



Lecture complex

Discipline: Fundamentals of surgical diseases

Discipline code: HAN4308

Name of the OP: 6 In 101 " Medicine"

The amount of study hours / credits: 150 hours / 5 credits

Academic course and semester: 4th year and 8th semester

Lecture length: 15 hours



The lecture complex was developed in accordance with the working curriculum of the discipline "Fundamentals of Surgical diseases" (syllabus) and discussed at a meeting of the department.

Protocol № 11 " 25 " 06 2025G.

Head of the Department: Candidate of Medical Sciences, Acting Associate Professor G. E.

Kaldygozova (signed)_



Lecture No. 1

1. Topic:

1.1. Acute surgical pathology of the abdominal cavity. Acute appendicitis. Etiology, pathogenesis, classification. Clinic, diagnosis. Differential diagnosis. Treatment.

2. Purpose:

1. The student should know:

1.1. Anatomy and physiology;

1.2. Etiology and pathogenesis;

1.3. Pathological anatomy;

1.4. Classification;

1.5. clinical symptoms;

1.6. clinical course;

1.7. clinical course of complicated forms;

1.10. Diagnostics;

1.11. differential diagnosis;

1.12. principles of treatment;

1.13. chronic forms.

3. Lecture abstracts:

* Acute appendicitis – rupture of the appendix (Buyen) - is the most common among the abdominal organs. Acute appendicitis is an acute surgical disease with polyethological disease.

* Frequency from 40 to 10,000 people

* In the 16th century, the scientist Pars described the appendix for the first time and frostbitten the opening of an abscess in the right iliac region.

• In 1884, R. Fitz coined the term "appendicitis."

* First operations:

* Krenlein (a German surgeon) removed an inflamed appendix in 1884.

Trojans removed the vermiform appendage in 1890

Etiology and pathogenesis of acute appendicitis:

* Reflex (neuro-humoral)

* Hematogenous thrombosis of appendicitis

* Lymphogenic

* Immunological

* Alimentary

* Parasitic

* Bauginospasm

* Foreign objects (button, shemysh)

Classifications of acute appendicitis

Classification according to Kolesov;

1. Appendicular tingling

2. acute simple superficial (catarrhal) appendicitis

3. destructive acute appendicitis

(a) phlegmonous

b) gangrenous

c) perforating

Complications of acute appendicitis

- a) appendicular infiltrate
- b) appendicular abscess
- c) pylephlebitis
- d) peritonitis

Classification according to Abrikosov (morphol.) 1946

I. superficial and catarrhal (6-12 hours developing)

II. Phlegmonous (12-24 hours)

A) simple

B) phlegmonous and ulcerative

C) appostemous-filled with purulent walls.

D) empyema (the cavity is filled with pus)

E) phlegmonous-perforated

III. Gangrenous (24-48 hours)

1st-gangrenous-a) perforated, b) without perforation

Gangrenous 2-a) perforated, b) without perforation

Bladed syndromes of acute appendicitis:

* 1. Dyspeptic

* nausea, vomiting 1-2 times, stool disorders

* 2. pain

* begins in the epigastric region and passes after 5-6 hours to the right iliac region-not firm, worth enduring, rarely periodic, increases with movement

* 3. inflammatory syndrome

* temperature (37.2-37.5) leukocytosis, neutrophilosis, left shift, increased ESR

* 4. Peritoneal syndrome

* intoxication, Hippocratic surface, tongue covered with plaque, abdomen swollen, does not participate in the respiratory act (Winter's syndrome), pain in the projection of the appendix, worsening peristalsis, + symptoms of Shchetkin-Blumberg, Mendel, Krymov muscle rigidity in the right area

Symptoms.

* Kocher 4/1 20% -migration of pain in the epigastric region to the right iliac region

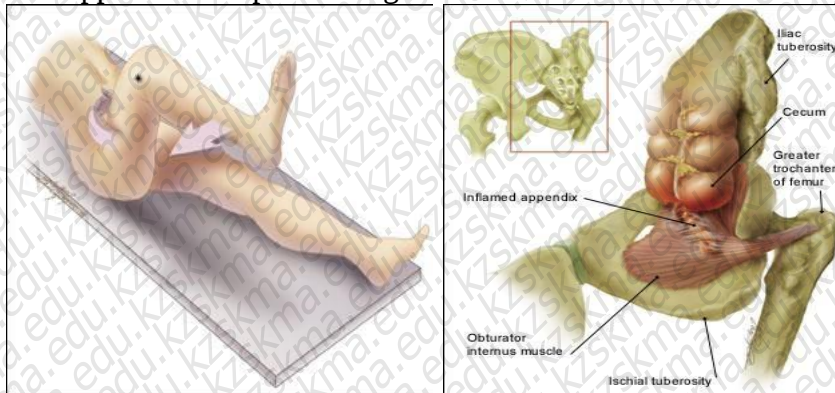
* Sitkovsky 80% - pain when the patient lies on his left side.

* Rovzing 67% - causes pain in the right abdominal cavity when you simultaneously compress the colon and sigmoid colon, which descend from top to bottom. This means that if there is an inflammatory process, the gas in the sigmoid colon causes pain by stretching the appendix.

* Voskresensky 78% (shirt symptom) the presence of the boundaries of cutaneous hyperesthesia under the right abdomen when the patient slides from the epigastric region to the abdominal cavity. Its appearance is associated with the return of blood from the superior iliac vein.

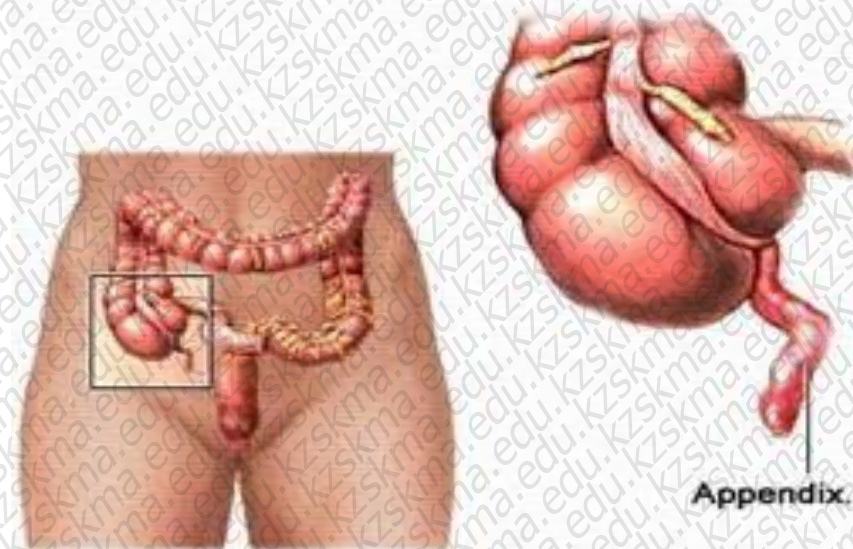
Razdolsky 50% - hyperesthesia of the skin in the right iliac region, A slap in the face with fingers

* Samples 60% - pain in the right iliac region increases when you lift your right leg without bending it
Copa-an increase or appearance of pain during the rotational movement of the hip joint with the lifting



of the right leg.

4. Иллюстрационный материал:(презентация ұсынылады)



5. Literature

Basic literature

- Nurmakov A. J. Surgical diseases: textbook / A. J. Nurmakov, D. A. Nurmakov; KazNMU named after S. D. Asfendiyarov. Alma Ata: New book, 2021. 496 p.
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Nurakhmanov, B. D. Clinical surgery: textbook. - ; Rec. to print the Center. Method. Council of KazNMU named after S. D. Asfendiyarov. Almaty: Evero Publ., 2012. 525c.

Lagoon, M. A. The course of faculty (Analitical) surgery in pictures, tables and schemes : the manual / M. A. Lagoon, B. S. Kharitonov. - ; Ministry of Education and science of the Russian Federation. Recommended by I. M. Sechenov First Moscow State Medical University. - M. : GEOTAR-Media, 2017. - 436 p.

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Zhumabekov, A. T. emergency first aid: an educational and methodical manual. Almaty: TechSmith, 2023, p. 112.

Seksenbaev, D. S. acute surgical diseases of the abdominal cavity : an educational and methodical manual / D. S. Seksenbaev. Shymkent: SKGFA, 2017. 263 P.

Kuznetsov, N. A. Surgical patient care: textbook / N. A. Kuznetsov, A. T. Brontwein ; Ministry of Education and Science of the Russian Federation. - ; ". - M. : GEOTAR-Media, 2013. - 288 p. : ill.

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Surgical pain. A guide to practical exercises [Electronic resource]: textbook. the manual / edited by A. F. Chernousov. - The electron. text data. (41.6 Mb). Moscow : GEOTAR-Media, 2012. 496 p.

Surgical diseases [Electronic resource]: textbook / edited by A. F. Chernousov. - The electron. text data. (69.1 Mb). Moscow : GEOTAR-Media, 2012. 664 p.

Some issues of general surgery.

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volumes. Volume 1. / Ed. headed by M. I. Kuzin; The textbook has been translated into Kazakh under the general editorship of A. N. Baimakhanova. Moscow: GEOTAR-Media, 2018. 560 p.

<http://rmebrk.kz / 1174592> Nurakhmanov B. Zh., Ibadildin A. S., Amantayeva K. K., Surgery-aaurular diseases, textbook. Almaty, VS "Evero", 2020. - p. 701

https://www.elib.kz/ru/search/read_book/669 / Madikenovich of life. Surgical diseases. - educational lectures.

- Almaty: Evero-2020. - 309 P. https://www.elib.kz/ru/search/read_book/559/A J. Nurmakov, D. A. Nurmakov Surgical diseases. Almaty: "Evero", 2020. - 472 pages

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- 2020. - 432 p. https://www.elib.kz/ru/search/read_book/828 Kaldybaev M. A. General surgery – textbook (volume II). -2020. - 448 p. https://www.elib.kz/ru/search/read_book/829 Kaldybaev M. A.

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Vasiliev V. K. and others. General surgery: A textbook. / V. K. Vasiliev, A. P. Popov, A.D.

Tsybikzhapov. - St. Petersburg: Lan, 2014. - 272c. <http://rmebrk.kz/book/1178354> general surgery.

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Rychagov, P. V. Garelik. Minsk: Higher School, 2008. 543 P. - URL:

<https://www.iprbookshop.ru/20100.html> general surgery. Volume 2 : textbook / G. P. Rychagov, P. V.

