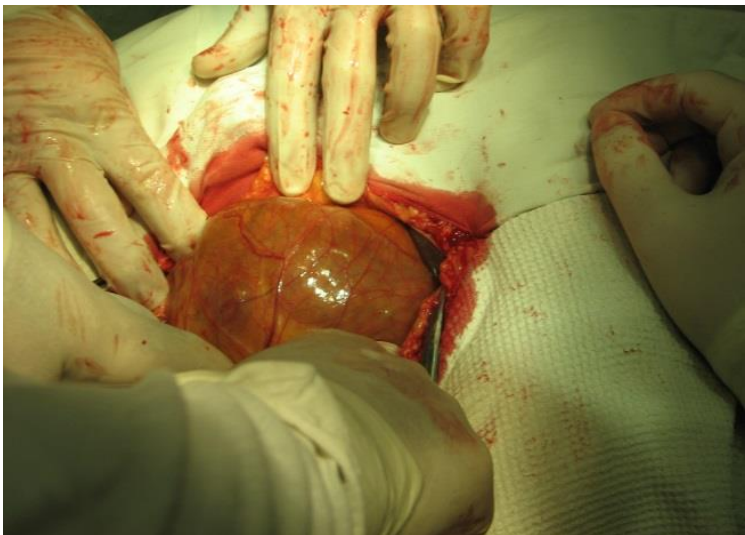


EMERGENCY ABDOMINAL SURGERY

Study guide

Edited by Ph. D., Associate Professor O. L. Sytnik





Ministry of Education and Science of Ukraine
Ministry of health of Ukraine
Sumy State University
Educational and Scientific Medical Institute

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Recommended by the Academic Council of Sumy State University

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This study guide contains information about basic principles of diagnostics and treatment of patients with acute surgical abdominal diseases.

For English-speaking students of higher educational institutions of III–IV levels of accreditation and postgraduates: internists, surgeons, family physicians.

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LIST OF CONVENTIONAL ABBREVIATIONS

WBC	– white blood cell count.
AXR	– abdominal X-ray.
US	– ultrasound.
CT	– computerized tomography scan.
MRI	– magnetic resonance imaging.
CVP	– central venous pressure.
MPI	– Mannheim peritonitis index.
ERCHPG	– endoscopic retrograde cholangiopancreatography.
NSAID	– not steroid anti-inflammatory drugs.
SIRS	– systemic inflammatory response syndrome.
FGDS	– fibergastroduodenoscopy.
SBO	– small bowel obstruction.
LBO	– large bowel obstruction.

INTRODUCTION

The publication of this tutorial is caused by providing of the new Master's training program for the discipline "Surgery".

Surgery is very important for all physicians. It starts at the IY-th academic year after finishing fundamental disciplines such as pathologic anatomy, pathologic physiology, pharmacology, topographic anatomy, operative surgery and initial clinical disciplines: general surgery, propaedeutic of internal diseases. Surgery forms the basic knowledge for successful study of the special branches of internal diseases.

The general purpose is: assimilation of theoretical and practical knowledges about etiology, pathogenesis, typical clinical manifestations, and methods of diagnosing, conservative treatment, operative treatment, rehabilitation of patients with acute surgical abdominal diseases for higher educational institutions of III–IV levels of accreditation and postgraduates: internists, surgeons, family physicians.

Concrete purposes are:

- interpretation of etiology, pathogenesis, classifications, typical clinical manifestations of the acute surgical abdominal diseases;
- formation of algorithms of diagnosis, conservative and operative treatment, postoperative treatment and rehabilitation;
- differential diagnostics of acute surgical abdominal diseases;
- evaluation of the results of lab. tests and additional methods of investigations in patients with acute surgical abdominal diseases;
- identification of the indications for laparoscopic and open techniques.

CHAPTER 1. ACUTE APPENDICITIS

Acute appendicitis is nonspecific inflammation of the vermiform appendix. It starts in the inner lining of it and spreads to its other parts.

Hystory:

- The vermiform appendix was described by Leonardo da Vinci (1472).

- In 1642 Saracenus reported about abscess in right iliac region.

- Melier in 1827 correctly ascribed the inflammation of the appendix.

- Reginald H. Fitz, an anatomopathologist at Harvard, described appendicitis in 1886 and advocated early surgical intervention.

- The first surgeon who correctly diagnosed acute appendicitis, performed appendectomy and had the patient recover was Senn from Canada, but his case was not reported until 1961.

- In 1889 McBurney described diagnostic and therapeutic management of acute appendicitis.

- At the end of the 19th century the English surgeon H. Hancock successfully performed appendectomy in a patient with acute appendicitis.

- Since 1987, many surgeons have begun to treat appendicitis laparoscopically. This procedure has been improved and standardized.

Frequency and problems. The incidence of acute appendicitis is around 7 % of the population in the United States and in European countries.

In Asian and African countries, the incidence is probably lower because of the dietary habits.

Persons of any age may be affected, with highest

incidence occurring during the second and third decades of life.

The frequency is depended from patients age: in children before 1 year – 3,48 cases on 10 000 of population, in children from 1 till 14 years – 11,4, from 15 till 59 years – 114,9, from 60 till 69 years – 29,7, 70 years and more – 15,8.

Appendicitis occurs more frequently in males than in females, with a male-to-female ratio of 1.7:1.

Statistics report that 1 from 5 cases of acute appendicitis is misdiagnosed.

The normal appendix is found in 15–40 % of patients after an emergency appendectomy.

The mortality is about 0.2–0.3 %, elderly patients have a mortality rate 5 %.

The main causes of mortality:

- later hospitalisation – 46,1 %;
- technic mistakes of operation – 5,2 %;
- mistakes of policy – 12.0 %;
- mistakes of postoperative treatment – 7,7 %;
- decompensated diseases of other systems – 9,3 %.

Anatomy. The appendix is a wormlike extension of the cecum, and its average length is 8–10 cm (ranging from 2–20 cm). Several lymphoid follicles are scattered in its mucosa. The number of follicles increases when individuals are aged 8–20 years.

From without inwards the structure of appendix is as follows:

1. Serous coat is composed of peritoneal coat, which covers the whole of the appendix except along the narrow line of attachment of the mesoappendix.

2. Muscle coat. It consists of outer longitudinal muscles and inner circular muscles as seen in case of small intestine. The longitudinal muscle is formed by coalescence of the three taeniae coli at the junction of the caecum and appendix. Thus the taeniae, particularly the anterior taenia may be used as a

guide to locate an elusive appendix. The inner circular muscle is continuation of the same muscle in the caecum. The peculiarity of the musculature of the appendix is that there are a few gaps in the muscular layer called “hiatus muscularis”. Through this infection from the submucosal coat directly comes to peritoneum and regional peritonitis occurs.

3. Submucosal coat. The submucous coat of the appendix is very rich in lymphoid tissue. It contains lymphoid follicles which are known as “abdominal tonsil”.

4. The mucous coat resembles that of large intestine.

Various anatomical positions of appendix are (fig. 1):

1. Retrocaecal position (the commonest irregular position – 70 %) – the appendix lies behind the caecum although in majority of cases in an intraperitoneal location. Only in case of long retrocaecal appendix the tip of the appendix remains in the retroperitoneal tissue close to the ureter.

2. Pelvic position (second most common irregular position – 25 %).

3. Subcaecal (2 %).

4. Subhepatic (3 %) – that means the tip of the appendix is towards the liver.

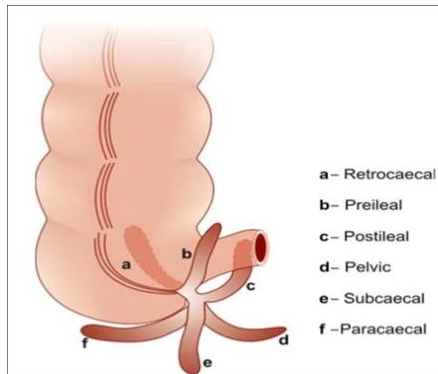


Figure 1 – Variants of appendix anatomical positions

(https://www.google.com/search?rlz=1C1GCEB_enUA1010UA1010)

Etiology of acute appendicitis:

1. Obstruction of the appendix lumen (fecoliths, hyperplasia of submucosal lymphoid follicle, intestinal helminthes, vegetables, fruit seeds, barium from previous X-rays). The causes of the obstruction include lymphoid hyperplasia secondary to irritable bowel disease or infections (more common during childhood and in young adults), fecal stasis and fecoliths (more common in elderly patients), parasites (especially in Eastern countries), or, more rarely, foreign bodies and neoplasms. Lymphoid hyperplasia of the appendix may be related to Crohn disease, mononucleosis, amebiasis, measles and respiratory infections.

2. The bacteriology flora. Most frequently seen organisms are Escherichia coli, enterococci, bacteroides (gram-negative rod), nonhaemolytic streptococci, anaerobic streptococci and CI. Welchii.

3. Diet which is relatively rich with fish and meat.

Appendicitis usually has 3 stages:

1. Oedematous stage. Appendicitis may have spontaneous regression or may evolve to the second stage. The mesoappendix is commonly involved with inflammation.

2. Purulent (phlegmonous) stage. Spontaneous regression rarely occurs. Appendicitis usually evolves beyond perforation. Peritonitis may be possible.

3. Gangrenous stage. Spontaneous regression never occurs.

Classifications:

As a result inflammation process develop from:

simple (catarrhal or superficial) appendicitis till *destructive* appendicitis (phlegmon, gangrene) and *complicated* appendicitis.

Kolesov's classification of acute appendicitis (1972)

1. Appendicular colic.
2. Simple superficial appendicitis (fig. 2).

3. Destructive appendicitis:
 - a) phlegmonous (fig. 3);
 - b) gangrenous (fig. 4);
 - c) perforated.
4. Complicated appendicitis:
 - a) appendicular mass;
 - b) appendicular abscess;
 - c) diffuse purulent peritonitis.
5. Other complications of acute appendicitis (pylephlebitis, sepsis, retroperitoneal phlegmon, local abscesses of abdominal cavity).



Figure 2 – Acute simple (superficial) appendicitis: hyperemia of appendix with vessels. The mesoappendix is normal. Spontaneous regression is possible



Figure 3 – Acute phlegmonous appendicitis: hyperemia and enlargement of appendix, fibrin

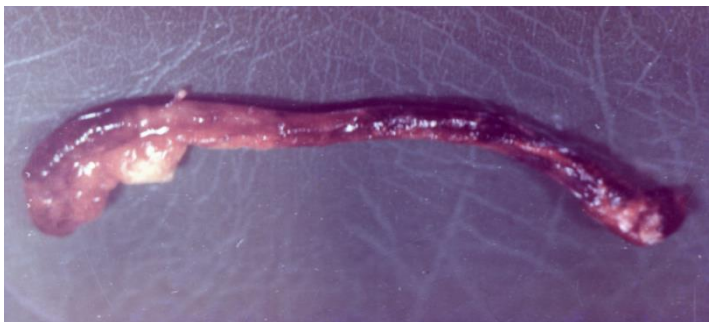


Figure 4 – Acute gangrenous appendicitis:
black or green color, total destruction of the wall

Clinical manifestations and diagnostics of acute appendicitis:

In 2–6 hours it moves to the place of appendix existence (the Kocher's sign). At coughing patients mark strengthening of pain in a right iliac area – it is a positive cough symptom (Cheremskih's sign).

The pain syndrome. The disease begins with a sudden pain in the abdomen. It is localized in a right iliac area, has moderate intensity, **constant** character and usually does not irradiate. With 70 % of patients the pain arises in epigastric area or other part of abdominal cavity - it is an “epigastric phase” of acute appendicitis.

The dispepsy syndrome. Together with pain, nausea and vomiting that have reflex character can disturb a patient, more frequently in young patients and during the “epigastric phase”.

The intoxication syndrome. The temperature of body of most patients rises, but it is a low grade fever. The general condition of patients is usually normal and gets worse only in case of growth of destructive changes in appendix. The puls rate is increased according the body temperature.

Percussion and auscultation of abdominal cavity – usually normal dates.

The palpation of abdominal wall:

Painfulness is the basic and decisive sign of acute appendicitis during the examination by palpation in a right iliac area. Tension of abdominal wall muscle is a positive symptom of peritoneum irritation.

The Blumberg's sign. After gradual pressing by fingers on a front abdominal wall from the place of pain quickly, but not acutely, the hand is taken away. Strengthening of pain is considered as a positive symptom in that place. Obligatory here is tension of muscles of front abdominal wall.

The Voskresensky's sign. By a left hand the shirt of patient is drawn downward and fixed on pubis. By the taps of 2–4 fingers of right hand epigastric area is pressed and during exhalation of patient quickly and evenly the hand slides in the direction of right iliac area, without taking the hand away. Thus there is an acute strengthening of pain.

The Rovsign's sign. By a left hand a sigmoid bowel is pressed. By a right hand by balloting palpation a descending bowel is pressed. Appearance of pain in a right iliac area is considered as a sign characteristic of appendicitis.

The Sitkovsky's sign. A patient, that lies on left, feels the pain which arises or increases in a right iliac area. The mechanism of intensification of pain is explained by displacement of blind gut to the left, by drawing of mesentery of the inflamed appendix.

The Obrazcov's sign. With the position of patient on the back by index and middle fingers the right iliac area of most painful place is pressed and the patient is asked to heave up the straightened right leg. At appendicitis pain increases acutely.

The Bartomier's sign. Is the increase of pain intensity during the palpation in right iliac area of patient in position on the left side. At such pose an omentum and loops of small intestine is displaced to the left, and an appendix becomes accessible for palpation.

The Rozdolsky's sign. At percussion there is a painfulness in a right iliac area.

The psoas (Roup's) sign. This test is performed by having the patient lie on his left side. The examiner men slowly extends the patient's right thigh, thus stretching the ileopsoas muscle. This will produce pain to make the sign positive. This indicates presence of inflamed appendix in close proximity to the psoas muscle. This is possible in retrocaecal appendicitis.

The Ivanov's sign. The deformation of abdominal wall (the umbilicus moves to the place of appendix existence) as a result of abdominal wall muscles tension and constriction.

The Yaure - Rosanon's sign. At pressure in Pty trigonum there is a painfulness (retrocaecal appendix position).

The Gabay's sign. After gradual pressign by finger in Pty trigonum the hand is taken away. Strengthening of pain is considered as a positive symptom in retrocaecal appendix position.

The Gubergritz's sign. Painfulness and skin hyperaesthesia in Scarpal trigonum (nervus genitofemoralis is involved with inflammation in retrocaecal appendix position).

The Coup's 1 sign. A patient lies on his left side. The examiner men extends the patient's right lag and moves it back and laterally. A patient feels the pain in a right iliac area.

The Coup's 2 sign. A patient lies on his back. The examiner men extends the patient's right leg and rotate it laterally. A patient feels the pain in a right iliac area.

The Mihelson's sign (acute appendicitis in pregnant women). A patient lies on her right side and feels a pain in the right iliac region.

The Krymov's sign. The examiner men palpate by finger (using external inguinal ring) the patient's posterior inguinal canal wall. A patient feels pain.

The Dumbadze's sign. The examiner men by finger performs balloting movements in umbilical ring to the right iliac area. A patient feels pain.

The Zhendrinsky's sign (differential diagnosis of acute appendicitis and acute adnexitis). Woman lies on her back. The examiner men presses by finger the abdominal wall in Cummel point (2 cm lower and righter from umbilicus) and asks to sit. In acute appendicitis feels increasing of pain, in acute adnexitis - decreasing of pain.

The Arsen's sign. A patient lies on his left side. The examiner men fixates by hand the abdominal wall in a right iliac area in upper position for some seconds. After that he removes the hand. The abdominal wall moves in lower position, and a patient feels pain.

Acute appendicitis in children. With children of infancy acute appendicitis can be seen infrequently, but, quite often carries atypical character. All this is conditioned, mainly, by the features of anatomy of appendix, insufficient of plastic properties of the peritoneum, short omentum and high reactivity of child's organism. The inflammatory process in the appendix of children quickly makes progress and during the first half of days from the beginning of disease there can appear its destruction, even perforation. The child, more frequent than an adult, suffers vomiting. Its general condition gets worse quickly, and already the positive symptoms of irritation of peritoneum can show up during the first hours of a disease. The temperature reaction is also expressed considerably acuter. In the blood test there is high leukocytosis.

Acute appendicitis of the people of declining and old ages can be met not so often, as of the persons of middle ages and youth. This contingent of patients is hospitalized to hospital rather late: in 2–3 days from the beginning of a disease. Because of the promoted threshold of pain sensitiveness, the intensity of pain in such patients is small,

therefore they almost do not fix attention on the epigastric phase of appendicitis. More frequent are nausea and vomiting, and the temperature reaction is expressed poorly. Tension of muscles of abdominal wall is absent or insignificant through old-age relaxation of muscles. But the symptoms of irritation of peritoneum keep the diagnostic value with this group of patients. Thus, the sclerosis of vessels of appendix results in its rapid numbness, initially-gangrenous appendicitis develops. Because of such reasons the destructive forms of appendicitis prevail, often there is appendicular mass.

With pregnant women both the bend of appendix and violation of its blood flow are causes of the origin of appendicitis. Increased in sizes uterus causes such changes. It, especially in the second half of pregnancy, displaces a blind gut together with an appendix upwards, and an overdistension of abdominal wall does not create adequate tension. Together with that, psoas-sign and the Kocher's sign, Bartomier's sign have a diagnostic value at pregnant women.

Appendicitis at retrocecal and retroperitoneal location. Thus an appendix can be placed both in a free abdominal cavity and retroperitoneal. An atypical clinic arises, as a rule, at the retroperitoneal location. The patients complain at pain in lumbus or above the wing of right ileum. There they mark painfulness during palpation. Sometimes the pain irradiates to the pelvis and in the right thigh. The positive Rozanov's sign - painfulness during palpation in the right Pti triangle is characteristic. In transition of inflammatory process on an ureter and kidney in the urines analysis red corpuscles can be found.

The pelvic location of appendix. In such patients the pain is localized above the right Poupart's ligament and above pubis. At the very low placing of appendix at the beginning of disease the reaction of muscles of front abdominal wall on an inflammatory process can be absent. With transition of

inflammation of an urinary bladder or rectum either the dysuric signs or diarrhea develop. Distribution of process on internal genital organs provokes signs characteristic of their inflammation.

Appendicitis at the medial placing of appendix. The appendix in patients with such pathology is located between the loops of intestine, which is the large field of irritation of peritoneum. At these anatomic features mesentery is pulled in the inflammatory process, acute dynamic of the intestinal obstruction develops in such patients. The pain in the abdomen is intensive, widespread, the expressed tension of muscles of abdominal wall develops.

For the subhepatic location of appendix the pain is characteristic in right hypochondrium. During palpation painfulness and tension of muscles can be marked.

Left-side appendicitis appears infrequently and, as a rule, in case of the reverse placing of all organs; however it can occur at a mobile blind gut. In this situation all signs which characterize acute appendicitis will be exposed not on the right, as usually, but on the left.

Special examinations:

White Blood Cell Count (WBC). The white blood cell count in the blood usually becomes elevated with infection. Unfortunately, appendicitis is not the only condition that causes elevated white blood cell counts. Almost any infection or inflammation can cause this count to be abnormally high. Therefore, an elevated white blood cell count alone cannot be used as a sign of appendicitis.

Urinalysis. Urinalysis is a microscopic examination of the urine that detects red blood cells, white blood cells and bacteria in the urine. Urinalysis usually is abnormal when there is inflammation or stones in the kidneys or bladder. The urinalysis also may be abnormal with appendicitis because the appendix lies near the ureter and bladder. If the inflammation

of appendicitis is great enough, it can spread to the ureter and bladder leading to an abnormal urinalysis. Therefore, a normal urinalysis suggests appendicitis more than a urinary tract problem.

Abdominal X-Ray (AXR). An abdominal X-ray may detect the fecalith that may be the cause of appendicitis. This is especially true in children.

Chest films may be performed to exclude any disease of the base of the right lung as disease in this area may irritate the spinal nerve to simulate the symptoms of appendicitis.

Ultrasound (US). Ultrasound can identify an enlarged appendix or an abscess. Nevertheless, during appendicitis, the appendix can be seen in only 50% of patients. Ultrasound also is helpful in patients with renal colic and in women.

Ultrasound target sign of acute appendicitis can be detected (fig. 5). Transverse ultrasound scan through an inflamed appendix shows an intact echogenic submucosal layer and a fluid-filled lumen (F), resulting in a “target” appearance.

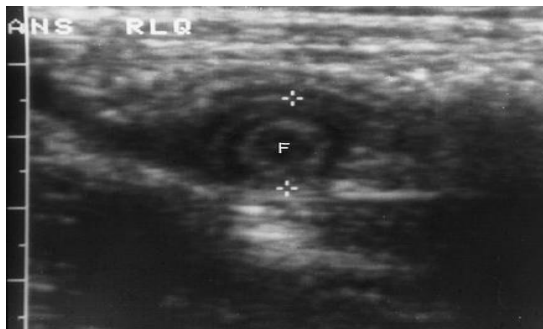


Figure 5 – Acute appendicitis with target sign
(https://www.google.com/search?rlz=1C1GCEB_enUA1010UA1010&)

Computerized tomography (CT) scan. In patients who are not pregnant, a CT scan of the area of the appendix is useful in diagnosing appendicitis and periappendicular

abscesses (fig. 6) as well as in excluding other diseases inside the abdomen and pelvis that can mimic appendicitis.

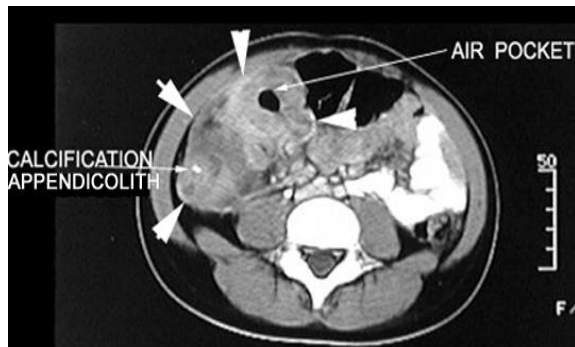


Figure 6 – Acute appendicitis with periappendicular abscesses (https://www.google.com/search?rlz=1C1GCEB_enUA1010UA1010&)

Laparoscopy. Laparoscopy is a surgical procedure in which a small fiber optic tube with a camera is inserted into the abdomen through a small puncture made on the abdominal wall. Laparoscopy allows a direct view of the appendix as well as other abdominal and pelvic organs. If appendicitis is found, the inflamed appendix can be removed with the laparoscope.

Differential diagnosis of acute appendicitis:

Food toxicoinfection. Complaints for pain in the epigastric area of the intermittent character, nausea, vomiting and liquid emptying are the first signs of disease. The state of patients progressively gets worse from the beginning. Next to that, it is succeeded to expose that a patient used meal of poor quality. However, here patients do not have phase passing, which is characteristic of acute appendicitis, and clear localization of pain. Defining the symptoms of irritation of peritoneum are not succeeded, the peristalsis of intestine is increased.

Acute pancreatitis. In anamnesis in patients with this pathology there is a gallstone disease, violation of diet and use

of alcohol. Their condition from the beginning of a disease is heavy. Pain is considerably more intensive, than during appendicitis, and is concentrated in the upper half of abdomen. Vomiting is frequent and does not bring to the recovery of patients.

Acute cholecystitis. The high placing of vermiform appendix in the right half of abdomen during its inflammation can cause the clinic somewhat similar to acute cholecystitis. But unlike appendicitis, in patients with cholecystitis the pain is more intensive, has cramp-like character, is localized in right hypochondrium and irradiate in the right shoulder. Also the epigastric phase is absent. The attack of pain can arise after the reception of spicy food and, is accompanied by nausea and frequent vomiting by bile. In anamnesis patients often have information about a gallstone disease. During examination intensive painfulness is observed in right hypochondrium, increased gallbladder and positive Murphy's and Ortner's signs.

Perforated peptic ulcer. Diagnostic difficulties during this pathology arise up only on occasion. They can be in patients with the covered perforation, when portion of gastric juice flows out in an abdominal cavity and stays too long in the right iliac area, or in case of atypical perforations. On the abdominal X-ray gram under the right copula of diaphragm free gaze can be found.

The apoplexy of ovarya more frequent is with young women and, as a rule, on 10–14 day after menstruation. Pain appears suddenly and irradiate in the thigh and perineum. At the beginning of disease there can be a collapse. However, the general condition of patients suffers insignificantly. When not enough blood was passed in the abdominal cavity, all signs of pathology of abdominal cavity organs calm down after some time. Signs, which are characteristic of acute anemia, appear at considerable hemorrhage. Abdomen more frequent is soft and

painful down, (positive Kulenkampff's sign: acute pain during palpation of stomach and absent tension of muscles of the front abdominal wall).

Extra-uterine pregnancy. A necessity to differentiate acute appendicitis with the interrupted extra-uterine pregnancy arises, when during the examination the patient complains at the pain only down in the stomach, more to the right. Taking it into account, it is needed to remember, that at extra-uterine pregnancy a few days before there can be intermittent pain in the lower part of the abdomen, sometimes excretions of "coffee" color appear from vagina. In anamnesis often there are the present gynecological diseases, abortions and pathological passing of pregnancy. For the clinical picture of such patient inherent sudden appearance of intensive pain in lower part of the abdomen. Often there is a brief loss of consciousness. During palpation considerable painfulness is localized lower, than at appendicitis, the abdomen is soft, the positive Kulenkampff's sign is determined. Violations of menstrual cycle testify for pregnancy, characteristic changes are in milk glands, vagina and uterus. During the vaginal examination it is sometimes possible to palpate increased tube of uterus. The temperature of body more frequently is normal. If hemorrhage is small, the changes in the blood test are not present. The convincing proof of the broken extra-uterine pregnancy is the dark color of blood, taken at puncture of back fornix of vagina.

Right-side kidney colic. For this disease pain at the level of kidney and in lumbus is inherent, hematuria and dysuric signs which can take place at the irritation of ureter by the inflamed appendix. Intensity of pain in kidney colic is one of the basic differences from acute appendicitis. Pain at first appears in lumbus and irradiate downward after passing of ureter in genital organs and front surface of the thigh. In diagnostics urogram survey is important, and if necessary –

chromocystoscopy. Absence of function of right kidney to some extent allows eliminating the diagnosis of acute appendicitis.

Diabetic abdomen – indicates abdominal pain and vomiting which precede coma.

Treatment of acute appendicitis:

Acute appendicitis is treated by surgery to remove the appendix (appendectomy). The operation may be performed through a standard small incision in the right lower part of the abdomen, or it may be performed using a laparoscopy, which requires three to four smaller incisions. In patients with diffuse or general peritonitis middle line laparotomy is preferred.

Do not administer analgesics and antipyretics to patients with suspected appendicitis who have not been evaluated by the surgeon.

Preparation of patients undergoing appendectomy is similar for both open and laparoscopic procedures. Perform complete routine laboratory and radiologic studies before intervention. Venous access must be obtained in all patients diagnosed with appendicitis. Venous access allows administration of isotonic fluids and broad-spectrum intravenous antibiotics prior to the operation. The abdomen is washed, antiseptically prepared, and then draped.

Open appendectomy:

Surgical incisions for open appendectomy are:

– line oblique incision over the McBurney point (i. e., two thirds of the way between the umbilicus and the anterior superior iliac spine);

– vertical incisions (i.e., the Battle pararectal) are rarely performed because of the tendency for herniation;

– middle line laparotomy.

After cutting of peritoneum abdominal cavity is opened. Note the character of any peritoneal fluid to help confirm the diagnosis and then suction it from the field. If it is purulent, collect and culture the fluid.

The convergence of taenia coli is detected at the base of the appendix, beneath the Bauhin valve (i. e., the ileocaecal valve), and the appendix is then viewed. If the appendix is hidden, it can be detected medially by retracting the caecum and laterally by extending the peritoneal incision.

After exteriorization of the appendix, the mesoappendix is held between clamps, divided, and ligated. The appendix is clamped proximally about 5 mm above the caecum to avoid contamination of the peritoneal cavity and is cut above the clamp by a scalpel. The appendix must be ligated to prevent bleeding and leakage from the lumen. The appendix may be inverted into the caecum with the use of a purse – string suture or Z – kind stitch.

The caecum is placed back into the abdomen. The abdomen is irrigated. When evidence of free perforation exists, peritoneal lavage with several liters of warm saline is recommended. After the lavage, the irrigation fluid must be completely aspirated to avoid the possibility of spreading infection to other areas of the peritoneal cavity.

Using of a drain is not commonly required in patients with acute appendicitis, but obvious abscess with gross contamination requires drainage.

