom independent training (interdisciplinary integration)

№	Disciplines	Know	To be able
1	Propaedeutics of children's diseases	The main anatomical and physiological features of the body depending on the age of the child	Ability to collect anamnesis, examine the child by systems, determine the parameters of physical development, prescribe feeding and nutrition to young children, prescribe care for healthy and sick children, evaluate the results of a couple of clinical research methods.
2	Faculty of Pediatrics, Neonatology, Medical Genetics, Pediatric Surgery.	Differential approach to the child depending on the child's condition	Clinic, classification, diagnosis, differential diagnosis of diseases accompanied by abdominal pain, diarrhea, fever, eating disorders. Prescribing treatment, providing emergency care.
3	Clinical pharmacology.	Drugs used to treat diseases	Knowledge of pharmacodynamics and pharmacokinetics of drugs required for the treatment of the above diseases and conditions, the ability to prescribe them.

4	Children's infectious diseases.	Differential approach to the treatment of a sick child depending on the etiology of the disease	Knowledge of clinic, classification, diagnostics, differential diagnosis of infectious diseases accompanied by diarrhea, prevention, treatment

5. Content of the topic

Functional disorders of the gallbladder and sphincter of Oddi

Code 82.8 - dyskinesia, or dysfunction (functional disorder) of gallbladder

Code 83.4 - spasm of the Oddi sphincter (SFO)

Definition. Functional disorders of the biliary tract (FDBT) - impaired motility of the gallbladder and / or tone of the sphincter apparatus (sphincter of Oddi) due to uncoordinated, untimely, insufficient or excessive contraction of the gallbladder and / or sphincter apparatus, accompanied by a violation of the bile duct. The disease is polyetiological and genetically determined.

The pathophysiological formation of functional disorders of the biliary tract (FDBT) in children is due to a violation of neuro-humoral mechanisms of regulation of the rapid function of the gallbladder and sphincter tone Oddi, Lutkens, Mirizzi. Motor activity of the biliary tract is regulated by parasympathetic and sympathetic divisions of the autonomic nervous system, gastrointestinal hormones (cholecystokinin, secretin, gastrin, glucagon), thyroidin, oxytocin, corticosteroid and sex hormones.

In the structure of the pathology of the biliary tract, FDBT is, according to various authors, 65-85%, mainly in various diseases of the digestive system in school-age children. Isolated FDBT is most common in preschool children.

Classification of functional disorders of the gallbladder and sphincter of Oddi (Roman IV Consensus)

Types of functional disorders of the biliary system according to the Roman criteria

<table>
<tr><td rowspan="3">E. Disorders of the gallbladder and sphincter of Oddi</td><td rowspan="2">E1. Biliary pain</td><td>E1a. Functional biliary vesicular disorder</td></tr>
<tr><td>E1b. Functional disorder of the biliary sphincter of Oddi</td></tr>
<tr><td colspan="2">E2. Functional disorder of the pancreatic sphincter Oddi</td></tr>
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Classification of functional disorders of the biliary tract (GA Lezhenko, 2014)

Localization	▪ Gallbladder ▪ sphincter of Oddi (SFD)
Etiology	▪ Primary ▪ Secondary

Functional condition	▪ Hypofunction / hyperfunction of JM ▪ Spasm / insufficiency of SFD
Clinical forms	▪ Hyperkinetic-hypertensive ▪ Hyperkinetic-hypotonic ▪ Hypokinetic-hypotonic ▪ Hypokinetic-hypertensive *

* there may be options for combination with normal sphincter tone or gallbladder kinetics

Initial research:

Laboratory: co-program; serum biochemical study: cholesterol, alkaline phosphatase, GGTP, total bilirubin and fraction, AST, ALT, amylase and lipase. Microscopic and biochemical examination of bile (in the presence of biliary sludge).

Instrumental: dynamic ultrasound cholecystography - echosonographic examination to determine the functional state of the gallbladder and sphincter of Oddi (such as functional disorders of the biliary tract.)

Indications: FE GDS, IV cholecystography, fractional duodenal sounding, bacteriological, biochemical and microscopic examination of bile.

Symptoms, syndromes, physical status .

History - the duration of the disease is more than 3 months, hereditary predisposition, abnormalities in the development of the gallbladder and biliary tract.

Complaints of hyperfunction : pain in the right hypochondrium, umbilical region is paroxysmal, 20-30 minutes after eating, physical or emotional stress, loss of appetite, fatigue, emotional lability.

Complaints of hypofunction : dull, aching pain in the right hypochondrium after eating after 60-90 minutes, exercise; nausea, vomiting, bitter taste in the mouth, fatigue, emotional lability, dizziness, etc.

Physical status: the pain on palpation in the right hypochondrium, umbilical region, possible positive bladder symptoms (Kera, Murphy, Vasilenko), in the hypokinetic type - an increase in the size of the liver (soft, mobile, painless, decreases rapidly after the use of cholekinetics), possibly - distal hyperhidrosis, pathological dermographism, predisposition to arterial hypertension, functional systolic murmur.

Clinical syndromes: dyspeptic, painful, astheno-vegetative, cholestasis.

The most significant in FDBT is pain. Its characteristics:

Biliary pain - episodes of pain localized in the epigastrium or right upper quadrant of the abdomen, lasting at least 3 months during the last six months, characterized by the following symptoms:

1. The duration of episodes is 30 minutes or more.
2. Recurrent symptoms that occur at different intervals (not daily).
3. The pain gradually reaches a constant level.
4. The pain is moderate or severe, disrupting daily activities or forcing you to go to the emergency department.
5. The pain does not decrease after the act of defecation.
6. Pain does not decrease when changing body position.
7. Pain does not decrease after taking antisecretory drugs.
8. Excluded organic pathology, which explains the above symptoms.

Additional criteria - a combination of pain with one symptom or more of the following:

• Pain may be associated with nausea and vomiting.
• Pain may occur in the back and / or right shoulder blade.
• Pain may help the patient to wake up at night.

Diagnosis: the diagnosis of FDBT (dyskinesia, or biliary tract dysfunction) is determined by a set of complaints, clinical and anamnestic data, laboratory and instrumental research methods.

Laboratory tests:

1. Coprogram - increase in neutral fat, a significant increase in the amount of fatty acids, intracellular starch, fiber (lack of bile secretion).

2. Biochemical examination of blood serum - increase in cholesterol, alkaline phosphatase, GGTP, total bilirubin due to the direct fraction (possible changes in gallbladder hypofunction, SFO dysfunction). Amylase / lipase levels within normal limits.

Instrumental research:

1. Ultrasound examination.

Dynamic ultrasound cholecystography using cholekinetic breakfast (egg yolks, sorbitol solution, hofitol solution) is used to assess the motor function of the gallbladder and the tone of the biliary tract sphincters. Measurement of gallbladder (GB) volume is performed on an empty stomach and after stimulation after 5, 15, 30, 40, 60 minutes. At ultrasound cholecystography phases of reduction of a gall bladder are registered. The first phase lasts 4-6 minutes, due to the state of the SFO, the prolongation of this phase indicates the spasm of the SFO. The second phase lasts about 15 minutes, due to the state of the SFO, the volume of GB decreases by 29-31%. The third phase lasts up to 30 minutes, due to the condition of the Lutkens sphincter, the volume of GB is further reduced by 30-35%. The fourth phase - further reduction of JM by 33 % - 65% compared to the initial volume. In the hyperkinetic type of GB dysfunction, the volume of the gallbladder is reduced by more than 65% for 60-90 minutes. after taking cholekinetics; in the hypokinetic type - less than 33%**.**

The size of the gallbladder should be estimated depending on the surface area of the body, not on the age of the child.

2. Duodenal probing followed by biochemical examination (concentration of total bile acids, cholesterol, phospholipids, bilirubin, determination of lithogenicity indices) and bile microscopy (detection of cholesterol crystals, calcium bilirubin) to assess the lithogenicity of bile.

3. Endoscopic retrograde cholecystopancreatography (ERCP) - enlargement of the common bile duct more than 10 mm and contrast delay in the common bile duct (GVHD) more than 45 minutes. indicates an increase in the tone of the SFD. It is carried out to clarify the dysfunctional disorders of the SFD and the diagnosis of mechanical interference in the WFD.

4. FEGDS - assessment of the mucous membrane of the esophagus, stomach, duodenum; duodenal papilla.

Diagnostic criteria for functional gallbladder disorder:

biliary pain in combination with normal liver enzymes, conjugated bilirubin, amylase / lipase (in the hypokinetic type of dysfunction may be moderate biochemical syndrome of cholestasis - increased serum cholesterol, alkaline phosphatase, GGTP, direct GGTP; gallbladder dysfunction according to ultrasound results.

Diagnostic criteria for functional biliary disorder of the SFO:

Biliary pain in combination with normal amylase / lipase levels; possible increase in the level of transaminases, alkaline phosphatase, direct fraction of bilirubin, over time associated with pain attacks; expansion of common bile duct after carrying out the test stimulated by fatty food - a sign of dysfunction of SFO (hypertonia).

Diagnostic criteria for functional pancreatic disorder:

Pain in the left hypochondrium, decreases when leaning forward (pancreatic type); girdle pain (combined type). The pain may be accompanied by the following symptoms: onset after eating; appearance at night; nausea and / or vomiting. Pain in combination with increased serum amylase / lipase levels, possible biochemical signs of cholestasis. Signs of SFO dysfunction (hypertonia) according to ultrasound results. Based on the study of biochemical parameters (level of liver enzymes, conjugated bilirubin, amylase / lipase), the level of functional disorders can be assessed.

Characteristics of therapeutic measures.

1. Dietary treatment: the organization of a food mode, 4 - 5 meals are shown, products with cholekinetic action at FDBT with hyperfunction are excluded; at FDBT with hypofunction it is expedient to use products with cholekinetic action.

2. Drug therapy.

A. *Hyperkinetic (hypertensive) type:*

- sedatives - phytopreparations, homeopathic remedies, sodium bromide, tranquilizers in age doses; (duration of the course and the choice of drug depends on the severity of neurological disorders);
- cholespasmolytic drugs: drotaverine at a dose of 1-1.5 mg / kg 3 times a day, pinaverium bromide 50 - 100 mg 3 times a day for children over 12 years, gimecromon for children over 5 years 200 - 600 mg per day in 3 doses, mebeverine to children from 12 years on 200 mg 2 times a day, prifinium bromide from the moment of a birth. Course duration 7 - 15 days; if necessary, extend the course, the drug must be changed;
- choleretics (drugs that stimulate the synthesis of bile): true (stimulating the synthesis of bile acids) - herbal and homeopathic remedies - alcohol 1-2 tablets three times a day after meals, flamingo 1/2 tablet three times a day 30 minutes before food, berberine 0.005-0.01 g three times a day before meals, nicodine 0.5-1 g three times a day before meals, cholenzyme, oxafenamide, febichol ; hydrocholeretics (stimulating the synthesis of the water component of bile) - mineral water 3 - 5 ml per kg of body weight three times a day, valerian drugs (age doses). Drugs are prescribed for a course of 10-14 days.
- physiotherapeutic procedures: thermal procedures (ozokerite, paraffin applications, diathermy to the right hypochondrium), induction of thermia, electrophoresis with antispasmodics to the right hypochondrium, ultrasound;
- balneotherapy - mineral waters of low mineralization and low z a saturation (Berezovskaya, Naftusya, etc.) 3-5 ml / kg of mass per 1 reception, 3 times a day for 1-1.5 months;
- phytotherapy (choleretic fees , mainly choleretic action).

