Ministry of Health of the Republic of Belarus
Educational Institution
“Gomel State Medical University”

Department of Pathological Anatomy

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METHODICAL GUIDELINES

for conducting a practical class
in the academic discipline “Pathological Anatomy”
for students
of the 3rd year, Faculty of International Students (instruction in English),
enrolled in the specialty
7-07-0911-01 “General Medicine”

Topic: “Gastrointestinal tract diseases: gastritis, peptic ulcers, colitis”

Duration: 3 hours

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EDUCATIONAL AND INSTRUCTIONAL GOALS, OBJECTIVES, AND MOTIVATION FOR MASTERING THE TOPIC

Educational Goal:

- To study the definition, classification, etiology, pathogenesis, morphology, complications, and outcomes of gastritis, peptic ulcer disease, and colitis. To analyze acute gastritis (erosive, hemorrhagic, suppurative, phlegmonous) and chronic gastritis (type A – autoimmune, type B – H. pylori-associated, type C – chemical/reactive). To discuss the Sydney classification of chronic gastritis (activity, atrophy, intestinal metaplasia, H. pylori density). To examine peptic ulcer disease: gastric and duodenal ulcers, their etiology (H. pylori, NSAIDs, hyperacidity, stress, smoking), pathogenesis (imbalance between aggressive and protective factors), macroscopic features (round/oval shape, punched-out margins, clean base, radiating mucosal folds), microscopic features (four zones: exudate, fibrinoid necrosis, granulation tissue, fibrous scar), complications (hemorrhage, perforation, penetration, obstruction, malignant transformation – gastric ulcer). To study ulcerative colitis and Crohn disease: differences in location, morphology (continuous vs. skip lesions, mucosal vs. transmural inflammation, crypt abscesses vs. granulomas, pseudopolyps, cobblestone mucosa, fistulas, strictures), complications (toxic megacolon, perforation, carcinoma risk). To examine ischemic colitis and infectious colitis (pseudomembranous colitis due to C. difficile).

Instructional Goal:

- Within the framework of the educational process for this academic discipline, the student must acquire not only theoretical knowledge, practical skills and abilities in the specialty but also develop their personal potential. They should cultivate qualities of responsibility and patriotism, be prepared to actively participate in the economic, socio-cultural, and public life of the country, recognize the social significance of their future professional activity, understand the norms of medical ethics and deontology, and learn to observe academic and labor discipline. In the course of studying the educational material, students should realize the importance of maintaining a healthy lifestyle and, as an example in the future, when performing professional duties, set an example for those around them and their patients.

Objectives: Upon completion of the practical session, the student must:

***Know*:**

- The classification of gastritis and the morphological features of acute and chronic gastritis;
- The etiology, pathogenesis, and macroscopic/microscopic features of peptic ulcers;
- The complications and outcomes of peptic ulcer disease;
- The differences between ulcerative colitis and Crohn disease;
- The morphological features of pseudomembranous colitis;
- The malignant potential of chronic inflammatory bowel disease.

Be able to:

- Distinguish gastric from duodenal ulcers based on macroscopic features;
- Identify H. pylori on histological sections (special stains: Giemsa, Warthin-Starry, immunohistochemistry);
- Recognize crypt abscesses, granulomas, and pseudopolyps in inflammatory bowel disease;
- Diagnose pathological processes and diseases based on the description of macroscopic and microscopic changes in organs and tissues of the body.

Master:

- Basic techniques for working with a microscope;
- Skills in clinical-anatomical analysis;
- The fundamentals of synthetic generalization of morphological diagnostic signs of diseases and their correct interpretation in cause-and-effect relationships.

Motivation for Mastering the Topic:

Diseases of the gastrointestinal tract, including gastritis, peptic ulcers, and inflammatory bowel disease, are extremely common in clinical practice. Peptic ulcer disease affects millions worldwide, with significant morbidity from complications such as hemorrhage and perforation. Inflammatory bowel disease (ulcerative colitis and Crohn disease) often affects young adults and requires lifelong management, with an increased risk of colorectal carcinoma. Knowledge of the morphological features of these diseases is essential for gastroenterologists, surgeons, internists, and pathologists for accurate diagnosis, treatment planning, and cancer surveillance.

QUESTIONS

1. Diseases of esophagus. Esophagitis. Definition. Etiology. Pathogenesis. Morphology. Complications and outcomes.
2. Gastritis. Definition. Etiology. Pathogenesis. Classification. Morphology. Complications and outcomes.
3. Peptic ulcer disease: gastric ulcer, duodenal ulcer. Definition. Etiology. Pathogenesis. Classification. Morphology. Complications and outcomes.
4. Appendicitis. Definition. Etiology. Pathogenesis. Classification. Morphology. Complications and outcomes.
5. Inflammatory bowel disease (IBD): ulcerative colitis, Crohn's disease. Differential diagnosis. Definition. Etiology. Pathogenesis. Classification. Morphology. Complications and outcomes.

