VOLGOGRAD STATE
MEDICAL UNIVERSITY
Department of Pathological Anatomy

Stomach disease. Gastritis. Peptic ulcer (peptic ulcer). Tumors of the stomach. Bowel disease. Ischemic colitis. Nonspecific ulcerative colitis. Crohn's disease. Appendicitis.

Guidelines for students
III year of medical faculty
for practical exercises in pathological anatomy

Volgograd, 2020

DIGESTIVE DISEASES

1. The purpose of the lesson. To study the etiology, pathogenesis, morphological characteristics, complications and outcomes of diseases of the digestive system.

2. Requirements for the level of the student in the development of the discipline - pathological anatomy. The student must know:

1. Definition, etiology, patho- and morphogenesis of acute and chronic gastritis.
2. Definition, etiology, pathogenesis, pathological anatomy, complications of gastric ulcer and duodenal ulcer.
3. Definition, classification, pathological anatomy of stomach cancer.
4. Definition, etiology, classification, pathological anatomy of acute appendicitis.
5. Definition, etiology, classification, pathological anatomy, complications of ulcerative colitis and Crohn's disease.

3. Theoretical aspects.

GASTRITIS

Gastritis is an inflammation of the gastric mucosa.
Classification of acute gastritis.
By etiology and pathogenesis:
1. exogenous gastritis - occurs with the direct action of external food, drug poisoning or infection,
2. endogenous gastritis with the indirect action of internal agents, for example, uremic in chronic renal failure.

Acute gastritis can be isolated, but more often accompanied by involvement in the process of the intestinal mucosa (gastroenteritis). It occurs due to infection, nutritional errors (overeating, eating too rough and spicy foods), taking medications (salicylates, sulfonamides, corticosteroids, etc.), with poisoning, as a manifestation of an allergy.

The prevalence of the process:
1. diffuse
2. focal: fundal, antral, pyloric antrum.

By morphology:
1. catarrhal,
2. fibrinous,
3. purulent (phlegmonous),
4. necrotic.

The most common form of acute gastritis is simple (catarrhal) gastritis, other forms (fibrinous, phlegmonous, necrotic gastritis) are rare. With simple gastritis, the mucous membrane is full-blooded, swollen, with small hemorrhages, saturated with serous or serous-mucous exudate, infiltrated with leukocytes located in its own layer and between the cells of the superficial epithelium. Hypersecretion of mucus by epithelial cells, their degeneration, necrobiosis and desquamation are noted. As a result of the death of the epithelium, small erosions with hemorrhages occur. In newborns, miliary ulcerative gastritis is described, which progresses rapidly and can result in perforation, its origin is unclear.

Depending on the severity of the lesion, changes in the mucosa range from vasodilation and edema of lamina propria to erosion and hemorrhage. Erosion is a section of the mucous membrane with a partial violation of the epithelium, while in the ulcer there is a violation of the muscular layer of the mucosa. Erosions in acute gastritis are usually multiple, so bleeding from them can be very dangerous. However, usually quick (within 24-48 hours) healing occurs by regeneration. With frequent relapses of acute gastritis, chronic gastritis can develop.

Chronic gastritis is a long-term current disease based on dysregenerative processes: atrophy, hyperplasia, metaplasia, sclerosis, dysplasia. Signs of inflammation are less pronounced.

Chronic gastritis is mostly combined with damage to the duodenum (gastroduodenitis). Among the causes of chronic gastritis, violations of the diet and diet, various diseases of the digestive system, infections, neuroendocrine and metabolic disorders, allergic conditions, Helicobacter pylori are of primary importance.

Classification of chronic gastritis (CG)

By pathogenesis:

1. Gastritis type A (autoimmune) is characterized by damage to the fundus of the stomach,
2. Gastritis type B (non-immune) is localized in the antrum.

Currently, the following forms of chronic gastritis are distinguished:

1. autoimmune chronic gastritis;
2. Helicobacter-associated chronic gastritis;
3. chemical (reflux) gastritis;
4. other forms of gastritis.

Autoimmune chronic gastritis

In patients with this type of chronic gastritis in the blood - antibodies against parietal cells of the stomach and against receptors to the external Castle factor. In these patients, hypochlorhydria, up to anchlorhydria, and B12-deficient anemia are observed. The association of autoimmune gastritis with macrocytic anemia is called pernicious anemia.

The gastric body (fundus gastritis) is more often affected: there is damage to specialized parietal (parietal) cells (gland atrophy) and the replacement of lamina propria with fibrous tissue, infiltration by lymphocytes and plasma cells. The mucous membrane of the antrum is not damaged. In some places of the integumentary-fossa epithelium of the fundus of the stomach, intestinal metaplasia can be observed. With this type of metaplasia, mucus producing cells are replaced by goblet cells containing acidic glycoproteins. In severe cases, suction and Panet cells may appear. This metaplasia is a pre-tumor state. However, cancer in patients with intestinal metaplasia develops quite rarely.

Helicobacter-associated chronic gastritis

The most common cause of chronic gastritis is Helicobacter pylori infection. This microorganism is gram-negative, populates the most protected space located near the surface of the epithelium under the mucous barrier, where the pH is neutral. Helicobacter pylori is not a simple commensal, since it damages surface cells, which leads to accelerated desquamation of cells and infiltration of the gastric mucosa by polymorphonuclear leukocytes and chronic inflammatory cells. Helicobacter pylori is found in 90% of cases with the activation of chronic gastritis type B, but is never detected with type A. Helicobacter pylori is never found in the mucous membrane of the duodenum and does not cause intestinal metaplasia.

