MINISTRY OF HEALTH OF UKRAINE
"Ukrainian Medical Stomatological Academy"

«Approved»
on meeting the
department of Pediatric Surgery
Protocol № 1 of 28.08.2020

The Head of the department
O.V. Pelypenko

METHODICAL INSTRUCTIONS

FOR STUDENTS` SELF-WORK

WHILE PREPARING FOR PRACTICAL LESSONS

Educational discipline	Pediatric Surgery
module №3	Urgent Pediatric Surgery
Theme of the lesson	Inflammatory diseases of abdominal cavity organs: peritonitis in children. Appendicular peritonitis, primary peritonitis, peritonitis of newborn.
Course	V
Faculty	foreign students preparation

POLTAVA 2020

1. **The topic basis**: the topic “peritonitis in children. Appendicular peritonitis, primary peritonitis, peritonitis of newborn.” is very important for future doctors in their professional activity, positively influences the students in their attitude to the future profession, forms professional skills and experience as well as taking as a principle the knowledge of the subject learnt.

2. The aims of the training course:

1.To master the basic list of surgical diseases at children caused by the inflammatory diseases of abdominal cavity organs.

2.To recognize basic clinical manifestation and local symptoms of inflammation of abdominal cavity organs.

3. To differentiate symptoms of inflammatory diseases of abdominal cavity organs, which need surgical intervention.

4. To interpret the auxiliary methods of research (CT, USD, X-ray, thermometric, rectal examination), laboratory and biochemical analyses, diagnostic laparoscopy.

3. Basic knowledge, skills, habits necessary for studying the subject (interdisciplinary integration).

Names of previous disciplines	Obtained skills
1. Anatomy, topographic anatomy.	Conducting palpation of different parts of the intestine, knowledge of the features of the anatomy of the abdominal cavity in different age periods.
2. Pathological physiology.	Clinical interpretation of laboratory studies.
3. Pathological anatomy.	Compare the morphological changes inherent in different types of acute appendicitis, peritonitis.
4. Microbiology.	Own the technique of sampling material for research. Interpret the results of the microbiological study.
5. Propaedeutics of childhood diseases	Collect complaints, anamnesis of the disease, conduct an examination of the child and additional methods of examination for diseases of the abdominal cavity.
6. Topographic anatomy and surgical surgery.	Own the methodology (diagrams) of operational access and methods of surgical interventions on the organs of the abdominal cavity in children of different age groups.
7. General surgery.	Demonstrate the methods of preparing a patient for therapeutic and diagnostic interventions and surgical interventions.
8. Hospital surgery.	To conduct differential diagnostics and substantiate the methods of diagnosis, treatment of acute appendicitis and abdominal cavity diseases in children.
9. Clinical pharmacology.	Classify the drugs necessary for the treatment of acute surgical diseases, taking into account the pharmacokinetics and methods of action of drugs in

	children of different age groups.

Thereotical questions for the lesson:

1. Give the basic list of surgical diseases at children caused by the inflammatory diseases of abdominal cavity organs.
2. What are the basic clinical manifestations and local symptoms of inflammatory diseases of abdominal cavity organs?
3. What are the basic clinical manifestations and local symptoms of the inflammatory diseases of abdominal cavity organs in new-born ones?
4. What are indications for conservative treatment and its character at peritonitis in new-born ones?
5. Peculiarities of preparation for surgery at inflammatory diseases of abdominal cavity organs.

4. Maintenance of the subject:

PERITONITIS

Peritoneum is multilayered membrane which lines the abdominal cavity, and supports and covers the organs within it. The part of the membrane that lines the abdominal cavity is called the parietal peritoneum. The portion that covers the internal organs, or viscera, is known as the visceral peritoneum and forms the outer layer (serosa) of most of the intestinal tract. The supportive peritoneum forms sheets of greatly modified membranes called mesenteries. These tissues hold the organs of the digestive tract in position and convey nerves, blood vessels, and lymphatic ducts to the viscera. The space between the visceral and parietal membranes contains a watery fluid that permits the abdominal organs to slide freely against the abdominal wall.