The wound closure begins by closing the peritoneum with a running suture. Then, the fibers of the muscular and fascial layers are reapproximated and closed with a continuous or interrupted absorbable suture. Lastly, the skin is closed with subcutaneous sutures or staples.

In cases of perforated appendicitis, some surgeons leave the wound open, allowing for secondary closure or a delayed primary closure until the fourth or fifth day after operation. Other surgeons prefer immediate closure in these cases.

Laparoscopic appendectomy. According to the preferences of the surgeon, a short supraumbilical incision is

made to allow the placement of a Hasson cannula or Veress needle. Pneumoperitoneum (10–14 mm Hg) is established and maintained by insufflating carbon dioxide. Through the access, a laparoscope is inserted to view the entire abdomen cavity. A 10-mm port is inserted through supraumbilical incision for camera. Another two 5-mm ports are placed in the right and left inguinal regions for manipulations. The appendix is grasped and retracted upward to expose the mesoappendix. The mesoappendix is divided, ligated or coagulated. The appendix may be transected with a linear Endostapler, or, alternately, the base of the appendix may be suture ligated in a usual manner to that in an open procedure. The appendix is removed using a laparoscopic pouch to prevent wound contamination.

Postoperative treatment:

Administer intravenous antibiotics postoperatively. The length of administration is based on the operative findings and the recovery of the patient. In complicated appendicitis, antibiotics may be required for many days or weeks.

Antiemetic's and analgesics are administered to patients experiencing nausea and wound pain. The patient is encouraged to ambulate early.

In patients with complicated appendicitis, a clear liquid diet may be started when bowel function returns.

Follow-up care:

After hospital discharge, patients must have a light diet and limit their physical activity for a period of 2–6 weeks based on the surgical approach (i. e., laparoscopic or open appendectomy). The patient should be evaluated by the surgeon in the clinic to determine improvement and to detect any possible complications:

- wound infection especially in patients with gangrenous or perforated appendicitis;
- ileus;
- cecal fistulas;
- pelvic or abdominal abscess.

Complications of acute appendicitis:

Appendicular mass. In majority of cases as soon as the appendix becomes gangrenous, omentum and coils of small intestine cover the appendix all around. There is no discrete collection of pus inside. This is an attempt of the nature to prevent general peritonitis even if rupture of the appendix occurs. Usually such appendicular mass develops on die 3rd day after the commencement of an attack of acute appendicitis. This is a tender mass on the right iliac fossa. This mass usually resolves by conservative treatment. In untreated cases of when the patient does not react to the conservative treatment such appendicular mass may turn into an appendicular abscess and becomes larger in size.

Treatment of the appendicular mass. In these cases conservative treatment should be started immediately. Nature has already localized, the lesion and it is better not to disturb such localization. Surgery at this stage is difficult and dangerous as it is difficult to find appendix due to adhesions and ultimately fecal fistula may form.

Conservative treatment includes intravenous fluid with dextrose saline and Ringer solution as and when required; nasogastric aspiration; antibiotic therapy. A broad spectrum antibiotic should be given intramuscularly. Metronidazole may be given intravenously.

The conditions for stopping the conservative treatment are: a) arising pulse rate; b) vomiting or increase in gastric aspiration; c) increasing abdominal pain - suggesting spreading peritonitis; d) increasing of the size of the mass.

About 90% of cases resolve without any problem. The patient is kept under observation for further 4 to 5 days after resolution of the mass. Patient is instructed to have appendectomy done 6 to 8 weeks after his discharge.

Appendicular abscess. A progressive supportive process in an appendicular mass forms an appendicular abscess walled off by the omentum, inflamed caecum and coils of small intestine. Such abscess may follow rupture of the appendix. The most common site of the abscess is in the lateral part of the iliac fossa (from retrocecal appendicitis). The second common position is in the pelvis. In untreated cases lethal form of peritonitis is produced by secondary rupture of appendicular abscess.

At abscessing of infiltrate the condition of a patient gets worse, the symptoms of acute appendicitis become more expressed, the temperature of body, which in most cases gains hectic character, rises, the fever appears. Next to that, pain in the right iliac area increases. Painful formation is felt there. In the blood test high leukocytosis is present with the acutely expressed change of leukocyte formula to the left. The temperature of body rises to 38,0–39,0 °C. During the rectal examination the weakened sphincter of anus is found. The front wall of rectum at first is only painful, and then its overhanging is observed as dense painful infiltrate.

Treatment of appendicular abscess. Immediate drainage under antibiotic cover is the treatment of choice. Appendectomy is not performed. A pelvic abscess may be drained in the female into the vagina and in the male into the rectum. If the appendix is not removed when the abscess is drained, interval of appendectomy should be done 6 to 8 weeks after.

Pylephlebitis. Ascending septic thrombophlebitis of portal venous system (pylethrombophlebitis) is a grave but rare complication of gangrenous appendicitis. Septic clots from involved mesenteric veins produce multiple pyogenic abscesses in the liver. It is more frequent in patients with acute retrocecal

appendicitis. It is heralded by chills, spiking fever, right upper quadrant pain and jaundice. Pylephlebitis is a complication of both appendicitis and after-operative period of appendectomy.

In case with rapid passing of disease the icterus appears, the liver is increased, kidney-hepatic insufficiency makes progress, and patients die in 7–10 days from the beginning of disease.

At gradual subacute development of pathology the liver and spleen is increased in size, and after the septic state of organism ascites arises.

Peritonitis. Peritonitis is happened when bacterial and other contents of the appendix leak into the abdomen.

CHAPTER 2. ACUTE PERITONITIS

Inflammation and 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.

Relevant anatomy. The peritoneum is the largest and most complex serous membrane in the body. It forms a closed sac by lining the interior surfaces of the abdominal wall (anterior and lateral), by forming the boundary to the retroperitoneum (posterior), by covering the extraperitoneal structures in the pelvis (inferior), and by covering the undersurface of the diaphragm (superior). This parietal layer of the peritoneum reflects onto the abdominal visceral organs to form the visceral peritoneum. It thereby creates a potential space between the 2 layers (i. e., the peritoneal cavity).

The peritoneum consists of a single layer of flattened mesothelial cells over loose areolar tissue. The loose connective tissue layer contains a rich network of vascular and lymphatic capillaries, nerve endings, and immune-competent cells, particularly lymphocytes and macrophages. The peritoneal surface cells are joined by junctional complexes, thus forming a dialyzing membrane that allows passage of fluid and certain small solutes. Pinocytotic activity of the mesothelial cells and phagocytosis by macrophages allow for clearance of macromolecules.

Normally, the amount of peritoneal fluid present is less than 50 ml, and only small volumes are transferred across the considerable surface area in a steady state each day. The peritoneal fluid represents a plasma ultrafiltrate, with electrolyte and solute concentrations similar to that of neighboring interstitial spaces and a protein content of less than 30 g/L, mainly albumin. In addition, peritoneal fluid contains small numbers of desquamated mesothelial cells and various numbers and morphologies of migrating immune cells (reference range is < 300 cells/ml, predominantly of mononuclear morphology).

The organs are divided into:

- intraperitoneal (stomach, small bowel, colon, sigmoid colon);
- mesoperitoneal (liver, ascending and descending colon);
- extraperitoneal.

Functions of peritoneum:

1. Protection. Attachments of protection are:

- mechanical;
- cellular immunity;
- humoral immunity;

– greater omentum often covers wound, organ perforation, place of perforation;

– peritoneal exudation has antibacterial properties.

2. Exudation.

3. Resorption.

4. Interoreception.

Parietal peritoneum is innervated by intercostal nerves. After irritation of parietal peritoneum visceromotor reflex has appeared. As a result patient has local pain and mussel's tenderness.

Visceral peritoneum is innervated by sympatic and parasympatic nerves. Therefore visceral pain isn't localized. Doctors have to remember, that inflammation of pelvic peritoneum (it doesn't have somatic innervations) isn't accompanied with mussel's tenderness.

5. Plastic function. After traumatic injury or starting of inflammation layers of fibrin have formed. They confine infection.

Classifications:

The first classification of peritonitis was suggested by Miculich (1886): 1) septic, 2) purulent, 3) progressive fibrino – purulent, 4) local. In 1912 Grekov defined the phases of peritonitis: early (1–2 days), late (3–5 days), final (6–21 days).

Modern classification of acute peritonitis

(Shalimov A.A., 1981)

According to origin

A) Primary. It occurs in the absence of an apparent intra-abdominal source of infection and is observed almost exclusively in patients with ascites formation from chronic liver disease.

B) Secondary. The common etiologic entities of secondary peritonitis include: esophagus Boerhaave syndrome (spontaneous rupture), malignancy, trauma, iatrogenic, peptic ulcer perforation, cholecystitis, acute pancreatitis, bowel

perforation, ischemic bowel, strangulated hernia, bowel obstruction, diverticulitis, ulcerative colitis and Crohn disease, appendicitis.

According to aetiology:

A) Microflora of the digestive tract (E. coli, Enterococcus, Pseudomonas, Proteus, Streptococcus, Staphylococcus, Anaerobic infection).

B) Microflora non-connected with the digestive tract (tuberculous infection, gonococcal infection).

C) Aseptic (pancreatogenic, bilious).

D) Carcinomatous.

E) Parasitogenic.

According to cause:

A) Traumatic.

B) Postoperative.

C) Inflammatory.

D) Perforated.

According to character of exudate:

A) Serous.

B) Serofibrinous.

C) Fibrinous.

D) Fibrinopurulent.

E) Purulent.

F) Putrefactive.

According to spreading of infection:

A) Local (inflammatory process is localized only in 1 anatomy region).

B) Diffuse (inflammatory process is localized in 2–5 anatomy regions).

C) Total (inflammatory process is localized in 6–9 anatomy regions).

Phases of peritonitis:

A) Reactive (< 24 hours).

B) Toxic (24–72 hours).

C) Terminal (> 72 hours).

Pathophysiology. Peritonitis is a serious manifestation of the surgical infection. The leading parts are: a) pathogenic microflora; b) intoxication; c) hypovolemia; d) deep metabolic disturbances.

The first phase – reactive phase:

1. From the moment the aggressive factor has gotten into abdominal cavity, the signs of inflammation (edema, hyperemia, and exudation) start.

10 minutes – appearance of exudate into abdominal cavity.

2 hours – formation of adhesions between peritoneum, bowels and large omentum starts.

18 hours – formation of friable mass has been completed.

2. Activation of function of hypothalamo-pituitary-adrenal axis.

3. Activation of biologically active substances: kinins, biogenic amines. They control blood circulation and intestinal motor function.

4. Activation of immune response.

5. Starting of disturbances of microcirculation.

6. Development of adynamic ileus. But barrier function of bowel wall is survived.

The second phase – toxic phase:

1. The leading factor at this stage is intoxication. Exactly intoxication usually determines the result of treatment. The constituents of intoxication are:

– peritoneal exudate which is absorbed;

– microflora (exo- endotoxins);

– proteolytic enzymes, which are released after destruction of leucocytes (cathepsins);

– toxic substances, which are produced and absorbed into parietic bowels (phenols, skatoles).

2. Adynamic ileus. Distended bowel wall loses barrier function and translocation of microorganisms starts. Free liquid is collected into intestinal lumen– “sequestration of liquid into the third space” (transudate, digestive juice).

3. The organism can't limit the infection and inflammatory process spreads into all abdominal cavity.

4. Immune response is depressed.

The third phase – terminal phase:

This is a stage of septic shock and multiple organ failure. The changes are irreversible. Intoxication, disturbances of metabolism and hemodynamic are fatal.

Toxic hepatitis: liver is the first organ which gets toxic substances per portal vein system. Liver metabolic disorders start: protein and pigmental metabolism, function of disintoxication is decreasing.

Toxic nephropathy: proteinuria, microhematuria, azotemia.

Toxic myocardiodystrophy: hollowness of tones, tachycardia, extrasystole, ST segment depression.

Toxic alteration of lungs: respiratory distress syndrome (“shock lung”).

Clinical diagnostic of acute peritonitis:

Clinical manifestations of acute peritonitis depend on:

- disease which causes the peritonitis;
- stage of peritonitis;
- spreading of peritonitis;
- characteristics of microflora;
- activity of immune response.

The diagnosis of peritonitis is usually clinical. Essentially, all patients present with some degree of abdominal pain. This pain may be acute or more insidious in onset. Initially, the pain is often dull and poorly localized (visceral peritoneum) and then progresses to steady, severe, and more localized pain (parietal peritoneum). If the infectious process is

not contained, the pain becomes diffuse. In certain disease entities (e. g., gastric perforation, severe acute pancreatitis, intestinal ischemia), the abdominal pain may be generalized from the beginning.

Anorexia and nausea are frequently present. Vomiting may occur because of the underlying visceral organ pathology or secondary to the peritoneal irritation.

On physical examination, patients with peritonitis most often appear unwell and in acute distress. Fever with temperatures that can exceed 38 °C is usually present, but patients with severe sepsis may present with hypothermia. Tachycardia is caused by the release of inflammatory mediators and intravascular hypovolemia caused by anorexia and vomiting, fever, and third-space losses into the peritoneal cavity and bowels. With progressive dehydration, patients may become hypotensive, they may demonstrate decreased urine output, and, with severe peritonitis, they may present septic shock.

On abdominal examination, essentially all patients demonstrate tenderness to palpation. In most patients (even with generalized peritonitis and severe diffuse abdominal pain), the point of maximal tenderness roughly overlies the pathologic process (i. e., the site of maximal peritoneal irritation). Patients with severe peritonitis often avoid all motion and keep their hips flexed to relieve the abdominal wall tension.

The abdomen is often distended, with hypoactive-to-absent bowel sounds. This finding reflects a generalized ileus and may not be present if the infection is well localized.

Occasionally, the abdominal examination reveals an inflammatory mass. Examining the abdomen of a patient with peritonitis and mass, the patient should be supine to left lateral position for better relaxation of the abdominal wall.

Rectal examination often elicits increased abdominal pain, particularly with inflammation of the pelvic organs but rarely indicates a specific diagnosis. A tender inflammatory mass toward the right may indicate appendicitis, and anterior fullness and fluctuation may indicate a pelvic abscess. In female patients, vaginal and bimanual examination may lead to the differential diagnosis of pelvic inflammatory disease.

A complete physical examination is important. Thoracic processes with diaphragmatic irritation (e.g., empyema), extraperitoneal processes (e.g., pyelonephritis, cystitis, acute urinary retention), and abdominal wall processes (e. g., infection, rectus hematoma) may mimic certain signs and symptoms of peritonitis. Always examine the patient carefully for the presence of external hernias to rule out intestinal incarceration.

Remember that the presentation and the findings on clinical examination may be entirely inconclusive or unreliable in patients with significant immunosuppression (e. g., severe diabetes, steroid use, posttransplant status, HIV), in patients with altered mental state (e. g., head injury, toxic encephalopathy, septic shock, analgesic agents), in patients with paraplegia, and in patients of advanced age.

The Rozanov sign. The patient lies on his back. You ask him to breathe deep. After that you ask the patient to extra breathe. The past step is impossible because of pain, because it is necessary to stretch the anterior abdominal wall muscle. This sign help you to differentiate the abdomen and pleural (pleurisy, pneumonia) inflammation.

The Mendel sign. The light superficial anterior abdominal wall precaution occurs the fluid collection or the source of inflammation.

The Razdolsky sign. The “hammers” anterior abdominal wall percussion occurs the source of inflammation..

The Drahter sign (pediatric surgery). You hold by one hand the foot of patient's legs. You free hand percusses the patient's heel. The child holds his arms on the area of inflammation.

The Winter sign. The abdominal wall does not participate in breathing because of the dramatic tension.

The Blumberg sign. You gradual press by fingers on the abdominal wall and patient feels a pain (the Mortol sign). After that you quickly, but not acutely, take you hand away. Strengthening of pain is considered as a positive symptom in that place. Obligatory there is a tension of front abdominal wall muscles. Absence of front abdominal wall muscles tension usually demonstrates the intraabdominal bleeding (the Kulenkampf sign).

The Voskresensky ("the shirt") sign. You move you hand from epigastric area (from xiphoid process) down (to the right, left iliac regions or to the pubis). The inflamed peritoneum, free abdominal exudate collection manifest by increasing of pain.

The Mortol sign. You gradual press by fingers on the abdominal wall and patient feels a pain.

The Madelung sign. The difference in body axillar and rectal temperature is more than 2 °C.

The "Douglas cry". The pain in the finger rectal examination or bimanual gynecologic examination.

The "Hippocrates face". Patient face is pale, covered with cold sweat, lines are sharpened.

The subphrenical space abscess:

The Senator sign. The m. rector spinae tension dues to vertebral immovability.

The Duchenne sign. The epigastric area is involving during inspiration and protruding during expiration.

The Litten sign. The involving of intercostal spaces during inspiration.

Lab. Studies:

CBC with differential, serum electrolytes with renal function. Most patients with intra-abdominal infections demonstrate leukocytosis ($> 11,000$ cells/ml) with a shift to the immature forms on the differential cell count. Patients in severe sepsis, patients who are immunocompromised, and patients with certain types of infections (e. g., fungal, cytomegaloviral) may demonstrate absence of leukocytosis or leucopenia.

Blood chemistry may reveal dehydration and acidosis.

Liver function tests if clinically indicated.

Amylase and lipase if pancreatitis is suspected.

UA is essential to rule out urinary tract diseases (e. g., pyelonephritis, renal stone disease); however, patients with lower abdominal and pelvic infections often demonstrate WBC in the urine and microhematuria.

In patients with diarrhea, evaluate a stool for specific culture (i. e., Salmonella, Shigella, cytomegalovirus) if the patient's history suggests infectious enterocolitis.

Aerobic and anaerobic blood cultures.

Peritoneal fluid evaluation (i. e., paracentesis, aspiration of abdominal fluid collections, intraoperative peritoneal fluid cultures).

Imaging studies:

AXR. Plain films of the abdomen (e. g., supine, upright, and lateral decubitus positions) are often the first imaging studies obtained in patients presenting with peritonitis. Their value in reaching a specific diagnosis is limited.

Free air is present in most cases of anterior gastric and duodenal perforation but is much less frequent with perforations of the small bowel and colon. Remember that the presence of free air is not mandatory with visceral perforation and that small amounts of free air are missed easily on plain films.

US. Abdominal US may be helpful in the evaluation of right upper quadrant (e. g., perihepatic abscess, cholecystitis, biloma, pancreatitis, pancreatic pseudocyst), right lower quadrant, and pelvic pathology (e. g., appendicitis, tubo-ovarian abscess, Douglas pouch abscess), but the examination is sometimes limited because of patient discomfort, abdominal distension, and bowel gas interference.

Ultrasonography may detect increased amounts of peritoneal fluid, but its ability to detect quantities of less than 100 ml is limited. The central (perimesenteric) peritoneal cavity is not visualized well with transabdominal ultrasonography. Examination from the flank or back may improve the diagnostic, and providing the ultrasonographer with specific information of the patient's condition and the suspected diagnosis before the examination is important. With an experienced ultrasonographer, a diagnostic accuracy of greater than 85 % has been reported in several series.

Over the past several years, ultrasound-guided aspiration and placement of drains has evolved into a valuable tool in the diagnosis and treatment of abdominal fluid collections.

CT of the abdomen and pelvis remain the diagnostic study of choice for peritoneal abscess and the related visceral pathology. CT scan is indicated in all cases where the diagnosis cannot be established on clinical grounds and findings on abdominal plain films. Whenever possible, the CT scan should be performed with enteral and intravenous contrast. CT scans can detect small quantities of fluid, areas of inflammation and other GI tract pathology, with sensitivities that approach 100%.

Peritoneal abscesses and other fluid collections may be aspirated for diagnosis and drained under CT guidance.

Nuclear medicine scans (e.g., gallium Ga 67 scan, indium In 111-labeled autologous leucocyte scan, technetium Tc 99m-iminoacetic acid derivative scan).

These diagnostic studies have little use in the initial evaluation of patients with suspected peritonitis or intra-abdominal sepsis. They are most frequently used in the evaluation of fever of unknown origin or in patients with persistent fever despite adequate antibiotic treatment and negative CT scan findings.

Magnetic resonance imaging (MRI) is an emerging imaging modality for the diagnosis of suspected intra-abdominal abscesses.

Contrast studies. Conventional contrast studies (upper gastrointestinal tract and colorectal contrast enema, fistulogram, contrast studies of drains) are reserved for specific indications in the setting of suspected peritonitis or peritoneal abscess.

Steps of treatment:

Patient with acute peritonitis is treated surgically after not long time (some hours) preoperative preparation. Contraindications for surgical treatment are absent.

The general principles guiding the treatment of intra-abdominal infections are:

- 1) to control the infectious source;
- 2) to eliminate bacteria and toxins;
- 3) to maintain organ system function;
- 4) to control the inflammatory process.

1. Care for patients:

- local hypothermia,
- gastric decompression by nasal tube,
- preparation of the operative field.

2. Correction of dehydration under central venous pressure (CVP) control is conducted with solutions of electrolytes and glucose solution.

3. Antibiotic therapy starts during rehydration. Use empiric broad-spectrum parenteral antibiotic coverage. (Second-generation cephalosporin, Penicillins with anaerobic

activity, Quinolones with anaerobic activity, Quinolone and Metronidazole, Aminoglycoside and Metronidazole).

4. Analgesic agents or NSAID.

5. Prevention of thromboembolic complications in the presence of risk factors.

6. Correction of oxygen exchange with Medical oxygen inhalation through a nasal catheter.

7. Inotropic myocardium support (Dopamine 0.5% solution).

8. Stimulation of diuresis (with CVP = 80-120 mm of water).

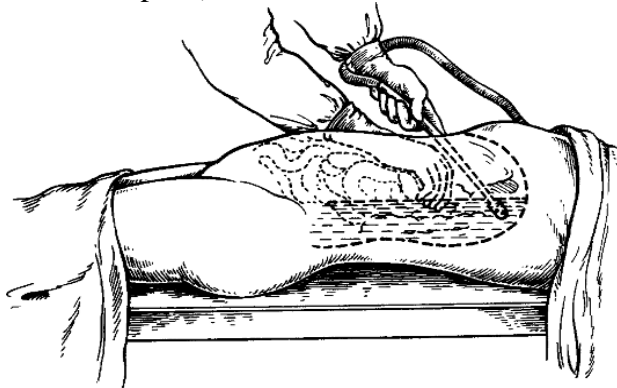
9. Standard premedication on the operating table: Atropine solution 0.1% intravenous injection.

10. Multicomponent intravenous anesthesia with muscle relaxation and artificial ventilation of lungs.

11. Operation

– Surgical access (middle line laparotomy, laparoscopy).

– Primary abdominal cavity sanitation (exudate evacuation, antiseptics).

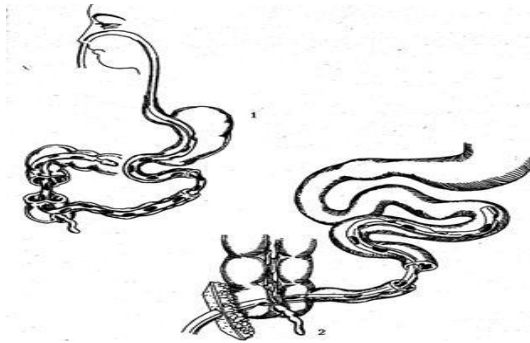


– Elimination of the causes of peritonitis (cholecystectomy, appendectomy, suturing of organ perforation and other interventions).

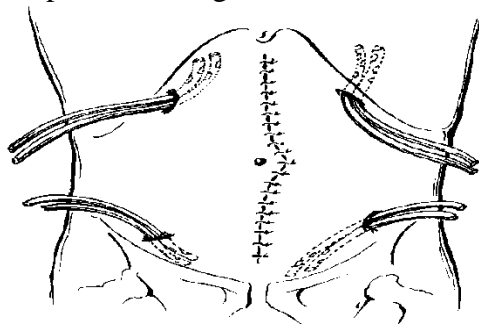
– Small bowel intubation: removing of toxic bowel content, stabilization of bowel motor function, enteral nutritional supporting, prevention of postoperative adhesion bowel obstruction.

Main techniques of intubation:

- 1 – nasointestinal intubation;
- 2 – intubation using caecostomy.



– Final abdominal cavity sanitation (in all 15l of antiseptics) and 4 – points drainage



– Job completion using Mannheim Peritonitis Index (MPI):

Risk factor	Score
Age > 50 years	5
Female gender	5

Organ failure	7
Malignancy	4
Pre-operative duration of peritonitis >24 hours	4
Origin of sepsis non colonic	4
Diffuse generalized peritonitis	6
Exudate	
Clear	0
Fibrinous, purulent	6
Fecal	12

MPI provides for three degrees of peritonitis gravity.

I degree – index less than 21 – closure of operative wound.

II degree – index from 21 to 29 – closure of operative wound and laparostomy for indications.

III degree – more than 29 – only second-look operation or laparostomy.

12. Therapy after surgery includes articles 2 – 8 and in addition:

- parenteral nutrition during 3 days after surgery, enteral nasointestinal tube nutrition after;

- stimulation of peristalsis: Metoclopramide 0.5 % solution of 2 ml / m 3 times; cleansing enema;

- recovery of lung function: breathing exercises and aerosol therapy (3 days).

Follow-up care:

The follow-up care is directed mostly by specifics of the underlying disease process and the presence or absence of chronic complications (e. g., enterocutaneous fistulae). Patients with simple peritoneal infections after appendicitis or cholecystitis are usually cured and do not require long-term follow-up care. Patients with peritoneal operations for perforated peptic ulcer disease, Crohn disease, pancreatitis, and

others often require lifelong medical therapy and treatment of recurrent complications.

CHAPTER 3. ACUTE CHOLECYSTITIS

Acute cholecystitis is nonspecific inflammation of gallbladder. After frequency this disease takes second place after acute appendicitis and makes about 10 % in relation to all acute surgical diseases of organs of abdominal cavity. The bile ducts anatomy is demonstrated at fig.7.

Acute cholecystitis occurs most commonly because of an obstruction of the cystic duct from cholelithiasis. Ninety percent of cases involve stones in the cystic duct (i. e., calculous cholecystitis), with the other 10 % representing acalculous cholecystitis.

Acalculous acute cholecystitis is related to conditions associated with biliary stasis, including debilitation, major surgery, severe trauma, sepsis, long-term total parenteral nutrition and prolonged fasting.

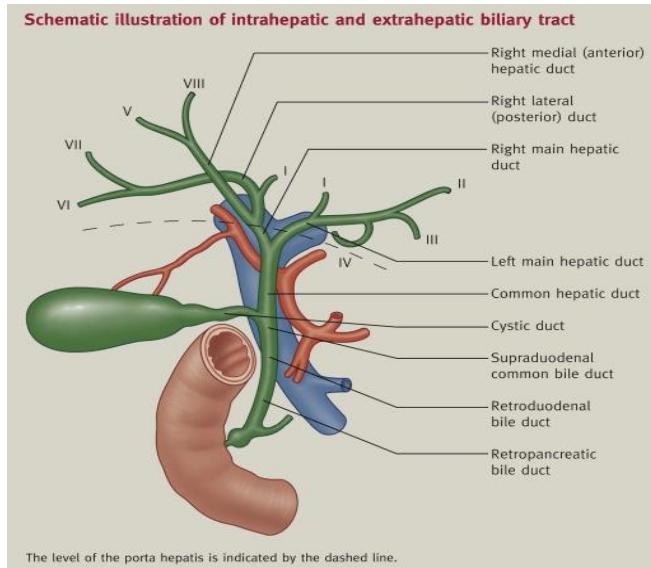


Figure 7 – The bile ducts anatomy
 (<https://els-jbs-prod-cdn.jbs.elsevierhealth.com>)

Although bile cultures are positive for bacteria in 50–75 % of cases, bacterial proliferation may be a result of cholecystitis and not the precipitating factor.

Risk factors for cholecystitis include increasing age, female sex, certain ethnic groups, obesity or rapid weight loss, drugs and pregnancy.

As a result inflammatory process develops from simple (catarrhal or superficial) cholecystitis till destructive cholecystitis (phlegmonous, gangrenous) and complicated cholecystitis.

Classification. Acute cholecystitis is divided into:

- I. Acute calculous cholecystitis.
- II. Acute non-calculous cholecystitis.
 1. Catarrhal.
 2. Phlegmonous.
 3. Gangrenous.

4. Complicated:

- a) hydropsy;
- b) empyema;
- c) biliary pancreatitis;
- d) choledocholithiasis;
- e) hepatitis;
- f) cholangitis;
- g) mass;
- h) abscess;
- i) hepatic-kidney insufficiency;
- j) peritonitis (local, general).

Clinical manifestations and diagnostics of acute cholecystitis:

Most patients with acute cholecystitis describe a history of biliary pain. Some patients may have documented gallstones. The disease, as a rule, begins after violation of diet: intake of much of fatty foods.