Garelik, A. N. Nekhaev [et al.]; edited by G. P. Rychagov, P. V. Garelik. Minsk: Higher School,

2006. 492 P. URL: <https://www.iprbookshop.ru/20101.html> Kolb, L. I. General surgery : a textbook / L. I. Kolb, S. I. Leonovich, I. V. Yarovich. — Minsk : Higher School, 2006. - 444 p. - ISBN 978-985-

06-1378-3. - Text: electronic // Digital educational resource IPR SMART: [website]. - URL:

<https://www.iprbookshop.ru/20102.html> Fundamentals of surgical patient care = Nursing care in

surgery: a textbook / A. I. Badretdinova, A. S. Klimenko, N. G. Kostsova [et al.]. - Moscow: Peoples' Friendship University of Russia, 2019. - 148 P. - URL: <https://www.iprbookshop.ru/104237.html>

6. Security Questions (Feedback Feedback)

Questions

1. Specify the definition of acute appendicitis, an acute nonspecific process of inflammation of the appendix.
2. specify the pathoanatomic forms of acute appendicitis –catarrhal, phlegmonous, gangrenous
3. Specify the location of the appendix-retrocecal, retroperitoneal, medial, pelvic, subhepental, left
4. Which artery is the blood-sucking appendix - A. arrendicis vermiformis, an effective iliac-transverse incision for smartphones.
5. Name the complications of acute appendicitis-appendicular infiltrate, pyelophlebitis, peritonitis, sepsis
6. What diseases should be distinguished from acute appendicitis? - peptic ulcer of the stomach and duodenum, acute cholecystitis, acute pancreatitis, urolithiasis, ectopic pregnancy
7. What are the main symptoms of acute appendicitis-pain in the right iliac region, muscle tension in the right iliac region, peritoneal irritation
8. Name additional symptoms of acute appendicitis. Kocher, Sitkovsky, Rovzing, Voskresensky, Bortalomye-Mikhelson, and others
9. What additional research methods are needed to identify complications of acute appendicitis?
10. What is the treatment strategy for acute appendicitis?

Lecture No. 2.

1. Topic:

1.1. cholelithiasis. Cholecystitis. Etiology, pathogenesis, classification. Clinic, diagnosis. Differential diagnosis. Treatment.

2. Purpose:

1. The student should know: (Cholecystitis)

- 1.1. Topographic anatomy and physiology of the gallbladder and biliary tract
- 1.2. methods of examination of the gallbladder and biliary tract
- 1.3. etiology and pathogenesis of diseases of the gallbladder and biliary tract
- 1.4. clinical signs of acute and chronic cholecystitis, cholelithiasis
- 1.5. complications of gallbladder and biliary tract diseases
- 1.6. diagnosis of gallbladder and biliary tract diseases
- 1.7. differential diagnosis of gallbladder and biliary tract diseases
- 1.8. principles of conservative treatment of diseases of the gallbladder and biliary tract
- 1.9. indications for surgical treatment of cholecystitis and cholelithiasis
- 1.10. acute methods of treatment of diseases of the gallbladder and biliary tract

3. Lecture abstracts:

* Acute cholecystitis is an inflammatory disease that leads to damage to the gallbladder wall, the formation of gallstones, and motor and tonal disorders of the gallbladder system.

Acute cholecystitis is one of the most common surgical diseases, second in frequency only to appendicitis.

Classification of cholecystitis:

1. acute calculous cholecystitis and acute stone-free cholecystitis;

* - acute catarrhal cholecystitis;

* - phlegmonous cholecystitis;

* - gangrenous cholecystitis.

2. chronic cholecystitis:

* - chronic stone-free cholecystitis;

* - chronic calculous cholecystitis.

Catarrhal cholecystitis-in the area of the right hypochondrium and epigastrium

, the presence of pain is transmitted to the lumbar region, under the right shoulder, and in the neck.

Easily accessible vomiting, frequent urge. Subfebrile body temperature,

Tachycardia is up to 100 seconds per minute. On palpation, there is severe pain under the right rib. in

the projection of a special gallbladder Ortner-Grekov, Murphy, Mussy-Georgievsky

The symptoms are positive, sometimes we may notice an enlarged gallbladder. In the study of CVD,

we observe a small leukocytosis of $10-14 \times 10^9 / l$, an increase in ESR is observed

Phlegmonous cholecystitis is a very pronounced clinical picture

, we can observe a painful cough when we rest, when we move. High fever, tachycardia 110-120 s

min. The Shchetkin-Blumberg symptom is positive in the upper right part of the abdomen, a small

leukocytosis of $20-22 \times 10^9 / l$ is observed in the study of CVD. an increase in ESR is observed

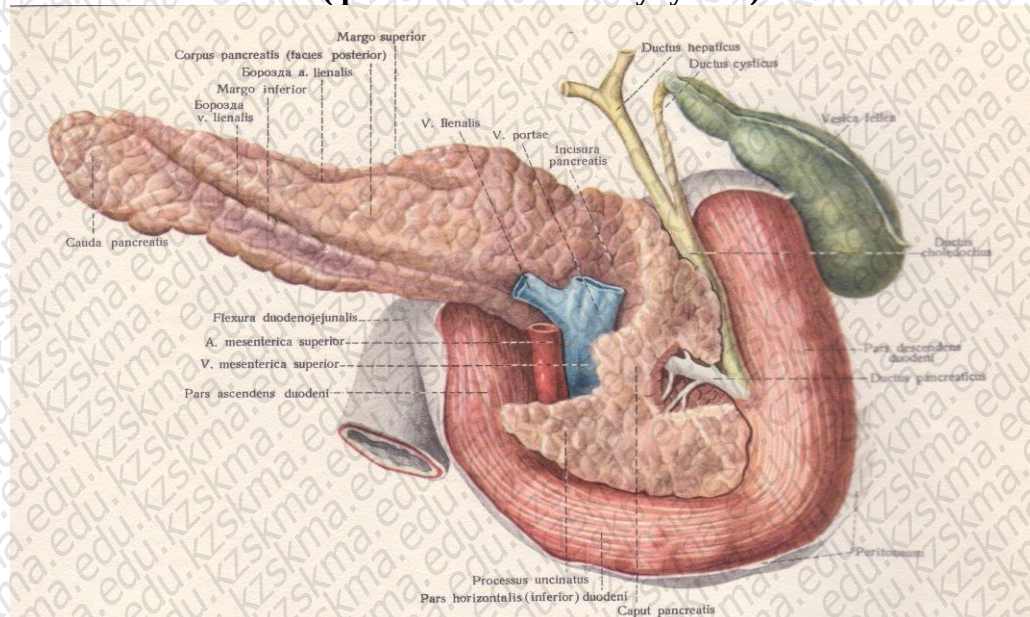
Macroscopic: the gallbladder is enlarged, the walls are thickened, inside there is a purulent exudate of

pale blue color.

Gangrenous cholecystitis is a complication of the phlegmonous form. We observe pronounced intoxication, the manifestation of local or disseminated purulent peritonitis (with perforation of the gallbladder)

The patient's condition is serious

4. Illustrative material:(presentation of the usynylade)



5. Literature

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6. Security Questions (Feedback Feedback) Questions

1. cholelithiasis. Etiology. Pathogenesis. Clinic, diagnosis, and treatment.
2. complications of cholelithiasis. Choledocholithiasis. Obstructive jaundice. Clinic. Diagnosis and treatment.
3. Acute cholecystitis. Etiology. Pathogenesis. Classification.
4. Clinic and diagnosis of acute cholecystitis.
5. Differential diagnosis of acute cholecystitis.
6. Tactics and treatment of acute cholecystitis. Endoscopic methods of treatment of patients with acute cholecystitis.
7. Dropsy and empyema of the gallbladder. Etiopathogenesis. Clinic, diagnosis, and treatment.
8. Chronic cholecystitis. Clinic, diagnosis, and treatment.
9. special laboratory research methods in the diagnosis of diseases of the gallbladder and biliary tract.
10. acute cholecystopancreatitis. Clinic, diagnosis, and treatment.

Lecture No. 3.

1. Topic:

1.1. diseases of the pancreas. Etiology, pathogenesis, classification. Clinic, diagnosis. Differential diagnosis. Treatment.

2. Purpose:

1. The student should know:

1. The student should know: (Pancreatitis)

1.1. Topographic anatomy and physiology of the pancreas.

1.2. methods of pancreatic examination.

1.3. etiology and pathogenesis of pancreatic diseases.

1.4. clinical signs of acute and chronic pancreatitis.

1.5. complications of pancreatic diseases.

1.6. diagnosis of pancreatic diseases.

1.7. differential diagnosis of pancreatic diseases.

1.8. conservative treatment of acute pancreatitis

1.9. indications for surgical treatment of pancreatitis.

1.10. acute methods of pancreatitis treatment.

3. Lecture abstracts:

Pancreatitis is a non-bacterial inflammation of the pancreas caused by pancreatic enzymes

The pancreas, translated from Greek, means "everything is eaten."

The pancreas is an elongated organ located behind the stomach. I-II goes in front of the lumbar vertebra, and the tail ends near the ribs X-XI.

Length 10-23cm, width 3-9cm, thickness 2-3cm, weight 70-80g

His tube opens 75 percent into the large thorax (duodenum 12), as described by Wirsung in 1642 and Oddi in 1720.

In 4 percent of cases, the Wirsung tube spontaneously opens into the duodenum.

The wirsung tube opens together with the holedochus, the vater tube, by 33.6 percent.

A common ampoule and a 5% Oddi sphincter are connected in 2 test tubes. compose.

In tube 2, it is located far from the kidney of the vater and the sphincter of Oddi

Wirsung tube-descends into the holedoch.

Vessels supplying blood to the pancreas

A. Gastroduodenalis, a. Hepatica communis

A. Pancreatoduodenalis inferior, a. mesenterica superior

A. Lienalis - often supplies the caudal part with the body of the gland

The composition of pancreatic juice:

Amylase is cleaved in the active state, breaks down carbohydrates, hydrolyzes starch and converts it into maltose.

Lipase is partially active, and its activating bile acids and calcium salts digest fats and fatty acids, glycerin.

A protease is an enzyme that breaks down proteins into amino acids. And enzymes involved in proteolysis-trypsinogen, carbopeptidase, collagenase, deinuclease, etc.

Etiology and pathogenesis

The etiology of acute pancreatitis can be caused by various causes:

1. diseases of the gallbladder and biliary tract:

Cholecystitis, cholelithiasis, choledochal inflammation, etc

. II. diseases of the stomach and duodenum:

Chronic gastritis, peptic ulcer diseases, etc.

Patterns and symptoms.

A sign of mondora is the appearance of purple (violet) spots on the body and face;

Gray-Turner's sign is cyanosis in the peripheral abdominal regions: - these symptoms are interpreted as the action of enzymes and impaired hemodynamics.

Holsted (1901) - cyanosis of the skin of the abdomen

A sign of Kurte is pain, which is a horizontal line in the epigastric region 6-7 cm above the navel.

The Resurrection sign is a weakening or blurred pulsation of the aortic root during palpation of the epigastric region.

Mayo-Robson-pain when pressing on the left spine-rib angle with three fingers.

A sign of Makhov is hyperesthesia of the skin on the upper side of the navel.

A sign of Chukhrienko is pain in the epigastric region when tapping the palm horizontally below the navel.

Kacha-spontaneous pain, hyperesthesia of the I-III zone
, yellowness around the veins-navel

Grunveld-petechiae near the navel

Bovine-transverse desiccation of the colon

Gusenbauer-upper displacement of the stomach, transverse displacement of the colon downwards – pancreatic cyst.

Janelidze-pancreatitis differs from a heart attack in that the pain increases with pressure.

