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SHORT COURSE OF SYSTEMIC PATHOLOGY
Part 4

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Посібник містить короткий виклад теоретичног
матеріалу основних тем системної патоморфології, ш
відповідає програмі, затвердженій МОЗ України і ЦМК з вищ
медичної освіти. У посібнику представлені цифрові мікро-т
макрофотознімки, викладений їх опис та наведені приклад
тестових і ситуаційних завдань до кожного заняття.

Для англомовних студентів вищих медичних навчальни
закладів III-IV рівнів акредитації.

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Content module 6

Theme 28 *Diseases of the nervous system Cerebro-vascular illness.*

Concrete aims:

- *To interpret the concept of cerebro-vascular illness, risk faktors, background diseases at it.*
- *To estimate causes, pathogenesis and morphological displays of ischemic, hemorrhagic and mixed stroke of cerebrum.*
- *To interpret morphological changes at a spontaneous intracranial and intracerebral hemorrhage, which arise as a result of defeats of vessels of different etiology.*
- *To interpret morphological changes in illness of Alzheimer's*
- *To interpret morphological changes in demielinisated diseases (a sclerosis is dissipated).*
- *To interpret morphological changes in a lateral amyotrophic sclerosis.*
- *To interpret morphological changes in postreanimated encephalopathy.*
- *To interpret etiologic factors and morphological changes at the infectious diseases of the central nervous system*

Subject Actuality: diseases of the nervous system, especially connected with vascular pathology, occupy a considerable place in the general structure of morbidity, invalidisation and death rate of man. With them there are doctors of all specialities in practical activity. That is why knowledge of morphological bases of this pathology are needed not only at the study of their clinical displays but also for a clinic-anatomic analysis, determination of tactic of treatment and prophylaxis.

Aim: to learn etiology, pathogenesis, morphological displays of cerebro-vascular illness, illness of dissipated Alzheimer's sclerosis, lateral amyotrophic sclerosis, postreanimation encephalopathy, infectious diseases of cerebrum, and also able to distinguish their clinical-morphological forms and most frequent complications.

Task: 1. To know etiology, pathogenesis, morphological displays of illnesses of the nervous system.

2. To learn to diagnose the morphological displays of the noted illnesses on macroscopic and microscopic levels.

3. Able to distinguish clinical-morphological forms and most frequent complications, and also reasons of death in illnesses of the nervous system.

The main questions for the individual training:

Cerebro-vascular illness. Determination. Epidemiology and risk factors, classification, background diseases. The defeat of brain is at ischemia.

Heart attack (ischemic stroke) of cerebrum. Clinical displays, reasons, pathogenesis. A value of atherosclerosis of cerebral arteries and defeats of arteries is in other diseases. Morphological classification and description, consequences.

Selective necrosis of neurons (ischemic encephalopathy). Reasons,, morphological description.

Hemorrhagic stroke. A heart attack is mixed.

Defeat of vessels of brain of different etiology. Aneurysm is at atherosclerosis and other diseases. Primary and second thrombosis of veins of brain and venous sinus of brain-tunic. Reasons, clinical value, complication.

Spontaneous intracranial hemorrhage. Intracerebral hemorrhage (intracranial haematoma). Subarachoid hemorrhage.

Theme 29 Illness of Alzheimer. A sclerosis is dissipated. Lateral amyotrophic sclerosis. Postreanimation encephalopathy. Illnesses of the peripheral nervous system. Infectious diseases (viral, tick encephalitis)

The main questions for the individual training:

Changes are at a senescence, degenerative processes and dementia. Primary and second dementia. Illness of Alzheimer Etiology, pathogenesis, morphological description, clinical displays. Illness of Lance. Illness of Huntington. Clinical -morphological description.

Demielinisation diseases. Classification. A sclerosis is dissipated. Sharp disseminated encephalomyelitis. Sharp hemorrhagic leukoencephalitis. Etiology, patho- and, morphological description, clinical displays.

Lateral amyotrophic sclerosis: etiology, pathogenesis and morphological changes. Postreanimation encephalopathy, pathological anatomy and evolution of mosaic hearth anoxic damages of head and spinal brain. Features of morphogenesis and pathoanatomical diagnostics.

Disease of peripheral nerves and paraganglia. Damage of peripheral nerves. Degenerative changes are in peripheral nerves. Peripheral neurology. Diabetic, uremia and other forms of neurology.

Festering infections: meningitis (leptomeningitis, pachimeningitis), abscess of cerebrum. Etiology, morphological description, consequences. Meningococcal meningitis: ways of passing to the infection, morphology, clinical syndromes, complications, reasons of death.

Unfestering infections of the central nervous system. Tubercular meningitis and tuberculoma. Neurosyphilis: tertiary and parenchimatous. clinical-morphological description.

Mycosis infections: etiology, morphological description.

Viral infectious diseases of the central nervous system. Classification, typical clinical displays. Aseptic meningitis. Sharp viral encephalitis. Morphological features. Neuroinfections which are caused by viruses of simple herpes, windy pox and belting herpes. Morphological description, clinical displays. Cytomegalovirus infection. Enterovirus disease of the central nervous system. Poliomyelitis.

Diseases of the central nervous system of, which are caused by arbovirus. Tick encephalitis. Hydrophobia. Pathogenesis, morphological description, clinical displays, reasons of death. Slow viral neuroinfections and prion illnesses (Kuru, illness of Kreyttsfeldt – Yakob's, prion illnesses of animals). Epidemiology, ways of passing to the infection, pathogenesis, morphological description, clinical displays. Influencing of HIV on the central nervous system.

Lesson equipment:

Macropreparations: atherosclerosis of arteries of cerebrum, ischemic attack of cerebrum, postinfarktion cyst of brain, hemorrhage in a cerebrum, spinal cord at the dissipated sclerosis, spinal cord at a lateral amyotrophic sclerosis (illness of Sharko), cerebrum at postreanimation encephalopathy.

Micropreparations: hemorrhagic heart attack of cerebrum (paints. hematoxylin and eosine), ischemic stroke of cerebrum (paints. hematoxylin and eosine), cerebral cortex in illness of Alzheimer (paints. hematoxylin and eozine), front horns of spinal cord at a lateral amyotrophic sclerosis (paints. Hematoxylin and eosine), perivenous demielinisation at the dissipated sclerosis (paints. Hematoxylin and eosine), selective necrosis of neurons (ischemic encephalopathy) (paints. hematoxylin and eosine).

Sliding seats and tables which are in the archive of department, for example: a hemorrhage is in a cerebrum.

I Preauditorium individual training for the practical training

Theoretic Material Summary

Cerebro-vascular disease is illnesses of cerebrum, which arises up on the soil of violation of circulation of the blood. In case of their large frequency of morbidity and death rate they are selected in the independent group of diseases with the proper code in international classification of diseases. A background for them is atherosclerosis and hypertensive illness, born anomalies of development of vessels of brain, arteritis, and hemorrhagic diathesis. Risk factors can be saccharine diabetes, atherosclerosis of coronal arteries, cardiac insufficiency, obesity, smoking of cigarettes, alcoholism

Etiology and pathogenesis. Direct reasons: spasm, thrombosis, tromboembolism of cerebral and precerebral arteries. A considerable place is taken a psychoemotional overstrain.

Classification. Distinguished transitory ischemic encephalopathy, selective necrosis of neurons, ischemic and hemorrhagic stroke.

Morphology. At the transitory ischemia of cerebrum in edema, dystrophic changes, is marked in nervous mews, single shallow hemorrhage, laying of hemosiderin at chronic motion.

Selective necrosis of neurons can have diffuse character or focus – after the attacks of hypotension. Diffuse selective necrosis of neurons, which is observed at the stop of heart, leads to death of patients in a few days. Thus in the cerebrum of displays of heart attack does not find. Only on a microscopic level widespread necrosis of neurons appears especially in hippocampus, III, V, VI layers of bark of cerebrum. Focus selective necrosis of neurons arises up after the attacks of hypotension and often meets in areas between the arterial pools of brain and cerebellum. The most remote from arterial vessels structures of cerebrum suffer.

An ischemic stroke(heart attack) arises up as a result of stopping of arterial blood supply of cerebrum through thrombosis of the atherosclerotic changed vessels of brain. The morphological display of ischemic stroke can be ischemic, hemorrhagic and a heart attack is mixed. In the mixed heart attack it is possible to find the areas of both ischemic and hemorrhagic heart attack. He more frequent arises up in the grey matter of brain. At an ischemic stroke circulatory ischemic necrosis which looks as a cell of grey softening cerebral matter develops in the brain;

A hemorrhagic stroke shows up: by a intracranial haematoma, hemorrhagic impregnation of matter of cerebrum, subarachnoid hemorrhage. A spontaneous intracranial hemorrhage often meets at hypertension (cerebral hemorrhage) and break of aneurysm of arteries (subarachnoid hemorrhage). Reason of spontaneous intracranial hemorrhages can be bleeding at a sharp leucosis, hemorrhage in the tumor of primary or metastasic origin. A cerebral hemorrhage (hemorrhagic stroke, cerebral apoplexy) develops at the break of microaneurysm of artery which is often formed for patients with arterial hypertension. At a hemorrhagic stroke, saturating with blood of the damaged area of brain is marked additionally with development of haematoma of brain. In the place of hemorrhage fabric of brain collapses and is softened – red softening to the brain. In the first days a cerebral haematoma is an area of brain, which is presented the blasted matter of brain from pr

The primary (uninfectious or stagnant or marantic) thrombosis of veins of brain and venous sinus of brain-tunic develops more frequent for the exhausted or dehydrated children which suffer a heavy infectious disease, rarer – for adults with stagnant cardiac insufficiency, at hematological diseases, at complications pregnancy or post-natal period. Investigation is development of venous heart attacks of

cerebrum. The second or septic thrombosis of veins and venous sinus arises up at nasty infectious illnesses, infections of middle ear, opened breaks of bones of skull.

Infectious diseases of the nervous system.

Festering infections in a cerebrum cause meningitis-inflammation of his space or encephalitis – inflammation of matter of cerebrum. After localization meningitis can be: pachymeningitis is inflammation of hard brain-tunic, leptomeningitis is inflammation of vascular and arachnoid.

Leptomeningitis arises up after penetration of infection (meningococcus, pneumococcus, intestinal stick is in subarachnoid space. Characteristic is the hematogenic infecting and air-drop way of infection. At morphological research find pus in subarachnoid intracranial and spinal spaces, at crinkles hemispheres and on the basis of brain. In ventricles a turbid cerebrospinal liquid, fibrin, pus, appears on walls and vascular interlacement, hydrocephaly.

Pachymeningitis – sharp inflammation of hard brain-tunic more frequent arises up at distribution of festering inflammatory process from the bones of skull at otitis, breaks of bones of skull.

Abscess of cerebrum in unfesterings infections in a cerebrum most often arise up the second time at tuberculosis and syphilis. At tuberculosis in a cerebrum often tubercular meningitis develops and tuberkuloma.

Tubercular meningitis arises up at hematogenic distribution of exciter. Macroscopically exudate has a cheese (caseation) kind and appears in the cisterns of basis of brain and round a spinal cord. In vascular and arachoid space it is possible to see shallow by the diameter of 1-2мм white humps. Develops the difficulty current of cerebrospinal liquid and hydrocephaly develops. Fibrous-caseation exudate, infiltration by lympho- and plasmocytes, macrophagocytes. *Tuberkuloma* is the hearth of caseation necrosis is

encapsulated meets in the large hemispheres of brain or in a cerebellum for children.

The defeat of cerebrum at syphilis can show up as tertiary and parenchimatous neurosyphilis.

Tertiary neurosyphilis shows subsharp meningitis from lymphocytes and by plasmocytes infiltration of subarachoid space, typically is periarteritis and obliterated endarteritis is a characteristic sign of meningovascular syphilis. Investigation can be ischemic defeats of cerebrum, and also counterfoils cranial and spinal nerves. There are rubbers with necrosis of fabric of brain.

Parenchimatous neurosyphilis shows up a subsharp encephalitis with making progress paralysis, by psychical disorders, making progress dementia and atrophy of cerebrum. Macroscopically the narrow appear and rounded bend, wide furrows, extended ventricles, sometimes granulomatous epedidimitis. Microscopically limphoplasmocytes perivasculyar infiltrate in the matter of brain and subarachoid space is found.

Mycosis infections of the nervous system are always the second defeat at mycosis. Among neuromycosis select cryptococcosis, mucormycosis, candidosis and etc. Cryptococcosis shows up as a subsharp meningitis; in exudate the masses of encapsulated Cryptococcus is found. In superficial layers of cortex of brain there are cysts which are filled Cryptococcus. Often opportunism mycosis joins with forming of abscesses of brain. Mucormycosis arises up for patients with saccharine diabetes and characterized the defeat of frontal particles of cerebrum.

The viral infections of cerebrum are characterized as the development of aseptic meningitis and encephalitis. Viruses can get in the brain of hematogenic by a way or perineural. *Aseptic* meningitis often develops at child's age, caused by

enterovirus or virus of epidemic parotitis. Morphological displays are insignificant.

A sharp viral encephalitis is characterized by the presence of lymphocytes and plasmocytes in the subarachoid space, lymphoplasmocytes perivascular muffs, mononuclear inflammatory infiltrate, which consists of lymphocytes, plasmatic mews and macrophagocytes, diffuse hyperplasia of microglia and oligodendria with formation of rod-shaped and amoebo like mews, astrotcytosis, areas of destruction of matter of cerebrum, chromatolisis, intranuclear and intracellular including, necrosis of neurons. Encephalitis which characterized caused by virus of simple herpes in addition by development of areas of heart attacks with the defeat of white and grey matters. Hydrophobia is caused by rabdovirus

Changes of the central nervous system are at a senescence, degenerative processes, dementia.

At senescence after 65 often find atrophy changes in the hemispheres of cerebrum, bulges of spaces, extended furrows especially in frontal and temporal particles. Some refining of cortex of large hemispheres, diminishing of amount of grey matter and expansion of the system of ventricles appears on a cut. At a microscopy the insignificant loss of neurons is found, there are senile name-plates in a grey matter, sometimes grainy and vacuole degeneration of nervous fibres.

Dementia shows disorders of higher nervous activity and arises the development of areas, of destruction or disorganization in the bark of cerebrum, white matter, subcortical kernels.

Causes of dementia: 1-primary dementia: illness of Alzgeimer, Lance, Gentington, Parkinson; 2- second dementia: vascular pathology (plural heart attacks of cerebrum, system red lupus), cranial-cerebral trauma (posttraumatic encephalopathy, subdural haematoma), infections

(neurosyphilis with making progress paralysis and by psychical disorders, illness of Kreyttsfeld-Yakob, AIDS), hydrocephalia at normal intracranial pressure, heavy intoxications and metabolic disorders.

Illness of Alzheimer's or presenile and senile imbecility, which shows up making progress degenerative changes in the nervous system, which begin to develop after 40-65years and accompanied laying of pathological albumen, – senile an amyloid and by neurofibrilar changes, atrophy of brain and hydrocephaly.

A lateral amyotrophic sclerosis (illness of Sharko) is a making progress disease of the nervous system with the defeat of motive neurons of front and lateral posts of spinal cord and peripheral nerves. Spastic paresthesia of muscles are developed; hands with atrophy of muscles but by the increase of tendon and pariosteum reflexes. Causes of illness can be a chronic viral infection, immunological and metabolic violations. At morphological research find atrophy of front motive counterfoils of spinal cord, compression of lateral cortical spinal ways, atrophy of precerebral bend of brain, atrophy of skeletal muscles. At a microscopy the dystrophic and destructive changes of nervous mews appear in the front horns of spinal cord, them demielinisation with swelling, disintegration and death of axial cylinders. Sometimes demielinisation spreads on peripheral nerves with the defeat of pyramid ways on all of draught. There is lymphoid infiltrate in tissue.

The dissipated sclerosis is characterized by the development of cells of demielinisation of white substance of cerebrum and spinal brain with next excrescence of glia and sclerosis. Clinically illness begins for a young people and shows up shaking, nystagmus, scanned language, increase of tendon reflexes, spastic paralyses, disorders of sight. Consider a viral infection reason of disease with development of

processes of autoimunisation. Morphologically in tissue of cerebrum are found the cells of grey color round ventricles. Visual nerves are often damaged, chiasm, visual ways. At the microscopy of hearth of the demielinisation find round vessels (perivenous demielinisation) from lymphocytes and by mononuclear infiltration. At progress of illness the perivascular cells of demielinisation meet, typical name-plates are formed with blasted olygodendrocytes.

Sharp disseminated encephalomyelitis develops early in life after viral infection (epidemic parotitis, measles, windy pox, german measles) and accompanied by the diffuse defeat of head and spinal brain as hearths of perivenous demielinisation, inflammatory edema, neutrophil, and later lymphomacrophagic infiltrations. In development of illness a considerable place is taken by immune reactions.

Sharp hemorrhagic leucoencephalitis develops after viral infections, septic shock, medicinal therapy and others like that. Morphologically the edema of cerebrum is found, numerous point hemorrhages in a white matter, hearths of necrosis, wall, vessels, perivascular area of demielinisation with neutrophil, and later by lymphoplasmocytes infiltration. In development of illness a considerable place is taken by immunopatological processes.

II Algorithm of the practical part of the lesson.

Study and be ready for the verbal description of the macropreparations

1 Atherosclerosis of arteries of cerebrum. The vessels of basis of cerebrum are deformed as a result of presence of atherosclerotic name-plates which are translucent through a wall and disposed as files. Gleam of vessels in the places are narrowed, and in places, free of them, find out aneurysm. Predetermines narrowing of gleam of artery atherosclerotic name-plates permanent anaemia fabrics of cerebrum, hypoxia

conduces to the dystrophic and sclerotic changes, that such changes conduce to the decline the function of brain which is clinically expressed age-old forgetfulness.

2 *Hemorrhage in a cerebrum.* The hemisphere of cerebrum is megascopic, twirling of it is oblate, furrows are smoothed out. In the place of hemorrhage fabric of brain is blasted with formation of cavity, filled with convolute blood. Dug up cerebral arteries comes, as a rule, during a hypertensive crisis, in the place of microaneurism.

3 *Ischemic infarction of brain.* On the cut of hemisphere of cerebrum the area of destruction of matter of brain of grey color is visible by a size to 8см, which does not have a clear structure and reminds porridge-like mass.

4 *Postinfarktion cyst of cerebrum.* On the cut of hemisphere of cerebrum a cavity is visible in the matter of brain by a size to 3см. On periphery of cyst the brown colouring of fabric of brain is marked.

5 *A cerebrum at postreanimation encephalopathy.* On preparation of the cerebrum of the sick which long time reanimation measures were conducted after the offensive of stop of heart is presented. Fabric of cerebrum has soft but viscous consistency without saving of clear forms, structure of white and grey matters.

Study the micropreparations of the theme and be ready to show on the picture the main points of the pathologic process with the following descriptions:

1 Hemorrhagic attack of cerebrum. Colouring of hematoxilin and eosin. In the matter of cerebrum evidently red spots are blood out of vessels. It is an edge of haematoma.

2 *Ischemic stroke of cerebrum.* Colouring of hematoxylin and eosin. In the matter of cerebrum evidently area of fabric which fully lost neurons and is coloured in a rose color is an ischemic heart attack in the center of which the matter of cerebrum is

fully blasted. Find a vessel in which evidently there is a blood clot. **To designate:** 1-attack of tissue of cerebrum, 2- neurons without nucleus, 3- blood clot in a vessel .

Situation Tasks:

1 A patient is 85 years delivered in a clinic with the phenomena of hemiparesis. To it conducted itself inadequately, forgot the pas, and couldn't go back into the apartment. What reason of the indicated symptoms?

2 Patient with hypertensive illness lost consciousness suddenly, hemiparesis developed. Are there what possible morphological changes from the side of cerebrum?

Answers to the Situation Tasks:

1 Atherosclerosis of vessels to cerebrum, atrophy of bark, senile silliness.

2 A hemorrhage in the cerebrum.

Test Tasks:

1 For a woman, 92, which died at the phenomena of cardiovascular insufficiency, at life the phenomena of forgetfulness were marked, it is set on a section, that bend of cerebrum narrow, furrows are deep. In the vessels of basis of brain numerous atherosclerotic name-plates which close 2/3 orifice. Specify the credible displays of pathology of cerebrum.

A. Sharp ischemia.
B. It was swollen.
C. Swelling.
D. Heart attack.
E. Atrophy.

2 У of patient on a section the thrombosis of left middle cerebral arteria is found. The hearth of grey softening of

cerebrum is found in the area of left hemisphere;. It is needed to define a pathological process which developed for a patient.

A. Ischemic attack.
B. Hemorrhagic attack.
C. Swelling.
D. Abscess.
E. Atrophy.

3 For a woman years, which long time suffered from the atherosclerotic defeat of vessels of cerebrum, on a section, diminishing of sizes of hemispheres, smoothing of crinkles and deepening of fissure are found. What is the reason of atrophy changes in main of dying woman?

A. From insufficient blood supply.
B. From high pressure.
C. From actions of physical and chemical factors.
D. From decline of function.
E. From violation of innervation.

Answers to the Test Tasks:
1. E.; 2.A.; 3.A.

Illustrations to theme

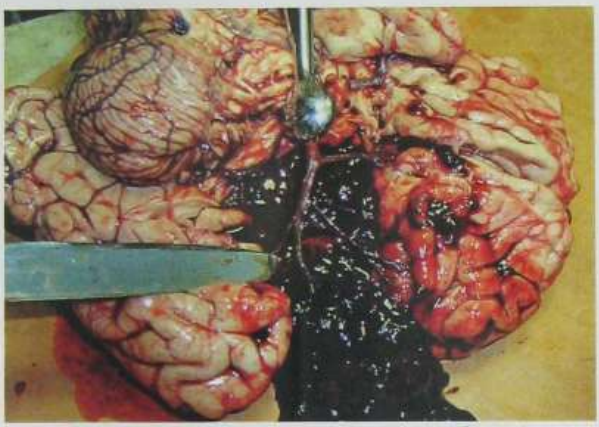

Figure 1 – Atherosclerosis of arteries of cerebrum and hemorrhage in a cerebrum.

Figure 2 – Hemorrhage in a cerebrum.

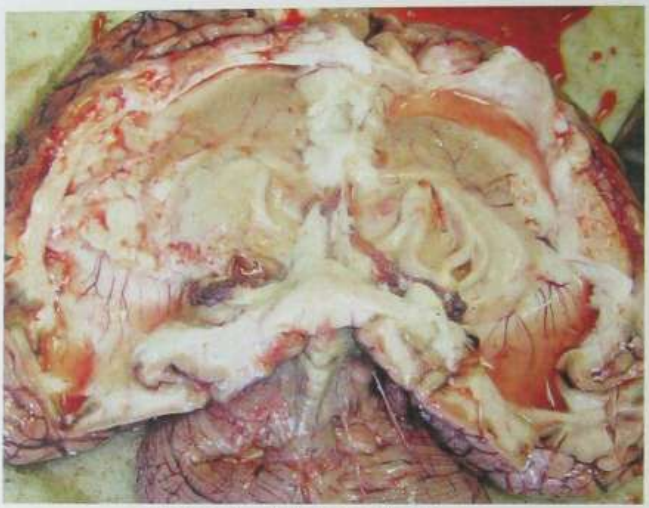

Figure 3 – Hydrocephaly.