B. *Hypokinetic (hypotonic) type:*

- tonics - tincture of ginseng, Chinese lemongrass, Eleutherococcus (age doses);
- choleretics (including drugs containing bile extract. Course duration 2 - 3 weeks.
- cholekinetics (drugs that stimulate the contraction of the gallbladder): artichoke leaf extract 1/4 - ½ tsp. 3 times a day, magnesium sulfate 25% for children under 3 years - 1 tsp., 3 - 7 years for 1 dec. l., 8 - 14 years 1 tbsp. 2 - 3 times a day; sorbitol 20% solution, xylitol 20% solution (0.1-0.2 g per kg body weight per reception) 2-3 times a day, galsten (age doses), vegetable oils (sunflower, corn, olive), flax seeds;
- prokinetics: domperidone for children from 3 to 12 years at 0.25 - 0.5 mg / kg body weight, children over 12 years at 10 mg 3 times a day for 15 - 30 minutes. before eating;
- tubage 2 times a week № 5 - 7 at the hypotonic form with mineral water (average mineralization) on 100 - 200 ml on reception, 25% solution of magnesium sulfate on 20 - 50 ml, 10-20% solution of sorbitol or xylitol on 50-100 Jr.
- physiotherapeutic procedures: electrophoresis of magnesium sulfate in the area of the right hypochondrium, sinusoidal modulated currents with a solution of mud, electrical stimulation of the gallbladder;
- balneotherapy: mineral waters of average mineralization and average gas saturation (Luzhanskaya, Morshinskaya, Polyana Kvasova, etc.) on 3-5 ml on 1 kg of weight on reception, 3 times a day within 1 month;
- phytotherapy (choleretic fees with choleretic, cholekinetic action).

Duration of inpatient treatment: 2 weeks (possible treatment in a day hospital or outpatient).

Purpose of treatment:

1) Stop the symptoms of functional disorders of the biliary tract.
2) Normalize the kinetic and tonic function of the biliary tract.
Requirements for treatment results: no clinical manifestations and ultrasound signs of FDBT.
Dispensary supervision. 3 years outside the paroxysmal period. It is possible to remove from dispensary supervision in the absence of pathological changes after laboratory and instrumental inspection.
Examination by a pediatric gastroenterologist 2 times a year, pediatrician - 2 times a year; otolaryngologist, dentist - if necessary.
The scope of control and diagnostic examinations:
coprogram - once a year, analysis of feces for worm eggs and protozoa - once a year, ultrasound of the abdominal cavity once a year, duodenal sounding and biochemical examination of bile - once a year (if indicated).
Anti-relapse treatment: 2 times a year (in the first year), then - once a year. The amount of treatment depends on the period of the disease and the child's condition (diet therapy, balneotherapy, phytotherapy, exercise therapy, physiotherapy).
It is recommended to carry out sanatorium treatment in 3-6 months after exacerbation. Spa-mud resorts are recommended.

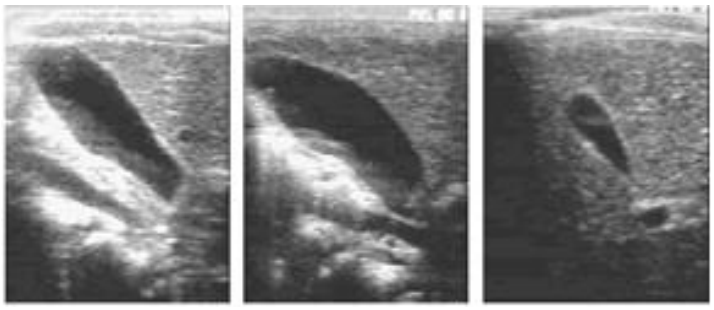
Addition

Studies have shown that uncoordinated, insufficient or excessive contraction of the gallbladder and its sphincters, which is controlled by the autonomic nervous system, is not always accompanied by clinical symptoms. It occurs in healthy children and is the limit of normal and pathology, but the long-term existence of dysfunction usually causes morphological changes. Violation of the bile passage occurs due to a complex set of regulatory effects, both neuroendocrine (gastrin, secretin, glucagon, etc.) and purely nervous (vagosympathetic and interoceptive effects).

According to the analysis of indicators of dynamic echocholecystography, depending on the state of motility of the biliary tract in healthy children, the following types were identified:

I - changes in the motility of the biliary tract are absent (normotonic-normokinetic type);

II - isolated increase in the tone of the sphincter of Oddi (hypertensive-normokinetic type);
III - increase in the tone of the sphincter of Oddi together with increased contractile function of the gallbladder (hypertensive-hyperkinetic type) - Figure 1;

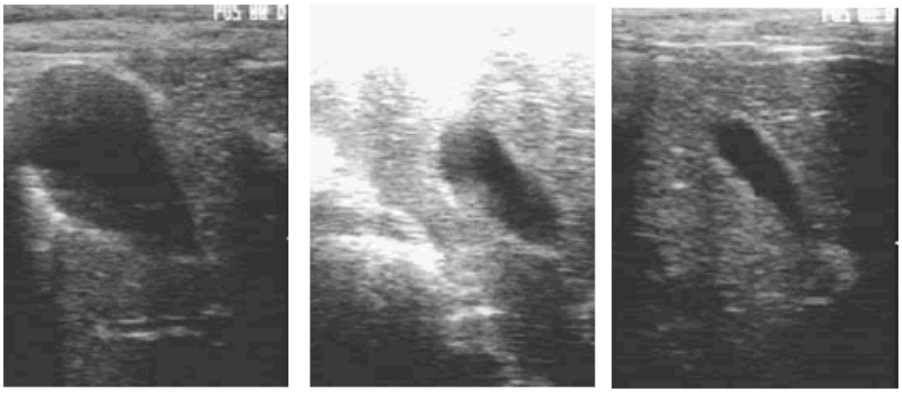


initial at 5 minutes at 60 minutes

hypertensive-hyperkinetic type of contraction of the gallbladder

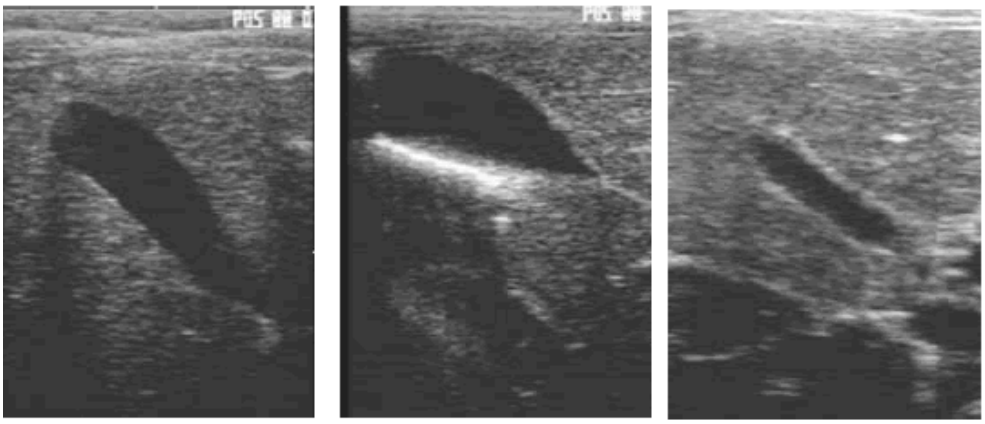
IV - isolated decrease in the tone of the sphincter of Oddi (hypotonic-normokinetic type);

V - decrease in the tone of the sphincter of Oddi together with increased contractility of the gallbladder (hypotonic-hyperkinetic type) - Figure 2



initial at 5 minutes at 60 minutes
hypotensive-hyperkinetic type of contraction of the gallbladder

VI - isolated increase in the contractile function of the gallbladder (normotonic-hyperkinetic type) - Figure 3



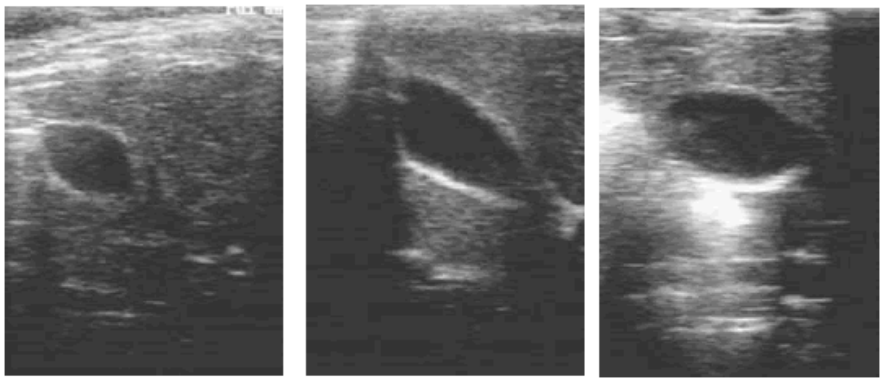
initial at 5 minutes at 60 minutes
normotensive-hyperkinetic type of contraction of the gallbladder

VII - isolated increase in the tone of the sphincter of Oddi at a later date (at the 15th minute; delayed hypertensive-normokinetic type);

VIII - increase in the tone of the sphincter of Oddi at a later date (15th minute) with increased contractile function of the gallbladder (delayed hypertensive-hyperkinetic type).

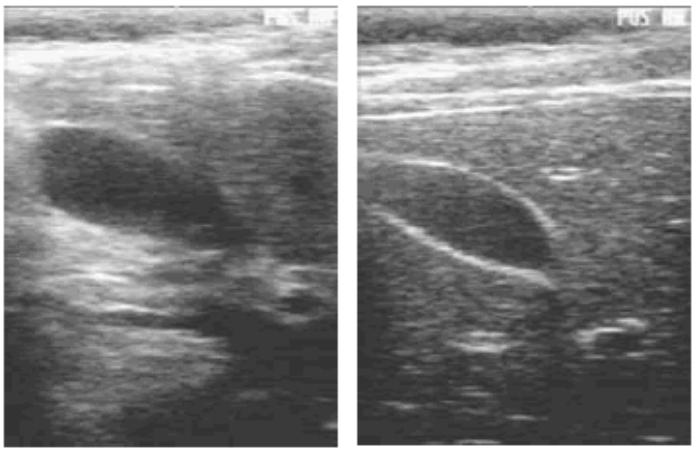
It should be noted that 16.6% of children in this group had a paradoxical reaction to the load of a trial breakfast - instead of reducing the sphincter of Oddi and increasing the volume of the gallbladder, there was a weakening, its volume at the 5th minute of observation decreased. Delayed spasm of the sphincter of Oddi was observed in types VII and VIII. The increase in gallbladder volume occurred at the 15th minute (16.7% of children). This allowed us to distinguish these types separately, as a variant of the individual norm.

In children with biliary dysfunction, you had two more variants of gallbladder contractility: IX - increase in the tone of the sphincter of Oddi together with a reduced ability of the gallbladder to contract (hypertensive-hypokinetic type) - Figure 2, 4.



initial at 5 minutes at 60 minutes
hypertensive-hypokinetic type of contraction of the gallbladder

X - isolated decrease in the ability of the gallbladder to contract (normotonic-hypokinetic type) - Figure 5.



initial at 90 minutes
normotensive-hypokinetic type of contraction of the gallbladder

Some of the identified types of motility of the biliary tract are similar to the manifestations of biliary dysfunction, but do not disrupt the passage of bile and are due to the peculiarities of autonomic regulation inherent in this age.