The acute inflammatory response caused by Helicobacter pylori develops as a result of the action:

complement components that are released upon activation by an alternative pathway and are chemotactic for polymorphonuclear leukocytes;

low molecular weight chemotactic factor secreted by bacteria;

interleukin-8, which is secreted by epithelial cells, macrophages and endothelial cells. Polymorphonuclear leukocytes secrete proteases and active oxygen-containing metabolites that cause gland atrophy. In lamina propria, plasma cells are synthesized that synthesize IgA, IgG and IgM against this microorganism.

With Helicobacter-associated chronic gastritis, the entire stomach is affected. However, this lesion is uneven. In most patients, the antrum and body are involved in the process, in which gland atrophy, fibrosis and intestinal metaplasia gradually develop. In patients with this localization of the process, the risk of developing ulcers and tumors of the stomach increases. The second type of change is inflammation, mainly in the antrum, without damaging the body of the stomach. In these patients, there is an increased acidity of gastric juice, which increases the risk of duodenal ulcers.

Chemical (reflux) gastritis

With regurgitation of bile and alkaline duodenal juice into the stomach, increased desquamation of the epithelium occurs, compensatory hyperplasia of proliferating cells in the bottom of the gastric fossa, vasodilation and edema of lamina propria, which is a manifestation of reflux gastritis. Reflux gastritis is most often observed:

in patients after operations that damage the pylorus;

as a result of impaired intestinal motility in gallstone disease and after cholecystectomy;

in patients with impaired anthro-duodenal motility, which can be either primary or secondary as a result of a pathological response to hormones such as cholecystokinin and secretin, which normally increase the tone of pylorus with an increase in acidity in the duodenum.

In reflux gastritis, the cells of the antrum begin to secrete intensely gastrin, which blocks the action of cholecystokinin and secretin on the muscle fibers of the pylorus.

With the prolonged existence of reflux gastritis, ulceration is possible.

A similar histological picture is observed with long-term oral use of non-steroidal anti-inflammatory drugs (NSAIDs).

Other forms of gastritis:

1. lymphocytic;
2. eosinophilic;
3. granulomatous.

Lymphocytic gastritis, the main histological manifestation is the presence of numerous mature lymphocytes in the surface layers of the epithelium. This form is sometimes found in patients with specific erosions along the enlarged folds of the mucosa. The etiology and relationship with Helicobacter-associated gastritis has not been established.

Eosinophilic gastritis is characterized by swelling of the mucosa and the presence of numerous eosinophils in the inflammatory infiltrate. It is assumed that eosinophilic gastritis is an allergic response to a food antigen to which the patient is sensitized.

Granulomatous gastritis is a rare form of gastritis in which epithelioid cell granulomas form. These granulomas can be a manifestation of Crohn's disease or sarcoidosis, but in rare cases it can be cryptogenic.

By morphology:

1. Chronic superficial gastritis.
2. Chronic atrophic gastritis

Currently, on the basis of histochemical and electron microscopic studies, forms of chronic gastritis are considered as phases of its development, accompanied by a structural reorganization of the mucous membrane. Changes can be focal with a primary lesion of the bottom or antrum of the stomach (fundus, antrum gastritis) or common (common gastritis).

In domestic and foreign literature there are many works on Helicobacter pylori infection. Impact study

Helicobacter pylori (HP) eradication during peptic ulcer disease allowed the US National Institute of Health (1994), the European Group for the Study of HP (1996), and the Russian Gastroenterological Association (1997) to recommend Helicobacter pylori therapy for all patients with peptic ulcer disease. Currently, a large number of modes of eradication therapy have been developed, but there is no consensus on their effectiveness and the optimal duration of treatment. In recent years, some authors have noted a decrease in the effectiveness of anti-helicobacter therapy, which is associated with the resistance of the pathogen to antibacterial drugs. Other authors claim that antibacterial drugs stop the reproduction of HP and stimulate the formation of a mostly breathable, but not cultivated cocciform structure. And today we have a small number of long-term observations.

With superficial gastritis, regurgitation of barium in the antrum, thickening and tortuosity of the folds in the antrum, and accelerated evacuation of barium from the stomach are determined by x-ray. Most patients have a normal X-ray picture.

Endoscopically in the area of the stomach’s body there is revenge, sometimes small focal hyperemia of the mucous membrane, the folds are relief, and in some places hypertrophied. In the pylorus, the folds are swollen with signs of hypersecretion. Sometimes there is an increased tone of the pylorus with pylorospasm.

When histopathological and electron microscopic examination noted: the epithelium of the gastric ridges and the fossa epithelium is represented by high cylindrical cells, the nuclei of which are rounded in shape, are located in the basal part of the cell. The boundaries of the nuclei are clear, chromatin is evenly distributed.

Flattened cells with a basophilic cytoplasm are found on the tops of the ridges, especially often in the pyloric section of the stomach. The nuclei in such cells are displaced to the apical part or are located at different levels, creating a multi-row picture, the chromatin in them is densified. There are also bubble-like light nuclei with fuzzy borders. The cytoplasm in such mucocytes is basic, the intercellular borders are poorly contoured. The infiltrate of the lamina propria of the mucous membrane mainly consists of plasma cells and lymphocytes with a small admixture of eosinophilic and neutrophilic fields

PEPTIC ULCER DISEASE

Definition Peptic ulcer disease is a chronic disease with periods of exacerbation. The main manifestation of the disease is a chronic recurrent ulcer of the stomach or duodenum.

According to localization and pathogenesis, peptic ulcer is isolated with localization of ulcers in the stomach and in the duodenum.

Etiology and pathogenesis.

Factors of aggression of the gastric mucosa:

1. Helicobacter pylori infection,
2. hydrochloric acid of gastric juice,
3. pepsinogen of gastric juice,
4. stress
5. smoking
6. drugs
7. alcohol intake,
8. violation of the regime and nature of nutrition,
9. heredity.