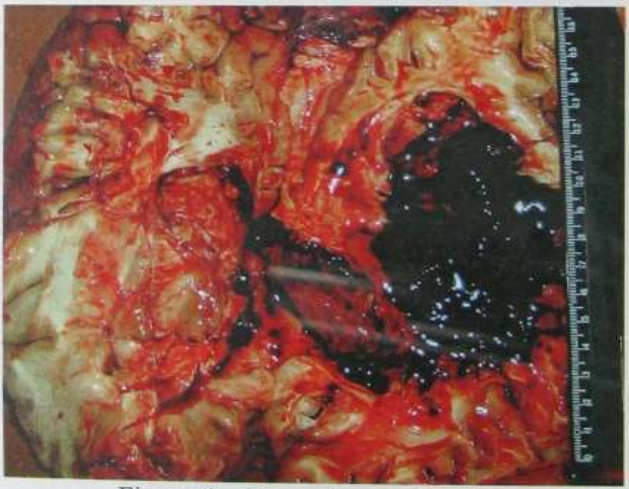

Figure 4 – Haematoma of brain.

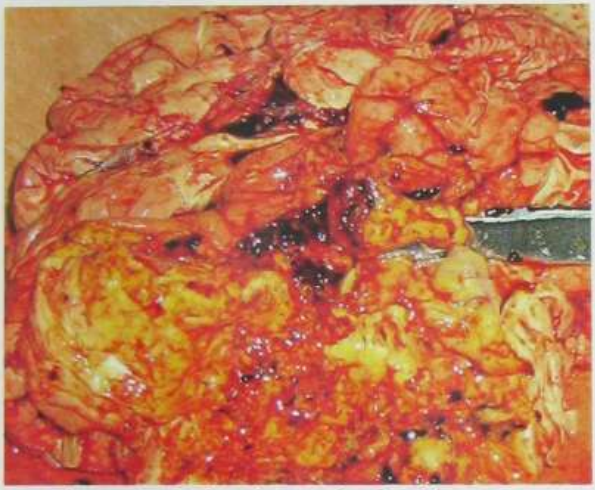

Figure 5 – Ischemic attack of cerebrum

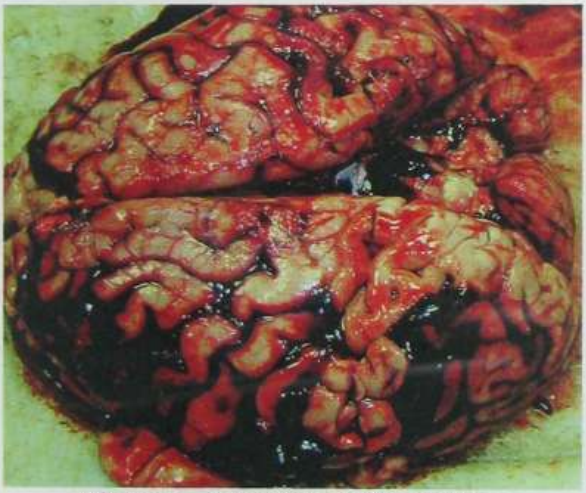

Figure 6 – Subarachnoid hemorrhage.

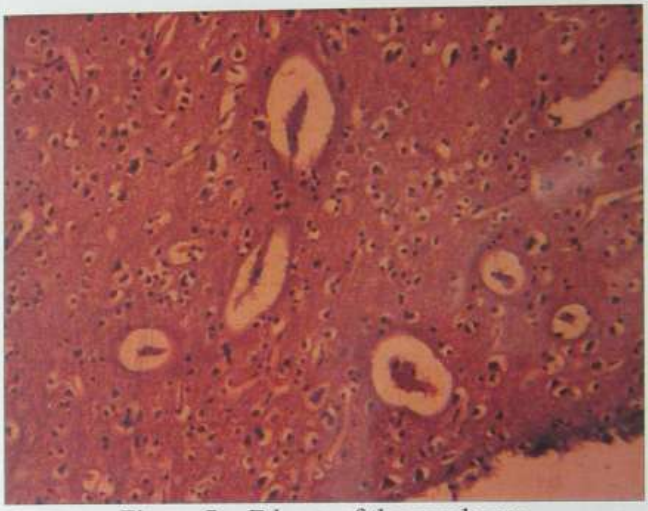

Figure 7 – Edema of the cerebrum.

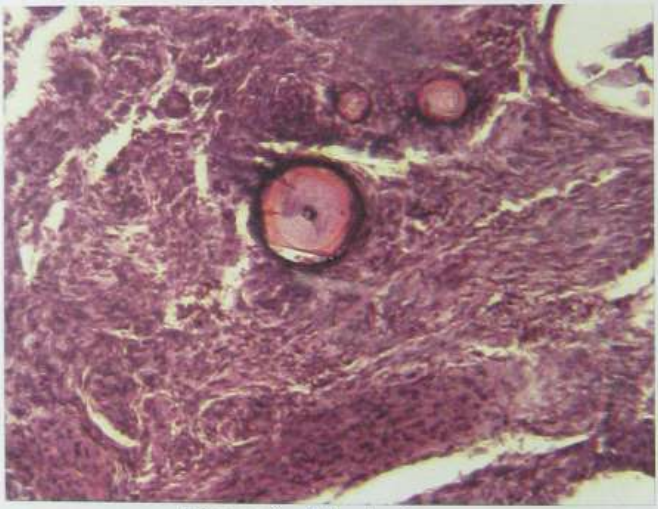

Figure 8 – Meningioma.

Theme*30 The diseases of respiratory organs*
Specific aims:

- *To estimate the role of aerohematic barrier, mechanisms of clearning of lungs in development of pulmonary pathology.*
- *To interpret etiology, patho- and morphogenesis, clinical-morphological signs, complications, consequences of diseases of upper respiratory tract.*
- *To interpret etiology, patho- and morphogenesis, clinical-morphological signs, complications, consequences of acute bronchitis.*
- *To interpret modern classification of acute pneumonias.*
- *To interpret etiology, patho- and morphogenesis, clinical-morphological signs, complications, consequences of acute pneumonias.*
- *To interpret etiology, patho- and morphogenesis, clinical-morphological signs, complications, consequences of acute destructive processes of lungs.*
- *To interpret etiology, patho- and morphogenesis, clinical-morphological signs, complications, consequences of diseases of lungs of vascular origin.*
- *To definition of chronic nonspecific diseases of lungs according to modern classification.*
- *To interpret etiology, and morphogenesis, morphological signs complications and consequences of chronic bronchitis.*
- *To interpret etiology, and morphogenesis, morphological signs complications and consequences of chronic obstructive emphysema.*
- *To interpret etiology, and morphogenesis, morphological signs complications and consequences of bronchoectatic disease.*
- *To interpret etiology, and morphogenesis, morphological signs complications and consequences of bronchial asthma.*

- *To interpret etiology, and morphogenesis, morphological signs complications and consequences of diffuse interstitial diseases of lungs.*
- *To interpret the pre-tumors (pre-cancer) conditions and changes in tumorous diseases of bronchi and lungs.*
- *To interpret the morphological and clinical features of malignant tumors of bronchi and lungs, pathways of their metastasis.*

Subject Actuality: the diseases of respiratory organs occupy the 3-4 place after traumatism, cardio-vascular illnesses and tumor processes. Quantity of them steadily grows not only in the structure of morbidity but also in invalidisation and death rate. Mainly it is polyetiological illnesses which can be as independent nosology (croupous pneumonia), sign or complication (bronchopneumonia). Knowledge is purchased at the study of this theme necessary for understanding of clinical signs of illness at the study of them on clinical departments and in practical work of doctor for a clinical-morphological analysis and bases of pathogenic therapy.

Aim: to learn etiology, pathogenesis, classification, morphological signs, complications and consequences of diseases of breathing organs.

Task:

1 To know the classification of pneumonias, bronchitis, cancer of lungs, etiology, features of pathogenesis of croupous and focus pneumonias.

2 To learn the features of pathogenesis of croupous and focus pneumonia.

3 To be able to diagnose the stages of croupous pneumonia by macro- and microscopic signs.

4 To learn to diagnose croupous pneumonia, focus pneumonias, cancer on macro and microscopic level.

5 To know and to be able to distinguish complication and consequences of illnesses of respiratory organs.

The main questions for the individual training:

1 As a result of morphological features of structure of lungs sharp inflammations have mainly exudate character. pneumonia is classified after etiology, character of exudate, prevalence and duration of motion of process.

2 In etiopathogenesis illnesses, except for the infectious, infectiously toxic influencing, an especially important value has reactivity of organism and age sick. In our time infectiously allergic pathology comes forward on the first plan.

3 Croupous pneumonia is independent nosology unit, bronchopneumonia – complication or sign of other illnesses.

4 A chronic bronchitis comes forward the basic sign of chronic diseases of lungs, emphysema, bronchioectasis and cancer develops on his basis.

5 The chronic diseases of lungs are accompanied by the changes of the right heart, exchange of matters (to dystrophy), violation of hemo- and lymphodinamic.

Lesson equipment:

Macropreparations: croupous pneumonia, carnification of lung, focus bronchopneumonia, abscess of lung, empyema of pleura, pertaining to apostema nephritis, bronkhoektasis,, chronic abscess of lung, emphysema of lung, pulmonary heart, eccentric hypertrophy of heart, amyloidosis of kidney, cancers of bronchial tube, peripheral cancer of lung, anthrakosis of lung.

Micropreparations: croupous pneumonia in the stage of grey hepatization, carnification of lungs at croupous pneumonia, festering bronchopneumonia, chronic abscess of lungs, bronchioectasis, emphysema,, cancer of lungs (planocellular nonkeratoid), amyloidosis of kidney, silicosis (anthracosis) of lungs.

Tables, sliding seats and electron diffraction pattern, which are in the archive of department, for example: resorption of

fibrin at croupous pneumonia, intracapillary sclerosis at emphysema, croupous pneumonia, focus pneumonia, abscess of lungs, empyema of pleura, pertaining to apostema nephrite, emphysema of lungs, pulmonary heart, eccentric hypertrophy of myocardium, amyloidosis of kidney.

I Preauditorium individual training for the practical training

Theoretic Material Summary

Pneumonia (pneumonia) is a disease, which unites the large group of various after etiology, pathogenesis and morphological description of inflammations of respiratory department of lungs. There are three ways of penetration of exciter of pneumonia in lungs - bronchogenic, hematogenic and lymphogenic. The first of them has a leading value. At first an inflammatory process takes bronchiole, and then spreads on parenchyma of lungs (bronchopneumonia). If inflammation has mostly productively exudative character, passes to the interalveolar partitions, it is talked about interstitial pneumonia (pneumonia interstitials). Next to it, there is an independent infectious disease, which shows up in that among a complete health sharply catches a fire fibrinous inflammation of parenchyma of lungs is parenchymatos (croupous pneumonia) pneumonia.

Croupous pneumonia – in 95 % cases caused by pneumococcus of Frenkel, rarer – diplobacillus of Fridlender, by streptococcus, staphylococcus, and stick of Pfeiffer. A cold which reduces immunobiological reactivity comes forward as a provoking factor. Illness often arises up in persons with alcoholism, avitaminosis, cardiac insufficiency, chronic overstrain. Morphological changes at croupous pneumonia test a certain evolution, which enables to select a few stages of process (K.Rokitansky) - stage of wave (from 12 god to 3

days), stage of red hepatization (1-3 days), stage of grey hepatization (2-6 days), stage of completion.

Pneumonia begins with the small hearth of inflammation in the back or back-lateral departments of lungs round the colonies of pneumococcus. Inflammation spreads a contact way and quickly enough takes one or a few pulmonary particles. In the stage of wave a lung is megascopic in a volume, tissue of it filling out and sanguineous. In the stage red hepatization exudate is enriched with fibrin and red corpuscles. Lungs after closeness remind a liver, on a cut – crimson. The color of phlegm is ferruginous. On the 4-6th day composition of exudate changes – red corpuscles disappear, but the number of neutrophils which phagocyte pneumococcus grows. A surface of lungs is grey color on the cut (stage of grey grained detritus it is possible to find tailings of fibrins hepatization). In the period of convalescence exudate resolves.

Complications of croupous pneumonia are divided into lung and extralung. Carnification belongs to the first, empyema of pleura, abscess formation, and gangrene. Extralung complication are pneumococcus inflammatory processes in different organs (lymphadenitis, meningitis, peritonitis, arthritis, etc.). The term "focus pneumonia (bronchopneumonia)" unites different originally inflammations of lungs the general line of which is localization of primary process in bronchial tubes. From here inflammation passes to pulmonary tissue and can be limited acinus, by a particle, segment or particle. Focus pneumonia occurs more frequently, than croupous. As children and people have an independent disease for years. Focus pneumonia is complicated by sharp respiratory and viral diseases (flu, measles). It can arise up at insufficiency of circulation of blood, especially on a background of the stagnant phenomena in lungs (stagnant pneumonia), at the protracted confinement to bed mode for

heavy and weakened patients (hypostatic pneumonia), in postoperation period.

In most cases the cause of bronchopneumonia is the aerogenic infection, but hematogenic is not eliminated and lymphogenic ways of infecting. The process begins with bronchiole and passes to alveolar motions. To the bronchitis can join peribronchitis. From peribronchial tissue a process passes to the nearby alveolar ways (peribronchial pneumonia). Inflammation of alveolar tissue quite often is preceded by the slump of alveolar ways. It can be consequence of clench from outside or corking of bronchial tube with exudates and next suction of air from alveolar ways which lost connection with respiratory ways.

Atelectasis is an active slump of pulmonary tissue, which can arise up at the shortage of surfactant, a collapse is a passive slump under pressure of exudate, air or tumor. The exception of part of alveolar ways from a respiratory function causes development of vicarious (compensate) emphysema. Exudate at bronchopneumonia consists of serous liquid with the admixture of leucocytes, falling off cells of alveolar epithelium, red corpuscles, at times to the fibrin. That is why serous, festering, desquamation, hemorrhagic and fibrinous pneumonia is distinguished.

Macroscopically we separate inflammatory focuses which correspond to the staggered bronchial tubes or particles appear in lungs. They burst above the surface of cut, have yellow grey, grey or red color, dense by touch, sink in water. A turbid liquid which does not contain the blisters of air flows down during squeezing of them. From bronchiole mucus-festering exudate is pressed out.

Bronchopneumonia mostly ends with convalescence, but complications - gangrene of lungs is possible, carnification. Interstitial (intermediate) pneumonia spreads mainly on intermediate tissue, here in gleam of alveolar ways.

Intermediate pneumonia belongs to the atypical forms. It is met at viral infections, croupous pneumonia. The process begins with bronchitis with the following distribution on lymphatic ways (lymphangitis) or vasculitis (system red lupus). Productive inflammation prevails at times (measles). More frequent is festering lymphangitis. Distinguished peribronchial, interlobular and interalveolar pneumonia. Macroscopically rather yellow ribbons which mark off particles one from one induration evidently. Sometimes at festering inflammation the intervals of sequestrum and particles become separated. Such pneumonia assists development of interstitial emphysema. Complication are abscess formation, empyema, mediastinitis.

Pneumonia of children has some features:

a) inflammatory process develops mainly in the respiratory departments of lungs;

b) infecting takes place antenatal intrauterine or through aspiration of amniotic waters;

c) hyaline membranes appear as a result of high permeability of blood vessels;

d) infection is more frequent than in adults, spreads outside lungs – on kidneys, liver, cerebrum.

Bronchitis is divided into sharp and chronic bronchitis (bronchitis acuta, bronchitis chronica). Among the etiologic factors of the sharp inflammation of bronchial tubes of primary value is given to viruses and bacteria which cause respirator diseases. From physical factors it follows to select the pathogenic action of dry or cold air, dust, from chemical is breathing in tobacco smoke, steams of chlorine, oxides of nitrogen and etc. The inherited insolvency of barrier mechanisms of mucus, insufficiency of cellular and humoral (IGA) protective factors of local value, assists to the development of bronchitis. In reply to the pathogenic influencing of gland and goblet cells of mucus bronchial tubes

producing of mucus increases. It results in peeling of ciliary prismatic epithelium, baring of mucus and penetration of infection through the membrane of bronchial tube.

Acute bronchitis can be of independent nosology or the secondary sign of the row of other illnesses (croupous pneumonia, uremia and others like that). In mucus bronchial tubes almost all forms of catarrhal inflammation are developed – serous, festering, fibrinous, fibrinous-hemorrhagic, mucus. Destruction of mucus is sometimes possible with the development of ulcers. In such cases it is talked about destructively ulcerous bronchitis. Predominance of that or other form of catarrh depends on the type of pathogenic factor and resistance of organism. Inflammation begins from a mucus membrane (endobronchitis), then passes to the muscular layer (endomesobronchitis) and in a terminal phase takes all of layers (panbronchitis). Certainly, an inflammatory process can be stopped at the development on a certain layer.

Existing of sharp bronchitis can be complicated with bronchopneumonia or peribronchial by intermediate pneumonia. Bronchopneumonia is mostly the result of aspiration of the infected mucus in the respiratory department of lungs. Peribronchial intermediate pneumonia arises up as a result of transition of inflammation not only on peribronchial but also on interstitial tissue.

Serous and mucus catarrh quickly ends with convalescence. Festering, fibrinous and fibrinous-hemorrhagic catarrh, and also an ulcerous-destructive bronchitis have the protracted motion and often pass to the chronic form or pneumonia.

Chronic inflammation of bronchial tubes is shown up in such forms:

a) chronic mucus or festering catarrh with atrophy of mucus, by the cystous regeneration of glands and metaplasia of prismatic epithelium in multi-layered flat;

b) chronic productive inflammation is with formation of polyposis from granulation tissue (polyposis chronic bronchitis);

c) deformation of bronchial tube at ripening of granulation tissue, excrescence of connecting tissue in a muscular layer, sclerosis and atrophy of mucus (deforming chronic bronchitis).

Chronic bronchitis with the protracted motion, except for sclerotic changes, is accompanied by dystrophy elastic, muscular and cartilaginous frameworks. That is why during a cough, when intrabronchial pressure grows sharply, in the areas of the least resistance the membrane of bronchial tube is changed broadens and bursts. So saccade bronchioectasis appear. At diffuse expansion of bronchial tube they have a cylinder form. Chronic bronchitis is always accompanied by the violation of drainage function of bronchial tubes, which causes the delay of their maintenance in lower departments, closing of road clearance of bronchiole and the development of bronchiolung complications (obstructive emphysema, chronic pneumonia, pneumofibrosis).

Bronchioectasis (bronchektasia) is born and purchased expansions of bronchial tubes as cylinders or sack. Born bronchioectasis arise up in connection with violation of forming of the bronchial tree. They are marked with the chaotic location of structures of membranes of bronchial tubes. Sometimes bronchiole are closed blindly in parenchyma of lungs, cysts appear. In such cases it is talked about a cystous lung. Bronchioectasis is purchased related to the sharp bronchitis, pneumonia, and collapse of lungs.

On a form expansion of bronchial tubes saccade bronchioectasis (local thrusting out of membrane) and cylinder bronchioectasis (diffuse expansion of road clearance of bronchial tube) are distinguished. Expansions of shallow

bronchial tubes mark as bronchioloectasis. Lungs in such cases have a cellular kind (pulmo cisticus).

The time of bronchioectasis there are the phenomena of chronic inflammation in the membrane of bronchial tubes, metaplasia of prismatic epithelium in multi-layered flat, dystrophic changes from the side of elastic fibres, cartilaginous tissue and leyomicyte, sclerosis. In the cavities of bronchioectasis saved mucus and festering exudate. On this soil there are abscesses, perifocal festering pneumonia, perifocal fibrous, obstructive emphysema. Sclerosis develops in vessels that at presence of plural bronchioectasis and emphysema results in the development of hypertension in the small circle of blood circulation and hypertrophy of the right ventricle of heart. The signs of hypoxia appear with next violation of trophism of tissues. A very typical sign is a bulge of nail phalanxes of fingers of hands and feet as "drumsticks".

Combination of changes in lungs and after their scopes (pulmonary heart, general amyloidosis, hypoxic signs, sclerosis, etc.) at presence of bronchioectasis examined as new nosology is bronkhoectatic illness.

Emphysema of lungs is the pathological state of pulmonary tissue, which is characterized by the promoted maintenance of air in it. Vesicular, chronic diffuse obstructive, chronic, focus, vicarious, primary panacinaric, senile and interstitial emphysemas are distinguished. Development of vesicular emphysema is related to the chronic bronchitis, broncholitis and by their consequences – plural bronchoectasis. It is found out, that there is a deficit of inhibitors of protease at these diseases – to elastase, collagenase. Insufficiency of important they inhibitor;1-antitrypsin can be genetically conditioned. Activation of elastase and collagenase causes the destruction of interalveolar partitions with confluence of alveolar ways in greater cavities.

Chronic diffuse obstructive emphysema (emphysema pulmonum obstructium diffusum chronicum) arises up at the time of chronic diffuse bronchitis. The development of it is taken to the valvular mechanism. It happens because of the mucus clot which appears in shallow of bronchial tubes and bronchiole, at inhalation skips air in alveolar ways, but does not allow him to go out during exhalation. Air is saved in acinus, which are broaden as a result of the insufficiency of elastic and collagenase fibres. At overwhelming expansion of respiratory bronchiole and acinus talk about centeracinus emphysema, and in the case of expansion of large bronchial tubes and acinus – about panatsinarnu emphysema. Stretching of membranes of acinus results in thinning of interalveolar partitions, expansion of interalveolar por and formation of vesicular blisters. The capillary net of partitions empties. Thus, there is the considerable diminishing of area of interchange of gases and a vent function of lungs is violated. Devastation of capillary net of alveolar ways together with the sclerosis of interalveolar capillaries conduces to development hypertension of small circle of circulation of blood and hypertrophy of the right ventricle of heart (pulmonary heart).

Chronic focus emphysema (emphysema pulmonum focale chronicum) arises up as a result of expansion of acinuses and respiratory bronchiole round the old hearths of tubercular inflammation or postatack scars. At confluence of a few bubbles it is talked about bullous emphysema. Bubbles, which are located under pleura, can break through in a pleura cavity and cause spontaneous pneumothorax. This type of emphysema is not accompanied by hypertension of the small circle of blood circulation, as a capillary river-bed is damaged on the limited area of lungs.

Vicarious emphysema (emphysema pulmonum vicarum s. compensatorium) is also called compensate. It arises up after the amputation of part lungs or one of lungs. This type of

emphysema is accompanied by compensate hypertrophy and hyperplasia of structures of lungs, which remaine. The couse of primary (idiopathic) emphysema is unknown. For it such signs are typical, as atrophy of membranes of alveolar ways, reduction of capillary membrane, and hypertension of small circle of blood circulation.

Development of senile emphysema, more precisely are emphysemas in old men, related to age-old involution of lungs.

Interstitial emphysema (emphysema pulmonum interstetiale) is characterized by the penetration of air in intermediate tissue. The couse of such phenomenon is the break of alveolar ways at strong coughing motions. Through the cellulose of root lungs air gets to intercellular spaces of mediastinum (pneumomediastinum), hypodermic cellulose of neck, thorax, chairman (hypodermic emphysema). At pressure on the areas exaggerated air of skin to hear a characteristic crunch (crepitation).