Peritonitis is defined as inflammation of the peritoneum. Peritonitis is often caused by introduction of an infection into the otherwise sterile peritoneal environment through perforation of the bowel, such as a ruptured appendix or colonic diverticulum. The disease may also be caused by introduction of a chemically irritating material, such as gastric acid from a perforated ulcer or bile from a perforated gall bladder or a lacerated liver. In women, localized peritonitis most often occurs in the pelvis from an infected fallopian tube or a ruptured ovarian cyst.

Inflammation and/or infection of the peritoneal cavity are commonly encountered problems in the practice of clinical medicine today. In general, the term peritonitis refers to a constellation of signs and symptoms, which includes abdominal pain and tenderness on palpation, abdominal wall muscle rigidity, and systemic signs of inflammation. Patients may present with an acute or insidious onset of symptoms, limited and mild disease, or systemic and severe disease with septic shock. The peritoneum reacts to a variety of pathologic stimuli with a fairly uniform inflammatory response.

Classification.

Depending on the underlying pathology, the resultant peritonitis may be infectious or sterile (ie, chemical or mechanical).

Peritoneal infections are classified as primary (spontaneous), secondary (related to a pathologic process in a visceral organ), or tertiary.

Primary peritonitis (also referred to as idiopathic or spontaneous peritonitis) has been defined as an infectious process involving the peritoneal cavity that has no intra-abdominal source.

Secondary peritonitis is a chronic or acute inflammation caused by bacteria entering the peritoneum following perforation of the gastrointestinal tract, for example, ruptured appendix. The irritant can be gastric juice, small bowel contents or faeces from the colon.

Tertiary peritonitis is persistent or recurrent infection after adequate initial therapy.

The intra-abdominal infection may be localized or generalized, with or without abscess formation.

Microbiology includes a mixture of aerobic and anaerobic organisms. The most commonly isolated aerobic organism is *Escherichia coli*, and the most commonly observed

anaerobic organism is *Bacteroides fragilis*. A synergistic relationship exists between these organisms. In patients who receive prolonged antibiotic therapy, yeast colonies (*candidal species*) or a variety of nosocomial pathogens may be recovered from peritoneal fluids.

Skin flora may be responsible for abscesses following a penetrating abdominal injury.

The type and density of aerobic and anaerobic bacteria isolated from intra-abdominal fluid depend upon the nature of the microflora associated with the diseased or injured organ.

Clinical features.

Initial diagnosis is based on symptoms and a physical examination. Abdomen will be tender, and often distended. Children with peritonitis will often feel the need to protect their abdominal area from touch. In neonates abdominal distention usually accompanied by erythema and edema of abdominal wall ad scrotum.

Many patients have a significant septic response, volume depletion, and catabolic state. This syndrome may include high cardiac output, tachycardia, low urine output, and low peripheral oxygen extraction. Initially, respiratory alkalosis due to hyperventilation may occur. If left untreated, this progresses to metabolic acidosis. Sequential multiple organ failure is highly suggestive of intra-abdominal sepsis.

Once an initial diagnosis and assessment of the probably causes has been made, tests are carried out.

Tests for spontaneous and secondary peritonitis include:

- Culture of peritoneal fluid (which is found in the peritoneal cavity and acts as a lubricant between the layers of the peritoneum) - a bacteriological laboratory test to identify infectious organisms in the fluid.
- Chemical examination or laboratory analysis of peritoneal fluid.
- Blood culture to determine the presence of microorganisms in the blood.

Other possible tests for peritonitis, especially spontaneous peritonitis, include:

- An abdominal X-ray to rule out other possible reasons for symptoms such as abdominal pain.
- An abdominal ultrasonography

SPECIFIC TYPES OF ACUTE PERITONITIS

Meconium Peritonitis

Meconium peritonitis is an aseptic peritonitis caused by spill of meconium in the abdominal cavity through one or several intestinal perforations which have taken place during intrauterine life.

