ПАТОЛОГИЧЕСКАЯ АНАТОМИЯ
ВНУТРЕННИХ И ИНФЕКЦИОННЫХ БОЛЕЗНЕЙ
(учебно-методическое пособие для практических занятий
для студентов медицинских ВУЗов, обучающихся
на английском языке)

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Учебно-методическое пособие для практических занятий по патологической анатомии, рекомендуемое для студентов 3-го курса, обучающихся на английском языке в высших медицинских образовательных учреждениях

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INTRODUCTION

The study of the structural bases of diseases and mechanisms of their development (pathogenesis) is the subject of pathological anatomy at medical high schools.

The course includes the study of the following points:
- morphology of diseases during every stage of their development (morphogenesis),
- structural principles of recovery,
- complications,
- outcomes and results of illnesses;

Pathological anatomy learning course study the pathomorphology of internal and infectious diseases.

Every student has to learn to correlate morphological and clinical manifestations of diseases in every stage of their development. It allows to master the skills of clinico-anatomical and physiological mentality and synthesis of diagnostic criterias of illnesses.

The practical academic manual worked out in accordance with academic curriculum contains topics, plans, descriptions of grosses and slides, clinical cases and control questions.

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UNIT 1
ATHEROSCLEROSIS, PRIMARY ESSENTIAL HYPERTENSION,
ISHEMIC DISEASE OF THE HEART

Grosses:

1. **Myocardial hyper trophy**. Describe this preparation. Explain the reason of thickening of heart ventricles (or left ventricles).

2. **Atherosclerosis of aorta**. Describe this preparation. Estimate the surface of intima, presence or absence of thrombus, aneurism.

3. **Myocardial infarction**. It is the infarction of 2-4 days duration. Describe this gross. Describe shape, color and density of focus. Give the definition of infarction.

4. **Primary shrunken kidney**. Describe the dimensions, density and character of surface.

5. **Atherosclerosis of the brain vessels**. Describe this preparation. Pay your attention to twisting and irregularly thickening of vessel's wall.

6. **Chronic myocardial aneurism**. Describe thickness, density and color of hastrum the wall of cardiac left ventricle. Tell, please, in what stage of myocardial infarction has chronic aneurism been developed and what was on this place before?

7. **Gangrene of the foot**. What types of gangrene do you know? What kind of atherosclerotic gangrene can you find more often? Write down the descriptions of all grosses in your copybooks.

**Slide №141 (D)**
Atherosclerosis of aorta
(hematoxylin and eosin)

Small magnification: Find features of atherosclerotic plaque in the aorta. The structure demonstrates a fibrous cap and the central lipid core of the plaque with typical cholesterol clefts in view as oval-stretched emptinesses. Sometimes regions of calcification may occur in the plaque.

Large magnification: You can see empty drops of cholesterol under the fibrous cap on pink homogeneous background of necrosis. In addition, look at the media - it is infiltrated by cholesterol and elastic membranes are destroyed.

**Slide № 201**
Atherosclerosis of coronary artery
(hematoxylin and eosin)

Small magnification: there is cross section of coronary artery. Vessel's wall is irregularly thickened and vessel's lumen is contracted because in large magnification there is deposit of lipid as oval-stretch emptinesses, around it there is the development of connective tissue. You may see necrosis – pink homogeneous areas. Also in vessel's wall there may be the deposit of calcium as small blue-violet particles. Elastic membranes are destroyed. The muscle layer is very thin (it's athrophy).

Please, write down the most important points near the drawing of this slide:
1. deposit of lipids,
2. connective tissue development (fibrosis),
3. necrosis,
4. atrophy of muscle layer.

Draw the coronary artery's wall with the atherosclerotic plaque.

**Slide № 143**

**Acute myocardial infarction**
(hematoxylin and eosin)

Small magnification: there is myocardium with bright-pink homogeneous zone, where in large magnification nuclears are absent. Around necrotised zone leukocyte infiltration is seen. In distinction from that zone in normal myocardium nuclears are present and cell's limits are clear.

Please, write down the next points near the drawing of this slide:
1. necrotic zone,
2. zone of reactive inflammation (leukocyte's infiltration),
3. normal myocardium.

**Slide № 97**

**Postinfarctive myocardial sclerosis**
(hematoxylin and eosin)

There is myocardium. There are areas of diffuse myocardial fibrosis, especially around the small blood vessels in the interstitial tissue of the myocardium. Also there is a large area of connective tissue in the place where earlier there was infarction zone.

Please, write down the next points near the drawing of this slide:
1. small-focal and diffuse cardiosclerosis (caused by atherosclerosis of coronary arteries),
2. large-focal cardiosclerosis (post-necrotic).

**Slide № 140**

**Arteriolsclerotic kidney**
(kidney in case of primary essential hypertension, primary shrunken kidney)
(hematoxylin and eosin)

Small magnification: pay your attention to the cortical layer of the kidney.
There are primarily diffused vascular changes, which produce parenchymatous changes secondarily as a result of ischemia. There is a variable degree of atrophy of parenchyma. They include the following changes:
1. glomerulars: a) hyalinosis,
   b) sclerosis,
   c) athrophy,
   d) local hypertrophy,
2. arteriolas: a) hyalinosis,
   b) sclerosis,
3. tubuli: athrophy,
4. arteries of muscle type: myoelastofibrosis (dimensions of it as to glomerular or a little larger),
5. sclerosis of stroma.

Every lesson you have to deside some problems (tasks). For example today there are several clinical cases:

1. The corpse of the patient, for a long time suffered from arterial hypertension, has been brought for the dissection. The hyalinosis of arterioles of kidney, brain and other organs was found at histological investigation.

   1. How does a blood vessel with hyalinosis look like?
2. What kind of blood vessels is hyalinosis developed in?
3. What types of the vascular hyalinosis do you know?
4. Please, describe their structure and call the diseases when every type of hyalinosis develops.

2. The corpse of the patient with the diagnosis of the acute ischemic heart disease and acute heart failure has been brought for the autopsy. The diagnosis has been confirmed at the dissection.

1. What morphological changes of the heart (macro- and microscopic) can confirm the diagnosis of the acute ischemic heart disease?

3. The patient with the chronic ischemic heart disease came to the hospital with the severe heart insufficiency. He died despite of the treatment. The pathoanatomist found the chronic heart aneurysm at the dissection.

1. What is a chronic heart aneurysm?
2. What does its wall form?
3. What mortal complications do chronic heart aneurysm accompany?
4. What other morphological changes (except aneurysm) are there in the heart in chronic ischemic heart disease?
5. What morphological signs of chronic heart insufficiency do you know?

4. The man with the obesity, arterial hypertension and smoking about 2 boxes of cigarettes a day during 10 years has come to the hospital with the attacks of severe retrosternal pain. The large focal myocardial infarction of the left ventricle was diagnosed. The death occured on the third day.

1. Please, describe gross changes that had been observed in the heart at the dissection.
2. Please, describe microscopic changes in the heart in this case.
3. What changes could have been found in the coronary arteries?
4. What risk factors did the patient have for development of myocardial infarction?

5. The patient who is suffering from arterial hypertension for a long time has come to the neurological clinic with the complaints of loss memory, disturbances of muscular coordination and gait disorders.

1. What diagnosis has been made according to the clinical symptoms?
2. What morphological changes are possible there in the brain in case of arterial hypertension during a long period of time?

6. The diagnosis of the mesenterial arteries atherosclerosis with the complication and peritonitis development was made at the dissection.

1. How did mesenterial arteries look like at visual inspection?
2. What possible complications are there in the mesenterial arteries caused by atherosclerosis?
3. What complications can be developed by peritonitis?
4. What clinico-morphological forms of atherosclerosis do you know?
7. The patient of 70 years old with the severe atherosclerosis of the cerebral arteries lost his consciousness suddenly and died in a day. The pathoanatomist determined the cerebral infarction at the dissection.

1. What morphological type of infarction is developed most often in the brain in case of atherosclerosis?
2. What are possible outcomes of this infarction?
3. What can you observe in the patient’s cerebral blood vessels in this case?
4. What cerebral blood vessels can be damaged in case of the cerebral infarction?

8. The corpse of the patient with the symptoms of chronic heart failure due to chronic ischemic heart disease has been brought for the dissection.

1. What changes in the heart can confirm the diagnosis of chronic ischemic heart disease?
2. What extra heart changes can confirm the chronic heart failure?

9. The man of 45 years old has come to the reception ward of the hospital with the complaints of acute retrosternal pain which has radiated to the left shoulder-blade and left arm. At ECG investigation the myocardial infarction of the anterior wall of the left ventricle has been diagnosed. The patient died after 6 hours from the beginning of the pain attack. The red thrombus in a branch of a coronary artery has been found by pathanatomist at the dissection.