COURSE OF THE SESSION

Diseases of esophagus

These are disorders in which there is motor dysfunction of the esophagus, manifested clinically by dysphagia.

These include **achalasia**, **hiatus hernia** and **esophageal diverticula**.

Achalasia of the esophagus is a neuromuscular dysfunction due to which the cardiac sphincter fails to relax during swallowing and results in progressive dysphagia and dilatation of the esophagus.

Etiology. The exact etiology is not known. It may be congenital. Emotional stress has been believed to contribute to the onset of the disease. Some investigators have demonstrated total absence of nerve fibers and ganglia of Auerbach's plexus in the terminal few cm of the esophagus in achalasia.

Pathologic changes. There is dilatation above the short contracted terminal segment of the esophagus. Muscularis propria of the wall may be of normal thickness, hypertrophied as a result of obstruction, or thinned out due to dilatation. Secondary esophagitis may supervene and cause esophageal ulceration and hematemesis.

Hiatus hernia

Hiatus hernia is the herniation or **protrusion of the stomach** through the esophageal hiatus of the diaphragm. Esophageal hiatal hernia is the cause of **diaphragmatic hernia** in 98% of cases. The condition is diagnosed radiologically in about 5% of apparently normal asymptomatic individuals. In symptomatic cases, especially the elderly women, the clinical features are heartburn (retrosternal burning sensation) and regurgitation of gastric juice into the mouth, both of which are worsened due to heavy work, lifting weights and excessive bending.

Etiology. The basic defect is the failure of the muscle fibers of the diaphragm that form the margin of the esophageal hiatus. This occurs due to shortening of the esophagus which may be congenital or acquired.

Esophageal diverticula

Diverticula are the **outpouchings of esophageal wall** at the point of weakness. They may be congenital or acquired.

Congenital diverticula occur either at the upper end of the esophagus or at the bifurcation of trachea.

Acquired diverticula may be of 2 types:

1) **Pulsion** (Zenker's) type– is seen in the region of hypopharynx and occurs due to esophageal obstruction such as due to chronic esophagitis, carcinoma etc. The mucosa and submucosa herniate through the weakened area or through defect in the muscularis propria.

b) **Traction** type – occurs in the lower third of esophagus from contraction of fibrous tissue such as from pleural adhesions, scar tissue of healed tuberculous lesions in the hilum, silicosis etc.

Complications of diverticula include **obstruction, infection, perforation, haemorrhage and carcinoma**.

Massive hematemesis

Hematemesis (**vomiting of blood**) may occur due to vascular lesions.

1. **Esophageal varices** are tortuous, dilated and engorged esophageal veins, seen along the longitudinal axis of esophagus. They occur as a result of elevated pressure in the portal venous system, most commonly in **cirrhosis of the liver** and after hepatic vein thrombosis (**Budd-Chiari syndrome**). The lesions occur as a result of bypassing of portal venous blood from the liver to the esophageal venous plexus. The increased venous pressure in the superficial veins of the esophagus may result in ulceration and massive bleeding.

2. **Mallory-Weiss syndrome**. In this condition, esophageal lacerations and bleeding may occur following minor trauma such as reflex relaxation of the muscularis resulting in overstretching and subsequent tearing at the esophago-gastric junction. The condition is common in chronic alcoholics who develop vomiting and hematemesis.

3. **Rupture of the esophagus** may occur following trauma, during esophagoscopy, indirect injury (e.g. due to sudden acceleration and deceleration of the body) and spontaneous rupture (e.g. after overeating, extensive aerophagy, etc.).

4. Other causes: bursting of **aortic aneurysm** into the lumen of esophagus, **hiatus hernia**, **esophageal cancer**, purpuras, hemophilia.

Reflux esophagitis

Reflux of the gastric juice is the **commonest cause of esophagitis**.

Gastroesophageal reflux, to an extent, may occur in normal healthy individuals after meals and in early pregnancy. However, in some clinical conditions, the gastroesophageal reflux is excessive, resulting in inflammation of the lower esophagus:

1) Sliding hiatus hernia, 2) Chronic gastric and duodenal ulcers, 3) Nasogastric intubation, 4) Persistent vomiting, 5) Surgical vagotomy, 6) Neuropathy in alcoholics, diabetics, 7) Esophagogastrostomy.