Protection factors of the gastric mucosa:

1. secretion of mucus,
2. Castle's intrinsic factor,
3. the allocation of bicarbonates,
4. epithelial factor,
5. blood circulation,
6. neural and muscle components.

Pathological anatomy.

The basis of peptic ulcer is a chronic recurrent ulcer. Ulcers are most often single, have a rounded shape, in diameter from several mm to 3-6 cm (giant ulcers).

Ulcer morphogenesis.

There are three stages: erosion, acute ulcer, chronic ulcer.

1. Erosion - a shallow defect (damage) of the mucous membrane within the epithelium. Erosion is formed with necrosis of the mucous membrane. Causes: stressful situations, various infectious diseases, brain operations or other volumetric operations, etc. Erosions are usually multiple, localized mainly along the lesser curvature of the body and pyloric stomach and less often in the duodenum; have a different shape; their size is from 1-2 mm to several centimeters. The bottom of the defect is covered with fibrinous coating, the edges are soft, even and do not differ in appearance from the surrounding mucous membrane. Healing of erosion - by epithelization (complete regeneration) in 3-4 days without scarring, with an unfavorable outcome - the transition to an acute ulcer.

2. Acute ulcer - a deep mucosal defect, penetrating to the actual muscle plate of the mucous membrane and deeper. Reasons: similar to those with erosion. Acute ulcers are more often single, round or oval in shape, in the form of a pyramid; their size is from several mm to several cm. Localization is a small curvature. The bottom of the ulcer is covered with fibrinous plaque; the edges are even, do not rise above the surrounding mucous membrane and do not differ from it in color. Microscopically: at the edges of the ulcer - a mild or moderate inflammatory process; after rejection of necrotic masses at the bottom of the ulcer - thrombosed or gaping vessels. Healing - scar formation in 7-14 days (incomplete regeneration), less often - an unfavorable outcome - a transition to a chronic ulcer.

3. Chronic ulcer - characterized by severe inflammation and proliferation of scar (connective) tissue in the area of the bottom, walls and edges of the ulcer. The ulcer is round or oval, less often - linear, slit-shaped or irregular in shape, of various sizes and depths. The edges of the ulcer are dense (callous ulcer), even, in the proximal part - undermined, in the distal - gentle.

4. Morphology of a chronic ulcer during an exacerbation:

During the exacerbation period, three layers are distinguished in the bottom of the ulcer:

1) the upper layer is a purulent-necrotic zone,
2) the middle layer is granulation tissue,
3) the lower layer is scar tissue that penetrates the muscle membrane.

During the period of exacerbation, the size and depth of the ulcer increase.

During remission, the purulent necrotic zone decreases. Granulation tissue grows, which matures and turns into a coarse-fibrous connective (scar) tissue. The processes of sclerosis in the area of the bottom and edges of the ulcer intensify. The bottom of the ulcer is exacerbated. Cicatrization of an ulcer does not lead to a cure for peptic ulcer, since at any time there may be an exacerbation of the disease.

Complications of peptic ulcer:

1. Ulcer-destructive - bleeding, perforation, penetration (develop with an exacerbation of the disease).
2. Cicatricial and ulcerative - stenosis of the input and output sections of the stomach (develop during the period of remission of the disease).
3. Inflammatory - gastritis, duodenitis, perivisceritis (develop during an exacerbation).
4. Malignancy (transition to cancer) ulcers.
5. Combined complications.

Bleeding is the most common complication; it develops in 25-33% of patients with peptic ulcer disease. This is an arrosive bleeding that occurs during an exacerbation of peptic ulcer. A duodenal ulcer is more often complicated by bleeding than a stomach ulcer. Bleeding is provoked by physical overstrain, abdominal trauma, various stressful situations, and the use of various medications (salicylates, glucocorticoids). It is characterized by vomiting of blood, chalk, collapse or shock. Death can occur from acute posthemorrhagic anemia or from hemorrhagic shock.

Perforation is the formation during the period of an exacerbation of an illness of a hole in the wall of the stomach and (or) the duodenum 12, which communicates with the abdominal cavity, omental bursa, retroperitoneal space, with hollow organs with the formation of fistulas. Perforation may develop with the formation of encapsulated pockets. Perforation of an ulcer in the abdominal cavity leads to diffuse purulent peritonitis, which can cause death.

Penetration - the spread (exit) of an ulcer to adjacent organs or tissues. Most often, penetration into the small omentum, pancreas (can lead to digestion of the organ), liver, hepatic-duodenal ligament, into the anterior abdominal wall (with the formation of inflammatory tumor-like infiltrate) is observed.

Stenosis is characterized by a sharp narrowing of the input and output sections of the stomach due to the growth of scar (connective tissue) during the period of remission of the disease. Pyloroduodenal stenosis is most common, which disrupts the passage of food and leads to vomiting and loss of water by the body. Pyloroduodenal stenosis is formed due to deformation of the pylorus and bulb of the duodenum, resulting from cicatricial changes in the intestinal wall and adhesions during perigastritis and periduodenitis. The process progresses at every relapse of peptic ulcer.

Inflammatory complications occur during an exacerbation around an ulcer. Perivisceritis ends with the formation of adhesions in the abdominal cavity, can lead to severe deformation of the stomach and duodenum. Against the background of perivisceritis, ulcer penetration into the surrounding organs is often formed.

Malignancy (transition to cancer) of an ulcer occurs only with a stomach ulcer of 3-5%.

Combined complications - a combination of several complications (perforation and bleeding, bleeding and penetration).