Bronchial asthma is a chronic disease of allergic nature, which is characterized by the attacks of expiration shortness of breath. Two main forms of bronchial asthma are selected – atopic and infectiously allergic.

An atopic form arises up at the time of operating on the respiratory tracts of allergens of uninfectious origin. In the half of cases illness is predefined a room dust in the complement of which high-allergic carbohydrates - products of disintegration of cellulose enter from a cotton plant. In addition, in a room dust the special type of tick with which link the origin of bronchial asthma in child's age is found. From other allergens such, as vegetable pollen, epidermis and wool of animals, medications (acetylsalicylic acid, morphine), domestic chemicals have a most value (detergents, varnishes). The infectiously allergic form of bronchial asthma develops in patients with broncho-pulmonary pathology, caused infectious agents – viruses, bacteria, mushrooms. Pathogenesis of both

forms of bronchial asthma is similar. Immunological, pathochemical and patophysiological stages are selected. At an atopic form the immunological stage is characterized by the hyperproduction and piling up of IgE. These antibodies are adsorbed in the cells of bronchiole and at the repeated hit of antigen in respiratory tracts co-operate with it after the mechanism of anaphylaxis. The reaction of immediate type is formed; the attack of shortness of breath arises up in a few minutes after the action of antigen. At infectiously allergic bronchial asthma the immunological stage is opened out after the mechanism of hypersensitiveness of slow type, where the leading role is played not by antibodies, but by sensibilised lymphocytes. The shortness of breath appears in 12-36 hours after a contact with an allergen.

During the pathochemical stage under the act of complexes an antigen-antibody active substances are released-histamine acetylcholine, prostaglandin, leukotriene. They violate the function of cells -targets, stopped up in the membranes of bronchiole, – leyomiocytes, goblet and other cells. It shows up in bronchiospasm, hypersecretions of mucus and edema of mucus bronchiole. Eventually vent possibilities them limited strongly. Exhalation especially suffers, when due to the additional tension of respiratory muscles high intrapulmonar pressure is created. Bronchiole stick together, and exhalation is bothered or in general becomes impossible. Violation of breathing in patients with bronchial asthma shows up as the repeated attacks of shortness of breath. During an attack there is infiltration of membranes of bronchiole by eozinophiles, neutrophiles, labrotcytes, and T-lymphocytes. There is an edema of mucus and submucose, obturation of bronchiole by mucus in which eozinophiles appear and an epithelium peeling. In pulmonary tissue sharp obstructive emphysema develops with the focus of atelectasis . Respiratory insufficiency which can lead to death of patient

during an attack, comes as a result. Before the chronic signs of bronchial asthma the phenomena of diffuse chronic bronchitis, bulge belong and hyalinosis of basal membrane of bronchiole, sclerosis of intraalveolar partitions, chronic obstructive emphysema of lungs, hypertension of small circle of blood circulation, hypertrophy of right ventricle of heart.

Interstitial illnesses of lungs are characterized by the primary inflammatory process in intraalveolar connecting tissue (pneumonitis), they are also called fibroutic alveolitis. They end up with the development of diffuse pneumofibrosis.

Distinguish three nosology forms of fibrous alveolitis:

1) idiopathic fibrous alveolitis;

2) exogenous allergic alveolitis (lung „farmer", „poultry farmer", „cattle-breeder", „textile worker", „pharmaceutist";

3) toxic fibrous alveolitis.

Causes:

1) viral, bacterial, mycosis infection;

2) dust with the antigens of animal and vegetable origin;

3) medical preparations:, immunosuppressors, antitumor antibiotics, antidiabetic preparations and others like that

In pathogenesis the basic role is played by the immunocomplex damages of capillaries between alveolar partitions and stroma of lungs with next cellular immune cytolysis.

Pathological anatomy is presented by three stages:

1) diffuse or granulematose alveolitis with infiltration neutrophiles, lymphocytes, plasmatic cells;

2) disorganization of alveolar structures and pneumofibrosis;

3) forming of cellular lungs with the development of the alveolar-capillary block, panatcynaric emphysema,

bronkhioloectasia, hypertension in the small circle of blood circulation, hypertrophy of the right ventricle.

The syndrome of Khammen-Rich is a sharp form of fibrous alveolitis, that occurs at the system diseases of connecting tannin, viral active hepatitis.

Pneumofibrosis is a chronic process in lungs, which develops after the previous diseases of pulmonary tissue or interstit. It is characterized by excrescence of connecting tissue, deep alteration of microcirculations, the development of hypertension in the small circle of blood circulation with the next hypertrophy of the right ventricle and the forming of pulmonary heart, hypoxia of pulmonary tissue, its alteration and deformation.

Professional diseases of lungs

Silicosis and anthracosis belong to the group of pneumoconiosis – professional illnesses which are caused by the action of industrial dust.

The cause of silicosis is the protracted inhalation of dust which contains the free oxide of silicon of SiO2. The crystalline oxide of silicon in a tissue liquid slowly dissolves and grows into colloid solution of silicic acid. The latter damages the tissue of lungs and initiates a fibrous process.

The same role in the pathogenesis of silicosis is played by the damage of the membrane of phagolysosoms by the particles of quartz, as a result of that hydrolytic enzymes are outpoured in the cytoplasm of macrophagocytes. The products of autolysis of macrophagocytes stimulate the proliferation activity of fibroblasts.

The course of silicosis is mostly chronic. In mucus and submucose membranes of nose, larynx, trachea, interstitial of lungs, lymphatic nodes the phenomena of atrophy, sclerosis and formation of silicate nodes appear. They have a round or polygonal form, the color is grey or grey black. In one cases

silicate nodes are built from concentric located hyaline of connective tissue bunches, in other - from the wrong directed collagenase bunches. In both cases a free dust or dust appears in macrophagocytes. They are called as dustcontaining cells– coniophagocytes.

Three forms of silicosis are distinguished. At a miliary form shallow nodes prevail by a size from millet corn. At a tumor form silicate nodes are large, they remind a tumor and occupy the greater part of pulmonary fate or and all of fate. The diffusely sclerotic form is characterized by the negligible quantity of miliary nodes and the predominance of diffuse excrescence of connecting tissue after motion of bronchial tubes, vessels and intraalveolar partitions.

During all forms of silicosis the development of chronic bronchitis, pneumosclerosis, hypertension of small circle of blood circulation, hypertrophy of right ventricle of heart are observed. Sometimes a silicate node can be added disintegration with the formation of silicate cavity. In genesis of cavities a substantial value has instability of newformed connecting tissue. In particular, it is less steady to collagenosis. Tuberculosis often joins silicosis. In such cases illness is called silicotuberculosis.

Anthracosis arises up at the protracted inhalation of coal dust. The illness is characterized by the development of connecting tissue in the places of lying of coal dust – in intraalveolar partitions, after motion of bronchial tubes and vessels. Connecting tissue overgrows round the accumulations of dust, not shown out coniophagocytes through a bronchial tree or lymphatic vessels. Such nodes are called anthracotic.

At the overload of lymphatic nodes by coal dust and their sclerosis there is the stagnation of lymph, hypoxia and acidosis of stroma of lungs. On this background - so-called black induration of lungs is developed.

Anthracosis is accompanied by chronic bronchitis,, emphysema, pulmonary hypertension and focus pneumonia. As a result of disorders of blood circulation and direct influencing of coal dust sometimes there is necrosis and softening of pulmonary tissue with the formation of cavities. This form of anthracosis is accompanied by haemoptysisand, reminds the second tuberculosis, through what it is called black consumption.

The cancer of lungs occupies the first place among malignant tumors in men and the second – in women. The death rate because of it is 26 %.

The cancer of bronchial tubes arises up mainly in smokers (90 %). An important role also belongs to the carcinogenic substances which penetrate blood and lymph.

To the precancer states belong chronic bronchitis, bronchioectasis,, and to the precancer changes – hyperplasia, displasia and metaplasia of epithelium.

As a rule, the cancer of lungs develops from the epithelium of bronchial tubes (bronkhogenic, central cancer), rarely – from the epithelium of bronchiole and alveolar epithelium (pneumogenic, peripheral cancer). Pathogenesis of central cancer is related to such precancer changes, as basal-cellular hyperplasia, displasia and flat cellular metaplasia of epithelium of bronchial tubes. For morphogenesis of peripheral cancer the main characteristic is wider spectrum of pre-tumor changes. Foremost, they are related to the development of pneumosclerosis after the carried inflammation, to the heart attack and others like that. Terms which are instrumental in malignant transformation are created in scar, namely is depositing of carcinogens, local immunosuppression, violation of intercellular connections.

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According to localization the following forms are selected:

1) periapical (central) cancer which developes from an epithelium a barrel lobular and initial part of bronchial tube, grows as a node or polypus of white color and dense consistency;

2) peripheral cancer which developes from the peripheral department of bronchial tube and its branches, and also from alveolar epithelium, exophiticaly grows for a long time, often developes in the area of scar;

3) the mixed (massive) cancer shows by itself soft tissue of white color, which can occupy all of fate or whole lung.

On character of growth endophitic (endobronchial) and exophitic (exobronchial and peribronchial) cancers are distinguished.

On macroscopic form a cancer is plague-like, polypus, endobronchial diffuse, ramified and nodal ramified cancer.

On microscopic structure flatcellular (epidermoid) cancer, undifferentiated, anaplastic caner (finecellular, largecellular, oastmealcellular), golden-flatcellular cancer, of bronchial glands – adenoidno-cystous and mucoepidermoid are selected.

Distribution to outside of organ is a characteristic for a central cancer. At endophitic growth he passes to tissue of mediastinum, cardiac shirt, pleura. Peripheral and mixed cancers spread within the limits of organ, germinating tissue of bronchial tubes and pleura. The cancer of lungs metastases by lymphogenic and hematogenic ways. Lymphogenic metastases arise up in peribronchial, bifurcational, neck and other lymphatic nodes, hematogenic – in a cerebrum, bones (mainly vertebrae), and adrenal glands. For a central cancer lymphogenic metastases are typical, for peripheral – hematogenic. First clinical sign of peripheral cancer, which developes in the area of scar and has small enough sizes

(microcarcinoma), related to the plural hematogenic metastases.

Permanent complication of cancer of lungs, especially central, is the development of atelectasis. Pneumonias, abscesses, bronchioectasis, bleeding which mask motion of cancer, develope as a result of the violation of the drainage function. The distribution on pleura causes the development of serous-hemorrhagic and hemorrhagic pleuritis, and also to carcinomatosis of pleura. Cachexy during the cancer of lungs developes later than during the cancer of stomach.

The pleuritis is the inflammation of pleura more frequently arises up as the complication some of visceral pathologies. Often occurs at the time of the diseases of lungs: pneumonias, heart attack, cancer, tuberculosis and etc., at the time of rheumatism and other system diseases of connecting tissue (allergic pleuritis), at the time of the diseases of kidneys (pleuritis of uremia). According to the character of inflammations pleuritis serous, fibrinous, serozno-fibrinous, festering, hemorrhagic are distinguished.

II Algorithm of the practical part of the lesson

Study and be ready for the verbal description of the macropreparations:

1 *Croupous pneumonia.* Synonyms is fibrinous, partial, pleuropneumonia which represents the type of exudative inflammation, volume of defeat of organ and one of the permanent signs of illness. Macropreparation shown itself by cut lungs, on which clear border between overhead and lower particlesare evident. The surface of the cut of overhead particle is dark grey, and low– light-grey, fine-grained, (croupous inflammation is in the stage of grey hepatization). Such color is conditioned by the presence of gleam in alveolar ways of exudate which consists of fibrin and leucocytes. The term

"hepatization" is related to the considerable compression of part which gives lungs similarities to the liver in consistency.

2 *Focus bronkchopneumonia.* Bronchopneumonia unites different in relation to an origin inflammation of pulmonary tissue, for which beginning of process in bronchiole is general and successive transition of inflammation on bronchial tubes and pulmonary tissue. Macropreparation shows itself by cut lungs of grey color. Above the surface of cut something with wrong form is bursting, different size focuses of rather yellow grey color, which are merging. The surface of these areas is grainy. In the focus of inflammation bronchial tube is evident. For diagnostics of inflammation a pathologist should carve the piece of tissue and dip it into water. It must drown. On the initial stages of inflammation it can not sink. Then a dissector is oriented after the unevenness of color of surface of a cut and turbidity of squeezing out liquid. On the presented macropreparation of focus of inflammation grainy, yellow grey color. Consequently, it is possible to think that exudate can have fibrinous character. If it has hemorrhagic, festering or desquamation character, the surface of a cut is smooth and of the proper color. Judging on a volume the defeat of probed lungs, bronchopneumonia can be taken to largfocus. The cause of it can be pneumococcus IV, III, streptococcus, staphylococcus, diplobacillus of Friedlander, toxic substances (uremia), allergens. Alongside with the described higher bronchopneumonic focuses are evident here and there are areas of pale grey color, which answer the extended alveolar ways (vicarious emphysema), and more dark areas, to atelectasis. Mainly it is the sign of the violation of the drainage function of bronchial tubes and compensatory-adaptive process.

3 *Carnification of lungs.* Macropreparation show itself by cu lungs of grey color, fleshy consistency, reduced lightness. Among pulmonary parenchyma present excrescence of fibrec connecting tissue is evident which appears as a result of the

organization of fibrinous exudate in transparent alveolar ways. Such pathological process in pulmonary tissue arises up as a complication of croupous pneumonia.

4 *An abscess in lungs*. Preparation is made from the part of lungs, in which the clear border between overhead and lower particles is evident. Low– darkly grey color, that corresponds to a normal structure of lungs. Overhead homogeneously grey (stage of grey hepatization of inflammation) with the presence of two cavities with unequal pyogenic membranes, and also sequester of pulmonary tissue. Such abscesses are a pulmonary complication of croupous pneumonia as a result of disorder of blood circulation in the staggered tissue (stasis, the thrombosis of capillaries are widespread) and joining of putrid infection. Define, what pulmonary complications of illnesses are possible as a result of deepening of putrid microorganisms in preliminary necrosed tissue.

5 *Empyema of pleura*. Preparation is a cut lung with visceral and parietal pleura. Serous membranes are thickened. In a cavity there is exudate of yellow color which consists of fibrin and leaves to the rot. At the time of croupous pneumonia parapneumonic fibrinos pleuritis constantly develope and can be complicated with the development of empyema.

6 *Pertaining to apostema nephrite*. A kidney is megascopic. Under a capsule yellow ulcer with red rim is evident. It is hematogenic "descending" form of intermediate pyelonephritis which is the sign of pneumococcus sepsis belongs to extrapulmonar complications of croupous pneumonia.

7 *Pneumosklerosis*. On a cut of the lungs excrescence of connecting tissue of white color is evident after the motion of bronchial tubes and vessels, it is more expressed at a gate of lungs, less on the periphery. Morphologically two types of pneumosclerosis are distinguished:

1) Focus rough, that scars are after abscesses, after the uncompleted croupous pneumonia in the places of carnification

2) The reticulated is dissipated – after bronchopneumonia. At the time of bronchopneumonia an inflammatory process spreads mainly intracanal, that after the motion of bronchial tubes up to respiratory bronchiole. At uncompleted bronchopneumonia sclerosis, as the finishing stage of inflammation, also spreads after motion of bronchial tubes and vessels which are located next to bronchial tubes – peribronchialy and perivasculary sclerosis which arrives at respiratory bronchiole and on intraalveolar membranes. That is why such pneumosclerosis is called diffuse reticulated. As a result of sclerosis of membrane of bronchial tubes not sticking together, their gleam is opened, ends of transversal cut of bronchial tubes of lungs come forward above the surface of cut, goose feathers are cut away as though. Consequently, diffuse reticulated is presented in preparation, as a result of uncompleted bronchopneumonia.

8 *Chronic abscess of lungs*. Preparation shows the cut of pulmonary tissue, in which there is cavity, membranes of it are unequally thickened, deformed. In the gleam of the cavity there is negligible quantity of festering exudate. The membrane of the cavity is built from rough fibrose of connecting tissue and adjoining indurated parenchyma of lungs.

9 *Emphysema of lungs*. A lung is withdrawn from the organism of man who died at the peak of bronchial asthma attack. A lung is of pale grey color, biglobular structure is as a result of swelling of acinuses. There is squeezing in pulmonary tissue in place of location of heart. Edges of lungs are round. On a section it is evident, that the cuttings edges of lungs cover a heart, mediastinum, come above each other. Lungs are fluffy by touch, a surface is smooth, brilliant. At pressure of a finger there is pit. At a cut there is a crepitate sound. Such emphysema develops as a result of the violation of the bronchial communicating, when valve-like mechanisms operate. If at the section of thorax lungs of sticking together, it

testifies to sharp emphysema. At the time of chronic emphysema lungs after the section of thorax of do not stick together. Consequently, chronic panacinaric emphysema is presented on the preparation.

10 *Pulmonary heart*. A heart is sharply megascopic in sizes 16x12x10 (a norm 10x8x6), mainly due to a right half. Such hypertrophy of myocardium of mainly the right ventricle develops as a complication of chronic diseases of lungs. The sclerosis of pulmonary vessels develops at pneumosclerosis, in particular branches of pulmonary artery. The membranes of vessels are thickened, gleams are narrow or even obliterated. That is why pressure in pulmonary artery rises and arises up hypertension in the vessels of small circle of blood circulation. Compensate hypertrophy of myocardium develops in the right ventricle; a heart is increased in sizes mainly due to the tension of the cavity of the right ventricle. So a pulmonary heart develops.

11 *Amyloidosis of kidney*. A kidney is considerably megascopic in sizes – 15x8x7 (a norm 10x6x5), on the cut it is of white color, greasy kind, a border absents between layers, medullar and cortical layers are not determined macroscopically. Such kidney is dense by touch. The second amyloidosis of kidneys develops as a complication at the time of chronic pneumonias, especially at the time of bronchoectatic illness, abscesses of lungs. The products of disintegration of pulmonary tissue, festering exudate, are sucked in blood, hyperproteinemia and disproteinemia develops. The products of disintegration of tissues, pathological proteins during excretion by kidneys, litter an albuminous filter –corpuscles, membranes of capillaries. The immune mechanism of amiloidforming is included in a sensibilisated organism. Kidneys are inlarged and indurated in connection with their obstruction with proteins. Kidney corpuscles are eliminated from a function, uremia which

sometimes is the cause of death at the time of chronic pneumonias develops.

12 *Cancer of bronchial tube*. On the cut of pulmonary tissue it is evidently, that the gleam of the left bronchial tube is fully filled with a tumor, obturation by a tumor. The tumor of white color, germinates in pulmonary tissue, in periapical lymph node. Such tumor early shows up clinically as a result of the broken drainage function of the main bronchial tube. Histologically such cancer is often squamous-cell carcinoma as a result of the previous metaplasia of prismatic ciliated epithelium of bronchial tube in multi-layered flat at chronic bronchitis. Squamous-cell carcinoma of bronchial tube are more frequently nonkeratoid but can be also keratoid. Also there can be the cancer of prismatic (glandular epithelium).

13 *Peripheral cancer of lungs*. The tumor is of white color, largenesses, grows from bronchial tubes in pulmonary tissue. Such cancer takes place from segmental bronchial tubes or bronchiole, more frequently, rarer planocellular cancer. More frequently germinates in pleura. Complication is a serous-hemorrhagic pleuritis.

14 *Anthracosis of lungs*. In macropreparation the cut of lungs lobe is presented, in which evidently numeral spots of black with unclear contours are - a node form of anthracosis. Histologically every node is built of a scar tissue both in intraalveolar membranes and in alveolar ways and round them. In a center there are amorphous masses with the particles of coal dust. In the overhead departments of preparation – the large black fields which appeared as a result of confluence of nodes - a diffuse form of anthracosis. Anthracosis, as a rule, is accompanied by the development of pulmonary heart, pulmonary-cardiac insufficiency. Tuberculosis of lungs often joins.

Study the micropreparations of the theme and be ready to show on the picture the main points of the pathologic process with the following descriptions:

1 *Croupous pneumonia.*

1) Tinted with hematoxylin and eozin. In the gleam of most alveolar ways there are a plenty of leucocytes (a lot of dark blue kernels) and fibrin. In separate areas in gleam of alveolar ways there are less leucocytes , in them fibrinous exudate are well seen as pale-blue or rose color of thin filaments which interlace between itself, as though magnificent ball of cotton wool. Together with fibrinous exudate neutrofil leucocytes and moderate leukocyte infiltration of membrane of bronchial tubes are seen. Vessels are extended, sanguineous. In gleam of the separate extended vessels there is a plenty of leucocytes. Consequently, in preparation the stage of grey hepatization of croupous pneumonia.

2) Tinted with hematoxyline-eozin and fuxeline is for the exposure of elastic fibres. Elastic fibres are in the membranes of vessels, in intraalveolar membranes, painted in a darkly cherry blossom. In the gleam of alveolar ways there are neutrophil leucocytes, alveolar epithelium is peeling, in the separate areas of alveolar ways fibrinous exudate is evident as rose color balls of thin fibres which are agglutinated between itself and thickly interlaced. Vessels are extended, sanguineous, lymphostasis in lymphatic vessels. On the basis of histological research of micropreparations it is possible to set a morphological diagnosis is the stage of grey hepatization of croupous pneumonia. To **designate:** 1- leucocytes are in gleam of alveolar ways; 2- filaments of fibrin are in gleam of alveolar ways; 3- extended vessels.

2 *Carnification of lungs at croupous pneumonia.* Tinted with hematoxyline and eosin and after the method of Van Gizon. In the gleam of alveolar ways there is fibrinous exudate with the admixtures of leucocytes. The organization of fibrinous

exudate takes place in almost all alveolar ways – the germination connecting tissue which is painted in red color, fibres are oriented in one direction, the kernels of fibroblasts, and also single leucocytes, are evidently prolated. In addition it is evident that the large fields of connecting tissue infiltrated with festering exudate. Placed intraalveolar membranes are sharply thickened and infiltrated with festering exudate. The carnification of lungs is the sign of transition of croupous pneumonia into a chronic process.

3 *Bronchopneumonia*. Microscopically it is characterized by four basic morphological signs:

1) Bronchiolitis is inflammation of membrane of bronchial tubes, the membrane of bronchial tubes is blasted, and the gleam is filled with festering exudate. All layers of membrane of bronchiole are infiltrated with leucocytes, sometimes tailings of peeling mucus are evident in the gleam of bronchiole are hyperchromal kernels of epithelium, located a chainlet as trabekul together with festering exudate, mucus is fully blasted placed, there are bronchiole with uncrippled mucus, but the gleam of them is filled with festering exudate. The gleams of bronchiole, as a rule, are extended. At times bronchiolitis has the appearance of microabscess of round or oval form (hearth accumulation of leucocytes) microscopically, when all layers of bronchiole membrane are blasted.

2) Peribronchial pneumonia – around bronchiolitis in the gleam of alveolar ways there is inflammatory exudate, as a rule, festering, rarer serous-festering or serous-hemorrhagic, bronchial epithelium is peeling. Intraalveolar membranes are thickened, infiltrated with leucocytes, at times blasted. An inflammatory process from bronchial tubes spreads on adjoining pulmonary tissue.