1. What stage of the myocardial infarction did the patient die in?
2. What morphological changes were there in the myocardium in this stage?
3. What additional methods of microscopy can you use for the myocardial infarction confirming?
4. What were the probable causes of death of the patient?

10. The corpse of the patient with the diagnosis of the cardiac form of arterial hypertension has been brought for the autopsy. It’s known that the patient suffered from chronic coronary insufficiency.

1. What morphological changes could have been observed in the patient’s organism?

11. The woman of 75 years old who is suffering from atherosclerosis for a long time, has a progressive loosening of memory, doesn’t recognize relatives, her behaviour becomes non-adequate and the communication with her is difficult.

1. What pathological process does take place in the patient’s brain and cerebral blood vessels?
2. What changes of brain tissue do develop due to this disturbance of cerebral blood vessels?
3. What is the name of this clinical condition?
UNIT 2
RHEUMATIC FEVER. CEREBROVASCULAR DISEASES

Grosses

1. Fibrinous pericarditis
A Russian variant - «hairy heart». Describe the appearance of pericardium: transparency, color, «bread and butter» effect.

2. Acute warty endocarditis.
Describe the size, square, thickness and spare line of the cusp. Call the risk factors of thrombus formation in both cases.

3. «Nutmeg liver». This gross was described at the first term. Learn liver appearance - density, surface, size, section's color. Why is liver called so?

Slide N 133
Valve in case of primary thromboendocarditis (acute warty endocarditis)
(hematoxylin and eosin)
Small magnification: You can see the normal configuration of mitral valve's cusp. A cusp with homogeneous dark pink areas in the center of it. On the surface of cusp there is a thrombus.

Large magnification: Look at the central part of the cusp - find the homogeneous zone (fibrinoid necrosis) and inflammation around it. Remember about the cell's composition of the inflammatory reaction. Depict this slide with large magnification and compare it with the slide N 134.

Slide N 134
Valve in case of secondary thromboendocarditis (relapsing warty endocarditis)
(hematoxylin and eosin)
Small magnification: You can see the deformation of the cusp. On the surface there is the thrombus. A zone of fibrinoid necrosis is appeared in the central part of the cusp.

Large magnification: the cusp's deformation is a result of its sclerosis and collagenization of acute thrombus. Among collagen fibers there is a lot of blood vessels - it is a diagnostic feature of the secondary acute attack. New acute changes are presented in the center of the valve zone - fibrinoid necrosis with the inflammation and trombus on the surface of the valve. The endocardial line is not clear. Depict this slide with large magnification on the one list together with the slide N 133.

Slide N 1 (D)
Liver in case of chronic venous congestion («nutmeg» liver) - as a result of mitral stenosis after acute attacks of the rheumatic heart disease.
(hematoxylin and eosin)
This slide was described and depicted in your copybooks in the first term (unit N 5). Study it once more with large magnification: you should find and remember the following signs: 1. Congested dilated central vein 2. Congested central zone of the lobule 3. Atrophy and necrosis of hepatocytes 4. Fatty degeneration on hepatocytes on the peripheral zones of the lobules.

The comparison of acute warty and relapsing warty endocarditis

<table>
<thead>
<tr>
<th>Features</th>
<th>Acute warty endocarditis</th>
<th>Relapsing warty endocarditis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Cusp deformation</td>
<td>-</td>
<td>+</td>
</tr>
</tbody>
</table>
2. Cusp surface appearance smooth rough
3. Size (square) of cusp normal decreased
4. Sclerosis - +
5. Vascularbation - +
6. Fibrinoid necrosis + +
7. Inflammation + +
8. Acute (young) thrombus + +
9. Old (sclerotic) thrombus - +

Clinical cases
1. The child has been hospitalized at the pediatric hospital with the diagnosis of rheumatic fever. He complained of pain in small joints, which were swollen, the painful nodes appeared in surrounding tissues.
   1. What changes were there in joints and surrounding tissues in rheumatic fever?
   2. Describe, please, the dynamic of morphological changes.

2. The combined mitral valvular disease were formed in the patient after rheumatic fever attack. The chronic heart insufficiency appeared in some years. Despite of the persistent treatment the patient died.
   1. What changes of chronic heart insufficiency were confirmed at the dissection?
   2. Describe, please, all possible morphological changes of the heart and other organs.

3. The woman of 50 years old had a long time history of rheumatic fever with heart failure. She died from chronic heart insufficiency. The combined mitral valvular disease was found at the dissection.
   1. Describe, please, morphological changes of the valve’s casps which were observed by pathologist at visual inspection and histological investigation.
   2. What morphological changes must have been found in the lesser circulation?
   3. What morphological changes were found in the organs and tissues of the greater circulation at the dissection?
   4. What does it mean “combined mitral valvular disease”?

4. The patient with the rheumatic combined mitral valvular disease which is complicated by the circulatory decompensation is in a ward of the therapeutic department.
   1. What changes can be developed in the lungs of the patient and why?
   2. Describe, please, possible structural changes in the lungs and liver in this patient.
UNIT 3
PULMONARY DISEASES: ACUTE PNEUMONIAS. GRIPPE

Grosses

1. **Lobar pneumonia** (stage of grey hepatization) - This type involves a whole lobe. The lung's lobe is dry, grey and dense. The pleura demonstrates a fibrinous inflammation - it is grey and rough.

2. **Bronchopneumonia** - In the bronchopneumonia pattern, foci or inflammatory consolidation are distributed in patches through out one or several lobes, most frequently bilateral and basal. Well developed lesions up to 3 or 4 cm in diameter are lightly elevated, dry, granular, grey-red to yellow and poorly delimited at their margins. A confluence of these foci may occur in severe cases producing the appearance of a lobar consolidation. The lung's substance immediately surrounding areas of consolidation is usually hyperemic and edematous. Compare lobar pneumonia and bronchopneumonia's features.

**Slide N 75 (D)**
**Lobar pneumonia**
(hematoxylin and eosin)

Small magnification: The pulmonary structure is essentially changed. All alveoli have no air. There is a great venous congestion.

Large magnification: The alveoli are filled with fibrinous exudate and there are a large number of neutrophils, blood congestion.

You have already depicted this slide in the first term (unit 7). Examine this slide once more and remember it.

**Slide N 72**
**Bronchopneumonia (focal pneumonia)**
(hematoxylin and eosin)

Small magnification: Among air alveoli you can find airless foci - every focus consists of bronchus or bronchioles, adjacent alveolar spaces and blood vessels.

Large magnification: Examine this slide in details - 1. find the blood vessel - its lumen contains a lot of erythrocytes - the main sign of venous congestion during inflammation. 2. Then study the bronchial wall and lumen, describe and depict general features of acute bronchitis – there are degeneration of column epithelium, inflammatory cells inside the bronchial lumen and the wall. There is an inflammatory reaction inside the alveoli - they demonstrate serous-purulent or purulent exudate.

Depict this slide with large magnification and compare their signs with lobar pneumonia.

**Slide N 128 (D)**
**Staphylococcal pneumonia**
(hematoxylin and eosin)

Examine all areas of this slide and find a central part of inflammatory focus – it is necrotic zone (bright pink). Zones of different exudates are localized around it. At first there is purulent exudate. Then - serous-purulent, then - only serous or hemorrhagic. This fact is the key sign of staphylococcal pneumonia.

You can not depict this slide - this is only demonstration.

**Slide N 151 (D)**
**Necrotic tracheitis in case of grippe**
(hematoxylin and eosin)

This is a fragment of trachea. Pay attention to changes in inner layers. You can see necrosis of mucous membrane with colonias of microbes. In submucous layer greate blood congestion and
diffuse leukocytes infiltration are observed. This condition may be in moderate or severe severity of disease. You can not depict this slide - this is only demonstration.

Clinical cases
1. The patient with a heavy form of the grippe (influenza) was admitted to the infectious hospital. In some time unpleasant feelings appeared in heart, the heart sounds were muffled. The diagnosis of myocardiodystrophy was made.
   1. What morphological changes were in the heart in picture of myocardiodystrophy?
   2. What was their cause?
   3. What was the morphogenetic mechanism of this dystrophy development?

2. The corpse of patient who died from lobar pneumonia was brought to the pathoanatomy department. The diagnosis of lobar pneumonia was confirmed by autopsy: there was fibrinous inflammation in the lungs and pleura.
   1. How does the lung and pleura look like at visual inspection and microscopic investigation?
   2. What pulmonary complications of lobar pneumonia do you know?