GASTRIC CANCER

Gastric cancer is one of the most common human cancers. According to the statistics of incidence, stomach cancer ranks first in many countries, in particular, in the Scandinavian countries, in Japan, in Ukraine, in Russia and other CIS countries. However, in the United States in the last twenty years there has been a marked decrease in the incidence of gastric cancer. A similar trend was noted in France, England, Spain, Israel, etc. Many experts believe that this happened due to the improvement of food storage conditions with the widespread use of refrigeration units, which reduced the need for preservatives. In these countries, the consumption of salt, salted and smoked products decreased, the consumption of dairy products, organic, fresh vegetables and fruits increased.

The high incidence of gastric cancer in the above countries, with the exception of Japan, according to many authors, is due to the use of foods containing nitrates. From nitrates, nitrosamines are formed in the stomach by conversion. The direct local action of nitrosamines is believed to be one of the most important causes of both cancer of the stomach and cancer of the esophagus. The high incidence of gastric cancer in Japan is associated with the consumption of large quantities of smoked fish (containing polycyclic carbohydrates), and not due to the high content of nitrosamines in foods.

Currently, gastric cancer has become more common at a young age, in the age groups of 40-50 years. The largest group of gastric cancers is adenocarcinomas and undifferentiated cancers. Cancers occur, as a rule, against the background of chronic inflammatory diseases of the stomach.

Precancerous conditions include chronic atrophic gastritis, in the genesis of which Helicobacter pylori, adenomatous polyps play a role. Precancerous histological changes in the

gastric mucosa include incomplete intestinal (colonic) metaplasia and severe dysplasia. However, some authors believe that stomach cancer can develop de novo, without previous dysplastic and metaplastic changes.

Background changes can lead to a precancerous process - severe epithelial dysplasia. Dysplasia is a violation of the phases of regeneration, in which proliferation (multiplication) of undifferentiated cells occurs, and differentiation (maturation) occurs late.

Classification of gastric cancer.

According to the localization of cancer:

1. pyloric (50%),
2. small curvature (27%),
3. cardiac (15%),
4. great curvature,
5. fundal,
6. total.

By the nature of growth and shape.

1. Cancer with exophytic growth (a tumor with such growth grows in the lumen of the stomach):

• plaque-like,
• polypous,
• mushroom,
• ulcer cancer (saucer-like cancer),
• ulcer-cancer - occurs with the malignancy of a chronic ulcer.

2. Cancer with endophytic growth (the tumor grows in the thickness of the stomach wall):

• diffuse,
• infiltrative-ulcerative.

3. Mixed cancer (exophytic and endophytic growth).

Histological forms:

1. adenocarcinoma (differentiated and poorly differentiated),
2. skirr (fibrous cancer),
3. cricoid cell (mucous cancer),
4. solid cancer.

Metastasis of gastric cancer is carried out - lymphogenous, hematogenous and implantation (contact) by.

Of particular importance are lymphogenous metastases to regional lymph nodes located along the lesser and greater curvature of the stomach, to the lymph nodes of the greater and lesser omentum. They appear first and determine the volume and nature of surgery.

Distant lymphogenous metastases include metastases to the lymph nodes of the liver gates (periportal), parapancreatic and paraaortic. The most important localization include lymphogenous metastases:

“Virch metastases” - to supraclavicular lymph nodes (usually to the left) (orthograde);

“Kruckenberg ovarian cancer” - in both ovaries (retrograde);

"Schnitzler metastases" - into the peritoneum of the posterior Douglas space and lymph nodes of pararectal tissue (retrograde).

In addition, lymphogenous metastases to the pleura, lungs, peritoneum are possible, although in the latter they are often implanted when the tumor germinates the serous membrane of the stomach wall.

Hematogenous metastases in the form of multiple nodes are found in the liver, lungs, and bones.

Implantation metastases manifest themselves in the form of multiple tumor nodes of various sizes in the parietal and visceral peritoneum, which are accompanied by fibrinous hemorrhagic exudate.

Complications of stomach cancer:

1. bleeding
2. anemia (acute and chronic),
3. perforation and peritonitis,
4. stenosis,
5. focal pneumonia,
6. intestinal obstruction,

7. obstructive jaundice,
8. cachexia.

APPENDICITIS

Definition Appendicitis is an inflammation of the appendix of the cecum, giving a characteristic clinical syndrome.

Etiology and pathogenesis. The main reason is autoinfection with Escherichia coli against the background of stagnation of the contents of the process.

Clinical and anatomical forms of appendicitis: acute and chronic.

Morphological forms of acute appendicitis: 1) simple, 2) superficial, 3) destructive (phlegmonous, apostematous, phlegmonous-ulcerative, gangrenous).

With simple and superficial appendicitis, changes are reversible, with destructive appendicitis, they are not reversible.

Acute simple appendicitis: stasis in the capillaries and venules, swelling of hemorrhages, congestion of siderophages, marginal standing of leukocytes and leukodiapedesis are characteristic. These changes are most pronounced in the distal appendix. In the intramural nervous system - dystrophic changes.

Acute superficial appendicitis. Macro: the process is swollen, its serous membrane is dull and full-blooded. Micro: foci of purulent inflammation of the mucous membrane (primary affects), on top of such a cone-shaped focus facing the lumen of the process, are surface defects of the epithelium.

These stages continue during the first day.

Phlegmonous appendicitis. Macro - the process is enlarged, the serous membrane is dull and full-blooded, with fibrinous plaque; the wall of the process on the incision is thickened, pus is released from the lumen; mesentery - swollen, hyperemic. Micro - diffuse neutrophil infiltration.

Apostematous appendicitis - against the background of diffuse purulent inflammation of the appendix - multiple small abscesses (abscesses).

Phlegmonous ulcerative appendicitis - against the background of diffuse purulent inflammation of the appendix - ulceration of the mucous membrane.