Endoscopically, the **demarcation** between normal squamous and columnar epithelium at the junctional mucosa **is lost**. The affected distal esophageal mucosa is red, erythematous, friable and bleeds on touch. In advanced cases, there are features of chronic disease such as nodularity, strictures, ulcerations and erosions. Microscopically, the reflux changes in the distal esophagus include **basal cell hyperplasia** and deep elongation of the papillae touching close to the surface epithelium. Inflammatory changes vary according to the stage of the disease. In early stage, mucosa and submucosa are infiltrated by **neutrophils**; in chronic stage, there is **lymphocytic infiltration and fibrosis** of all the layers of the esophageal wall.

Barrett's esophagus

This is a condition in which, following reflux esophagitis, the **squamous epithelium** of the lower esophagus is replaced by **columnar epithelium**. The condition is seen more commonly in later age and is caused by factors producing gastroesophageal reflux disease.

Barrett's esophagus is a **premalignant condition** in which changes of dysplasia and carcinoma have been reported.

Pathologic changes. Endoscopically, the affected area is red and velvety. Microscopically, the most common finding is the replacement of squamous epithelium by **metaplastic** columnar cells. Inflammatory and dysplastic changes of the columnar epithelium or glands may be present.

Longstanding cases of Barrett's esophagus have 5-8% risk of developing carcinoma of the esophagus.

Acute gastritis

Acute gastritis most often develops after exposure to various **chemicals** (eg, alcohol, junk food) or certain **drugs** (especially non-steroidal anti-inflammatory drugs). These substances cause rapid **exfoliation** (desquamation) of epithelial cells and a **decrease in mucus secretion**, which is accompanied by a decrease in the function of the protective barrier against the action of acid. The pathogenesis of this process is a decrease in the synthesis of prostaglandins. The consumption of spicy, cold or hot food can also play a role.

Acute neutrophilic gastritis (in which the main morphological feature is infiltration with leukocytes) is characteristic of the primary response to infection caused by Helicobacter pylori. This condition is temporary, which in most people proceeds without clinical manifestations and after 3-4 weeks turns into chronic gastritis. Only in a small number of patients the infection spontaneously disappears and recovery occurs. Other infections, such as salmonella, staphylococcus, etc., can also lead to the development of acute gastritis.

Acute gastritis

Acute gastritis can develop under the influence of toxic products of endogenous origin, for example, in **uremia**.

According to the area of damage, we can distinguish:

- acute diffuse gastritis;
- acute focal gastritis (can be predominantly fundic, antral, pyloric, pyloroduodenal).

Depending on the severity of the lesion, changes in the mucosa range from **vasodilation and edema** of the lamina propria to **erosion and hemorrhage**. Erosions in acute gastritis are usually multiple, so bleeding from them can be very dangerous. However, there is usually a rapid (within 24-48 hours) healing by regeneration. With frequent recurrences of acute gastritis, chronic gastritis may develop.

Chronic gastritis

Chronic gastritis is the commonest histological change observed in biopsies from the stomach. The microscopic change is usually **poorly correlated to the symptomatology**, as the change is observed in about 35% of endoscopically normal mucosal biopsies. The condition occurs more frequently with advancing age; average age for symptomatic chronic gastritis being 45 years which corresponds well with the age incidence of gastric ulcer.

Etiopathogenesis of chronic gastritis

In the absence of clear etiology of chronic gastritis, a number of etiologic factors have been implicated. All the causative factors of acute gastritis described above may result in chronic gastritis too. Recurrent attacks of acute gastritis may result in chronic gastritis. Some other causes are as under:

1. **Reflux of duodenal contents** into the stomach, especially in cases who have undergone surgical intervention in the region of pylorus.

2. Associated disease of stomach and duodenum, such as **gastric or duodenal ulcer**, gastric carcinoma.

3. **Chronic hypochromic anemia**, especially associated with atrophic gastritis.

4. **Immunological factors** such as autoantibodies to gastric parietal cells in atrophic gastritis and autoantibodies against intrinsic factor.

The mechanism of chronic gastric injury by any of the etiologic agents is by cytotoxic effect of the injurious agent on the gastric mucosal epithelium, thus breaking the barrier and then inciting the inflammatory response.

Classification of chronic gastritis

The current classification of gastritis is based on time course (acute versus chronic), histological features, anatomic distribution, and underlying pathological mechanisms.