These data indicate that the prognostic criteria for prenosological disorders of the bile ducts are the deadline for gallbladder contraction and the presence of hypokinetic motility, and a marker of impaired biliary tract in primary school children is not only the condition of the sphincters, but the gallbladder, its hypokinetic type contractility. Hypertensive-hypokinetic and normotonic-hypokinetic types of contractility are not observed in healthy children. They are unique to children with biliary dysfunction.

Determining the value of the contractility of the gallbladder for different types of its motility significantly improves and accelerates the interpretation of the results of ultrasonography. It is calculated as the ratio of the maximum volume of the gallbladder to the minimum that was recorded during the cholekinetic test (CC, %):

CC = Vn / Vm, where KS is the coefficient of contractility of the gallbladder, Vi is the initial (fasting) volume, Vm is the minimum volume.

DIFFERENTIAL-DIAGNOSTIC CRITERIA FOR DISEASES OF THE BILIARY TRACT
(A.A. KOZLOVSKY, 2008)

Criterion	Form of dyskinesia		Chronic cholecystocholangitis
	HYPERKINETIC	HYPOKINETIC	
Anamnesis	Neurotic reactions, emotional stress, lability of the autonomic nervous system	Negative emotions, physical activity	Weakness, lethargy, signs of intoxication, polyhypovitaminosis
Family predisposition	+	+	+
Seasonality of exacerbation	Autumn-spring period	Not typical	Autumn-spring period
Duration of the disease	Up to 1 year	1-1.5 years	1-1.5 years
The nature of the pain	Paroxysmal, intermittent	Aching, dull, constant	Paroxysmal, dull, constant
Relationship with errors in nutrition	30-40 minutes after eating a cold meal	After 1-1.5 hours after meals, especially fatty foods	1.5-2 hours after eating, especially fatty foods
Irradiation of pain	Not typical	Not typical	In the right shoulder and shoulder blade
Vomiting	+	+/-	+
Nausea	+	+	+
Constipation	-	+	+
Eructation	-	+	+
Muscle tension	-	-	+
Bladder symptoms	Expressed vaguely	Expressed vaguely	Positive
Enlarged liver	-	+/-	+
Intoxication	-	-	+
Changes in blood biochemical parameters	Missing	A slight increase in alkaline phosphatase activity	Increased activity of transaminases, alkaline phosphatase, bilirubin

Changes in the general analysis of blood	Missing	Missing	Leukocytosis, neutrophilia, increased shoe, anemia

Gallstone disease in children is a hereditary disease of the hepatobiliary system, which is based on a violation of the metabolism of cholesterol and (or) bilirubin, which is accompanied by a chronic inflammatory process with a progressive course, leading to sclerosis and gallbladder dystrophy and characterized by gallstones bladder and (or) in the bile ducts.

ICD-10 code:

K80 gallstone disease (housing, cholelithiasis).

K80.2 gallstone without cholecystitis (cholecystolithiasis).

K80.3 gallstone (choledocholithiasis) with cholangitis (not primary sclerosing).

K80.4 gallstone (choledocholithiasis) with cholecystitis.

K80.5 gallstone (choledocholithiasis) without cholangitis or cholecystitis.

K80.8 other forms of cholelithiasis

Classification

In Ukraine, the classification proposed by HH Mansurov (2002) is accepted as a basis. According to this classification, there are 4 stages of the disease:

Stage I - initial (pre-stone, physico-chemical):

A. Thick inhomogeneous bile.

B. Formation of biliary sludge (putty bile, microliths and their combinations).

Stage II - the formation of gallstones:

A. By localization (in the gallbladder, common bile duct, hepatic ducts).

B. By the number of concretions (single, multiple).

B. By composition (cholesterol, pigment, mixed).

D. According to the clinical course:

a) latent;

b) with the presence of clinical symptoms:

• painful form with biliary colic;

• dyspeptic form;

• under the guise of other diseases.

Stage III - chronic recurrent calculous cholecystitis.

Stage IV - complications (acute cholecystitis, edema of the gallbladder, fistulas, secondary dyspeptic cirrhosis, scar strictures).

Both clinical and anamnestic data of the disease and various laboratory and instrumental studies are important for the diagnosis of housing and communal services.

1. In the clinical analysis of blood leukocytosis, acceleration of ESR which is an indicator of inflammatory process and characteristic of III, IV stages of a disease is possible.

2. Biochemical examination of blood serum is characterized by signs of cholestasis - an increase in the level of total bilirubin due to the direct fraction, alkaline phosphatase, the level of total cholesterol, triglycerides, LDL.

3. Ultrasound finds echopositive inclusions in the gallbladder (sludge, microliths, stones).

4. Survey radiography of the abdominal cavity - in the presence of X-ray-positive stones allows you to visualize the shadows in the gallbladder.

5. Oral cholecystography, MRI are performed according to the indications as clarifying methods to visualize the bile ducts.

According to OV Tyazhka et al. (2014), it is important in the diagnosis of housing and communal services may be the determination of elevated levels of phosphatidylcholine in the blood with a simultaneous decrease in phosphatidylethanolamine, which indicates a violation of the permeability of cell membranes. The author also established threshold levels and ratios of serum and bile phospholipids, which allows to predict the probability of cholelithiasis.

Ultrasound is rightly considered the main and screening method in the diagnosis of housing and communal services in both children and adults. Its sensitivity in the localization of stones in the gallbladder exceeds 97%, and in the presence of acoustic shadow reaches 99%.

Treatment of children with gallstones depends on the stage of the disease and includes, according to the current protocol (2013), the following areas:

1. *Dietary correction* - table № 5 according to Pevzner with supplements in the form of vegetables, fruits and other functional foods (dietary fiber).

2. *Mode* - limitation of physical activity in stage II of the disease (formation of gallstones). Adherence to these measures is due to the fact that with increased physical activity may move the stones in the bile ducts, resulting in obstruction of their stones.

3. *Drug treatment.*

I stage of housing and communal services (initial) in the presence of dense inhomogeneous bile:

- real choleretics - drugs that stimulate bile secretion and the formation of bile acids: a) containing bile acids - hologon, alohol, liobil; b) drugs of chemical synthesis - tsikvalon, nicodine; c) herbal remedies - corn stigmas, hofitol, holosas; hydrocholeretics - increase bile secretion due to the aqueous component: mineral water, valerian preparations;
- cholekinetics - drugs that stimulate the contraction of the gallbladder: of plant origin - artichoke leaf extract 1 / 4-1 / 2 tsp. three times a day, turmeric root powder 50-100 mg three times a day; magnesium sulfate 25% for children under 3 years - 1 tsp, 3-7 years - 1 des. sp., 8-14 years - 1 tbsp. l. 2-3 times a day; 20% solution of sorbitol, xylitol (0.1-0.2 g per 1 kg of body weight per dose) 2-3 times a day, vegetable oils, flax seeds. The duration of the course of choleretic therapy is 2-3 weeks;
- litholytics - a suspension of ursodeoxycholic acid (UDCA) at a dose of 10 mg / kg (one tablespoon contains 250 mg of UDCA). At body weight up to 7 kg the daily dose is equal to 1/4 spoon, to 12 kg - 1/2 spoon, from 13 to 18 kg - 3/4 spoon, from 19 to 25 kg - on 1 spoon a day. The daily dose is divided into 3 doses, with priority given in the evening, before bedtime. The duration of the course of therapy is 1.5–2 months, repeated courses are conducted 2-3 times a year.

I stage of housing and communal services in the presence of microliths:

- choleretics;
- litholytic therapy lasting 6-12 months.

Stage II housing and communal services - the formation of gallstones:

- consultation and observation of the surgeon;
- strict bed rest;
- in an attack of *biliary colic:*

- heat on the right hypochondrium;

- analgesics: metamizole sodium (50% - 0.1 ml per year of life) and its combinations;

- antispasmodics: drotaverine (2% - 0.5–2 ml), papaverine (2% - 1–2 ml), platyphylline (0.2% - 1 ml); prifinium bromide (riabal) - from an early age at a dose of 1 mg / kg per day p / w, i / m, i / v;

- with prolonged pain - novocaine paranephric blockade;

- antibacterial therapy is indicated for severe intoxication and inflammatory reaction of peripheral blood. Drugs of choice are antibiotics that form a therapeutic depot in the gallbladder - a group of penicillins, macrolides, cephalosporins, in case of intolerance to these groups of antibacterial drugs - furazolidone, which accumulates in the bile in sufficient concentration for therapeutic effect. Antibacterial drugs are prescribed in standard age doses. The course of antibacterial therapy is 7-10 days;

- litholytic therapy lasting from 6 to 24 months. (before dissolving or reducing the size of concretions);

- hepatoprotectors (silymarin and other phytopreparations) in age doses are prescribed for a course of 2-3 months, repeated courses are conducted 2-3 times a year;

- enterosorbents (enterosgel, etc.) for 10–14 days, 3–4 courses per year.

The pancreas is the largest and most important gland of the digestive system and at the same time an important endocrine gland, which is involved in the regulation of carbohydrate metabolism.

PANCREATITIS is a disease of the pancreas of inflammatory and degenerative nature.

The basis of the pathological process is the enzymatic digestion of its tissues (autolysis) due to intra-organ activation of enzymes. This process occurs under the influence of damaging agents: mechanical, metabolic, toxic, allergic, vascular, neurotrophic on the background of high pressure in the pancreatic ducts.

Morphological substrate of pancreatitis: edema, local cellular infiltration of tissue, destructive processes, vascular damage, sclerosis, parenchymal atrophy.

ACUTE PANCREATITIS (AP)

Etiology. The most common causes of AP in children are the following factors:

I. Factors that directly damage the parenchyma of the software:

- viruses (mumps, hepatitis virus, Coxsackie B enterovirus, infectious mononucleosis, rubella, chicken pox, herpes, cytomegalovirus, measles, influenza);

- bacterial infections: pseudotuberculosis, dysentery, salmonellosis, peritonitis, sepsis;

- blunt trauma of the pancreas as a result of a strong blow to the abdomen (often cycling, sports).

II. Obstructive disorders that lead to increased pressure in the pancreatic ducts:

- anatomical anomalies (annular or lobular pancreas, choledochal cyst);

- changes in the common bile duct or Fater's nipple (papillitis, Oddi's sphincter hypertension, choledocholithiasis, choledochal cyst, choledochal stricture, etc.);

- pathology of the duodenum: duodenitis, duodenostasis, partial duodenal obstruction. Increased pressure in the duodenum, exceeding the resistance of the sphincter of Oddi, can lead to pathological reflux - duodenobiliary and duodenopancreatic, the latter also contributes to intraductal activation of enzymes, stagnation and damage to the gland. In chronic duodenitis, especially atrophic, the death of some endocrine cells of the duodenum (S-cells that produce secretin and L-cells that secrete cholecystokinin) and violation of endocrine regulation of the pancreas is possible. In particular, with secretin insufficiency, the pressure in the duodenum increases, there is a spasm of the sphincter of Oddi, the pressure in the pancreatic ducts increases and the secretion of the liquid part of the juice is suppressed in comparison with the secretion of enzymes. This increases the viscosity of the secretion and the concentration of protein, which leads to the intraductal formation of protein precipitates (protein plugs);

- helminthiasis (obstruction of the duodenal papilla by roundworms, opisthorchiasis, fasciolosis, clonorchosis);

- hepatobiliary pathology. In patients with inflammatory biliary diseases, bile acquires more pronounced aggressive properties: increased concentration of unconjugated bile acids, saturation with lipid peroxidation products. Therefore, when biliary reflux occurs in these patients, the damaging effect on the pancreas is significantly enhanced.