The pain syndrome:

The disease begins with a sudden pain in the abdomen. It is localized in a right upper quadrant, has moderate or strong intensity, constant character and usually irradiates to the right shoulder, under the right scapular bone, processus xiphoideus of sternum, and sometimes to the area of the heart.

The dispepsy syndrome:

Frequent symptoms which disturb a patient, are nausea, vomiting, at first by gastric content, and later – with a part of bile. Afterwards feelings of swelling of stomach, delay of emptying and gases often join to them.

The intoxication syndrome:

The temperature of body of most patients rises, but it is a low grade fever. The general condition of patients is usually normal and gets worse only in case of growth of destructive

changes in gallbladder. The puls rate is increased according the body temperature.

Percussion and auscultation of abdominal cavity – usually normal dates.

The palpation of abdominal wall:

Painfulness and tension of abdominal wall muscle in the right upper quadrant are detected. During palpation painfulness in the place of crossign of right costal arc with the external edge of direct muscle of abdomen can be observed (the *Kehr's point*). By superficial and deep palpation of right hypochondrium, as a rule, painfulness, increased gallbladder can be exposed.

The Botkin sign. Irradiation of pain in the heart area over the n. phrenicus course in the anterior mediastinum on the front wall of the pericardium.

The Bereznyahovsky – Eleker sign. Irradiation of pain in the supraclavicular area over the course of n. phrenicus, reaching the scapula area and shoulder 1/3 external upper surface.

The Karavanov – Spector (Co-Tui – Meyer – Schlesigner) sign (a sign of acute cholecystitis and local peritonitis). The deformation of abdominal wall (the umbilicus moves to the place of gallbladder existence) as a result of abdominal wall muscles tension and constriction.

The Ronshov sign. The jaundice of skin around umbilicus.

The Sale sign. Easign breathing in the lower part of the right lung (auscultation).

The Ionash sign. Pressure on the mastoid processus in the place of trapezoidal muscle fixation (occipital nerve, a branch of the cervical plexus, location), increases pain in the gallbladder area.

The Mussy-Georgievsky sign. Pressure between the legs of the sternum-clavicular-mastoid muscle to the right, increases pain in the gallbladder area.

The Karavanov sign. Pressure on the chest wall lower the right scapula angle increases pain in the right upper abdominal wall quadrant.

The Boas – Skvyrsky sign. The pain after pressure righter VIII–X thoracic vertebrae on the back.

The Mackenzy sign. Hypersensitivity in the right hypochondria area.

The Lyahovytsky sign. Pressure on the xiphoid process increases pain in the gallbladder area.

The Zakharyn sign. Pressure by finger-tips on the gallbladder area increases pain.

The Ker sign. Moderate finger pressure at the Ker point (the right side of the rectus abdominis muscle near the costal arch) during inspiration, or without it appears or increases pain.

The Ortner sign. Light perpendicular knocking on the right costal arch appears or increases pain in the in the gallbladder area.

The Murphy sign. A patient lies on his back. A doctor puts his left hand on the right costal arch so that the thumb presses abdominal wall in the projection of the gallbladder; the patient is asked to take a deep breath, which is stopped because of pain (gallbladder and liver moves down with friction).

The Obraztsov sign. A sharp pain when a doctor presses by hand under the right costal arch (as palpation of the liver).

The Courvoisier sign. You palpate enlarged, not painful gallbladder in patient with jaundice (sign of distal bile duct obstruction, usually by tumor).

Blumberg's sign may be positive in patients with peritonitis.

In elderly patients, pain and fever may be absent, and localized tenderness may be the only presenting sign.

Patients with acalculous cholecystitis may present similarly to patients with calculous cholecystitis, but acalculous cholecystitis frequently occurs suddenly in severely ill patients without a prior history of biliary colic.

Special examinations

CBC. Patients with uncomplicated cholelithiasis or simple biliary colic typically have normal laboratory test results. Acute cholecystitis is associated with polymorphonuclear leukocytosis.

Choledocholithiasis with acute common bile duct obstruction initially produces an acute increase in the level of liver transaminases (alanine and aspartate aminotransferases), followed within hours by a rising serum bilirubin level. If obstruction persists, a progressive decline in the level of transaminases with rising alkaline phosphatase and bilirubin levels may be noted over several days. Concurrent obstruction of the pancreatic duct by a stone in the ampulla of Vater may be accompanied by increases in circulating lipase and amylase levels.

In patients with suspected gallstone complications, blood tests should include a complete blood count with differential, liver function panel, and amylase and lipase. Repeated testing over hours to days may be useful in evaluating patients with gallstone complications.

US. Ultrasonographic findings that are suggestive of acute cholecystitis include the following: pericholecystic fluid, gallbladder wall thickening greater than 4 mm and sonographic Murphy sign. The presence of gallstones also helps to confirm the diagnosis (fig. 8, 9).

The sensitivity and specificity of *CT/MRI* scans for predicting acute cholecystitis have been reported to be greater than 95 %. Spiral CT scans and MRI have the advantage of being noninvasive, but they have no therapeutic potential and are most appropriate in cases where stones are unlikely.

CT findings suggestive of cholecystitis include wall thickening (> 4 mm), pericholecystic fluid, subserosal edema, intramural gas, and sloughed mucosa (fig. 10).

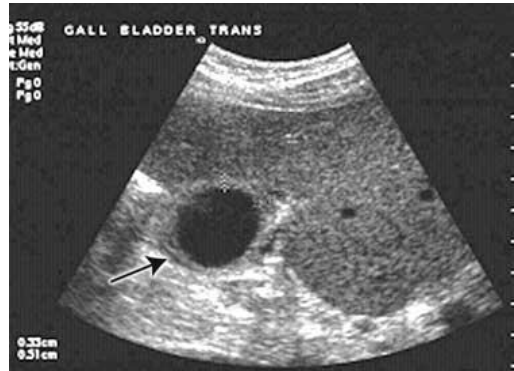


Figure 8 – US examination showed a thickened gallbladder wall and pericholecystic fluid
(<https://www.google.com/search?q=sonographic+murphy+sign&tbm>)

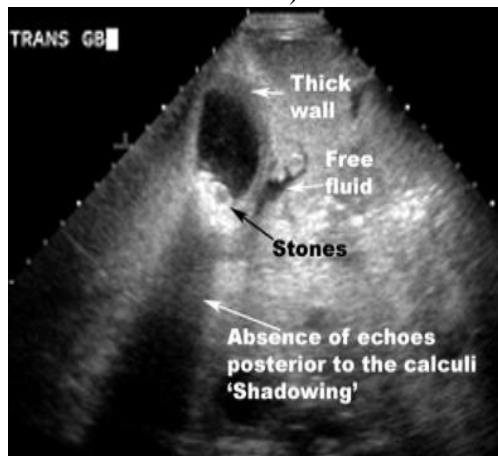


Figure 9 – US findings: thick gallbladder wall, stones in gallbladder, absence of echoes posterior to the calculi
(<https://www.google.com/search?q=sonographic+murphy+sign&tbm>)

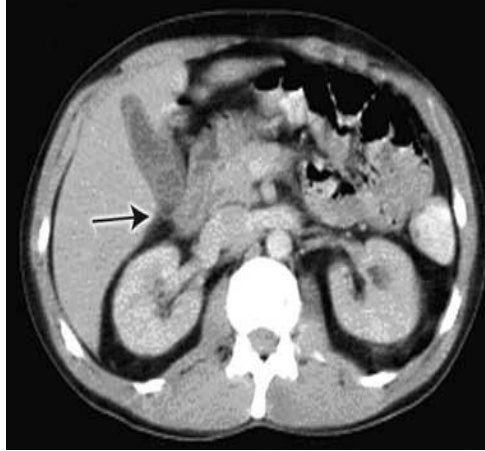


Figure 10 – CT of acute cholecystitis

(<https://www.google.com/search?q=CT+findings+of+cholecystitis>)

Endoscopic retrograde cholangiopancreatography (ERCHPG) may be useful in patients at high risk for common duct gallstones if signs of common bile duct obstruction are present (fig. 11).



Figure 11 – ERCHPG

(<https://www.google.com/search?q=Endoscopic+retrograde+cholangiopancreatography&tbm>)

ERCHPG allows visualization of the anatomy and may be therapeutic by endoscopic papillosphincterotomy + removal of stones from the common bile duct. Disadvantages include the need for a skilled operator, high cost, and complications such as acute pancreatitis, which occurs in 3–5 % of cases.

Differential diagnosis of acute cholecystitis:

Perforated peptic ulcer. For this disease the Mondor's triad (“knife-like” pain, tension of muscles of front abdominal wall and ulcerous anamnesis) and positive *Spizharny's sign* are characteristic (disappearance of hepatic dullness). On the abdominal X-ray gram under the right copula of diaphragm free gaze can be found.

Right-side kidney colic. Pain at right-side kidney colic also can be localized in right hypochondrium. However, it is always accompanied by disorders of urination. Kidney pain often irradiates downward along passing of ureter. For this pathology micro- or macrohematuria, presence of renal concrements exposed at sonography and on the urograms. Absence of renal function during chromocystoscopy can be characteristic.

Acute appendicitis. It is needed always to remember, that the subhepatic location of the pathologically changed appendix is also able to show up pain in right hypochondrium. However, for patients with acute appendicitis beginning of pain in epigastric area, absence of hepatic anamnesis, expressed dyspeptic phenomena, inflammatory changes from the side of gallbladder at sonography are inherent.

Heart attack, myocardial infarction. The so called cholecysto-cardial syndrome which often imitates stenocardia pain, suspicion of heart attack or myocardial infarction can develop. Electrocardiography examination is decisive in establishment of diagnosis. However, laparoscopy is applied in doubtful cases.

Acute pancreatitis. Acute pancreatitis is accompanied by the expressed upper abdominal pain. During palpation in left costal-vertebral corner patients feel painfulness (*Mayo-Robson's sign*).

Treatment of acute cholecystitis:

For acute cholecystitis, initial treatment is conservative:

- local abdominal hypothermia;
- bowel rest;
- intravenous hydration;
- analgesia with not steroid anti-inflammatory drugs (NSAID);
- spasmolytical therapy;
- intravenous antibiotics.

Although not initially an infective process, broad-spectrum antibiotics are used and should be guided by local microbiological policy to target the most common organisms found in the biliary tract. These include *Escherichia coli*, *klebsiella*, *enterobacter* and *enterococcus* species. Anaerobes are less significant but include *clostridium* and *bacteroides* species.

Following successful conservative treatment most patients are discharged from hospital for future elective laparoscopic cholecystectomy. If acute cholecystitis resolves, laparoscopic cholecystectomy may be done 4–6 weeks later. Delayed surgery carries the risk of recurrent biliary complications.

For frail and elderly patients who have only a single attack, or mild recurrent episodes, conservative management may be the mainstay of treatment.

Indications for urgent operation are:

- peritonitis;
- inefficacy of conservative treatment during 24–48 hours (retention of abdominal pain and muscles resistance,

increasing of body's temperature and polymorphonuclear leukocytosis, revealing of Blumberg's sign, jaundice).

In cases of severe inflammation, shock, or if the patient has higher risk for general anesthesia (required for cholecystectomy), the managing physician may elect to have an interventional radiologist insert a percutaneous drainage catheter into the gallbladder ("percutaneous cholecystostomy tube") and treat the patient with antibiotics until the acute inflammation resolves. The patient may later warrant cholecystectomy if their condition improves.

Gallbladder removal, cholecystectomy, can be accomplished via open surgery or a laparoscopic procedure. Laparoscopic procedures can have less morbidity and a shorter recovery stay. Open procedures are usually done if complications have developed or the patient has had prior surgery to the area, making laparoscopic surgery technically difficult. A laparoscopic procedure may also be "converted" to an open procedure during the operation if the surgeon feels that further attempts at laparoscopic removal might harm the patient. Open procedure may also be done if the surgeon does not know how to perform a laparoscopic cholecystectomy. Preparation of patients undergoing cholecystectomy is similar for both urgent open and laparoscopic procedures.

Surgical incisions for open cholecystectomy are: line oblique incision in right subcostal region (is rarely performed because of the tendency for herniation); upper middle line laparotomy. Laparoscopic cholecystectomy is performed using 4 considerably smaller incisions (fig. 12).

The phases of operation are: visual inspection of abdominal cavity and operative confirmation of diagnosis; puncture of gallbladder if it is distending; dissection and visualization of Kalo's trigonum structures; cutting of cystic duct and artery with ligation or clipping (fig. 13); removing of

gallbladder; control of bleeding and bile leak; sanitation and drainage of abdominal cavity.

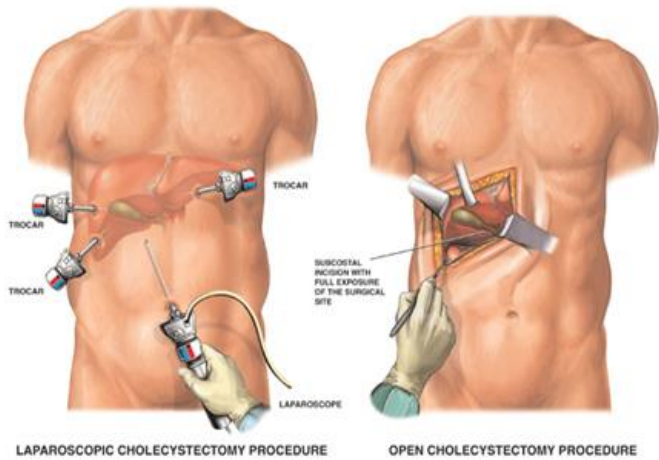


Figure 12 – Open and laparoscopic procedures

<https://www.google.com/search?q=Open+and+laparoscopic+procedures&tbm=isch&ved>

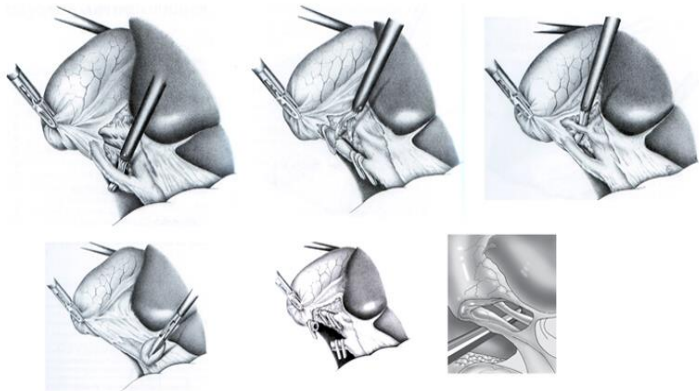


Figure 13 – Dissection of Kalo's trigonum structures

<https://www.google.com/search?q=Dissection+of+Kalo%E2%80%99s+trigonum+structures&tbm>

Complications of cholecystectomy:

- bile leak ("biloma");
- bile duct injury (about 5–7 out of 1 000 operations.

Open and laparoscopic surgeries have essentially equal rate of injuries, but the recent trend is towards fewer injuries with laparoscopy. It may be that the open cases often result because the gallbladder is too difficult or risky to remove with laparoscopy);

- abscess;
- wound infection;

bleeding (liver surface and cystic artery are most common sites);

- hernia;

– organ injury (intestine and liver are at highest risk, especially if the gallbladder has become adherent/scarred to other organs due to inflammation (e.g. transverse colon);

– deep vein thrombosis, pulmonary embolism (risk can be decreased through use of sequential compression devices on legs during surgery);

- fatty acid and fat-soluble vitamin malabsorption.

Postoperative treatment

Administer intravenous antibiotics postoperatively. The length of administration is based on the operative findings and the recovery of the patient. Antiemetics and analgesics are administered to patients experiencing nausea and wound pain. A liquid diet may be started when bowel function returns.

Follow-up care

After hospital discharge, patients must have a light diet and limit their physical activity for a period of 4 weeks – 3 months based on the surgical approach (i. e., laparoscopic or open cholecystectomy). The patient should be evaluated by the surgeon in the clinic to detect any possible complications.

Following cholecystectomy, about 5–10 % of patients develop chronic diarrhea. This is usually attributed to bile salts.

The frequency of enterohepatic circulation of bile salts increases after the gallbladder is removed, resulting in more bile salt reaching the colon. In the colon, bile salts stimulate mucosal secretion of salt and water.

Postcholecystectomy diarrhea usually is mild and can be managed with occasional use of over-the-counter antidiarrheal agents. More frequent diarrhea can be treated with daily administration of a bile acid.

Following cholecystectomy, a few individuals experience recurrent pain resembling biliary colic. The term “postcholecystectomy syndrome” is sometimes used for this condition. Many patients with postcholecystectomy syndrome have long-term functional pain that was originally misdiagnosed as being of biliary origin. Diagnostic and therapeutic efforts should be directed at the true cause.

Some individuals with postcholecystectomy syndrome have an underlying motility disorder of the sphincter of Oddi, termed biliary dyskinesia, in which the sphincter fails to relax normally following ingestion of a meal.

Prognosis For uncomplicated cholecystitis, the prognosis is excellent, with a very low mortality rate.

CHAPTER 3. ACUTE PANCREATITIS

The pancreas is a gland located in the upper, posterior abdomen and is responsible for insulin production and the manufacture and secretion of digestive enzymes. In acute pancreatitis there are not only symptoms of acute inflammation in the pancreas are present. There are the signs of hemorrhages and necrotic processes, which are caused by autodigestion of tissues by pancreatic enzymes. Secondary infection and multiorgan system failure may be associated with autodigestion

process. The overall mortality rate of patients with acute pancreatitis is 10–15 %. In patients with severe disease (necrosis and/or organ failure), the mortality rate is approximately 30 %.

Anatomy of hepato-pancreato-duodenal region is demonstrated at figure 14.

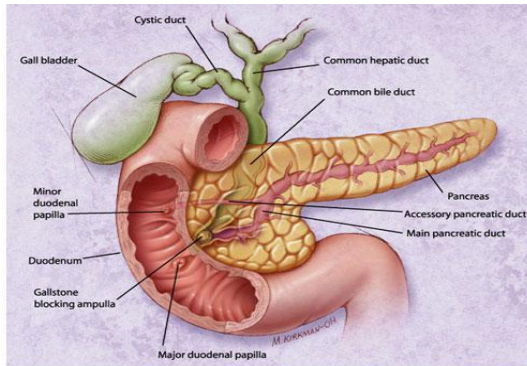


Figure 14 – Anatomy of hepato-pancreato-duodenal region (https://www.google.com/search?rlz=1C1GCEB_enUA1010UA1010&sxsrf)

Etiology:

- Bile and digestive – pancreatic reflux.
- Obstruction and hypertension of biliary and pancreatic ducts.
- Blood supply disturbance of pancreas.
- Allergic and toxic process.
- Peptic ulcer disease.
- Injuries.

A number of factors are capable of initiating acute pancreatitis although their mechanism of action is not known. Alcoholism and biliary tract disease are the processes most commonly associated with pancreatitis: together they account for 80 % of acute cases.

The most common cause of acute pancreatitis is gallstones passing into the bile duct and temporarily lodging at the sphincter of Oddi. The risk of a stone causing pancreatitis is inversely proportional to its size.

Alcohol. Most commonly, the disease develops in patients whose alcohol ingestion is habitual over 5–15 years. Alcoholics are usually admitted with an acute exacerbation of chronic pancreatitis.

Acute pancreatitis may be the first manifestation of a tumor causing pancreatic duct obstruction: pancreatic carcinoma should be considered in non-alcoholic patients in whom no biliary disease is identified.

Infectious agents which have been implicated as a cause of pancreatitis include mumps virus, coxsackie virus, Mycoplasma and parasites.

Many drugs are capable of causing acute pancreatitis: these include thiazide diuretics, 6-mercaptopurine, azathioprine, estrogens, frusemide, methyldopa, sulphonamides, tetracycline, pentamidine, enalapril, and procainamide.

Patients with types I and V hypertriglyceridaemia frequently suffer from pancreatitis, usually associated with extremely high levels of serum triglycerides. However, hypertriglyceridaemia can also be the result rather than the cause of pancreatitis.

Postoperative pancreatitis can be fatal and is often due to iatrogenic pancreatic trauma. Pancreatitis following cardiopulmonary bypass is being recognized with increasing frequency. The majority of cases are mild or even subclinical, but evidence of severe pancreatitis is found in 25 % of patients who die following cardiac surgery.

Abdominal trauma causes an elevation of amylase and lipase levels in 17 % of cases and clinical pancreatitis in 5 % of cases. Pancreatic injury occurs more often in penetrating

injuries (e. g., from knives, bullets) than in blunt abdominal trauma (e. g., from steering wheels, horses, bicycles). Blunt injury may crush the gland across the spine, leading to a ductal injury in that location.

Toxins. Exposure to organophosphate insecticide can cause acute pancreatitis. In Trinidad, the sting of the scorpion *Tityus trinitatis* is the most common cause of acute pancreatitis.

Hypercalcemia cause can lead to acute pancreatitis. Causes include hyperparathyroidism, excessive doses of vitamin D, familial hypocalciuric hypercalcemia and total parenteral nutrition.

Developmental abnormalities of the pancreas. Two developmental abnormalities are associated with pancreatitis: anomaly of pancreatic ducts and annular pancreas.

Sphincter Oddi dysfunction can lead to acute pancreatitis by causing increased pancreatic ductal pressures.

Pathophysiology:

Acute pancreatitis may occur when factors involved in maintaining cellular homeostasis are out of balance. The initiating event may be anything that injures the acinar cell and impairs the secretion of zymogen granules, such as alcohol use, gallstones and certain drugs. In addition, acute pancreatitis can develop when ductal cell injury leads to delayed or absent enzymatic secretion.

Once a cellular injury pattern has been initiated, cellular membrane trafficking becomes chaotic, with the following deleterious effects:

- 1) lysosomal and zymogen granule compartments fuse, enabling activation of trypsinogen to trypsin;
- 2) intracellular trypsin triggers the entire zymogen activation cascade;

3) secretory vesicles are extruded across the basolateral membrane into the interstition, where molecular fragments act as chemoattractants for inflammatory cells.

Activated neutrophils then exacerbate the problem by releasing superoxide or proteolytic enzymes (cathepsins B, D, and G; collagenase; and elastase).

Finally, macrophages release cytokines that further mediate local (and, in severe cases, systemic) inflammatory responses. The early mediators defined to date are tumor necrosis factor- α , interleukin-6, and interleukin-8.

These mediators of inflammation cause an increase in pancreatic vascular permeability, leading to hemorrhage, edema, and eventually pancreatic necrosis. As the mediators are excreted into the circulation, systemic complications can arise, such as bacteremia due to gut flora translocation, acute respiratory distress syndrome, pleural effusions, gastrointestinal hemorrhage and renal failure.

In mild pancreatitis the inflammatory response is well controlled. There may be edema, usually confined to the pancreas, but tissue necrosis is uncommon. In severe pancreatitis the response is uncontrolled, leading to more widespread tissue injury and the many systemic manifestations of the disease. An inflammatory exudate rich in proteolytic enzymes, kinins, and vasoactive substances escapes from the pancreas into the lesser sac, retroperitoneum and peritoneal cavity. It can then be absorbed into the systemic circulation leading to shock, respiratory failure and renal failure.

The etiology of shock in the early phase of acute pancreatitis is multifactorial. Sequestration of fluid in the interstitium, or the third space (intestinal tract), results in intravascular fluid depletion. However, restoration of a euvolemic status, as indicated by central venous pressure and pulmonary capillary wedge pressure measurements may not restore normal blood pressure and hemodynamic. Kinins,

serotonin and vasoactive amines have been implicated as mediators of diminished peripheral vascular resistance and increased vascular permeability. Patients have high concentration of these substances in the peritoneal fluid during acute pancreatitis. Removal of this fluid by peritoneal lavage reverses the hemodynamic alterations.

A failure of system vascular resistance increases appropriately in the face of hypovolemia and inability of the myocardium to compensate appropriately for this loss of peripheral resistance by increasing cardiac output. This is attributed to a myocardial depressant factor.

Circulated lipases and phospholipases destroy surfactant. Development of atelectasis and pneumonia is possible. Plevritis may occur as a result of pleura injuries by enzymes.

The mechanisms underlying the development of renal failure are: hypotension and hypovolemia, reductions in glomerular filtration, toxic injuries of renal cells.

Classifications:

Savelyev V. S. (1983):

Clinical – anatomical forms

- Edematous pancreatitis.
- Fatty pancreatitis.
- Hemorrhagic pancreatitis.
- Mixed pancreatitis.
- Purulent pancreatitis.

Periods:

- Period of hemodynamic disturbances and pancreatogenic shock.
- Period of functional insufficiency of parenchymatous organs.
- Period of degenerative and purulent complications.

Atlanta's classification, Beger H. G., 1991:

- Interstitial pancreatitis.

- Pancreonecrosis (aseptical, inflectional).
- Parapancreatitis (fluid in parapancretical fatty tissue).
- Pseudocyst of pancreas.
- Abscess of pancreas.

Complications:

The first, initial period of the disease takes about first 14 days from the beginning of the disease. Aseptic autolysis, necrobiosis and necrosis of the pancreas and extraperitoneal parapancreatic tissue due to the formation of a systemic response of the body. The second period of the disease starts after 2–3 weeks from the beginning of the disease. Infection and suppuration of pancreatic necrosis and/or necrotized retroperitoneal fatty tissue during this period is possible.

The first period of acute pancreatitis.

Table 1 – Systemic complications. They are treated with conservative therapy

Organ / system	Pathological conditions
Lungs	Pleurisy Atelectasis RDSS Pneumonia
Heart	Pericarditis Arrhythmia
Circulatory system	Hypovolemia Shock
CNS	Encephalopathy Patcher's syndrome (angiopathic retinopathy)
Homeostasis	Hypocalcaemia Hyperglycaemia Hypokalaemia Hypoalbuminemia Hyperlipidemia
Coagulation	DIC syndrome

Continuation of table 1

Organ / system	Pathological conditions
Kidneys	Oliguria Anuria
Intestinal tract	Dynamic bowel obstruction Erosive gastrointestinal bleeding

2. Enzymatic peritonitis. Diagnosis is based on clinical data and ultrasound (free fluid in the abdomen). Minimally invasive drainage of the abdominal cavity is indicated: videolaparoscopic drainage, if it is impossible to perform it – drainage of the abdominal cavity with separate mini-incisions in the iliac and subcostal areas. The fluid obtained during the drainage operation is examined for α -amylase activity and sent for bacteriological examination. If an urgent operation is performed for “peritonitis” and acute pancreatitis is diagnosed intraoperatively, the operation has been completed with drainage of the abdominal cavity in the iliac and hypochondriac areas. The manipulations on the pancreas are not performed to prevent from additional iatrogenic infection.

3. Limited free fluid collections. Ultrasound-controlled percutaneous puncture and drainage is performed.

4. Biliary hypertension (mechanical jaundice with gallstone disease). Surgery is indicated: laparoscopic removal of inflamed gallbladder and removal stones from the extrahepatic ducts (choledocholithotomy and external drainage of the choledochus or endoscopic papillosphincterotomy with choledocholithoextraction). In patients with edematous acute pancreatitis the manipulations on the pancreas are not performed to prevent from additional iatrogenic infection.

5. Parapancreatic mass – mass in the projection of the pancreas.

Clinical signs of parapancreatic infiltrate are: palpated infiltrate in the projection of the pancreas (local component) and resorbive fever (systemic component of inflammation), which are the result of aseptic inflammatory response for necrosis in the pancreas and parapancreatic cells.

Laboratory and instrumental methods of infiltrate diagnosis:

– laboratory parameters of the systemic inflammatory response syndrome (SIRS): leukocytosis with a shift to the left, lymphopenia, increased concentrations of fibrinogen, C-reactive protein;

– us signs of parapancreatic infiltrate (increased size of the pancreas, blurred contours, and the presence of hyperechotic zone without clear contours in the parapancreatic tissue;

– CT of the abdominal cavity to control dynamics of mass.

Outcomes of parapancreatic mass are:

a) resorption of mass, in which there is a reduction of local and general manifestations of acute inflammatory reaction;

b) aseptic sequestration of pancreatic tissue with the development of pancreatic cysts;

c) development of purulent complications.

Treatment of parapancreatic infiltrate:

– continuation of basic infusion-transfusion therapy;
– therapeutic nutrition in the form of enteral or parenteral nutritional support;

– systemic antibiotics therapy, including lymphotropic antibiotics therapy.