3) Atelectasis. As a result of obturation of the gleam of bronchial tubes with inflammatory exudate, peeling mucus halts its drainage function and of the proper area develop

lungs. Microscopically atelektazi is present compact airless tissue, pulmonary picture is effaced, tissue of focus or diffusely infiltrated with leucocytes. Vessels are sanguineous.
4) Emphysema of lungs. Compensate in nearby with atelectasis areas emphysema develops – alveolar ways are stretched, overcrowded with air, intraalveolar membranes are torn placed. Incomplete obturation of bronchial tubes assists also to the development of emphysema, as a result air gets in alveolar ways, but does not fizzle out is a valve effect of the damaged bronchial tubes. Placed in the area of emphysema intraalveolar membranes are thickened due to the inflammatory infiltration. **To designate**: 1- bronkhiolitis; 2- the area of peribronchial pneumonia; 3- atelectasis; 4- emphysema.
4 *Chronic abscess of lungs*. At the microscopic review of the cut of lungs cavity with maintenance is evident. It is histologically possible on the basis of the stored structures to set that the extended gleam of bronchial tube (bronchioectasis). The membrane of bronchial tube is thickly infiltrated with neutrophils, lymphocytes. A mucus membrane is fully blasted. In the gleam there is mucus with the admixtures of neutrophils. Analysing these information, it is possible to consider that abscess has a bronchogenic origin. Considerable excrescence of the fibred connecting tissue is marked in perifokal departments. Intraalveolar membranes are thickened due to the excrescence of connecting tissue and lymphocytic infiltration. Placed connecting tissue occupies the whole fields. The bricked up separate alveolar ways and unreal bronchiolis appear among its fibers. Next to that it is possible to see bronchiole with the deformed gleams. Their mucus is covered with prismatic epithelium, there are leucocytes in the gleam. The membrane of bronchiole is thickly infiltrated by lymphocytes. The membranes of blood vessels are thickened due to the hypertrophy of internal muscular layer and perivascular sclerosis. To **designate**: 1- extended gleam

bronchial tube; 2- infiltration of membrane of bronchial tube by neutrophils and lymphocytes; 3- thickened intraalveolar membranes; 4- thickened membranes of vessels.

5 *Cancer of lungs (bronchopulmonar squamous-cell carcinoma without a cornification*). Pay attention to the nest location of epithelium. Epitteliocytes are of different forms. Next to hyperchromal there are hypokhromal, plural pathological mitosis. Atypical cells take place in mucus, in peribronchial and perivascular spaces, that testifies to infiltrative growth. **To designate**: 1-epitheliocytes of different form with hyperchromal nucleus; 2- mitosis; 3- atypical cells: 3a- in mucus, 3b- peribronchial and 3c- perivascular spaces.

6 Amyloidosis of kidney. At colouring the congo-red is marked the considerable laying to the amyloid in the membranes of vessels, capillary loops, basal membranes of canals and in the stroma of organ. Some corpuscles are quite transferable by amyloid. **To designate**: 1- amyloid in the corpuscles of kidneys; 2- amyloid in canals of kidneys.

7 *Bronchioectasis and pneumosclerosis*. Preparation is tinted with hematoxylin-eozin. The considerable excrescence of fibred connecting tissue which replaces almost all parenchyma of lungs. Separate alveolar ways and considerable accumulation of lymphocytes are determined only. Transparent bronchial tubes and bronchiole is deformed due to polyposis and considerably extended. Mucus bronchial tubes are covered with prismatic epithelium. A basal membrane is thickened and hyalinised. There is an accumulation of festering exudate in the gleam of the separate bronchioloectasia. The membranes of blood vessels are thickened, the gleam is narrowed. **To designate**: 1 excrescence of fibred connecting tissue; 2- deformed transparent bronchial tubes and bronchiole; a 3- accumulation of festering exudate is in gleam of separate bronchioloektasia ; 4- the bulge of membrane of blood vessels.

Situation tasks.

1 Young man after supercooling felt weakness, stuffiness, pain at breathing in the right half of thorax, a fever appeared. At percussion the sound is objectively dull, the absence of the vesicular breathing and noise of friction of pleura in the area of lower particle of right lungs. Death came in a week from the beginning of disease at the phenomena of pulmonary-cardiac insufficiency. On autopsia a lower particle of right lungs is densed, with stratification of fibrin on a pleura, on a cut there is tissue of grey color, fine-grained, airless with the presence of cavity filled a pus.

1. What disease developed?
2. What is the noise of friction of pleura connected with?
3. What stage of disease is it ?
4. What pulmonary complication developed?

2 In a man after the resection of stomach, stuffiness, fever, appeared on the third days (to 38,5 S). The moist wheezes in the lower departments of lungs, roentgenologic are shallow hearths of inflammation in 8-10 segments of lungs. What disease complicated a postoperation period? Specify its kind after pathogenesis?

3 A patient has suffered from the chronic inflammatory diseases of lungs for a long time. Died from hypernitrogenemiatic uremia. On autopsia the following things found out the compression of lungs with considerable expansion of gleam of bronchial tubes, reticulated, hypertrophy of right ventricle, dense kidneys cortical layer of which is of yellow white color are megascopic, pyramids pinky. What disease did a patient have? What complications developed? What is the cause of uremia?

4 A patient entered a clinic with high temperature, a cough with the selection of phlegm of unpleasant smell. Roentgenologicaly in lungs a cavity with the level of liquid was found out. Lobectomia was conducted. The cavity which

unites with a bronchial tube was filled with a pus. What disease is it? What are the possible complications of the illness?
5 In a patient who suffers from a chronic bronchitis, roentgenologicaly the darkening round a right main bronchial tube was found. During bronchoscopia a biopsy was taken. At histological research the accumulation of cells of multi-layered flat epithelium with the phenomena of polymorphism and plenty of mitosis was found. What process developed in lungs?
6 In a patient roentgenologicaly the darkening in the peripheral departments of lower part lungs with the presence of cavity was found . Pulmonectomia is conducted. Histologically the accumulation of glandular structures with the phenomena of polymorphism and increased mitotic activity of epithelium cells, area of necrosis was found. What disease took place? Name the type of illness.

Answers to the situation tasks:

1 Croupous pneumonia, with fibrinous pleuritis, the stage of grey hepatization, abscess of lungs.
2 Focus pneumonia, postoperation.
3 Bronchioectasis, uremia, amyloidosis of kidneys is purchased.
4 Bronchogenic abscess of lungs, empyema of pleura, mediastinitis, meningitis, abscess of brain, sepsis, amyloidosis, uremia.
5 Cancer of lungs, bronchogenic, squamous-cell
6 Cancer of lungs, pneumogenic, peripheral, with disintegration.

Test Tasks:

1 A patient with the symptoms of the difficulty of breath and cough uremia. At bronchoscopia mucus bronchial tubes are sanguineous, filling out with shallow hemorrhages. In the

gleam of bronchial tubes there is much mucus. Specify the form of bronchitis.
A. Primary acute.
B. Secondary sharp.
C. Primary chronic.
D. Secondary chronic.
E. Deformate.

2 The patient with alcoholism suddenly had a cough with the selection of festering phlegm with the veins of blood cough and fever. Died from hepatargia. It is discovered on a section, that the lower particle of the right lung is densed, heavy and grey. Histologically – alveolar ways are filled with fibrin and leucocytes, venous stagnation. Specify the stage of croupous pneumonia. A. Rush.
B. Red hepatization.
C. Grey hepatization.
D.Resorbtion .
E. Carnification.

3 An old man had a moist cough and fever after supercooling. Died on the fourth day from pulmonary-cardiac insufficiency. On a section: the lower particles of lungs are densed with the deposits of fibrin on a pleura, after consistency remind a liver, on a cut grey brown. Specify the credible disease.
A. Croupous pneumonia.
B.Pneumonia of Fridlender.
C. Influenza bronchopneumonia.
D. Streptococcus bronchopneumonia.
E. Intermediate pneumonia.

4 An old man with the stomach cancer had pneumonia from which he died. On a section: the basal departments of lungs from two sides are of grey color. Histologically: exudate from

mucus, neutrophiles and filaments of fibrin, is distributed in alveolar ways unevenly: in one there is much, in other- few. The hearths of emphysema, atelectasis are determined. Bronchitis is expressed. Specify the type of pneumonia.
A.Croupous.
B. Bronchopneumonia.
C. Interstitial.
D. Fridlender's.
E. Legionella.

5 A child after adenoviral infection had lowlobular bilateral pneumonia from which he died. On a section the hearths of suppuration and necrosis were found out. Round the hearths of necrosis there is serous-hemorrhagic inflammation. Specify the type of pneumonia after the credible etiology.
A. Viral.
B. Staphylococcus.
S. Streptococcus.
D. Mycoplasmic.
E. Pneumococcus.

6 Croupous pneumonia was diagnosed in a clinic. In a week festering meningitis developed from which the man died. It is discovered on a section, that the lower fate of right lungs grew into airless fleshy tissue. Histologically – the masses of fibrin in alveolar ways germinated granulation tissue. Specify the complication of croupous pneumonia.
A. Gangrene.
B. Carnification.
C. Pneumocirrhosis.
D. Black induration.
E. Brown induration.

7 A man, with alcoholism, suddenly had moist cough with the selection of ferruginous sputum, fever developed. Croupous pneumonia was diagnosed in a clinic. He died of the progressing pulmonary-cardiac insufficiency. It was discovered on a section, that a lung was of black brown color with the widespread of necrosis. Grey brown liquid flows down from a surface. Specify the complication of croupous pneumonia.
A.Gangrene.
B.Carnification.
C. Pneumocirrhosis.
D. Black induration.
E. Brown induration.

8 At pathoanatomical research of lungs of a child who died of measles encephalitis, the presence of panbronchitis was found out. An inflammatory process passed to surrounding tissue and spread on adjoining intraalveolar partitions, that predetermines their bulge. In alveolar ways there is exudate which consists of alveolar macrophagocytes and single neutrophiles. Specify the form of pneumonia.
A.Croupous .
B. Bronchopneumonia.
C. Peribronchial.
D. Interlobular.
E. Interalveolar.

9 At pathoanatomical research of the lungs of a dying person of festering mediastenitis is set that inflammation has flegmonous character in mediastinal pleura with "stratification" lungs on a fate. Specify the form of pneumonia.
A. Croupous.
B.Bronchopneumonia.
C. Peribronchial.
D. Interlobular.

E. Interalveolar.

10 During a birth the premature removing of a layer by the layer of placenta. Pneumonia was marked from which a newborn child died. It is discovered on a section that lungs are inlarged in a volume, dense. Histologically in bronchiole and alveolar waysmeconium, and clots of fat, bronchitis and festering alveolitis were found out. Specify the credible form of pneumonia.

A. Croupous.
B. Intermediate.
C. Aspirate.
D. Lipid.
E. Peribronchial.

Answers to the test of task from a theme:

1. B; 2. C; 3. A; 4. B; 5. B; 6. B; 7. A; 8. C; 9. D; 10. C.

Illustrations to theme

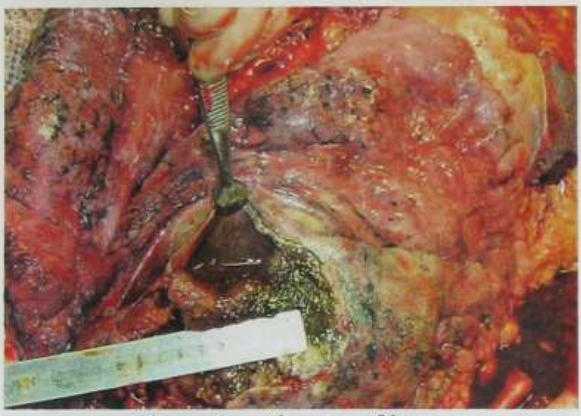


Figure 1 – Abscess of lung.

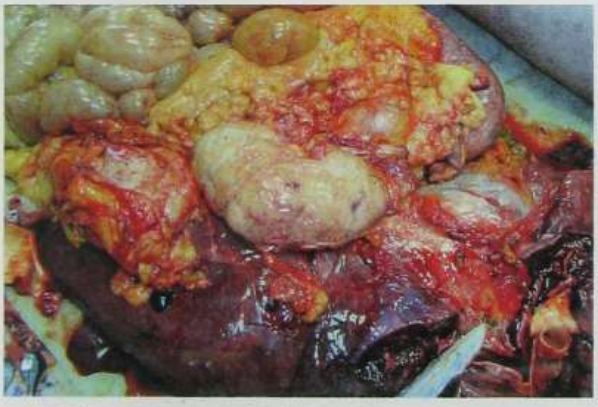


Figure 2 – Amyloidosis of kidney.

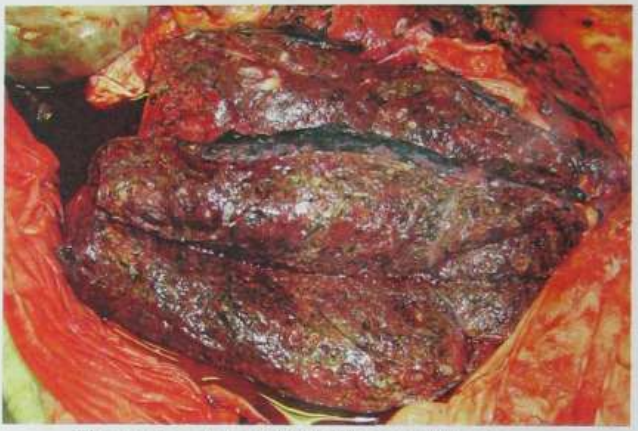


Figure 3 – Interstitial illness of lung.

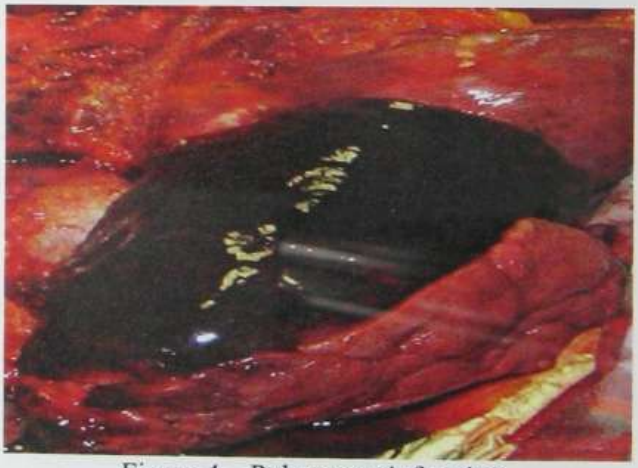


Figure 4 – Pulmonary infarction.

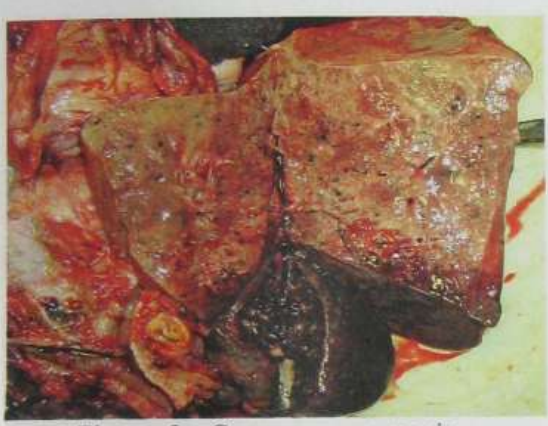


Figure 5 – Croupous pneumonia.

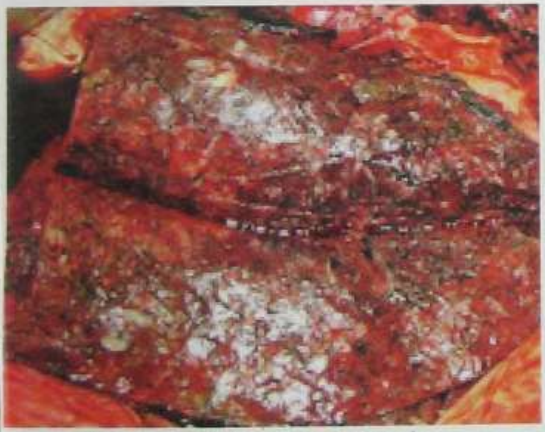


Figure 6 – Bronchopneumonia.

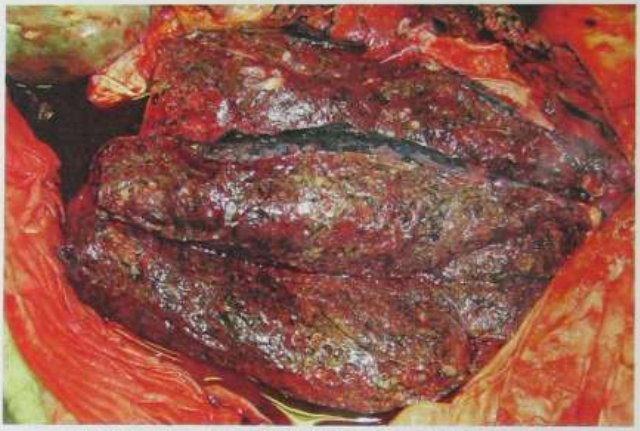


Figure 3 – Interstitial illness of lung.

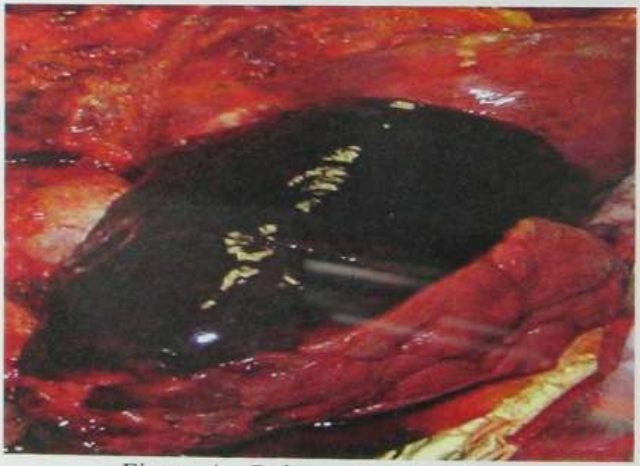


Figure 4 – Pulmonary infarction.

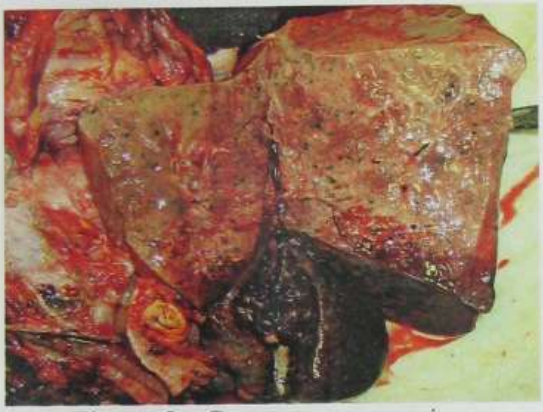


Figure 5 – Croupous pneumonia.

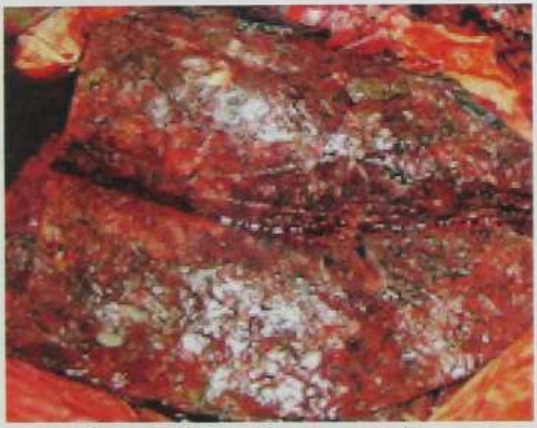


Figure 6 – Bronchopneumonia.

Figure 7 – Pneumosclerosis.

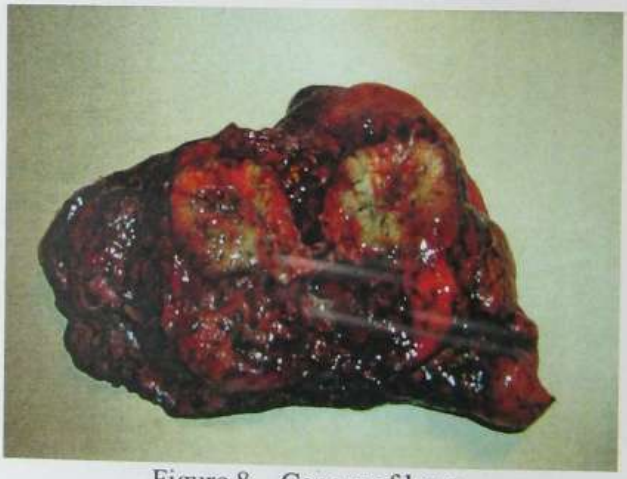


Figure 8 – Cancer of lung.

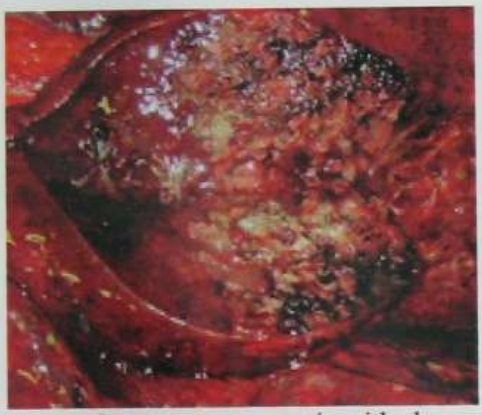


Figure 9 – Croupous pneumonia with abscesses.

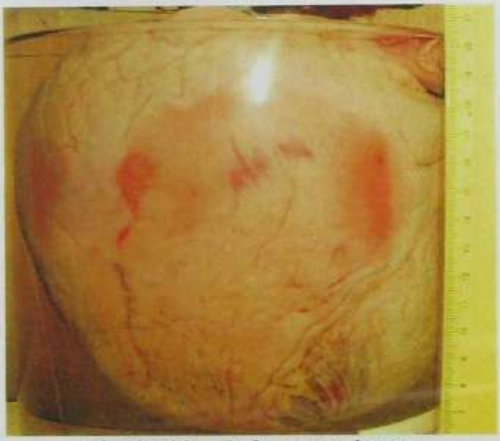


Figure 10 – Pulmonary heart.

Figure 11 – Purulent Plevritis.

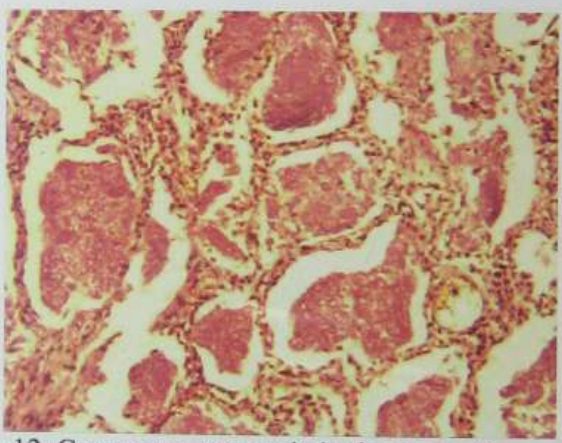


Figure 12. Croupous pneumonia in the stage of grey hepatization.