III. Dysmetabolic causes, first of all - *hypercalcemia* (consequence of hyperparathyroidism or hypervitaminosis D). Increasing the calcium content in acinar cells stimulates the secretion of enzymes, but not bicarbonate and the liquid part of the juice, so the viscosity of the secretion increases, the outflow deteriorates, calcium is deposited in the form of phosphate (stones in the ducts). Intraorganic activation of trypsin by calcium is possible.

IV. Acute circulatory problems *in* (thrombosis, embolism, prolonged spasm of blood vessels of the pancreas) can occur when:

- essential hyperlipidemia - there is obstruction of the vessels of the gland by drops of fat and atheromatous plaques;

- systemic diseases (nodular periarteritis, Kawasaki disease, necrotic angiitis, other vasculitis).

V. Toxic and drug lesions:

- poisoning by lead, mercury, arsenic, phosphorus, etc.;

- drugs: L-asparaginase, azathioprine, hypothiazide, furosemide, estrogens, metronidazole, tetracyclines, sulfonamides (sulfasalazine), valproic acid, high doses of glucocorticoids.

VI. Allergies.

- possible development of AP on the background of food allergies, as well as after vaccinations. At first there is a sensitization of an organism, and then development of antibodies to fabric of a pancreas. At the subsequent receipt of an allergen the anaphylactic organ-specific reaction like the Arthus phenomenon with defeat of a pancreas develops;

- alimentary factor in its pure form can cause damage to the pancreas only in chronic protein deficiency in food (kwashiorkor). Excessive consumption of fatty, fried foods may be only an additional factor that provokes the manifestation of the disease against the background of other reasons listed above. In 25% of children with AP, the etiology can not be established.

Pathogenesis. Due to the increase in pressure in the duodenum, duodenal juice containing enterokinase enters the duct of the pancreas, where it converts inactive trypsinogen into active trypsin. This leads to enzymatic destruction of acinar cells of the body. With the damage of acinar cells and the release of cellular cytokinases, another mechanism of activation of proteolytic enzymes - trypsin, kallikrein, carboxypeptidase, elastase. There is a "deviation" in the blood. Activation of kallikrein causes the development of acute interstitial edema of the pancreas. The amount of active trypsin exceeds the ability of the pancreas to produce a trypsin inhibitor. The imbalance between trypsin and its inhibitor in favor of the former promotes the activation of other proteolytic proenzymes: chymotrypsinogen and procarboxypeptidase. As a result of these changes there are processes of autolysis of pancreatic tissue.

Proteolysis is accompanied by the accumulation of histamine, kinins (bradykinin, kalidin) and progressive changes in hemodynamics. The coagulating and fibrinolytic properties of blood are disturbed, which causes thrombosis and hemorrhage. During obstruction of the pancreatic duct, the juice of the pancreas is not evacuated to the duodenum, but enters the parenchyma and stroma of the organ. At first aseptic necrosis of a pancreas develops, further, in case of accession of an infection, transformation into purulent pancreatitis can occur.

The accumulation of excessive amounts of active enzymes and proteolysis products in the blood leads to their penetration through the serous membrane into the abdominal cavity, intoxication of the body and the development of poly-organ failure syndrome.

Recently, the influence of lipase, phospholipase-A, elastase is of great importance in the pathogenesis of pancreatitis.

A certain place in the mechanism of disease development belongs to the vascular component. Thus, during diseases of the gastroduodenal area, reflex spasms of the blood vessels of the pancreas are often noted, which leads to a violation of local trophism and even necrosis of the gland tissue. The possibility of infection in the pancreas by hematogenous and lymphogenic routes has been confirmed. At the same time, the hematogenous and lymphogenic routes can spread active pancreatic enzymes in the body, causing the development of acute mesadenitis. Penetrating directly into the surrounding tissues with a large number of nerve plexuses, enzymes cause pain, neuro-trophic disorders.

Among the theories of the development of pancreatitis, an important place is occupied by the theory according to which the activation of the trypsinogen-trypsin system plays a major role in the pathogenesis of this disease. Under the conditions of activation of the kallikrein-kinin system due to the formation of polypeptides of bradykinin and kalidin, vasodilation occurs, the permeability of vascular walls increases, then trophism and metabolism are disturbed. Together with the considered processes the migration of leukocytes into interstitial tissue increases, their disintegration stimulates the formation of leukokinins, which aggravates circulatory disorders, changes in glandular tissue.

It is believed that acute pancreatitis may occur due to autodigestion, which develops against the background of functional overload of the pancreas (overeating). There is also a theory that the main pathogenetic link in the occurrence of acute pancreatitis is decompensated acidosis and hypercorticism (spontaneous or iatrogenic). As a result, there are significant biochemical changes in acinar cells, proteolytic enzymes are released. Injury, infection, disruption of lymph flow cause the penetration of enzymes into the pancreas.

Pathomorphology. In the initial stage of morphological changes, damage to acinar cells is accompanied by edema of interstitial tissue. The gland increases in size, swells, the interstitium is infiltrated with leukocytes (interstitial or edematous-serous pancreatitis). Later, on the background of mucosal edema, hemorrhagic exudation may develop (erythrocyte joins leukocyte infiltration). The gland becomes denser, becomes brown (serous-hemorrhagic pancreatitis). With timely and adequate treatment, the process can stop at the stage of edematous-serous pancreatitis.

In case of disease progression, pancreatic necrosis develops. It is usually mixed - hemorrhagic and fatty. At the same time the volume and density of a pancreas considerably increase. Large foci of necrosis can result in the formation of cavities (pseudocysts), and their infection leads to the development of purulent-necrotic pancreatitis. The course of pancreatic necrosis is usually accompanied by pronounced hemorrhagic changes not only in the pancreas, but also in the extraperitoneal tissue and remote areas of the abdominal cavity.

Classification. Most researchers distinguish between acute and chronic pancreatitis; primary and secondary - by origin (reactive and intoxication).

There are 2 stages of AP on the basis of clinical and morphological data:

- interstitial (edematous-serous);
- destructive (pancreatic necrosis).

Clinic. There is a sudden paroxysmal pain in the upper abdomen above the navel, under the left rib, sometimes in the epigastrium, forcing the child to take a forced position (bent on the left side or abdomen). Often the pain as a belt covers the abdomen, sometimes radiating to the left back, chest, left arm, shoulder, leg. Pain syndrome may be accompanied by dyspeptic symptoms: flatulence, constipation or weak stools, nausea, vomiting. Characteristically, vomiting does not reduce the intensity of pain. The general condition is significantly disturbed. In children, increased mobility or immobility. The skin is pale, grayish-cyanotic. Suffering facial expression, dry baked lips, periorbital cyanosis. Sometimes you can see a small hemorrhagic rash on various parts of the body (spotted or spotty-papular rash, similar to a cow). Pulse frequent, weak filling. Arterial dystonia is possible. Subfebrile or short-term febrile body temperature. Breathing is shallow, slightly more often than normal. On superficial palpation, you can detect muscle tension that is located across the navel (Kerte's symptom), tension of the left oblique abdominal muscle in the form of a painful condensed cord extending from the middle of the left costal arch to the outer edge of the rectus abdominis. Pain at the level of 3 - 5 cm above the navel on the outer edge of the rectus abdominis (Duck point). Many patients have a positive Voskresensky symptom (absence of aortic pulsation in the area of the projection of the pancreas due to its edema). Pain is often observed on palpation in the left costal-vertebral angle (Mayo-Robson symptom).

Along with local symptoms, children have symptoms of damage to other organs and systems - a plurivisceral symptom complex develops. Many patients with acute pancreatitis suffer from indigestion in the small intestine. Clinically, this is manifested by flatulence, weakening of bowel movements, an increase in the frequency of bowel movements to 3-4 times a day, coprological changes - steato- and creatorrhea.

Many patients with AP have lesions of several organs of the digestive system or other organs and systems, which may be manifested by polymorphic symptoms.

At the beginning of the disease there are tachycardia, weakened heart tones, hypotension. In the future, you can detect bradycardia, heart rate disorders. Possible toxic damage to the heart muscle, which is determined by instrumental examination. If on the 1st day the size of the liver may be within normal limits, there is only some sensitivity of its edge during palpation, then on the 2nd day show a significant increase in the size of the liver due to its toxic lesions, subicteric or icteric skin, sclera. The urinary system is also affected - there is proteinuria, minor microhematuria, decreased urine weight in patients with moderate disease. In severe cases, anuria, hyperazotemia are observed.

Further development of the pathological process leads to the appearance of retroperitoneal syndrome - flatulence, vomiting, angina, various hemorrhagic effusions into the pleural cavity. Lesions of the adrenal glands are accompanied by hypotension, lethargy, hypothermia. As a consequence of intoxication in 2 - 3 days there are cerebral symptoms in the form of excitement or depression, dizziness, prostration.

The results of laboratory tests.

1) neutrophilic leukocytosis, eosinophilia, thrombocytopenia, increased ESR;

2) increased activity of pancreatic enzymes (due to edema, destructive processes, the evacuation of enzymes in the duodenum is disrupted, which in turn leads to their "deviation" into the blood, urine, effusion) - blood and urine amylase - is observed 2-3 hours before pain manifestations and can last up to several days. Especially significant is the increase in pancreatic isoamylase (with an increase in the content of pancreatic isoamylase indicates an inflammatory process in the pancreas, a decrease - on the destructive changes in it);

3) increase in lipase of blood and urine in 2-3 times as display of deeper defeat of gland with disease progression in 5-7 days. Lipasemia (lipazuria) is pathognomonic for pancreatitis and indicates destructive changes in the pancreas;

4) increase in trypsin activity by 10 - 40 times (using radioimmunological methods in the presence of trypsin inhibitor);

5) decrease in the level of kallikreinogen in the blood serum;

6) hypo- or hyperglycemia;

7) reducing the calcium content below 1.75 mmol / l;

8) X-ray examination reveals blurred contours of the lumbar muscle on the left. Contrast radioscopy indicates a shift of the stomach to the left, up and forward, and the colon - down. Due to the increase in the size of the pancreas there is a displacement (unfolding) of the loop of the duodenum, violation of the relief of the medial wall of its descending part, sometimes its narrowing;

9) Ultrasound - in the acute period of pancreatitis reveal an increase in size and violation of the echostructure of the pancreas.

The diagnosis of acute pancreatitis is based on the anamnesis of the disease, the positive symptoms of pancreatic lesions, cytolysis syndrome (primarily hyperamylasemia and hyperlipasemia), instrumentally detected changes in the parameters and structure of the gland.

Complication. Early: shock, liver failure, renal failure, DIC syndrome, bleeding, diabetes. Late: pseudocysts, fistulas, abscesses and phlegmon of the pancreas, peritonitis.

Differential diagnosis of AP is carried out with many diseases that are accompanied by intense abdominal pain: perforation of the ulcer, acute intestinal obstruction, hepatic colic, acute appendicitis, acute cholecystitis, etc.

Treatment. Suspicion of AP is an indication for emergency hospitalization. The scope of prehospital therapy for AP includes the use of analgesics (analgin, baralgin, tramal) and antispasmodics (no-spa, papaverine, atropine, platyphylline).