Shoffar-pain in the right flight-projection of the pancreatic head

Fitz-acute hemorrhagic pancreatitis

Soft abdomen with peritonitis

Dynamic kneading-intestinal paresis

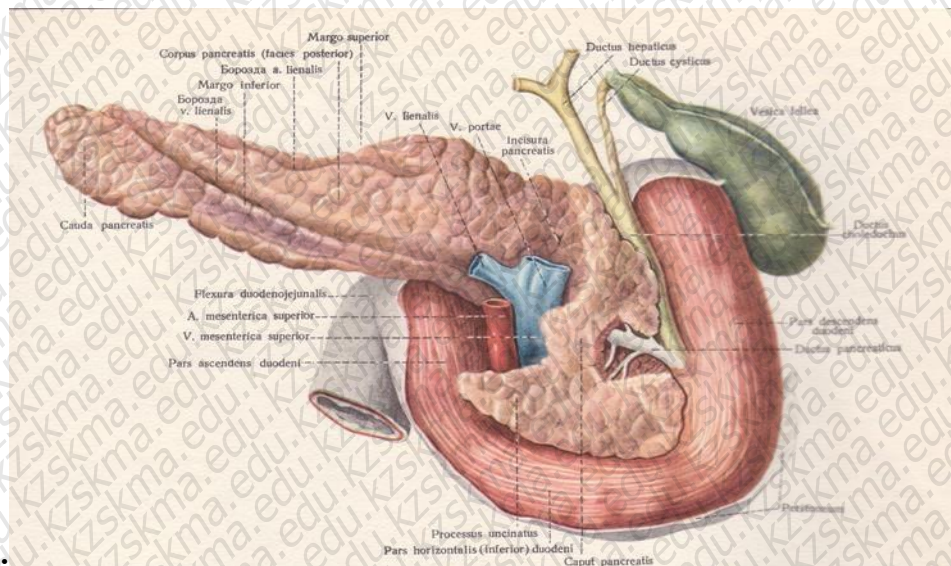
Lumbar pain in the upper abdomen

The activity of pancreatic enzymes in various body fluids plays an important role in the diagnosis of acute pancreatitis. An increase in the level of amylase in the blood is especially common (90%), less often-amylase in the urine (75%). But with necrosis of the gland, the amount of amylase decreases to normal recovery.

The norm of amylase according to the Karalei method is

3.3-8.9 mg / sec/ L. In blood

, up to 44 mg / sec/ L. In urine.



4. Illustrative material:

5. Literature

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Friendship University of Russia, 2019. 148 p. - URL: <https://www.iprbookshop.ru/104237.html>

6. Security Questions (Feedback Feedback)

Questions

1. acute pancreatitis. Etiopathogenesis. Pathanatomy. Classification.

2. Clinic and diagnosis of acute pancreatitis.

3. differential diagnosis of acute pancreatitis. Complications of acute pancreatitis. Clinic, diagnosis and treatment

4. conservative treatment of acute pancreatitis.

5. tactics and indications for surgical treatment of acute pancreatitis. Surgical methods of treatment.

6. methods of drainage of bile ducts (indications, methods).

7. Modern methods of diagnosis of obstructive jaundice.

8. Indications for external and internal drainage of the bile ducts after Choledochotomy.

9. differential diagnosis of obstructive jaundice

10. radical surgery for obstructive jaundice.

11. diagnosis and treatment of acute cholangitis.

Lecture No. 4.

1. Topic:

Acute surgical pathologies of the abdominal cavity. Peptic ulcer of the stomach and duodenum.

1.1. Gastric and duodenal ulcer. Etiology, pathogenesis, classification. Clinic, diagnosis. Differential diagnosis. Treatment.

2. Purpose:

1. The student should know:

- 1.1. Anatomy and physiology of the stomach and duodenum
- 1.2. methods of examination of the stomach and duodenum
- 1.3. etiology and pathogenesis of peptic ulcer disease
- 1.4. clinical symptoms of uncomplicated peptic ulcer disease
- 1.5. complications of gastric and duodenal ulcers
- 1.6. clinical symptoms of complicated peptic ulcer disease
- 1.7. diagnosis of advanced forms of peptic ulcer disease
- 1.8. differential diagnosis of complicated forms of ulcers.
- 1.9. principles of treatment of advanced forms of gastric ulcer.
- 1.10. Classification of bleeding
- 1.11. determination of the degree of blood loss
- 1.12. the clinical picture of bleeding.
- 1.13. clinic and diagnosis of gastric and duodenal ulcer perforation
- 1.14. conservative and operative methods of treatment of a bleeding wound
- 1.15. differential diagnosis of Gastroduodenal bleeding
- 1.16. therapeutic tactics of Gastroduodenal bleeding
- 1.17. indications for surgical treatment of Gastroduodenal bleeding
- 1.18. surgical tactics of Gastroduodenal bleeding
- 1.19. Differential diagnosis of genital ulcers.
- 1.20. treatment of penetration WOUNDS
- 1.21. Classification of pyloric stenosis
- 1.22. Clinic and diagnosis of pyloric stenosis
- 1.23. treatment of gate stenosis.
- 1.24. clinic and diagnosis of gastric and duodenal ulcers
- 1.25. differential diagnosis of a perforation wound
- 1.26. surgical methods of Perforation wound treatment
- 1.27. the dependence of the volume of surgical intervention on the perforation ulcer various factors
- 1.28. principles of surgical and conservative treatment of complex forms peptic ulcer disease.

3. Lecture abstracts:

Peptic ulcer of the stomach and duodenum is a very common disease. The incidence in adults in developed countries is 8-12%. Peptic ulcer is 4 times more common in men than in women. In recent years, the incidence of peptic ulcer of the stomach and duodenum has become increasingly common among young people.

The size of the gastric ulcer ranges from several mm to 5-6 cm or more, and the depth exceeds 20 mm. They grow only in the mucous membrane, often destroying all the layers of the stomach.

A microscopic wound distinguishes 4 layers in the acute phase at the base

Etiology: HP is found in 90% of stomach ulcers and 100% of duodenal ulcers. HP's habitual habitat is the antrum of the stomach. HP, which inhabited the duodenum, leads to the development of ulcers and erosions. With the healing and elimination of HP, the motor disorders of the stomach and duodenum decrease, gastrinemia decreases, the inflammatory process is eliminated, wounds heal quickly and its recurrence stops.

Along with HP, other factors are involved in the development of peptic ulcer disease, such as exposure to nicotine, alcohol, cravings for spicy foods, and gastropathic medications (aspirin, indomethacin, reserpine). Classification.

I. Depending on the location of the wound:

1. stomach ulcer

2. Duodenal ulcer.

3. Gastric ulcer and duodenal ulcer.

II. Due to the secretory function of the stomach:

1. Normal acidity

2. High acidity

3. low acidity

III. 1. complicated

2. uncomplicated.

IV. by etiology:

1. Helicobacter is associated with pylori.

2. Helicobacter is not related to pylori.

V. Clinical phase.

I. Exacerbation: newly formed ulcer; onset of epithelialization

II. suppression of exacerbations: scar-free ulcer healing; cicatricial ulcerative deformity.

III. Remission (positive).

VI. severity:

mild; moderate, severe.

Peptic ulcer of the stomach:

- cardiac and subcardial department;

- body and abdominal angle;

- antral part;

- the pyloric canal.

The duodenum:

- duodenal bulb;

- post-bulbar department (intra-cranial grooves).

By the number and diameter of the groove:

- single cases;

- a lot;

- small (up to 0.5 cm);

- average (0.6-1.9);

- large (2.0-3.0);

- giant (>3.0).

In clinical form:

- type;

- atypical (with atypical pain syndrome, without pain, asymptomatic).

By the nature of the course of the disease:

- acute (newly identified groove);

- chronic: with rare complications (once every 2-3 years); with monthly complications (2 times a year and more often).

Clinic

Often, the pain is transmitted to the left side of the chest to the arch of the sternum, lumbar or thoracic spine. The pain is associated with eating. With a stomach ulcer, pain occurs within 30 to 60 minutes of eating. it appears later, that is, early pain. For duodenal ulcers: 2-3 hours later, there will be a slight aura, as well as "pain on an empty stomach." According to the mechanism of occurrence, night pain is closer to "fasting pain", which often occurs between 11 p.m. and 3 a.m. Peptic ulcer is vomiting with acidic stomach contents, which really causes a characteristic symptom aura and brings relief. Sometimes patients themselves cause a gag reflex because of this. Sometimes there are other dispersive disorders: heartburn, nausea, belching, constipation.

Vomiting is the second classic symptom of peptic ulcer disease. Vomiting brings relief, the vomit contains acidic gastric juice, undigested food mass, cream, blood attacks.

Heartburn-a burning sensation in the back occurs in 70-80% of patients. This means increased hypersecretion and acidity of gastric juice. Heartburn can occur before, after eating, or without any contact. Patients will have to drink soda.

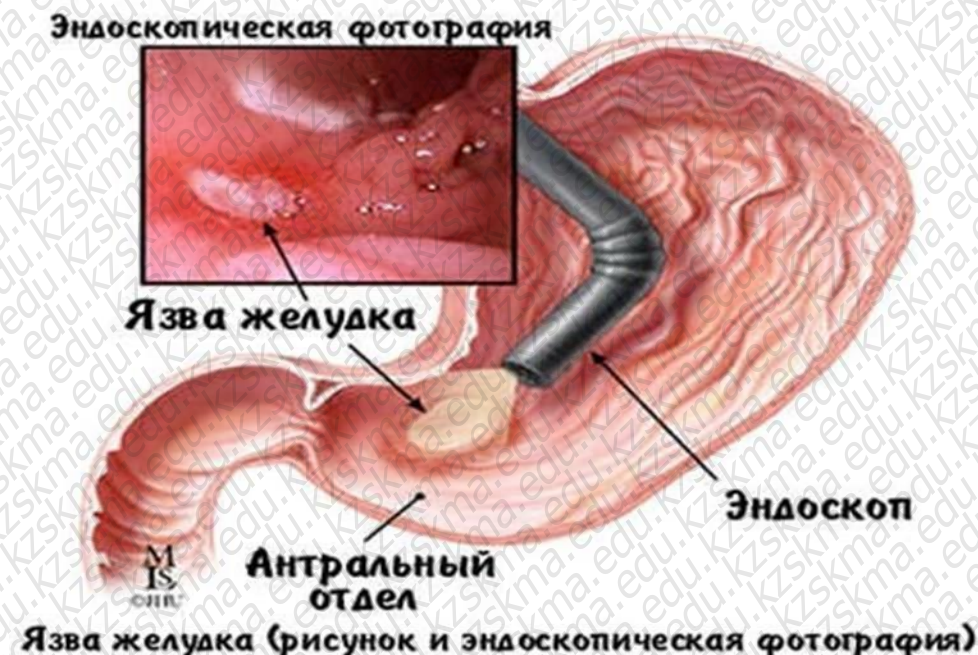
Stuttering-develops as a result of antiperistalsis of the stomach with a deficiency of the cardiac sphincter of the esophagus. Belching is caused by air or a small amount of acidic, odorless food. Smelly belching occurs when the stomach rots with achilia

The first stage. The pain is especially felt in the epigastric region and to the right of the midline. Feeling pain on an empty stomach or within 2-3 hours after eating. then, especially at night. Dyspeptic changes are also observed.

Stage II. Pain in the epigastric region persists, but not much. During endoscopy, the wound is cleared of fibrin plaque and swells around it.

Stage III. Pain occurs only on an empty stomach, there are no changes in the mucous membrane of the duodenum and stomach, there are no dyspeptic disorders.