Etiology/Pathophysiology. The most common causes of meconium peritonitis are ischemic lesions of the small bowel associated with mechanical obstruction (atresia, volvulus, intussusception, congenital bands, Meckel diverticulum, internal hernia, meconium ileus). However, in some cases it is impossible to find its etiology, in spite of pathological changes. Meconium is a sterile mixture and consists of desquamated epithelial cells, vernix, lanugo hair, and intestinal secretions containing cholesterol and mucopolysaccharides. When meconium spills into the peritoneum it acts as an irritant and an inflammatory serosal reaction. A secondary inflammatory response results in the production of fluid (ascites), fibrosis, calcification and sometimes cyst formation.

There are 3 types of meconium peritonitis:

1. *Fibroadhesive* - an intense adhesive peritoneal reaction with no active leak of bowel contents. This type produces obstruction by adhesive bands and the site of perforation is usually sealed off.

2. *Cystic* - a localized cavity formed by adjacent loops of bowel. This condition prevents communication of the perforation with the remainder of the viscera.

3. *Generalized* - no adhesions or calcifications, seen in cases where the bowel perforation occurs just before birth. Calcified meconium is scattered throughout the peritoneal cavity and the bowel loops are adherent by thin fibrinous adhesions.

The ***prenatal sonographic findings*** vary depending on several factors: the etiology, the time interval since perforation and the degree of inflammatory response. It may be seen as early as 13 weeks gestation. In the typical case, diffuse hyperechoic punctate echoes with or without acoustic shadowing may be seen in the abdominal cavity, on the hepatic surface and in the scrotal sac. In addition, depending upon the etiology, ascites, polyhydramnios or fetal bowel distention may be present. Polyhydramnios, reported in approximately 50% of patients, may be caused by peristaltic deficiency associated with decreased swallowing activity. If the inflammatory response remains localized a meconium pseudocyst may occur. This appears sonographically as a cystic heterogeneous mass with an irregular, calcified wall.

In the absence of an intra-abdominal mass such as a meconium pseudocyst, the major differential diagnostic possibilities for bright echoes within the abdomen is “hyperechogenic bowel”. In this condition, the bowel appears as bright as bone. This is often normal, particularly in the third trimester, but has been described to be associated with cystic fibrosis and chromosomal abnormalities.

Postnatal diagnosis is based on clinical and radiological, and ultrasonographic findings of intestinal obstruction, and occasionally one or more of the following: calcification, pneumoperitoneum, cyst formation or ascites. Typical symptoms of meconium peritonitis are abdominal distension immediately or it soon after birth, bile-stained vomiting and failure to pass meconium. X-ray and ultrasound examination show the intestinal ileus, the ascites when it exists, the ground-glass appearance of the abdomen due to the meconium and rarely the presence of a pneumoperitoneum, since the quick formation of adhesions prevents the intestinal gas from escaping. Intra-abdominal calcifications are characteristic and can easily be seen on plain abdominal films.

Early diagnosis is a decisive factor for the prognosis of these newborns, because the commencement of bacterial colonization of the meconium starts after birth.

Treatment. The indication for operation in newborns with meconium peritonitis is a clear sign of intestinal obstruction or perforation. The diagnosis of meconium peritonitis without intestinal obstruction or pneumoperitoneum does not constitute an indication for operation. Infants with neonatal meconium calcification, meconium ascites with hydrocele, or calcified meconium found in the hernial sac do not require operation, but they have to be observed and feeding withheld for 48 hours. With an absence of clinical symptoms, enteral feeding can be started with caution, gradually progressing to formula. Antibiotic coverage is desirable.

The ***prognosis*** depends upon the etiology. Bowel perforations may heal and the ascites and bowel dilatation may resolve, leaving only peritoneal calcifications as the only sonographic sign of meconium peritonitis. While cystic fibrosis is universally seen in cases of meconium ileus, it is seen in only 7-40% of cases of meconium peritonitis

Primary peritonitis

Primary peritonitis (also referred to as idiopathic or spontaneous peritonitis) has been defined as an infectious process involving the peritoneal cavity that has no intra-abdominal source. The infection may reach the abdomen through hematogenous or lymphatic routes or by direct extension from the vagina. With the exception of the ends of the fallopian tubes, the peritoneal cavity is a completely closed space that can be penetrated by foreign bodies such as ventriculoperitonel shunts and peritoneal dialysis catheters. In prepubertal and adolescent girls, retrograde spread of fluid out the through the fallopian tubes may account for the "presence of an ascending vulvovaginitis or “swimming pull peritonitis”.