Diet. In the first 3 days - hunger, which provides maximum sparing of the pancreas, only fractional drinking of alkaline mineral waters is allowed. At heavy destructive forms exclude also liquid use, put a constant nasogastric tube for suction of gastric contents. When the condition improves, the diet is expanded very gradually: first, grated porridge is introduced in water, then in milk, slimy soup, weak tea, crackers, omelet, then cheese, milk jelly, jelly in milk, white bread. From the 7th day, gradually introduce vegetable and mashed potatoes, steamed meatballs, boiled fish, curd and fruit paste. In the future, some fat restriction (up to 80-90%), easy to digest carbohydrates and a moderate increase in protein (up to 130%) are recommended. Exclude juicy and choleretic products and dishes: raw fruits and vegetables, juices, vegetable fats, meat and vegetable broths, fried, spicy dishes, mushrooms, coffee, chocolate. Dishes that contain natural inhibitors of enzymes - egg white, oatmeal, soy, potatoes. The diet should be strictly adhered to for 6 months, then the principle of mechanical and chemical sparing is preserved, fried, spicy, sour food is excluded.

In the period of exacerbation against the background of restrictions on oral nutrition, it is very important to prescribe parenteral and enteral nutrition. Mixtures of amino acids (aminosteryl, aminosol, alvesin, polyamine and others) are administered intravenously (30 - 40 drops per 1 min). It is recommended to add electrolyte solutions to them, taking into account the acid-base balance. Along with them, fat emulsions are used to bind active lipase and fill the deficiency of fatty acids in the blood at the rate of 12 g of fat per 1 kg of body weight: 10-20% solutions of intralipid or lipofundin intravenously with heparin at a rate of 20-30 drops per 1 min.

Mixtures of amino acids can be administered enterally (intraduodenally and even intraeminally) in the morning on an empty stomach, in a state heated to 37 ° C, in a volume of 50 to 200 ml, depending on age every other day, for up to 5-7 procedures. This route of administration of amino acids does not cause side effects, is easily tolerated by patients and has a pronounced therapeutic effect.

Enteral nutrition is carried out with mixtures based on protein hydrolysates with a high degree of hydrolysis with the inclusion of medium-chain triglycerides, low-fat dairy products and with a modified fat component. Gradually switch to a mixture with a low degree of hydrolysis (Table), because these products are absorbed in the intestine without prior fermentation. They can be administered intraduodenally in a warm state through a tube.

Drug therapy

I. Analgesics:

- analgesics: baralgin, analgin, tramal, in severe cases, you can prescribe narcotic analgesics - promedol 2% (but not morphine, because the latter causes spasm of the sphincter of Oddi);

- antispasmodics: papaverine, no-shpa, halidor;

- cholinolytics with predominant antispasmodic action: platyphylline, buscopan, metacin.

The method of administration and dose depend on the degree of pain and severity of the condition - in mild forms appoint oral administration 3 times a day, in severe cases - intravenous drip in the form of a cocktail, which includes analgesic, antispasmodic, 0.5% solution of novocaine (15 - 20 ml) in 0.9% sodium chloride solution.

It would be good to supplement this therapy with antihistamines and antiserotonin drugs (tavegil, peritol, suprastin). This helps reduce pancreatic edema and potentiates the analgesic effect.

II. Inhibition of functional activity of the pancreas. Achieved as a direct effect on its exocrine function, and indirectly - by affecting gastric secretion, as hydrochloric acid is a natural stimulant of the pancreas.

- cholinolytics: 0.1% solution of atropine 0.1 - 0.2 ml twice subcutaneously, gastrocepin, pirencepin, telenzepine 1 tablet 3 times 30 minutes before meals;

- antacids: almagel, maalox, magaldrate 1 tablespoon or tablet every 2-3 hours (6-7 times a day);

- H_2-histamine blockers, II-III generation (ranitidine or famotidine) 1-2 tablets per day;

- Na-K-ATPase inhibitors (omeprazole) 1 capsule once a day;

- somatostatin preparation - sandostatin (octreotide). Depending on age, prescribe 25-50-100 mcg 2-3 times a day intravenously or subcutaneously for 5-7 days. The drug is used in severe forms of pancreatitis.

III. Reduction of enzymatic toxemia (performed in severe forms of acute pancreatitis):

- proteolysis inhibitors: contrikal, trasilol, gordox, zimofen - inhibit kallikrein, trypsin, chymotrypsin, plasmin by forming an inactive complex. The dose is selected depending on the degree of enzyme and the condition of the child. Kontrikal is usually first prescribed at 5000-10000 IU IV drip in a solution of 0.9% sodium chloride. Then the dose is increased to 500-1000 IU / kg. After reducing the enzyme, the dose is reduced and canceled.

- detoxification therapy is carried out in severe forms of AP: glucose-saline solutions (5% glucose solution with 0.9% sodium chloride solution), 10% albumin, plasma, vitamins C, B_6 in age dosages.

- in severe cases, plasmapheresis or hemosorption is indicated.

IV. Against the background of drug suppression of pancreatic function, enteral nutrition is combined with the appointment of small doses of pancreatic enzymes that do not contain bile (pancreatin, Mezim-forte, pancitrate, Kreon) 1 tablet 3 times a day after or during meals.

Activity and combination of some enzyme preparations

Characteristic	Licrease	Pancreatin	Pancitrate	Festal	Mezim-forte	Kreon
Form	Micro-granules	Tablets	Micro-granules	Tablets	Tablets	Micro-granules
Lipase	12000	12500	10000	6000	3500	8000
Amylase	14000	12500	9000	4500	4200	9000
Proteases	660	1000	500	300	1950	450
Packaging	2 blisters of 12 drops	Vial of 100 tablets	50 capsules	10 blisters of 10 tablets	40 blisters of 20 tablets	2 blisters of 10 capsules

The effectiveness of enzymes and dose adequacy is assessed by the dynamics of clinical signs (disappearance of pain and dyspeptic syndromes), normalization of the coprogram and the level of duodenal enzymes, blood and urine, positive dynamics of the child's body weight. It should

be noted that in children with AP and hyposecretory type of function, despite clinical improvement, recovery of exocrine function does not occur, so the question of enzyme replacement therapy is decided strictly individually. Prolonged uncontrolled use of these drugs inhibits its own enzyme production by the feedback mechanism.

V. For the prevention of purulent complications in severe forms of pancreatitis prescribe broad-spectrum antibiotics (cephalosporins, aminoglycosides). Purulent complications (abscesses, phlegmon, peritonitis) are indications for surgical treatment of AP.

VI. In the period of abatement of acute phenomena continue taking pancreatic enzymes, prefer an intermittent course: 2 weeks of reception, 10 days break.

Shown phytotherapy: celandine, calendula, marshmallow root - 1 tablespoon collection per 1 cup of water, drink 1/3 cup 3 times 15 minutes before meals 1 month, then - low-mineralization mineral water (Borjomi, Slavyanivska , Essentuki №4, Myrhorodska, Naftusya) in a warm state without gas 50-100 ml 5-6 times a day.

Dispensary observation after AP is carried out for 3 years: 1st year - 4 times, 2nd year - 2 times a year. In addition to the clinical examination, it is recommended to examine the urine for amylase, coprogram, ultrasound. After 3 years, the child is transferred to the risk group for chronic pancreatitis with a single annual examination for 2 years.

ICD-10 code: K 86.1 Chronic pancreatitis

Chronic pancreatitis (CP) is a polyetiological disease with a phase-progressive course, focal or diffuse-degenerative, destructive changes of acinar tissue, exocrine and endocrine functions.

Epidemiology

Pancreatitis, according to modern data, in the structure of diseases of the digestive system in children is from 15 to 25%. In Ukraine, the incidence of pancreatitis reaches 3.1 ‰ of the pediatric population (Unified clinical protocols of medical care for children with digestive diseases, 2013). Boys and girls get sick with the same frequency.

Etiology

According to the origin of CP is divided into primary and secondary. In childhood, the most common secondary pancreatitis, developing against the background of diseases of other digestive organs. In the development of secondary pancreatitis, according to GV Rymarchuk (2003), the pathology of the duodenum and biliary tract occupies a significant place. The main factors in the development of pancreatitis of biliary origin are abnormalities in the development of the gallbladder and biliary tract (52%), chronic cholecystitis (42%) and gallstone disease (6%). At the same time, foreign authors (Michael H. Ma et al., 2012) in the study found that the most etiologically significant factor in the development of pancreatitis associated with biliary pathology is housing (76%), with 21% of cases of pancreatitis developed against the background of biliary sludge. Among other reasons, the authors distinguish the following: dysfunction of the sphincter of Oddi (5%), bifurcated pancreas (5%), other abnormalities of the pancreatobiliary system (pancreatic cyst, stenosis of the pancreatic duct, annular pancreas, cyst% choledochus).

Since CP is usually a consequence of acute pancreatitis, most of the etiological factors of the latter can be attributed to the causes of CP. These include the following factors:

- acute viral and bacterial infections (SARS, mumps, viral hepatitis, enterovirus infections, dysentery, scarlet fever, sepsis);
- helminthiasis (opisthorchiasis, strongyloidiasis, ascariasis);
- chronic inflammatory bowel disease (Crohn's disease, nonspecific ulcerative colitis);
- systemic processes (connective tissue diseases, endocrine pathology, hyperlipidemia, hypothyroidism, hyperparathyroidism, hypercalcemia of various origins, chronic renal failure, cystic fibrosis);
- allergic pathology;

- abdominal injuries;
- toxic injuries, in particular, drug (tetracycline antibiotics, sulfonamides, diuretics, etc.).

Primary chronic pancreatitis occurs much less frequently (14%). It includes a hereditary form, as well as a congenital deficiency of enzymes - Schwachmann syndrome, Johansen - Blizzard syndrome and others.

Pathogenesis

In the development of CP in children there are three main pathogenetic variants: obstructive, immunopathological and dysmetabolic. The possibility of combining pathogenetic variants in one patient is allowed. Schematically, the main pathogenetic links in the development of CP are shown in Fig. 1.

Classification

According to the classification of CP proposed by RV Rymarchuk (1998), it is divided into:

- by origin - primary, secondary;
- by the nature of the course - recurrent, with constant pain, latent;
- by the period of the disease - exacerbation, subremission, clinical and laboratory remission;
- by severity - mild, moderate, severe;
- by functional state - hyposecretory, hypersecretory, obstructive, normal type of pancreatic secretion;
- by the nature of complications - calcification, false cyst, pancreatolithiasis, insufficiency of incretory function.

Clinical characteristics

The clinical picture of CP in children is characterized by significant variability of symptoms and gradual development.

The subclinical stage is characterized by deterioration of general health, decreased appetite, intermittent abdominal pain of a mild nature. During this period, there are signs of microcirculation disorders - acrocyanosis, ecchymosis, rarely hemorrhagic rash, increased fermenthemia.

In the transition to the stage of exacerbation there is a dominant syndrome of chronic pancreatitis - *pain*. The main criteria for pain in CP:

- Localization - epigastric region (77%), right and left hypochondrium (58 %), in young children - umbilical region (VG Maidannik, 2010).
- Progressive increase in pain.
- Irradiation to the back, left and right halves of the chest - shingles.
- Appearance after errors in food (fatty, fried, rough, cold food).
- Hunger pains at night (48%).
- Duration - from 1 to 4-5 hours, in some cases - up to several days.
- Stopped by taking analgesics and antispasmodics.
- Intensifies in the supine position.
- Weakens in a sitting position, with the torso tilted forward, on the side with the legs close to the abdomen.