1. Type A gastritis (**autoimmune** gastritis). Type A gastritis involves mainly the **body-fundic mucosa**. It is also called autoimmune gastritis due to the presence of circulating antibodies and is sometimes associated with other autoimmune diseases such as Hashimoto's thyroiditis and Addison's disease. As a result of the **antibodies against parietal cells** and **intrinsic factor**, there is depletion of parietal cells and impaired secretion of intrinsic factor. These changes may lead to significant **gastric atrophy** where intestinal metaplasia may occur, and a small proportion of these patients may develop pernicious anemia. Due to depletion of gastric acid-producing mucosal area, there is hypo- or achlorhydria, and hyperplasia of gastrin-producing G cells in the antrum resulting in hypergastrinemia.

2. Type B gastritis (H. pylori-related, or **bacterial**). Type B gastritis is the most common subtype. It mainly involves the region of **antral mucosa** and is more common. It is also called hypersecretory gastritis due to **excessive secretion of acid**, commonly due to infection with H. pylori. These patients may have associated duodenal or gastric ulcer. Unlike type A gastritis, this form of gastritis has no autoimmune basis nor has association with other autoimmune diseases.

3. Type C gastritis (**chemical** gastritis). Type C gastritis is caused by chemical injury due to **bile reflux and drugs**. Especially nonsteroidal anti-inflammatory drugs (NSAIDSs) like ibuprofen are implicated. Histological changes can vary and include edema, foveolar hyperplasia of antral mucosa, a mild chronic inflammation, vascular congestion, reactive epithelial changes, and smooth muscle hyperplasia in the lamina propria. This type of gastritis is not generally associated with an increased risk of gastric cancer.

Pathologic changes of chronic gastritis

Macroscopically, the features of all forms of gastritis are inconclusive. The gastric mucosa may be **normal**, **atrophied**, or **edematous**. Chronic gastritis is essentially a histological diagnosis.

Chronic Superficial Gastritis. As the name suggests there is inflammatory infiltrate consisting of plasma cells and lymphocytes in the superficial layer of the gastric mucosa, but there are no histological changes in the deep layer of mucosa containing gastric glands. Chronic superficial gastritis may resolve completely or may progress to chronic atrophic gastritis.

Chronic Atrophic Gastritis. In this type of gastritis, there is inflammatory cell infiltrate in the deeper layer of the mucosa and atrophy of the epithelial elements including destruction and metaplasia of the glands.

Intestinal metaplasia involves antral mucosa more frequently. Characteristic histologic feature is the presence of intestinal type goblet cells; Paneth cells and endocrine cells may also be present. Parietal cells are very few or absent. Intestinal metaplasia, focal or extensive, in atrophic gastritis is significant because its incidence is high in populations having high prevalence rate of gastric cancer like in Japan.

Rare forms of gastritis

With **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 mucosal folds. The etiology and relationship with Helicobacter-associated gastritis has not been established.

Eosinophilic gastritis is characterized by mucosal edema 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.

Peptic ulcer disease

Peptic ulceration is a violation of the integrity of the epithelial cover and underlying tissues of the digestive tract as a result of damage to them by acid and pepsin. According to the clinical course, ulcers are divided into acute and chronic.

The cause of the development of **acute ulcers** can be:

1.**Severe course of acute gastritis**. Deep spread of erosions in acute gastritis occurs usually with the use of NSAID or alcohol, during treatment with corticosteroids, which leads to the appearance of deep ulcers.

2.**Strong stress**. Acute ulcers can occur as a result of various factors that lead to stress, for example, with extensive burns, brain injuries. In this case, ulcers are formed as a result of **mucosal ischemia**, which leads to a decrease in its resistance to acid.

3.Pronounced **increase in acidity**. Increased acidity, for example, in patients with gastrin -secreting tumors (**Zollinger-Ellison syndrome**), leads to the formation of multiple ulcers in the antrum, duodenum and even jejunum.

Chronic ulcers

The cause of the development of **chronic ulcers** can be:

1.**Helicobacter pylori infection**.

2.**Chemical exposures**, including steroid drugs and non-steroidal anti-inflammatory drugs.

3.**Chronic distress syndrome**.

Chronic peptic ulcers most often form at the junction of different types of mucous membranes. So, for example, in the stomach, ulcers are observed at the point of transition of the body to the antrum , in the duodenum - in the proximal area on the border with the pylorus, in the esophagus - in the stratified epithelium in front of the esophageal-gastric junction, postoperative ulcers are localized in the stoma (in the anastomosis).

That is, **ulcers appear in those places where acid and pepsin come into contact with an unprotected mucosa**.

Pathogenesis of chronic ulcers

For many years it was thought that the cause of peptic ulcers was **hyperacidity**. However, in many cases, patients observed normal and even reduced acidity of gastric juice. Conversely, ulceration was rarely observed in patients with hyperacidity. In addition, during treatment with antacids (drugs that reduce acidity), relapses were observed in many cases.