The first reported case of fatal spontaneous bacterial peritonitis may have been in 1581 in a girl who expired after an episode of painless diarrhea. By the early 1900s, as many as 10% of abdominal operations in children fell into the category of primary peritonitis an associated mortality of up to 50%. Today, this condition represents less than 1% of all pediatric laparotomies because the diagnosis is made and treatment initiated without the need for operation. Most cases of primary peritonitis in the pediatric age group are associated with nephrotic syndrome or chronic hepatic states in which ascites or cirrhosis is present. This group includes infants and children with biliary atresia, cystic fibrosis, hepatic fibrosis, and lupus erythematosus. Organisms isolated from the peritoneal cavity will vary according to the various

associated conditions. For instance, gram-positive organisms, including *Streptococcus pneumoniae* and group A streptococci, and variety of gram-negative species are most commonly found in patients with nephrotic syndrome. The same gram-positive groups are cultured in patients with underlying liver disease, whereas the spectrum of gram-negative isolates includes *Esherichia coli*, Klebsiella *pneumonia*, and *Pseudomonas* species.

Clinical history. Children with primary peritonitis have acute abdominal pain associated with a febrile illness, nausea, vomiting, diarrhea, or other viral-like prodromes. The time course may be more protracted then that for secondary peritonitis, and diffuse rebound tenderness is often present. The absence of localized pain may result from irritation of visceral organ surfaces. Diagnostic paracentesis may be helpful in the presence of ascites, as is sampling of the dialysate in children with renal failure on peritoneal dialysis program. In children with impaired hepatic function, cirrhosis, and ascites, the primary peritonitis that develops is often associated with the gram-negative organisms that normally populate the intestinal tract, such as *E. coli* and *K.pneumoniae*, whereas the gram-positive isolates include *S. pneumoniae* and *Staphylococcus aureus*.

The literature identified a group of healthy children with idiopathic peritonitis and simultaneously positive cultures for group A streptococci from trachea, pharynx, or tonsils.

When the clinical picture is not improving despite intravenous antibiotics, diagnostic laparoscopy or laparotomy is indicated. An appendectomy can be done safely even in the presence of cloudy exudate, and the bowel surface can be inspected for secondary causes of the infection. After the surgical intervention, antibiotics are continued until the leukocytosis normalizes and the ileus resolves.

Abdominal Abscess

Intra-abdominal abscesses are localized collections of pus that are confined in the peritoneal cavity by an inflammatory barrier. This barrier may include the omentum, inflammatory adhesions, or contiguous viscera. The abscesses usually contain a mixture of aerobic and anaerobic bacteria from the GI tract.

Pathophysiology. Bacteria in the peritoneal cavity, in particular those arising from the large intestine, stimulate an influx of acute inflammatory cells. The omentum and viscera tend to localize the site of infection, producing a phlegmon. The resulting hypoxia in the area facilitates growth of anaerobes and impairs bactericidal activity of granulocytes. The phagocytic activity of these cells degrades cellular and bacterial debris, creating a hypertonic milieu that expands and enlarges the abscess cavity in response to osmotic forces. If untreated, the process continues until bacteriemia develops, which then progresses to generalized sepsis with shock.

Clinical.

Intra-abdominal abscesses are highly variable in presentation. Persistent abdominal pain, focal tenderness, spiking fever, prolonged ileus, leukocytosis, or intermittent polymicrobial bacteriemia suggest an intra-abdominal abscess in patients with predisposing primary intra-abdominal disease or following abdominal surgery. If a deeply seated abscess is present, many of these classic features may be absent. The only initial clues may be persistent fever, mild liver dysfunction, persistent GI dysfunction, or nonlocalizing debilitating illness.

The diagnosis of an intra-abdominal abscess in the postoperative period may be difficult because postoperative analgesics and incisional pain frequently mask abdominal findings. In addition, antibiotic administration may mask abdominal tenderness, fever, and leukocytosis.