Occurrence of a pain syndrome is connected with increase in pressure in pancreatic channels, an inflammation of intrapancreatic nerve trunks, stretching of a capsule of a pancreas, involvement in process of a duodenum and irritation of a solar plexus.

The next syndrome, characteristic of CP, is the *syndrome of dyspeptic disorders*, which is manifested by decreased appetite, nausea, periodic vomiting at the height of the pain attack, belching, heartburn.

In the initial stage of the disease, intestinal dysfunction is manifested by periodic constipation. During the manifestation, liquid defecation is characteristic, due to the development of the syndrome of impaired absorption, which is manifested by pancreatic steatorrhea with the appearance of "fatty" shiny feces.

Along with pain and dyspeptic syndromes, there are always signs of *chronic intoxication*: increased fatigue, general weakness, frequent headaches, emotional lability. In CP, there are signs of autonomic imbalance with a predominance of parasympathicotonia. There may be low-grade fever, tachycardia and low blood pressure. One third of children lose weight, which in some patients can reach 5-9 kg.

At objective inspection in the period of an exacerbation pain in certain zones of a stomach is defined by palpation: choledochopancreatic (Shoffar's triangle), epigastric, left hypochondrium. Characteristic of the appearance of a number of symptoms typical of exacerbation of CP:

1) Mayo-Robson symptom - pain on palpation in the left costal-vertebral angle;

2) Kacha symptom - the zone of hyperesthesia in the left hypochondrium, corresponding to the zone of innervation of the VIII thoracic segment on the left;

3) Grotto's symptom - thinning of the subcutaneous fat fold on the left in the area of the projection of the pancreas.

Diagnosis

Determination of the enzyme spectrum in blood, urine and feces is extremely important. Methods for determining the isoenzyme spectrum of amylase, lipase, trypsin and its inhibitors, elastase-1 are informative.

Amylase is one of the indicator enzymes. Extrapancreatic factors have little effect on its content. The increase in amylase levels is observed in 2–12 h from the beginning of exacerbation of chronic pancreatitis, and during the next 2–4 days its rapid decrease occurs. The level of lipase rises a little later (after 3-4 days). The exacerbation phase of chronic pancreatitis is also characterized by an increase in trypsin levels and a simultaneous decrease in the value of the inhibitor / trypsin ratio.

Foreign authors (Michael H. Ma et al., 2012) analyzed the levels of amylase, lipase, and AST in children with pancreatitis of biliary origin. The values of these enzymes exceeded those in children with pancreatitis of other etiologies in 1.6; 1.5; 2.3 times respectively.

However, it should be borne in mind that the normal activity of blood and urine enzymes does not preclude exacerbation of chronic pancreatitis. Provocative tests with proserine or glucose are used to confirm the diagnosis of chronic pancreatitis. After that, the level of blood amylase normally increases to 60% of the initial, and in severe pancreatic insufficiency - up to 30%.

At a severe course of a disease decrease in level of enzymes is noted. The most optimal for assessing the exocrine function of the pancreas in children is to determine the level of *fecal elastase-1* using an enzyme-linked immunosorbent assay - a non-invasive method with high sensitivity.

Pancreatic elastase (elastase-1) - an enzyme synthesized by the pancreas, when entering the duodenum is involved in the digestive process along with other enzymes. Unlike most enzymes, elastase-1 is not destroyed in the intestine, which is why its concentration in feces is the main indicator of exocrine function of the pancreas.

Reference values of fecal elastase-1:

- norm - more than 200 mcg / g of fecal masses;
- moderate to mild exocrine insufficiency of the pancreas - 100-200 mg / g of feces;
- severe exocrine insufficiency of the pancreas - less than 100 mg / g of feces.

It should be noted that the sensitivity of functional tests, in particular the determination of the level of elastase-1, is insufficient to detect a mild deficiency of pancreatic enzymes (O. Yu. Belousova et al., 2013). This must be taken into account, because this kind of disorder pediatricians and pediatric gastroenterologists have to deal with most often.

Instrumental diagnostics

Among the instrumental non-invasive methods of diagnosis of chronic pancreatitis in children, the most informative is ultrasound. Ultrasound method of examination allows to determine the size of the pancreas and the degree of echogenicity of the parenchyma. Normally, the thickness of the head of the pancreas in children ranges from 8 to 18 mm, body - from 5 to 15 mm and tail - from 5 to 16 mm, the width of the main duct does not exceed 2 mm. At an exacerbation of chronic pancreatitis diffuse or local increase in a head, a body or a tail of a pancreas, expansion of a channel, contours become indistinct, echogenicity of body increases. In such cases, it is necessary to conduct repeated studies to assess the dynamics of the gland. With a long course of chronic pancreatitis, the contours of the gland become uneven, there is an inhomogeneous increase in echogenicity, signs of fibrosis, foci of calcification. Also, ultrasound can detect calculosis in the ducts of the gland, pseudocysts, in case of trauma to the abdominal cavity - hematoma or rupture of the pancreas.

However, it should be noted that the standard ultrasound method can not always verify the diagnosis of chronic pancreatitis, because changes in echogenicity of the organ and increase in its size primarily indicate in favor of reactive pancreatitis or dyspancreatism (pancreatopathy) and are not necessarily signs of severe inflammation. Much more informative, according to modern authors (O. Yu. Belousova, 2013), are available modified methods of ultrasound, such as the method of *postprandial ultrasound assessment of the pancreas* (GV Rymarchuk et al., 2001) and the method of quantification indicators of volumetric blood flow in the superior mesenteric artery before and after food load (*assessment of postprandial hyperemia* - SI Polyakova, 2005). According to the method of postprandial ultrasound assessment of the pancreas, in children in the morning on an empty stomach determine the position, contour, structure, echogenicity, the presence of pathological inclusions, the state of the main pancreatic duct of the pancreas, measure the linear size of the pancreas before and after the test 90. The obtained results are evaluated as follows:

- increase or decrease in the size of the software by less than 5% - chronic pancreatitis;
- change in size by 5–15% - reactive changes in the pancreas;
- resizing by more than 15% is a normal postprandial reaction.

An additional method of diagnosing CP, for clarification, is computed tomography (sensitivity of the method about 90%, specificity - 85%), which allows to detect changes in the size of the pancreas, its contours, foci of necrosis and calcification, pseudocysts and cysts, as well as enlargement pancreatic duct.

Functional disorders of the pancreas - dyspancreatism (pancreatopathy). Reactive pancreatitis

Chronic pancreatitis in children often differentiate with functional disorders of the pancreas in which most disturbed exocrine function of the body (dyspankreatyzm) of low-on clinical symptoms. At a dyspancreatism inflammatory changes of a pancreas are absent, process develops on type of a viscero-visceral reflex and is limited, probably, to transient hypostasis. Usually dyspancreatism accompanies diseases of other digestive organs (gastroduodenal area and hepatobiliary system) and is characterized by short-term pain mainly in the left hypochondrium, loss of appetite, sometimes - unstable bowel movements, steatorrhea, rarely - creatorrhea. Sometimes palpation can reveal a slightly tense and sensitive area of the left costal-vertebral angle, where the retroperitoneal pancreas is projected and directly adjacent. Data of ultrasound examination of the pancreas indicate its normal size or a slight increase. At a dyspancreatism disturbance of associative allocation of the basic enzymes of a pancreas - amylase, lipase and trypsin is always observed. However, only the normal ratio between enzymes can be disturbed and not always any of them goes beyond normal values (Yu. V. Belousov, 2012). These disorders are, from the standpoint of pathophysiology, premorbid condition, or prenosological, which precedes and may contribute to the development of the disease. In this state, the body's defenses and adaptive forces are overstrained or weakened, but clinical manifestations in the form

of individual symptoms have not yet been observed. The premorbid period is characterized by changes at the molecular or subcellular level and may result in the restoration of body or individual organ functions or go into a prenosological state, which, in turn, is characterized by the first clinical symptoms (loss of appetite, rarely transient pain in the left hypochondrium, sometimes - unstable stools). However, this is not yet a clinical picture of CP, and the inflammatory changes that underlie it are still absent.

It should be noted that both variants of classification proposed by RV Rymarchuk (1998) and a group of experts of the Scientific Center for Child Health of the Russian Academy of Medical Sciences (2004), above, do not include the concept of functional disorders - pancreatopathy or dyspancreatism, which occur in pediatric practice. quite often.

It is also important to emphasize the close pathogenetic link between functional disorders of the pancreas and the *pancreatic variant of Oddi's sphincter dysfunction* . In the diagnosis of this dysfunction, the doctor should rely on the Roman criteria IV, which assume the presence of the following signs:

1. Pain in the left hypochondrium, decreases when leaning forward (pancreatic type); girdle pain (combined type).

2. Pain may be accompanied by the following symptoms: onset after eating; appearance at night; nausea and / or vomiting.

3. Pain in combination with increased serum amylase / lipase levels, possible biochemical signs of cholestasis

4. Signs of SFO dysfunction (hypertonia) according to ultrasound results

The term "reactive pancreatitis / reactive pancreatopathy" refers to the defeat of the pancreas, which occurs against the background of any diseases, especially digestive organs (gastroduodenal, biliary pathology), ie this condition is secondary. According to some authors (TV Gasilina, SV Belmer, 2009), it is not actually "pancreatitis". In the treatment of the underlying disease, this condition is reversible. Clinical symptoms in reactive pancreatitis are short-lived and stop before the underlying disease. Morphologically, changes in reactive pancreatitis are characterized by interstitial edema associated with impaired lymph outflow and the development of enzymatic insufficiency of the pancreas, which is usually reversible. RA Faizullina (2013) on the basis of laboratory and instrumental examination proposed two options for reactive software changes. The first option has an acute course, accompanied by pain in the "pancreatic" points (Mayo - Robson, Desjardins, Kacha), a short-term increase in the activity of pancreatic blood enzymes, ultrasound shows an increase in the size of the pancreas, sometimes a decrease in parenchymal echogenicity. The second variant is characterized by a prolonged course, with pain in the "pancreatic" points (Mayo - Robson, Desjardins, Duck, etc.), increased activity of pancreatic enzymes in the blood and duodenal contents, and after stimulation there is a decrease in the secretion of enzymes in the duodenal contents. At ultrasound - increase in the size of the software, sometimes increasing the echogenicity of the parenchyma or single foci of compaction on the background of reduced or unchanged echogenicity.

Treatment

In the treatment of diseases of the pancreas in children requires an adequate individual approach. During the exacerbation of CP, the child is hospitalized.

Regime

The mode of the maximum physical and psychoemotional rest is provided.

Diet therapy

Extremely important place is occupied by a strict diet for the first 24-48 hours - drinking rose hip broth, slightly mineralized alkaline mineral waters (Borjomi, Slavyanov, Smirnov, Morshyn).

Then, after stabilization, grated porridge on water, slimy soups without oil, white bread, low-fat cheese (12-18% fat), unsweetened tea are introduced into the diet. Grated boiled vegetables are added from the 5th day, and from the 7th-9th day - boiled meat and fish in grated form. Fresh fruits and vegetables can be included in the diet only from the 15th to the 20th day.

It is necessary to individualize the diet as much as possible, taking into account the age of the sick child, his personal characteristics, as well as the presence of concomitant pathology. Not earlier than in a month the child is transferred to the table № 5P according to Pevzner, which contains an increased amount of protein (up to 130% of physiological needs) and a reduced amount of fat (up to 80%), with physiological or reduced carbohydrate content, while extractives are excluded.