Gangrenous appendicitis is the outcome of all forms of acute appendicitis. Necrosis develops in the appendix tissue. It is secondary, as it develops as a result of the transition of purulent inflammation to the surrounding tissues (periappendicitis), including the process of the mesentery of the process (mesenteriolitis), which leads to thrombosis of the appendicular artery. Macro - the process is thickened, black, its serous membrane is covered with dirty greenish fibrinous-purulent deposits. Micro: extensive foci of necrosis with colonies of bacteria, hemorrhages, blood clots in the vessels, ulceration of the mucous membrane.

Complications of acute appendicitis:

1. perforation (the formation of holes in the wall of the process),
2. diffuse purulent peritonitis,
3. “appendicular infiltrate” or abscess (develops as a result of local spread of the inflammatory process to periappendicular tissues).
4. empyema of the appendix (accumulation of pus in the lumen of the appendix),
5. purulent thrombophlebitis of the vessels of the mesentery, pylephlebitis, pylephlebitic abscesses of the liver.

Chronic appendicitis develops after acute appendicitis and is characterized by sclerotic and atrophic processes, against which inflammatory-destructive changes may appear.

Complications of chronic appendicitis:

1. dropsy of the process (develops with cicatricial obliteration of the proximal part of the process, in which serous fluid accumulates in the lumen of the process, and the process turns into a cyst).
2. mucocele (accumulation in the lumen of the cyst of the secretion of glands - mucus).
3. myxoglobulosis of the appendix (the formation of spherical structures - myxoglobulis - in the lumen of the appendix as a result of its peristalsis).
4. pseudomyxoma of the peritoneum (a process resembling a tumor - myxoma, develops when a cyst breaks and the mucus and its cells form into the abdominal cavity when implantation of these cells on the peritoneum is possible.

Crohn's disease.

The disease mainly affects the terminal portions of the ileum, but it is believed that any part of the digestive tract can be involved in the process. In addition to lesions of the intestine, as well as ulcerative colitis, there can be systemic changes in the form of migrating polyarthritis, sacroiliitis (inflammation of the sacroiliac joint), lesions of the articular-ligamentous apparatus of the spine with the development of ankylosis, erythema nodosum (subcutaneous nodules), and thickening of the fingertips hands in the form of drum sticks.

Macroscopically in the affected intestine, the serous membrane appears dull and gray. The mesentery of the affected segment of the intestine is thickened, swollen, sometimes fibrosed. Mesenteric fat envelops the intestines. The wall of the intestine is thickened due to edema, inflammatory infiltration, areas of fibrosis and hypertrophy of the muscle membrane. As a result, the lumen of the intestine in the zone of changes is narrowed (the so-called cord-like intestine). Crohn's disease is characterized by a clear delineation of the affected segments of the intestine from the adjacent and not involved in the process.

At the early stage of the disease, surface ulcers occur, and as the process progresses, the ulcers merge into long, sinuous, linear necrotic stripes oriented along the length of the intestine. The surface of the intestine due to the alternation of zones of necrosis and unchanged mucous membrane resembles a cobblestone pavement. Between the folds of the mucous membrane thin gaps may appear, which often extend deep into the intestinal wall up to the serous membrane. At later stages, fistulas or adhesions between the loops of the intestine or between the intestine and other organs may develop.

Under the microscope, in the early stages of Crohn's disease, focal accumulations of polymorphonuclear leukocytes can be seen in the surface layer, and lymphocytes in the deeper layer in the mucous membrane. Individual crypts may also contain neutrophils. In the later stages, a significant amount of polymorphonuclear leukocytes accumulate in the area of the crypts of the small or large intestine and abscesses are formed that cause the destruction of the mucous membrane and lead to ulcers. Ulcers have different depths and clear boundaries.

Chronic forms of Crohn's disease, which are characterized by severe destructive processes, are much more common. In the small intestine, the villi become shorter and thicker, and in the large intestine they lose uniform distribution and become branched. The destruction of crypts is accompanied by progressive atrophy of the mucous membrane of the colon. In glandular cells, metaplasia is observed either in the direction of the antral glands (pyloric metaplasia) or in the distal colon in the direction of the lining with exocrinocytes with acidophilic granules (Panet cells) (normally these cells are absent in this section). In all layers of the intestinal wall there are accumulations of lymphocytes. In any layer of the intestinal wall in about 50% of patients can be found granulomas without necrosis, consisting of epithelioid and giant multinucleated cells (resemble sarcoid granulomas).

Of the complications of Crohn’s disease, the following should be called local: the development of fibrous strictures, in particular, in the terminal parts of the ileum, as well as the formation of fistulas in other parts of the intestine, bladder, vagina, on the skin near the anus. Due to cicatricial stenosis, intestinal obstruction may develop. Common complications: severe electrolyte disturbances, specific malabsorption of vitamin B, 2 followed by the development of pernicious anemia, generalized malabsorption, inflammation of the choroid (uveitis), perincholangitis, hydronephrosis due to damage to the ureters, systemic amyloidosis. In Crohn's disease, the risk of cancer of the gastrointestinal tract is 5-6 times higher than in healthy people.

Crohn's disease and ulcerative colitis are combined into a group of idiopathic bowel diseases. Crohn's disease is a granulomatous disease that most often affects the small and large intestines. Ulcerative colitis is limited to the colon, and granulomas do not form with it. The pathogenesis of these diseases is based on the inability of the intestinal immune system, which supports the homeostasis of the intestinal mucosa and regulates inflammatory reactions, to adequately respond to antigens (intestinal flora, food antigens). The result of such a mismatch is immune-mediated damage. The following mechanisms of immunopathological processes are

possible: the inability of epithelial cells of the small and large intestine to act as antigen-presenting cells; violation of the production of cytokines; change in the function of natural killers; the formation of cytotoxic anti-epithelial antibodies. One of the main sources of mediators that damage the wall of the colon and small intestine, which is observed in chronic ulcerative colitis and Crohn's disease, are neutrophils. Eosinophils, mast cells and fibroblasts are somewhat less significant in the production of mediators. The importance of immunological mechanisms in the pathogenesis of Crohn's disease and ulcerative colitis is also evidenced by systemic lesion.