4. Illustrative material



5. Literature

Basic literature

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6. Security Questions (Feedback Feedback) Questions

1. anatomical and topographic data on the stomach. Methods of investigation of stomach diseases. Conservative and surgical treatment of the ulcer was painful.
2. bleeding from a gastroduodenal ulcer. Etiopathogenesis. Clinical forms of bleeding from gastric and duodenal ulcers. Differential diagnosis.
3. Conservative treatment of bleeding from gastroduodenal ulcer.
4. tactics and surgical treatment of bleeding from gastroduodenal ulcer. Surgical methods of treatment.
4. Gastric ulcer and duodenal ulcer. Clinic and diagnosis.
5. Differential diagnosis of gastric and duodenal ulcers in acute surgical diseases of the abdominal cavity.
6. Tactics and types of surgical intervention for gastric and duodenal ulcers. Complications
7. Stomach ulcer and 12-finger closed and atypical perforation intestine. Clinic, diagnosis, and treatment.
8. Penetrating and callous ulcers of the stomach and duodenum. Clinic, diagnosis and treatment.
9. pyloroduodenal stenosis of ulcerative etiology. Etiopathogenesis.

10. Clinic and diagnosis of Pyloroodenar stenosis. Preparation of the operation.**Surgical methods of treatment. Postoperative care.**

Lecture No. 5.

1. Subject: external abdominal cracks (light)

1.1. external abdominal cracks. Etiology, pathogenesis, classification. Clinic, diagnosis. Differential diagnosis. Treatment.

2. Purpose:

1. The student should know:

1.1. Definition of the concept of light.

1.2. Classification of cracks.

1.3. etiology and pathogenesis of light

1.4. diagnosis and diagnosis of anterior abdominal wall cracks.

1.5. differential diagnosis of cracks of various localization

1.6. methods and principles of surgical treatment.

1.7. mechanisms of violation of the lumen of the anterior abdominal wall.

1.8. clinic and diagnosis of compression hernias.

1.9. Differential diagnosis of compression cracks.

1.10. features and scope of surgical interventions for compression hernias.

3. Lecture notes:

Light is the transfer of abdominal organs into neighboring cavities or under the skin through natural or artificial pores closed by the abdominal cavity. There are 2 types: internal and external.

External cracks are when the organs of the abdominal cavity come out under the skin along with the peritoneum through artificial or physiological defects.

And the case when the stomach is not covered with a veil is called an event.

Classification of light.

1. depending on the areas where the crack begins in the abdominal cavity and the exit routes under the skin:

a) vertical

b) oblique

2. by the time of appearance:

a) congenital (the vaginal process of the abdominal cavity does not completely overlap, its cavity is in contact with the abdominal cavity. Later, a crack forms in which the vaginal growth becomes a light sac.)

b) acquired

3. according to the clinical course:

a) uncomplicated light

b) complicated light (narrowing, inflammation, light displacement, coprostasis)

Inguinal hernia (Hernia inguinale) is the most common type of abdominal wall hernia. All types of external cracks account for 70-80% of them. And men account for 90-97% of the total number of inguinal hernias. The inguinal hernia is located within the inguinal triangle (the Hungarian triangle) in the lower part of the abdominal wall.

Etiology

Local causes common causes

1. local:

A) vulnerability and weakness of the layers of congenital muscular aponeurosis

B) surgical, occupational and household injuries, additional diseases

2. General:

A) constitutional peculiarity of a person (asthenics)

B) the gender of the sick person

C) age

D) severe heredity

Pathogenesis

* The course of development of an oblique inguinal hernia is divided into several stages:

1. The initial stage

2. The core stage

3. the stage of complete inguinal hernia formation

4. the stage of light entering the scrotum

* Vertical inguinal hernia does not have such stages of development, its exit is not long and it does not enter the scrotum,

Clinic

During physical exertion, the patient suddenly has a bulge in the groin, and this place becomes painful, and discomfort occurs when walking. If there is uncompressed light, it protrudes when amplified in the vertical position and returns inside in the horizontal position.

Inguinal hernia should be distinguished from hydrocele, varicocele, femoral hernia.

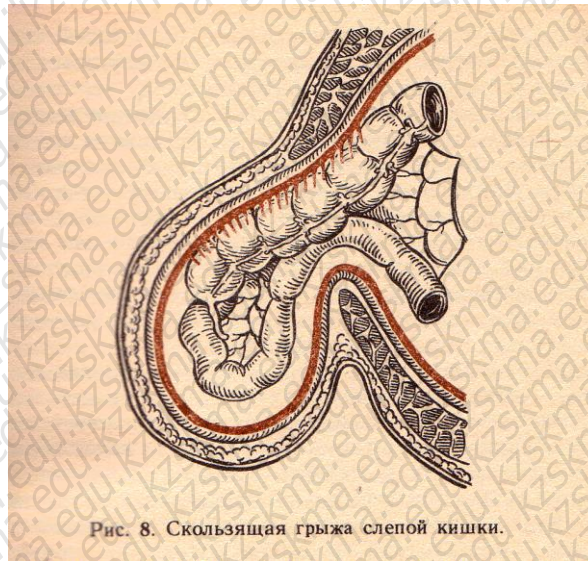
With an inguinal hernia that does not move, the scrotum may be enlarged and resemble a hydrocele, but with a hydrocele, the convex shape is not pear-shaped, rounded, oval, with a dense tip and smooth surface. With a hydrocele, the percussion sound is dull, and the inguinal hernia may have tympanitis. The most important thing is diaphanoscopy.

Unlike varicocele, palpation causes a snake-like expansion of the veins of the spermatic cord, the expanding veins easily loosen when the scrotum is pressed and lifted upward.

With a femoral hernia, the bulge of brightness is located below the inguinal ligament.

An umbilical hernia is a protrusion of the abdominal organs from the umbilical region through a weak spot in the abdominal wall. In women, umbilical hernia is twice as common as in men, due to the tension of the umbilical ring during pregnancy. The Zharyk gate is the umbilical Sakina.

Femoral cracks are less common in men than inguinal ones. Women aged 40-60 are 15-20 times more likely to get sick than men. This is due to the fact that women have a wide femur and a high degree of its muscles and vessels, as well as low strength of the Pupart ligament.



4. Illustrative material:



5. Literature

Basic literature

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Questions

1. external abdominal hernias. Etiopathogenesis. Classification of hernias.
2. inguinal hernias. The anatomy of the intestinal canal. Direct and oblique, congenital and acquired inguinal hernias. Clinic. Differential diagnosis.
3. Indications and contraindications for surgical treatment of inguinal hernias. Surgical methods of treatment.
4. Umbilical hernias. Etiopathogenesis. Clinic. Diagnostics. Surgical methods of treatment.
5. hernias of the white line of the abdomen and other rare places. Clinic, diagnosis and treatment.
6. Squeeze your belly with light. Etiopathogenesis. Pathanatomy. Clinic. Differential diagnosis.
7. incorrect hernia. The differential diagnosis is incorrect and pinched hernia. Hernia inflammation. A mobile hernia. Features of the clinic, diagnosis, and surgical treatment.

Lecture No. 6.

1. Topic:

1.1. acute intestinal obstruction. Etiology, pathogenesis, classification. Clinic, diagnosis. Differential diagnosis. Treatment.

2. Purpose:

1. The student should know:

- 1.1. etiology of acute intestinal obstruction.
- 1.2. pathogenesis of acute intestinal obstruction.
- 1.3. classification of acute intestinal obstruction.

- 1.4. clinical signs of acute intestinal obstruction.
- 1.5. methods of diagnosis of acute intestinal obstruction.
- 1.7. differential diagnosis of acute intestinal obstruction.
- 1.8. principles of conservative treatment.
- 1.9. indications for surgical treatment.
- 1.10. acute methods of treatment of acute intestinal obstruction.

3. Lecture abstracts:

Acute intestinal obstruction is a symptomatic complex based on a violation of the polyethological liquidity (passage) of the intestinal contents

Etiology.

Congenital character (atresia of the intestine, rectum and other parts), Hirschsprung's disease: the number of Auerbach plexuses in the intestinal wall decreases sharply, which leads to peristalsis disorders, common mesentery of the cecum and ileum, dolichosigma, defects of the diaphragm and peritoneum

Acquired character. Adhesions, inflammatory infiltrates, hematomas, tumors, foreign bodies, bile and feces

Classification

- Dynamic
- spasticity
- paralytic
- Mechanical
- obturation
- strangulation
- combined (adhesive resistance belongs to it)

By origin

- innate
- walking
- By the level of interference
- high (small intestine)
- low (thick)
- In the clinical area
- full
- partially
- sharp
- chronic

Paralytic intestinal obstruction is a condition that occurs with intestinal paresis or paralysis. The most common causes of this type of obstruction are peritonitis, surgical trauma (during abdominal surgery), and the spilling of blood, urine, or bile into the abdominal cavity.

Spastic intestinal obstruction

This occurs with intestinal cramps and can be caused by various factors.:

- irritation of the intestine from its opening;
- mechanical irritation of the intestine;
- with reflex irritation;
- functional and organic damage to the nervous system;
- spasm of intestinal vessels,
- dysentery;
- poisoning with morphine, lead, nicotine.

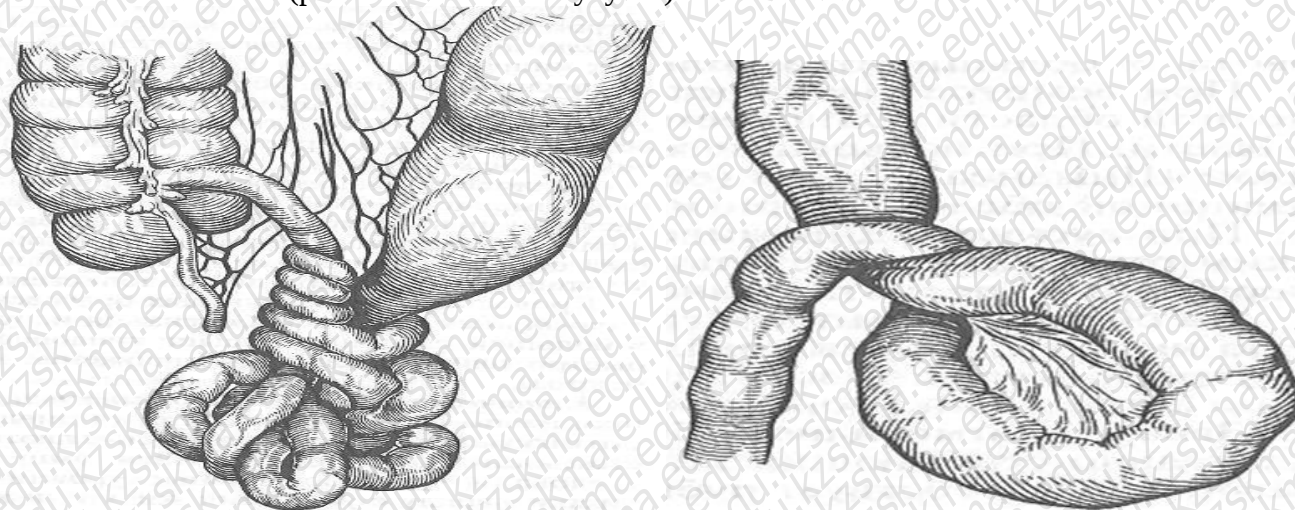
Strangulation intestinal obstruction-intestinal obstruction with circulatory disorders in mesenteric and intra-abdominal vessels is caused by their compression, which leads to the rapid development of the pathological process and the early onset of necrobiological changes in the compressed intestine.