3. The patient had suffered from arterial hypertension. During hypertonic crisis the brain hemorrhage had been developed. The patient lived for 7 days without consciousness. At the autopsy besides the brain damage the foci of pneumonia were observed in the lower parts of both lungs.
   1. What clinico-morphological type of pneumonia was there in this case?
   2. Describe the lung at visual inspection?
   3. What predisposing factors of pneumonia development were there in this case?
   4. If the patient did not die, what processes would be in the lung in recovery?

4. Patient G. of 37 years old had fallen ill with a heavy form of pneumococcal pneumonia and died from heart insufficiency. At dissection the enlarged, dense, whitish-grey, unaired left lower lobe of the lung was observed. There were also pleura changes.
   1. What clinico-morphological form of acute pneumonia was there in this case?
   2. What stages of development does this pneumonia have and how long does every stage take for its development?
   3. Describe, please, the lung is gross appearance at visual inspection and the histological features in every stage.
   4. What pathological process was developed in the pleura? Call, please, its macro- and microscopical characteristics.
   5. What are possible causes of death in this form of pneumonia?

5. The child died in the children's department from confluent bacterial bronchopneumonia. The etiology defined two suggestions: either staphylococcal or streptococcal pneumonia.
   1. What are the distinguishing structural criteria of these two types of pneumonia?
   2. What can you say about streptococcal etiology in this case and what is about staphylococcal one?
   3. What possible complications may be in these pneumonias?

6. The corpse of the 34 years old patient has been brought for the dissection with the diagnosis of lobar pneumonia which has been complicated by heart insufficiency. At autopsy some morphological changes confirming the diagnosis were found. There was the total damage of the left lung in stage of grey hepatization.
1. Describe, please, macro- and microscopic picture of grey hepatization stage.
2. What other stages of lobar pneumonia do you know?
3. What extra pulmonary changes can be developed in case of lobar pneumonia?
4. What is the etiology of lobar pneumonia?

7. The patient of 60 years old was operated on for gastric carcinoma. After the operation he stayed in heavy condition and pneumonia was developed. The patient died from cardiac-pulmonary failure.

1. What clinico-morphological form of acute pneumonia was there in this case?
2. What are the main morphological features of this pneumonia?
3. What predisposing factors of the pneumonia development were there in this case?
4. How can you avoid the pneumonia development in postoperative period?
5. Is pneumonia the basic disease or complication in this case?

8. The symptoms of cardiac insufficiency have appeared in the patient with bilateral bronchopneumonia: heart sounds are muffled, there are arrhythmia and changes of ECG.

1. What is bronchopneumonia?
2. Give, please, its morphological characteristics (macro- and microscopical).
3. What morphological changes in the heart did cause its functional disorders?
4. Why were they developed?

9. The patient was admitted to the hospital during the epidemic period of the grippe with the complaints of chills, aching joints, coughing, headache, pronounced dyspnea, and temperature rising up to 40°C. At auscultation the multiply moist rales were listened to. Despite of the treatment the patient died in 12 days due to severe pneumonia.

1. What clinico-morphological form of the grippe was there which caused death?
2. What morphological changes were there in the bronchi and lungs?
3. How did the lungs look like at visual inspection?
4. What is the figurative name of this lung?

10. During “American Legion” congress in 1976 the 48 years old man was complaining of rising temperature up to 38,7°C and pain in the chest in coughing. There was no lung pathology. The temperature retuned to normal after antibiotics treatment. The rate of immunofluorescent Legionellas antibodies was examined in 6 months. It was positive.

1. What clinical form of legionellosis was there in the patient?
2. Call, please, the cause of the disease. Is the disease pass from person to person, what are the ways of spreading?
3. Why was the congress of the “American Legion” mentioned in this case?
4. Why does it take place in the history of this disease researching?
5. What changes may occur in lungs if the process becomes severe?
UNIT 4
PULMONARY DISEASES: CHRONIC DISEASES OF THE LUNG

Grosses

1. Bronchiectasis and the most important complication - amyloidosis of the kidney and spleen. There are three organs - the lung, the kidney and the spleen. Look at the lung and find the main structural changes, describe the great sclerosis, dilation of bronchial lumen and purulent exudate inside it. The kidney and spleen demonstrate the main complication - amyloidosis. Describe the appearance of these organs and answer the questions. What can you say about the connection between pulmonary changes and amyloid formation's in the kidney and spleen? What is the name of such kidney?

2. Central (bronchogenic) cancer of the lung. Find tumorous tissue, describe size, form, colour and boundaries of it with pulmonary tissue.

Slide N 36
Carnification of the lung
(hematoxylin and eosin)

Find and depict the following sign of the chronic process in the lung – growth of connective tissue in the lumen of alveoli when purulent (fibrinous) exudate is replaced by fibrotic tissue.

Slide N 103 (D)
Diffuse interstitial disease of the lung
(hematoxylin and eosin)

This is a piece of the lung. There is focal (granulomas) and diffuse cellular infiltration with lymphocytes, histiocytes, monocytes, macrophages and so on in the alveolar septas. There is replacement fibrosis in the interstitial septal wall.

Slide N 103 a (D)
Chronic bronchitis, bronchiectasis, carnification, chronic abscess in the lung
(hematoxylin and eosin)

Find and depict the following signs of the chronic process in the lung:
1. Note the marked interstitial fibrosis
2. Chronic bronchitis with the enlargement of the bronchial wall, chronic inflammatory process inside its wall, fibrosis of its wall, squamous metaplasia of the epithelium.
3. Carnification - collagenization of exudate inside the alveoli.
4. Chronic abscess with its piogenic membrane - it has 3 layers - internal – purulent exudate, medium - granulation tissue and external - connective tissue.
5. Bronchiectasis - the permanent dilatation lumens of bronchi, inflammation inside these walls, shedding of epithelium and purulent exudate inside the lumen of bronchus.

Slide N 16
Pulmonary emphysema
(hematoxylin and eosin)

This is a piece of the lung. You can see a combination of permanent dilatation of air spaces distal to the terminal bronchioles and the destruction of the walls of dilated air spaces.

Slide N 316 (D)
Small cell (poorly-differentiated) pulmonary carcinoma
(hematoxylin and eosin)

This is a piece of the lung. You can see invasive growth of immature tumor which consists of generally small cells look like limphocytes. In large magnification you see that these cells have round, oval or polygonal form. In a part of slides there are foci of necrosis, hemorrhages in tumor.
Clinical cases
1. In the patient who was suffering from chronic bronchitis, tumor of lung was found. To confirm the diagnosis the bronchoscopy was made. At this examination the formation was observed which was attached to bronchial wall. At histological examination the patient was found to have carcinoma from bronchial epithelium.

1. What macro- and microscopical variants of lung carcinoma do you know?
2. Where does lung carcinoma spread?
3. What peculiarities of metastatic spreading of this tumor do you know?

2. The 47 years old patient has come to the surgery department with a high temperature and moist cough. On X-ray examination the cavity with the fluid was observed in the right lung. The operation of lobectomy was performed. At investigation of the operative material the pathoanatomist has found the cavity of 7 sm. in diameter which has contained a yellowish-green, bad smelled fluid. The wall of the cavity is thick, whitish-grey and dense.

1. What pathological process was there in the lung?
2. What could has been preceded of this pathological formation in the lung?
3. What morphological changes determined the chronic course of this process?
4. What do you think, why was the operation indicated for the patient?
5. What complications of this disease have been avoided by operation?

UNIT 5
DISEASES OF STOMACH AND INTESTINE

Grosses
1. **Chronic ulcer of the stomach.** Describe the appearance, localisation, size and depth of the ulcer and condition of mucosal layer around the ulcer. Then describe the base and margins of once. Call the possible complications of the gastric ulcer.
2. **Acute (phlegmonous) appendicitis.** The inflamed appendix below is red, swollen and covered with fibrinous patch.
3. **Malignant tumor of the stomach (exophytic - fungiform type).** A large tumors node is seen on the stomach small curvature. Describe this node and name the general signs of stomach tumor. What does it mean – differentiation, atypism, metastasis, a general influence on the organism?
4. **Malignant tumor of the stomach (endophytic - diffused type).** Confine the size of the wall, it’s colour, density an possible borders of the tumor’s growth. Compare with exophytic type of growth. What do you think about the prognosis of both variants?
5. **Metastasis of Krukenberg.** It is a pair of secondary tumor (retrograde lymphogenic metastasis) in both ovaries.

Slide N 186 (D)
**Chronic atrophyc gastritis**
(hematoxylin and eosin)

Small magnification: you can see a thin mucosal layer of the stomach.
Large magnification: In mucosal layer glands are decreased in quantity and size – there is atrophy. In lamina propria of mucosal membrane diffuse lymphocyte's infiltration and fibrosis are observed.