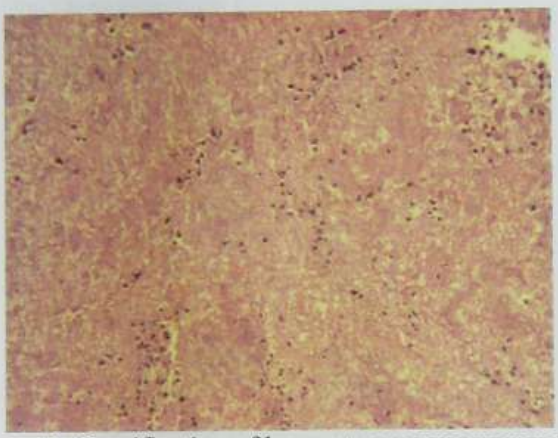


Figure 13. Carnification of lungs at croupous pneumonia.

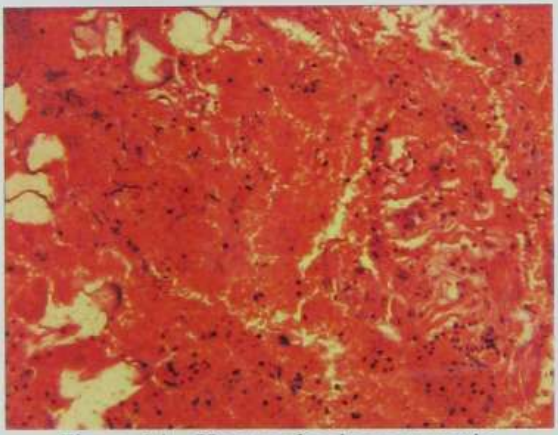


Figure 14 – Haemorrhagic pneumonia.

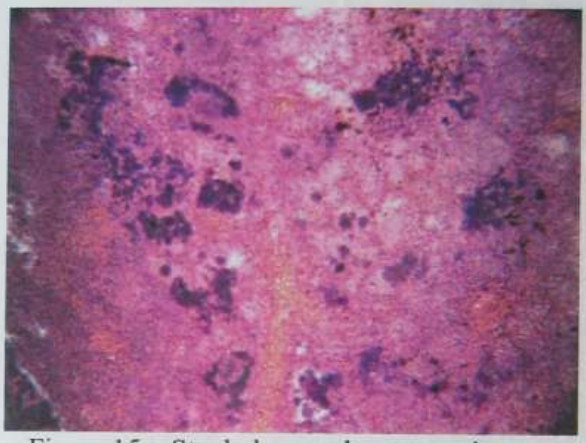


Figure 15 – Staphylococcal pneumonia.

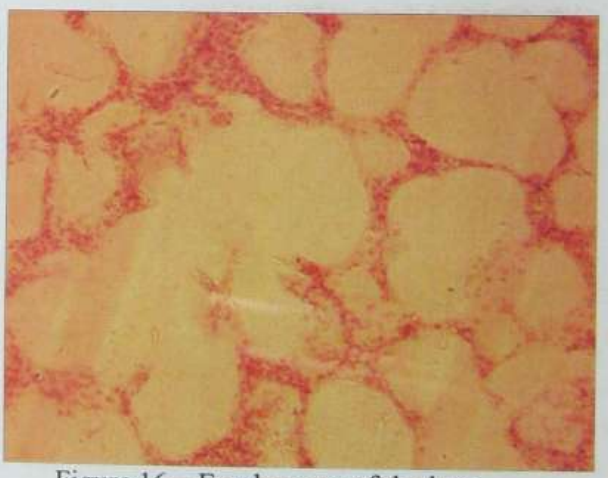


Figure 16 – Emphysema of the lung.

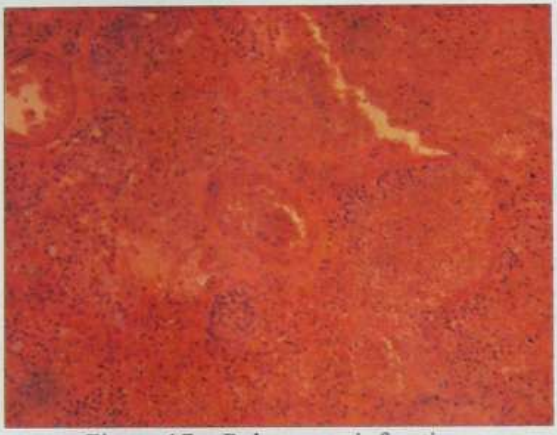


Figure 17 – Pulmonary infarction.

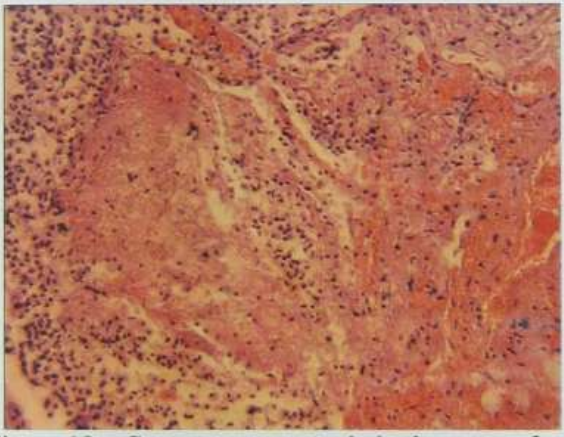


Figure 18 – Croupous pneumonia in the stage of red hepatization.

Theme 31 Diseases of Esophagus, Stomach and Intestine

Specific aims:

- *interpreting the clinicopathologic presentations of congenital and acquired esophagus diseases;*
- *interpreting the etiology, clinicopathologic description, complications and consequences of benign and malignant tumours of esophagus;*
- *explaining the protective barriers of the mucous tunic of stomach;*
- *interpreting the etiology (including Helicobacter pylori), pathogenesis, principles of classification and morphologic forms of chronic gastritis defined on the basis of gastrobiopsy studies;*
- *interpreting chronic gastritis as a precancerous condition;*
- *interpreting the etiology, pathogenesis and morphologic presentations of erosions and acute ulcers;*
- *interpreting the etiology, patho- and morphogenesis, and morphologic presentations of chronic ulcer in the periods of exacerbation and remission, its complications and consequences;*
- *interpreting the epidemiology, etiology, classification, morphologic presentations, macroscopic and histologic forms, metastasis peculiarities of stomach cancer;*
- *interpreting congenital intestinal anomalies;*
- *interpreting the etiology, pathogenesis and clinicopathologic presentations of intestinal diseases of various origin;*
- *interpreting the notion of malabsorption syndromes, their pathogenesis and causes, the clinicopathologic presentations of diseases connected with malabsorption*

syndrome, and the role of morphologic examination of biopsy material in the diagnosis of intestinal diseases;

- *interpreting the classification, etiology, pathogenesis, clinicopathologic description and complications of acute and chronic appendicitis;*
- *interpreting the clinicopathologic description and consequences of peritonitis, primary and secondary peritoneal tumours;*

Subject Actuality: due to the prevalence of digestive tract diseases and the development of specific (endoscopic, X-ray, biochemical, bioptic, etc.) methods of research and treatment, the profession of gastroenterologist was singled out as a separate medical speciality. Due to the access to mucous tunics, gastroenterologists constantly use in their practice the morphologic method – biopsy examination. Yet, the problems of gastroenterology are of great interest not only for particular specialists but for a wide range of medical workers of other specialities. The knowledge of structural bases of gastrointestinal tract pathology ensures understanding and proper treatment of certain stages of a disease progression, enables predicting and averting possible complications.

Aim: to study the etiology, pathogenesis, pathologic anatomy and complications of esophagus, stomach and duodenum diseases, appendicitis and peritonitis.

Task:

1 To know the etiology, pathogenesis and classification of pharynx and esophagus diseases, gastritis, stomach ulcer, appendicitis and peritonitis.

2 To learn to identify the morphologic presentations of gastrointestinal tract diseases.

3 To be able to explain the possible complications and consequences of gastrointestinal tract diseases.

The main questions for the individual training:

Esophagus diseases. Congenital diseases. Anatomic anomalies (atresia, fistulas, stenosis, congenital webs and rings). Clinicopathologic description. Diseases connected with the oesophagus's motor function disorder. Achalasia. Esophagus diverticulums (congenital and acquired). Rupture of esophagus mucous tunic (Mellori-Weiss syndrome).Varicose oesophagus veins. Esophagitis. Barrett esophagus. Etiology, patho- and morphogenesis, clinicopathologic description, complications, consequences. Esophagus tumours. Benign tumours: classification. Malignant tumours. Esophageal carcinoma. Epidemiology, classification, morphogenesis, morphologic description, complications, consequences, prognosis.

Stomach diseases. Protective barriers of the mucous tunic of stomach. Congenital stomach anomalies. Diaphragmatic hernia. Pyloric stenosis (congenital, acquired). Clinicopathologic description.

Gastritis. Definition. Acute gastritis. Etiology, pathogenesis, morphologic forms. Clinicopathologic description. Chronic gastritis, the essence of the process. Etiology, pathogenesis. Principles of classification. Forms defined on the basis of gastrobiopsies studies, morphologic description. Complications, consequences. Chronic gastritis as a precancerous condition.

Stomach ulcer. Definition. General description of peptic (chronic) ulcers of various localizations. Epidemiology, etiology, patho- and morphogenesis, its peculiarities in pyloroduodenal and mediogastric ulcers. Morphologic description of chronic ulcer in the periods of exacerbation and remission. Complications, consequences. Acute stomach ulcers: etiology, pathogenesis, morphologic description, consequences.

Stomach diseases of various etiology. Hypertrophic gastropathy. Classification, morphologic variants, clinicopathologic peculiarities. Varicose of stomach veins.

Helicobacter pylori's role in the pathogenesis of stomach diseases.

Stomach tumours. Classification. Hyperplastic polyps. Stomach adenoma. Morphologic description. Malignant stomach tumours. Stomach cancer. Epidemology, etiology, classification principles. Metastasis peculiarities. Macroscopic and histologic forms.

Congenital intestinal anomalies. Atresia and stenosis. Mekel's diverticulum. Congenital megacolon (Hirschsprung's disease). Etiology, clinicopathologic description.

Vascular intestinal diseases. Intestinal ischemia. Intestinal infarction. Angiodysplasia. Hemorrhoidal boluses. Epidemiology, etiology, pathogenesis, clinicopathologic description, complications, consequences.

Enterocolitis. Diarrheic syndrome: definition, main types, causes. Infectious enterocolitis. Necrotizing enterocolitis. Colitis connected with antibiotic therapy (pseudomembranous, medicamentous colitis). Etiology, pathogenesis, clinicopathologic description, prognosis. Intestinal inflammations of various origin.

Malabsorption syndromes. Pathogenesis and causes of malabsorption syndromes. Clinicopathologic description of diseases connected with malabsorption syndrome. The role of morphologic examination of biopsy material in the diagnosis of intestinal diseases. Diseases with malabsorption syndrome having a specific morphologic character: classification, histologic features. Diseases with malabsorption syndrome having no specific morphologic character: classification, histologic features. Gluten enteropathy. Tropical aphtae. Intestinal lipodystrophy (Whipple's disease). Syndrome of

excessive bacterial growth in small intestine. Disaccharidase insufficiency. Abetalipoproteinemia.

Idiopathic intestinal inflammations. Nonspecific ulcerative colitis. Regional enteritis (Crohn's disease). Epidemiology, etiology, patho- and morphogenesis, morphologic description, clinical representations, complications, consequences, prognosis.

Diverticulosis and intestinal obstruction. Diverticulums: definition, epidemiology, etiology, pathogenesis, morphologic description, clinical representations, consequences, prognosis. Intestinal obstruction: causes (infarct, hernias, adhesions, intussusceptions, volvulus), clinicopathologic description.

Tumours of large and small intestine. Epidemiology, nomenclature.

Pretumour diseases, nonneoplastic masses. Hyperplastic polyps. Juvenile polyps. Peitz-Jiggers polyps. Epidemiology, etiology, clinicopathologic description, prognosis.

Epithelial tumours. Benign tumours. Adenomas: epidemiology, classification, clinicopathologic description, prognosis. Familial adenomatous polyposis. Adenoma and cancer: the conception of multistage cancerogenesis in the large intestine. Large intestine cancer. Epidemiology, etiology, classification, macro- and microscopic morphologic description, clinical presentations, prognosis. Small intestine tumours: classification, clinicopathologic peculiarities. Carcinoid tumours: classification, histogenesis, morphologic description, clinical syndromes, complications, prognosis.

Appendix diseases. Normal appendix: anatomic and histologic peculiarities. Appendicitis. Classification, epidemiology, etiology, pathogenesis. Morphologic description and clinical presentations of acute and chronic appendicitis. Complications. The disease peculiarities in children and elderly people. Appendix tumours. Classification, clinicopathologic description, prognosis.

Peritoneal diseases. Peritonitis. Etiology, pathogenesis, classification, clinicopathologic description, consequences. Condensing retroperitonitis (Ormond's disease). Mesenteric cyst.

Primary and secondary peritoneal tumours. Morphologic description.

Lesson equipment:

Macropreparation: necrotic tonsillitis, esophageal carcinoma, atrophic gastritis, hypertrophic gastritis, acute stomach ulcer, chronic stomach ulcer, perforated stomach ulcer, penetrating stomach ulcer, acute duodenal ulcer, patteliform gastric carcinoma, scirrhous gastric carcinoma, phlegmonous appendicitis.

Micropreparation: necrotic tonsillitis, erosive gastritis, chronic atrophic gastritis with glands change, Helicobacter pylori on the surface of mucous tunic of stomach and between epitheliocytes in chronic atrophic gastritis, chronic superficial gastritis, chronic stomach ulcer, gastric adenocarcinoma, phlegmonous appendicitis.

Slides, tables available in the department's archives, e.g.: necrotic tonsillitis, atrophic gastritis, hypertrophic gastritis, acute stomach ulcer, chronic stomach ulcer, perforated stomach ulcer, penetrating stomach ulcer, acute duodenal ulcer, phlegmonous appendicitis.

I Preauditorium individual training for the practical training

Theoretic Material Summary

Tonsillitis (as a side effect causes digestive function disorder) or angina is an infectious disease with evident inflammatory changes of the lymphoid tissue of pharynx and tonsils. Tonsillitis is caused by streptococci, staphylococci, adenoviruses, etc. The inflammation is usually caused by

general or local supercooling. Important to the disease etiology is the sensitization of the body. More often the disease occurs in teenagers and adults up to 40 years of age, rarely – in babies and elderly people. It can be explained by age-related peculiarities of pharyngeal lymphoid apparatus development and the body's reactivity.

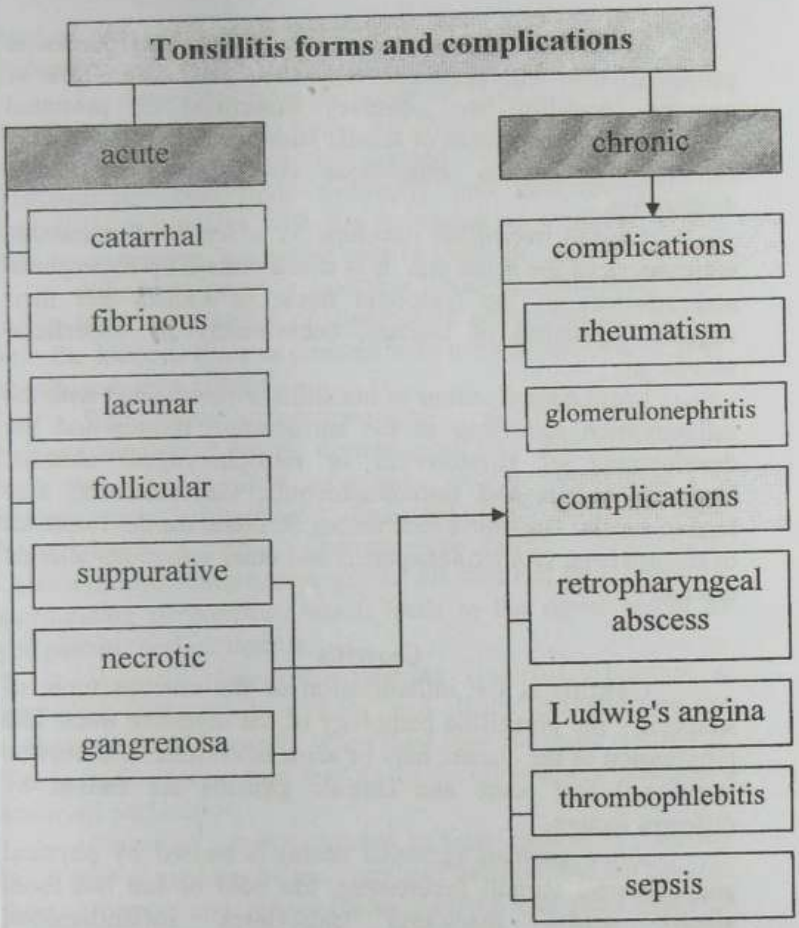
Tonsillitis progression may be acute or chronic. Acute tonsillitis is divided according to the inflammation character into catarrhal, fibrinous, suppurative, lacunar, follicular, necrotic tonsillitis and angina gangrenosa.

At the time of *catarrhal tonsillitis* the mucous tunic of tonsils and palatal arches is drastically plethoric, cyanotic, swollen and covered with serous-mucous (catarrhal) exudation.

Fibrinous tonsillitis usually occurs in diphtheria and manifests itself in diphtheritic inflammation. The mucous tunic of tonsils is covered with white-yellow coat which are difficult to remove.

Suppurative tonsillitis is characterized by the enlargement of tonsils due to their swelling and neutrophil infiltration. According to the suppurative inflammation character, this type is subdivided into quinsy (angina) and abscessing tonsillitis.

Lacunar tonsillitis is characterized by the accumulation of serous, mucous or suppurative exudation at the bottom of lacunas. It can be seen on the surface of



swollen tonsils in the form of yellow coats that are easy to remove.

In *follicular tonsillitis* tonsils are large and hyperaemic, follicles are considerably bigger in size, with central suppurative fluidizing.

Necrotic tonsillitis and angina gangrenosa occurs in people affected with scarlet fever and leucosis. At the time of necrotic tonsillitis one observes superficial or profound necrosis of mucous tunic of tonsils with haemorrhages. It may develop into angina gangrenosa characterized by tissue destruction.

Chronic tonsillitis develops as a result of numerous recurrences of the acute one. It is characterized by hyperplasia and sclerosis of the lymphoid tissue of tonsils and their capsule, dilatation of lacunas, occasionally by superficial ulcers.

Local *complications* of tonsillitis are connected with the inflammation spreading to the surrounding tissues and the development of paratonsillar or retropharyngeal abscess, Ludwig's angina and thrombophlebitis. Generalization may lead to sepsis. Tonsillitis recurrences facilitate the development of rheumatism, glomerulonephritis and other infectious-allergic diseases.

Gastritis

Gastritis is the inflammation of the mucous tunic of stomach – the prevailing pathology of the digestive tract. The progression of the disease may be acute or chronic. It should be mentioned that acute and chronic gastritis are caused by different factors.

Acute gastritis (gastritis acuta) is caused by physical and chemical stimuli (overeating, too cold or too hot food, alkalis, acids), medicines (salicylates, sulfanilamides, corticosteroids), microorganisms, mushrooms, exo- and endotoxins, for example in uraemia. The inflammation of the mucous tunic of stomach may be diffuse and focal (focal gastritis). The latter is divided into fundic, antral, pyloroantral and pyloroduodenal. According to the exudation character, one

singles out catarrhal (simple), fibrinous, suppurative (phlegmonous) and necrotic (corrosive) gastritis.

In *catarrhal (simple) gastritis* (gastritis cataralis s. simplex) the mucous tunic is thickened, swollen and hyperaemic, its surface is covered with much mucus. Histologically one finds dystrophy and desquamation of superficial epithelium with the formation of erosions. When they are numerous, it is called erosive gastritis.

Fibrinous gastritis (gastritis fibrinosa) manifests itself in the form of catarrhal or diphtheritic inflammation. In this case the mucous tunic is covered with a fibrinous coat of grey or yellow-brown colour.

Suppurative (phlegmonous) gastritis (gastritis phlegmonosa) is a heavy disease occurring due to stomach traumata, stomach ulcer, ulcerative gastric carcinoma. The mucous tunic is drastically thickened, folds are thick with haemorrhages and fibrinous-suppurative deposition. Leucocytic infiltration impregnates all stomach layers and the surrounding peritoneum, which leads to the development of perigastritis and peritonitis.

Necrotic (corrosive) gastritis (gastritis necrotica s. corrosiva) is the result of acids' and alkalis' influence on the mucous tunic of stomach, when they coagulate and destroy it. The necrotic process may lead to the development of phlegmon and even perforation.

Catarrhal gastritis treated in time ends with recovery but it may sometimes be recurrent and develop into the chronic form. Necrotic and phlegmonous gastritis end with sclerotic deformation of the organ – gastric cirrhosis.

Chronic gastritis (gastritis chronica) is a separate disease with its own etiology and pathogenesis, rarely connected with acute gastritis. Chronic gastritis is characterized by long-lasting dystrophic and necrobiotic changes of mucous tunic epithelium in combination with regeneration disorder and

structural change of the mucous tunic. The process ends with atrophy and sclerosis. The factors that can derange the regenerative process are important for the etiology of chronic gastritis. First and foremost, these are exogenous factors – eating pattern disorder, alcohol abuse, the effect of thermal, chemical and mechanical stimuli. Among the endogenous factors the greatest attention is paid to autoinfection, in particular Helicobacter pylori, to chronic autointoxication, endocrine and cardiovascular diseases, allergic reactions and duodenogastric reflux. Regeneration disorders mainly consist in the slowing-down of parietal cells differentiation. There appear immature cells that perish very soon, before the differentiation is completed. So, chronic gastritis is not an inflammatory process but a manifestation of regeneration disorder and dystrophy.

According to the topography, chronic gastritis is divided into fundic, antral and pangastritis. According to the etiology and the pathogenesis, one distinguishes between gastritis A, B and C. The prevailing one is *gastritis B* – nonimmune gastritis. It is caused by Helicobacter pylori, intoxications, alcohol abuse and malnutrition. According to Houston classification one singles out 3 types of chronic gastritis: nonatrophic, atrophic and specific forms. *Gastritis A* is autoimmune gastritis caused by the appearance of antibodies to parietal cells and characterized by the lesion of the fundic part. It often goes together with other autoimmune diseases and is accompanied by the decrease of hydrochloric acid secretion and the development of pernicious anaemia. *Gastritis C* is reflux gastritis caused by duodenogastric reflux and characterized by the lesion of the antral part. It often occurs after stomach resection.