The patient is laying down on his abdomen. Choose the paravertebral area of the m. erector spinae, which is located 2 cm outside the processus transversus on the horizontal line connected the lower corners of scapulas. Having painted a

virtual line, a needle is moved to get a depth of 1.5–2.0 cm (almost to the processus transversus) and 32 IU of lidase (lymphoretic) are injected, after which No – Spary (antispasmodic) is injected, followed by 5 000 IU of heparin (antithrombotic drug), ketalgin (diclofenac), as an analgesic and nonspecific anti-inflammatory drug, followed by the antibiotic ceftriaxone (1.0). There is a 5-minute pause between the injections of each subsequent drug.

The second period of acute pancreatitis.

1. Acute post-necrotic pancreatic pseudocysts.

Postnecrotic cavities formation usually follows an attack of necrotic form of acute pancreatitis. They may extend into the retroperitoneal space and bowel mesentery. The local inflammatory response causes the formation of pseudocyst wall composed of well-vascularized granulation tissue. The pseudocyst cavity may contain enzymatic fluid and free necrotic tissues (sequestration). Pseudocyst communication with the pancreatic duct is possible.

Clinical signs of parapancreatic infiltrate are: palpated infiltrate, possible gastrointestinal disorders (anorexia, nausea and vomiting) and resorbitive fever.

Laboratory and instrumental methods of infiltrate diagnosis:

– laboratory parameters of SIRS: leukocytosis with a shift to the left, lymphopenia, increased concentrations of fibrinogen, C-reactive protein;

– ultrasound signs of pseudocyst cavity (size and location of pseudocyst cavity, type of content).

– CT of the abdominal cavity to control location, spreading and dynamics of pseudocyst cavity.

2. Purulent complications

2.1. Pancreatic abscess. This is a result of infection (with microorganism translocation) of necrotized tissues or limited free fluid collections.

2.2. Retroperitoneal space phlegmon. This is a result of infection (with microorganism translocation) of retroperitoneal space exudation.

2.3. Purulent peritonitis. The most common causes are infection of the exudate (in enzymatic peritonitis) or perforation of the pancreas / omental sac abscess into the abdominal cavity.

Clinical manifestations and diagnosis of acute pancreatitis purulent complications.

Clinical manifestations:

a) the presence of signs of abdominal sepsis, which are not eliminated by intensive anti-inflammatory therapy;

b) infiltration of tissues around the pancreas, which can be detected by visual inspection and palpation of the anterior abdominal wall;

c) peritoneum irritation signs.

Instrumental and laboratory manifestations of purulent complications:

a) high levels of acute inflammatory markers (increasing of fibrinogen by 2 times or more, C-reactive protein, procalcitonin, etc.);

b) CT – controlled growing of fluid formations, detection of devitalized tissues and/or the presence of gas balls around the pancreas in the parapancreatic tissue, hyperechoic content in the fluid formations;

c) ultrasound – controlled growing of fluid formations, hyperechoic content in the focus of fluid formations;

d) positive results of bacteriological examinations of the material obtained by needle puncture under the control of ultrasound from liquid collections.

Treatment of the second period of acute pancreatitis complications:

The main goal is to remove infected exudate and necrotic tissues, suppress the inflammatory process and ensure an efficient supply of energy and plastic substances.

Kind of surgical manipulation / operation depends of the location and spreading of process. The most useful manipulations / operations are:

- percutaneous ultrasound – controlled puncture and drainage;

- laparoscopic external drainage;

- open surgery: single- or multi-stage necrosequesrectomy, external drainage, laparostomy.

Antibacterial therapy is a mandatory component of complex therapy of the second period of AP complications and only complements surgical treatment.

A. Systemic antibiotic therapy must be prescribed for the best results, including lymphotropic antibiotics therapy. The patient is laying down on his abdomen. Choose the paravertebral area of the m. erector spinae, which is located 2 cm outside the processus transversus on the horizontal line connected the lower corners of scapulas. Having painted a virtual line, a needle is moved to get a depth of 1.5–2.0 cm (almost to the processus transversus) and 32 IU of lidase (lymphoretic) are injected, after which No – Spany (antispasmodic) is injected, followed by 5 000 IU of heparin (antithrombotic drug), ketalgin (diclofenac), as an analgesic and nonspecific anti-inflammatory drug, followed by the antibiotic ceftriaxone (1,0). There is a 5-minute pause between the injections of each subsequent drug.

B. In the scheme of antibacterial therapy are used drugs with a broad spectrum of action, according the kind, location, spreading and types of pathogens that are most common in necrotic pancreatitis.

C. Correction of the selected scheme is possible in 4–5 days according to the results of bacteriological

examination with antibioticogram of the obtained material (content of pseudocyst, abscess, and intraabdominal exudate).

D. Antimicrobial therapy must be carried out considering the potential toxic reactions of the drug, assessing the severity of the underlying and concomitant pathology of the surgical patient.

Criteria that indicate sufficient antibacterial therapy:

- positive dynamics of the main symptoms of infection;
- regression of other signs of systemic inflammatory reaction (tachycardia, tachypnea), normalization of leukocyte formula;
- positive dynamics of the functional state of the gastrointestinal tract (restoration of motility, the possibility of nutrition);
- negative result of blood bacteriological examination (hemoculture).

Complications of acute necrotic pancreatitis

Arosive bleeding – one of the most dangerous complications of pancreatic necrosis, which occurs due to erosion of both small and large vessels in the focus of pancreatic necrosis and inflammation, regardless of the time of the disease.

Hollow organs perforation

Hollow organs fistula formation

Clinical manifestations and diagnostics of acute pancreatitis:

Most patients with acute pancreatitis can describe a history of biliary pain. The disease, as a rule, begins after violation of diet: intake of much of fatty foods. The following physical examination findings vary with the severity of the disease.

The pain syndrome. The disease begins with a sudden pain in the abdomen. It is localized in the upper abdomen, usually in the epigastric region, but it may be perceived more on the left or right side, depending on which portion of the

pancreas is involved. The pain can get very strong intensity, constant character and usually irradiates to the right or left shoulder, under the right shoulder, and to the patient's back ("belt like pain"). The duration of pain varies but typically lasts more than a day.

The dispepsy syndrome. Characteristic symptoms which disturb a patient are nausea and frequent vomiting not bringing relief. Afterwards feeling of swelling of abdomen often joins to them. Diarrhea can also occur.

The intoxication syndrome. The temperature of body of most patients rises, but it is a low grade fever. The puls rate is increased according the body temperature.

Percussion – tympanitis can be detected.

Auscultation of abdominal cavity – suppression of peristaltic sounds (gastric and transverse colonic ileus).

Some of patients have *jaundice*.

In severe cases, hemodynamic instability is evident and hematemesis or melena sometimes develops (erosive-hemorrhagic gastropathy). In addition, patients with severe acute pancreatitis are often pale, diaphoretic and listless.

The palpation of abdominal wall.

Abdominal tenderness, muscular guarding and distension are observed in most patients.

The Kerte sign. The anterior abdominal wall resistance at pancreatic localization proection.

The Mayo-Robson sign. The moderate pressure in the left costo – vertebral corner appears pain, including the projection of the pancreas.

The Chuhriyenko sign. The moderate shocks of abdominal wall from front to back over the umbilicus increases pain in the area of the pancreas.

The Voskresensky sign. The absence or decreasing of abdominal aorta pulsation over the umbilicus (caused by edema of the pancreas and parapancreatic tissue).

The Ninder sign. The pressure by hand on the area of the left upper quadrant determined aortic pulse caused by edema of the pancreas and parapancreatic tissue.

The Chauffard zone. The triangular area is formed by: median abdominal line, bisectrix of the angle between median line and horizontal umbilical line, and rib arc.

The Osipova sign. The patient lies on his back. Doctor brings both hands in the lumbar area and tries a patient - there is increased pain in the area of the pancreas.

The Gerbrih sign. The percussion of abdomen determines tympanitis on the pancreas projection caused by parietic transverse colon.

The KJuneo sign. The mass is determined by palpation in the projection of the pancreas.

The Cullen sign. The appearance of yellow- violet skin discoloration on the umbilical area.

The Grey-Turner sign. The appearance of yellow-violet skin discoloration on the side walls of the abdomen.

The Mondor sign. The cyanotic spots on the face and trunk.

The Grunwald sign. The peripheral vascular microcirculation disorders due to ecchymosis around the umbilicus and on the buttocks.

Special examinations. The main tasks of special investigations are:

- Differential diagnosis with other abdominal and extraabdominal diseases.
- Detect the form (pancreatitis or pancreonecrosis).
- Detect the previous system disorders for immediate correction.

Lab. Studies.

Amylase and lipase serum level. Serum amylase and lipase levels are typically elevated in persons with acute pancreatitis. Amylase or lipase levels at least 3 times above the

reference range are generally considered diagnostic of acute pancreatitis.

The level of serum amylase or lipase does not indicate whether the disease is mild, moderate, or severe, and monitoring levels serially during the course of hospitalization does not offer insight into prognosis.

Liver-associated enzymes. Determine alkaline phosphatase, total bilirubin, aspartate aminotransferase and alanine aminotransferase levels to search for evidence of gallstone pancreatitis.

Calcium, cholesterol and triglycerides. Determine these levels to search for an etiology of pancreatitis (hypercalcemia or hyperlipidemia) or complications of pancreatitis (hypocalcemia resulting from saponification of fats in the retroperitoneum). However, be wary of the fact that baseline serum triglyceride levels can be falsely lowered during an episode of acute pancreatitis.

Serum electrolytes, creatinine, and glucose. Measure these to look for electrolyte imbalances, renal insufficiency, and pancreatic endocrine dysfunction.

CBC. Hemoconcentration at admission (an admission hematocrit value greater than 47 %) has been proposed as a sensitive measure of more severe disease. Leukocytosis may represent inflammation or infection.

C-reactive protein. A C-reactive protein (CRP) value can be obtained 24–48 hours after presentation to provide some indication of prognosis. Higher levels have been shown to correlate with a propensity toward organ failure. A CRP value in double figures (i. e., > 10 mg/dl) strongly indicates severe pancreatitis. CRP is an acute-phase reactant that is not specific for pancreatitis.

Arterial blood gases. Measure ABGs if a patient is dyspneic. Whether tachypnea is due to acute respiratory

distress syndrome or diaphragmatic irritation must be determined.

Trypsin and its precursor trypsinogen-2 in both the urine and the peritoneal fluid have been evaluated as possible markers for acute pancreatitis but are not widely used.

Imaging studies.

AXR. This modality has a limited role in acute pancreatitis. These radiographs are primarily used to detect free air in the abdomen, indicating a perforated viscus, as would be the case in a perforated ulcer. In some cases, the inflammatory process may damage peripancreatic structures, resulting in a colon cut-off sign, a sentinel loop, or an ileus. The presence of calcifications within the pancreas may indicate chronic pancreatitis.

Chest radiography can help to detect lung and pleural complications.

US. This is the most useful initial test in determining the etiology of pancreatitis and is the technique of choice for detecting gallstones. In the setting of acute pancreatitis, sensitivity is reduced to 70–80 %. In addition, the ability to identify choledocholithiasis is limited. Ultrasonography cannot measure the severity of disease. Some complications (pancreatic mass, pancreatic abscess, abdominal or retroperitoneal abscess, pseudocyst of pancreas, peritonitis) can be diagnosed.

CT. This is generally not indicated for patients with mild pancreatitis unless a pancreatic tumor is suspected (usually in elderly patients). CT scanning is always indicated in patients with severe acute pancreatitis and is the imaging study of choice for assessing complications. Scans are seldom needed within the first 72 hours, because inflammatory changes are often not radiographically present until this time.

Contrast-enhanced CT of the pancreas is diagnostic and can show (fig. 15, 16):

- enlargement of pancreas due to edema;
- peripancreatic inflammation: linear strands in the peripancreatic fat;
- phlegmon;
- hemorrhagic: enlarged pancreas with increased density due to hemorrhage;
- necrosis;
- fluid in the paracolic gutter;
- fluid collections: a simple peripancreatic fluid collection will not have a well-defined capsule;
- pseudocysts: as liquefaction of necrotic pancreatic tissue progresses it will gradually take on the appearance of localized fluid collection pseudocyst;
- abscesses: diffusely enlarged pancreas with air pockets.

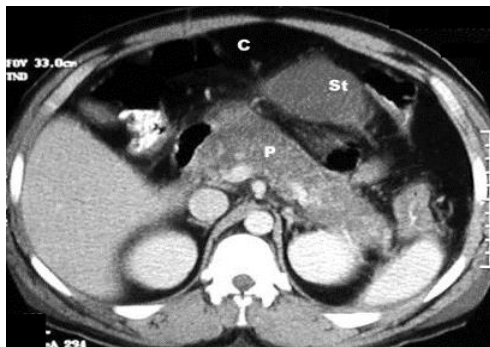


Figure 15 – CT scan in acute pancreatitis

(<https://radiopaedia.org/articles/acute-pancreatitis>)

Post Contrast CT findings reveal diffusely enlarged pancreas with low density from edema. C: colon, St: stomach, P: pancreas

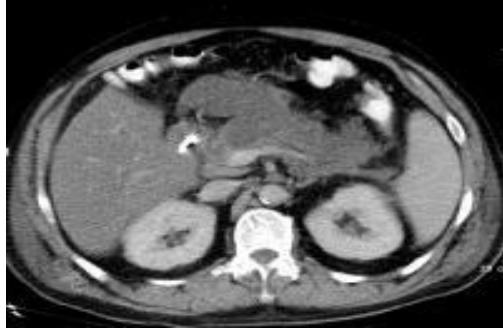


Figure 16 – Acute pancreatitis, pancreatic necrosis
(<https://radiopaedia.org/articles/acute-pancreatitis>)

Note the no enhancing pancreatic body anterior to the splenic vein. Also present is peripancreatic fluid extending anteriorly from the pancreatic head.

Abdominal CT scans also provide prognostic information based on the following grading scale developed by Balthazar:

A – normal.

B – enlargement.

C – peripancreatic inflammation.

D – single fluid collection.

E – multiple fluid collections.

The chances of infection and death are virtually nil in grades A and B but steadily increase in grades C through E. Patients with grade E pancreatitis have a 50 % chance of developing an infection and a 15 % chance of dying.

Dynamic spiral CT scanning is used to determine the presence and extent of pancreatic necrosis. Focal or diffuse areas of unenhanced parenchyma on the second study suggest pancreatic necrosis.

Magnetic resonance cholangiopancreatography has an emerging role in the diagnosis of suspected biliary and pancreatic duct obstruction.

Endoscopic ultrasonography is an endoscopic procedure that allows a high-frequency ultrasound transducer to be inserted into the gastrointestinal tract. EUS is often helpful in evaluating the cause of severe pancreatitis, particularly microlithiasis and biliary sludge, and can help identify periampullary lesions better than other imaging modalities.

Procedures:

ERCHPG is dangerous in patients with acute pancreatitis and should never be used as a first-line diagnostic tool in this disease.

The indications for this procedure are:

1) if a patient has severe acute pancreatitis that is believed, and is seen on other radiographic studies, to be secondary to choledocholithiasis;

2) if a patient has biliary pancreatitis and is experiencing worsening jaundice and clinical deterioration despite maximal supportive therapy.

CT-guided needle aspiration. This procedure is used to differentiate infected necrosis and sterile necrosis in patients with severe necrotizing pancreatitis.

Treatment of acute pancreatitis.

For acute pancreatitis, initial treatment is conservative.

Minimizing of pancreatic secretion is the main task.

Bowel rest, nasogastric tube.

Local hypothermia.

Inhibitors of pancreatic secretion: octreotide 0.1 mg subcutaneously.

H₂-receptor or H-pump blockers. They are useful for inhibition of pancreatic enzymes secretion and prevention of acute upper digestive tract ulcerations.

Atropine.

5-floruracil (500 mg/d).

Analgesics, spasmolytics. Narcotic analgetics are dangerous in patients with acute pancreatitis because of sphincter Oddi constriction.

Relief of vomiting. Drainage of the stomach with a probe, metaclopramid (cerucal, reglan) 10–20 mg; osetron (ondansetron, navoban, tropisetron).

Fluid replacement. The most important requirement in the early treatment of pancreatitis is maintenance of adequate hydration. If the patient becomes hypovolemic, and the splanchnic circulation is compromised, the pancreas may become ischemic, with the potential for the development of complicated pancreatitis.

With considerable reduction of arterial blood pressure - dopamine 5–7 mkg/kg of the body weight; for relief of angiospasm – benzoexamethonium 0.15 mg/kg of the body weight or pentamine 0.25–0.3 mg/kg of the body weight every 6 hours; for reduction of vascular permeability – hydrocortisone 1,000–1,500 mg/day.

Detoxication:

1) administration of diuretics: torasemide 5 % from 4 ml or furosemide 1 % from 2 ml per day under the control of diuresis;

2) metabolic solutions on a polyionic basis: 400 ml i/v 1 time a day;

3) multicomponent combined infusion solutions;

4) extracorporeal detoxication (plasmapheresis, hemosorbition).

Treatment of hypoxaemia. Fluid replacement, normalization of peripheral microcirculation is adequate in majority of patients. In the most severely ill patients intubation and ventilatory support is indicated.

Nutritional support. Patients with severe acute pancreatitis often cannot be fed for several days. Once severe pancreatitis has developed total parenteral nutrition should be

instituted. Intravenous fat emulsions do not exacerbate pancreatitis in patients with normal triglyceride levels. If triglyceride levels are raised, however, fat emulsion should not be used. Total parenteral nutrition should be continued until the patient appears clinically well.

Antibiotic therapy. Antibiotics are generally ineffective in preventing the late septic complications of acute pancreatitis, and their use may even promote selection of organisms that are more difficult to treat later on. The best variants are:

- third and fourth – generations of cephalosporin's;
- quinolones and metronidazole;
- carbapenems.

Antiprotease therapy. Gordox, Contrical. They are useful and effective only as inhibitors of kinins.

Treatment of metabolic disturbances includes correction of hypocalcaemia, hypoalbuminaemia, hypomagnesaemia, hypokalaemia, control of blood glucose.

Surgical care. In patients with acute pancreatitis common indications for surgery are:

- Progressive peritonitis.
- Progressive multiorgan system failure.
- Gallstones pancreatitis without effect of conservative treatment during 24–48 h.
 - Erosive hemorrhage.
 - Bowel perforations.
 - Septic complications.

Operations:

- Peritoneal lavage and drainage.
- Gallstones pancreatitis: cholecystectomy and, if still necessary, common bile duct tube.
 - Abdominization.
 - Necrectomy.
 - Surgical management in necrotizing process into the retroperitoneum, perirenal spaces, and mesentery.

- Drainage by percutaneous aspiration of peripancreatic fluid collections.
- Percutaneous drainage of pancreatic abscesses (under CT or US control).

CHAPTER 5. PERFORATED PEPTIC ULCER

The frequency of peptic ulcer disease is estimated 6 % to 15 % in the general population. Most individuals do not have clinically significant ulcer.

Approximately 10 % of young men have *H. pylori*, and the proportion of people with the infection increases steadily with age. The incidence of duodenal ulcer has been decreasing over the past 3–4 decades.

Lifetime prevalence of perforation in patients with peptic ulcer disease is approximately 5 %. With NSAID – related ulcers, the incidence of perforation is 0.3 %. Emergency operations for peptic ulcer perforation carry a mortality risk of 6–30 %. Most deaths result from postoperative complications.

Etiology.

1. Contributes factor: progressive destruction of stomach or duodenal wall.

Ulcers of the stomach and duodenum are caused chiefly by the effects of hydrochloric acid, produced by the parietal cells of the stomach, and by lack of protection of the mucosa against this acid. Acid production is the most important factor as far as duodenal ulcer is concerned, but cannot be the only factor, since the severity of duodenal ulcers and their responses to therapy do not vary directly with the amount of gastric acid secreted.

Additional factors are important in the production of gastric ulcers. Gastric contents can be retained for a much longer period than duodenal contents and are not neutralized rapidly. The mucous membrane of the stomach must normally be protected continuously from the damaging effect of the hydrochloric acid which it secretes. This protective layer consists of an adherent layer of mucus which is separated from the gastric mucosa by a bicarbonate layer that normally neutralizes any hydrochloric acid diffusing back through the mucous layer. The mucous layer also protects the gastric mucosa against other noxious agents such as bile, alcohol, and aspirin and other irritating drugs.

Secretion of acid by the parietal cells of the stomach is influenced by several mechanisms. Stimulation by the vagus nerves accounts for about 50 % of acid secretion. Gastrin that is secreted by the antral mucosa causes 40 to 45 % of the secretion and the remaining 5 to 10 % is due to gastrin that comes from the intestinal tract from various APUD cells.

Production of gastric mucus should presumably vary according to the blood supply of the organ. Experimental studies have shown that prostaglandins increase the mucosal blood supply and increase the bicarbonate level and thickness of the mucous layer; they may, therefore, play an important role in the protection of the gastric mucosa.

Causes of gastric ulcers vary in importance depending upon the location of these ulcers. In 1965 Johnson observed the different behavior of ulcers in different portions of the stomach and classified them into three groups. In patients with type 1 ulcers, located in the body of the stomach, gastric acid secretion as determined by secretory tests is low. Here inadequate mucosal protection and back-diffusion of hydrochloric acid through the protective bicarbonate and mucous layer into the gastric mucosa seem to be involved in the disease. Silent suggested it is in this location that

alternating waves of gastric acid from above and refluxing alkaline secretions from the duodenum destroy the protective layer of mucus.

Johnson's type 2 gastric ulcers (those combined with a present or past duodenal ulcer) and type 3 (those in the prepyloric area) behave more like duodenal ulcers; here the effects of acid production appear to be more important.

A peptic ulcer occurs when an alteration occurs in the aggressive and/or protective factors such that the balance is in favor of gastric acid and pepsin. Any process that increases gastric acidity (e.g., individuals with increased maximal and basal acid output), decreases prostaglandin production (e. g., NSAIDs), or interferes with the mucous layer (e. g., *Helicobacter pylori* infection) can cause such an imbalance and lead to peptic ulcer disease.

H. pylori infection that follows an antral predominant pattern leads to an inflammatory state in which high levels of tumor necrosis factor-alpha (TNF-alpha) and other cytokines are produced. These stimulate gastric acid production directly by increasing gastrin release from G cells and inhibit somatostatin production by antral D cells. This leads to a net increase in gastric acid secretion, which leads to an increased acid load in the duodenum, overwhelming the mucosal defense.

H. pylori can then colonize the duodenal mucosa and adhere to cells. This adherence leads to a variety of second-messenger signals, which invoke an immunologic response against those cells causing mucosal damage by host neutrophils and other inflammatory cells. *H. pylori* also affect the gastric and duodenal mucous layer because this organism produces proteases that degrade the protective mucous layer. Moreover, *H. pylori* infection decreases the production of epidermal growth factor, which normally promotes healing of gastric and duodenal mucosa. *H. pylori* organisms produce urease. Urease

hydrolyzes urea to ammonia and carbon dioxide. Hydroxide ions produced by equilibration of ammonia with water may damage the gastric and duodenal mucosa.

2. Causes factor: increasing of pressure in stomach lumen (more food intake, physical training etc.).

Classifications:

Forms of perforation (Savelyev V. S., 1976):

- perforation of duodenal or gastric ulcer into the free abdominal cavity;
- covered perforation;
- atypical perforation (into the lesser sac, retroperitoneal fatty tissue).

Clinical stages (Neimark N. I., 1972):

- stage of shock;
- stage of illusion;
- stage of peritonitis.

Clinical manifestations. The main signs are:

Triad of Mondor. The "knife like pain", "total abdominal muscles resistens" and "ulcer history."

The Dyelafua sign. The "knife like pain" abdominal pain.

The Krasnobayev-Kryuvelye *sign*. The expressed muscle tension of the anterior abdominal wall, especially over the source of inflammation.

The Winter sign. The abdominal wall does not participate in breathing because of the tension.

The Chuhayev sign. The direct abdominal muscles are tensed and expressed their relief.

The Dzbanovsky-Chuhayev sign. The sudden tension of anterior abdominal wall muscles due to transverse skin crinkles above the umbilicus.

The Ratner – Vicker sign. The severe muscle tension of the abdominal wall in the epigastric or (and) right hypochondriac area.

The Bernstein sign. The scrotum skin is corrugated, the testicles are pulled to the external inguinal canal rings, the penis is turned up and right.

The Hjusten sign. It is defined by auscultation: the heart tones spread up to the umbilicus and "silver bell" tone (the released gas pass from the perforated organ through the liquid).

The Brunner sign. The accumulation of content between the stomach and the diaphragm causing the friction noise.

The Grekov sign. The "vagal pulse" (bradycardia) + + sharp abdominal pain.

The Clarke sign. Free gas between the liver and abdominal wall due to lack of hepatic blunting.

The Spizharny sign. Percussion detects a tympanitis on the liver projection because of free gas upper abdominal collection.

De Quervain sign. In the slanting areas of the abdomen, mostly over the right iliac fossa or on the side channels where the blood or abnormal fluid are accumulated, the blunting percussion sound is determined.

The Podlah sign. This is a sign of atypical peptic ulcer perforation. Free gas passes through the esophageal gate of the diaphragm to the mediastinum and the left supraclavicular area, where subcutaneous emphysema can be detected.

The Vihiatso sign. This is a sign of atypical peptic ulcer perforation. Free gas passes through the lig. hepatoduodenale or (and) lig. teres hepatis, and in the umbilical area subcutaneous emphysema can be detected.

The Neimark test. To 2–3 ml of fluid getting from the abdominal cavity, add 4–5 drops of 10 % iodine solution. In the presence of duodenal fluid, content becomes dark blue.

Clinically the perforations are distinguished into three phases: shock, stage of illusion and peritonitis (Mondor, 1939).

Stage of shock (lasts for about 6 hours). The aggressive content of the stomach causes a chemical burn of the peritoneum.

- Intensive upper abdominal pain at the time of perforation (Delafua compares it with “pain due to stabbing with a dagger”).

- Elicer’s sign – irradiation of pain to the right shoulder or scapula.

- Tenderness of abdominal wall. In elderly patients tension of muscles of abdominal wall is absent or insignificant through old-age relaxation of muscles.

- Percussion of liver will reveal attenuated dullness due to free gas.

- Auscultation will reveal a silent abdomen.

- Blumberg's sign.

- Temperature is normal.

- Blood pressure may be lost.

- Pulse is rapid.

Stage of illusion (lasts for about 6–12 hours). The intensity of pain decreases due to reducing of the gastric content concentration as a result of peritoneum exudate secretion. At the same time, the translocation of microorganisms starts (migration from the intestinal lumen into the free abdominal cavity).

- As more exudate accumulates, chemical irritation of peritoneum becomes less, the patient feels better with less pain.

- The same signs may be found by rigidity becomes less.

- Abdominal distention will start.

- Temperature becomes elevated.

Stage of purulent peritonitis.

- The patient is extremely toxic with tachycardia and high temperature.

- The abdomen is rigid, distended and silent.

- Blumberg's sign.
- Blood pressure is often lost.

Covered perforation (A. M. Shnicler, 1912). In this pathology the perforation is closed by fibrin, by omentum or sometimes – by food particle. After that some amount of stomach content and air left into the abdominal cavity. This protective mechanism leads to relief from stomachache, but moderate tenderness of abdominal wall in right epigastria region persists (Ratner's sign). On percussion hepatic dullness is doubtful. During AXR examination it is not always possible to mark gas in right hypochondrium.

Consequences of the covered perforation are: repeated perforation with development of classic clinical signs; subdiaphragmatic or subhepatic abscess; complete closing of defect by surrounding tissue with gradual convalescence of patient.

The atypical perforation is the perforation, at which gastric or intestinal content does not enter the abdominal cavity, but enters the retroperitoneal space (ulcers of posterior wall of duodenum), large or small omentum (ulcers of lesser curvature of stomach), hepatoduodenal ligament.

In such patients during the perforation pain is not acutely expressed. During palpation insignificant rigidity of muscles of anterior abdominal wall is observed. On occasion, especially in the late stages of disease, can be hypodermic emphysema and crepitation.

The differential diagnosis includes acute cholecystitis, acute pancreatitis, strangulating intestinal obstruction, acute appendicitis, perforation of some other portion of the intestinal tract, and mesenteric thrombosis. A past history of ulcer disease is predictive, but in 30 % of patients perforation is the first manifestation of peptic ulcer disease.

Diagnosis program:

AXR examination of abdominal cavity organs for presence of free gas (pneumoperitoneum, fig. 17).

Fibergastroduodenoscopy (FGDS) + AXR examination.

US of abdominal cavity (pneumoperitoneum, fig. 18) + laparocentesis (peritoneal exudate).

Laparoscopy.

Laparotomy.

CT with oral contrast may also demonstrate extravasation of contrast in the presence of a perforated ulcer. If history and physical examination raise the possibility of perforation, upright chest radiography and an upper digestive tract contrast study with water-soluble contrast are the diagnostic studies of choice. Do not perform CT scanning in this situation.