Obstructive intestinal obstruction-develops as a result of obstruction or external compression of the lumen of the intestinal tube. The breeches are not involved. The most common cause is cancer. The obturation valve is usually a thick contact obstacle. They can roll gallstones, which is noticeable at the beginning of a spark - when fistulas form.

During invagination, obturation is combined with strangulation as a result of the insertion of part of the intestine along with the mesentery into the lumen located below or above the intestinal tube.

Adhesions (adhesions) can rotate the intestinal lumen due to excessive bumps in the "bivalve" area, creating "windows" in the abdominal cavity that cause strangulation of individual intestinal loops.

4. Illustrative material (presentation of the usynyada)



5. Literature

Basic literature

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6. Security Questions (Feedback Feedback)

Questions

1. acute intestinal obstruction. Etiopathogenesis. Classification.
2. Differential diagnosis of acute intestinal obstruction.
3. tactics and treatment of acute intestinal obstruction
4. Obstructive intestinal obstruction. Etiopathogenesis. Clinic, diagnosis and treatment.
5. Strangulatory intestinal obstruction. Clinic, diagnosis, and treatment.
6. Intestinal inversion. Pathanatomy. Clinic, diagnosis and treatment.
7. Intestinal node. Pathanatomy. Clinic, diagnosis and treatment
8. Intestinal invagination. Etiopathogenesis. Pathanatomy. Clinic, diagnosis and treatment.
9. Sticky intestinal obstruction. Etiopathogenesis. Clinic, diagnosis, and treatment.
10. Dynamic intestinal obstruction. Etiopathogenesis. Clinic, diagnosis and treatment.

Lecture No. 7.

1. Topic:

Peritonitis is an inflammation of the peritoneum.

2. Purpose:

1. The student should know:

- 1.1. Anatomy and physiology;
- 1.2. Etiology and pathogenesis;
- 1.3. Pathological anatomy;
- 1.4. Classification;
- 1.5. clinical symptoms;
- 1.6. clinical course;
- 1.7. clinical course of complicated forms;
- 1.10. Diagnostics;
- 1.11. differential diagnosis;
- 1.12. principles of treatment;

1.13. chronic forms.

3. Lecture abstracts:

Peritonitis refers to acute or chronic inflammation of the parietal and visceral lobes of the abdominal cavity. Peritonitis is one of the very serious and dangerous complications that occur after inflammation of the abdominal organs or their injuries. The reason for this is that with peritonitis, a purulent inflammatory process occurs in a space that anatomically structurally clogs and complicates, and also has a number of specific features associated with its development in this space. Peritonitis is a very dangerous complication.

Grouping of peritonitis by degree of condition (V. S. Mayat, V. D. Fedorov, 1973)

Local: a) limited (inflammatory cancer, abscess); b) unlimited (the limit of limitation is not defined);

A) diffuse (the inflammatory process covers less than two layers of the abdominal cavity);

b) the inflammatory process (the inflammatory process covers more than two layers of the abdominal cavity);

c) general or total (inflammation has completely covered the parietal and visceral leaves of the abdominal cavity);

K. S. Simonyan (1970), B. D. Savchuk (1979) and others. The need for classification (grouping) by stage (stages) of development along with the degree of peritonitis has been shown. According to the authors, the burden of peritonitis depends not only on the level of its condition, but also on how the complication occurs and develops. Therefore, the authors point out the need to divide the period of occurrence of peritonitis into three stages (stage). They:

The reactive stage (staria). This includes the first 24 hours of the disease onset period.;

Toxic (poisoning), i.e. the toxic stage (with an interval of 24-72 hours);

Terminal stage (the disease lasts more than 72 hours).

At the first, i.e., the reactive stage of peritonitis, there are local and general symptoms (soreness, stiffness of the abdomen, muscles due to contraction, vomiting, palpitations, etc.).

At the toxic (poisoning), or toxic stage, the above-mentioned local signs, general symptoms characteristic of poisoning of the body (change in the shape of the face, pallor of the skin, vomiting, increased body temperature, the appearance of changes in the blood characteristic of purulent inflammation, etc.).

At the terminal, i.e., the final stage of peritonitis, the condition of a sick person reaches a very bad form. A detailed description of the clinical manifestations of these stages is discussed in detail below.

- Depending on the clinical course: a) acute; b) chronic;

- Depending on the type of exudate: a) serous; b) fibrinous; c) Purulent; d) hematopoietic (hemorrhagic; e) fecal; f) putrefactive (putrefactive); g) additive (serous-firinous, fibrinous-Purulent, etc.).

Depending on the condition: a) limited (abscess or inflammatory tumor-infiltrate); b) diffuse (limited, moderate, general or total).

- Depending on the stage (phase) of development: a) reactive; b) toxic; c) terminal.

Etiology and pathogenesis. The basis of the occurrence of surgical peritonitis is the settlement of virulent microorganisms in the abdominal cavity. In most cases, as noted above, the infection occurs after inflammation of the organs located in the abdominal cavity, or due to a violation of their integrity, for various reasons. These include acute appendicitis, cholecystitis, pancreatitis, abdominal pain, and gynecological diseases. Now, if in some cases, due to injuries to the anterior layers of the abdomen or complications of the last period after surgery, the integrity of the abdominal organs is disrupted, the microflora enters the abdominal cavity from these organs (traumatic, i.e. peritonitis that occurs after injury or surgery). In addition, microbes can enter the abdominal cavity hematogenously from purulent foci lying far from it. Although some sick people (especially female patients) have all the symptoms of peritonitis, their source, that is, the main cause, cannot be determined (cryptogenic peritonitis).



Clinical manifestations. Clinical manifestations of peritonitis are manifested in different ways. Since they depend on the time of the onset of the disease, the stage of development, the volume of its spread in the abdominal cavity, the virulence of the microflora, the general condition of the patient, etc.

Therefore, when detecting peritonitis, it is worth carefully reviewing the medical history of patients, identifying and finding out if there are signs of acute diseases that cause peritonitis.

In the reactive stage of the disease (24 hours before), especially in the early stages, when patients complain of very severe (intense) pain. It is determined whether this feeling increases with the slightest movement of a sick person, coughing, or deep, even breathing. If the pathological focus causing Peritonitis is located in the upper layer of the abdominal cavity, there is a spread of painful sensations in the back, scapular muscle, shoulder (irradiation), and now that the focus is in the pelvic cavity, painful sensations can spread towards the pelvic, sacral, intercostal areas.

At the stage of intoxication (with an interval of 24-72 hours), pain and some of the mentioned local symptoms worsen somewhat, and signs of intoxication of the body also appear. The stomach gets bigger and drier, the stomach and stools become rarer, and vomiting becomes more frequent. The heart rate (pulse) reaches 110-120 in 1 minute, blood pressure drops, and body temperature rises (38-39 °C).

At the terminal stage of the disease (over 72 hours), the patient's general condition is in very poor condition. There are general and local symptoms characteristic of a high degree of intoxication. The pain covers all areas of the enlarged, dried abdomen, vomiting and hiccups are often observed. The patient's tone changes, his facial features change, his eyes light up and go inside, the skin layer turns yellow, his face becomes covered with sticky sweat ("Hippocratic run"). The tongue dries ("brush-shaped tongue"). With a pulse reading of 1 min 120-140, the respiratory rate reaches 1 min 30-40. Blood pressure drops sharply. There is an increase in the volume of free fluid in the abdominal cavity. In addition, there are signs of liver, kidney, and brain function failure (hepatargia, oliguria, impaired consciousness, encephalopathy, etc.).

The detected changes in the results of clinical, laboratory, and biochemical studies largely correspond to the stage of peritonitis. In these studies, changes in the reactive stage of peritonitis are not pronounced, on the contrary, in its toxic and terminal stages they are pronounced (leukocytosis, increased ESR, signs of hypovolemia, changes in liver transferases, increased bilirubin, creatinine, etc.).

If there are difficulties in the diagnosis of peritonitis, instrumental examinations (X-ray, ultrasound, laparoscopy, laparocentesis) should be widely used. These research methods are of great importance for detecting peritonitis or the symptoms of the diseases that cause it.

Treatment. Treatment of peritonitis is carried out depending on its clinical form, stage of development, extent of the condition, degree of metabolic changes and dysfunctions of the main organs, types of microorganisms, etc. Therefore, the treatment of peritonitis is a very difficult and difficult job.

Scientists from the country and abroad have made a great contribution to this problem (Aliev M., 1982, Izimbergenov N., 1998, Ospanov A., 1999, Lokhvitsky St., 1985, Madykenov O., 1978, Seksenbaev D., 1998, Nurmakov A. Zh. et al., 2000, Kuzin M. I., 1982, Savelyev B.C., 1982, Pendulum B.C., Fedorov V. D., 1976, Savchuk B. D., 1987, etc.). These scientists draw attention to the fact that the process of treating peritonitis should be carried out comprehensively, without interrupting the patient at first glance. It should be noted that doctors or ambulance staff who first saw sick people at the prehospital stage without a full diagnosis of peritonitis, it is better not to give them medications that eliminate the feeling of illness, since such drugs can change the clinical picture of the disease and negatively affect the correct diagnosis. After delivery to the hospital, when the diagnosis of peritonitis is fully established, the disease requires immediate comprehensive treatment, and it is important that the measures included in this complex are fully implemented.

4. Illustrative material: presentation.



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6. Security Questions (Feedback Feedback) Questions

Lecture No. 8

1. Topic: (diseases of the rectum. Hemorrhoids.)
 - 1.1. diseases of the rectum. Hemorrhoids. Etiology, pathogenesis, classification. Clinic, diagnosis. Differential diagnosis. Treatment.
2. Purpose:
 1. The student should know:
 - 1.1. Definition of Hemorrhoids.
 - 1.2. classification of hemorrhoids.
 - 1.3. etiology and pathogenesis of hemorrhoids.
 - 1.4. Clinic and Diagnosis of hemorrhoids.
 - 1.5. differential diagnosis of hemorrhoids of various localization
 - 1.6. methods and principles of surgical treatment.
3. Lecture abstracts:

Hemorrhoids

This is a disease characterized by a gradual expansion of the cavernous bodies of the rectum. Hemorrhoids are a common disease of the rectum. It is observed in 10% of adults, and among diseases of the rectum, the proportion of hemorrhoids is 40%.

Etiology.

- Congenital insufficiency of the vascular system

- From stagnation of blood due to disruption of the process of normal blood flow in the rectal veins;
- From the effects of mechanical factors, namely from constipation or from the fact that the work performed does not require a lot of movement.;
- Compression of rectal veins during pregnancy;
- The body's susceptibility to diseases
- Inefficiency of nutrition;
- Pregnancy

Classification.