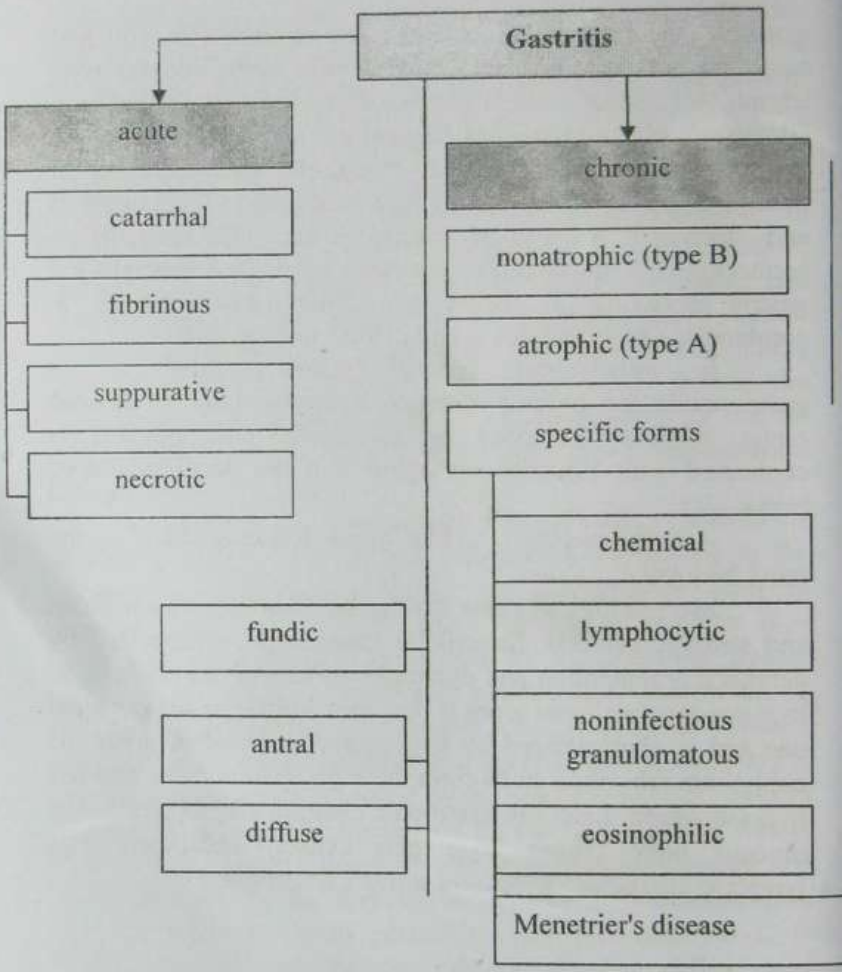
Chronic atrophic gastritis is characterized by a qualitatively new peculiarity – glands atrophy that precedes the development of sclerosis. From the endoscopic point of view,

stomach cushions are either smoothed or look like villi and resemble polyps. They are covered with epitheliocytes with edging and goblet cells (intestinal metaplasia of epithelium). Glands atrophy and mucoid degeneration of epithelium cause the disorder of pepsin and hydrochloric acid secretion. It manifests itself clinically in the increased gastrin level in blood and decreased acidity of gastric juice. This gastritis is connected with autoimmune processes, pernicious anaemia and gastric carcinoma. It occurs in patients' close relatives, in combination with thyroiditis and diffuse toxic goiter.

In *nonatrophic chronic gastritis* there is no gastrinaemia, the hydrochloric acid secretion is in the normal range, slightly decreased or increased. This gastritis is connected with Helicobacter pylori and the development of peptic ulcer.

Among specific gastritis forms reflux gastritis is the most frequent.

Morphologically one distinguishes between superficial and atrophic gastritis. Superficial gastritis is characterized by deranged regeneration and dystrophy of superficial epithelium. In some mucous tunic areas it becomes similar to the cuboidal one and is characterized by hyposecretion. In other areas the epithelium resembles in its form the high prismatic one and has hypersecretion. Later the dystrophic changes affect glands. The mucous tunic proper layer gets densely infiltrated with lymphocytes, plasmocytes and solitary neutrophils.



Menetrier's disease (giant hypertrophic gastritis) is a specific chronic gastritis form in which the mucous tunic is greatly thickened and looks like convolutions of the brain. The morphologic basis of the disease is the proliferation of

glandular epithelium cells, hyperplasia of glands, the infiltration of mucous tunic with lymphocytes, plasmocytes, epithelioid and giant cells, and the formation of cysts.

The exacerbation of chronic gastritis manifests itself in stroma edema, hyperaemia, considerable cellular infiltration with the increase of neutrophil level, occasionally microabscesses and erosions may occur. At remissions there are no such manifestations.

With the most evident processes of deranged regeneration and structural formation which lead to cellular atypia (dysplasia), chronic gastritis is often the basis for the development of gastric carcinoma.

Peptic Ulcer

Peptic ulcer is a general chronic recurrent disease that affects stomach or duodenum. The ulcer has a polycyclic progression and is characterized by seasonal exacerbations.

According to the contemporary view, the main role in the disease's etiology is played by psycho-emotional and physical overstress. Under stress conditions the system 'hypothalamus – adenohypophysis – adrenal gland cortex' gets activated and glucocorticoid production eventually increases. This hormone stimulates gastric secretion and increases the acidity of gastric contents. At the same time it decreases mucus secretion, hinders protein synthesis and cell reproduction in the mucous tunic of stomach. The ulcerogenic action of glucocorticoids also makes itself felt in cases when they are introduced for medical purposes.

Direct damaging influences on the stomach are also important– constant eating of too hot, too coarse or too spicy food, eating pattern disorder, alcohol abuse and smoking. In recent years a significant role has been allotted to Helicobacter pylori that destroys the mucous barrier of the stomach and

makes its mucous tunic vulnerable to the digestive action of gastric juices.

The etiologic role of the hereditary factor is also proved. Peptic ulcer is associated with blood group 0 (I) and the presence of Rh-antigen. It is the prevailing tonus of the parasympathetic part of the vegetative nervous system over the sympathetic part that lies in the basis of hereditary susceptibility. The vagotonia stimulates gastric secretion and creates favourable conditions for the development of ulcer.

The pathogenesis of peptic ulcer may be imagined as imbalance between the factors that damage and protect the mucous tunic. Among the damaging factors are acid gastric juices and various physical and chemical stimulations; among the protective ones – the mucous barrier, adequate blood supply, high regenerative capacity of the mucous tunic, alkalinity of saliva and pancreatic juice. All influences that cause the predominance of damaging factors over the protective ones play a certain role in the etiology and pathogenesis of ulcer.

The morphogenesis of chronic recurrent gastric or duodenal ulcer includes the following stages: erosion, acute ulcer and chronic ulcer.

Erosion (erosio) is a superficial defect of mucous tunic that does not go deeper than its muscle plate. Such defects are usually acute and very rarely chronic. They occur as a result of necrosis of a mucous tunic area with subsequent haemorrhage and rejection of the mortified tissue. At the bottom of such defects one finds muriatic haematin of black colour and at its edges – a leucocytic infiltration.

In the course of ulcer development, erosions, especially on the lesser curvature of stomach, do not close up. Under the influence of gastric juices the layers of the stomach wall necrotize deeper and deeper and the erosion develops into an *acute peptic ulcer* (ulcus acutum pepticum) of round or oval

form. The lesser curvature is known to be a 'food pathway', so it is easily traumatized. Its glands secrete very active digestive juice. The lesser curvature is rich in receptors and extremely reactive but its folds are rigid, and at the contraction of the muscular layer they cannot cover the defect. This causes bad closing up of lesser curvature injuries and the development of the acute ulcer into a chronic one (ulcus chronicum). That's why chronic ulcers are usually located on the lesser curvature, in the antral and pyloric part. Ulcers in the cardial part and on the greater curvature of stomach are rare.

Chronic ulcer may penetrate into the serous tunic. Its edges look like cushions, they are dense, sometimes callous (callous ulcer), its bottom is smooth or rough. The edge of ulcer, turned to the esophagus, is undermined and the mucous tunic hangs over the defect. There forms a pocket in which gastric contents accumulate. The edge of ulcer, turned to the hilus, is sloping. Microscopically the bottom of such ulcer is represented by connective tissue, and in the mucous tunic one finds chronic inflammation at the edges of the defect.

The indications of exacerbation of peptic ulcer are the appearance of fibrinoid necrosis isolated with a leucocytic layer and granulation tissue, and fibrinoid changes of vessel walls at the bottom of ulcer. At closing up, one can observe at the bottom of ulcer connective tissue with obliterated vessels, the epithelization of mucous tunic defect.

The morphogenesis of duodenal ulcer is identical. According to the localization, one distinguishes between bulbar ulcer (on the front or the back wall of the bulb), postbulbar (below the bulb) and "kissing" ulcers (located opposite each other on the front and the back wall of the bulb).

All complications of peptic ulcer are divided, according to V.Samsonov, into the following groups: ulcerative-destructive – haemorrhage, perforation, penetration (into pancreas, large intestine wall, liver, etc.); inflammatory –

gastritis, duodenitis, perigastritis, periduodenitis; ulcerocicatricial – narrowing of the upper and the lower (outlet) part of stomach, deformation of stomach, narrowing of the duodenal lumen, deformation of the duodenal bulb; ulcer malignization – the development of cancer; combined complications.

Haemorrhages occur in the period of exacerbation due to the fibrinoid necrosis of vessel walls (arrosive haemorrhage). An affected person vomits with "coffee grounds", the colour is determined by muriatic haematin. Fecal masses get the colour and the consistency of tar. Such fecal masses are called melaena.

Perforation (perforatio) usually occurs with ulcers on the front wall of the duodenal bulb. The inrush goes into the abdominal cavity, the retroperitoneal space and the lesser omentum. Perforation occurs in the period of exacerbation and leads to diffuse peritonitis – suppurative-fibrinous inflammation of peritoneum.

Penetration (penetratio) is spreading of ulcer beyond stomach or duodenum when other organs' tissues become the bottom of ulcer – pancreas, lesser omentum, transverse colon, gallbladder, liver. Penetration causes the digestion of the neighbouring organ's tissue by gastric juices and the inflammation of the organ.

Inflammatory complications lead to the formation of commissures. Occasionally ulcer may be complicated by phlegmon.

At closing up, ulcer leaves a thick scar that often causes pyloric stenosis. Food masses are retained in the stomach, an affected person often vomits. This causes loss of water, salts and hydrochloric acid, and the development of chlorohydropenic uraemia (gastric tetany).

Combined complications are the simultaneous occurrence of the above mentioned variants.

Stomach Cancer (Gastric Carcinoma)

More often it occurs in men older than 50.

Among the etiologic factors are endogenous nitrosoamines, exogenous nitrates, Helicobacter pylori. The precancerous conditions are adenomatous polyp, chronic atrophic gastritis, chronic stomach ulcer, gastric stump, pernicious anaemia, heavy epithelial dysplasia of the mucous tunic of stomach.

According to the localization, gastric carcinoma usually occurs in the pyloric part and on the lesser curvature.

According to the character of growth, one singles out the following clinicoanatomic (macroscopic) forms of gastric carcinoma: *exophytic expansive* growth (plaque-forming, polypous, fungous, ulcerative); *endophytic infiltrating* growth; *exoendophytic* growth. One singles out early gastric carcinoma that grows not deeper than the submucous layer.

According to the histologic structure, one distinguishes between adenocarcinoma, undifferentiated carcinoma, squamous cell carcinoma, squamous cell adenocarcinoma, unclassified carcinoma.

Metastasis of gastric carcinoma: lymphogenic (lymph nodes along the greater and the lesser curvature of stomach, Virkhov's metastasis – left supraclavicular lymph nodes, Krukenberg's tumour – both ovaries, Schnitzler's metastasis – pelvic (perirectal) fat tissue), hematogenic and implantation metastasis.

Appendicitis

Appendicitis is an acute or chronic inflammation of appendix with characteristic clinical symptoms.

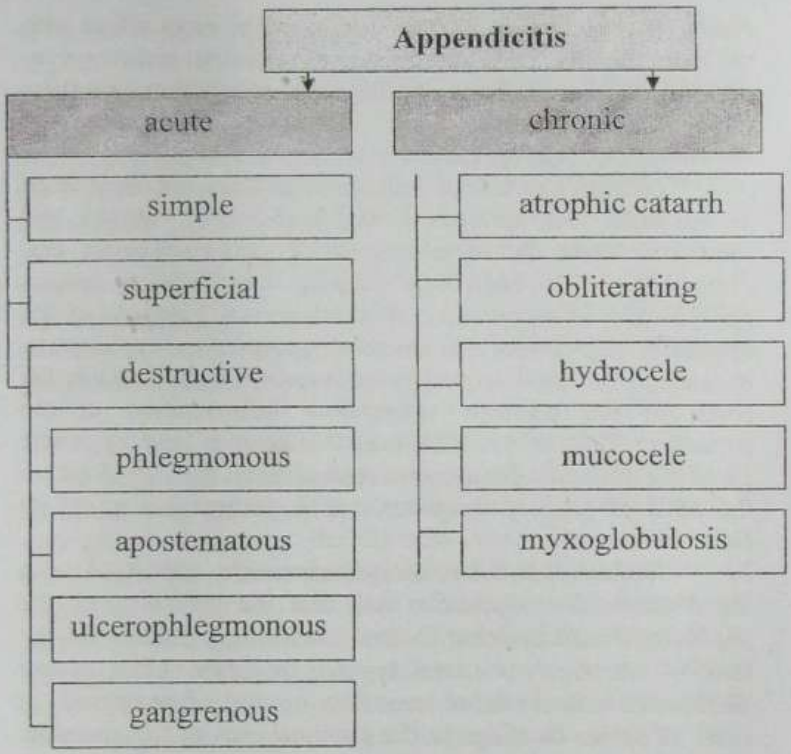
It is caused by an activated enterogenous autoinfection. Vascular disorders of neurogenic nature in the appendix wall are considered to be the starting mechanism of the disease development. Vasospasm leads to blood and lymph stasis,

haemorrhages, the organ's trophism derangement, the development of dystrophic and necrobiotic changes of its tissues. This ensures infectious organisms' invasion and the development of suppurative inflammation. Deranged peristalsis, atony and kinking of appendix, formation of fecal boluses in the lumen, presence of parasites and foreign bodies are favourable for the appendicitis development.

One distinguishes between the acute and the chronic clinicoanatomic form of appendicitis.

Acute appendicitis, in its turn, has three morphologic forms that may be regarded as consecutive stages of the inflammatory process – simple, superficial and destructive (phlegmonous, apostematous, ulcerophlegmonous, gangrenous).

In the first hours after the attack of the disease there develops *simple appendicitis*. It is characterized by blood and lymph circulation disorders, namely – stasis, edema, haemorrhages, marginal elevation of leucocytes, leucodiapedesis.



Superficial appendicitis manifests itself morphologically in primary affect. This term denotes a focus of suppurative inflammation with mucous tunic erosion on the basis of circulation disorders. The appendix grows thick and the serous tunic becomes plethoric and dingy.

From the primary affect, localized usually in the distal part of the appendix, the suppurative inflammation spreads over the whole organ – there develops *phlegmonous appendicitis*. The appendix is thick, its serous tunic is dingy and covered with fibrinous exudation. From the lumen there comes pus. The mesentery is swollen and hyperaemic. If the

suppurative process is limited around the primary affect with the formation of small abscesses, it is called *apostematous appendicitis*. On the basis of phlegmonous inflammation there often occur mucous tunic ulcers. It is typical of *ulcerophlegmonous appendicitis*.

Diffuse suppurative inflammation does not limit itself to appendix but spreads to the surrounding tissues and mesentery with the development of periappendicitis and mesenteritis. At mesentery lesion there often occurs appendicular artery thrombosis which causes gangrene of the appendix. That is how the *secondary gangrenous appendicitis* appears. It is called secondary because the thrombosis is the result of the previous suppurative inflammation of the appendix. That is the difference between it and *appendix gangrene* (primary gangrenous appendicitis) that develops on the basis of primary thrombosis or thromboembolism of its artery.

Acute appendicitis complications are connected with the destruction of appendix wall and the spreading of the suppurative inflammation to the surrounding tissues. At the time of ulcerophlegmonous appendicitis there often occurs perforation with the subsequent development of peritonitis. In cases of lumen blockage in the proximal part of the appendix pus accumulates in the distal part. The organ resembles a sack with pus (appendix *empyema*). The spreading of the inflammation to the surrounding tissues is called periappendicitis, to the blind gut – perityphlitis, to the mesentery – mesenteritis. The latter may end with suppurative thrombophlebitis of mesentery vessels. Further spreading of the process leads to the inflammation of liver veins (pylephlebitis), thromboembolism of portal vein branching and the formation of pylephlebitic abscesses in the liver.

Chronic appendicitis develops after endured acute one and is characterized by atrophic and sclerotic changes. In cases

of self-recovery acute inflammation ends with the development of granulation tissue in the zone of primary affect. There have been cases when the appendix lumen was completely filled with granulation and fibrous tissue (obliteration of appendix). Sometimes scar tissue obliterates only the proximal part and in the distal one serous fluid accumulates. The appendix turns into a cyst (hydrocele). If glands intensely produce mucus, it fills the cyst contents (mucocele). Very rarely the mucus turns into mucous globules (myxoglobulosis). Occasionally, due to cyst rupture, the mucus flows out into the abdominal cavity and the mucus-producing cells settle down and become the source of pseudomyxoma peritonei.

Regional Enteritis (Crohn's Disease)

Regional enteritis is a chronic inflammatory lesion of intestinal wall, usually of the terminal part of ileum, though it may affect all parts of the digestive tract. There develops a nonspecific granulomatous inflammation without necrosis (it resembles sarcoidosis) and with submucous tunic fibrosis and narrowing of the intestinal lumen. Typical of the disease is the alternation of affected and unaffected areas. In the mucous tunic one finds deep transverse and longitudinal ulcers, edema of the submucous tunic. Macroscopically the mucous tunic resembles a cobblestone pavement. Among the complications are diarrhea, malabsorption syndrome, intestinal obstruction, fistulas and degeneration into cancer.

II Algorithm of the practical part of the lesson

Study and be ready for the verbal description of the macropreparations:

1 Necrotic tonsillitis. The macro-specimen represents a complex of organs: tongue, trachea, pharynx. The tonsils and arches are swollen, with dotty haemorrhages. One can clearly see the ulceration, the bottom of ulcers is of grey-black colour.

Necrotic tonsillitis is characteristic of scarlet fever and acute leucosis.

2 Atrophic gastritis. The mucous tunic of stomach is atrophied and completely without folds – "bald mucous tunic".

3 Acute stomach ulcer. The mucous tunic of stomach is hypertrophied, the folds are clearly seen. In the upper part of the specimen one can see a mucous tunic defect of round form 2 cm in diameter. At the upper edge of the ulcer, from the cardial side, the mucous tunic hangs over the defect; the lower edge is turned to the pylorus and sloping. The bottom is uneven due to black necrotic masses that have not yet been torn away.

4 Chronic stomach ulcer. The mucous tunic of stomach is atrophied and almost without folds. One can see a deep defect of large size (4 x 2 cm). Its bottom is dark brown and rough. The edges of the ulcer are slightly thickened and undermined. The dark brown colour of the bottom is caused by muriatic haematin synthesized out of haemoglobin and hydrochloric acid. The roughness of the bottom is caused by necrotic masses that have not yet been torn away, and the thickening of the edges – by excrescence of connective tissue.

5 Perforated stomach ulcer. In the specimen one can see an opene stomach (cut along the greater curvature), in the middle there is the lesser curvature with very few folds. The mucous tunic folds beyond the curvature are clearly seen. On the lesser curvature, closer to pylorus, one can see a through defect, the upper edge hangs over, the lower one is sloping, the ulcer edges are slightly thickened. *Explain the perforation morphogenesis*.

6 Penetrating stomach ulcer. The mucous tunic of stomach has clearly seen folds. One can observe a defect of large size, the edges are undermined and thickened. There is an adhesion of stomach and pancreas in the area of the ulcer.

7 Acute duodenal ulcer. Below the pyloric fold one can see a mucous tunic defect of oval form. The upper edge hangs over

the defect, the lower one is sloping. The ulcer edges are not thickened, the bottom is smooth with dark brown spots.
8 Chronic duodenal ulcer. The intestinal wall is deformed due to drastic thickening of the defect edges. The ulcer is deep, its walls are white.
9 Phlegmonous appendicitis. The appendix is thickened, the peritoneum is rough in some areas due to fibrinous-suppurative exudation deposition. Especially thickened is the basis of the appendix and the blind gut wall, the mesentery.

Study the micropreparations of the theme and be ready to show on the picture the main points of pathologic process with the following descriptions:
1 *Necrotic tonsillitis*. The specimen is tinted with hematoxylin and eosin. In the specimen one can observe mucous tunic necrosis spreading to the below lying tissues. There is an inflammatory leucocytic infiltration around it. The picture of the characteristic organ structure is erased, only in some areas lymphoid tissue nests are preserved. **Designate**: 1 – lymphoid follicle, 2 – necrosis focus, 3 – leucocytic infiltration.
2 *Erosive gastritis*. The specimen is tinted with hematoxylin and eosin. The mucous tunic is swollen, hyperaemic, the epithelium is desquamated. Numerous haemorrhages, red colour and inflammation focuses, nests of densely located neutrophils, inflammatory infiltration. Dystrophic and necrotic changes in the muscular layer. Vascular hyperaemia, slight haemorrhages in the below lying layers. Erosive gastritis develops after intoxication with alkalis, acids, etc. **Designate**: 1 – epithelium desquamation, 2 – inflammatory exudation.
3 *Chronic stomach ulcer*. The specimen is tinted with hematoxylin and eosin. With the naked eye one can see a mucous tunic defect (niche). Microscopically one can see that the mucous tunic is destroyed. The ulcer edges are represented by necrotizing tissue delimited by a scarcely evident

demarcation zone and granulation tissue, and at the periphery there is connective tissue. In the muscular layer one can observe excessive excrescence of fat tissue that is a variety of connective tissue. On the basis of histologic examination one can set the diagnosis – exacerbation of chronic stomach ulcer.
Designate: 1 – epithelium necrosis, 2 – granulation tissue, 3 – connective tissue, 4 – muscular layer.
4 *Phlegmonous appendicitis*. The specimen is tinted with hematoxylin and eosin. All appendix layers are infiltrated with inflammatory exudation – one can see accumulations of neutrophil leucocytes. The mucous tunic is to a large extent destroyed, in the appendix lumen there are fecal masses (a homogenous, pink-blue mass without nucleuses) and inflammatory exudation – accumulations of neutrophil leucocytes. The vessel lumens are drastically widened and plethoric. On the serous tunic there is a fibrinous-suppurative deposition – peritonitis.
Designate: 1 – neutrophil infiltration of the wall, 2 – vessels plethora, 3 – fibrinous-suppurative exudation on the serous tunic.
5 *Stomach ulcer penetration into pancreas*. The specimen is tinted with hematoxylin and eosin. With the naked eye one can see a blue tissue (pancreas) with a defect filled with light pink tissue (connective tissue). Microscopically one finds in the connective tissue many vessels with thick walls, haemorrhages, fat tissue (areas with densely located nucleuses – many cells). So, the bottom of the chronic ulcer is in the pancreas tissue.
Designate: 1 – stomach wall, 2 – pancreas.
6 *Adenocarcinoma (ulcer - cancer)*. Microscopically one can see a mucous tunic defect. The bottom of the ulcer is represented by connective tissue of pink colour. In the below lying tissues one can observe deranged glandular structures and infiltration growth. In the deranged glands nucleuses are located not basally but chaotically. The nucleuses are of

different form, size, colour intensity, there are numerous mitoses.

Designate: 1 – the bottom of ulcer, 2 – excrescence of atypical cells.

7 *Obliterating appendicitis*. The specimen is tinted according to van Gison's method. The appendix is thinned. The wall layers are moderately infiltrated with leucocytes. Sclerotic changes prevail in the wall – the excrescence of fibrous connective tissue. One can find inflammatory infiltrations with prevailing lymphocytes. The appendix lumen is obliterated due to excrescence of granulation tissue (there are a lot of black nucleuses) that differentiates to a mature fibrous connective structure. There is no mucous tunic.