Figure 17 – X-Ray examination of abdominal cavity, pneumoperitoneum
(https://www.google.com/search?rlz=1C1GCEB_enUA1010UA1010&sxsrf)

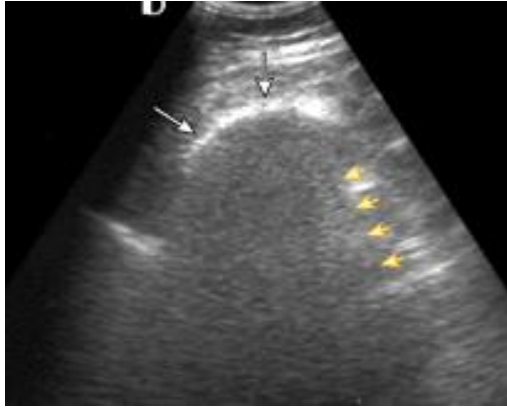


Figure 18 – US of abdominal cavity, pneumoperitoneum
(https://www.google.com/search?rlz=1C1GCEB_enUA1010UA1010&sxsrf=ALiCzsZbcimunMIMKENVX)

Lab. Studies.

CBC count. (Leukocytosis with left shift is found in most cases.)

Serum gastrin level. Gastrin levels greater than 1 000 pg/ml are suggestive of gastrinoma.

Serum *H. pylori* antibody detection. Antibodies (immunoglobulin G [IgG]) to *H. pylori* can be measured in serum, plasma, or whole blood. However, results from whole blood tests using finger sticks are less reliable.

Urea breath tests. Urea breath tests are used to detect active *H. pylori* infection by testing for the enzymatic activity of bacterial urease. In the presence of urease produced by *H. pylori*, labeled carbon dioxide (heavy isotope, carbon-13, or radioactive isotope carbon-14) is produced in the stomach, absorbed into the bloodstream, diffused into the lungs, and exhaled.

Fecal antigen tests. Fecal antigen testing is used to identify active *H. pylori* infection by revealing the presence of

H. pylori antigens in stools. This test is more accurate than antibody testing and less expensive than urea breath tests.

Treatment of perforated peptic ulcer.

Tactic of treatment in patients with perforated peptic ulcer is surgical.

Conservative treatment is temporary measure when surgery is impossible. Conservative treatment includes the following (Taylor's method):

- nasogastric decompression;- replacement of fluid and electrolytes;

- proton pump inhibitor;

- broad-spectrum antibiotics;

- hypothermia of abdominal wall.

Conservative treatment is associated with high risk of septic complications (formation of abscess, peritonitis).

Surgical treatment.

Preoperative details. Fluid resuscitation should be initiated as soon as the diagnosis is made. Essential steps include insertion of a nasogastric tube to decompress the stomach and a Foley catheter to monitor urine output. Intravenous infusion of fluids is begun, and broad-spectrum antibiotics are administered. In select cases, insertion of a central venous line may be necessary for accurate fluid resuscitation and monitoring. As soon as the patient has been adequately resuscitated, emergent surgery should be performed.

Simple closure of perforation. Simple closure of perforation may be performed by laparotomy or laparoscopy.

The 1st step of the operation is the exploration of the abdominal cavity, which allows confirming the diagnosis of generalized peritonitis. In addition, it allows differentiating between the septic and the clinical peritonitis, and especially it permits to determine the possibility of the laparoscopic repair.

A very important step of the operation is the aspiration of peritoneal fluid, which should be as complete as possible. This is followed by extensive irrigation of the abdominal cavity.

The next step is the exact localization of the perforation, which sometimes may be covered by the liver, gallbladder or omentum. In this case, the perforation is identified on the anterior aspect of the duodenum.

Ideal repair of the perforation is direct closure by absorbable or non-absorbable sutures. Treatment of perforation may include also an epiploplasty in addition to the closure. An omental flap is chosen and is placed over the suture and fixed with 1 or 2 absorbable stitches. When a reliable direct suture of the opening cannot be achieved, closure can be completed by application of biological glue. The operation is completed by final extensive abdominal irrigation.

Ulcer excision, pyloroduodenoplasty.

Ulcer excision and pyloroduodenoplasty is the simplest open surgical treatment for duodenal ulcer and in selected instances it can be used in the treatment of bleeding, obstruction, perforation. The operation carries low morbidity and mortality rates.

The operation usually consists of a Judd procedure. A longitudinal excision is made around the ulcer defect beginning from distal stomach to the proximal duodenum and closed transversely, so that the action of the pyloric valve is obliterated. In other cases, when the proximal duodenum is badly deformed by scar, a Finney procedure (essentially a side-to-side gastroduodenostomy) or a Jaboulay procedure (gastric resection plus side-to-side gastroduodenostomy) is used after ulcer excision.

Various types of vagotomy.

Three types of vagotomy require consideration. Bilateral truncal vagotomy denervates the entire stomach and

the gastrointestinal tract to the left colon. When combined with other operations it is extremely effective in reducing the number of recurrent ulcers. However it does carry some deleterious side-effects, reducing the ability of the stomach to empty and being followed by other late motility disturbances, occasional vagus diarrhea, and reflux alkaline gastritis. Selective vagotomy denervates the entire stomach but leaves nerves to the gallbladder, pylorus, and bowel intact. From a practical point of view this operation is more difficult and time-consuming than truncal vagotomy, appears to have nearly equivalent results, and has attracted few supporters. Proximal gastric vagotomy can be used without pyloroplasty.

Partial gastrectomy.

The usual procedure involves resection of the distal two-thirds of the stomach followed by either a Billroth I or a Billroth II anastomosis. In our opinion the operation is neater and more accurate when sutures are inserted by hand, although stapling instruments can be used. Partial gastrectomies are usually performed for type 1 gastric ulcer and for combination of duodenal ulcer perforation with decompensate stenosis.

Postoperative treatment.

Administer intravenous antibiotics postoperatively. The length of administration is based on the operative findings and the recovery of the patient. Antiemetics and analgesics are administered to patients experiencing nausea and wound pain. A liquid diet may be started when bowel function returns. To prevent from ulcer recurrence and their complications proton pump inhibitor or H₂ – receptors antagonist is administered.

Follow-up care.

After hospital discharge, patients must have a light diet and limit their physical activity for a period of 4 weeks – 3 months based on the surgical approach (i.e., laparoscopic or open procedure). The patient should be evaluated by the surgeon in the clinic to determine improvement and to detect

any possible complications. Continuous supportive therapy (for a month or even years) with half the dose of proton pump inhibitors or H₂ – receptors antagonists is administered.

Prognosis.

For uncomplicated surgery, the prognosis is excellent, with a very low mortality rate.

CHAPTER 6. PEPTIC ULCER ACUTE HEMORRAFE

Peptic ulcer acute hemorrhage is a common occurrence throughout the world. In France, a recent report concludes that the mortality from peptic ulcer acute hemorrhage has decreased from about 11 % to 7 %; however, a similar report from Greece finds no decrease in mortality.

Patients typically present with an ulcer that has bled or is actively bleeding. Approximately 80 % of ulcers stop bleeding. The overall mortality rate is approximately 10 %. Rebleeding or continued bleeding is associated with increased mortality. Comorbidities increase the probability of rebleeding in patients after endoscopic therapy. An increasing amount of evidence in the literature states that therapy with high-dose proton pump inhibitors may decrease the rate of rebleeding after endoscopic therapy. By increasing the gastric pH above 6, the clot is stabilized.

This patient population has become progressively older, with significant comorbidities that increase mortality.

Pathophysiology.

Duodenal ulcer disease is strongly associated with *Helicobacter pylori* infection. The organism causes disruption of the mucous barrier and has a direct inflammatory effect on gastric and duodenal mucosa. Eradication of *H. pylori* has been

demonstrated to reduce the risk of recurrent ulcers and, thus, recurrent ulcer hemorrhage.

NSAIDs are the second major etiology of ulcer hemorrhage because of their effect on cyclooxygenase-1, which leads to impaired mucosal defense to acid. The use of cyclooxygenase-2 inhibitors has been shown to reduce the risk of ulcer hemorrhage, although only when not combined with aspirin therapy. Recent concerns have been raised about an increase in myocardial infarction and stroke in patients taking selective cyclooxygenase-2 inhibitors.

The combination of *H. pylori* infection and NSAID use may increase the risk of ulcer hemorrhage.

As the ulcer burrows deeper into the gastroduodenal mucosa, the process causes weakening and necrosis of the arterial wall, leading to the development of a pseudoaneurysm. The weakened wall ruptures, producing hemorrhage. The flow through the vessel varies with the radius: small increases in vessel size can mean much larger amounts of blood flow and bleeding. Visible vessels usually range from 0.3-1.8 mm.

Massive hemorrhage has been reported from larger vessels. The larger vessels are located deeper in the gastric and duodenal submucosa and serosa. Larger branches of the left gastric artery are found high on the lesser curvature, while the pancreaticoduodenal artery and its major branches are located posteroinferiorly in the duodenal bulb.

The size of the vessel is important in the prognosis in that larger vessels cause faster blood loss, with more severe hypotension and more complications, especially in older patients.

The main disturbances in patients with acute peptic ulcer bleeding are:

1. Acute loss of blood leads to rapid reduction of cardiac output.

2. Activation of simpatico-adrenal system results in peripheric vasoconstriction and centralization of blood supply.

3. Mechanisms of compensation are supplemented with hemodilution (displacement of interstitial liquid into vessels) and increasing of aldosterone production).

4. After loss before 20 % of blood volume circulation the bleeding stops and in absence of hemorrhagic compensation the disturbances of circulation of blood are not observed.

5. After loss more 20 % of blood volume circulation without the proper compensation of hemorrhage such patients can survive, however always there are considerable disturbances of blood circulation with disturbance of functions of liver and kidneys.

6. Inadequate peripheric blood supply leads to hypoxia, stimulates anaerobic glycolysis and results in free radical metabolites (metabolic acidosis).

7. Rheological disturbances result in embolism and thrombosis in microcirculation bloodstream.

8. Arteriovenous shunts open, multiple organ failure develops (“sock” lungs, kidneys, liver).

9. Loss of blood cells results in immunodeficiency.

Classifications.

Stages of loss of blood (Shalimov A. A.,1987):

- 1) 20 % of volume of blood circulation.
- 2) 20–30 % of blood circulation.
- 3) More then 30 % of blood circulation.

Forrest endoscope classification (Forrest J. A. H., 1974):

- Forrest I. Active bleeding ulcer.
- Forrest II. Temporary hemostasis. (risk of bleeding recurrence 40 %). Forret IIA, nonbleeding visible vessel, Forrest IIB, bleeding ulcer with adherent blood clots; Forrest IIC, ulcer with pigmentation.

- Forrest III. There aren't signs of bleeding (risk of bleeding recurrence 4 %).

History.

It is needed to remember, that for diagnosis anamnesis is very important. The patient history findings include weakness, dizziness, syncope associated with hematemesis (coffee ground vomitus), melena (black stools with a rotten odor) and hematochezia (red or maroon stool).

More patients tell about their peptic ulcer disease. Sometimes bleeding has occurred repeatedly or patients has undergone surgery for perforated ulcer in the past. In some patients a gastric or duodenum ulcer was not diagnosed before and correctly collected anamnesis revealed that the patient had stomach-ache. Patients often tell that pain in upper part of abdomen which occurred a few days prior to bleeding, suddenly disappears the onset of bleeding (the Bergmann's sign).

Patients may have a history of previous dyspepsia (especially nocturnal symptoms), ulcer disease, early satiety, and NSAID or aspirin use. Many patients with peptic ulcer acute hemorrhage who are taking nonsteroidal anti-inflammatory drugs present without dyspepsia but with hematemesis or melena as their first symptom. Low-dose aspirin (81 mg) has been associated with peptic ulcer acute hemorrhage with or without the addition of NSAID therapy. Patients with a prior history of ulcers are at an especially increased risk for peptic ulcer acute hemorrhage when placed on aspirin or NSAID therapy and should receive continuous acid suppression with a proton pump inhibitor. Because recurrence of ulcer disease is common, history findings are relevant.

Patients may present in a more subacute phase with a history of dyspepsia and occult intestinal bleeding manifesting

as a positive fecal occult blood test result or as iron deficiency anemia.

A history of recent aspirin ingestion suggests that the patient may have nonsteroidal anti-inflammatory drug gastropathy with an enhanced bleeding diathesis from poor platelet adhesiveness.

A history of chronic alcohol use of more than 50 g/d or chronic hepatitis (B or C) increases the risk of hemorrhage, gastric antral vascular ectasia, or portal gastropathy.

The presence of postural hypotension indicates more rapid and severe blood loss.

Clinical manifestation.

1. The Mendel sign. The finger – tips percussion demonstrates the ulcer presence location.

2. The Bergman sign. The reduction of epigastric area pain intensity after bleeding starts (stomach acid neutralization).

3. The Taylor sign. The linking blood results in the intensive peristalsis (irritation of bowels by blood).

In patients with peptic ulcer disease bleeding starts mainly at night. Vomiting can be the first sign, mostly, with gastric localization of ulcers. Vomiting, as a rule, is "coffee-ground" in appearance. Sometimes it is with fresh blood or blood clots.

The black stool is the permanent symptom of ulcer bleeding, with an unpleasant smell ("melena") that can take place few times in day.

Bloody vomiting and "melena" is accompanied by worsening of the general condition of patient. An acute weakness, dizziness, noise in a head and darkening in eyes, sometimes – loss of consciousness. A collapse with the signs of hemorrhagic shock can also develop.

In patients with ulcer bleeding there are typical changes of hemodynamic indexes: the pulse is rapid, weak filling and tension, arterial pressure is mostly reduced. These indexes need

to be observed in the dynamics, as they can change during the short interval of time.

There is pallor of skin and visible mucosa at an examination. The stomach sometimes is moderately enlarged, but more frequently it is pulled in, soft on palpation. In upper part it is possible to notice hyperpigmented spots – as a result of prolonged application of hot-water bottle. Pain on deep palpation in the area of right hypochondrium (duodenal ulcer) or in a epigastric area (gastric ulcer) is often observed in penetrated ulcers.

In the examination of patients with the gastrointestinal bleeding digital examination of rectum is obligatory. It needs to be performed at the first examination, because information about the presence of black excreta ("melena") is important. In addition, it is sometimes possible to expose the tumor of rectum or haemorrhoidal nodes which are also the source of bleeding.

Clinical variants.

It is necessary always to remember that complication of peptic ulcer by bleeding happens considerably more frequently, than it is diagnosed. The clinical signs and staging of disease depends on the degree of loss of blood. Usually, 50–55 % of moderate bleeding (microbleeding) are unseen. In fact profuse bleeding with the loss 50–60 % of the volume of circulatory blood could stop the heart and cause the death of patient.

For loss of 20 % of volume of blood circulation (I stage) typically: rapid pulse rate to 90–100 per min., decreasing of arterial blood pressure to 90/60 mm Hg. The excitability of patient changes by lethargy, however consciousness is clear, breathing is frequent. After the bleeding stops and in absence of hemorrhagic compensation the disturbances of circulation of blood are not observed.

In patients with the II stage of hemorrhage (loss of 20–30 % of blood circulation) the general condition needs to be estimated as average. Expressed pallor of skin, sticky sweat,

lethargy. Pulse rate – 120–130 per min., weak filling and tension, arterial blood pressure – 90–80/50 mm Hg. In the first few hours the spasm of vessels (centralization of blood circulation) arises after bleeding. Without the proper compensation of hemorrhage such patients can survive, however always there are considerable disturbances of blood circulation with disturbance of functions of liver and kidneys.

The III stage of hemorrhage (more than 30 % of blood circulation). The pulse rate in such patients is 130–140 per min., and arterial blood pressure – from 60 to 0 mm Hg. Consciousness is almost always darkened, acutely expressed adynamic. Central vein pressure is low. Oliguria is observed, that can change to anuria. Without active and directed correction of hemorrhage the patient can die.

Amount of bleeding doesn't always correspond to the general condition of patient. It can depend on compensatory mechanism of the organism, rate of loss of blood and the presence of accompanying pathology.

It is needed to remember, that the ulcer bleeding can accompany the perforation of ulcer. During perforation, ulcers are often accompanied by bleeding. Correct diagnosis of these two complications has important value in tactical approach and in the choice of method of surgical treatment. In fact simple suturing of perforated and bleeding ulcer can lead to complications in the postoperative period, as profuse bleeding and cause the necessity of repeat operation.

Differential diagnosis/

With wide use of gastroduodenoscopy the question of differential diagnosis of bleeding is not significant. However such a problem arises due to impossibility to use this method of examination when the general condition of patient is bad or taking into account other reasons. Differential diagnosis is conducted in bleeding of no ulcer origin, which can arise in different parts of digestive tract.

For bleeding from the *varicose veins of esophagus* in patients with portal hypertension with liver cirrhosis. This type of bleeding is massive and leading to considerable hemorrhage. Vomiting of fresh blood, expressed tachycardia, falling of arterial pressure are observed. In such patients it is possible to find the signs of cirrhosis of liver and portal hypertension.

The cancer of stomach in the destruction stage can be also complicated by bleeding. Such bleeding often are not massive, and chronic character is carried mostly with gradual growth of anemia. Worsening of the general condition of patient, loss of weight, decreasing of appetite and waiver of meat are inherent in this pathology.

The gastric bleeding can be related to the diseases of the cardio vascular system (atherosclerosis, hypertensive disease). This is mainly seen in the older people.

Other diseases to be differentiated from ulcer bleeding are: the Mallory-Weiss syndrome, benign tumors of stomach and duodenum (more frequent leiomyoma), hemorrhagic gastritis, acute (stress) erosive defects of stomach, arteriovenous fistula of mucosa.

Diagnosis program:

1. Anamnesis and physical examination.
2. Digital examination of rectum.
3. EFGDS.
4. Lab studies.
5. Other necessary instrumental examinations.

Lab studies.

CBC with platelet count and differential: *CBC* is necessary to assess the level of blood loss. Where possible, having the patient's previous results is useful to gauge the level of blood loss. *CBC* should be checked frequently (4-6h) during the first day.

Basic metabolic profile: it is useful to evaluate for renal comorbidity.

Measurement of coagulation parameters is necessary to assess for continued bleeding. Abnormalities should be corrected rapidly.

Prothrombin time/activated partial thromboplastin time.

Liver profile: the liver profile can identify hepatic comorbidity and suggest underlying liver disease.

Calcium level: a calcium level is useful to identify the patient with hyperparathyroidism as well as to monitor calcium in patients receiving multiple transfusions of citrated blood.

Gastrin level: a gastrin level can identify the rare patient with gastrinoma as the cause of peptic ulcer bleeding and multiple ulcers.

Imaging studies.

Chest and abdominal radiographs should be ordered to exclude aspiration pneumonia, effusion, and esophageal perforation; abdominal scout and upright films should be ordered to exclude perforated organs and ileus.

Barium contrast studies are not usually helpful and can make endoscopic procedures more difficult (i.e., white barium obscuring the view) and dangerous (i. e., risk of aspiration).

CT scan and ultrasonography may be indicated to evaluate liver disease with cirrhosis, cholecystitis with hemorrhage, pancreatitis with pseudocyst and hemorrhage, aortoenteric fistula, and other unusual causes of upper gastrointestinal hemorrhage.

Nuclear medicine scans may be useful to determine the area of active hemorrhage.

Angiography may be useful if bleeding persists and endoscopy fails to identify a bleeding site. As salvage therapy, embolization of the bleeding vessel can be as successful as emergent surgery in patients who have failed a second attempt of endoscopic therapy.

Procedures.

Nasogastric lavage. This procedure may confirm recent bleeding (coffee ground appearance), possible active bleeding (red blood in the aspirate that does not clear), or a lack of blood in the stomach (active bleeding is possible but does not exclude an upper gastrointestinal lesion).

A nasogastric tube is an important diagnostic tool, and tube placement can reduce the patient's need to vomit. Placement for diagnostic purposes is not contraindicated in patients with possible esophageal varices.

The characteristics of the nasogastric lavage fluid (e. g., red, coffee grounds, clear) and the stool (e. g., red, black, brown) can indicate the severity of the hemorrhage. Red blood with red stool is associated with an increased mortality rate from more active bleeding compared with negative aspirate findings with brown stool.

Tactic and choice of treatment method.

Main tasks in treatment of patients with acute peptic ulcer hemorrhage are:

1. Stopping of flow of blood.
2. Removal of consequences of bleeding (correction of blood volume circulation, cardiac, renal, liver disturbances and others).
3. Antysecretory therapy.
4. Moving off blood from intestine lumen (clysis).
5. Immunocorrection.

Stopping of bleeding may be achieved using 3 ways: endoscope, conservative therapy, operation.

Most patients (85–90 %) respond to endoscopic therapy. After fiber-optic endoscopy inspection and visualization of the source of bleeding endoscopy hemostasis has been performed using one from next ways:

– periulcerous injection of vasoconstrictors and procoagulators;

- electro coagulation;
- laser coagulation;
- plasma coagulation;
- covering over the bleeding ulcer with polymer film;
- clipping of vessels.

A combination of therapies has become more common.

Injection therapy is applied first to better clarify the bleeding site, especially in the actively bleeding patient; then, heater probe or bipolar probe coagulation is applied. Injection therapy can also be performed prior to endoscopic placement of hemoclips. Injection therapy is useful prior to laser therapy to reduce the heat sink effect of rapidly flowing blood prior to laser coagulation.

The use of argon plasma coagulation for ulcer hemorrhage, however, there are significant theoretical and experimental considerations from animal models about the use of a noncontact monopolar cautery in this situation.

The bipolar probe consists of alternating bands of electrodes producing an electrical field that heats the mucosa and the vessel.

The electrodes are coated with gold to reduce adhesiveness. The probes are stiff to allow adequate pressure to the vessel to oppose the walls and thus produce coagulation when the energy is transmitted (heat energy by the heater probe, electrical-field energy by the bipolar probe). Careful technique is required to heat-seal the perforated vessel.

Alternatively, epinephrine (1:10,000, 1:20,000) can be injected in 0.5 ml aliquots around the base of the vessel. This causes coagulation by compression, with perhaps some additive effect of activation of platelet factor 3 or vasoconstriction. Other solutions that have been used for injection are ethanol (more necrosis), hypertonic saline, sterile water, and cyanoacrylate.

Epinephrine injection is often used to reduce the volume of bleeding so that the lesion can be better localized and then treated with a coaptive technique (i.e., heater probe, gold probe). Such combination therapy has become more frequent and has evolved into the standard technique.

Laser therapy is rarely used. To perform laser coagulation, the area near the vessel is first injected with epinephrine to reduce blood flow (reducing the heat-sink effect); then, the laser is applied around the vessel (producing a wall of edema). Caution must be observed to avoid drilling into the vessel with the laser, causing increased bleeding.

Hemostatic clips have recently become available in the United States. With careful placement of the clip, closing the defect in the vessel is possible. Usually, multiple clips are applied. The clipping devices are single-use devices with a single clip deployed with each instrument.

Argon plasma coagulation is a technique in which a stream of electrons flows along a stream of argon gas. The coagulation is similar to monopolar cautery with the current flow going from a point of high current density, the point of contact of the gas with the mucosa, to an area of low current density, the conductive pad on the patient's body. The current flows through the body in an erratic path to the pad. This monopolar cautery technique is similar to the laser technique in that energy is delivered to the vessel for coagulation with apposition of the vessel walls. This technique was not effective for visible vessels larger than 1 mm.

Contraindications to emergency fiber-optic endoscopy include:

- severe cardiac and lung decompensation;
- acute myocardial infarction;
- impaired cardiopulmonary status and bleeding diathesis;

Emergency esophagogastroduodenoscopy may be more difficult or impossible if the patient has had previous oropharyngeal surgery or radiation therapy to the oropharynx.

The presence of a Zenker diverticulum can make intubation of the esophagus more difficult.

After endoscopy hemostasis conservative therapy has been started.

It must include:

- prescription of hemostatic (intravenously the aminocaproic acid 5 % ~ 200–400 ml, chorus calcium 10 % – 10,0 ml, vicasol 1 % – 3,0 ml);
- increasing of the volume of circulatory blood (gelatin, reopoliglukine, salt blood substitutes);
 - preparations of blood (fibrinogen – 2–3 g, cryoprecipitate);
 - blood substitutes therapy (red corpuscles mass, washed red corpuscles, plasma of blood);
 - H₂-receptor antagonists or proton pump inhibitors;
 - antacids and adsorbents (almagel, phosphalugel, maalox – for 1–2 dessert-spoons through 1 hour after food intake).

It is important to wash stomach with ice water and the use 5 % solution of aminocaproic acid 1 tablespoon every 20–30 minutes.

Absolute indications for surgical treatment are:

- 1) prolonged bleeding;
- 2) recurrent bleeding;
- 3) perforated bleeding ulcer.

Relative indications for surgery may be established in patients with high risk of recurrent bleeding: Forrest I or II attached to “gigantic” (more than 20 mm in diameter) ulcers, combination of bleeding with stenosis and penetration.

It's better to perform surgery in the patient at high risk of recurrent bleeding during 24–48 hours after removal of consequences of acute bleeding.

Angiographic embolization is an option in the patient at high risk for surgical intervention. Angiographic embolization of the gastroduodenal or the left gastric arteries may be effective, but there are dangers involved. A foreign body may slip from the gastroduodenal into the hepatic artery and lead to hepatic necrosis; one in the left gastric artery may lead to necrosis of the upper portion of the stomach.

Operations for massively bleeding duodenal ulcers.

The site of bleeding must be determined. If the source is the gastroduodenal artery, it must be ligated as the first step. This procedure involves suture of the artery, either above the duodenum as it emerges from the hepatic artery or within the duodenal lumen, suture of the caudal portion of the artery within the duodenum or of the two major branches; the superior pancreaticoduodenal and the right gastroepiploic; and of the transverse pancreatic artery. Heavy, non-absorbable sutures are used. In other instances the duodenum may be boggy and bleeding from multiple areas; in such instances it may be impossible to identify single vessels to ligate.

As soon as bleeding has been controlled, a definitive operation can be carried out. We believe that ulcer excision and pyloroduodenoplasty with/without selective vagotomy is best. A major determinant in the selection is the experience of the surgeon. If vagotomy wasn't performed, H₂-receptor antagonists or proton pump inhibitors has been prescribed for prevention of ulcer recurrence and its complications.

Partial gastrectomy (the usual procedure involves resection of the distal two-thirds of the stomach) is more difficult, but gives slightly better control of bleeding because it removes areas of gastritis and duodenitis that are potential sources of postoperative bleeding.

Operations for bleeding gastric ulcers.

Ulcer excision and pyloroduodenoplasty is preferred for bleeding ulcers.

For gastric ulcer resection either a Billroth I or II anastomosis can be made.

Total gastrectomy cannot be determined in a patient who continues to bleed.

Chapter 7. BOWEL OBSTRUCTION

Bowel obstruction is most common in surgery practice. It often leads to different complications and death of patients. Physician has to diagnose a case, to define the tactics of treatment, to choose the optimum method of treatment in patients with bowel obstruction.

Classification(<https://slideplayer.com/slide/3722974/13/images/4/Classification.jpg>):

Classification

Classification of intestinal obstruction can be made according to –

- Pathology
- Anatomy
- Nature of presentation and
- Pathological changes in the bowels

1) Classification according to pathology

- Dynamic: where peristalsis is working against a mechanical obstruction
- Adynamic: where there is absence of peristalsis without obstruction
 - Absent peristalsis (e.g. Paralytic ileus)
 - Non- propulsive form (e.g. Mesenteric vascular occlusion and pseudo-obstruction)

2) Classification according to anatomy

- Small bowel obstruction –
 - High or
 - Low
- Large bowel obstruction

3) Classification according to the nature of presentation

- Acute
- Chronic
- Acute on chronic
- Subacute

4) Classification according to pathological changes

- Simple: where the blood supply is intact
- Strangulated: where there is direct interference to blood flow

A small-bowel obstruction (SBO).

It is caused by a variety of pathologic processes. The most common cause of SBO is postsurgical adhesions. Postoperative adhesions can be the cause of acute obstruction within 4 weeks of surgery or of chronic obstruction decades later. The incidence of SBO parallels the increasing number of laparotomies performed in developing countries. Other etiologies of SBO include malignant tumor (20 %), hernia (10 %), inflammatory bowel disease (5 %), volvulus (3 %).

SBOs can be partial or complete, simple (i. e., nonstrangulated) or strangulated. If not diagnosed and properly treated, vascular compromise leads to bowel ischemia and further morbidity and mortality. Because as many as 40 % of patients have strangulated obstructions, differentiating the characteristics and etiologies of obstruction is critical to proper patient treatment.