Slide N 145 (D)
**Erosion(s) of the stomach**
(hematoxylin and eosin)
Small magnification: Only mucosal defect is visible (as a result of superficial necrosis and it following separation). In the margins and base of erosion there are leukocytes infiltration and brown pigment. Do not depict this slide. Describe the wall of erosion. What pigment can be formed in the erosive area?

**Slide N 144**

**Chronic ulcer of the stomach**
*(hematoxylin and eosin)*

Small magnification: The stomach wall is visible with deep defect of internal, submucosal and muscle layers. Determine the low part of ulcer (base of ulcer) and around part of it.

Large magnification: Look at the base of the ulcer and confirm that the necrotic zone is presented here. Under it you can see the granulated tissue and the scar. Inside the deep layers you can find the old brown pigment – hydrochloride hematin and blood vessels with sclerosis of the wall.

Depict this slide with large magnification and define the stage of chronic ulcer. Describe the base of ulcer in the remittent stage and depict by the scheme.

**Slide N 146**

**Acute flegmonous appendicitis**
*(hematoxylin and eosin)*

Small magnification: Determine all layers of the appendix and find the inflammatory morphological sings.

Large magnification: Confirm the leucocytes-rich exudate in the all layers of the wall. Name the type of appendicitis in this case and call other types of it.

Depict this slide with large magnification.

Clinical cases
1. The resected stomach with a sticking out carcinoma in the lumen of the small curvature has been brought to the pathoanatomical laboratory. Gastric adenocarcinoma has been diagnosed at the histological investigation.

   1. What is the name of the tumor growth in the lumen of the organ?
   2. Give, please, examples of well- and poorly-differentiated adenogenic carcinomas.
   3. What is adenocarcinoma?
   4. What are the localizations of gastric carcinoma’s metastases and what are the ways of its spreading?

2. The removed stomach has been brought to the pathoanatomical laboratory due to its tumor suggestion. The stomach wall of pyloric and fundal portions is thick, plicas are big and deformed. There is dense, whitish tissue at incision. The diagnosis of cancer has been made.

   1. What is the name of this macroscopic form of carcinoma?
   2. What histological picture does this type of gastric carcinoma usually characterize?
   3. What are the sides of localizations of gastric carcinoma,s metastases?

3. A severe form of epithelial displasia was found in gastric mucosa at gastroscopy in the patient with a long time history of stomach functional disorders.

   1. What morphological changes did appropriate to this diagnosis?
   2. Why is displasia dangerous?
   3. What morphological changes may be there in different forms of displasia (mild and average, except severe) and what must the doctor do in every certain case?
4. The 5 years old child has eaten much different irritated food, at the New Year's table. In some hours he began to complain of abdominal pain, nausea and vomiting. The temperature was normal. After some time the self-beeling became better, except anorexia only. The diagnosis of gastritis was made.

1. What form of gastritis was there in this case according to the clinical course and morphology?
2. What morphological changes of gastric mucosa (at visual inspection and histological investigation) are there in this form of gastritis?
3. What is the usual outcome of this gastritis?
4. What other morphological forms of acute gastritis do you know? Describe please their morphological appearances.

5. The patient with the acute abdominal pain has been urgently transferred to the surgical clinic. The pain has appeared suddenly (“knife-like pain”). The patient is known to have had a long time history of peptic ulcer.

1. Describe, please, peptic ulcer`s morphology (macro- and microscopic changes) in all phases of the disease.
2. What complication could have been suspected in this case?
3. What was necessary to do and why?
4. What groups of peptic ulcer complications do you know?

6. The gastrobiopsy is often taken from the chronic gastritis patients for the diagnosis confirming.

1. What classifications of chronic gastritis do you know?
2. What structural changes could have been observed by biopsy in gastric mucosa material with different morphological types of chronic gastritis?

7. The pain in epigastrium and vomiting appeared suddenly in the woman. She came to the doctor two days after the onset. The temperature was 38°C. The woman was admitted to the hospital and was operated on. During the operation thick appendix was found, its serous membrane was hyperemic covered by fibrinouse and purulent stains.

1. What form of acute appendicitis was found in the patient?
2. What histological changes of the appendix did correspond to this form?
3. What complications could be combined with this form of acute appendicitis?

8. The patient of 50 years old, has a long time history of chronic gastritis with the typical symptoms characterizing the disease. He came to the doctor complaining of increased pain in the abdomen (stomach). The gastroscopy with biopsy was performed. The histological diagnosis was chronic atrophic gastritis.

1. On the base of what histological changes was this diagnosis made?
2. Why was gastroscopy indicated with biopsy for the patient?
3. Why is chronic gastritis dangerous?
4. What morphological forms of chronic gastritis do you know?
5. What etiological factors take place in the chronic gastritis development?
UNIT 6
LIVER DISEASES

Grosses

1. Portal cirrhosis of the liver
The characteristic diffusely small-nodular surface reflects the interconnection between nodular regeneration and scarring. The average nodules size is 3 mm in this dilatated view. Describe the results of these types of the cirrhosis – portal hypertension.

2. Secondary biliary cirrhosis of the liver
This gross demonstrates the fine nodularity and bile staining of the final-stage of biliary cirrhosis. It is micronodular cirrhosis. Call the extrahepatic features of this type of cirrhosis.

3. Postnecrotic cirrhosis of the liver
It is characterized by irregular areas of massive fibrosis alternating with other areas of more delicate scarring, producing nodules of variable size, but at whole it is macronodular cirrhosis.

4. Stones in gallbladder
Chronic cholecystitis demonstrates a thickened gallbladder wall and luminal stones. Call the possible composition of gallstones. Describe the sequence of stones formation in gallbladder.

Slide N 171-A
Toxic degeneration of the liver (massive progressive necrosis of the liver).
(hematoxylin and eosin)
Small magnification: The general structure of the liver is destroyed.
Large magnification: Slide showing the destruction of hepatic lobular architecture with zonal necrosis. The massive necrosis of hepatocytes is marked in the liver. The small inflammatory reaction is seen between necrotic hepatocytes.
Depict this slide with large magnification and explain the causes of the morphological changes in the liver. Call the following possible outcomes of this disease.

Slide N 148 (D)
Chronic hepatitis with high activity
(hematoxylin and eosin)
Small magnification: Slide shows portal tract expansion with inflammatory cells.
Large magnification: Focal necrosis is marked in the liver. The inflammatory cells are deposited at the side of the necrotic hepatocytes.
Depict this slide with large magnification, describe the causes of this process and call other types of chronic hepatitis and fill the gaps in the table.

Morphological comparison of different types of hepatitis

<table>
<thead>
<tr>
<th>N</th>
<th>Features</th>
<th>high activity (active)</th>
<th>low activity (persistent)</th>
<th>cholestatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Necrosis of hepatocytes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Localisation of inflammatory cells</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Degree of fibrosis</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Slide N 234  
**Portal cirrhosis of the liver**  
(hematoxylin and eosin)  
Small magnification: The liver shows the disruption of normal lobular architecture. The border between lobuli is not clear, by marked every lobular has a small size (<3mm), the majority of lobuli is not show central viens – they have the figurative name - “false lobuli”.  
Large magnification: You can see fibrotic tissue between lobuli with inflammatory cells – there are lymphoid aggregates. The fibrotic tissue forms the narrow septas, it is developed by separated fibroblasts. Depict this slide with large magnification.

Slide N 122 (D)  
**Portal cirrhosis of the liver**  
(picrofuchsin by van Gieson)  
Study this slide and recognize the connective tissue and hepatocytes.

Slide N 100 A  
**Secondary biliary cirrhosis of the liver**  
(hematoxylin and eosin)  
Small magnification: The general structure of the liver is changed. It is characterized by drowing of fibrotic septas which divide the liver on small falce lobules. There is an extensive proliferation of bile ducts. Other cholestatic features in the parenchima may be with expressed degeneration and formation of bile lakes. Small regenerative nodes are formed there.  
Large magnification: A portal tract is markedly expanded by an infiltrates of lymphocytes and plasmatic cells. You can find the granulomatous reaction to a bile ducts. Connective tissue has a delicate structure as thin fibrotic septas.

Slide N 189 (D)  
**Postnecrotic cirrhosis of the liver**  
(hematoxylin and eosin)  
Small magnification: The structure of the liver is changed. The liver shows the lobuli of different size (several centimeters in diameter, macronodules). Between them fibrous septas (broad scars) are seen as a result of septic collapse.  
Large magnification: Find the common features of postnecrotic cirrhosis - large false lobules and the massive fibrous scar contains numerous arteries, veins and bile ducts crowded together as a result to destruction of several whole hepatic lobules. Depict this slide with small magnification and compare it with other forms of cirrhosis.