According to clinical signs

- 1) Operational
- 2) chronic
- 3) relapsing (relapsing)

Depending on the location (anatomical characteristics):

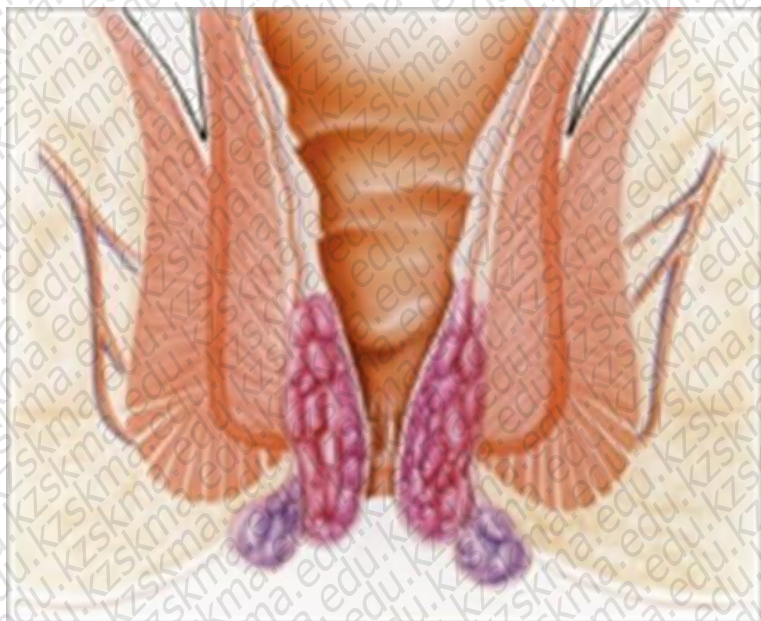
- 1) external hemorrhoids-dilated veins are located at the level of the crested line, that is, in the area bordering the skin of the creamy layer of the rectum
- 2) Internal hemorrhoids – dilated veins are located above the level of the clamp (sphincter).

Depending on the degree of protrusion of the dilated venous nodes:

- a) grade 1 – the nodules come out only during a large bowel movement (act of defecation), then spontaneously, without additional help, back into place.
- b) grade 2 – after defecation or during heavy physical exertion, dislocated nodes do not fall into place on their own, so it becomes necessary to lower them into place with additional movements (hands).
- c) grade 3-nodes come out and do not fall back into place when the patient is in an upright position, when walking or with minor physical activity the load

Clinic.

- Unfavorable sensations in the anus
- Pain
- The feeling of itching is a certain complication of the bowel movement process
- Bleeding from the Anus (symptoms of anemia)



4. Illustrative material: presentation.

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6. Security Questions (Feedback Feedback)

Questions

1. Hemorrhoids and its complications. Etiopathogenesis. Clinic, diagnosis and treatment

2. cracks of the rectum. Etiopathogenesis. Clinic, diagnosis and treatment.

Lecture No. 9.

1. Topic: (diseases of the lungs and pleura. Abscess and gangrene of the lungs. Spontaneous pneumothorax. Acute purulent pleurisy. Pyopneumotorax. Chronic empyema of the pleura. Bronchiectatic disease.)

1.1. diseases of the lungs and pleura. Abscess and gangrene of the lungs. Spontaneous pneumothorax. Acute purulent pleurisy. Pyopneumotorax. Chronic empyema of the pleura. Bronchiectatic disease. Etiology, pathogenesis, classification. Clinic, diagnosis. Differential diagnosis. Treatment.

2. Purpose:

1. The student should know:

1.1. identification of lung abscess and gangrene. Spontaneous pneumothorax. Acute purulent pleurisy. Pyopneumotorax. Chronic empyema of the pleura. Bronchiectatic disease.

1.2. Classification of lung abscesses and gangrene. Spontaneous pneumothorax. Acute purulent pleurisy. Pyopneumotorax. Chronic empyema of the pleura. Bronchiectatic disease.

1.3. Etiology and pathogenesis of lung abscess and gangrene. Spontaneous pneumothorax. Acute purulent pleurisy. Pyopneumotorax. Chronic empyema of the pleura. Bronchiectatic disease.

1.4. Clinic and diagnosis of lung abscess and gangrene. Spontaneous pneumothorax. Acute purulent pleurisy. Pyopneumotorax. Chronic empyema of the pleura. Bronchiectatic disease.

1.5. differential diagnosis of lung abscess and gangrene. Spontaneous pneumothorax. Acute purulent pleurisy. Pyopneumotorax. Chronic empyema of the pleura. Bronchiectatic disease

1.6. methods and principles of surgical treatment.

3. Lecture abstracts:

A lung abscess is often a purulent disintegration of necrotic tissue areas characterized by the presence of a gallbladder within the segment, several pus-filled cavities, and amniotic infiltrates..

Etiology microbes spread through the lungs through mine routes: 1)aerogenic - airborne; 2) hematogenic - with blood; 3) lymphogenic abscess the cause of the abscess is most often hemolytic streptococcus, gram-negative flora. 57% of them are resistant to antibiotics. The inflammatory process spreads to the aorta and exposes it to selectase. Bronchiectasis of the bronchi disrupts blood circulation in the lungs, increasing congestion and thrombosis. The tendency to necrosis of lung tissue and suppuration is increasing.

The main cause of panda abscess is bronchial blockage, blood vessel thrombosis, compression by swollen lung tissue, and pulmonary infarction. That is, the presence of a lung abscess can lead to lung deactivation, circulatory disorders, and targeted putrefaction of lung tissue by microbial toxins.

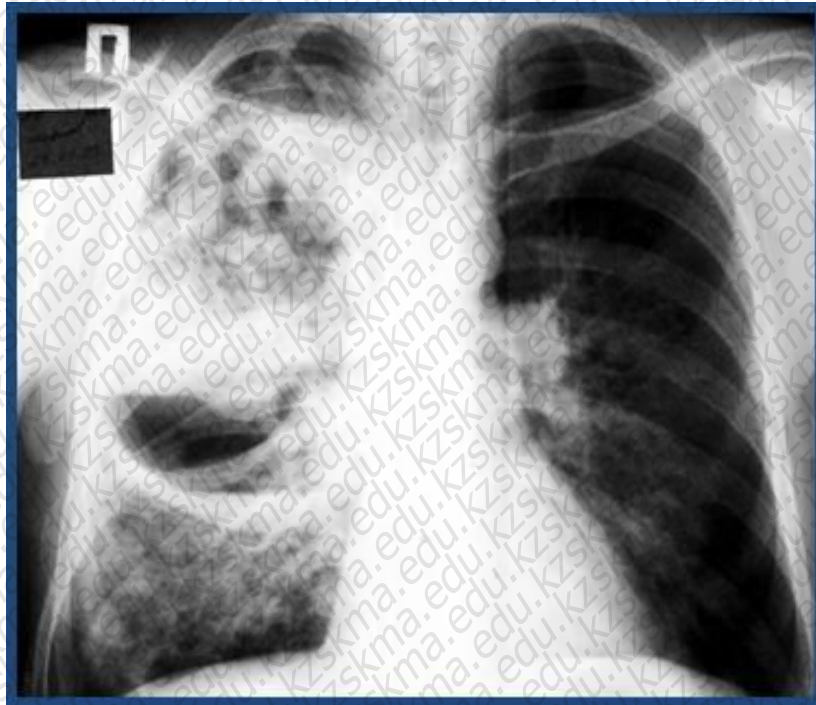
Aspiration abscess is inhaled when a foreign body enters the bronchi, stomach contents during surgery and in the postoperative period.

Classification The most effective classification is that of V. S. Savelyev and E. G. Grigoriev (2004). I. Depending on the etiology: 1) post-traumatic 2) hematogenic 3) aspiration 4) post-Pneumonic II. By clinical and morphological character 1) acute abscess A) single B) multiple 2) pulmonary gangrene A) limited B) spreading

III. Due to complications: pleural empyema A) bronchopleural discharge B) bronchopleural discharge 3) pulmonary blood 4) pulmonary sepsis

Clinical manifestations the clinical course of an acute abscess depends on the phase of sweat development. On the first facade, necrosis of the lung tissue and the formation of an abscess are observed. On the second facade, there is a rupture of pus in the corresponding bronchus.

4. Illustrative material: presentation.



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6. Security Questions (Feedback Feedback) Questions

1. Lung abscess and gangrene. Anatomical and physiological data. Etiopathogenesis. Classification.
2. Clinic, diagnosis and treatment of lung abscess and gangrene.
3. Clinic, diagnosis and treatment of acute purulent pleurisy.
4. Clinic, diagnosis and treatment of Pyopneumothorax.
5. Clinic, diagnosis and treatment of chronic pleural empyema.
6. Clinic, diagnosis and treatment of bronchiectasis.

Lecture No. 10

1. Topic: vascular surgery. Varicose veins. varicose veins.

Vascular surgery. Varicose veins. Mandibular gills.

Blockage of the arteries. Atherosclerotic lesion of the arteries. Vanishing endarteritis: Raynaud's disease and syndrome. Etiology, pathogenesis, classification. Clinic, diagnosis. Differential diagnosis. Treatment.

2. Purpose:

1. The student should know:

1.1. detection of varicose veins, arteriosclerosis, endarteritis.

1.2. classification of varicose veins, arteriosclerosis, and endarteritis.

1.3. Etiology and pathogenesis of varicose veins, arteriosclerosis, endarteritis.

1.4. clinic and diagnosis of varicose veins, arteriosclerosis, and endarteritis.

1.5. differential diagnosis of varicose veins, arteriosclerosis, endarteritis of various localization

1.6. methods and principles of surgical treatment.

3. Lecture abstracts:

Varicose veins

Latin.varis means node.

Varicose veins or varicose veins disease are characterized by the formation of pocket-like and snake-like formations on the vein wall, elongation of the veins, and the absence of valves.

Normally, the pressure in the Vein is 75-120 mmhg. It will be b, and if the lid is deficient, 500-800 MMHG, B will be. Patients can stand or walk a lot. pressure 1000-2000 MMHG will be B.

Pathogenesis:

This chapter of the disease is still fully understood and remains an unsolved problem. To this day, various opinions are expressed and expressed about the causes and development of varicose veins (Lidsky A. T., 1958, Mamamtavrishvili D. G., 1964, Savelyev V. S. et al., 1972, etc.). Among them, mechanical, infectious, hormonal theories can be noted. These views suggest that a disease can have multiple causes rather than just one.

Recently, a special place in traditional formulations has been given to "endothelial dysfunction", i.e. at first there is damage to the endothelium and smooth muscle cells of the vein, which eventually leads to tension and stretching of the vein and deficiency of the portal apparatus. Microcirculation disorders and changes in the trophic structure of the vascular wall are of particular importance in the development of this disease.

International classification of CEAR

I. Clinical classification. (C)

C0. The symptoms of varicose veins are imperceptible. Patients complain of a feeling of heaviness in their legs.

C1. Reticulated veins and vascular asterisks (telangiectasia) are visually revealed. Some patients experience leg muscle cramps at night.

C2. Dilated veins are visible, rather than temporal veins.

C3. Swelling appears on the soles, ankles, calves, which does not disappear even after a short rest.

C4. Visualization shows signs of lipodermatosclerosis (dermatitis, hyperpigmentation of the ankle joint).

C5. The above-mentioned skin changes and healing wound.

C6. The above-mentioned skin changes and permanent trophic ulcer.