Frequency.

SBO accounts for 20 % of all acute surgical admissions. Mortality and morbidity are dependent on the early recognition and correct diagnosis of obstruction. If untreated, strangulated obstructions cause death in 100 % of patients. If surgery is performed within 36 hours, the mortality decreases to 8 %. The mortality rate is 25 % if the surgery is postponed beyond 36 hours in these patients.

Pathophysiology.

Obstruction of the small bowel leads to proximal dilatation of the intestine due to accumulation of GI secretions and swallowed air. This bowel dilatation stimulates cell secretory activity resulting in more fluid accumulation. This leads to increased peristalsis both above and below the obstruction with frequent loose stools and flatus early in its course.

Vomiting occurs if the level of obstruction is proximal. Increasing small-bowel distention leads to increased

intraluminal pressures. This can cause compression of mucosal lymphatics leading to wall lymphedema. With even higher intraluminal hydrostatic pressures, increased hydrostatic pressure in the capillary beds results in massive third spacing of fluid, electrolytes, and proteins into the intestinal lumen. The fluid loss and dehydration that ensue may be severe and contribute to increased morbidity and mortality. Strangulated SBOs are most commonly associated with adhesions and occur when a loop of distended bowel twists on its mesenteric pedicle. The arterial occlusion leads to bowel ischemia and necrosis. If left untreated, this progresses to perforation, peritonitis, and death. Bacteria in the gut proliferate proximal to the obstruction. Microvascular changes in the bowel wall allow translocation to the mesenteric lymph nodes. This is associated with an increase in incidence of bacteremia due to *Escherichia coli*, but the clinical significance is unclear.

History.

Abdominal pain (characteristic with most patients). Pain, often described as crampy and intermittent, is more prevalent in simple obstruction. Often, the presentation may provide clues to the approximate location and nature of the obstruction. Usually pain that occurs for a shorter duration of time and is colicky and accompanied by bilious vomiting may be more proximal. Pain lasting as many as several days, which are progressive in nature and with abdominal distention, may be typical of a more distal obstruction. Changes in the character of the pain may indicate the development of a more serious complication (i. e., constant pain of strangulated or ischemic bowel).

Nausea.

Vomiting. This is associated more with proximal obstructions.

Diarrhea (an early finding).

Constipation (a late finding) as evidenced by the absence of flatus or bowel movements.

Previous abdominal or pelvic surgery and/or previous radiation therapy (may be part of patient's medical history).

History of malignancy (particularly ovarian and colonic).

Physical signs.

The Tiliyaks – Vikker sign. The cramp – like abdominal pain.

The Shlange sign. Sometimes you visually peristaltic movements of the intestine, which is intensified after abdominal palpation.

The Hirschsprung sign. The anal sphincter “hiatus” in bowel invagination.

The Kruvelye sign. The bleeding from the anus in bowel invagination..

The Leedsky sign. The auscultation determines "long, different sonority sounds, even short sounds".

The Spasokukotsky sign. In small bowel obstruction auscultation you determine “the sound of falling drop”.

The Roush sign. Palpation of elastic, moderately painful, “tumor-like” formation in the abdominal cavity causes the tenesmus.

The Mondor sign. In the later periods of acute bowel obstruction some anterior abdominal wall muscle resistance can be revealed.

The Val sign. The enlarged bowel loop is palpated, sometimes with asymmetry of abdomen.

The Sklyarov sign. “Splashing”. The moderate shocks of abdominal wall + auscultation: they determine the splashing of liquid witch is accumulated in the alimentary canal lumen.

The Grekov – "Obukhov Hospital" sign. The rectal digital examination demonstrates empty rectum and “hiatus” of anal sphincter.

The Shymon-Dance sign. The right iliac region by palpation is felt “free” because the invagination of the cecum.

The Mathieu sign. The finger-tips percussion above the umbilicus due to "splashing".

The Kivul sign. The percussion over the dilatated with gas and liquid bowel loop detects tympanitis with metallic shade.

Test of Bebuk. The presence of red blood cells in the washing waters after the enema.

The Tsege-Manteufel sign. Only 500 ml. of water can be passed in the rectum with cleaning enema.

Abdominal distention. Duodenal or proximal small bowel has less distention when obstructed than the distal bowel has when obstructed.

Hyperactive bowel sounds occur early as GI contents attempt to overcome the obstruction. Hypoactive bowel sounds occur late.

Exclude incarcerated hernias of groin, femoral triangle, and obturator foramina. Proper genitourinary and pelvic examinations are essential.

Look for the following during rectal examination. Gross or occult blood, which suggests late strangulation or malignancy.

Check for symptoms commonly believed to be more diagnostic of intestinal ischemia, including the following:

- fever;
- tachycardia (> 100 beats/min);
- peritoneal signs.

No reliable way exists to differentiate simple from early strangulated obstruction on physical examination. Serial abdominal examinations are important and may detect changes early.

Lab Studies.

Serum chemistries: Results are usually normal or mildly elevated.

Creatinine. Elevations may indicate dehydration.

CBC may be elevated with a left shift in simple or strangulated obstructions. Increased hematocrit speaks to volume state (i.e., dehydration).

Lactate dehydrogenase tests

Urinalysis

Liver panels

Imaging studies.

AXR. Order plain radiographs first for patients in whom SBO is suspected. At least 2 views, supine or flat and upright, are required. Too findings were more predictive of a higher grade or complete SBO: presence of air-fluid differential height in the same small-bowel loop and presence of a mean level width greater than 25 mm. When these findings are present, the obstruction is most likely high grade or complete. When both are absent, a low (partial)-grade SBO is likely or nonexistent. Absent or minimal colonic gas indicates SBO (*fig.19*).

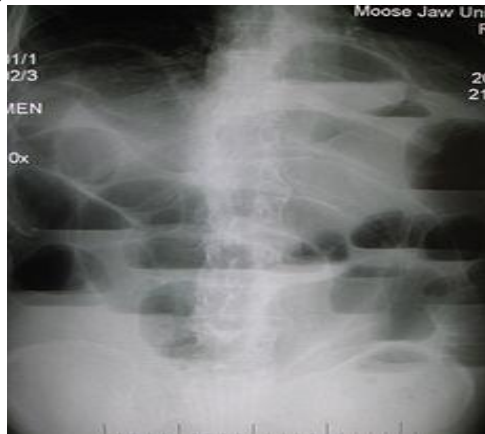


Figure 19 – Multiple air fluid levels (<https://aneskey.com/wp-content/uploads/2017/02/00184.jpeg>)

Small bowel loops contain transverse folds known as ‘plica circularis’. These folds are well seen in this patient with small bowel obstruction. Usually the colon is decompressed and hardly visible (fig. 20).

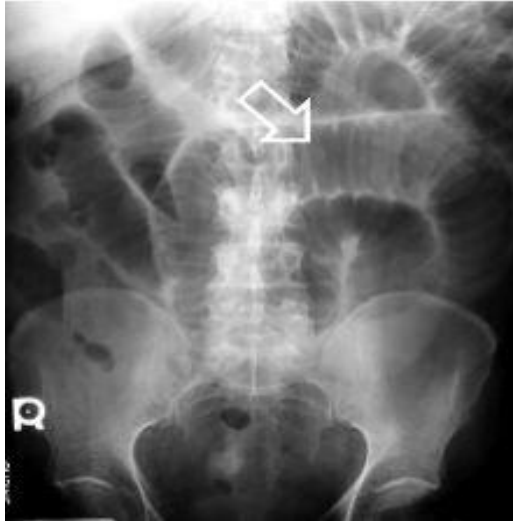


Figure 20 – Plica circularis

(<https://aneskey.com/wp-content/uploads/2017/02/00184.jpeg>)
Enteroclysis.

This is valuable in detecting presence of obstruction and in differentiating partial from complete blockages. This study is useful when plain radiographic findings are normal in the presence of clinical signs of SBO or if plain radiographic findings are nonspecific. It distinguishes adhesions from metastases, tumor recurrence, and radiation damage. Enteroclysis offers a high negative predictive value and can be performed with 2 types of contrast. Barium is the classic contrast agent used in this study. It is safe and useful when diagnosing obstructions provided no evidence of bowel

ischemia or perforation exists. Barium has been associated with peritonitis and should be avoided if perforation is suspected.

CT scanning is useful in making an early diagnosis of strangulated obstruction and in delineating the myriad other causes of acute abdominal pain, particularly when clinical and radiographic findings are inconclusive. It also has proved useful in distinguishing the etiologies of SBO, i.e., extrinsic causes such as adhesions and hernia from intrinsic causes such as neoplasms or Crohn disease. It also differentiates the above from intraluminal causes such as bezoars. CT scanning is about 90% sensitive and specific in diagnosing SBO. CT scanning is the study of choice if the patient has fever, tachycardia, localized abdominal pain, and/or leukocytosis. It is capable of revealing abscess, inflammatory process, extraluminal pathology resulting in obstruction, and mesenteric ischemia. CT scanning enables the clinician to distinguish between ileus and mechanical small bowel in postoperative patients. Obstruction is present if the small-bowel loop is greater than 2.5 cm in diameter dilated proximal to a distinct transition zone of collapsed bowel less than 1 cm in diameter (fig. 21).



Figure 21 – Note the dilated small bowel loops with a focal transition zone to distal collapsed bowel
(<https://assets.cureus.com/uploads/figure/file/61687>)

A smooth beak indicates simple obstruction without vascular compromise; a serrated beak may indicate strangulation. Bowel wall thickening indicates early strangulation. CT scanning is useful in identifying abscesses, hernias and tumors.

US is not more invasive than CT scanning. It may reliably exclude SBO in as many as 89 % of patients. Specificity is reportedly 100 %.

Ultrasonography signs of SBO:

- dilatation of small bowel lumen;
- “pendulous” movements of bowel content.

Treatment.

A strangulated obstruction is a surgical emergency. In patients with a complete SBO, the risk of strangulation is high and early surgical intervention is warranted. Patients with simple complete obstructions in whom nonoperative trials fail also need surgical treatment but experience no apparent disadvantage to delayed surgery.

Adhesions. Decreasing intraoperative trauma to the peritoneal surfaces can prevent adhesion formation.

Malignant tumor. Obstruction by tumor is usually caused by metastasis. Initial treatment should be nonoperative; surgical resection is recommended when feasible.

Inflammatory bowel disease. To reduce the inflammatory process, treatment generally is nonoperative in combination with high-dose steroids. Consider parenteral treatment for prolonged periods of bowel rest. Undertake surgical treatment, bowel resection, and/or stricturoplasty if nonoperative treatment fails.

Intraabdominal abscess. CT-guided drainage is usually sufficient to relieve obstruction.

Radiation enteritis. If obstruction follows radiation therapy acutely, nonoperative treatment accompanied by

steroids is usually sufficient. If obstruction is a chronic sequel of radiation therapy, surgical treatment is indicated.

Acute postoperative obstruction. This is difficult to diagnose because symptoms often are attributed to incisional pain and postoperative ileus. Treatment should be nonoperative. If nonoperative treatment fails, surgical treatment is indicated.

Prognosis. With proper diagnosis and treatment of the obstruction, prognosis is good. Complete obstructions treated successfully no operatively have higher incidence of recurrence than those treated surgically.

Large bowel obstruction (LBO).

LBO may be caused by neoplasms or anatomic abnormalities such as volvulus, incarcerated hernia, stricture or constipation. LBO from an anatomic abnormality leads to colonic distention, abdominal pain, anorexia, and, late in the course, feculent vomiting. Persistent vomiting may result in dehydration and electrolyte disturbances.

LBO is a surgical entity. The morbidity and mortality often are related to the surgical procedure used to relieve the colonic obstruction and, in the long term, to the underlying disease that caused the obstruction.

Colonic obstruction is most common in elderly individuals because the incidence of neoplasms and other causative diseases is higher in this population.

Hirschsprung disease resembles colonic obstruction in the pediatric population.

History.

History focuses initially on the failure to pass stools or gas. One should attempt to distinguish complete bowel obstruction from partial obstruction, which is associated with passage of some gas or stools, and from ileus. Further historical questioning may be directed at the patient's current and past history in an attempt to determine the most likely

cause. Complete obstruction is characterized by the failure to pass either stools or flatus and the presence of an empty rectal vault upon rectal examination, unless the obstruction is in the rectum. Partial obstruction, in which the patient appears obstipated but continues to pass some gas or stools, is a less urgent condition. Distinguishing colonic ileus from organic obstruction is important. Ileus may be suggested by abdominal pain as a dominant feature of the clinical presentation, by peritoneal signs, or by the presence of pronounced fever and leukocytosis. LBO, or even constipation, may be accompanied by some degree of fever or leukocytosis. Similarly, based on peritoneal signs, distinguishing the tender gas-filled and stool-filled colon observed in organic obstruction from a tender abdomen due to peritonitis is difficult. Obtaining a thorough history of previous bowel function, abdominal pain, and general systemic issues is important. History of chronic weight loss and passage of melanotic bloody stools suggest neoplastic obstruction. Conversely, a history of recurrent left lower quadrant abdominal pain over several years is more consistent with diverticulitis, a diverticular stricture, or similar problems. A history of aortic surgery suggests the possibility of an ischemic stricture.

Physical examination.

Abdominal examination. Perform the examination in standard fashion, i.e., inspection, auscultation, percussion, and palpation. Large bowel obstruction may be characterized by diminished or, in later stages, absent bowel sounds. The abdomen is distended and may be tender. The presence of true involuntary guarding or peritoneal signs should raise the specter of another intra-abdominal process, such as an abscess. The practice of seeking rebound tenderness is misleading and potentially cruel. Many patients without peritoneal signs complain vigorously after an aggressive rebound maneuver.

Seeking tenderness and pain by having the patient cough or by shaking the bed probably is more useful.

Examination of inguinal and femoral regions. This should be an integral part of the examination. Incarcerated hernias represent a frequently missed cause of bowel obstruction. In particular, colonic obstruction often is caused by a left-sided inguinal hernia with the sigmoid colon incarcerated in the hernia.

Digital rectal examination. Perform this to verify the patency of the anus in a neonate. The examination focuses on identifying rectal pathology that may be causing the obstruction and determining the contents of the rectal vault. Hard stools suggest impaction. Soft stools suggest obstipation. An empty vault suggests obstruction proximal to the level that the examining finger can reach. Fecal occult blood testing should be performed, and a positive result may suggest the possibility of a more proximal neoplasm.

Lab Studies.

Studies are directed at evaluating the dehydration and electrolyte imbalance that may occur as a consequence of large bowel obstruction and at ruling out ileus as a diagnosis. Suggestion of an abnormal anion gap also should prompt an arterial blood gas measurement and/or a serum lactate level measurement. A decreased hematocrit level, particularly with evidence of chronic iron-deficiency anemia, may suggest chronic lower gastrointestinal bleeding, particularly due to colon cancer. A stool test also should be performed, for similar reasons. Although bowel obstruction, or even constipation, may mildly elevate the WBC count, substantial leukocytosis should prompt reconsideration of the diagnosis. Ileus, secondary to an intra-abdominal or extra-abdominal infection or another process, is a possibility.

Imaging Studies. AXR demonstrates dilation of the large bowel and air fluid levels. Colonic air suggests the

anatomic location of the obstruction. A dilated colon without air in the rectum is more consistent with obstruction. The presence of air in the rectum is consistent with obstipation, ileus, or partial obstruction. This finding can be misleading, particularly if the patient has undergone rectal examinations or enemas. The characteristic bird's beak of volvulus may be seen.

If differentiation between obstipation and obstruction is required, imaging with contrast is indicated (fig. 22). If localization is required for surgical intervention, imaging with contrast is indicated. Water-soluble Gastrografin has important advantages over barium as a contrast agent and generally should be employed first. It usually does not cause chemical peritonitis if the patient has colonic perforation.

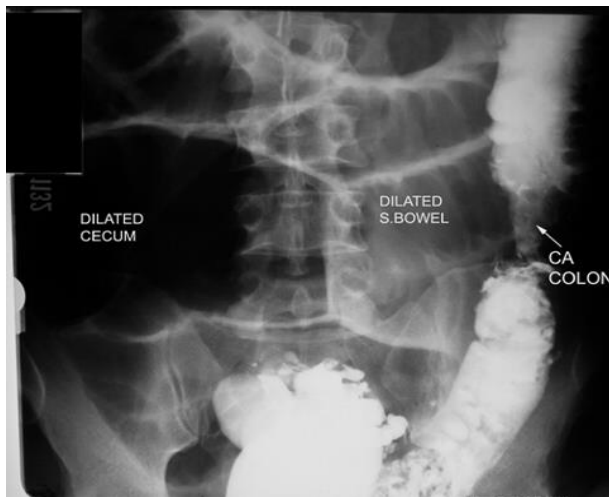


Figure 22 – Left side colonic cancer with obstruction
(<https://www.wjgnet.com/1948-5190/full/v11/i3/193.htm>)

CT scanning is not used initially in patients with large bowel obstruction unless a diagnosis has been made. CT scan, particularly with rectal contrast, may demonstrate a mass or evidence of metastatic disease.

Other tests. Fiber – optic endoscopy may be useful in evaluating left-sided colonic obstruction, including the anatomic location and pathology of the lesion. Because the cecum is not reached in such cases, the endoscopist must be alert to the possibility of incorrectly identifying anatomic landmarks and the location of the obstruction. Although flexible endoscopy is relatively comfortable for the patient and provides a better view than rigid sigmoidoscopy, the latter also may be used, depending on the availability of resources and training of personnel. Right-sided colonic obstruction is more difficult to evaluate without first administering an oral bowel preparation, which is contraindicated in the setting of bowel obstruction.

Procedures.

Endoscopic reduction of volvulus. This procedure is indicated for sigmoid volvulus when peritoneal signs are absent, which would imply dead bowel or perforation. It also is indicated when evidence of mucosal ischemia is not present upon endoscopy. An experienced person should perform the procedure. A rigid sigmoidoscopy may be used if a flexible instrument is not available. The endoscopist must have sufficient experience with this technique. Reduction of a volvulus does not imply cure. The sigmoid usually revolvulizes if definitive treatment is not carried out. These patients generally are admitted, subjected to mechanical bowel preparation, and managed surgically by sigmoid resection, unless contraindications are present. Barium enema for reduction of intussusception. This is useful and often successful in children in whom a pathological leading point for the intussusception is unlikely. It should be performed by an experienced radiologist because the risk of perforation is significant. In adults, typically a pathologic leading point for the intussusception is present. Success is far less likely, and patients still require surgery to deal with their pathology.

Cleansing enemas. Perform these if obstipation is suspected rather than true large bowel obstruction. Also perform them to prepare the distal colon for endoscopic evaluation. Endoscopic dilation and stenting of colonic obstruction. This procedure is indicated for colonic near total obstruction through which some small amount of lumen remains. The procedure may be palliative in a high-risk patient with an unresectable malignancy, accepting a risk of reobstruction of the stent, or preparatory to surgical resection. In cases in which the stent is deployed prior to surgery, it permits relief of the acute obstruction, resuscitation of the patient, and mechanical bowel preparation prior to a one-stage colonic resection and reanastomosis, thus avoiding temporary or permanent colostomy. The procedure should be performed only by an endoscopist experienced in such procedures. Surgical consultation and backup should be available, as the risk of perforation is increased during attempts at such procedures, with a potentially catastrophic result.

Medical Care. It involves resuscitation, correction of fluid and electrolyte imbalance, and nasogastric decompression to temporarily treat the obstruction and prevent vomiting and aspiration.

Surgical Care.

Surgical care is directed at relieving the obstruction. In most patients, the obstructing lesion is resected. Because the colon has not been cleansed, anastomosis often is risky. After resection, most surgeons perform a proximal colostomy if the obstruction is on the left side or ileostomy if it is on the right side. In patients with substantial comorbidity and surgical risk or in the presence of an unresectable tumor, a diverting proximal colostomy or ileostomy may be performed without resection. A diverting transverse loop colostomy may be the least invasive procedure for a very ill patient with a left colonic obstruction. It permits relief of the obstruction and further

resuscitation without compromising chances for a subsequent resection. A sigmoid colostomy without resection may be employed in patients with rectal obstruction that cannot be managed without a combined abdominoperineal approach. Cecostomy should not be performed because the diversion is inadequate. In younger patients some surgeons would consider primary anastomosis, rather than ileostomy, in the right colon, assuming no intraoperative hypotension, blood loss, or other complications are present. If resection and proximal colostomy or ileostomy is performed, a mucous fistula generally is extracted from the distal end, unless the obstruction is rectosigmoid, in which case the distal end may be oversewn or stapled and left to drain transanally. If the cause of the obstruction can be relieved nonsurgically, through procedures such as decompressing a volvulus, or if the obstruction is only partial, deferring surgery temporarily and supporting the patient while the large bowel is cleansed so that primary anastomosis may be performed more safely is preferable.

Further Outpatient Care.

Care after discharge focuses on surgical convalescence and, if relevant, the need to care for the disease that caused the obstruction. An obstructing colon cancer may require postoperative chemotherapy, depending on the stage of the disease. The patient who is chronically obstipated may need stool softeners. If the patient has received a colostomy or ileostomy, a decision regarding whether it is temporary or permanent may have been made at the time of discharge, depending on the patient's diagnosis, comorbidity, and postoperative convalescence. Most patients who retain a rectum are, at least in principle, candidates for reanastomosis at a subsequent stage. Generally, it is performed 2–3 months after the initial operation. Careful counseling and assessment are required before proceeding with the second procedure. Counseling is directed at the risks of the second procedure

because the patient must understand that this surgery is elective and that a colostomy or ileostomy is compatible with a reasonable lifestyle. Often, local colostomy support groups and meeting with other patients with colostomies are helpful at this time. Patients who had stool incontinence before their first operation, those with substantial surgical risks, and patients with decreased mental status who are cared for in nursing homes may potentially be better off without a reanastomosis. In addition, the remaining colon, both proximally and distally, must be evaluated radiographically or endoscopically to rule out synchronous colonic lesions such as neoplasms because the presence of the large bowel obstruction prevented this from being performed before the first procedure.

Prognosis.

Prior to surgical decompression, the patient's overall medical condition and presence of any comorbidities that define surgical risk determine the prognosis. After surgical decompression, prognosis is determined by the underlying disease.

Ileus.

After abdominal surgery, a normal physiological ileus occurs. This type of ileus spontaneously resolves within 2–3 days after sigmoid motility returns to normal. However, the terms postoperative adynamic ileus or paralytic ileus are defined as ileus of the gut persisting for more than 3 days following surgery. Ileus occurs from hypomotility of the gastrointestinal tract in the absence of a mechanical bowel obstruction. This suggests that the muscle of the bowel wall is transiently impaired and fails to transport intestinal contents. This lack of coordinated propulsive action leads to the accumulation of both gas and fluids within the bowel. Although ileus has numerous causes, the postoperative state is the most common scenario for ileus development. Frequently, ileus occurs after intraperitoneal operations, but it may also

occur after retroperitoneal and extra-abdominal surgery. The longest duration of ileus is noted to occur after colonic surgery.

Causes of adynamic ileus:

Most cases of ileus occur after intra-abdominal operations.

Sepsis.

Drugs (e. g., opioids, antacids, coumarin, amitriptyline, chlorpromazine).

Metabolic (e.g., low potassium, magnesium, or sodium levels; anemia, hyposmolality).

Myocardial infarction.

Pneumonia.

Trauma (e. g., fractured ribs, fractured spine).

Biliary and renal colic.

Head injury and neurosurgical procedures.

Intra-abdominal inflammation and peritonitis.

Retroperitoneal hematomas.

Pathophysiology.

According to some hypotheses, postoperative ileus is mediated via activation of inhibitory spinal reflex arcs. Anatomically, 3 distinct reflexes are involved: ultrashort reflexes confined to the bowel wall, short reflexes involving prevertebral ganglia and long reflexes involving the spinal cord. The long reflexes are the most significant. Spinal anesthesia, abdominal sympathectomy, and nerve-cutting techniques have been demonstrated to either prevent or attenuate the development of ileus. The surgical stress response leads to systemic generation of endocrine and inflammatory mediators that also promote the development of ileus. Rat models have shown that laparotomy, eventration, and bowel compression lead to increased numbers of macrophages, monocytes, dendritic cells, T cells, natural killer cells, and mast cells, as demonstrated by immunohistochemistry. Calcitonin gene-related peptide, nitric oxide, vasoactive intestinal peptide, and substance P function as inhibitory neurotransmitters in the

bowel nervous system. Nitric oxide and vasoactive intestinal peptide inhibitors and substance P receptor antagonists have been demonstrated to improve gastrointestinal function.

History. Patients with ileus typically present with vague, mild abdominal pain and bloating. They may report nausea, vomiting, and poor appetite. Abdominal cramping is usually not present. Patients may or may not continue to pass flatus and stool.

Physical examination. Patients may have distended and tympanic abdomens, depending on the degree of abdominal and bowel distension. The abdomen may be tender. A distinguishing feature is absent or hypoactive bowel sounds unlike the high-pitched sound of obstruction. The silent abdomen of ileus reveals no discernible peristalsis or succession splash.

Laboratory studies and blood work should focus on evaluations for infectious, electrolytic, and metabolic derangements.

Imaging studies:

On plain AXR, ileus appears as copious gas dilatation of both small intestine and colon. With enteroclysis, the contrast medium in patients with paralytic ileus should reach the cecum within 4 hours; if it remains stationary for longer than 4 hours, mechanical obstruction is suggested.

Medical Care.

Most cases of postoperative ileus resolve with watchful waiting and supportive treatment. Patients should receive intravenous hydration. For patients with vomiting and distension, use of a nasogastric tube provides symptomatic relief.

Underlying sepsis and electrolyte abnormalities, particularly hypokalemia, hyponatremia, and hypomagnesemia, may worsen ileus. These contributing conditions are easily diagnosed and corrected.

Discontinue medications that produce ileus (e. g., opiates). The use of postoperative narcotics can be diminished by supplementation with NSAID. NSAIDS may improve ileus by improving local inflammation and by decreasing the amount of narcotics used.

The presence of ileus does not preclude enteral feeding. Postpyloric tube feeding into the small bowel can be performed.

Use of prokinetic agents has had moderate success, a serotonin agonist, has reportedly been successful in treating ileus.

TESTS FOR CONTROL

Chapter 1 Acute appendicitis

Tests (one answer is correct)

1. Acute appendicitis is:

- A) inflammation of the appendix;
- B) inflammation of the appendix with its perforation;
- C) nonspecific inflammation of the inner lining of the vermiform appendix that spreads to its other parts;
- D) purulent inflammation of the inner lining of the vermiform appendix that spreads to its other parts.

2. Kolesov's classification of acute appendicitis:

- A) edematous stage, purulent stage, gangrenous stage;
- B) appendicular colic, destructive appendicitis (phlegmonous, gangrenous, perforated), complicated;
- C) no complicated and complicated;
- D) phlegmonous, gangrenous, perforated.

3. Abdominal pain in patients with acute appendicitis:

- A) sudden, intensive, localized in the right part of abdomen;

- B) often begins gradually in the right lower quadrant of the abdomen;
- C) usually begins as periumbilical or epigastric pain migrating to the right lower quadrant of the abdomen;
- D) often begins as periumbilical or epigastric pain, accompanies with vomiting, nausea high temperature.

4. Describe Rovsign 's sign:

- A) passive internal rotation of flexed right thigh with the patient in supine position causes pain;
- B) light percussion on McBurney's point will elicit pain;
- C) pain in the right lower quadrant is complained of when palpation pressure is exerted in the left lower quadrant;
- D) strengthening pain in the right lower square in the position of patient on the left side.

5. Describe Sitkoosky's sign:

- A) passive internal rotation of flexed right thigh with the patient in supine position causes pain;
- B) light percussion on McBurney's point elicits pain;
- C) pain in the right lower quadrant is complained of when palpation pressure is exerted in the left lower quadrant;
- D) strengthening pain in the right lower square in the position of patient on the left side.

6. Main method in diagnostic procedure is:

- A) X-ray examination;
- B) ultrasound;
- C) lab. studies;
- D) anamnestic and clinical examination.

7. Peculiarities of trends of acute appendicitis in elderly patients:

- A) often retrocaecal position of appendix;
- B) often pain accompanies with vomiting, nausea;
- C) no evident clinical picture + rapid destructive changes in appendix;
- D) often spontaneous regression of inflammation.

8. Patient 20 years old has pain in upper part of abdominal cavity with vomiting, nausea during last 2 hours. Optimal tactic is:

- A) urgent operation;
- B) antibiotics;
- C) hospitalization and observation at surgical department;
- D) observation at home by family doctor.