Clinical cases  
1. The patient with the symptoms of hepatic failure is in the hospital. His skin is yellow.  
   1. What is the name of pathological condition when the skin becomes yellow?  
   2. What other tissue changes may have the patient?  
   3. Due to accumulation of what pigment patient’s tissues become yellow?  
   4. What is the nature of this pigment?  
   5. Give, please, examples of clinical pictures and pathological conditions when this pigment can be accumulated in blood?  

2. The acute hepatic insufficiency has been developed in the patient after mushrooms poisoning. The patient has had hepatic coma clinically. A sharp decrease of liver sizes has occurred.  
   1. What disease has been developed in the patient?
2. What morphological stages does it have? What morphological changes of the liver are there in every stage of the disease (macro- and microscopic)?

4. What possible outcomes may this disease have?

3. The corpse of a 54 years old woman has been brought for the dissection. There was cholelithiasis in her anamnesis. At autopsy the jaundice, decreased liver with dense consistency, small nodular surface and green color have been marked.

1. What disease was detected at dissection?
2. What morphogenetic and morphological forms of the disease were there in this case?
3. What morphological changes must be found in hepatocytes, stroma, bile capillaries and topography of blood vessels in the liver?
4. What was the cause of death in this case?

4. The patient has a long time history of chronic alcoholism (a hepatic form of disease). At the last hospitalization the liver was found with small nodular surface at laparoscopy in this patient. There were distended veins of the anterior abdominal wall ("caput Medusae").

1. What pathological process was developed in the liver?
2. Call, please, morphogenetic and morphological types of the process in this case.
3. What possible microscopic changes were there in the liver?
4. Why were the veins of the anterior abdominal wall distended in the patient?
5. What are the possible causes of death in this case?

5. The 43 year old patient who was the surgeon felt ill with a viral hepatitis after the injury of the hand during the operation. He was treated for several years, but hepatitis became chronic. The severe morphological picture of chronic hepatitis was observed at puncture biopsy.

1. Why does acute hepatitis become in chronic in some cases?
2. What clinico-morphological forms of chronic hepatitis do you know?
3. Compare, please, these forms according to the morphological and clinical parameters?
4. What form of chronic hepatitis was there in the patient?

6. The corpse of the patient who died from liver cirrhosis has been brought for dissection. Portal liver cirrhosis has been observed.

1. What other morphogenetic types of liver cirrhosis do you know?
2. Make, please, comparison of all these liver cirrhosis types according to the etiology, taking into account clinical symptoms and macro- and microscopic changes.

7. Patient M. has a severe viral hepatitis in his anamnesis. The diagnosis is liver cirrhosis at present time. At laparoscopy the liver is decreased, its surface is greatly nodular (the diameter of the nodes is up to 5 sm.) and thick connective tissue septus are observed.

1. What type of liver cirrhosis is there according to morphogenesis and morphology?
2. What morphological peculiarities has this liver cirrhosis type?
3. What do you think, in outcome of what form of viral hepatitis in this liver may be cirrhosis developed?
4. How is the decompensation of this liver cirrhosis clinically realized?
8. At dissection of the dead patient of 50 years old, the decreased, dense, small nodular liver was found and there were varicose dilation of esophageal veins and veins of the cardiac portion of the stomach. In the stomach cavity there were 900 ml. of liquid blood and blood clots, in peritoneal cavity – 700 ml. of edematous fluid.

   1. What disease of the liver was there in the patient?
   2. Describe, please, the morphological changes of the liver.
   3. What was the cause of death in this patient?
   4. Explain, please, why was blood found in the stomach and was there edematous fluid in the peritoneal cavity?
   5. Give, please, examples of diseases which may cause liver cirrhosis.

9. The young woman has been urgently brought to the hospital with poisoning symptoms. Despite of the treatment the patient’s condition was worse and the death was due to hepatic failure. An acute decrease of the liver, with flabby consistency and yellow colour was found at autopsy.

   1. What disease had the patient?
   2. What stages of this disease do you know?
   3. In what stage did the death occur in this case?
   4. Describe please the microscopic picture of the liver in this stage.
   5. What other outcome of the disease is possible?
   6. What possible changes were there in the patient’s kidneys?

10. What different clinical forms of the viral hepatitis are known including the form with jaundice and without jaundice.

   1. What other clinico-morphological forms of acute hepatitis do you know?
   2. What morphological changes are there in liver in hepatitis with jaundice and without jaundice?
   3. Why is jaundice developed and what is its type?

11. The patient has been admitted to the infectious hospital with the suggestion of epidemic hepatitis. The diagnosis has been confirmed by laboratory analysis.

   1. What is the etiology of this hepatitis?
   2. What morphological changes are there in the liver in this hepatitis?
   3. What is the most frequent outcome of the disease?
   4. Call, please, all possible outcomes of viral hepatitis.
   5. What is the most severe form of viral hepatitis and why is it so?
UNIT 7
KIDNEY’S DISEASES

Grosses

1. **Hemorrhagic glomerulonephritis** - the kidney is enlarged and pale, often with small red spots on the cortical surface. The surface of the kidney is smooth. The kidney’s capsula is thin and transparent.

2. **The secondary nephrosclerosis** - the kidneys are symmetrically contracted. They have dense consistency and small-granular surface. On section the cortex is thinned.

3. **The kidney in case of toxin abuse** - the kidney has a normal size and smooth surface. On section the cortex is pale and the piramides are dark red in color.

4. **Hydronephrosis** - the kidney is with marked dilatation of the pelvis and calyces and thinning of the renal parenchyma. In early advanced cases the kidney may become transformed into a thin-walled cystic structure with striking parenchymal atrophy, total obliteration of the pyramids and thinning of the cortex.

5. **Renal calculi (urolithiasis)** – remember, that the stone’s containment are organic center and salt’s periphery. The general cause of their formation is metabolic disturbance. The main predispositions factors are inflammation, urine flow disturbance and change of urine contents.

6. **Amyloidosis of the kidney** - the kidneys have a similar appearance - they are symmetrically enlarged, pale and very dense. On section the cortex is enlarged, has a pale color. The border is not clear between layers. Give it's figurative name.

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**Slide N 184**

**Rapidly-progressive (crescentic) glomerulonephritis**

*(extracapillary proliferative glomerulonephritis).*

(hematoxylin and eosin)

Small magnification: Find the cortex of the kidney and examine glomeruli. Some of them have a normal structure. Majority of them are changed.

Large magnification: The picture is dominated by the formation of distractive crescents. Crescents are formed by the proliferation of parietal cells and by the migration of monocytes and macrophages into Bowman space. There may be lymphocytes. The crescents eventually obliterate Bowman’s space and compresses the glomerular tuft. At the slide you can see the collapsed glomerular tufts, crescent-shaped mass of proliferating cells and lymphocytes in the Bowman capsule.

Some of glomeruli are increased in size – functional hypertrophy - and around tubuli are appeared as cysts with bright-pink substance inside the lumen – these are protein cylinders.

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**Slide N 185**

**Chronic glomerulonephritis - secondary nephrosclerosis (secondary shrunken kidney)**

(hematoxylin and eosin)

Small magnification: Find the cortex and examine the glomeruli. You can not find the glomeruli with normal structure. The majority of glomeruli are decreased in size.

Large magnification: The glomerular's histology depends on the stage of the disease. At the end cases wide spread decreased glomeruli are seen. So, you can see a small pale-pink glomeruli (in hematoxilin-eosin). A marked atrophy of the associated tubules, irregular
interstitial fibrosis and lymphocytic infiltration also occur. Also hyalinosis of arterioles and myoelastofibrosis in arteries of muscle type are seen clearly.

Depict this slide with large magnification and compare it with the slide N 184.

**Slide N 183 (D)**

**Acute tubular necrosis (Sublimate kidney or kidney in case of corrosive chloride of mercury abuse)**

(hematoxilyn and eosin)

Small magnification: Find the cortex and convince it with the normal structure of glomeruli – they contain blue nuclei. Around them you can recognize the tubuli as pink circles.

Large magnification: With mercuric chloride, for example, tubular epithelial cells become totally necrotic (cells are without nuclears in the cytoplasm and are desquamated into the tubule lumen.

**Slide N 18 (D)**

**Amyloidosis of the kidney**

(hematoxilyn and eosin)

Examine this slide and compare it with N 19

Remember the slide № 19 from the first term. It was stained by Congo-red and only amyloid substances were coloured with red.

Determine the clinico-morphological stage of this process.

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1. The stomatological patient has a long time history of chronic osteomyelitis of the upper jaw. Despite of treatment, the facial edemas, protein in urine and other symptoms of kidney functional disorders appeared in some time.

   1. What complication were developed in the patient?
   2. Why are kidney changes possible in this patient?

2. The corpse of the patient who was suffering from secondary amyloidosis for a long time and died from combined insufficiency of kidneys, liver and adrenals glands, has been brought for dissection. The pathoanatomist made the diagnosis of secondary amyloidosis, of parenchymatous variant.