In patients with subphrenic abscesses, irritation of contiguous structures may produce shoulder pain, hiccup, or unexplained pulmonary manifestations such as pleural effusion, basal atelectasis, or pneumonia. With pelvic abscesses, frequent urination, diarrhea, or tenesmus may occur.

Relevant Anatomy. The 6 functional compartments within the peritoneal cavity include the (1) pelvis, (2) right paracolic gutter, (3) left paracolic gutter, (4) infradiaphragmatic spaces, (5) lesser sac, and (6) interloop potential spaces of the small intestine.

The paracolic gutters slope into the subhepatic and subdiaphragmatic spaces superiorly and over the pelvic brim inferiorly. In a supine patient, the peritoneal fluid tends to collect under the diaphragm, under the liver, and in the pelvis. More localized abscesses tend to develop

anatomically in relation to the affected viscus. For example, abscesses in the lesser sac may develop secondary to severe pancreatitis, or periappendiceal abscesses from a perforated appendix may develop in the right lower quadrant. Small bowel interloop abscesses may develop anywhere from the ligament of Treitz to the ileum. An understanding of these anatomic considerations is important for recognizing and draining these abscesses.

Lab Studies.

Hematologic parameters suggesting infection (eg, leukocytosis, anemia, abnormal platelet counts, abnormal liver function) frequently are present, although patients who are debilitated often fail to mount reactive leukocytosis or fever.Blood cultures indicating persistent polymicrobial bacteremias strongly implicate the presence of an intra-abdominal abscess. Because more than 90% of intra-abdominal abscesses contain anaerobic organisms, particularly B fragilis, postoperative Bacteroides species bacteremia suggests intra-abdominal sepsis.

Imaging Studies.

Plain abdominal radiographs, though rarely diagnostic, frequently indicate the need for further investigation.

Abnormalities on plain abdominal films may include a localized ileus, extraluminal gas, air-fluid levels, mottled soft tissue masses, absence of psoas outlines, or displacement of viscera.

In subphrenic or even subhepatic abscesses, the chest radiograph may show pleural effusion, elevated hemidiaphragm, basilar infiltrates, or atelectasis.

In experienced hands, ultrasonography has an accuracy rate greater than 90% for diagnosing intra-abdominal abscesses.

Ultrasonography is readily available, portable, and inexpensive. The findings can be quite specific when correlated with the clinical picture.

A drawback is that marked obesity, bowel gas, intervening viscera, surgical dressings, open wounds, and stomas can create problems with definition. In addition, the quality of the procedure is operator-dependent. These disadvantages may limit efficacy in postoperative patients.

CT scan has greater than 95% accuracy and is the best diagnostic imaging method. The presence of ileus, dressings, drains, or stomas does not interfere with reliability.

Identify any occult abscesses using serial images obtained from the diaphragm to the pelvis.

The appearance of an air bubble within a fluid collection or a low-attenuation extraluminal mass is diagnostic of an intra-abdominal collection.

CT scans can document inflammatory edema in the adjacent fat (obliteration of fat plane) and hyperemia in the abscess wall (enhancement).

Drawbacks include nonportability, relative difficulty in diagnosing intraloop abscesses, and, possibly, poor patient cooperation.

Recent intra-abdominal surgery also may pose a diagnostic problem in patients in whom intra-abdominal abscesses are suspected. CT scan is not recommended for use in the diagnosis of such abscesses until approximately the eighth postoperative day. By that time, postoperative tissue edema is reduced, and nonsuppurative fluids (eg, hematoma, seroma, intraoperative irrigation fluid) should be reabsorbed. In most postoperative patients, signs of intra-abdominal abscesses do not develop within the first 4-5 days.

Treatment.

Medical therapy. Antibiotic therapy involves the administration of parenteral empirical antibiotics. Begin therapy prior to abscess drainage, and conclude therapy when all systemic signs of sepsis resolve. Because abscess fluid usually contains a mixture of aerobic and anaerobic organisms, direct initial empiric therapy against both sets of microbes. This may be accomplished with antibiotic combination therapy or with broad-spectrum, single-agent therapy. Specific therapy then is guided by the results of cultures retrieved from the abscess.

In patients who are immunosuppressed, candidal species may play an important pathogenic role, and treatment with amphotericin B or fluconasol may be indicated.