Designate: 1 – lymphocytic infiltration, 2 – excrescence of connective tissue.

Situation Tasks:

1 The child has got symptoms of weakness, feels pain at swallowing, the skin is hyperaemic with red specks, the nasolabial triangle is pale. At examination the tonsils and arches are enlarged, hyperaemic, covered with mucus, the tongue is crimson, the palate is hyperaemic. Name the pathology of tonsils.

2 The patient complained of malaise in hypochondrium, nausea. At gastroscopy one has found "bald mucous tunic of stomach" in the area of the bottom and the body. Laboratory analysis has revealed decreased level of free hydrochloric acid and anaemia. Identify the gastritis type and the cause of decreased acidity and anaemia.

3 At incision one has found a penetrating defect in the stomach wall. Its edges are like cushions, dense, the peritoneum is dingy, there is a nebulous liquid in the abdominal cavity. Set the diagnosis and identify the complication.

4 An X-ray examination has revealed a filling defect in the antral part of stomach. At the examination of post-operational material one has established that the defect is of oval form, its edges are dense, the bottom is black. Identify the pathologic process, describe the possible microscopic changes of the defect bottom, name the possible complications.

5 The patient came to the doctor complaining of pain in the right hypochondrium that had lasted for two days. At the operation one found thickened appendix, hyperaemic serous tunic covered with fibrinous-suppurative exudation. Identify the disease, describe the microscopic changes and name the possible complications.

Answers to the Situation Tasks:

1 Catarrhal tonsilitis.

2 Gastritis type A; lesion of coating cells, gastromucoprotein deficiency.

3 Perforated ulcer; peritonitis.

4 Stomach ulcer; muriatic haematin, fibrinoid necrosis, granulation tissue, demarcation bank, scar tissue, vasculitis; ulcerative-destructive, inflammatory, ulcerocicatricial, malignization, combined complications.

5 Phlegmonous appendicitis; diffuse leucocytic infiltration; perforation, peritonitis, pylephlebitic abscesses, development into secondary gangrenous one.

Test Tasks:

1 Histologic examination of gastrobiopsy has revealed chronic atrophic gastritis with considerable decrease of coating cells number. It is also known that the patient has a high level of gastrinaemia and low level of hydrochloric acid in gastric juices, signs of anaemia. Name the most probable disease.

A. Chronic fundic gastritis.

B. Chronic stomach ulcer.

C. Reflux gastritis.
D. Stomach haemorrhage.
E. Giant hypertrophic gastritis.

2 A person affected by peptic ulcer suffers from frequent vomiting with undigested food and periodic convulsion. An X-ray examination of stomach revealed pyloric stenosis. What complication has developed in the patient's body?
A. Perforated ulcer.
B. Penetrating ulcer.
C. Acute ulcer.
D. Malignization.
E. Chlorohydropenic tetany.

3 A person affected by stomach ulcer began to vomit with "coffee grounds". What complication has developed?
A. Haemorrhage from esophagus.
B. Stomach haemorrhage.
C. Malignization of ulcer.
D. Perforation of ulcer.
E. Penetration of ulcer.

4 A person affected by stomach ulcer suddenly felt pain in epigastrium, a collapse developed. At examination one found that the abdominal wall was drastically tense and painful. A laparotomy revealed in the area of the pyloric part a through defect with dense edges and gastric contents flowing out of it, the mesentery was hyperaemic, dingy, with grey deposition. Set the diagnosis taking into consideration the clinical picture and the operational findings.
A. Penetrating stomach ulcer.
B. Perforated stomach ulcer.
C. Acute stomach ulcer.
D. Chronic stomach ulcer.
E. Malignant stomach ulcer.

5 At an operation on chronic appendicitis one has found excrescence of mucus-like tissue on the peritoneum and cystic dilatation of appendix. The latter is filled with mucus and mucous masses in the form of layers. What is the diagnosis?
A. Phlegmonous appendicitis.
B. Apostematous appendicitis.
C. Primary gangrenous appendicitis.
D. Mucocele.
E. False appendicitis.

6 Microscopic examination of an ablated appendix revealed diffuse suppurative inflammation of the organ and the mesentery as well as ulcerative defects of the mucous tunic. Identify the appendicitis form.
A. Acute simple appendicitis.
B. Acute superficial appendicitis.
C. Apostematous appendicitis.
D. Ulcerophlegmonous appendicitis.
E. Secondary gangrenous appendicitis.

7 A patient died of acute loss of blood. At autopsy a large quantity of blood and blood clots in the stomach and the intestines was found. In the stomach wall on the lesser curvature there was deep defect with dense edges and a patulous vessel at the bottom. Set the diagnosis.
A. Acute stomach ulcer.
B. Stomach cancer.
C. Exacerbation of chronic stomach ulcer.
D. Perforation of stomach ulcer.
E. Penetration of stomach ulcer.

8 Macroscopic examination of an ablated appendix has shown that the serous tunic is dingy and hyperaemic. The appendix is thickened, its mesentery is swollen and hyperaemic. Microscopically one can see that the inflammatory infiltration

involves all layers of the appendix wall. Identify the morphologic form of acute appendicitis.
A. Simple.
B. Superficial.
C. Phlegmonous.
D. Apostematous.
E. Gangrenous.

9 Macroscopic examination of an ablated appendix has shown that it is thickened and its serous tunic and mesentery is hyperaemic and dingy. Microscopically one can observe diffuse leucocytic infiltration and microabscesses. Identify the morphologic form of acute appendicitis.
A. Simple.
B. Superficial.
C. Phlegmonous.
D. Apostematous.
E. Gangrenous.

10 A laparoscopy revealed signs of peritonitis and thrombosis of mesentery vessels of appendix. The appendix was thickened, the serous tunic was of dirty-grey colour. Microscopically one found haemorrhages, vessel thrombosis and appendix wall necrosis. Identify the morphologic form of acute appendicitis.
A. Simple.
B. Phlegmonous.
C. Ulcerophlegmonous.
D. Apostematous.
E. Gangrenous.

Answers to the Test Tasks:

1.A; 2.E; 3.B; 4.B; 5.D; 6.D; 7.C; 8.C; 9.D; 10.E.

Illustrations to theme

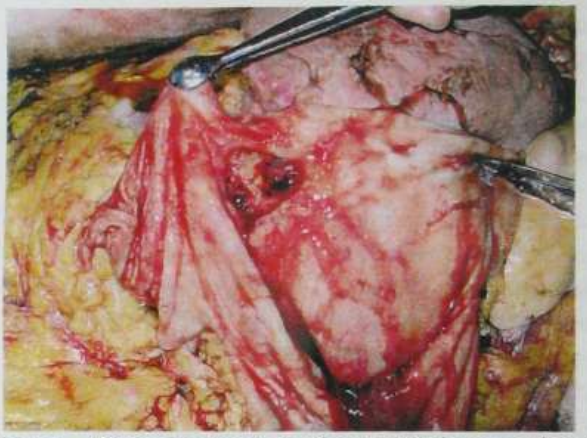


Figure 1 – Chronic stomach ulcer with haemorrhages.

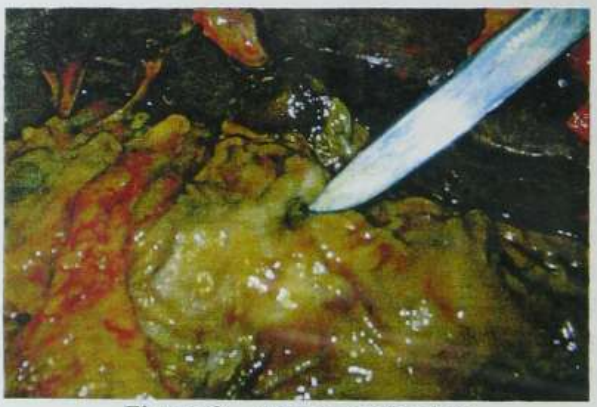


Figure 2 – Acute peptic ulcer.

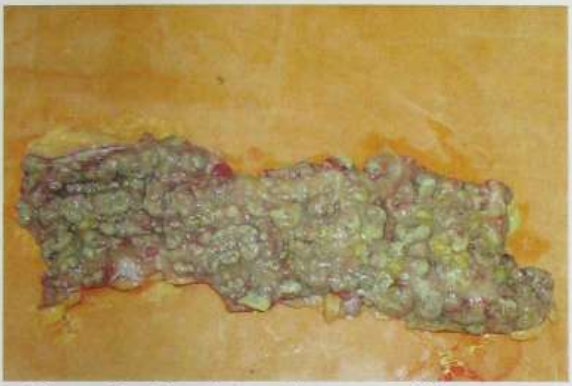


Figure 3 – Pseudomembranous colitis.

Figure 4 – Multi erosion of stomach.

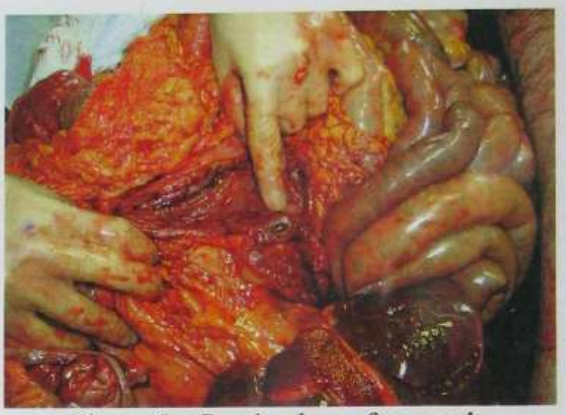


Figure 5 – Peptic ulcer of stomach.

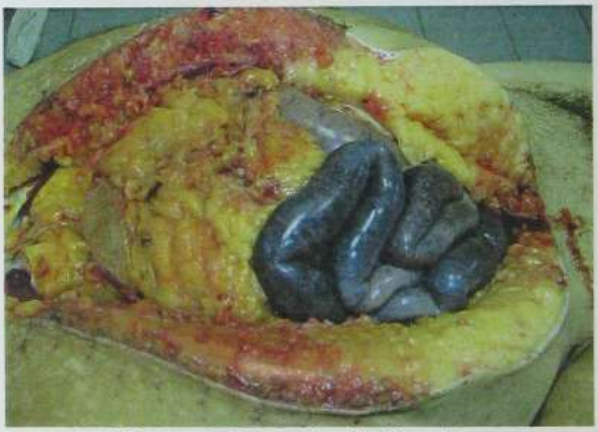


Figure 6 – Intestinal infarction.

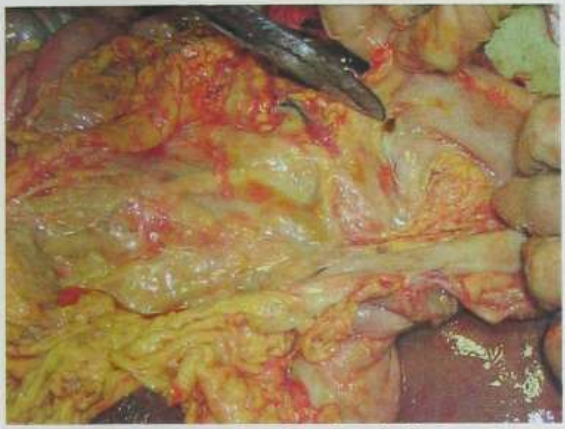


Figure 7 – Acute duodenal ulcer.

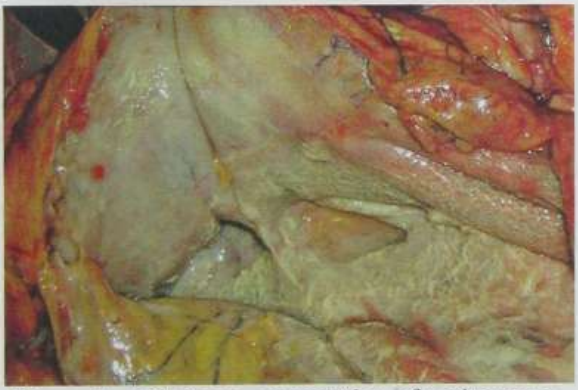


Figure 8 – Fibrinous-suppurative of peritoneum.

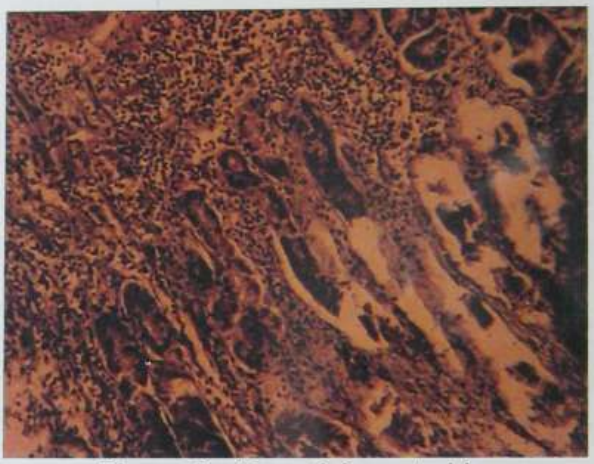


Figure 9 – Necrotizing enteritis.

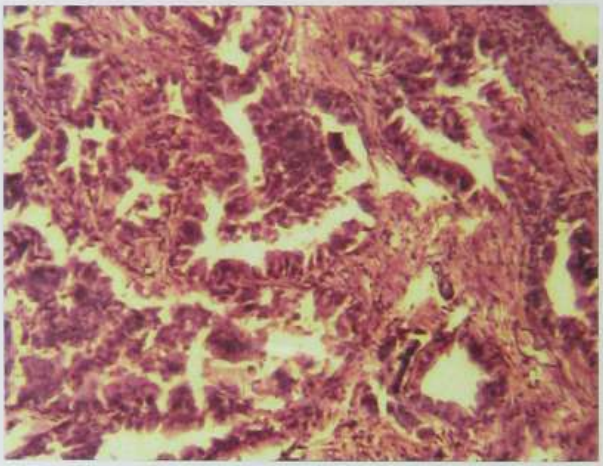


Figure 10 – Intestinal adenocarcinoma.

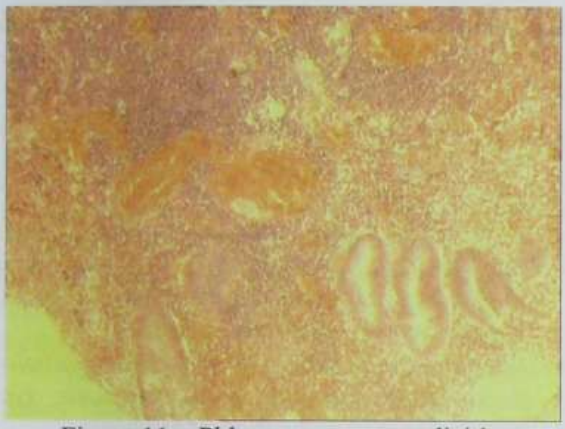


Figure 11 – Phlegmonous appendicitis.

Figure 12 – Acute duodenal ulcer.

Theme 32 Diseases of Liver, Gallbladder and Pancreas

Specific aims:

- *interpreting the main causes of liver diseases, their main clinical syndromes and biochemical signs;*
- *interpreting the etiology, pathogenesis, pathologic anatomy, extrahepatic manifestations and complications of massive hepatic necrosis and fatty hepatosis;*
- *interpreting the etiology and clinicopathologic presentations of the main cirrhosis types, morphologic classification of cirrhosis;*
- *interpreting morphologic and functional manifestations of cholelithiasis and acute and chronic cholecystitis.*

Subject Actuality: pathology of hepatobiliary organs is quite common. Most of all one has to do with liver diseases. The liver is remarkable for a great variety of its functions. No other organ has such a wide range of influence on the body's homeostasis. The liver's main functions are: metabolic, disintoxicational, biligenetic and that of biliary excretion. Besides, it is involved in digestion, blood coagulation, thermal regulation, hemodynamics, phagocytosis and other processes.

Liver lesion is caused by various factors. Among them are biologic agents (hepatitis virus, tubercle bacillus, spirochete, lamblias, amoebas, actinomycete, echinococci, ascarids); hepatotropic poisons, including medicines (tetracycline, PAS(A), sulfanilamides, steroid hormones), industrial chemicals (carbon tetrachloride, chloroform, arsenic), vegetal toxins (aflatoxin, muscarine); vaccines and serums. The following disorders are etiologically important blood circulation disorders in hepatic vessels (thrombosis,

embolism), tumours, endocrine and metabolic diseases (pancreatic diabetes, thyrotoxicosis, obesity), hereditary fermentopathies. The pathogenesis of the diseases manifests itself in two mechanisms:

1) direct lesion of hepatocytes in the form of dystrophy and necrosis;

2) immune lesion of hepatocytes by auto-antibodies.

Besides, physicians often have to do with diseases of gallbladder, bile duct and pancreas. All this arouses the necessity of knowing the structural bases of the given pathology.

Aim: learning the etiology, pathogenesis, pathologic anatomy and consequences of hepatosis, hepatitis, liver cirrhosis, cholecystitis, cholelithiasis, pancreatitis, tumescent processes in hepatobiliary system.

Tasks:

1 To know the morphologic description of massive hepatic necrosis, hepatitis, liver cirrhosis, cholecystitis, pancreatitis, liver and pancreas tumours, cholelithiasis.

2 To learn to distinguish between the hepatobiliary diseases on the basis of morphologic signs.

3 To be able to explain the possible complications of fatty hepatosis, hepatitis, liver cirrhosis, cholecystitis, pancreatitis and cholelithiasis.

The main questions for the individual training:

The causes and mechanisms of the development of liver diseases. Liver dysfunction: clinical syndromes and biochemical signs. The importance of biopsy in liver diseases diagnosis. General symptoms of necrosis, apoptosis and regeneration of hepatocytes; etiology, classification, patho- and morphogenesis, morphologic description and consequences. Hyperplasia and hypertrophy of hepatocytes.

The influence of metabolic disorders on liver. Fatty hepatosis. Etiology, clinicopathologic description, prognosis. Lipid accumulation diseases. Glycogen accumulation diseases.

Massive hepatic necrosis. Definition. Etiology, pathogenesis, pathologic anatomy, extrahepatic manifestations and complications. Hepatorenal syndrome.

Hepatitis: definition, classification. Acute viral hepatitis. Epidemiology, aetiology, ways of infection transmission, patho- and morphogenesis, clinicopathologic forms, morphologic description, viral markers, consequences. Clinical and biochemical signs of acute hepatitis. Chronic hepatitis. Etiology, morphologic description and classification, activity signs, consequences, prognosis.

Alcoholic lesion of liver. Alcoholic fatty liver. Alcoholic hepatitis. Epidemiology, patho- and morphogenesis, morphologic description, clinical presentations, complications and causes of death, consequences, prognosis.

Liver cirrhosis. Pathomorphologic signs and morphologic classification of cirrhosis. Etiologic classification of cirrhosis. Clinicopathologic description of the main types of cirrhosis. Postnecrotic liver cirrhosis. Alcoholic cirrhosis. Cirrhosis after viral hepatitis. Biliary cirrhosis (primary, secondary).

Liver tumours. Classification, epidemiology. Benign tumours. Liver cell adenoma. Intrahepatic bile duct adenoma. Hemangioma, morphologic description. Malignant tumours. Hepatocellular adenocarcinoma, epidemiology, etiology, complications, metastasis regularities. Extension levels of hepatocellular adenocarcinoma under TNM system. Hemangiosarcoma: morphologic description, clinical presentations, complications, prognosis. Secondary metastatic liver lesions.

Gallbladder and bile duct. Morpho-functional description. Composition of gall. Cholelithiasis. Etiology,

pathogenesis, types of gallstones. Cholecystitis. Definition. Acute and chronic cholecystitis. Etiology, pathogenesis, clinicopathologic description, complications and causes of death. Gallbladder and bile duct tumours. Cholangiocellular carcinoma: clinicopathologic description.

The diseases of the exocrine part of pancreas. Pancreatitis acute (pancreatonecrosis) and chronic. Epidemiology, etiology, pathogenesis, morphologic description, clinical presentations, complications and causes of death.

The tumours of the exocrine part of pancreas. Pancreas cancer: epidemiology, classification, morphologic description, prognosis.

Lesson equipment:

Macropreparation: fatty hepatosis, massive hepatic necrosis, liver abscess, micronodular (portal) liver cirrhosis, mixed liver cirrhosis, secondary biliary cirrhosis, postnecrotic liver cirrhosis, varicose of esophagus veins, calculous cholecystitis, gallstone, liver in mechanical (obstructive) jaundice.

Micropreparation: massive hepatic necrosis, fatty liver, interstitial suppurative hepatitis, acute viral hepatitis, monolobular (portal) liver cirrhosis, multilobular (postnecrotic) liver cirrhosis (tinted with hematoxylin + eosin, and picrofuchsin), biliary cirrhosis.

Slides, tables and electronogrammes available in the department's archives, e.g.:
postnecrotic liver cirrhosis, varicose of esophagus veins, calculous cholecystitis, gallstone.

I Preauditorium individual training for the practical training

Theoretic Material Summary

Hepatosis Massive hepatic necrosis (acute hepatosis) is characterized by the progressive necrosis of liver parenchyma. It is usually caused by exogenous (mushroom poison, chemical compounds) and endogenous (pregnancy, thyrotoxicosis) factors. In the progression of massive hepatic necrosis one singles out the stages of yellow atrophy, red atrophy and recovery. The duration of the disease is about three weeks.

During the first days one can observe fatty degeneration of hepatocytes in the centre of the particle. It is quite soon followed by necrosis and autolytic destruction. The liver becomes smaller, flaccid and turns yellow. That's why it is called yellow atrophy.

The detritus yields to resorption by macrophages. The stroma becomes "uncovered" and sinusoids, finding no resistance of hepatocytes, become overfilled with blood. The liver turns yellow with red specks (the stage of red atrophy). At this stage hepatic failure often develops.

Hepatic failure has general clinical representations of tissue turgor decline, xeroderma, skin icteritiousness and sclera, vessel "stars" and skin haemorrhages, enlargement or diminution of liver, there often occurs splenomegaly, ascites, edemas. The pathologic process progression provokes a complex of hepatic, mental and neurologic disorders. The affected person has fetor hepaticus, the liver aches at palpation and he/she suffers from fever and leucocytosis.

The gravity of hepatic failure is usually estimated according to how deep nervous and mental disorders go. Three stages of hepatic failure are singled out. The stage of psycho-emotional disorders is characterized by emotional instability: swift change of humour, depression or euphoria, insomnia at

night and sleepiness in daytime, headache, giddiness, memory weakening. The stage of neurologic disorders and impairment of consciousness manifests itself in sudden excitation which is followed by inhibition, tremor of hands, lips and eyelids. Progressive hepatic failure ends with coma (the third stage).

Hepatitis

Hepatitis is an acute or chronic liver disease characterized by dystrophic and necrobiotic changes of parenchyma combined with the inflammatory stroma infiltration. Hepatitis may be a separate nosologic unit (primary) or a manifestation of other diseases (secondary).

Primary hepatitis develops under the influence of hepatotropic viruses (viral hepatitis), alcohol (alcoholic hepatitis), medicines (medicamentous hepatitis), cholestasis (cholestatic hepatitis). Viral and alcoholic hepatitis are the most common forms of hepatitis.