Drug treatment

One of the important tasks in the acute period of chronic pancreatitis is the *elimination of pain* .

1. Baralgin, analgin, tramal are used as analgesics.

2. Of the antispasmodics, mebeverine (Duspatalin) is prescribed at a dose of 2.5 mg / kg twice a day for children over 12 years of age, papaverine (0.005-0.06 g twice a day); children at any age - pryfinium bromide (Riabal on 0,005-0,02 g 1-2 times daily or intramuscular injection of 0.5-1.0 ml twice daily for 7-10 days); school-age children - pinaveriyu bromide (dytsetel) 50-100 mg three times a day. Drugs administered parenterally in the first days of exacerbation and if severe, while improving health - *per os.*

Analgesic and antispasmodic drugs not only relieve the child of abdominal pain, but, importantly, eliminate the spasm of the sphincter of Oddi, thereby reducing intraductal pressure and ensuring the passage of pancreatic juice and bile into the duodenum.

In order to *suppress the functional activity of the pancreas,* it is advisable to prescribe drugs that reduce gastric secretion:

1) complex antacids (maalox, almagel, glucosil) 5–15 ml (0.5–1 pill) 2–3 times a day after meals 1.5–2 h;

2) blockers of H2-receptors of histamine III generation (famotidine, kvamatel) - a dose of 1-2 mg / kg per day for 1-2 doses with a gradual reduction of the dose to avoid the syndrome of "rebound";

3) regulatory peptides (somatostatin, dalargin, glucagon) - the most effective means to inhibit the enzymatic activity of software. In the treatment of pancreatitis octreotide considered the drug of choice - similar to endogenous somatostatin, which is humoral inhibitor of secretion of exocrine and endocrine pancreas and intestines. The drug is prescribed 25-50 mg 2-3 times a day intravenously or subcutaneously for 5-7 days;

4) proteolysis inhibitors (contrikal, gordox, trasilol, zymofen, etc.) - administered parenterally in severe forms of CP to reduce enzymatic toxemia. The dose in this case is determined by the degree of enzyme and the condition of the child.

As *detoxifying agents* and to eliminate metabolic disorders in the first days in severe cases, intravenous glucose-novocaine mixture, saline solutions, 5% solution of albumin, rheopolyglucin, ascorbic acid, B vitamins are administered intravenously.

Ondansetron (osetron), prokinetics (metoclopramide, domperidone) are prescribed to *relieve uncontrollable vomiting and nausea*.

In order to *prevent purulent complications* in severe forms of CP, broad-spectrum antibiotics (cephalosporins, macrolides) are prescribed.

4–6 days after the elimination of the pain syndrome, *pancreatic enzymes* that do not contain bile (pancreatin, creon, mesylate forte) are prescribed.

Enzyme therapy relieves pain by suppressing the hypersecretion of cholecystokinin-induced pancreatic juice, which reduces the pressure in the ducts of the pancreas, and restores the processes of cavity digestion (Review of expert recommendations of some European countries on the management of chronic patients). In pediatric practice, preference is given to microgranular enzyme preparations with an acid-resistant shell (creon, pangrol). Their advantage is to ensure maximum efficiency and safety, the possibility of the most accurate dosing, which is extremely necessary for adequate administration in accordance with the severity of disorders of the exocrine function of the pancreas of the child.

The main indication for the appointment of enzymes is the presence of steatorrhea - the appearance of neutral fat in the stool. Due to the fact that some children with insufficiency of exocrine function of the pancreas may be absent, the appointment of enzyme preparations in this case are guided by clinical or laboratory signs of malnutrition.

The dose should be calculated according to the lipase, as its enzymatic activity, together with the trypsin content, determines the effectiveness of the drug.

The initial dose of the enzyme preparation in children is 500 IU of lipase per 1 g of fat in the diet per day, the maximum daily dose should not exceed 10.000 IU of lipase per 1 kg of body weight per day.

It is necessary to strictly adhere to the principle of prescribing enzyme preparations, which consists in distributing the daily dose (no more than 10.000 U of lipase per 1 kg of body weight per day) in accordance with the amount of food consumed, that is, the number of doses of the drug should coincide with the number of feedings. The duration of enzyme intake is about 2 weeks.

Dispensary supervision

During the 1st year after inpatient treatment, an examination by a general practitioner - family medicine, pediatrician or pediatric gastroenterologist every 3 months (blood and urine amylase levels are monitored, coprogram). At pancreatic insufficiency appoint 3 - 4 times a year a course of enzymes for 2 weeks. The following years the examination of the child general practitioner - family medicine pediatrician or twice a year, pediatric gastroenterologists - 2 times a year, spend indications enzyme replacement therapy. Ultrasound examination is performed twice a year. In case of clinical and laboratory manifestations of the disease the child required hospitalization. From the 2nd year of dispensary observation (in the phase of incomplete clinical remission) the child is examined once every 6 months. (2 times a year), physical education classes are allowed in the preparatory group. An important condition for stabilizing the process and preventing further exacerbations of chronic pancreatitis is the use of repeated courses of sanatorium treatment. In 2 years after exacerbation of the disease (period of complete remission) dispensary observation and anti-relapse treatment is carried out once a year, shown spa treatment in remission, physical education classes are allowed in the main group.

Sanatorium treatment - 24-30 days.

Mandatory clinical examinations: clinical examination, the presence of symptoms of Mayo-Robson, Grotto.

Mandatory laboratory tests: general blood test, general urine test.

Additional laboratory tests: proteinogram, fecal elastase-1, blood and urine glucose, glucose tolerance test. Mandatory instrumental examinations: ultrasound of the abdominal cavity (if necessary). • Diet 4, 5 or 9 (diet № 1 or № 3). • Mineral waters of small and medium mineralization of hydrocarbonate and sulphate composition. They have antispasmodic, desensitizing, anti-inflammatory, immunocorrective action, stimulate the secretion of pancreatic juice, increase the content of bicarbonate, help restore the content of enzymes in the secretion of the pancreas. Assign 1-2 times a day, increasing the single dose under conditions of good tolerability to 3 times a day for 60-40 minutes. before meals (single dose of 5 ml / kg body weight).

• Peloidotherapy: mud applications are applied to the right hypochondrium and segmentally behind for 10-15 minutes. at a mud temperature of 38-40 ° C, every other day, alternating it with baths (sea, mineral, coniferous, radon, carbon dioxide).

For the course - 8-10 procedures.

• Ozokerite applications.

• Hardware physiotherapy:

- electroplating;
- electrophoresis of peloidine or peloidodistylate;
- sinusoidal modulated currents;
- decimeter range waves;
- electrophoresis.

• Balneotherapy.

• Climatotherapy includes aerotherapy, air, sunbathing, sea bathing according to the usual method.

• Motor mode: morning hygienic gymnastics, group physical therapy, dosed walking.

6. Materials for methodological support of the lesson

6.1. Tests

1. In the development of diseases of the biliary tract a significant etiological factor is:
a) the presence of dietary fiber in the diet
b) respiratory viral infections
c) regular nutrition
d) physical activity
+ e) abuse of food rich in animal fats

2. Primary dysfunction of the biliary tract due to the influence of the following factors:
a) giardiasis invasion
b) dysbiosis
+ c) constitutional features
d) gastroduodenal pathology
e) abnormalities of the gallbladder

3. What is the nature of pain typical of biliary tract dysfunction by hyperkinetic type:
a) "late" pain after eating fatty foods
b) shingles
+ c) short-term paroxysmal pain in the right hypochondrium
d) constant stabbing pains in the right hypochondrium
e) relief of pain after eating

4. At dysfunction of a biliary tract on hypokinetic type the most expressed:
a) cholestatic syndrome
b) diarrheal syndrome
c) astheno-neurotic syndrome
d) skin-allergic syndrome
+ e) dyspeptic syndrome

5. One of the diagnostic criteria of FDBT is:
a) weight loss
b) shingles
c) jaundice
+ d) pain in the right hypochondrium
e) alternation of constipation and diarrhea

6. At hypomotor dysfunction of a biliary tract apply:
+ a) holagogum
b) valerian tincture
c) drotaverine
d) bismuth preparations
e) mebeverine

7. When performing ultrasound, the volume of the gallbladder 1 hour after taking a choleretic breakfast decreased by more than 2/3. Assess the nature of the motor function of the gallbladder:
a) hypotension
b) mixed

+ c) hyperkinesia
d) hypokinesia
e) normal

8. Contributing factor in the pathogenesis of inflammation of the gallbladder wall are:
a) oxaluria
b) chronic tonsillitis
c) flatulence
d) systemic pathology
+ e) bile stasis

9. With exacerbation of chronic cholecystitis objectively detect the following symptoms:
+ a) muscle resistance in the right hypochondrium
b) jaundice
c) hemorrhagic rash
d) pallor of the nasolabial triangle
e) shortness of breath

10. The presence of which diagnostic criterion is most characteristic of chronic cholecystitis in contrast to functional disorders of the biliary tract:
a) nausea
b) seizure and
c) paroxysmal pain in the right hypochondrium
d) seasonality
+ e) symptoms of intoxication

11. Note the main criterion of chronic cholecystitis in children with ultrasound diagnosis:
a) deformation of the gallbladder
+ b) thickening of the gallbladder wall more than 2 mm
c) the presence of microliths
d) signs of bile stagnation
e) increase in the size of the gallbladder

12. Indications for antibacterial therapy in exacerbation of chronic cholecystitis are:
a) long-term errors in diet
b) weight gain
c) amiloria
+ d) increase in body temperature
e) dyslipidemia

13. Which variant of pathogenesis is most characteristic of chronic pancreatitis of biliary origin:
a) immunopathological
b) dysmetabolic
+ c) obstructive
d) toxic-metabolic
e) genetic mutations

14. Specify the most characteristic symptom of chronic pancreatitis:
a) rotten belching
b) heartburn
c) increased appetite
d) pain in the left iliac region

+ e) diarrhea

15. In the diagnosis of chronic pancreatitis in children, the most accessible is the following research method:
a) review radiography of the abdominal cavity
+ b) determination of fecal elastase-1
c) magnetic resonance imaging
d) endoscopic retrograde pancreatocholangiography
e) fibrogastroduodenoscopy

16. To relieve pain in the period of exacerbation of chronic pancreatitis, it is best to prescribe enzyme preparations containing the maximum amount:
a) lipase
b) amylase
+ c) trypsin
d) phospholipases
e) elastases

17. To correct exocrine insufficiency of the pancreas, it is best to prescribe enzyme preparations containing the maximum amount:
+ a) lipase
b) trypsin
c) amylase
d) chemotripsin
e) elastases