4. Lesson plan

1) To study the following macropreparations, to describe them according to the scheme for the description of macropreparations.

Macropreparations:

1. Macropreparation "Multiple erosions and acute gastric ulcers." Pay attention to the localization, shape and depth of ulcers and erosion, the consistency of the edges and the color of the bottom of acute ulcers and erosion.
2. Macropreparation "Chronic gastric ulcer". Pay attention to the localization, shape and depth of the ulcer, the consistency of the edges and the color of the bottom of the ulcer, and the state of the gastric mucosa.
3. Macropreparation "Cancer of the stomach." Pay attention to the localization, shape and surface of the tumor formation, the consistency, edges, condition of the gastric mucosa.
4. Macropreparation "Acute phlegmonous appendicitis." Pay attention to the size, wall thickness and lumen content of the appendix, luster, color, presence of overlays, blood supply to the vessels of the serous membrane; thickness, color, integrity of the mucous membrane.

2) Examine the following micropreparations, sketch them, indicate and mark the pathological changes with arrows, using the atlas of micropreparations.

Micropreparations:

1. The drug "Acute catarrhal gastritis" (stained with hematoxylin and eosin). Pay attention to the localization, amount and composition of exudate, the condition of the integumentary and glandular epithelium, blood vessels, the cellular composition of the infiltrate of the own plate of the gastric mucosa.
2. Micropreparation "Chronic atrophic gastritis with intestinal metaplasia" (stained with hematoxylin and eosin). Pay attention to the thickness of the mucous membrane and the number of glands; cell composition of superficial and glandular epithelium; the cellular composition of the infiltrate of the own plate of the mucous membrane; lymphoid tissue in the mucous membrane.
3. The micropreparation "Adenocarcinoma of the stomach" (stained with hematoxylin and eosin). Pay attention to the thickness of the mucous membrane, the amount of localization, the shape of the normal and tumor glands; the cellular composition of normal and tumor glands, to find signs of invasive growth, cellular nuclear atypia, figures of mitoses; cellular composition of the infiltrate of the own plate of the mucous membrane.
4. Micropreparation "Phlegmonous appendicitis" (stained with hematoxylin and eosin). Pay attention to the localization, prevalence and cellular composition of the infiltrate, the integrity of the mucous membrane, the lymphoid apparatus of the appendix, blood vessels, and the serous membrane.
5. Micropreparation "Nonspecific ulcerative colitis" (stained with hematoxylin and eosin). Pay attention to the localization, prevalence and cellular composition of the infiltrate, the condition of the crypts, the integrity and thickness of the mucous membrane, blood vessels.

6. Micropreparation "Crohn's Disease" (stained with hematoxylin and eosin). Pay attention to the localization, prevalence and cellular composition of the infiltrate; localization, size and cellular composition of granulomas; the integrity and thickness of the mucous membrane; blood vessels.

3) Examine the following electron diffraction patterns using an atlas of micropreparations.

Electron micrograph.

1. Gastrinoma. Mark electron-dense secretory granules in the cytoplasm of the tumor cell.

4) Solve the following situational tasks (cases) using the tutorial.
Situational tasks (cases).

Case 1

Patient S., 57 years old, died of acute cardiovascular failure, history of nausea, repeated vomiting such as "coffee grounds", intense pain in the epigastrium. At autopsy: in the lumen of the stomach about 2 liters of liquid contents such as “coffee grounds”, the intestinal contents are viscous, black (reaction to blood “+”; in the pyloric section of the stomach there is a deep mucosal defect (reaching almost to the serous membrane), with a diameter of 2, 2 cm, with dense thickened edges and a bottom (the phenomena of fibrinoid necrosis are microscopically determined at the bottom); at the bottom of the defect there is a vessel with a diameter of up to 0.2 cm, the lumen of the vessel is covered with a blood clot.

Questions: 1) What disease did the patient have? 2) What is the name of the gastric mucosa defect discovered at autopsy? 3) What is the stage of the disease? 4) What complication did the patient develop? 5) What is the name of the described intestinal contents called?

Case 2

Patient C., 49 years old, for several years noted periodically recurring pain in the epigastrium. The real deterioration came 3 days ago, when, after errors in the diet, intense pain in the epigastrium, nausea, vomiting, bloating appeared. Over the next 3 days, periodic recurrence of abdominal pain was noted; intoxication phenomena increased. He was admitted to the hospital in an extremely serious condition, and died with increasing effects of endotoxemia. At autopsy: in the abdominal cavity, about 1.5 liters of a yellow-greenish, viscous fluid; the peritoneum is dull, hyperemic, with multiple fibrin films impregnated with a greenish, viscous liquid; on the front wall of the stomach a hole with a diameter of 1.2 cm (from the side of the mucous membrane of the stomach - thickening of the edges of the defect).

Questions 1) What disease did the patient have? 2) What is the stage of the disease? 3) What complications developed?

Case 3

In patient S., 52 years old, who died from acute myocardial infarction, an autopsy revealed: acute myocardial infarction of the posterior wall of the left ventricle of the heart (about 7-10 days old); multiple rounded defects with a diameter of 0.3 to 1 cm on the gastric mucosa (microscopically: some of the defects penetrate the muscle layer, inflammatory tissue infiltration around them in the surrounding tissues; some of the other defects are located within the mucous membrane, there is no inflammatory infiltrate around them).

Questions: 1) What are the described defects in the gastric mucosa called? 2) What is the genesis of the formation of these defects? 3) What is the danger of the formation of these defects of the gastric mucosa for the patient?