Surgical therapy.

Drainage of pus is mandatory and is the first line of defense against progressive sepsis. Percutaneous CT-guided catheter drainage has become the standard treatment for most intra-

abdominal abscesses. It avoids possibly difficult laparotomy, requires anesthesia, prevents the possibility of wound complications from open surgery, and may reduce the length of hospitalization. It also obviates the possibility of contamination of other areas within the peritoneal cavity. CT-guided drainage delineates the abscess cavity and may provide safe access

for percutaneous drainage. When performed by experienced hands, it also prevents the possibility of injury to adjacent viscera or blood vessels.

After surgical drainage, clinical improvement should occur within 48-72 hours. Lack of improvement within this time frame mandates a repeat CT scan to check for additional abscesses.

Criteria for removal of percutaneous catheters include resolution of sepsis signs, minimal drainage from the catheter, and resolution of the abscess cavity as demonstrated by a sonogram or CT scan. Persistent drainage usually reflects the presence of an enteric fistula, and a CT scan with contrast should be performed. Frequently, this fistula can be documented by sonography.

Complications of percutaneous drainage include bleeding or inadvertent puncture of the GI tract.

Surgical intervention.

Open surgical drainage is mandatory if percutaneous drainage fails or if collections are not amenable to catheter drainage. The surgical approach may be either extraperitoneal or transperitoneal.

With accurate preoperative localization, direct surgical drainage may be possible through an extraperitoneal approach. This technique reduces the risk of bowel injury, spread of contamination, and bleeding. It also allows for a faster return of bowel function.

The transperitoneal approach is made safer by the judicious use of preoperative antibiotics. Although contamination of otherwise uninfected sites remains a major concern, this complication is particularly reduced if the organisms involved are sensitive to the chosen drugs. . Transperitoneal exploration is indicated for multiple abscesses not amenable to CT-guided drainage, such as interloop collections or an enteric fistula feeding the abscess.

Pelvic abscesses often are palpable as tender fluctuant masses impinging on the vagina or rectum. Draining these abscesses transvaginally or transrectally is best to avoid the transabdominal approach.

During the course of a laparotomy, the surgeon must use digital or direct exploration to be certain that all loculations are broken down and that all debris, such as hematomas or necrotic tissue, is evacuated.

Improved clinical findings within 3 days after treatment indicate successful drainage. Failure to improve may indicate inadequate drainage or another source of sepsis. If left untreated, the septic state inevitably produces multiple organ failure.

5. Additional materials for the self-control

A. Clinical cases

Case 1. In a 10 year-old patient on the 5-th day after operation for gangrenous appendicitis, diffuse peritonitis occurred diarrhea, pain during defecation, frequent urination. The tongue is moist, coated with white fur. The abdomen is soft, tender in the lower abdomen. The difference between morning and evening temperature is $1,5^0$ C.

What diagnosis have you made?

What tactics have you developed?

Case 2. For four days a 13 year-old girl suffered from abdominal pain, high temperature, vomiting. During the examination the girl was pale, inert. The body temperature was 37,7°C. Pulse was 92 beats per minute. The tongue was moist, coated with white fur. The abdomen was not distended. In the right lower quadrant of abdomen a painful, immobile swelling 6x6 cm is palpable. Shchotkin-Blumberg symptom is poorly positive.

What diagnosis have you made?

What tactics have you developed?

4. LITERATURE FOR STUDENTS

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Materials for self-study of the students

Main tasks	Notes (instruction)
Repeat: Anatomy of intestine, appendix vermiformis Physiology of appendical frolics, peritoneum Pathogenesis of inflammation	To sketch out the anatomy of peritoneal cavity Top represent the methods of diagnosis of acute appendicitis and peritonitis in children
Study: Pathogenesis of appendicitis, primary and secondary peritonitis Features of appendicitis and peritonitis in children The diagnostic possibilities of ultrasound examination, CT, MRI in children	To make differential diagnosis of acute appendicitis and nonsurgical diseases in children To make the indications to surgical treatment in children with abdominal pain To know modern diagnostically methods Toknowtheadvantagesand disadvantages of classic and laparoscopic appenectomy