This led to the idea that it is **not acidity that plays the main role** in the development of ulcers, but the **ratio of aggression factors and mucosal defense factors**. It is believed that in the genesis of duodenal ulcer, the main role is played by the increase in aggression factors, and in the development of gastric ulcer, the decrease in protective factors comes first. With a decrease in the latter, it is possible to develop ulcers even with low acidity.

Gastric juice is highly acidic (pH < 2), so the unprotected mucosa quickly undergoes **autodigestion**. Protection of the mucous membrane is carried out by the **muco-bicarbonate barrier** and the **surface epithelium**. The mucosal barrier plays a major role in protecting the mucosa. The superficial and pit cells of the mucosa secrete viscous neutral glycoproteins that form a mucus layer on the mucosal surface. Mucus itself has anti-acid properties, however, its protective power is enhanced by the presence of buffering ingredients, mainly bicarbonate ions.

The surface epithelium forms the second line of defense; to ensure this function, the correct functioning of both the apical membrane, which prevents the transport of ions, and the synthetic apparatus that produces bicarbonates, are necessary. Both of these functions depend on the **mucosal blood supply**.

Ulceration occurs as a result of **either violation and destruction of the mucous barrier, or violation of the integrity of the epithelium**. As a result of bile reflux, the mucous barrier is easily destroyed by its components. **Acid and bile together destroy the surface epithelium**, increasing the permeability and vulnerability of the mucous membrane. This leads to congestion and swelling in the lamina propria, which is seen in reflux gastritis.

The epithelial barrier may also be disrupted by the use of NSAIDs, as they **disrupt the synthesis of prostaglandins**, which normally protect the epithelium.

Helicobacter pylori infection also plays a significant role in the destruction of the epithelium, in which both cytotoxins and ammonium ions and an inflammatory reaction have a destructive effect.

Duodenal ulcer

Increased acidity plays a major role in the development of duodenal ulcers. In half of the patients **hypersecretion of acid** is observed, however, even with normal stomach acidity, the daily secretion cycle can be disturbed: there is no decrease in secretion at night. It is also known that when stimulated with gastrin in patients infected with Helicobacter pylori, acid synthesis is 2-6 times higher than in uninfected.

Factors damaging the anti-acid defense in the stomach usually do not affect the duodenum: Helicobacter pylori does not colonize the duodenal mucosa, the mucosa is resistant to the action of bile and alkaline ions of pancreatic juice, drugs are significantly diluted and absorbed before entering the intestine. However, Helicobacter pylori affects ulcer formation, because **infection promotes gastric hypersecretion**, which causes the development of **gastric metaplasia in the duodenum**, and then **colonization of the metaplastic epithelium of Helicobacter pylori** occurs, which leads to the development of chronic inflammation, which also provokes ulceration.

Morphological changes. Macroscopically, chronic ulcers usually have a round or oval shape. Their sizes usually do not exceed 2 cm in diameter. The depth of the ulcer is different, sometimes it reaches the serous membrane. The edges of the ulcer are clear, dense and rise above the normal surface.

Chronic peptic ulcers

Microscopically, chronic peptic ulcers have **4 histological zones**. From within outside, these are as under:

1. **Necrotic zone** – lies in the floor of the ulcer and is composed of fibrinous exudate containing necrotic debris and a few leucocytes.

2. **Superficial exudative zone** – lies underneath the necrotic zone. The tissue elements here show coagulative necrosis giving eosinophilic, smudgy appearance with nuclear debris.

3. **Granulation tissue zone** – is seen merging into the necrotic zone. It is composed of nonspecific inflammatory infiltrate and proliferating capillaries.

4. **Zone of cicatrization** – is seen merging into thick layer of granulation tissue. It is composed of dense fibrocollagenic scar tissue over which granulation tissue rests. Thrombosed or sclerotic arteries may cross the ulcer which on erosion haemorrhage.

Complications of peptic ulcers

Acute and subacute peptic ulcers usually heal without leaving any visible scar. However, healing of chronic, larger and deeper ulcers may result in complications:

1) **Haemorrhage**. Chronic blood loss may result in iron deficiency anemia. Severe bleeding may cause 'coffee ground' vomitus or melaena. A penetrating

chronic ulcer may erode a major artery (e.g. left gastric, gastroduodenal or splenic artery) and cause a massive and severe hematemesis and sometimes death.

2) **Perforation**. A perforated peptic ulcer is an acute abdominal emergency. Perforation occurs more commonly in chronic duodenal ulcers than chronic gastric ulcers. Following sequelae may result:

a) On perforation the contents escape into the peritoneal cavity, causing acute peritonitis.

b) Air escapes from the stomach and lies between the liver and the diaphragm giving the characteristic radiological appearance.

c) Subphrenic abscess between the liver and the diaphragm may develop due to infection.

d) Perforation may extend to involve the adjacent organs e.g. the liver and pancreas. This is known as penetration.