II. Etiological classification (E)

EU: congenital diseases

Man: primacy, the reason is unknown

Es: secondary, the cause is known: after thrombosis, after injury, etc

. EP: the cause of the disease could not be determined

III. Anatomical classification (A)

-AS. Superficial veins

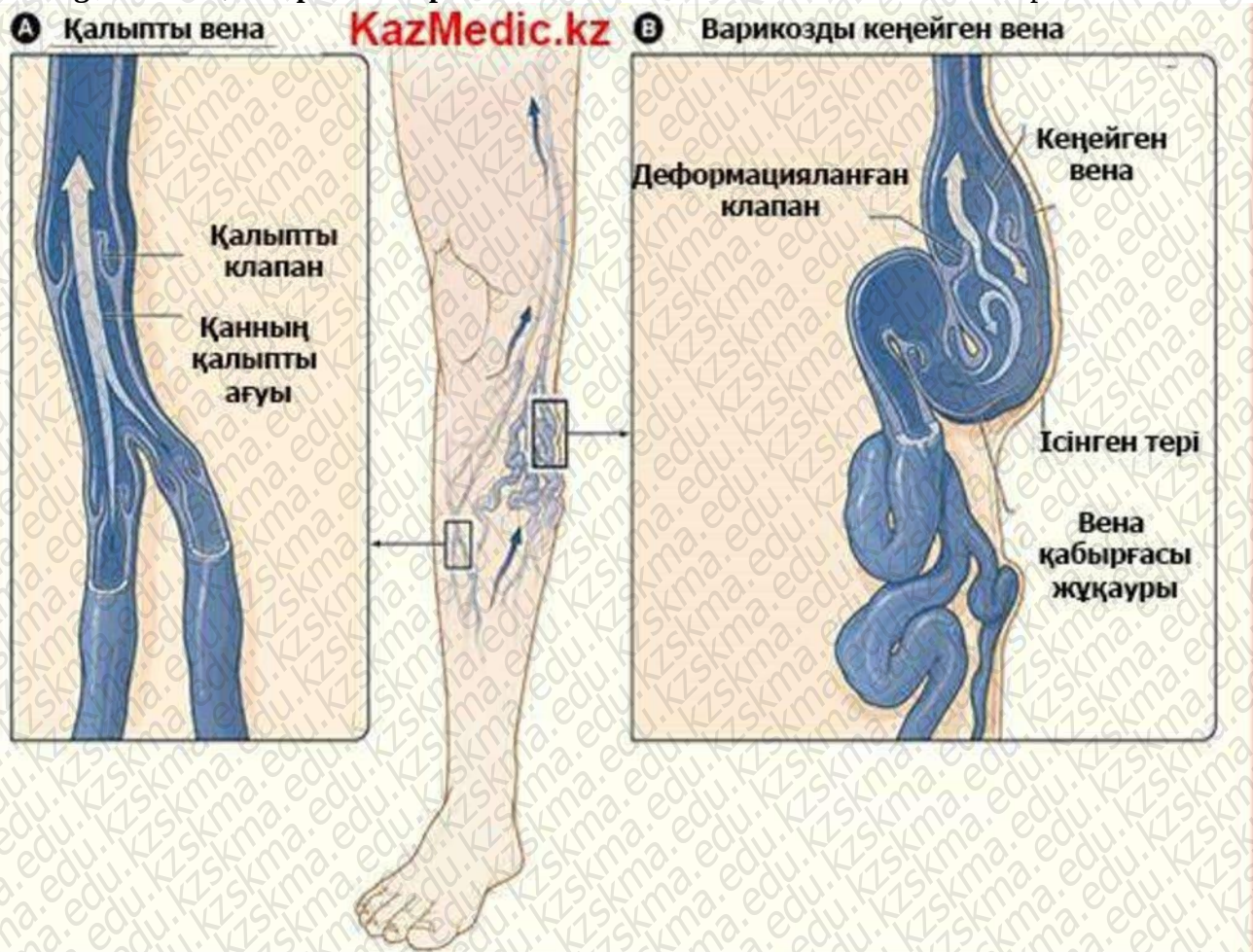
1. telangiectasia and reticular veins

2. large cutaneous vein above the knee

3. Large cutaneous vein below the knee

4. Small cutaneous vein

5. changes in veins, except for the pool and the KTV basin4.Illustrative material: presentation.



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6. Security Questions (Feedback Feedback)

Questions

1. varicose veins. Anatomical and physiological data. Etiopathogenesis. Classification.

2. Clinic, diagnosis and treatment of varicose veins.

3. functional tests and special methods for the study of varicose veins of the lower extremities.

4. thrombophlebitis of deep and upper veins. Etiology. Classification. Clinic, diagnosis and treatment.

Principles of anticoagulant therapy in acute thrombosis.

5. obliterating endarteritis. Etiology. Pathanatomy. Clinic,

treatment.

Lecture No. 11.

1. Topic:

Occlusive diseases of the arteries. Atherosclerosis is a lesion of the arteries.

2. Purpose:

1. The student should know:

1.1. occlusive diseases of the arteries. Detection of arteriosclerosis of the arteries, endarteritis.

1.2. occlusive diseases of the arteries. Classification of arteriosclerosis and arterial endarteritis.

1.3. Etiology and pathogenesis of varicose veins, arteriosclerosis, endarteritis.

1.4 occlusive diseases of the arteries. Arteries. clinic and diagnosis of atherosclerosis, endarteritis.

1.5. differential diagnosis of varicose veins, arteriosclerosis, endarteritis of various localization

1.6. methods and principles of surgical treatment.

3. Lecture abstracts:

Atherosclerosis (atheroscleross; gr. athra-porridge and sklrs-hardening) is an accumulation of cholesterol on the inner surface of blood vessels, which accumulates and concentrates like a nodule.

The place where Cholesterol is absorbed first turns into a hardened knot if the yellow stripe takes the form of a spot. These nodes are connected to each other. As a result, ulcers form on the inner layer of the root, calcium salts settle, the roots harden, and blood flow is obstructed. Atherosclerosis often occurs in the aorta, the largest blood vessel in the body. Due to atherosclerosis, thinned areas of the aorta become baggy and clog the blood vessels of both wide (aneurysms) and other organs. Because of this, sometimes the patient can die within a few minutes. Atherosclerosis of the iliac artery leads to a dead intestine, atherosclerosis of the foot leads to gangrene of the foot. Atherosclerosis is considered the main cause of heart contractions (angina pectoris), chronic coronary heart disease, and heart attack. It is mainly found in the elderly. Causes of atherosclerosis include high blood pressure, endocrine diseases, and obesity. The advanced form of atherosclerosis requires surgical treatment. To avoid atherosclerosis, you need to eat right, especially use vegetable oil, and exercise.[1][2]

Etiology

Currently, there is no unified theory of the occurrence of this disease. The following options are proposed, as well as their combinations: the theory of lipoprotein infiltration, the primary accumulation of lipoproteins in the vascular wall, the theory of endothelial dysfunction is the primary violation of the protective properties of the endothelium and its mediators, autoimmune is the primary dysfunction of macrophages and leukocytes, infiltration of their vascular walls, formation of a pathological clone of monoclonal primary smooth muscle cells, viral is the first viral lesion of the endothelium (herpes, cytomegalovirus, etc.), a genetically inherited defect of the vascular wall, chlamydia is basically, the primary lesion of the vascular wall of Chlamydia pneumoniae is chlamydia. An age-related increase in the level of hormonal gonadotropic and adrenocorticotrophic hormones leads to a higher synthesis of the building material for cholesterol.

Development

The development of atherosclerosis, that is, atherosclerosis, occurs in stages:

The initial stage I is vascular lipoidosis or the formation of lipid spots and stripes.

Stage II – fibrous-at this stage, collagen and connective tissue grow and surround "bubble" cells and cholesterol particles, forming a fibrous capsule.

Stage III is the stage of development of complex and complicated conditions.

The clinical picture.

The clinical picture of atherosclerosis depends on the area in which it is located, and not on the level of development of the pathological process. Sometimes a single atherosclerotic chamomile of the coronary artery leads to death, and severe atherosclerosis of the marginal arteries, thoracic or abdominal aorta may occur without severity and have no significant effect on human life expectancy. Atherosclerosis, being a progressive process, mainly affects the vessels of one or more areas. Daisies are often located on large roots and areas of the aorta where the roots spread. The vessels most susceptible to atherosclerosis are the aorta, crown, brain, kidneys, leg and abdominal arteries.

Basic principles of dietary therapy of atherosclerosis. The main approach to treatment is diet therapy. A group of experts from the European Society for the Study of Atherosclerosis (1987) published seven "golden" diet rules that restore lipoprotein metabolism:

Reducing the amount of fat.

A sharp decrease in the amount of saturated fatty acids that contribute to the development of hyperlipidemia (animal fat, butter, sour cream, eggs).

Widespread consumption of foods rich in unsaturated fatty acids.

Copious consumption of foods high in fiber and complex carbohydrates (vegetables, fruits).

The consumption of vegetable oil instead of butter when cooking.

Drastic reduction in the consumption of cholesterol-rich foods.

Limit the daily allowance of table salt to 3-5 g.

Diagnostics

Diagnosis of atherosclerosis-related diseases: interviewing the patient and identifying the symptoms of the disease: symptoms of coronary heart disease, symptoms of cerebrovascular accident, lameness rupture, symptoms of abdominal layer, etc.; General examination of the patient: signs of aging, listening to systolic murmur in the aortic focus; palpation of all arteries available for mandatory palpation: aorta, external iliac arteries, common femoral arteries, knee arteries, Lumbar and posterior large angular artery of the foot, radial and ulnar arteries, carotid arteries. detection of systolic murmur over the auscultation points of the arteries. in case of suspected damage to the arterial canal of the limb, a capillary reaction is determined. Determination of cholesterol concentration in blood and determination of blood lipid balance;

X-ray examination of the chest organs, X-ray endovascular research methods;

Ultrasound examination of the organs of the heart and abdominal cavity and the space behind the abdominal cavity; Dopplerography of the vessels of the extremities may be or better, ultrasound duplex and triplex scanning of the arteries of the brachiocephalic region, arteries of the extremities, aortic-axillary segment, as well as transcranial Doppler. diagnosis of arterial wall stiffness, including by volumetric sphygmography and determination of the cardio-ankle joint index (CAVI)

Medical treatment:

1. Statins are used - simvastatin, pravastatin, fluvastatin, atorvastatin.
2. Fibrates - fenofibrate, bezafibrate, clofibrate.
3. Multivitamin therapy is effective for patients with atherosclerosis.
4. enterosorbents.
5. sanatorium treatment. [3]

Drug therapy hypolipidemic drugs include the correction of arterial hypertension (especially systolic blood pressure), diabetes mellitus, and metabolic syndrome. However, normalization of the lipid spectrum is the most important. The drugs used for this purpose are divided into four main groups:

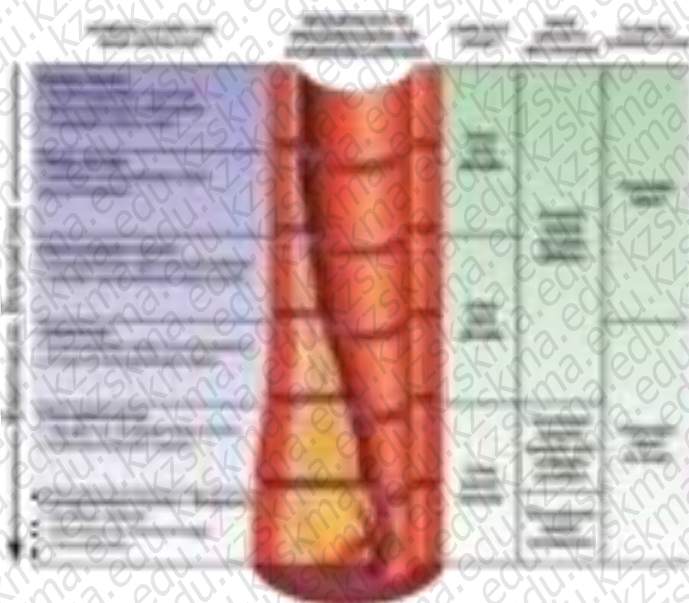
I-preventing the absorption of cholesterol

II-reduces the synthesis of cholesterol and triglycerides in the liver and reduces their concentration in blood plasma

III- release of atherogenic lipids and lipoproteins that enhance catabolism

Appendix IV[4]

4. Illustrative material: presentation.



5. Literature

Basic literature

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- Additional literature
- Kaldybaev, M. A. test questions of general surgery: a textbook / Ministry of Health of the Republic of Kazakhstan. - Karaganda: IP "Aknur", 2013. - 370 Pp.
- Madikenovich, O. Surgical diseases: textbook lectures / O. Madikenovich ; Ministry of Health of the Republic of Kazakhstan; educational educational and methodological section of KAZGMA for higher and postgraduate specialties. Almaty: Evero Publ., 2012, p. 405
- Zhumabekov, A. T. emergency first aid: an educational and methodical manual. Almaty: TechSmith, 2023, p. 112.
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- Total:
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Nurmakov, A. J. Surgical diseases [Electronic resource] : Textbook / A. J. Nurmakov, A. N. Baymakhanov. - The electron. text data. (59.9 Mb). Moscow: Litterra, 2017, p. 256. Hospital surgery. Syndromology [Electronic resource]: textbook. the manual / A. G. Abdullaev [et al.]; edited by N. O. Milanov, Yu. V. Biryukov, G. V. Sinyavin. - The electron. text data. (48.6 Mb). Moscow : GEOTAR-Media, 2013. 440 P. e-mail. Disk

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6. Security Questions (Feedback Feedback)

Questions

1. occlusive diseases of the arteries. Atherosclerosis of the arteries. Anatomical and physiological data. Etiopathogenesis. Classification.
2. occlusive diseases of the arteries. Clinic, diagnosis and treatment of atherosclerosis of the arteries.
3. thrombophlebitis of deep and upper veins. Etiology. Classification. Clinic, diagnosis and treatment. Principles of anticoagulant therapy in acute thrombosis.

4. obliterating endarteritis. Etiology. Pathanatomy. Clinic, treatment.

Lecture No. 12.

1. Topic: lesions and diseases of the esophagus.

1.1. diseases and lesions of the esophagus. Etiology, pathogenesis, classification. Clinic, diagnosis. Differential diagnosis. Treatment.

2. Purpose:

1. The student should know:

1.1. definition of the concept of diseases and lesions of the esophagus.

1.2. classification of diseases and lesions of the esophagus.

1.3. etiology and pathogenesis of diseases and lesions of the esophagus.

1.4. clinic and diagnosis of diseases and lesions of the esophagus.

1.5. differential diagnosis of diseases and lesions of the esophagus.

1.6. methods and principles of surgical treatment.

3. Lecture abstracts:

Esophagus (Latin esophagus-esophagus) is a department of the human digestive system connecting the oral cavity and pharynx with the stomach. The human esophagus is tubular, about 25 cm long, 16-22 mm in diameter (when stretched), and 3.5 – 5.6 mm thick. The esophagus consists of cervical, thoracic, and abdominal sections. Its wall consists of the inner cream, the middle fleshy, the outer thoracic and abdominal parts, and the cervical part (the outer connective membrane of a blood vessel). Achalasia of the esophagus is a relatively rare disease characterized by dystrophic changes in the intramural nervous plexus of the esophagus and cardia of unknown etiology, atony and enlargement of the esophagus, impaired peristalsis of its wall during swallowing and reflex opening of the cardia, including impaired passage of food into the stomach and prolonged sticking in the esophagus.

Esophageal injuries are divided into closed, which touch the mucous membrane, and open, which penetrate from the outside through the neck and chest.

Etiology and pathogenesis of esophageal injury. Closed esophageal injuries can occur during diagnostic and therapeutic measures during esophagoscopy, during cardiodilation, as well as when foreign bodies enter. If a solid probe is present in the esophagus for a long time, esophageal spasms may occur. Perforation of the esophageal wall can occur with various diseases of the esophagus – tumors, peptic ulcers, chemical burns. Injury to the esophagus can occur during surgery on the thoracic interval and lungs.

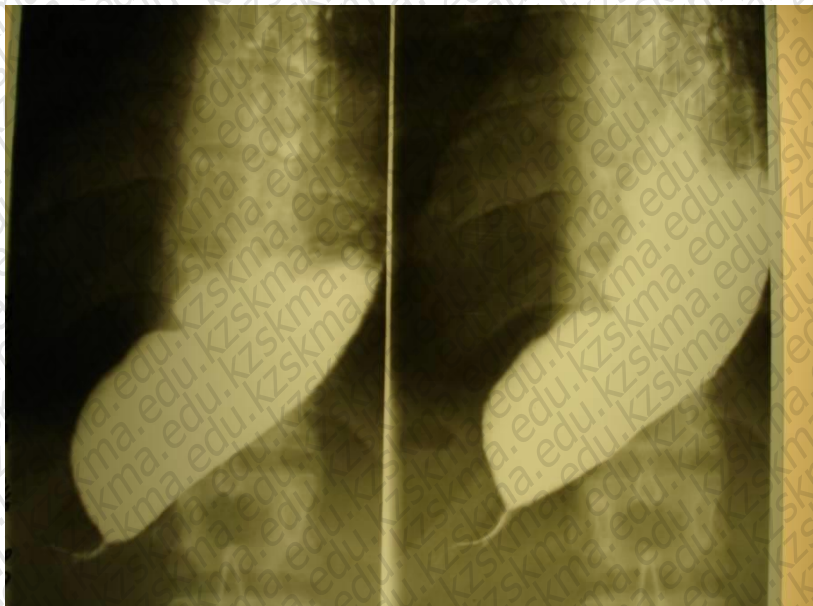
There are cases of spontaneous rupture of the esophagus. Predisposing factors include alcohol intoxication, overeating, weight lifting, increased intra-abdominal pressure during childbirth.

Spontaneous rupture of the esophagus sometimes occurs in a place where the esophagus is slightly above the junction with the stomach (Boerhaave syndrome). Rupture of the esophagus occurs when its contents divide into the chest, which in itself leads to mediastinitis. In case of damage to the pleural lobe, pleural empyema develops.

Open esophageal injuries occur with penetrating injuries to the neck, chest, or abdomen. They are usually accompanied by damage to many neighboring organs of the thyroid gland, trachea, lungs, heart, abdominal organs, and large blood vessels.



4. Illustrative material: Presentation.



5. Literature

Unwritten literature

Nurmakov A. J. surgical diseases: textbook / A. J. Nurmakov, D. A. Nurmakov ; KazNMU. S. D. Asfendiyarov. - Almaty: New book, 2021. - 496 p.

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Zhumabekov, A. T. emergency first aid: an educational and methodical manual. Almaty: TechSmith, 2023, p. 112.

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Kuznetsov, N. A. silversmithing in the science of surgery / N. A. Kuznetsov, A. T. Brontwein; Minister of Education and Science of the Russian Federation. - ; ". -M.: GEOTAR - Media, 2013. - 288 p.:

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<https://aknurpress.kz/reader/web/1460> General surgery .K. D. Usmanov, 2017/ <https://aknurpress.kz/login/1459> surgical diseases: textbook: in 2 volumes. Volume 2. / Ed. under the direction of M. I. Kuzin; textbook translated into Kazakh under the general editorship of A. N. Baymakhanova. - M.: GEOTAR-Media, 2018. - 528 p. - <http://rmebrk.kz / 1174591> surgical diseases: textbook: in 2 volumes. Volume 1. / Ed. headed by M. I. Kuzin; The textbook has been translated into Kazakh under the general editorship of A. N. Baimakhanova. Moscow: GEOTAR-Media, 2018. 560 p. <http://rmebrk.kz / 1174592> Nurakhmanov B. Zh., Ibadildin A. S., Amantayeva K. K., Surgery-aurular diseases, textbook. Almaty, VS "Evero", 2020. - p. 701 https://www.elib.kz/ru/search/read_book/669 / Madikenovich of life. Surgical diseases. - educational lectures. - Almaty: Evero-2020. - 309 P. https://www.elib.kz/ru/search/read_book/559/A J. Nurmakov, D. A. Nurmakov Surgical diseases. Almaty: "Evero", 2020. - 472 pages https://www.elib.kz/ru/search/read_book/670 Kaldybaev M. A. General surgery—textbook (Volume I). - 2020. - 432 p. https://www.elib.kz/ru/search/read_book/828 Kaldybaev M. A. General surgery – textbook (volume II). -2020. - 448 p. https://www.elib.kz/ru/search/read_book/829 Kaldybaev M. A. General surgery –textbook (volume III). -2020. - 200 p. https://www.elib.kz/ru/search/read_book/830/ Kovalev, A. I. General surgery: a course of lectures: A textbook. - 2nd ed., revised and the ball. - M.: Publishing House "Medical Information Agency", 2013. - 760c. <http://rmebrk.kz/book/1177583> Vasiliev V. K. and others. General surgery: A textbook. / V. K. Vasiliev, A. P. Popov, A.D. Tsybikzhapov. St. Petersburg: Lan Publ., 2014. 272c. <http://rmebrk.kz/book/1178354> general surgery. Volume 1: textbook / G. P. Rychagov, P. V. Garelik, A. N. Nekhaev [et al.]; edited by G. P. Rychagov, P. V. Garelik. Minsk: Higher School, 2008. 543 P. - URL: <https://www.iprbookshop.ru/20100.html> general surgery. Volume 2 : textbook / G. P. Rychagov, P. V. Garelik, A. N. Nekhaev [et al.]; edited by G. P. Rychagov, P. V. Garelik. - Minsk: Higher School, 2006. — 492 P. - URL: <https://www.iprbookshop.ru/20101.html> Kolb, L. I. General surgery : a textbook / L. I. Kolb, S. I. Leonovich, I. V. Yarovich. — Minsk : Higher School, 2006. - 444 p. - ISBN 978-985-06-1378-3. - Text: electronic / / Digital educational resource IPR SMART: [website]. - URL: <https://www.iprbookshop.ru/20102.html> Fundamentals of surgical patient care = Nursing care in surgery: a textbook / A. I. Badretdinova, A. S. Klimenko, N. G. Kostsova [et al.]. - Moscow: Peoples' Friendship University of Russia, 2019. - 148 P. - URL: <https://www.iprbookshop.ru/104237.html>

6. Security Questions (Feedback Feedback) Questions

1. The esophagus. Anatomical and physiological data. Etiopathogenesis. Classification.
2. clinic, diagnosis and treatment of diseases and lesions of the esophagus.
3. clinic, diagnosis and treatment of diseases and lesions of the esophagus.