   1. What is the secondary amyloidosis?
   2. What types of amyloidosis according to the etiology do you know?
   3. Where does amyloid deposit in parenchymatous type?
   4. Why were there kidney, liver and adrenals insufficiencies in this case?

3. Some changes suggesting the kidney amyloidosis have been found at dissection.

   1. How do kidneys look like at visual inspection in amyloidosis?
   2. What test can you make for amyloid recognizing at dissection?
   3. What histological stains can you use for the presence of amyloid confirmation?

4. The patient with the diagnosis of acute glomerulonephritis was in the nephrological clinic. Despite of the treatment the patient died from renal failure sooner. The autopsy confirmed the diagnosis. There were severe changes in glomeruli and ducts.

   1. What changes were there in glomeruli in acute glomerulonephritis?
2. What structural changes were there in tubules and in blood vessels?
3. What certain morphological changes do the most severe clinical manifestations have?

5. The patient with the renal and extra renal symptoms of glomerulonephritis has been admitted to the hospital.

1. Call, please, all renal and extrarenal symptoms of glomerulonephritis which you know and their pathogenesis.

6. The young man has come to the polyclinic with the complaints of facial oedemas. At laboratory examination the protein and hyaline casts were found in urine. The patient was hospitalized with the diagnosis of glomerulonephritis with nephrotic syndrome.

1. What is the nephrotic syndrome?
2. Why is it developed (its causes and pathogenesis)?
3. What changes were there in the tubular epithelium and what was the mechanism of their development?

7. Patient P. with the wide-spreading burns has been brought to the traumatological clinic in shock. In several hours he died. The changes of the kidneys were found at dissection. The kidneys were increased, flabby, capsule was taken off without any difficulties, the cortex was pale, and pyramids were dark red at incision.

1. What pathological process was there in the kidney?
2. What was its stage?
3. Call, please, other stages of this pathology.
4. What probable histological changes of the kidneys were there in this case?
5. Why kidney’s pathology was developed in all types of shock?

8. The patient with the manifestations of chronic renal insufficiency due to chronic glomerulonephritis had been hospitalized many times. The severe hypertension was occurred at the last time. The death was sudden due to acute cerebral circulation impairment. The changes confirming the chronic glomerulonephritis were found at autopsy and there was also brain hemorrhage in brain lateral ventricles.

1. What is the name of the kidney in chronic glomerulonephritis accompanied by chronic renal insufficiency?
2. How does this kidney look like at visual inspection?
3. Why was cerebral effusion developed?
4. What changes were there in the blood vessels?
UNIT 8
INTESTINAL INFECTIONS. SEPSIS

Grosses

1. **Fibrinous colitis in case of dysentery** – It is an internal wall of intestinum with ulceration and pseudomembrane formation (fibrin deposition). Call the stage and the onset of the disease. What complications might be you call in this stage?

2. **Small intestine in case of typhoid fever** – describe the Peyer's patches - they elevate up to 5-8-cm with the enlargement of draining mesenteric nodes resulting in oval ulcers with their long axes in the direction of bowel flow. Call the stage, the onset of the disease and general complications of typhoid fever.

3. **Septic thrombo-ulcerative endocarditis of the aortic valve.** It is the destructive aortic valve with irregular, large friable vegetations – there are thrombi with microbes. Describe the possible complications of thrombus formation.

4. **Embolic purulent nephritis** – describe the appearance, size and consistency of the kidney and pathological foci – localization, number, size, color and shape.

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**Slide N 114 (D)
Fibrinous colitis in case of the dysentery**
(hematoxylin and eosin)

Small magnification: It is the intestinal wall without typical structure of mucosa – you cannot see a normal intestinal glands – it is a deep necrosis of mucosa and sub mucosa with fibrinous inflammation.

Large magnification: Find the low-parts of intestinal glands as a result of deep necrosis – the surface of necrotic inner layes with fibrin deposition, neutrophilic reaction and marked edema. So mark necrosis of mucosa, fibrin deposition and inflammatory reaction. Name the stage of the dysentery with these local morphological changes.

**Slide N 169
Serous-desquamative enteritis in case of cholera.**
(hematoxylin and eosin)

Small magnification: You can see that intestine's wall becomes thick. In mucosal layer inflammatory infiltration and desquamation of superficial epithelium are defined.

Large magnification: Find the signs of congestion of the mucosal lamina propria and moderate infiltration of neutrophiles and mononuclearis inflammatory cells.

Depict this slide with large magnification and call the special complications of cholera.

**Slide N 149
Septic thrombo-ulcerative endocarditis**
(hematoxylin and eosin)

Small magnification: It is the aortic valve with thrombus on it

Large magnification: Find the zone of necrosis with erosions inside the valve with thrombus formation and expressed inflammatory infiltration, areas of sclerosis. Depict this slide.

**Slide N 74
Embolic purulent nephritis**
(hematoxylin and eosin)

Small magnification s: Observe whole slide a find the blue areas.
Large magnification: Look at the blue area. There are focal abundant neutrophilic infiltration. In the centers of these foci there is colonies of microbes dark-blue in colour. Depict this slide with large magnification and mark pyogenic embolus.

Clinical cases
1. The temperature has risen up to 41°C, the acute edema of the oral mucosa and neck tissues, cloudy consciousness, multiplied skin petechial hemorrhages have appeared in the patient of 21 years old in 3 days after teeth extraction. The death has come on the 2-d day after the symptoms appearance.

   1. What clinico-morphological form of sepsis was there in this case?
   2. What was type of the sepsis according to the gates of infection?
   3. What local morphological changes were there in the patient?
   4. What morphological changes were there in the stroma and parenchyma of the parenchymatous organs, in the hemopoetic organs and in the lymphoid tissue?

2. The woman of 44 years old, has come to the hospital with the odontogenic phlegmone of the mouth cavity and submandibullar region. Despite of the active surgical and antibiotic treatment the sepsis has been developed and the death occured due to this. The multiple abscesses of the lungs, liver, kidneys and the septic spleen were found at the dissection.

   1. What clinico-morphological form of the sepsis was there in this case?
   2. What primary septic focus was there in this case?
   3. What morphological changes could the pathoanatomist observed in the the septic spleen?
   4. What are the principles of sepsis classifications?

3. The patient with the symptoms of the heart damage (pain, arrhythmias, weakness of the heart sounds and others) has come to the reception ward of the hospital. The diagnosis of the myocardial infarction was not confirmed by ECG and the new diagnosis of the septic endocarditis was made.

   1. What possible morphological changes are there in the organism in septic endocarditis?
   2. What are the modern views of etiology and pathogenesis of this disease?

4. In the 46 year old patient of the suffering from rheumatic heart valvular disease for a long time the fever, jaundice, edemas, petechial skin hemorrhages and shortness of breath at rest appeared. On the conjunctiva of both eyeballs there was petechial rash. The patient died. The diagnosis of the septic endocarditis has been confirmed at the dissection.

   1. What clinico-morphological form of sepsis was developed there in the patient?
   2. By what objects have been valvular casps damaged in the sepsis?
   3. Compare, please, morphological changes of the valvular casps which developed in sepsis and rheumatic fever.
   4. What complications were possible there in the patient if he would survive?

5. The symptoms of “acute” abdomen (acute abdominal condition) have appeared in the patient with typhoid fever on the third week of the disease and peritonitis has been diagnosed. Despite of the performed treatment the patient died.
1. Describe, please, the genesis and dynamic of morphological changes of the intestine with typhoid fever.
2. Why has peritonitis been developed?
3. Call, please, other possible local complications in typhoid fever.

6. The young woman has been hospitalized at the Emergency Department with the complaints of headache, high temperature, weakness and apathy. At the external examination the following changes have been found: the skin was clear, the abdomen was slightly painful and in the lungs the vesicular respiration was heard. The diagnosis was uncertain. Approximately on the 10-th day the roseolous popular rash on the abdomen, s skin and the disorders of the intestine appeared. The patient was directed to the infectious department with the diagnosis of typhoid fever.

1. What stages of typhoid fever do you know?
2. What changes were developed in the intestine according to the stages?
3. Why does rash appear?
4. What possible complications are there out of intestine in typhoid fever?

7. The fibrinous colitis has been found in the patient with schigella dysentery at the rectoscopy. The diagnosis of the infection has been confirmed by bacteriologic tests.

1. Describe, please, macro- and microscopic appearances of fibrinous colitis.
2. What stages of schigella dysentery are there in its development?
3. What changes are there at the onset at the beginning and at the end of schigella dysentery in the sigmoid intestine and rectum?

8. The rectoscopy was made in the patient with a severe form of classic schigella dysentery on the 12-th day. Some morphological changes were found.