3) **Malignization**. Sometimes chronic gastric ulcers may transform into gastric carcinoma.

Appendicitis is a **primary inflammation of the appendix** of the caecum with a peculiar clinical syndrome. Therefore, not every inflammation of the appendix in clinical and anatomical terms should be considered as appendicitis (for example, when the inflammatory process spreads from nearby organs, when it is affected by tuberculosis, etc.)

There are two clinical and anatomical forms of appendicitis: acute and chronic.

Acute appendicitis is the most common cause of emergency operations in surgery. It occurs in all age groups, but is most common in adolescents.

The most common **cause** of acute appendicitis is **obstruction of the lumen** of the appendix by fecal concretions or enlarged submucosa as a result of lymphoid hyperplasia. At the same time, in the distal segment, there is an increased reproduction of microorganisms, such as Escherichia coli, Streptococcus faecalis and anaerobic bacteria. These bacteria then invade the mucosa and other linings of the appendix, causing acute inflammation.

Pathological changes. It is customary to distinguish the following main morphological forms of acute appendicitis:

1) **simple**;

2) **destructive** (which is divided into **phlegmonous**, **ulcerative**, **gangrenous**).

All these forms, in essence, are a morphological reflection of the **phases of an acute inflammatory process** in the appendix, which ultimately ends in necrosis. The duration of this process is 2-4 days.

Acute **simple appendicitis** is characterized by the presence of stasis in capillaries and venules, edema, hemorrhages, marginal standing of leukocytes, leukodiapedesis, most often in the distal appendix. Outwardly, the appendix looks normal, however, the diagnosis is confirmed by histological examination.

Changes of simple appendicitis may be reversible. However, usually they progress, and destructive appendicitis develops.

By the end of the first day, the neutrophilic infiltrate extends to the entire thickness of the appendix wall (**phlegmonous appendicitis**). Macroscopically, the inflamed appendix looks edematous and red, its surface is often covered with fibrinous-purulent exudate.

Sometimes, against this background, small multiple abscesses are detected, in which case such appendicitis is designated as **apostematous**.

Acute inflammation of the mucous membrane leads to the formation of ulcers and inflammation of the muscle layer - this is **phlegmonous-ulcerative appendicitis**.

Purulent-destructive changes are completed by the development of **gangrenous appendicitis**. The process in this form is thickened, its wall is gray-dirty in color, structureless with a fetid odor, pus is released from the lumen. Microscopically, there are extensive foci of necrosis with colonies of microbes, hemorrhages, blood clots in the vessels.

Complications of acute appendicitis. The local spread of the inflammatory process can lead to the **involvement of periappendicular tissues**, which is manifested by the development of an **abscess**. Perforation may result in peritonitis. Distant abscesses (eg, in the rectovesical and subdiaphragmatic spaces) may also form. Very rarely, inflammation spreads through the veins, which leads to the development of portal vein **thrombophlebitis** with the formation of multiple pylephlebitic liver abscesses.

Chronic appendicitis is characterized by the presence of **sclerotic and atrophic processes**, against which signs of inflammatory and destructive changes can be detected. There are adhesions with surrounding tissues. With cicatricial obliteration of the proximal section, serous fluid can accumulate in the lumen of the appendix and a cyst is formed. If the contents of the cyst are represented by mucus, such a complication is referred to as **appendiceal mucocele**.

Inflammatory bowel disease

The term inflammatory bowel disease (**IBD**) is commonly used to include 2 idiopathic bowel diseases having many similarities but the conditions usually have distinctive morphological appearance. These 2 conditions are **Crohn's disease** (regional enteritis) and **ulcerative colitis**:

1. **Crohn's disease** (or regional enteritis) is an idiopathic chronic ulcerative IBD, characterized by transmural, non-caseating granulomatous inflammation, affecting most commonly the segment of terminal ileum and/or colon, though any part of the gastrointestinal tract may be involved.

2. **Ulcerative colitis** is an idiopathic form of acute and chronic ulcero-inflammatory colitis affecting chiefly the mucosa and submucosa of the rectum and descending colon, though sometimes it may involve the entire length of large bowel.

Both these disorders primarily affect the bowel but **may have systemic involvement** in the form of polyarthritis, uveitis, ankylosing spondylitis, skin lesions and hepatic involvement. Both diseases can occur at any age but are more common in 2nd and 3rd decades of life. Females are affected slightly more often.