Case 4

Patient B., 65 years old, died of acute myocardial infarction. At autopsy: acute myocardial infarction of the posterior wall of the left ventricle with rupture and hemipericardium; in the abdominal cavity about 3 l of a clear, slightly yellowish liquid; on the mucous membrane of the stomach in the region of the anterior wall of the body of the stomach - a soft, whitish-gray

formation in the form of a saucer, with a diameter of up to 7 cm, sprouting all layers of the wall of the stomach, with decay in the center; in the liver - multiple rounded foci of decaying whitish-gray tissue. Microscopically: the formation of the stomach is represented by groups of bizarre glands with signs of tissue and cellular atypism.

Questions: 1) What disease of the stomach did the patient have? 2) What is the macroscopic and microscopic form of the described stomach disease? 3) What are the described formations in the liver called? 4) What is the name of the described fluid accumulation in the abdominal cavity?

Case 5

Patient K., 25 years old, became acutely ill: pains appeared in the right ileal region, nausea, single vomiting. 1.5 hours after admission to the hospital, the patient was operated on. An enlarged vermiform appendix (diameter up to 1.2 cm) of bright red color, with a dull serous membrane, which was removed, was found during surgery. Microscopically: there is a swelling of the wall of the appendix, focal hemorrhage, diffuse neutrophil infiltration.

Questions: 1) What disease did the patient have? 2) What is the form of the disease? 3) What other forms of the disease do you know? 4) What are the complications of this disease.

5) Answer the following questions of the current test control.

QUESTIONS OF THE CURRENT TEST CONTROL:

Select all correct answers.

1. Microscopic signs of catarrhal gastritis:

a) moderate edema,

b) slight hyperemia,

c) single leukocytes in their own plate of the mucous membrane,

g) the safety of the surface lining,

e) point hemorrhages.

Select all correct answers.

2. Microscopic signs of severe acute gastritis:

a) ulcers with tight edges,

b) erosion of the mucous membrane,

c) focal hemorrhages,

g) white blood cells above the basement membrane,

e) foci of desquamation of the surface epithelium.

Select all correct answers.

3. The pathogenesis of pernicious anemia in a patient with autoimmune gastritis:

a) the cessation of the production of HC1,

b) production of antibodies to Helicobacter pylori,

c) production of antibodies to parietal cells,

g) production of antibodies to the internal factor,

e) destruction of glands and atrophy of the mucous membrane.

Select all correct answers.

4. Chronic gastritis associated with Helicobacter pylori is characterized by:

a) damage to the antrum,

b) lymphoplasmacytic infiltration,

c) metaplasia

d) multiple erosions,

e) an increase in the mitotic activity of the epithelium of the cervical glands.

Select all correct answers.

5. Epithelial changes in chronic gastritis:

a) atrophy,

b) dysplasia

c) intestinal metaplasia,

g) hyperplasia

e) atypia.

Select all correct answers.

6. A 43-year-old woman with cirrhosis died of posthemorrhagic anemia. Possible bleeding sources:

a) veins of the esophagus,

b) aneurysm of the thoracic aorta,

c) uterine veins,

g) the veins of the cardia of the stomach,

e) aneurysm of the abdominal aorta.

Select all correct answers.

7. Etiological factors of esophagitis:

a) chronic gastritis,

b) reflux of gastric contents into the esophagus,

c) chemical burns,

g) treatment with large doses of antibiotics,

e) uremia.

Select all correct answers.

8. Histological signs of Barrett's esophagus:

a) intestinal metaplasia,
b) gastric metaplasia,
c) epithelial metaplasia,
g) hyperplasia of the epithelium.
Select all correct answers.
9. Edges of chronic gastric ulcer:
a) proximal and distal gently sloping,
b) proximal gently sloping
c) proximal undermined,
g) a distal gentle,
d) distal undermined.
Select all correct answers.
10. Complications of chronic gastric ulcer:
a) bleeding
b) perforation
c) stenosis and obstruction of the stomach,
g) gastroptosis,
e) malignancy.
Select all correct answers.
11. A 63-year-old man was operated on for bleeding from a chronic ulcer of lesser curvature of the stomach. Microscopically found in the bottom of the ulcer:
a) caseous necrosis,
b) fibrinous purulent exudate,
c) fibrous connective tissue,
g) fibrinoid necrosis,
e) purulent hemorrhagic exudate.
Select all correct answers.
12. Ulcerogenic promoters:
a) corticosteroids,
b) stress
c) aspirin
d) smoking
d) increase the tone of the vagus nerve.
Select all correct answers.
13. A young woman with extensive skin burns developed massive stomach bleeding that caused her death. Possible sources of gastric bleeding:
a) acute ulcers of the antrum,
b) a chronic ulcer of lesser curvature,
c) acute ulcers of the bottom and body of the stomach,
d) chronic pyloric ulcers.
Choose one correct answer
14. The background for the development of adenoma of the stomach is gastritis of the form:
a) chronic superficial,
b) acute erosive hemorrhagic,
c) acute fibrinous,
d) chronic with enterolization.
Select all correct answers.
15. Clinical and morphological characteristics of intestinal cancer of the intestinal type:
a) occurs more often before the age of 30,
b) has a high degree of differentiation,
c) develops against a background of chronic gastritis,
d) 2 times more often affects men,

e) develops from metaplastic epithelial cells.

Select all correct answers.

16. *A 40-year-old woman developed epigastric pain and decreased appetite. When fibrogastroscopy in the area of lesser curvature revealed a thickening of the wall of the stomach and its defect, which is regarded as an ulcerative infiltrative cancer. Clinical and morphological characteristics of diffuse type cancer:*

a) develops from epithelial cells,
b) occurs at a relatively young age,
c) histologically - cricoid-cell carcinoma,
g) occurs against a background of chronic gastritis,
e) has a low degree of differentiation.