6.2. The information necessary for the formation of knowledge and skills can be found in literary sources
- main:
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3. Order of the Ministry of Health of Ukraine dated January 29, 2013 № 59 "Unified clinical protocols of medical care for children with diseases of the digestive system"
4. D 362 State form of medicines. Issue ten. Kyiv, 2018 https://moz.gov.ua/uploads/1/5052-dn_20180510_868_dod_2.pdf
5. Differential diagnosis of the most common diseases of childhood. Textbook / ed. V.M. Dudnyk, 1st edition. Vinnytsia: Nilan Ltd., 2017. 560 p.
6. Karen J. Markdante, Robert M. Kligman. Fundamentals of Pediatrics according to Nelson: translation of the 8th English. edition: in 2 volumes. Volume 1. Kyiv: VSV "Medicine", 2019. XIV, 378 p.
7. Karen J. Markdante, Robert M. Kligman. Fundamentals of Pediatrics according to Nelson: translation of the 8th English. edition: in 2 volumes. Volume 2. Kyiv: VSV "Medicine", 2019. XIV, 426 p.
8. Emergency conditions in pediatric patients: Training. way. for students. honey. ZVO, medical interns. - 2nd type. Recommended MES recommended NMU Academic Council. OO Bohomolets / Marushko Yu, Chief GG etc. Kyiv: VSV "Medicine", 2020. 440 p.
9. Pediatrics: a national textbook: in 2 volumes / Ed. prof. Berezhnogo VV Kyiv, 2013. Vol.1. Kyiv, 2013. 1040 p.
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7. Lezhenko GA Dysfunction of the gallbladder and sphincter of Oddi in children / AG Lezhenko, AE Pashkova, AN Girya // News of medicine and pharmacy. - 2014. - № 18. - P. 12—14.
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9. Sorokman TV, Popelyuk A.-M.V. Features of the course of pathology of the pancreas in children. Gastroenterology. 2016. №62 (4). Pp. 43-46.
10. Hard OV Smischuk VV, Bryuzhina TS values biochemical studies of bile as an indicator of metabolic disorders fatty acids, phospholipids and cholesterol in children with cholelithiasis / Perynatolohyya and Pediatrics. 2015. № 1. P. 63-67.
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6.3 . Orienteering map for independent work with literature

№	The main tasks	Instructions	Answers
1	2	3	4
1.	Get acquainted with the literature and the purpose of the lesson	Get acquainted with modern ideas about etiopathogenesis, classification, clinical course and additional methods of diagnosing diseases in children	Know the risk factors for the development of the disease, modern classification, clinical picture of disease manifestations, hematological, immunological, radiological and functional signs of diseases and conditions that are part of organic and functional diseases of the biliary system and pancreas
2.	Epidemiology	Know the prevalence among children.	Know: the prevalence of the most common diseases and pathological conditions in the pediatric population .
3.	Etiopathogenesis	Know the causes and mechanism of gastrointestinal lesions in children	Know that these changes may be due to malformations, acquired and traumatic injuries, but more often they are the result of inflammatory diseases

4 .	Clinic	Describe the clinical picture	Remember the difference in the clinical manifestations of the disease
7.	Diagnosis	Know the schemes of diagnosis and treatment of gastrointestinal diseases	Use schemes for diagnosis and treatment of organic and functional diseases of the biliary system and pancreas

7. Materials for self-control over the quality of preparation.

A. Questions for self-control.

1. Relevance and prevalence of FDBT and chronic pancreatitis in children.
2. Etiopathogenesis and classification of FDBT in children.
3. Clinical manifestations of FDBT in children.
4. Laboratory and instrumental examination of a child with FDBT.
5. Differential diagnosis of FDBT and chronic cholecystitis.
6. Approaches to therapy and medical examination of patients with FDBT and chronic cholecystitis.
7. Etiopathogenesis of chronic pancreatitis.
8. Clinical manifestations of chronic pancreatitis.
9. Laboratory and instrumental examination of a sick child with chronic pancreatitis.
10. Differential diagnosis of chronic pancreatitis and pankreatopatiy.
11. Approaches to the treatment of chronic pancreatitis.
12. Rehabilitation measures for children with this pathology.

B. Tests for self-control:

1. In the development of diseases of the biliary tract a significant etiological factor is:
a) the presence of dietary fiber in the diet
b) respiratory viral infections
c) regular nutrition
d) physical activity
+ e) abuse of food rich in animal fats

2. Primary dysfunction of the biliary tract due to the influence of the following factors:
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a) hypotension
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+ c) trypsin
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e) elastases

17. To correct exocrine insufficiency of the pancreas, it is best to prescribe enzyme preparations containing the maximum amount:
+ a) lipase
b) trypsin
c) amylase
d) chemotripsin
e) elastases

B. Tasks for self-control:

1. Girl, 9 years old. Ill for 1 year. Complains of abdominal pain, mainly in the right hypochondrium, a feeling of heaviness and distension in this area. After eating fried, fatty foods, the pain intensifies. Constipation occurs periodically. On examination, the pallor of the skin is determined, the tongue is covered with a grayish-white plaque, on palpation of the abdomen there is pain in the right hypochondrium, positive symptoms Vasilenko, Kera, Murphy.

In the biochemical analysis of blood total bilirubin tial - 15.5 mmol / l, direct - 3.2 mmol / l, indirect - 12.3 mmol / l; ALT - 22 U / l, AST - 23 U / l; alkaline phosphatase - 123 U / l; total cholesterol - 4.1 mmol / l; amylase - 42 U / l. In the coprogram: a moderate amount of fatty acids, intracellular starch, digestible fiber.
Ultrasound examination of the abdominal cavity: gallbladder length - 66.7 mm , width - 25 mm, wall thickness 1.7 mm .

Task:

1. Establish a preliminary diagnosis.
2. Evaluate the data of laboratory and instrumental research.
3. What additional research methods are needed to clarify the diagnosis?

Answer:

1. Functional disorder of the biliary tract.
2. Indicators of biochemical analysis of blood within normal limits. In the coprogram - signs of impaired absorption of fats in the intestine, amyloria, which can be caused by a disorder of bile secretion. Ultrasound - signs of impaired motility of the gallbladder without inflammation.
3. Ultrasound examination to assess the motor-evacuatory function of the gallbladder with the use of a choleretic breakfast.

2. Boy, 13 years old. For 1.5 years, periodic abdominal pain, more in the right hypochondrium, which are exacerbated after eating errors (fried potatoes with ketchup, pork chops), stressful situations, are paroxysmal in nature, decrease after taking antispasmodics (No-spa). Also worried about fatigue, loss of appetite, periodic taste of bitterness in the mouth, heartburn.
Objectively: the child is malnourished, the skin is pale, the tongue is covered with a grayish-white plaque with a yellowish tinge, palpation of the abdomen determines the pain in the right hypochondrium, the positive symptoms of Kerr, Murphy, Ortner. In the general analysis of blood insignificant neutrophilic leukocytosis, ESR - 18 mm / h. In the biochemical analysis of blood total bilirubin - 19.6 mmol / l, direct - 6.8 mmol / l, indirect - 12.8 mmol / l; ALT - 32 U / l, AST - 29 U / l; LF - 165 U / l; total cholesterol - 5.8 mmol / liter.
Ultrasound examination of the gallbladder shows a constriction in the neck, length of JM - 68.5 mm , width of JM - 28 mm , wall of JM - 3.5 mm , compacted, a small amount of echo-suspension in the gallbladder cavity.

Task:

1. Formulate a diagnosis.
2. What are the characteristic clinical syndromes found in children?
3. Make a treatment plan.

Answer:

1. Chronic cholecystitis, period of exacerbation, moderate. Anomaly of gallbladder development (constriction in the neck).
2. Pain, dyspeptic, asthenovegetative.
3. Diet - table №5a according to Pevzner, with the exception of fried, fatty, spicy dishes, smoked products, butter dough products, meat broths, hot, cold and carbonated drinks; mode - limitation of physical activity, with a gradual expansion of physical activity; drug treatment: etiotropic antibacterial therapy, pathogenetic therapy - cholagogues (choleretics, cholekinetics), antispasmodics, vitamin therapy; course of tubage ("blind" sounding) N 8-10; mineral waters; physiotherapy.

3. A 14-year-old patient D. was admitted to the gastroenterology department with complaints of abdominal pain, mainly in the epigastrium, which occur after eating fatty, fried, excessively

consumed food, accompanied by nausea and irradiation in the back. In the anamnesis: these symptoms disturb during a year, sometimes pains arise at night, are stopped by antispasmodics (but-spa). One and a half years ago, the child received a closed abdominal injury. Recently, the condition has worsened, with heartburn, loss of appetite, weight loss, general weakness, and increased fatigue. Allergy history is burdened: food allergy to citrus, chocolate.
Objective: patient with asthenic physique, malnutrition, pale skin, acrocyanosis, tongue covered with grayish-white plaque, palpation of the abdomen reveals pain in the epigastric region, right above the navel and in the left hypochondrium, a positive symptom of Mayo-R.
Clinical blood test: Hb - 112 g / l, Er - 4,0 * 1012 / l, KP - 0,94; leukocytes - 13 * 109 / l, ESR - 19 mm / year.
Biochemical analysis of blood: total protein - 68 g / l; ALT - 29 U / l, AST - 22 U / l, LF - 115 U / l; total bilirubin - 12.5 μmol / l, amylase - 190 U / l (normal - 25-125).
Coprogram: A moderate amount of altered muscle fibers and neutral fat. The level of fecal elastase-1 is 140 μg / g of feces.
Ultrasound of the pancreas: the size of the head of the pancreas 22 mm, body - 16 mm, tail - 12 mm, width of the main duct - 2.5 mm, the contours of the gland are blurred, echogenicity is increased.

Task:

1. Formulate a diagnosis.
2. What are the main etiological factors of this disease?
3. Evaluate the data of laboratory and instrumental research.

Answer:

1. Chronic pancreatitis, exacerbation, recurrent, moderate.
2. Pathology of the duodenum and biliary tract; acute viral and bacterial infections; helminthiasis; chronic inflammatory bowel disease; system processes; allergic pathology; abdominal injuries; toxic and iatrogenic damage.
3. Clinical blood test: neutrophilic leukocytosis, accelerated ESR; in the biochemical analysis of blood - hyperenzymemia (increased amylase levels). Coprogram: creatorrhea, steatorrhea. The level of fecal elastase-1 corresponds to a moderate degree of exocrine insufficiency of the pancreas. Ultrasound of the abdominal cavity - signs of exacerbation of chronic pancreatitis: increase in the size of the pancreas, dilation of the main duct, blurred contours, increased echogenicity.

8 . Materials for classroom independent preparation.

8.1. The list of educational practical tasks that must be performed during practical classes.

1. Collect anamnesis, select data that indicate the disease.
2. Identify the most informative signs of the disease during the objective and laboratory-instrumental examination of the patient.
3. To establish the clinical diagnosis according to the modern classification.

9. Instructional materials for mastering professional skills and abilities.

9.1. Methods of work performance, stages of performance

1. To evaluate the received data of the anamnesis of life and illness, to allocate risk factors
2. Conduct a clinical examination of the patient.
3. Make a plan for additional examination.
4. Evaluate the results of laboratory and instrumental examination.
5. To formulate the clinical diagnosis according to the classification.
6. Prescribe treatment that is adequate for the specific situation

10. Materials for self-control of mastering knowledge, skills, abilities

Situational tasks:

1. Girl, 9 years old. Ill for 1 year. Complains of abdominal pain, mainly in the right hypochondrium, a feeling of heaviness and distension in this area. After eating fried, fatty foods, the pain intensifies. Constipation occurs periodically. On examination, the pallor of the skin is determined, the tongue is covered with a grayish-white plaque, on palpation of the abdomen there is pain in the right hypochondrium, positive symptoms Vasilenko, Kera, Murphy.

In the biochemical analysis of blood total bilirubin - 15.5 μmol / l, direct - 3.2 μmol / l, indirect - 12.3 μmol / l; ALT - 22 U / l, AST - 23 U / l; LF - 123 U / l; total cholesterol - 4.1 mmol / l; amylase - 42 U / l. In the coprogram: a moderate amount of fatty acids, intracellular starch, digestible fiber. Ultrasound examination of the abdominal cavity: gallbladder length - 66.7 mm , width - 25 mm , wall thickness JM 1.7 mm .

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