9. Optimal final method of differential diagnostic of acute appendicitis is:

- A) laparoscopy;
- B) ultrasound;
- C) lab. studies;
- D) anamnestic and clinical examination.

10. In preoperative preparation in patients with acute appendicitis we use:

- A) analgetics;
- B) spasmolitics;
- C) antibiotics;
- D) all answers are not correct.

11. In patients with acute appendicitis and clinical manifestation of local peritonitis surgeons use:

- A) McBurney's incision of abdominal wall;
- B) Shprengel's incision of abdominal wall;
- C) Koher's incision of abdominal wall;
- D) middle line laparotomy.

12. In patients with acute appendicitis and clinical manifestation of general peritonitis surgeons use:

- A) McBurney's incision of abdominal wall;
- B) pararectal incision of abdominal wall;
- C) Koher's incision of abdominal wall;
- D) middle line laparotomy.

13. Differential diagnostic of appendicular mass:

- A) colonic cancer;
- B) appendicular abscess;

- C) ovarium tumor;
- D) all answers are correct.

14. Final method of differential diagnostic between colonic cancer and appendicular mass is:

- A) laparoscopy;
- B) ultrasound;
- C) colonoscopy + biopsy;
- D) X-ray examination.

15. Final method of differential diagnostic between appendicular abscess and appendicular mass is:

- A) laparoscopy;
- B) ultrasound;
- C) colonoscopy + biopsy;
- D) lab. studies.

16. Clinical manifestation of appendicular abscess:

- A) temperature of body rises to 38,0–39,0 °C;
- B) increasing of the size of the mass;
- C) increasing abdominal pain – suggesting spreading peritonitis;
- D) all answers are correct.

17. Ultrasound examination helps in differential diagnostic between colonic cancer and appendicular mass by:

- A) detecting of adhesions between mass and bowels;
- B) absence of adhesions between mass and bowels;
- C) increasing of mass during 2 weeks;
- D) revealing of metastasis.

18. Treatment of the intraabdominal bleeding from mesoappendix after appendectomy:

- A) laparotomy and stopping of bleeding;
- B) conservative;
- C) surgical in young patients, conservative in elderly;
- D) laparoscopy drainage.

19. Treatment of the appendicular mass:

- A) surgical;

- B) conservative;
- C) surgical in young patients, conservative in elderly;
- D) laparoscopy drainage.

20. Treatment of the pelvic abscess in man:

- A) laparotomy or laparoscopy drainage;
- B) conservative;
- C) surgical in young patients, conservative in elderly;
- D) rectal drainage.

Standards of answers

- 1 – C; 2 – B; 3 – C; 4 – C; 5 – D; 6 – D; 7 – C; 8 – C; 9 – A;
10 – C; 11 – A; 12 – D; 13 – D; 14 – C; 15 – B; 16 – D;
17 – D; 18 – A; 19 – B; 20 – D.

Chapter 2 Acute peritonitis

Tests (one answer is correct)

1. Mark not correct answer. The organs are divided into:

- A) intraperitoneal;
- B) mesoperitoneal;
- C) extraperitoneal;
- D) mixed position.

2. Normally, the amount of peritoneal fluid present is less than:

- A) 10 ml;
- B) 50 ml;
- C) 100 ml;
- D) 150 ml.

3. Classification of peritonitis according to origin includes:

- A) perforated;
- B) serose;
- C) primary;
- D) reactive phase.

4. Classification of peritonitis according to cause includes:

- A) perforated;
- B) serose;
- C) primary;

D) reactive phase.

5. Classification of peritonitis according to character of exudate includes:

- A) perforated;
- B) serose;
- C) primary;
- D) reactive phase.

6. Classification of phases of peritonitis includes:

- A) perforated;
- B) serose;
- C) primary;
- D) reactive phase.

7. Classification of phases of peritonitis doesn't include:

- A) initial;
- B) reactive;
- C) toxic;
- D) terminal.

8. Pathophysiology of the reactive phase includes:

- A) respiratory distress syndrome;
- B) appearance of exudate into abdominal cavity;
- C) adynamic ileus when distended bowel wall loses barrier function;
- D) all answers are correct.

9. Pathophysiology of the toxic phase includes:

- A) respiratory distress syndrome;
- B) appearance of exudate into abdominal cavity;
- C) adynamic ileus when distended bowel wall loses barrier function;
- D) all answers are correct.

10. Pathophysiology of the terminal phase includes:

- A) respiratory distress syndrome;
- B) appearance of exudate into abdominal cavity;
- C) adynamic ileus when distended bowel wall loses barrier function;

D) all answers are correct.

11. General tactic of treatment of acute peritonitis is:

A) conservative;

B) surgical;

C) initial treatment is conservative + surgery for some indications;

D) surgical in young patients, conservative in elderly patients.

12. Tactic of treatment of tuberculous peritonitis is:

A) specific therapy;

B) surgical;

C) initial treatment is conservative + surgery for some indications;

D) surgical in young patients, conservative in elderly patients.

13. Preoperative preparation in acute peritonitis:

A) it is prescribed individually;

B) it isn't indicated;

C) it is necessary for all patients with acute peritonitis;

D) it is necessary in the terminal phase.

14. Antibacterial therapy in acute peritonitis:

A) it isn't indicated;

B) it is starting before the operation;

C) it is starting at the operation;

D) it is starting after the operation.

15. Contraindication for ultrasound- and CT-guided percutaneous drainage of abdominal abscess is:

A) subphrenic abscess;

B) multiple or multiloculated abscesses;

C) subhepatic abscess;

D) parapancreatic abscess.

16. Laparoscopic sanation of the abdominal cavity isn't effective in:

A) perforated peritonitis;

B) pancreatogenic peritonitis;

C) elderly patients;

D) terminal phase with multiloculated abscesses.

17. In patients with general peritonitis surgeons use:

- A) McBurney's incision of abdominal wall;
- B) pararectal incision of abdominal wall;
- C) lockal incisions of abdominal wall;
- D) middle line laparotomy.

18. The main reasons of surgical treatment in acute peritonitis are:

- A) adequate sanitation of abdominal cavity;
- B) source control;
- C) gastrointestinal decompression;
- D) all answers are correct.

19. Required quantity of solutions for adequate sanitation of abdominal cavity in patients with total peritonitis is:

- A) 5 liters;
- B) 10 liters;
- C) 15 liters;
- D) 20 liters.

20. The inspection method after operation is:

- A) open-abdomen technique;
- B) scheduled relaparotomy;
- C) scheduled relaparoscopy;
- D) all answers are correct.

Standards of answers

1 – D; 2 – B; 3 – C; 4 – A; 5 – B; 6 – D; 7 – A; 8 – B; 9 – C;
10 – A; 11 – B; 12 – A; 13 – C; 14 – B; 15 – B; 16 – D;
17 – D; 18 – D; 19 – C; 20 – D.

Chapter 3 Acute cholecystitis

Tests (one answer is correct)

1. Acute cholecystitis is:

- A) inflammation of gallbladder;
- B) inflammation of gallbladder with its perforation;
- C) nonspecific inflammation of gallbladder;
- D) purulent inflammation of gallbladder.

2. Risk factors for cholecystitis include:

- A) gallstones;
- B) pregnancy;
- C) cardiovascular events;
- D) all answers are correct.

3. Mark not correct answer about classification of acute cholecystitis:

- A) edematous stage, purulent stage, gangrenous stage;
- B) calculous and non-calculous;
- C) catarrhal, phlegmonous, gangrenous;
- D) not complicated and complicated.

4. Characteristic of pain in acute cholecystitis:

- A) sudden pain in the upper abdomen, has moderate intensity, constant character and not irradiate;
- B) colic pain;
- C) constant pain, may radiate to the right shoulder or scapula;
- D) "knife-like" pain.

5. Describe Murphy's sign:

- A) a delay of breathing during palpation of gallbladder on inhalation because of increasing of pain;
- B) a strengthening of pain at pressure on the area of gallbladder;
- C) a painfulness at the easy pattering on right costal arc by the edge of palm;
- D) an increasing of pain after palpation between the legs of right nodding muscle.

6. Describe Kehr's sign:

- A) a delay of breathing during palpation of gallbladder on inhalation because of increasing of pain;
- B) a strengthening of pain at pressure on the area of gallbladder;
- C) a painfulness at the easy pattering on right costal arc by the edge of palm;

D) an increasing of pain after palpation between the legs of right nodding muscle.

7. Describe Ortner's sign:

A) a delay of breathing during palpation of gallbladder on inhalation because of increasing of pain;

B) a strengthening of pain at pressure on the area of gallbladder;

C) a painfulness at the easy pattering on right costal arc by the edge of palm;

D) an increasing of pain after palpation between the legs of right nodding muscle.

8. Describe Mussy's sign:

A) a delay of breathing during palpation of gallbladder on inhalation because of increasing of pain;

B) a strengthening of pain at pressure on the area of gallbladder;

C) a painfulness at the easy pattering on right costal arc by the edge of palm;

D) an increasing of pain after palpation between the legs of right nodding muscle.

9. Peculiarities of trends of acute cholecystitis in elderly patients:

A) often pain accompanies with vomiting, nausea;

B) no evident clinical picture + rapid destructive changes in gallbladder;

C) often spontaneous regression of gallbladder;

D) all answers are correct.

10. Main method in diagnostic procedure is:

A) X-ray examination;

B) ultrasound;

C) lab. studies;

D) anamnestic and clinical examination.

11. Ultrasonographic findings of acute cholecystitis include:

A) gallstones;

- B) free liquid in abdominal cavity;
- C) gallbladder wall thickening greater than 4 mm;
- D) all answers are correct.

12. Tactic of treatment of acute cholecystitis is:

- A) only conservative;
- B) only surgical;
- C) initial treatment is conservative + surgery for some indications;
- D) individual.

13. Indications for surgical treatment are:

- A) peritonitis;
- B) retention of abdominal pain and muscles resistance;
- C) increasing of body's temperature and leukocytosis;
- D) all answers are correct.

14. Optimal term for operation in patients with inefficacy of conservative treatment is:

- A) 2 hours;
- B) 12 hours;
- C) 24–48 hours;
- D) 72 hours.

15. If acute calculous cholecystitis resolves, optimal tactic is:

- A) following conservative management may be the mainstay of treatment;
- B) laparoscopic cholecystectomy has be done 2 weeks later;
- C) laparoscopic cholecystectomy has be done 4 - 6 weeks later;
- D) open cholecystectomy has be done 4 - 6 weeks later.

16. Indications for open cholecystectomy in acute cholecystitis:

- A) total terminal peritonitis;
- B) surgeon does not know how to perform a laparoscopic cholecystectomy;
- C) complications have developed or the patient has had prior surgery to the area;

D) all answers are correct.

17. In cases of severe inflammation, shock, or if the patient has higher risk of operation the surgeon has to:

- A) perform only conservative treatment;
- B) perform immediately laparoscopic cholecystectomy;
- C) perform immediately open cholecystectomy;
- D) perform percutaneous cholecystostomy tube under ultrasound or CTscan control.

18. The most dangerous complication of laparoscopic cholecystectomy in patient with acute cholecystitis is:

- A) bile duct injury;
- B) cystic duct injury;
- C) liver injury;
- D) all answers are correct.

19. Treatment of the gallbladder mass:

- A) surgical;
- B) conservative;
- C) surgical in young patients, conservative in elderly;
- D) laparoscopy drainage.

20. Treatment of the intraabdominal bleeding from cystic artery after cholecystectomy:

- A) laparotomy and stopping of bleeding;
- B) conservative;
- C) surgical in young patients, conservative in elderly;
- D) laparoscopy drainage.

Standards of answers

1 – C; 2 – D; 3 – A; 4 – C; 5 – A; 6 – B; 7 – C; 8 – D; 9 – B;
10 – B; 11 – C; 12 – C; 13 – D; 14 – C; 15 – C; 16 – D; 17 – D;
18 – A; 19 – B; 20 – A.

Chapter 4 Acute pancreatitis

Tests (one answer is correct)

1. Acute pancreatitis is:

- A) inflammation of pancreas;
- B) inflammation of gallbladder and pancreas;

C) autodigestion of tissues by pancreatic enzymes, and secondary infection and multiorgan system failure may be associated with autodigestion process;

D) purulent inflammation of pancreas.

2. Risk factors for pancreatitis include:

A) gallstones;

B) pregnancy;

C) fatty foods;

D) all answers are correct.

3. Mark not correct answer about classification of acute pancreatitis:

A) edematous pancreatitis and pancreonecrosis;

B) fatty and hemorrhagic pancreonecrosis;

C) catarrhal, phlegmonous, gangrenous;

D) not complicated and complicated.

4. Characteristic of pain in acute pancreatitis:

A) intensive pain in the upper abdomen, has constant character;

B) colic pain;

C) moderate pain in the upper abdomen, may radiate to the right shoulder or scapula;

D) "knife-like" pain.

5. Describe Mayo – Robson's sign:

A) regional tension of anterior abdominal wall in epigastria region, along the projection of pancreas;

B) palpation pain in the left costal-vertebral angle;

C) abdominal distension in upper region;

D) absence of pulsation of abdominal aorta in epigastria region.

6. Describe Goby's sign:

A) regional tension of anterior abdominal wall in epigastria region, along the projection of pancreas;

B) palpation pain in the left costal-vertebral angle;

C) abdominal distension in upper region;

D) absence of pulsation of abdominal aorta in epigastria region.

7. Describe Voskresensky's sign:

A) regional tension of anterior abdominal wall in epigastria region, along the projection of pancreas;

B) palpation pain in the left costal-vertebral angle;

C) abdominal distension in upper region;

D) absence of pulsation of abdominal aorta in epigastria region.

8. Describe Korte's sign:

A) regional tension of anterior abdominal wall in epigastria region, along the projection of pancreas;

B) palpation pain in the left costal-vertebral angle;

C) abdominal distension in upper region;

D) absence of pulsation of abdominal aorta in epigastria region.

9. Describe Cullen's sign:

A) bluish discoloration around the umbilicus;

B) reddish-brown discoloration along the flanks resulting from retroperitoneal blood dissecting along tissue planes;

C) violet spots on the body and face;

D) cyanosis of skin of abdominal wall.

10. Describe Mondor's sign:

A) bluish discoloration around the umbilicus;

B) reddish-brown discoloration along the flanks resulting from retroperitoneal blood dissecting along tissue planes;

C) violet spots on the body and face;

D) cyanosis of skin of abdominal wall.

11. Describe Grey-Turner's sign:

A) bluish discoloration around the umbilicus;

B) reddish-brown discoloration along the flanks resulting from retroperitoneal blood dissecting;

C) violet spots on the body and face;

D) cyanosis of skin of abdominal wall.

12. Describe Holsted's sign:

- A) bluish discoloration around the umbilicus;
- B) reddish-brown discoloration along the flanks resulting from retroperitoneal blood dissecting along tissue planes;
- C) violet spots on the body and face;
- D) cyanosis of skin of abdominal wall.

13. Main method in diagnostic procedure is:

- A) X-ray examination;
- B) ultrasound;
- C) lab. studies;
- D) anamnestic and clinical examination.

14. Specific ultrasonographic findings of acute pancreatitis include:

- A) changing in size and structure of pancreas;
- B) free liquid in abdominal cavity;
- C) gallstones;
- D) all answers are correct.

15. Tactic of treatment of acute pancreatitis is:

- A) only conservative;
- B) only surgical;
- C) initial treatment is conservative + surgery for some indications;
- D) individual.

16. Indications for surgical treatment are:

- A) peritonitis;
- B) retention of abdominal pain and muscles resistance;
- C) increasing of body's temperature and leukocytosis;
- D) formation of mass.

17. Optimal term for operation in patients with gallstones pancreatitis:

- A) 2 hours;
- B) 12 hours;
- C) 24–48 hours;
- D) 72 hours.

18. If acute gallstones pancreatitis resolves, optimal tactic is:

- A) following conservative management may be the mainstay of treatment;
- B) laparoscopic cholecystectomy has be done 2 weeks later;
- C) laparoscopic cholecystectomy has be done 4–6 weeks later;
- D) open cholecystectomy has be done 4–6 weeks later.

19. Indication for peritoneal lavage is:

- A) formation of mass;
- B) retroperitoneal phlegmone;
- C) increasing of body's temperature and leukocytosis;
- D) peritonitis.

20. Treatment of the pancreatic mass:

- A) surgical;
- B) conservative;
- C) surgical in young patients, conservative in elderly;
- D) laparoscopy drainage.

Standards of answers

1 – C; 2 – D; 3 – C; 4 – A; 5 – B; 6 – C; 7 – D; 8 – A; 9 – A;
10 – C; 11 – B; 12 – D; 13 – C; 14 – A; 15 – C; 16 – A;
17 – C; 18 – C; 19 – D; 20 – A.

Chapter 5 Perforated peptic ulcer

Tests (one answer is correct)

1. Risk factors for perforated ulcer include:

- A) H. pylori infection;
- B) hydrochloric acid;
- C) NSAID events;
- D) all answers are correct.

2. Mark not correct answer about classification of perforated ulcer:

- A) perforation of duodenal or gastric ulcer;
- B) covered and atypical perforation;
- C) perforation with peritonitis and perforation without peritonitis;

D) perforation has 3 stages.

3. Describe Ratner's sign:

- A) an irradiation of pain to the right shoulder or scapula;
- B) a strengthening of pain at pressure on the area of stomach;
- C) a painfulness at the easy pattering on right costal arc by the edge of palm;
- D) a persisting moderate tenderness of abdominal wall in right epigastria region.

4. Describe Elicer's sign:

- A) an irradiation of pain to the right shoulder or scapula;
- B) a strengthening of pain at pressure on the area of gallbladder;
- C) a painfulness at the easy pattering on right costal arc by the edge of palm;
- D) persisting moderate tenderness of abdominal wall in right epigastria region.

5. Ratner's sign is a sign of:

- A) stomach perforation;
- B) covered perforation;
- C) stage of illusions;
- D) atypical perforation.

6. Initial method in diagnostic procedure is:

- A) X-ray examination;
- B) ultrasound;
- C) lab. studies;
- D) laparoscopy.

7. Tactic of treatment of perforated ulcer is:

- A) conservative;
- B) surgical;
- C) initial treatment is conservative + surgery for some indications;
- D) surgical in young patients, conservative in elderly patients.

8. Optimal term for operation in patients with perforated ulcer is:

- A) 2 hours;
- B) 12 hours;
- C) 24–48 hours;
- D) 72 hours.

9. Surgical procedures for perforated ulcer are:

- A) simple closure;
- B) ulcer excision;
- C) partial gastrectomy;
- D) all answers are correct.

10. Contraindication for laparoscopic surgery for perforated ulcer is:

- A) total terminal peritonitis;
- B) surgeon does not know how to perform a laparoscopic repair;
- C) fatal cardiovascular events;
- D) all answers are correct.

Standards of answers

- 1 – D; 2 – C; 3 – D; 4 – A; 5 – B; 6 – A; 7 – B; 8 – A; 9 – D;
10 – D.

Chapter 6 Peptic ulcer acute hemorrhage

Tests (one answer is correct)

1. Acute peptic ulcer hemorrhage is:

- A) destruction of vessel in ulcer with flow of blood;
- B) destruction of vessel in ulcer with flow of blood with hematemesis and melena;
- C) destruction of vessel in ulcer with flow of blood with hematemesis, melena and multiorgan system disturbances;
- D) all answers are correct.

2. Risk factors of peptic ulcer hemorrhage include:

- A) H. pylori infection;
- B) hydrochloric acid;
- C) NSAID events;
- D) all answers are correct.

3. Mark not correct answer about classification of peptic ulcer hemorrhage:

- A) bleeding duodenal or gastric ulcer;
- B) there are 3 stages of loss of blood;
- C) Forrest classification correspondences with loss of blood;
- D) Forrest classification correspondences with kind of hemostasis.

4. Characteristic of pain peptic ulcer hemorrhage:

- A) intensive pain in the upper abdomen, has constant character;
- B) pain has stopped after beginning of hemorrhage;
- C) moderate pain in the upper abdomen, may radiate to the right shoulder or scapula;
- D) colic pain.

5. Describe Bergmann's sign:

- A) pain has stopped after beginning of hemorrhage;
- B) palpation pain in the left costal-vertebral angle;
- C) abdominal distension in upper region;
- D) absence of pulsation of abdominal aorta in epigastria region as a result of stomach dilatation.

6. Diagnostic procedure in peptic ulcer hemorrhage includes:

- A) X-ray examination;
- B) ultrasound;
- C) digital examination of rectum;
- D) CT scan.

7. Diagnosis program includes:

- A) anamnesis and physical examination;
- B) digital examination of rectum;
- C) EFGDS;
- D) all answers are correct.

8. Tactic of treatment of peptic ulcer hemorrhage is:

- A) only conservative;
- B) only surgical;

- C) EFGDS hemostasis + conservative treatment + surgery for some indications;
- D) initial treatment is conservative + surgery for some indications.

9. Absolute indications for surgical treatment are:

- A) prolonged bleeding;
- B) recurrent bleeding;
- C) perforated bleeding ulcer;
- D) all answers are correct.

10. Optimal term for operation in patients with absolute indications for surgical treatment:

- A) 2 hours;
- B) 12 hours;
- C) 24–48 hours;
- D) 72 hours.

11. Relative indication for surgical treatment is:

- A) high risk of recurrent bleeding;
- B) massive bleeding;
- C) prolonged bleeding in older patients;
- D) suspicion about stomach cancer.

12. Optimal term for operation in patients with relative indications for surgical treatment:

- A) 2 hours;
- B) 12 hours;
- C) 24–48 hours;
- D) 72 hours.

13. Indication for angiographic embolization is:

- A) bleeding ulcer of stomach;
- B) high risk for surgical intervention;
- C) recurrent bleeding;
- D) bleeding recurrent ulcer after open surgery.

14. Contraindication for emergency EFGDS is:

- A) Zenker diverticulum;
- B) upper abdominal open surgery;

- C) severe cardiac and lung decompensation;
- D) all answers are correct.

15. Tactic of treatment of perforated bleeding ulcer is:

- A) conservative;
- B) surgical;
- C) initial treatment is conservative + surgery for some indications;
- D) surgical in young patients, conservative in elderly patients.

16. Optimal term for operation in patients with perforated bleeding ulcer is:

- A) 2 hours;
- B) 12 hours;
- C) 24–48 hours;
- D) 72 hours.

17. Surgical procedure for perforated bleeding ulcer is:

- A) simple closure;
- B) laparoscopy repair;
- C) ulcer excision;
- D) all answers are correct.

18. Surgical procedure for bleeding duodenal ulcer is:

- A) simple closure;
- B) ulcer excision + pyloroduodenoplasty;
- C) partial gastrectomy;
- D) all answers are correct.

19. Surgical procedure for bleeding gastric ulcer is:

- A) simple closure;
- B) ulcer excision + truncal vagotomy;
- C) partial gastrectomy;
- D) all answers are correct.

20. Surgical procedure for bleeding gastric ulcer in elderly patients is:

- A) simple closure;
- B) ulcer excision;
- C) partial gastrectomy;

D) all answers are correct.

Standards of answers

1 – C; 2 – D; 3 – C; 4 – B; 5 – A; 6 – C; 7 – D; 8 – C; 9 – D;
10 – A; 11 – A; 12 – C; 13 – B; 14 – C; 15 – B; 16 – A;
17 – C; 18 – B; 19 – C; 20 – B.

Chapter 7 Bowel obstruction

Tests (one answer is correct)

1. Small bowel blood supply is (arteries):

- A) a. mesenteric superior;
- B) a. mesenteric inferior;
- C) truncus celiacus;
- D) a. gastric dextra.

2. Large bowel blood supply is (arteries):

- A) a. mesenteric superior;
- B) a. mesenteric inferior;
- C) Riolany ark;
- D) all answers are correct.

3. Venous bowel outflow is:

- A) v. cava superior;
- B) v. cava inferior;
- C) portal vein;
- D) all answers are correct.

4. Mark not correct answer about clinical classification of bowel obstruction:

- A) acute;
- B) subacute;
- C) chronic;
- D) mechanical.

5. Classification of ileus:

- A) after abdominal injury;
- B) paralytic or spastic;
- C) after central nerve system injury;
- D) all answers are correct.

6. Classification of large bowel obstruction:

- A) obstructive;
- B) strangulated;
- C) acute and chronic;
- D) all answers are correct.

7. Main sign of bowel obstruction:

- A) constant pain;
- B) cramp-like pain;
- C) hyperthermia;
- D) weakness.

8. Main sign of small bowel obstruction:

- A) hyperthermia;
- B) vomiting;
- C) absence of gas and stool;
- D) weakness.

9. Main sign of large bowel obstruction:

- A) hyperthermia;
- B) vomiting;
- C) absence of gas and stool;
- D) weakness.

10. Intensive pain is observed in patients with:

- A) obstructive bowel obstruction;
- B) strangulated bowel obstruction;
- C) acute bowel obstruction;
- D) paralytic bowel obstruction.

11. Paralytic bowel obstruction usually occurs after:

- A) abdominal injury;
- B) laparoscopy;
- C) laparotomy;
- D) all answers are correct.

12. Obstructive bowel obstruction may be caused by:

- A) tumors;
- B) gallstones;
- C) coproliths;

D) all answers are correct.

13. In elderly patients the most frequent cause of obstructive bowel obstruction is:

A) tumors;

B) gallstones;

C) coproliths;

D) all answers are correct.

14. The most frequent localisation of tumors which are complicated with obstructive bowel obstruction is:

A) cecum;

B) ascendant colon;

C) transversal colon;

D) descendant and sigmoid colon.

15. In diagnostic program for bowel obstruction we use:

A) plain radiography;

B) enteroclysis;

C) ultrasonography;

D) all answers are correct.

16. Tactic of treatment of bowel obstruction is:

A) conservative;

B) surgical;

C) initial treatment is conservative + surgery for some indications;

D) surgical in young patients, conservative in elderly patients.

17. The main sign of nonviability of bowel:

A) changing of color;

B) absents of pulsation of mesenteric arteries;

C) decreasing of motor activity;

D) all answers are correct.

18. The limits of resection of small bowel are:

A) nonviability part + 40 cm before + 20 cm late;

B) nonviability part + 20 cm before + 10 cm late;

C) only nonviability part;

D) surgeon has to detect individually.

19. Optimal surgical procedure in patients with tumor of ascendant colon and bowel obstruction:

- A) tumor + 40 cm before + 20 cm late;
- B) right hemicolectomy + enterostomy;
- C) right hemicolectomy + ileotransversostomy;
- D) surgeon has to detect individually.

20. Optimal surgical procedure in patients with tumor of descendant colon and bowel obstruction:

- A) tumor + 40 cm before + 20 cm late;
- B) left hemicolectomy + colostomy;
- C) left hemicolectomy + transversosigmoidostomy;
- D) surgeon has to detect individually.

Standards of answers

1 – A; 2 – D; 3 – C; 4 – D; 5 – B; 6 – D; 7 – B; 8 – B; 9 – C;
10 – B; 11 – C; 12 – D; 13 – A; 14 – D; 15 – D; 16 – C;
17 – B; 18 – A; 19 – C; 20 – B.

PROBLEMS

Young man, 22 years old, has moderate pain in suprapubic region, dysuria. He is suffered from abdominal pain 2 days. Pain started as diffuse lower part abdominal pain end after 12 hours localized in suprapubic region. Temperature of body is 37,8 °C. Signs of peritonitis are negative.

1. What is provisional diagnosis:
 - A) acute cystitis;
 - B) acute prostatitis;
 - C) acute appendicitis;
 - D) renal colic?
2. What lab. study is the most informative:
 - A) white blood cell count;
 - B) urinalysis;

- C) CRP;
- D) all studies are not specific?
- 3. What investigation is the most informative:
 - A) excretory urography;
 - B) CT scan of abdominal cavity;
 - C) abdominal plain film;
 - D) cystoscopy?
- 4. Tactic of treatment:
 - A) emergency surgery;
 - B) analgetic + antibiotics;
 - C) analgetic + uroseptics;
 - D) surgery if conservative treatment is not successful.
- 5. The cause of misdiagnosis is:
 - A) patient's age;
 - B) patient's sex;
 - C) late hospitalization;
 - D) unusual pelvic position of appendix.

Standards of answers

1 – C; 2 – D; 3 – B; 4 – A; 5 – D.

Woman, 62 years old, has pain in right subcostal region, vomiting with bile. Pain started as a colic pain after fatty food intake, after 2 hours became constant. Temperature of body is 37,4 °C. During palpation painfulness and muscle resistance are observed in right subcostal region. Blumberg sign is negative.