Choose one correct answer

17. The most important morphological indicator of prognostic value in gastric cancer:

a) histological option
b) macroscopic form,
c) the depth of invasion,
g) mucus formation,
e) secondary changes.

Select all correct answers.

18. Microscopic characteristics of polypous cancer of the stomach:

a) glands of a bizarre shape,
b) cricoid cells,
c) mucus in the lumen of the glands,
g) creeping growth,
e) glands of the intestinal type.

Select all correct answers.

19. Complications of stomach cancer:
a) hemoptysis,
b) the dilatation of the gatekeeper,
c) perforation
g) exhaustion
e) gastric bleeding.

Select all correct answers.

20. The development of appendicitis is promoted by:
a) thrombosis of the arteries of the appendix,
b) clogging with coprolites,
c) blockage with gallstones,
g) compression of the veins of the appendix,
e) the reproduction of microbial flora.

Select all correct answers.

21. Histological signs of superficial appendicitis:
a) edema of the serous membrane,
b) a conical focus of purulent inflammation,
c) diffuse neutrophilic infiltration,
d) plethora
e) minor hemorrhages.

Select all correct answers.

22. Macroscopic signs of phlegmonous appendicitis:
a) normal sizes
b) fibrin filaments on the serous membrane,
c) pus in the lumen of the appendix,
g) thickening of the wall,

e) ulceration of the mucous membrane.

Choose one correct answer

23. A young man with a clinical picture of an acute abdomen underwent appendectomy. An enlarged vermiform appendix, covered with greenish-gray fibrinous-purulent overlays, was removed. Under a microscope, necrosis of the mucous membrane, hemorrhage, and microvascular thrombosis are visible in the wall of the process. Morphological form of appendicitis:

a) simple
b) surface
c) phlegmonous,
g) apostematous,
e) gangrenous.

Select all correct answers.

24. Complications of acute appendicitis:
a) phlebitic abscesses of the pancreas,
b) perforation of the process wall,
c) self-amputation of the appendix,
d) purulent thrombophlebitis of the mesenteric arteries,
d) pylephlebitic abscesses of the liver.

Select all correct answers.

25. The expansion of the lumen of the appendix due to the accumulation of mucous secretions is:

a) empyema
b) hydrocele,
c) mucocele,
g) pneumocele,
e) pseudomyxoma.

Choose one correct answer

26. During histological examination in the apical part of the appendix, a small node was found consisting of isolated complexes of monomorphic atypical cells. During immunohistochemical studies, chromogranin A was visualized in the cytoplasm of cells. Conclusion:

a) adenoma,
b) cystadenoma,
c) mucocele,
g) adenocarcinoma,
e) carcinoid.

Select all correct answers.

27. The causes of fibrinous-purulent peritonitis in acute appendicitis:
a) self-amputation of the appendix,
b) perforation of the process wall,
c) purulent thrombophlebitis of the mesenteric veins,
g) pylephlebitis,
d) liver abscesses.

4. List of recommended literature:

Basic literature:

1. “Basic pathology” Vinay Kumar, Ramzi S. Cotran, Stanley L. Robbins, 1997.

Additional literature:

1. “Pathology. Quick Review and MCQs” Harsh Mohan, 2004.
2. “Textbook of Pathology ” Harsh Mohan, 2002.
3. “General and Systemic Pathology” Joseph Hunter, 2002.
4. “General and Systematic Pathology ” Ed. J.C.E. Underwood – Edinburgh: Churchill Livingstone, 1996 (2th).
5. “Histology for Pathologist” Ed. S.S.Sternberg – Philadelphia: Lippincott Raven Publ, 1997 (2th).
6. “Histopathology. A Color Atlas and Textbook” Damjanov I., McCue P.A. – Baltimore, Philadelphia, London, Paris etc.: Williams and Wilkins, A Waverly Co., 1996.
7. “Muir’s Textbook of Pathology” Eds. R.N.M. MacSween, K. Whaley London: ELBS, 1994 (14th).
8. “Pathology” Eds. Rubin, J.L. Farber – Philadelphia: Lippincott Raven Publ, 1998 (3th).
9. “Pathology Illustrated” Govan A.D.T., Macfarlane P.S., Callander R. – Edinburgh: Churchill Livingstone, 1995 (4th).
10. “Robbins Pathologic Basic of Disease” Eds. R.S.Cotran, V.Kumar, T.Collins – Philadelphia, London, Toronto, Montreal, Sydney, Tokyo: W.B.Saunders Co., 1998 (6th).
11. “Wheater’s Basic Histopathology. A Color Atlas and Text” Burkitt H.G., Stevens A.J.S.L., Young B. – Edinburgh: Churchill Livingstone, 1996 (3th).
12. “Color Atlas of Anatomical Pathology” Cooke R.A., Steward B. – Edinburgh: Churchill Livingstone, 1995 (10th).
13. “General Pathology ” Walter J.B., Talbot I.C. – Edinburgh: Churchill Livingstone, 1996 (7th).
14. “Concise Pathology” Parakrama Chandrasoma, Glive R. Taylor.
15. “Pathology” Virginia A. LiVolsi, Maria J. Merino, John S. J. Brooks, Scott H. Saul, John E. Tomaszewski, 1994.
16. “Short lectures on pathology” Zagoroulko A., 2002
17. “Robbins pathologic basis of diseases” Cotran R., Kumar V., Collins T.
18. “General pathology” Dr. Fatma Hafez, 1979.
19. “Anderson’s Pathology ” Damjanov I., Linder J. – St. Louis: Mosby Inc., 1995 (10th).

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