1. What was the stage of the disease in this case?
2. What following changes were there in the intestine?
3. What regime must has been prescribed for the patient and why?

9. The woman of 32 years old was admitted to the oncology dispensary with the complaints of weakness, emaciation and liquid stool. Multiple, dense conglomerates of nodes were found in the abdomen at palpation. The diagnosis of malignant tumor of the abdominal cavity was made. Than the bilateral confluent bronchopneumonia developed and the patient died. At the dissection there was no tumor, but the generalized form of yersiniosis was found.

1. What is yersiniosis? What is the way of the infection? Who is the origine of the infection?
2. What clinico-morphological forms of yersiniosis do you know?
3. What morphological specialties are there in the organism in yersiniosis?

10. The young military man, came to the hospital with the severe form of yersiniosis, with the damage of the intestine, mesenterial lymphatic nodes and with the symptoms of peritonitis. He died in several days due to generalization of the infection.

1. What morphological changes were there in the intestine and lymphatic nodes in case of yersiniosis?
2. What was the form of yersiniosis on the beginning of the disease and at its end in this case?
11. The patient was admitted to the hospital with the symptoms of a severe gastroenteritis and with the complaints of profuse diarrhea and vomiting. At the examination manifestations of dehydration were observed. The cholera vibrio was found at the bacteriologic tests. Despite of the treatment the patient died.

1. What was the stage of cholera in this case?
2. What morphological changes were there in the stomach, intestine and soft tissues?
3. What are possible causes of death in cholera?

12. The man of 30 years old arrived from India. The profuse diarrhea and vomiting appeared suddenly. The temperature was high. Cholera was confirmed by bacteriological tests. The clinical manifestations of the disease changed in some time: the skin became dry and wrinkled, the temperature decreased and the patient died due to hypovolumic coma.

1. What the stage of cholera was there in the patient?
2. Describe, please, morphological changes of his inner organs.
3. Call, please, specific and nonspecific complications of cholera.

UNIT 9
TUBERCULOSIS

Grosses

1. Ghon complex.
   It is the child lung. Ghon complex consists of:
   1. A subpleural lesion - the grey-white focus is under the pleura in the lower part of the upper lobe.
   2. Lymphangitis (it is not visible in the gross) and
   3. Enlarged whitish lymphatic node are seen. Describe this gross and call the outcomes of it.

   Under the pleura and on the section of surface you can see numerous grey-white granulomas. Describe the gross and remember microscopic features of granulomas.

   Find the plural cavities and describe the possible complications of this process. Answer the question: why is it necessary to operate the kidney with tuberculosis?

4. Fibro-cavernous pulmonary tuberculosis.
   This is the lung of the old man. Section surface shows several cavities, one of them demonstrates irregular unclear cavity lined by caseous material, into the second cavity you can see the clear smooth surface (collagenization) of it. Both demonstrate the secondary type of tuberculosis, but in different clinical stages the first stage is an acute type, the second is a remission. Look at the background. The pulmonary tissue is seen as green-yellow with black coal points – it is a caseous pneumonia (inflammation with necrotic changes). Describe this gross and call the most important extrapulmonary (renal) complication and explain the mechanism of its development.
Slide N 124  
**Primary pulmonary tuberculous focus**  
(hematoxylin and eosin)  
Small magnification: It is the child's lung with aerial alveoli. Among them there is an airless focus.
Large magnification: examine this focus: caseous necrosis is seen in the center of it in bright pink color. Around it you can see a lot of cells. Recognize them: lymphocytes, neutrophils, and macrophages. Depict this slide with large magnification.

Slide N 164 (D)  
**Lymphatic node in case of primary tuberculosis**  
(hematoxylin and eosin)  
Small magnification: Debris of lymphatic tissue is seen only on the periphery of the lymphatic node. The central part shows caseous necrosis. You already depicted this slide in the first term (unit № 1).

Slide N 131 (D)  
**Miliary tuberculosis of the lung**  
(hematoxylin and eosin)  
Small magnification: Aerial alveoli are seen. Find the airless foci.
Large magnification: Examine the airless focus in detail: it shows caseous necrosis in the central part in bright pink colour. Around it the inflammatory reaction is seen. The internal layer is formed by epithelioid cells – they have an oval shape, extra central nuclear, these cells are product transformation of macrophages. Isolated polynuclear Langhans giant cells are visible among epithelioid cells. External layer is lymphocytes. Granulomas have no blood vessels.

Slide N 168 (D)  
**Miliary tuberculosis of the liver**  
(hematoxylin and eosin)  
Examine this slide with small and large magnification, find the granulomas and describe the complications of this process.

Slide N 135  
**Tuberculosis of the kidney**  
(hematoxylin and eosin)  
Examine this slide with small and large magnification, find the granulomas and describe the complications of this process.

Slide N 126  
**Secondary pulmonary fibro-focal tuberculosis**  
(hematoxylin and eosin)  
Small magnification: examine all zones of this slide and convince them with chronic background changers of pulmonary tissue – chronic bronchitis, pneumosclerosis, atelectasis, emphysema.
Large magnification: Find two foci and describe them – one of them demonstrates central necrotic zone and inflammatory reaction with epithelioid, giant Langhans cells and lymphocytes. Second focus shows central necrosis, thin inflammatory zone and collagen capsule. Depict this slide with large magnification and mark different features of both foci.

Clinical cases
1. The primary tuberculous complex (Ghon complex) has been developed in the infant of 4 years old. The diagnosis of the primary tuberculosis has been made and the treatment was
administered. However, the lymphogenic spreading of the infection has appeared and tuberculous bronchoadenitis has been developed.

1. Describe, please, the morphology of the primary tuberculous complex.
2. What morphological changes does the tuberculous bronchoadenitis characterize?
3. What pathways of primary tuberculous spreading do you know?

2. The corpse of the boy of 6 years old who died from hematogenic spreading of the primary tuberculosis was brought for the dissection. The autopsy confirmed the presence of primary tuberculous complex and signs of primary tuberculosis spreading, specifically miliary tuberculosis of the lungs.

1. What morphological changes did confirm the presence of primary tuberculous complex and miliary tuberculosis of the lungs?

3. It is important to know the main groups of secondary tuberculosis complications fo avoiding its development.

1. What groups of secondary tuberculosis complications do you know?
2. When and why can hemorrhages be developed? What is the mechanism of their development?
3. Call, please, other certain examples of secondary tuberculosis complications of every group.

4. The patient of 62 years old has the cavitary fibrocaseous form of tuberculosis with the cavern in the upper lobe of the right lung. The patient has been treated for many years. Not long ago he was admitted to the hospital with the decompensation of chronic heart insufficiency.

1. What is the way of the cavern development in tuberculosis?
2. What is the morphology of cavitary fibrocaseous tuberculosis?
3. Why is heart insufficiency possible in case of secondary tuberculosis?
4. Describe, please, its pathogenesis.

5. The rounded shape of shadow was found in the upper lobe of lung in the woman of 45 years old at the photoroentgenography during dispensary observation. On following examination the lung tuberculoma was found. The woman was operated on. The histological investigation confirmed the diagnosis.

1. What is tuberculoma? What is its morphology? In what clinico-morphological form of tuberculosis is tuberculoma developed?
2. How can tuberculoma be developed (describe the mechanism of its formation)?

6. At the dissection of two men died from phthisiatic dispensary the acute focal tuberculosis was found in one and there was the fibrocaseous tuberculosis in the second. Compare, please, two tuberculosis forms according to the morphology.

7. The man of 40 years old has come to the polyclinic with the complaints of weakness, fever, night sweating. The acute focal tuberculosis has been found on examination.

1. What macro- and microscopic characteristics does acute focal tuberculosis have?
2. What is the Abricosov focus and what is its structure?
8. The tuberculous spondylitis was developed in the child of 12 years old. To avoid the deformation of vertebra column the plaster bandage was applied and bed regime was prescribed.

1. In what clinico-morphological form of tuberculosis does tuberculous spondylitis occur?
2. What is the dynamic of morphological changes in tuberculous spondylitis course?
3. Why is plaster bandage necessary?
4. What changes are possible in tuberculous foci in recovery?

9. A severe form of tuberculosis of the kidney was found in the young man. The patient was operated on and the kidney was removed.

1. What macro- and microscopic changes of the kidney were there in the patient?
2. Why do clinical manifestations of the kidney tuberculosis appear later than the morphological changes?
3. What structures can be damaged after in intracanalicular spreading of tuberculous infection in the kidney?

UNIT 10
INFECTIONS OF CHILDHOOD. AIDS

Grosses

1. Purulent leptomenigitis.
   Describe the picture of soft brain membraines and determine the day and the stage of the disease. Write down the description in your copybooks.