Crohn's disease may involve **any portion of the gastrointestinal tract** but affects most commonly 15-25 cms of the terminal ileum which may extend into the caecum and sometimes into the ascending colon.

Grossly, the characteristic feature is **multiple**, well-demarcated **segmental bowel involvement** with intervening **uninvolved 'skip areas'**. The wall of the affected bowel segment is thick and hard, resembling a 'hose pipe'.

Serosa may be studded with minute granulomas. The lumen of the affected segment is markedly narrowed.

The mucosa shows 'serpiginous ulcers', while intervening surviving mucosa is swollen giving '**cobblestone appearance**'.

There may be deep fissuring into the bowel wall. Histologically, the characteristic features are:

1. **Transmural inflammatory cell infiltrate** consisting of chronic inflammatory cells (lymphocytes, plasma cells and macrophages) is the classical microscopic feature.
2. Non-caseating, **sarcoid-like granulomas** are present in all the layers of the affected bowel wall in 60% of cases and may even be seen in the regional lymph nodes.
3. There is **patchy ulceration of the mucosa** which may take the form of deep fissures, accompanied by inflammatory infiltrate of lymphocytes and plasma cells.
4. **Edema** and **lymphoid follicles hyperplasia** in the submucosa.
5. In chronic cases, **fibrosis** becomes increasingly prominent in all the layers disrupting muscular layer.

Complications of Crohn's disease are:

1. **Malabsorption** due to impaired absorption of fat, vitamin B12, proteins and electrolytes from the diseased small bowel.
2. **Fistula formation** may occur in long-standing cases. These may be internal fistulae between the loops of the intestine, or external fistulae such as enterocutaneous, rectal and anal fistulae.
3. **Stricture formation** may occur in chronic cases due to extensive fibrosis in the affected bowel wall.
4. **Carcinoma** may develop very rarely in the small intestine as a late complication of Crohn's disease.

Ulcerative colitis

Classically, ulcerative colitis **begins in the rectum**, and in continuity extends upwards into the sigmoid colon, descending colon, transverse colon, and sometimes may involve the entire colon. The colonic contents may rarely backflow into the terminal ileum in continuity, causing 'back-wash ileitis' in about 10% of cases.

Grossly, the characteristic feature is the **continuous involvement** of rectum and colon **without any uninvolved skip areas** as seen in Crohn's disease. The appearance of colon may vary depending upon the stage and intensity of the disease because of remissions and exacerbations.

Mucosa shows **linear and superficial ulcers**, usually **not penetrating the muscular layer**. The intervening intact mucosa may form inflammatory 'pseudopolyps.' The muscle layer is thickened due to contraction, producing shortening and narrowing of the affected colon with loss of normal haustral folds giving 'garden-hose appearance'.

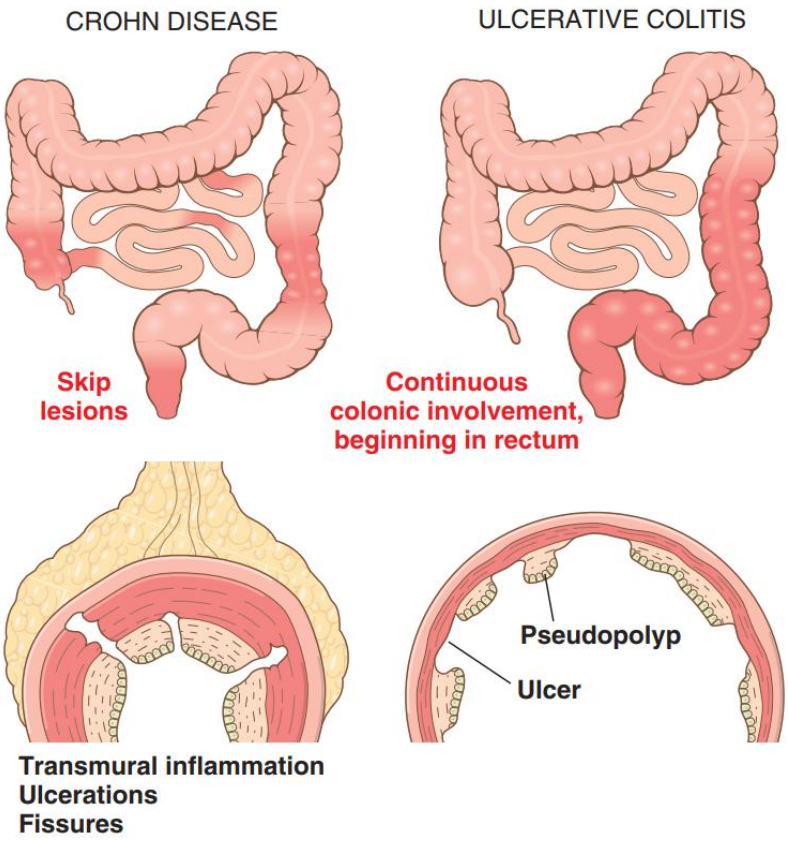
Histologically, ulcerative colitis because of remission and exacerbations, is characterized by alternating '**active disease process**' and '**resolving colitis**'. The changes in the 'active disease process' are:

1. **Crypt distortion**, **cryptitis** and focal accumulations of neutrophils forming crypt abscesses.
2. Marked congestion, dilatation and **hemorrhages** from mucosal capillaries.
3. **Superficial mucosal ulcerations**, usually not penetrating into the muscle layer, except in severe cases, and is accompanied by nonspecific inflammatory cell infiltrate of lymphocytes, plasma cells, neutrophils, some eosinophils and mast cells in the lamina propria.
4. Goblet cells are markedly diminished in cases of active disease.
5. Areas of **mucosal regeneration and mucodepletion** of lining cells.
6. In long-standing cases, epithelial **cytologic atypia** ranging from mild to severe dysplasia and sometimes developing into carcinoma in-situ.

Complications of ulcerative colitis are as follows:

1. **Toxic megacolon** (Fulminant colitis) is the acute fulminating colitis in which the affected colon is thin-walled and dilated and is prone to perforation and fecal peritonitis. There is deep penetration of the inflammatory cell infiltrate into muscle layer which is disrupted.
2. **Perianal fistula formation** may occur rarely.
3. **Carcinoma** may develop in long-standing cases of ulcerative colitis of more than 10 years duration.
4. **Stricture formation** almost never occurs in ulcerative colitis.

Crohn disease vs Ulcerative colitis


CROHN DISEASE
ULCERATIVE COLITIS
Skip lesions
Continuous colonic involvement, beginning in rectum
Pseudopolyp
Ulcer
Transmural inflammation
Ulcerations
Fissures

INFORMATIONAL AND METHODOLOGICAL PART

MAIN LITERATURE

1. Pathomorphology : textbook / ed. by I.V. Sorokina, V.D. Markovskyi, D.I. Halata. - 2nd ed. - Kyiv : AUS Medicine Publishing, 2020. - 327 p., [10] col. inserts : tab.

2. Klatt, E. C. Robbins and Cotran Atlas of Pathology : [with Student Consult Online Access] / Edward C. Klatt. - third edition. - Philadelphia : Elsevier, 2015. - 587 p. : il.

3. Robbins Basic pathology / [ed. by] Vinay Kumar, Abul K. Abbas, Jon C. Aster. - 9th ed. - [Philadelphia] : Elsevier ; Saunders. - 910 p. : ill., scheme, tab.

ADDITIONAL LITERATURE

1. Sobotta. Atlas of human anatomy. [V. 1]. General anatomy and musculoskeletal system / ed. by F. Paulsen, J. Waschke. - 15th ed. - München : Elsevier. Urban & Fischer, 2011. - 400 p. : ill., col. ill.

2. Sobotta. Atlas of human anatomy. [V. 2]. Internal organs / ed. by F. Paulsen, J. Waschke. - 15th ed. - München : Elsevier. Urban & Fischer, 2011. - 259 p. : ill., col. ill.

3. Sobotta. Atlas of human anatomy. [V. 3]. Head, neck, and neuroanatomy / ed. by F. Paulsen, J. Waschke. - 15th ed. - München : Elsevier. Urban & Fischer, 2011. - 370 p. : ill., col. ill.

4. Weir & Abrahams' imaging atlas of human anatomy / ed.: J. D. Spratt [and oth.] ; consultant eds.: J. Weir, P. H. Abrahams. – 5th ed. - [S.l.] : Elsevier, 2017. - xvi, 263 p. : ill.

NORMATIVE LEGAL ACTS AND DOCUMENTS

1. Order of the Ministry of Health of the Republic of Belarus No. 111 of 06/01/1993 "On further improvement of the pathological service of the Republic of Belarus"

Access mode: http://patan.by/menyu/administrativnyie-proczeduryi.html - The date fccess: 20.03.2022.

ELECTRONIC DATABASES

1. Physician's consultant. Electronic Medical Library = Consultant of the doctor. Electronic medical library [Electronic resource] / Publishing group "GEOTAR-Media", LLC "IPUZ". – Access mode: http://www.rosmedlib.ru/ . – Access date: 03/15/2022.

2. Springer Link [Electronic resource] / Springer International Publishing AG. – Access mode: https:link.springer.com. – Date of access: 03/15/2022.