- 1. What is provisional diagnosis:
 - A) acute cholecystitis;
 - B) acute gastritis;
 - C) acute appendicitis;
 - D) renal colic?
- 2. What lab. study is the most informative:
 - A) white blood cell count;
 - B) urinalysis;

- C) CRP;
 - D) all studies are not specific?
3. What investigation is the most informative:
- A) excretory urography;
 - B) ultrasound scan of abdominal cavity;
 - C) abdominal plain film;
 - D) chest film?
4. Tactic of treatment:
- A) emergency surgery;
 - B) conservative treatment;
 - C) gallstones – emergency surgery;
 - D) surgery if conservative treatment is not successful.
5. If conservative treatment of acute calculous cholecystitis is successful:
- A) surgery is indicated after next pain attack;
 - B) surgery is not indicated;
 - C) surgery is performed after 1 month;
 - D) surgery is performed after 6 months.

Standards of answers

1 – A; 2 – D; 3 – B; 4 – D; 5 – C.

Woman, 59 years old, has intensive upper abdominal pain, vomiting. Pain started after fatty meat intake 12 hours ago. Scleras are icteric. During palpation painfulness and muscle resistance are observed in upper abdomen. Blumberg sign is negative. Last 2 years patient noted periodical colic pain in wright subcostal region.

1. What is provisional diagnosis:
- A) acute cholecystitis;
 - B) acute hepatitis;
 - C) acute pancreatitis;
 - D) food toxicoinfection?
2. What lab. study is the most informative:
- A) white blood cell count;

- B) level of serum amylase;
 - C) CRP;
 - D) all studies are not specific?
3. What investigation is the most indicated:
- A) endoscopic retrograde cholangiopancreatography;
 - B) ultrasound scan of abdominal cavity;
 - C) abdominal plain film;
 - D) CT scan of abdominal cavity?
4. The most probable etiological factor:
- A) peptic ulcer;
 - B) hepatitis;
 - C) gallstones;
 - D) toxic factor.
5. Tactic of treatment:
- A) emergency surgery;
 - B) conservative treatment;
 - C) surgery if conservative treatment is not successful after 12–24 hours;
 - D) surgery if conservative treatment is not successful after 24–48 hours.

Standards of answers

1 – C; 2 – B; 3 – B; 4 – C; 5 – D.

Young man, 28 years old, has intensive diffuse abdominal pain. Pain started 2 hours ago after physical training. Temperature of body is 36,9 °C. Last year patient noted periodical pain in stomach. Tenderness of abdominal wall, Blumberg sign are present.

1. What is provisional diagnosis:
- A) acute cholecystitis;
 - B) acute pancreatitis;
 - C) perforated ulcer;
 - D) acute gastritis?

2. What lab. study is the most informative for differential diagnostics:

- A) white blood cell count;
- B) CRP;
- C) level of serum amylase;
- D) all studies are not informative?

3. What investigation is the most informative:

- A) gastroscopy;
- B) ultrasound scan of abdominal cavity;
- C) abdominal plain film;
- D) laparoscopy?

4. Tactic of treatment:

- A) emergency surgery;
- B) conservative treatment (Taylor method);
- C) surgery if conservative treatment is not successful after 12 hours;
- D) surgery if conservative treatment is not successful after 24 hours.

5. The best method for perforated duodenal ulcer is:

- A) ulcer excision;
- B) ulcer excision + vagotomy;
- C) ulcer excision + adequate antiulcer treatment;
- D) partial gastrectomy.

Standards of answers

1 – C; 2 – C; 3 – D; 4 – A; 5 – C.

Young man, 34 years old, has coffee ground vomiting and melena during 20 hours. Pulse rate is 100 per min., decreasing of arterial blood pressure is 90/60 mm Hg.

1. What is provisional loss of blood:

- A) “microbleeding”;
- B) I–II stages;
- C) III stage;
- D) profuse bleeding?

2. What investigation is the most informative:
- A) gastroscopy;
 - B) ultrasound scan of abdominal cavity;
 - C) abdominal plain film;
 - D) laparoscopy?
3. Tactic of treatment:
- A) emergency surgery;
 - B) medicamentous hemostasis;
 - C) endoscopic hemostasis;
 - D) endoscopic hemostasis + medicamentous hemostasis.
4. The best method for hemostasis control:
- A) puls rate;
 - B) blood pressure;
 - C) clinical observation;
 - D) endoscopic observation.
5. The best procedure for bleeding duodenal ulcer is:
- A) ulcer excision;
 - B) ulcer excision + vagotomy;
 - C) ulcer excision + adequate antiulcer treatment;
 - D) partial gastrectomy.

Standards of answers

1 – B; 2 – A; 3 – D; 4 – D; 5 – C.

Woman, 70 years old, has moderate crampy abdominal pain, constipation, abdominal distension during 4 days. 2 hours became constant. Temperature of body is 37,4 °C. During palpation diffuse painfulness without muscle resistance is observed. Blumberg sign is negative. Digital rectal examination – empty rectum.

1. What is provisional diagnosis:
- A) acute colitis;
 - B) small bowel obstruction;
 - C) large bowel obstruction;
 - D) coprosthesis?

2. What lab. study is the most informative for differential diagnostics:

- A) white blood cell count;
- B) CRP;
- C) hematocrit level;
- D) all studies are not informative?

3. What investigation is the most informative for differential diagnostics:

- A) CT scan of abdominal cavity;
- B) ultrasound scan of abdominal cavity;
- C) abdominal plain film;
- D) laparoscopy?

4. Tactic of treatment:

- A) emergency surgery;
- B) conservative treatment;
- C) surgery if conservative treatment is not successful;
- D) colonoscopic procedures.

5. The best method for control of conservative treatment efficiency:

- A) puls rate;
- B) X-ray observation;
- C) clinical observation;
- D) clinical + X-ray observation.

Standards of answers

1 – C; 2 – D; 3 – C; 4 – C; 5 – D.

Young man, 25 years old, has diffuse abdominal pain, vomiting. He is suffered from abdominal pain 4 days. Pain started as diffuse lower part abdominal pain, after some hours localized in right lower quadrant, became general again on the 3 day. Temperature of body is 38,5 °C. Tenderness of abdominal wall, Blumberg sign are present.

1. What is the most probable cause of peritonitis:

- A) acute cholecystitis;

- B) acute pancreatitis;
 - C) acute appendicitis;
 - D) perforated ulcer?
2. What lab. study is the most impotent for patient:
- A) CBC with differential, serum electrolytes with renal function;
 - B) urinalysis;
 - C) CRP;
 - D) liver tests?
3. What investigation is the most informative:
- A) ultrasound scan of abdominal cavity;
 - B) CT scan of abdominal cavity;
 - C) abdominal plain film;
 - D) which is necessary for preoperative preparation?
4. Tactic of treatment:
- A) emergency surgery;
 - B) conservative treatment (Taylor method);
 - C) surgery if conservative treatment is not successful;
 - D) emergency surgery after preoperative preparation.
5. Optimal surgical incision for patient:
- A) McBurney incision;
 - B) middle line laparotomy;
 - C) right side pararectal laparotomy;
 - D) right side transrectal laparotomy.

Standards of answers

1 – C; 2 – A; 3 – D; 4 – D; 5 – B.

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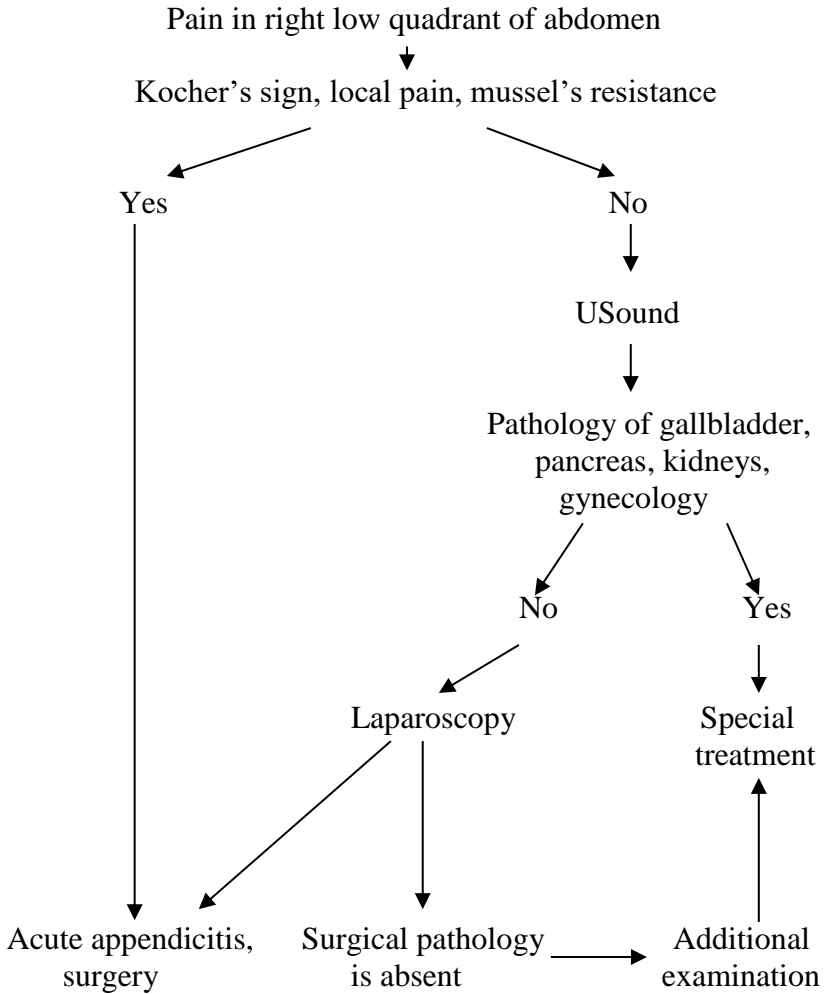
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THE LIST OF LINKS

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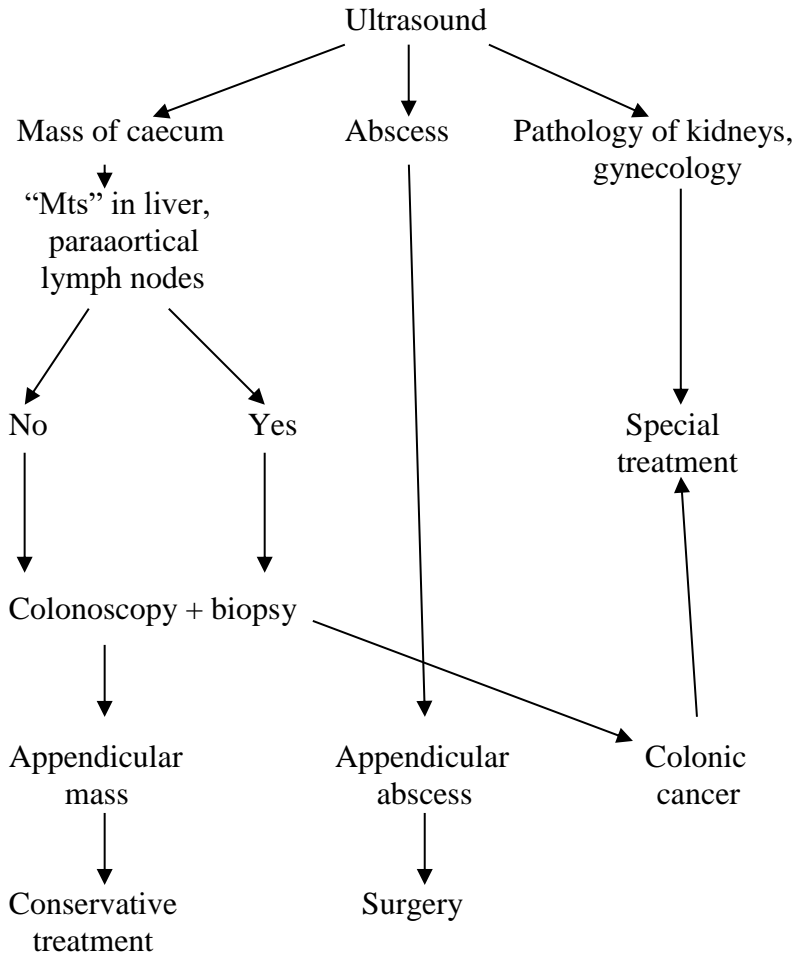
ADDITION A
Algorithm of diagnostic of acute appendicitis
(obligatory)



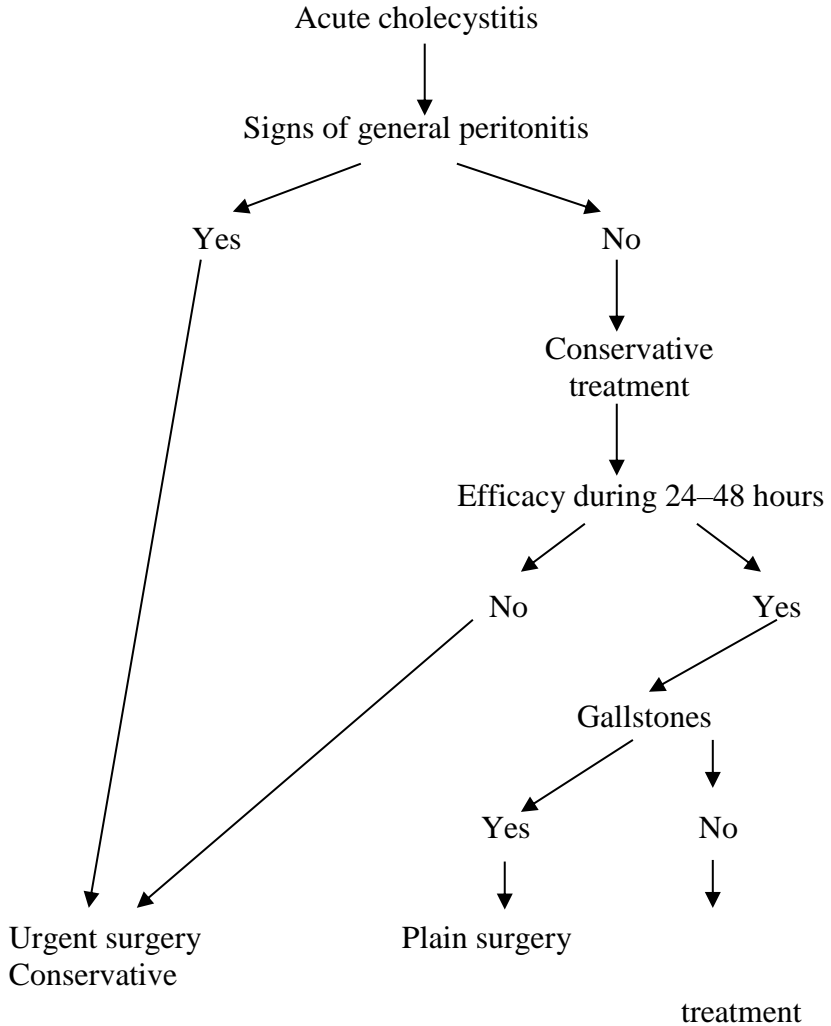
ADDITION B

Algorithm of diagnostic and treatment of appendicular mass and abscess (obligatory)

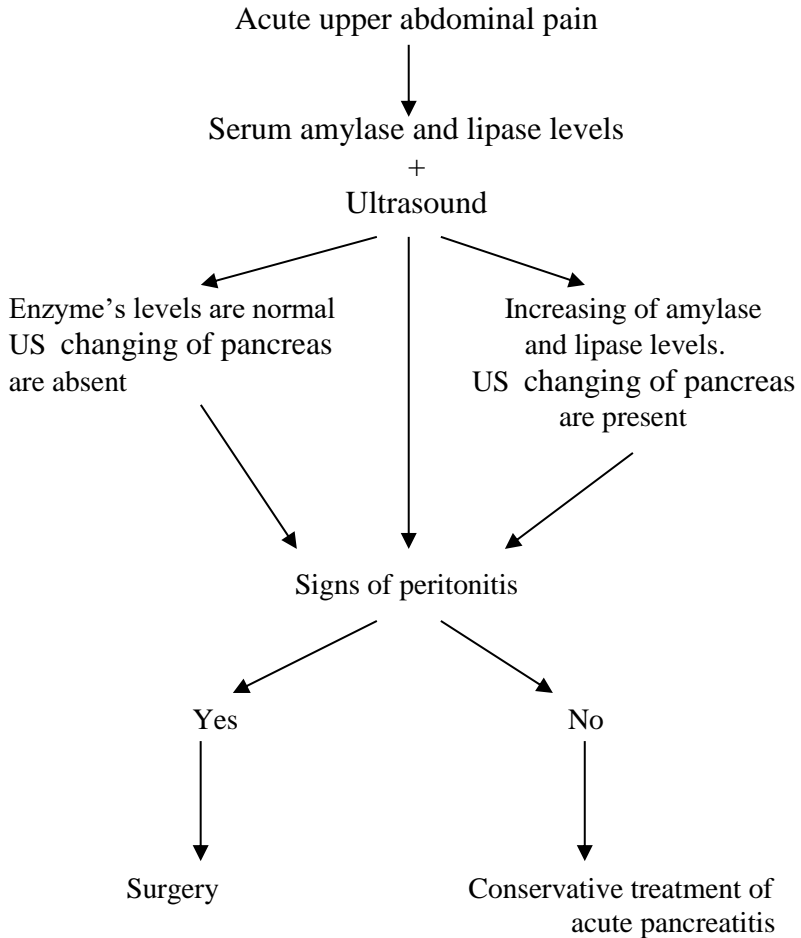
Mass in right low quadrant of abdomen



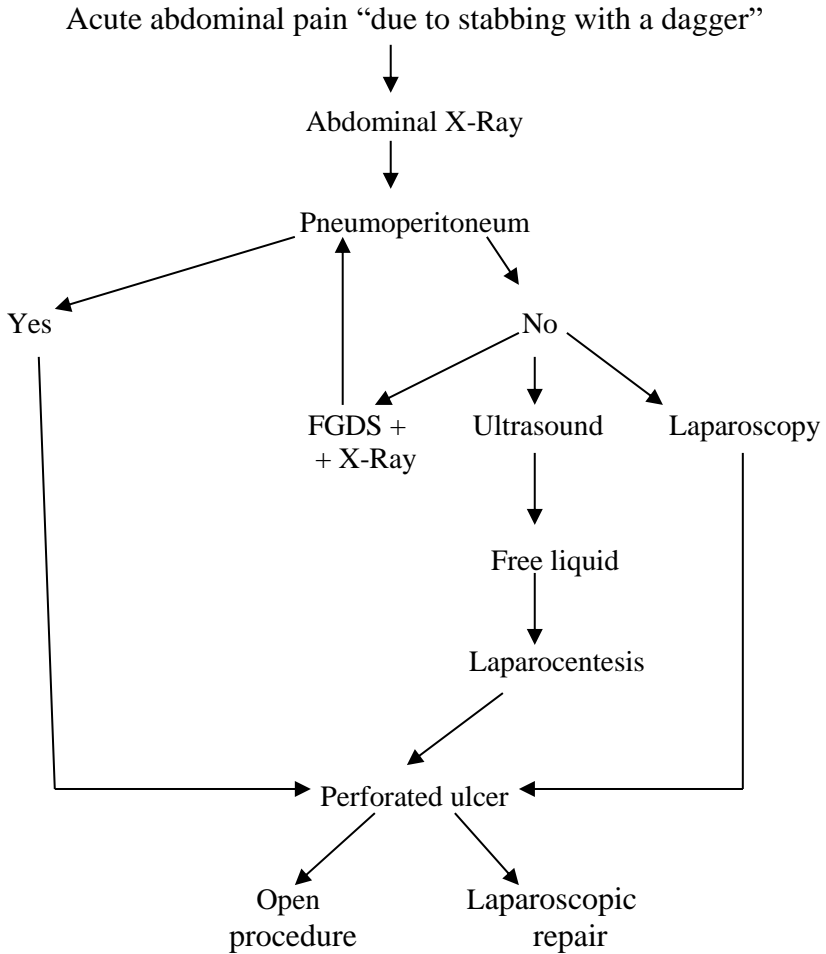
ADDITION C
Algorithm of treatment of acute cholecystitis
(obligatory)



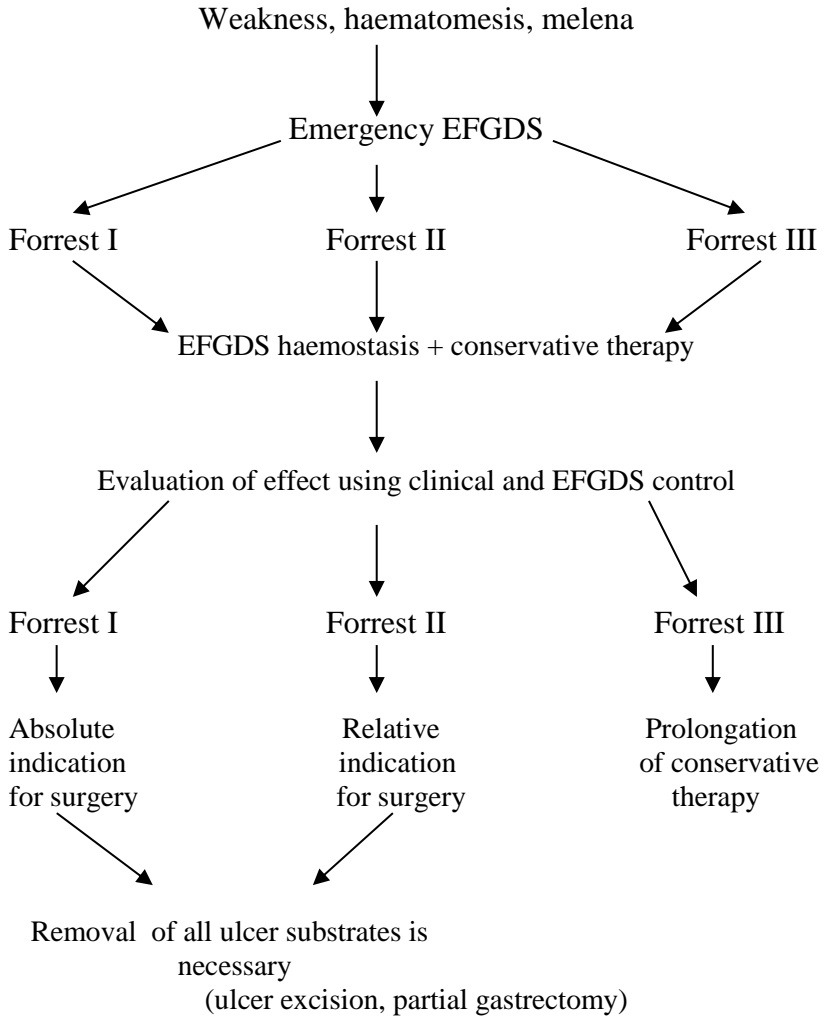
ADDITION D
Algorithm of diagnosis and treatment of acute pancreatitis
(obligatory)



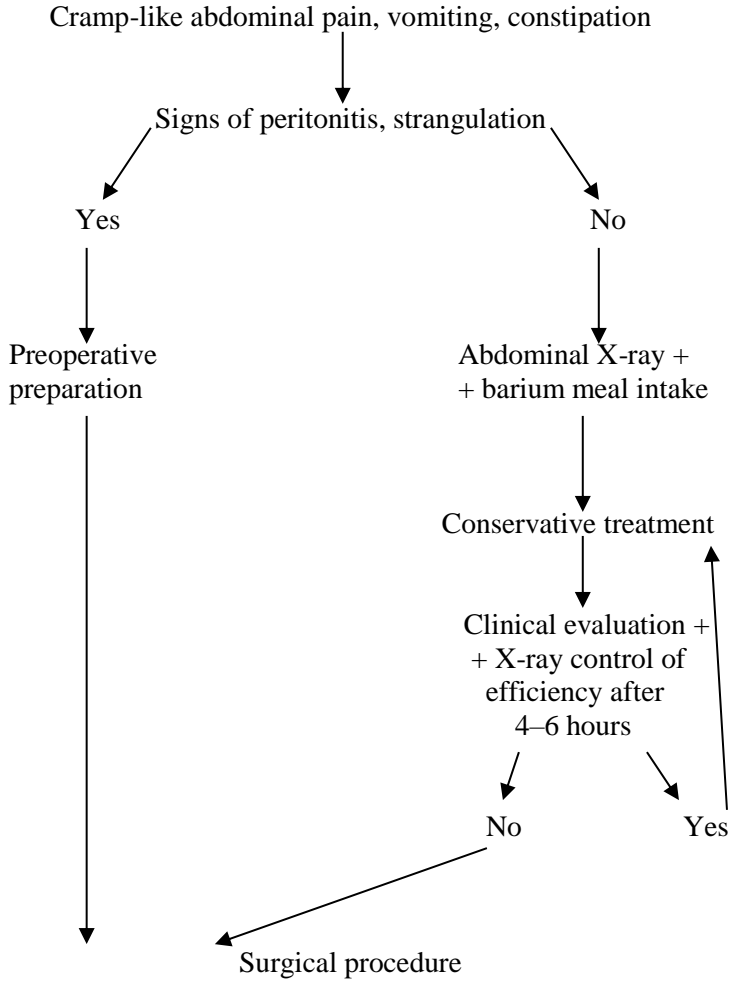
ADDITION E
Algorithm of diagnosis and treatment of perforated ulcer
(obligatory)



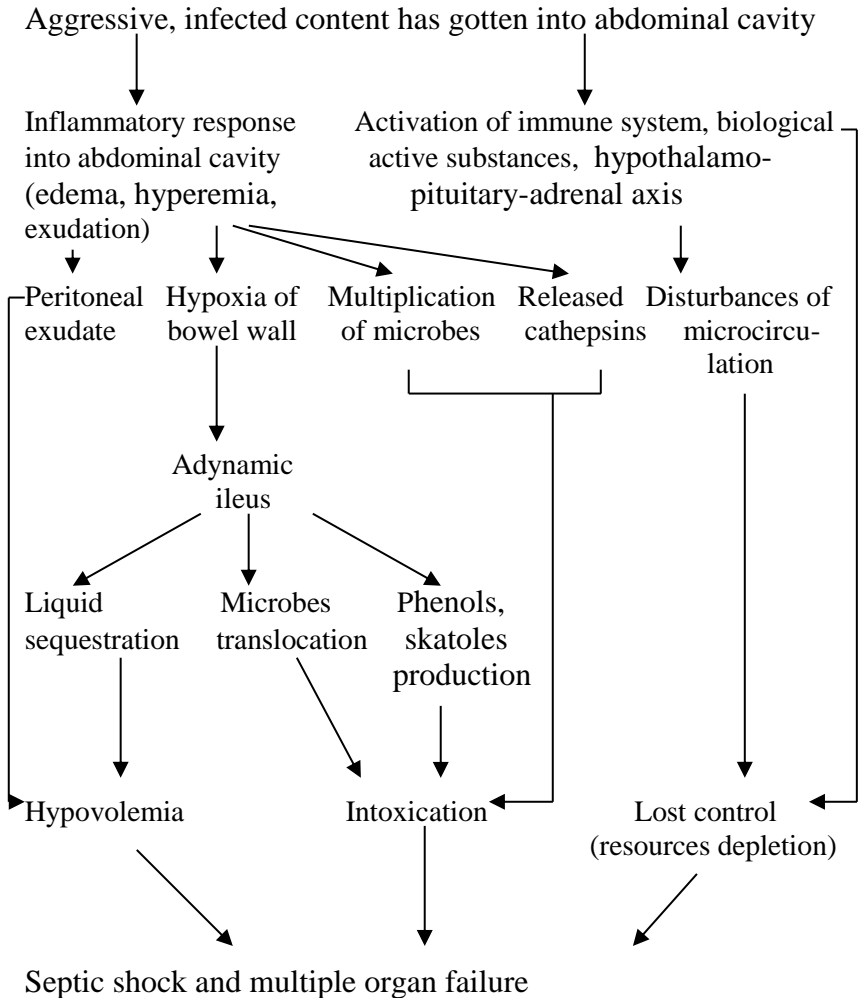
ADDITION F
Algorithm of diagnosis and treatment of bleeding ulcer
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ADDITION G
Algorithm of diagnosis and treatment of bowel obstruction
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ADDITION H
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Електронне навчальне видання

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П'ятикоп Геннадій Іванович**

НЕВІДКЛАДНА АБДОМІНАЛЬНА ХІРУРГІЯ

**Навчальний посібник
(Англійською мовою)**

За загальною редакцією кандидата медичних наук, доцента О. Л. Ситніка

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Навчальний посібник містить інформацію про основні принципи діагностики та лікування хворих на гострі хірургічні захворювання органів черевної порожнини.
Для англійськомовних студентів закладів вищої освіти III–IV рівнів акредитації, лікарів-інтернів, хірургів та сімейних лікарів.