2. Fibrinous laryngo-tracheitis in case of diphtheria.
   Larynx and trachea contain thin diphtheric (fibrinous) membrane, causing respiratory obstruction and death from mechnical asphyxia.

Slide N 113
Pneumonia in case of measles
(hematoxylin and eosin)

   Small magnification: It is the child's lung. Lesions of the bronchi and interstitial tissue of the lungs are observed.
   Large magnification: Pay attention to bronchi. Disease involves all membranes of the bronchi (panbronchitis) with diffuse profuse lymphocytes infiltration. There is increasing thickness of interalveolar septas as a result of diffuse inflammatory infiltration. Depict this slide with large magnification.

Clinical cases
1. The man of 57 years old, homosexual, has been hospitalized in a grave condition with the complaints of fever and increasing of all lymphatic nodes. Also the splenomegaly, infectious injuries of gastro-intestinal tract and skin tumor were found on examination. The diagnosis was AIDS.

   1. What are the stages in the AIDS clinical course?
   2. What stage was there in this case?
   3. What changes were there in the lymphatic nodes?
   4. What tumors are usually developed in AIDS?
2. A young man, the father of two children, became ill with AIDS after the blood transfusion. The disease had a rapidly-progressive clinical course; fever, diarrhea and emaciation were developed. It was the gastrointestinal form of AIDS.

1. Why were infectious injuries in AIDS called opportunistic?
2. What are clinico-morphological features of infectious diseases in AIDS?
3. What other organ systems can be affected in AIDS (excepting gastro-intestinal tract)?

3. The child has been admitted to the hospital in moderately grave condition and with average intoxication. There are signs of fibrinous inflammation on the mucous membranes of larynx and trachea. The diagnosis of diphtheria has been made after additional investigations.

1. What form of exudative inflammation is possible there on larynx and trachea mucosa in case of diphtheria?
2. What clinico-morphological forms of diphtheria do you know?

4. The acute pain on swallowing and heavy oedema of neck soft tissues had appeared in 5 years old child. The temperature had risen up to 39°C, the condition has become worth. The whitish-yellow membranes, difficulty fixing, were on the tonsils and soft palate. Despite of treatment the child has died.

1. What disease was there in this case?
2. What pathological process was developed on tonsils and soft palate?
3. What is the name of this form of disease?
4. What possible changes are there in the organism in this form?

5. The child with the confirmed diagnosis of diphtheria has been brought to the infectious hospital.

1. What forms of fauces diphtheria do you know?
2. Describe, please, their morphological appearances.
3. What complications are possible in fauces diphtheria?

6. The mother of 2 years old child called in a doctor due to the temperature rising, a sore throat and rash on the body, s skin except nasolabial triangle of the face. The doctor made the diagnosis of scarlet fever.

1. What changes are there in tonsils, oral cavity and lymphatic nodes in scarlet fever?
2. What was the period of the scarlet fever in this case?
3. Why did rash appear and what is its outcome?

7. The patient of 40 years old had progressive deafness after scarlet fever in childhood which was complicated by otitis.

1. What clinical forms of scarlet fever can be complicated by otitis?
2. Why and in what period of disease does it happen?
3. What complications can be developed in the first and second periods of scarlet fever?

8. The 1.5 years old baby died from meningococcal meningitis with the oedema and swelling of the brain. The death occurred on the third day after the onset of the disease.
1. Describe, please, gross and microscopic changes of the soft brain membrane in this time?
2. What following changes of the soft brain membrane are possible?

9. The 3 months old baby was in the epidemic focus of meningococcal infection. He had an acute onset of the disease, high temperature and rash. The acute adrenals failure (Waterhouse-Friderichsen syndrome) was developed soon and the patient died from bacterial shock.

1. What is the name of this form of meningococcal infection?
2. What morphological changes could have been observed at the dissection there in the patient who died from this form of disease?
3. What is the Waterhouse-Friderichsen syndrome?

10. The child had meningococcal infection but he did not recover from it. In this period there was hydrocephalus in the patient.

1. What is hydrocephalus and why was it developed in this case?
2. Describe, please, morphological changes of the brain membranes and brain in this case?
EXAMINATION GROSSES
1. Amyloidosis of the kidney
2. Stones in the gallbladder
3. Hemorrhagic infarction of the lung
4. Ischemic infarction of the spleen
5. Gangrene of the intestine
6. "Nutmeg" liver
7. Trombembolism of pulmonary artery
8. Hypertrophy of the heart
9. Stones in the kidney and hydronephrosis
10. Chondroma of the finger
11. Fibromyoma of a uterus
12. Papillary cystadenoma of the ovary
13. Carcinoma of the stomach (fungiform type)
14. Carcinoma of the stomach (diffused type)
15. Cancer of an uterus neck (cervix)
16. Cancer of a breast
17. Central cancer of the lung
18. Metastatic cancer of ovaries (tumor of Krukenberg)
19. Spleen in case of Hodgkin's diseases
20. Spleen in case of chronic myeloleukemia
21. Atherosclerosis of an aorta with mural thrombus
22. Atherosclerosis of the brain vessels
23. Primary shrunken kidney
24. Hemorrhage in a brain (hematoma)
25. Acute myocardial infarction
26. Chronic aneurysm of the heart
27. Acute verrucous endocarditis in case of rheumatic fever
28. Fibrinous pericarditis
29. Lobar pneumonia
30. Bronchopneumonia
31. Bronchiectasias, pneumosclerosis and amyloidosis of the kidney and spleen
32. Chronic ulcer of a stomach
33. Micronodular (portal) cirrhosis of a liver
34. Hemorrhagic glomerulonephritis
35. Thrombo-ulcerative endocardatitis
36. Embolic purulent nephritis in case of sepsis
37. Small hemorrhages under the epicardium
38. Intestine in case of a typhoid fever
39. Fibrinous (diphtheroid) colitis
40. Necrotic tonsillitis
41. Fibrinous laryngotracheitis
42. Purulent meningitis
43. The primary pulmonary tuberculous complex (Gonh complex)
44. Miliary pulmonary tuberculosis
45. Fibro-cavernous pulmonary tuberculosis (secondary tuberculosis)
46. Cavitary tuberculosis of the kidney
47. Shock kidney (necrotic nephrosis)

EXAMINATION SLIDES
1. № 1 Nutmeg liver
2. № 26 Fatty liver (Sudan III)
3. № 19 Amyloidosis of the kidney (Congo – red)
4. № 111 Brown induration of the lung
5. № 11 Hemorrhages in the brain
6. № 5 Mixed thrombus in the blood vessel
7. № 6 Hemorrhagic infarction in the lung
8. № 7 Ischemic infarction of the kidney
9. № 153 Edema of the lung
10. № 9 (183) Necrosis of kidney convoluted tubules epithelium
11. № 40 Glandular hyperplasia of endometrium
12. № 173 Purulent leptomeningitis
13. № 75 Fibrinous (lobar) pneumonia
14. № 76 Fibronous pericarditis
15. № 131 Tuberculous granulomas in the lung
16. № 62 Squamous-cell carcinoma with keratinization of the skin
17. № 64 Adenocarcinoma of the stomach (rectum)
18. № 104 Metastasis of the cancer to the lymphatic node
19. № 50 Fibroleuomyoma of the uterus (van Gieson)
20. № 55 Nondifferentiated fibrosarcoma
21. № 49 Chondroma
22. № 51 Cavernous hemangioma of the liver
23. № 66 Fibroadenoma of the breast
24. № 120 Leukaemic infiltration in the kidney in case of acute leukaemia
25. № 44 The liver in case of myeloleukaemia
26. № 118 The lymphatic node in case of Hodgkin's disease
27. № 201 Atherosclerosis of the coronary artery
28. № 143 Acute infarction of the myocardium
29. № 97 Postinfarctive cardiosclerosis
30. № 140 Arteriolosclerotic kidney (primary shrunken kidney)
31. № 72 Bronchopneumonia
32. № 134 The recurrence verrucous endocarditis
33. № 184 Rapidly-progressive (crescentic) glomerulonephritis
34. № 234 Portal cirrhosis of the liver
35. № 100-a Cholestatic (biliary) cirrhosis of the liver
36. № 144 Chronic ulcer of the stomach
37. № 146 Acute flegmonous appendicitis
38. № 114 Fibrinous (diphtheroid) colitis in case of the dysentery
39. № 169 Small intestine in case of cholera
40. № 74 Embolic purulent nephritis
41. № 124 Primary tuberculous focus in the lung
42. № 164 Necrosis of lymphatic node in case of tuberculosis
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ПАТОЛОГИЧЕСКАЯ АНАТОМИЯ ВНУТРЕННИХ И ИНФЕКЦИОННЫХ БОЛЕЗНЕЙ
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