Secondary hepatitis accompanies a wide range of diseases. They are infectious diseases (typhoid fever, dysentery, cytomegalia, yellow fever, malaria, tuberculosis, sepsis), thyrotoxicosis, rheumatic diseases, digestive tract pathology, intoxications.

Acute hepatitis can be exudative and productive. Exudative hepatitis is in its turn divided into serous and suppurative.

Chronic hepatitis is characterized by parenchyma destruction, cellular infiltration of stroma, sclerosis and changed regeneration. Three types of it are singled out – aggressive, where hepatocytes dystrophy and necrosis prevails, persistent, where cellular infiltration of portal areas and intraparticular stroma prevails, cholestatic characterized by cholestasis, cholangitis and cholangiolitis.

Light cases of hepatitis end with full recovery but massive liver lesion may lead to the development of cirrhosis.

Viral hepatitis is caused by hepatotropic viruses. Liver cells are damaged either by the allergic reaction of cytolytic type or by the hypersensibility of delayed type. Autoimmunisation is connected with a specific liver lipoprotein that forms as a result of virus replication in hepatocytes and acts as an auto-antigen. After the recovery the disease leaves typospecific immunity that's why the person may be affected by a different type of viral hepatitis.

The following clinicopathologic forms of viral hepatitis are singled out: acute cyclic (icteric), anicteric, necrotic (malignant), cholestatic, chronic.

At its peak the *cyclic (icteric) form* is characterized by the ballooning degeneration, focal and coagulation necrosis of hepatocytes. Groups of hepatocytes that have undergone coagulation necrosis form round homogenous eosinophilic structures which are forced out into perisinusoid spaces – Councilman's corpuscles. Cholestasis and necrosis of hepatocytes results in hepatocellular jaundice. At the same time there occurs lympho- and macrophage infiltration of portal tracts and sinusoids. Macroscopically, the liver is larger in size, the capsule is tense, dense and red *(large red liver).*

In the course of recovery the liver returns to normal size, hyperaemia decreases. The capsule is somewhat thickened and dingy; adhesions appear between the capsule and the peritoneum. Reparative processes prevail over the destructive ones, lympho- and macrophage infiltration becomes focal. The process ends with liver sclerosis that may develop into cirrhosis.

The *anicteric form* of viral hepatitis, compared to the icteric one, is characterized by less evident morphologic changes although at laparoscopy one finds the picture of large red liver. Ballooning degeneration and Councilman's corpuscles are rarely found in this form. But one can clearly observe proliferation of reticuloendotheliocytes. Lympho- and

macrophage infiltrations do not destroy the terminal plate, there is no cholestasis.

The *necrotic form* is first and foremost marked by the progressive necrosis of parenchyma. The liver rapidly diminishes in volume, becomes contracted and grey-brown in section. Microscopically one can observe necroses of hepatocytes, the accumulation of reticuloendotheliocytes, Councilman's corpuscles, "uncovered" stroma as a result of resorption of necrotic masses, haemorrhages, cholestasis in capillaries. If the affected person does not die of hepatic coma, postnecrotic liver cirrhosis develops.

The *cholestatic form* manifests itself in prevailing cholestasis with the development of cholangitis and cholangiolitis on the basis of hepatocytes destruction, and lympho-, macrophage and neutrophil infiltration of stroma. One often finds Councilman's corpuscles.

The *chronic form* of viral hepatitis is represented by active or persistent hepatitis. Active hepatitis develops on the basis of sclerotic liver changes. It is characterized by ballooning degeneration, necrosis of hepatocytes and inflammatory stroma infiltration. Liver regeneration proves incomplete which leads to the development of cirrhosis. The persistent form is characterized by prevailing infiltration of sclerosed portal areas with lymphocytes, histiocytes and plasmatic cells. Dystrophic hepatocyte changes are low-grade. Chronic persistent hepatitis rarely develops into cirrhosis.

In viral hepatitis death occurs due to acute or chronic hepatic failure.

Alcoholic hepatitis is an acute or chronic liver disease caused by alcoholic intoxication. Ethanol and acetaldehyde are hepatotropic poisons. Ethanol is neutralized by liver ferment - alcohol dehydrogenase. Its synthesis in liver is genetically predetermined and quantitatively specific for each individual. After a long period of alcohol abuse the alcohol

dehydrogenase's protective effect is not sufficient to safeguard the liver from affection and at a certain alcohol concentration there occurs hepatocyte necrosis. The cytotoxic effect of alcohol, even in small doses, manifests itself in the liver which has been previously affected by such diseases as chronic hepatitis, fatty hepatosis and cirrhosis. Cessation of alcohol consumption leads the process into a benign course. But if alcohol consumption continues, chronic hepatitis progresses and ends with liver cirrhosis as ethanol drastically suppresses regenerative potential of the organ.

At the time of acute alcoholic hepatitis the liver is larger in volume and dense, light brown areas alternate with brown-red ones. Microscopically one can observe necrosis of centrolobular hepatocytes. The so called alcoholic hyaline (Malori's corpuscles) can be found in their cytoplasm which is an important diagnostic sign. Peripheral hepatocytes are in the state of fatty degeneration. Necrosis areas and portal tracts are infiltrated with neutrophils. Occasionally, especially in previously affected liver, massive hepatic necrosis occurs. In most cases after the cessation of alcohol consumption the liver structure regenerates.

Chronic alcoholic hepatitis does not differ morphologically from active and persistent viral hepatitis. It is identified by the presence of Malori's corpuscles in the cytoplasm of hepatocytes and beyond the cells. Alcoholic hyaline is a fibrillar protein which is synthesized by hepatocytes under the influence of ethanol and causes their destruction. Chronic alcoholic hepatitis ends with the development of cirrhosis.

Liver Cirrhosis

Liver cirrhosis is a chronic diseases characterized by sclerosis, structural change and the deformation of liver. The pathomorphology of cirrhosis includes the following liver changes: hepatocytes dystrophy and necrosis, deranged

regeneration, diffuse sclerosis, structural change and deformation of the organ. At the time of cirrhosis the liver is dense and gibbous, its volume usually diminishes but in rare cases it may increase.

The cirrhosis development is based on hepatocytes dystrophy and necrosis. Their destruction leads to intense regeneration of preserved parenchyma. It results in the formation of nodular regenerates and false particles which are wrapped in connective tissue. The false particles are characterized by deranged angioarchitecture. They often lack the central vein or it is located in peripheral areas and connective tissue membrane develops in sinusoids. All this causes blood circulation disturbance in the liver. Increasing hypoxia leads to dystrophy and destruction of hepatocytes in nodular regenerates and to intense excrescence of connective tissue, which further disturbs the microcirculation. The process develops like a chain reaction with constant intensification of sclerotic changes.

The classification of sclerosis is based on etiologic, morphologic, morphogenetic and clinicofunctional criteria.

Postnecrotic cirrhosis develops after massive necrotic liver changes, for example after massive hepatic necrosis, viral or alcoholic hepatitis. The necrotized tissue resolves, the stroma and central veins collapse, triads are close to each other. Vast fields of connective tissue develop in these areas and from the organ's surface they look hollow. Big nodular regenerates appear. According to its morphology, it is usually a macronodular form of cirrhosis, more rarely – a mixed one.

Portal cirrhosis is a micronodular form. It develops as a result of the circulation deficiency, chronic alcoholic hepatitis, malnutrition and metabolic disorders. The connective tissue expands in the directions of portal tracts and penetrates into liver particles in the form of processes dividing the particles

into smaller false ones. Moderate cellular infiltration of stroma remains as a manifestation of previous hepatitis.

Biliary cirrhosis can be primary and secondary. *Primary cirrhosis* is the result of nonsuppurative destructive (necrotic) cholangitis and cholangiolitis. In response to destruction there occurs proliferation and cicatrisation of bile ducts, infiltration and sclerosis of the periportal areas, the destruction of peripheral hepatocytes and the formation of septa and false particles as in portal cirrhosis. The liver is enlarged, grey-green in section, and its surface is smooth or fine-grained.

Secondary biliary cirrhosis is caused by cholestasis (cholangiostatic cirrhosis) as a result of extrahepatic obstruction of bile duct (stone, tumour) or by bile duct infection with the development of bacterial, usually suppurative, cholangitis and cholangiolitis (cholangiolitic cirrhosis).

Classification of Liver Cirrhosis

According to etiology	According to morphology	According to morphogenesis	According to clinicofunctional criteria
Infectious (viral hepatitis, parasitic liver diseases) Toxic and toxicoallergic (alcohol, hepatotropic poisons, medicines, allergens)	Micronodular Macronodular	Postnecrotic Portal Biliary Mixed	According to the extent of hepatocellular deficiency (cholemia, hypoalbuminemia, hypothrombinemia, hypoonchia, haemorrhages, coma) According to the extent of portal hypertension (ascites, oesophagogastric haemorrhage)
Biliary (cholangitis, cholestasis)			According to the process activity (active, moderately active, inactive)
Metabolic-alimentary (insufficiency of proteins, vitamins and lipotropic factors, accumulation diseases)			
Circulatory (chronic venous stasis)			According to progression (progressive, stable, regressive)
Cryptogenic (of unidentified etiology)			

Morphologic symptoms of cirrhosis are dilatation and rupture of bile capillaries, which causes peripheral hepatocytes necrosis. Connective tissue expands according to the morphogenesis of portal cirrhosis. In secondary biliary cirrhosis the liver is enlarged, dense and green due to bile impregnation, in section one can see dilated ducts filled with bile.

Mixed cirrhosis appears as a result of portal one supplemented at a certain stage by necrotic liver changes.

Liver cirrhosis causes typical extrahepatic derangements: jaundice and haemorrhagic syndrome as a sign of hepatocellular deficiency, cholestasis and cholemia; exhaustion as a result of digestion disorders caused by stasis and atrophy of gastrointestinal tract in portal hypertension; splenomegalia as a result of reticuloendothelium hyperplasia and sclerosis. This leads to the development of extrahepatic portacaval shunts due to which some blood bypasses the liver and discharges the portal vein. Affected people have dilated veins in the esophagus, the hemorrhoidal plexus and in the stomach, dilated hypodermic veins in the thorax and the abdominal wall. The latter are called "Medusa heads". The varicosity of the above mentioned veins goes together with the thinning of their walls which is often the cause of profuse esophageal, gastric or hemorrhoidal haemorrhage. As a result of portal hypertension and the lesion of liver parenchyma where the degradation of antidiuretic hormone occurs, the transudate infiltrates into the abdominal cavity, sometimes in the volume of 10 litres. This phenomenon is called ascites. The ascitic fluid, accumulated in the abdominal cavity, compresses blood vessels and internal organs deranging the blood flow. In kidneys one finds signs of acute renal insufficiency (tubular epithelium necrosis) and, occasionally, hepatic immune complex glomerulonephritis, which cause the development of hepatorenal syndrome. In most cases people affected by

cirrhosis die of chronic hepatic failure. Besides, cirrhosis may be the basis for the development of liver cancer.

Cholecystitis

Among the pathologic processes in the gallbladder acute and chronic inflammation (cholecystitis) and gallstones are the most common.

At the time of *acute cholecystitis* the inflammation can be catarrhal, fibrinous and suppurative (phlegmonous). It is caused by ascending and descending infection on the basis of biliary dyskinesia and cholestasis. Important to its development are gallstones which traumatize mucous tunic often causing pressure sores. Acute cholecystitis is complicated by the breaking of gallbladder wall with the development of bile peritonitis. In cases of gallbladder duct obstruction and pus accumulation in the cavity gallbladder empyema develops. The spreading of the suppurative process beyond the organ is complicated by suppurative cholangitis, cholangiolitis and pericholecystitis with the formation of adhesions.

Chronic cholecystitis is the result of acute one. Morphologically it manifests itself in mucous tunic atrophy and sclerosis with lymphohistiocytic infiltration. Occasionally the petrification of the gallbladder wall and adenomatous excrescence of the mucous tunic occur.

Gallstones are often the cause of calculous cholecystitis. Such cases manifest themselves in the chronic inflammation with periodic exacerbations. The gallbladder wall may be broken by the stone causing the development of bile peritonitis. When the stone comes down into the general bile duct and causes its occlusion, obstructive jaundice develops.

Cholelithiasis

Cholelithiasis is a disease defined by the formation and presence of concrements in hepatic and extrahepatic bile ducts.

The main difference from calculous cholecystitis lies in the fact that in cholelithiasis the stones are in intrahepatic ducts. The disease is polyetiologic. The interaction of such factors as genetic susceptibility, malnutrition, metabolic disorders, bile duct infections and cholestasis creates conditions under which bile tends to form stones. What is considered to be important to the change of normal bile into lithogenic one is the decrease of the cholato-cholesterol index – the ratio between the contents of biliary acids and cholesterol in the bile. When the quantity of biliary acids is insufficient, cholesterol turns into sediment and gives stimulus to the formation of stones. But their formation also requires favourable local conditions – bile duct inflammation, mucus discharge, absorption disorders in gallbladder, local allergization. According to I.V.Davydovskyi, the main morphologic signs of cholelithiasis are the presence of Luschke's ducts, excrescence of non-striated muscles and glandular hyperplasia of gallbladder mucous tunic. Luschke's ducts are the channels that are lined with prismatic epithelium and reach muscular and subserous tunic of the organ. It is in them that bile accumulates, which facilitates the formation of stones. The second sign of cholelithiasis is the productive granulomatous inflammation. Granulomas appear as a result of ulcero-necrotic lesion of bile ducts and the gallbladder with bile penetration. As a result of regeneration its components get immured in connective tissue. Cholesterol crystallizes and turns into sediment. It is resorbed by gigantic cells of "foreign bodies" which form a granuloma.

Cholelithiasis may be complicated by choledochitis, cholangitis, cholangiolitis, pressure sores in the general bile duct and the gallbladder, bile peritonitis, obstructive jaundice, secondary biliary liver cirrhosis, reactive hepatitis and cholangiocellular liver cancer.

Liver Cancer

Primary liver cancer is the eighth on the list of cancers of other localization.

According to the macroscopic picture, one singles out nodular – one or several green nodes – and diffuse cancer, according to its growth pattern – infiltrating, expansive and mixed cancer. According to the histogenesis, one singles out hepatocellular and cholangiocellular cancer.

Hepatocellular cancer is the most common one. In 60-80% of cases it develops on the basis of liver cirrhosis. One often finds HbsAg in cancer cells.

According to the macroscopic picture, liver cancer may have trabecular, solid or trabecular-solid construction with cellular atypism, invasion into venous vessels and subsequent hematogenic metastasis.

Cholangiocellular cancer is more common among people older than 60. It grows out of bile duct epithelium and is not connected with cirrhosis. In the macroscopic picture it resembles a dense node of white colour. According to the microscopic structure, it is more often an adenocarcinoma, sometimes tumour cells secrete mucus. It usually spreads in a lymphogenous way.

More often one can find secondary metastatic malignant tumours in the liver which metastasize from the gastrointestinal tract, lungs, kidneys or mammary gland.

The malignant process in the liver may result in hepatic failure which is often the cause of death.

Pancreas diseases

Pancreatitis. One distinguishes between acute and chronic pancreatitis.

Acute pancreatitis is connected in 80 % of cases with cholelithiasis or with alcoholism. Important to the pathogenesis of the disease development is the ischemic lesion of the organ's

parenchyma due to arterial thrombosis; medication damage, etc. With the disease progression there appear white or yellow-white areas of fat necrosis in the surrounding tissues (steatonecrosis). The gland is swollen, sometimes one can observe haemorrhagic imbibition of parenchyma. In such cases the tissue turns black-brown with the areas of necrosis.

Chronic pancreatitis often occurs after a long period of alcohol consumption. Fibrosis, cicatricial narrowing of ducts, acinar tissue atrophy develops in the tissue. The gland is dense and grey; in some places one can find cysts with calcareous content.

Pancreas tumours are divided into benign (adenoma) and malignant (carcinoma). The head of pancreas is affected in 60% of cases, the body – in 20%, the tail – in 5%. Head carcinomas obstruct the outlet of general bile duct and cause obstructive jaundice.

II Algorithm of the practical part of the lesson

Study and be ready for the verbal description of the macropreparations:

1 Fatty hepatosis. In sagittal slice one can see that the organ is enlarged, the parenchyma is yellow, the surface is smooth. Fatty degeneration of liver develops mainly due to the infiltration. Fats enter the liver from the intestine through the portal vein. In the liver fats break up into their components out of which proper fats are synthesized in the tissues. In hypoxia the fats in the liver do not decompose completely and accumulate in hepatocytes. Various toxins of exo- or endogenous origin traumatize and damage hepatocytes reducing their function, so that incompletely decomposed fats accumulate in hepatocytes. The toxic lesion of hepatocytes leads to the destruction of their intracellular ultrastructures composed of lipoproteins and these destruction products (lipids) also accumulate in hepatocytes. Such mechanism of

fatty degeneration development is called decomposition. Decomposition plays a secondary role in the development of fatty hepatosis. The organ is enlarged due to fat accumulation.

2 Massive hepatic necrosis. The stage of yellow atrophy. In sagittal slice one can see that the organ is diminished, the parenchyma is ochre-yellow, the surface is wrinkled, the vessels are dilated. The liver diminution is caused by the evident destructive (necrotic) changes of hepatocytes. This leads to the wrinkling of the capsule and the dilatation of internal organs vessels.

3 Massive hepatic necrosis. The stage of red atrophy. In sagittal slice one can see that the organ is diminished, the parenchyma is motley: one can see red and grey spots on a yellow background. The liver capsule is wrinkled. The liver diminution is caused by evident necrotic changes of hepatocytes that go together with the dilatation of internal organs vessels, which are overfilled with blood, and cause red colouring. So the red areas are the parenchyma necrosis zones.

4 Liver abscess. In sagittal slice one can see that the parenchyma is destroyed almost all along and it represents a loosened porous mass. The parenchyma is preserved only under the capsule in the form of narrow stripe of homogenous tissue that looks like clay. Such vast liver abscesses are usually metastatic and of hematogenic origin. The inflammation of abdominal cavity organs, usually appendicitis, may be complicated by phlebitis with subsequent thrombosis – phlebothrombosis. Having come off, the septic thrombus enters the liver through the portal vein and an abscess develops there. Such abscess is called pylephlebitic, "pyle" – gate, "phlebitis" – the inflammation of venous wall. So the abscess that has developed is the complication of the inflammation of the portal vein wall.

5 Micronodular (portal) liver cirrhosis. The liver is enlarged, the surface is fine-gibbous all along. The organ's enlargement

indicates the initial stage of cirrhosis development where regenerative processes prevail over destructive ones. Small regeneration nodes indicate the fact that they have been preceded by small necrosis areas which is typical of portal cirrhosis. In section the liver is yellow; dense connective tissue network of light grey colour runs trough the parenchyma.

6 Mixed liver cirrhosis. The liver is enlarged, the surface is gibbous due to big and small nodes. In section one can see white connective tissue bars. Different sizes of regeneration nodes suggest that they have been preceded by necrosis of different extent usually caused by different factors. That is why such cirrhosis is called mixed.

7 Biliary liver cirrhosis. In section one can see considerably dilated intrahepatic bile ducts filled with thickened bile and small yellow stones. In the liver tissue one can observe a dense network of white connective tissue bars. The surface is fine-gibbous and the parenchyma is white with a green hue. The green colour is the indication of cholestasis.

8 Postnecrotic liver cirrhosis. In section one can see that the parenchyma is yellow-green due to fine unsaturated green specks; the surface is coarse-tuberous. The liver is drastically diminished in volume.

9 Varicose esophagus veins. In mucous tunic of the lower esophagus part one can see dark blue bars – dilated veins that bulge out into the esophagus lumen. It is one of portacaval shunts which expand at portal hypertension. The varicose node rupture is complicated by haemorrhage which in some cases becomes fatal.

10 Calculous cholecystitis is represented by two macro-specimen:

a) the gallbladder is enlarged, its wall is thickened, the lumen is filled with thickened bile and small stones;

b) the gallbladder is enlarged, its wall is thickened, the lumen is filled with small faceted stones.

11 Gallstone. The gallbladder is enlarged, its wall is thickened. The gallbladder is filled with one big stone with rough surface. In section one can see circular layered rings around the nucleus.

12 Liver in mechanical (obstructive) jaundice. The organ's size is not noticeably changed. In section one can see dilated intrahepatic bile ducts and the yellow-green hue of tissue. The surface is fine-gibbous.

Study the micropreparations of the theme and be ready to show on the picture the main points of pathologic process with the following descriptions:

1 *Massive hepatic necrosis, the stage of red atrophy*. The specimen is tinted with hematoxylin and eosin. Only solitary hepatocyte areas are preserved. They are a little more intensely coloured and have preserved nucleuses and radial orientation of trabecules. Around the intact liver tissue one can observe a homogenous anhistous mass – detritus (parenchyma destruction products) – and massive haemorrhages. **Designate**: 1 – preserved hepatocytes, 2 – detritus, 3 – haemorrhage.

2 *Fatty liver*. The specimen is tinted with hematoxylin and eosin. The liver's structure is preserved. In centrolobular hepatocytes one can find light-coloured vesicles – drops of fat that has been eluated at the preparation of histologic sections with alcohol. At the particles' periphery some hepatocytes have big hyperchromic nucleuses, some of them contain three nucleuses, which is the sign of regeneration. In some areas of interparticular stroma there are small clusters of cells: lymphocytes, fibroblasts and histiocytes, which indicate productive inflammation that precedes sclerosis. **Designate**: 1 – centrolobular hepatocytes, 2 – multinuclear hepatocytes, 3 – cellular infiltration.

3 *Interstitial suppurative hepatitis*. The specimen is tinted with hematoxylin and eosin. In the stroma one can observe

inflammatory infiltrations of various sizes with neutrophils prevailing in their cellular composition. The liver preserves the particular structure, in the particles hepatocytes are located radially, the spaces between trabecules are widened. In centrolobular hepatocytes the cytoplasm and nucleuses are of lighter colour (dystrophic effect) in comparison with the periphery. **Designate**: 1 – trabecules, 2 – cellular infiltration.

4 *Portal liver cirrhosis*. The specimen is tinted according to van Gison's method. The liver's structure is deranged – one can see false particles that are the areas of liver parenchyma fragmented with connective tissue after bridging necroses. Thus one can observe several central veins located eccentrically. The false particles are confined by thick layers of connective tissue. The sclerotic process progresses, which is indicated by the presence of granulation tissue in the stroma. Besides, one can see regeneration nodes – small areas of liver parenchyma in which the hepatocyte trabecules have no radial orientation and central veins are missing. So in this case we have progressive portal liver cirrhosis. **Designate**: 1 – false particle, 2 – connective tissue layers, 3 – eccentrically located central veins.

5 *Biliary liver cirrhosis*. The specimen is tinted according to van Gison's method. One can see dark brown granules in the lumen of dilated bile capillaries – cholestasis. The stroma is thickened due to connective tissue. Basically the liver's structure is preserved, but one can observe fatty degeneration of hepatocytes – light-coloured vesicles – which is neutral fat in hepatocytes which has been eluated at the preparation of sections with alcohol. One finds false particles and regeneration nodes. **Designate**: 1 – bile capillaries, 2 – connective tissue layers, 3 – fatty degeneration of hepatocytes.

6 *Acute viral hepatitis*. The specimen is tinted with hematoxylin and eosin. In the parenchyma one can observe inflammatory infiltrations of various sizes with lymphocytes

prevailing in their cellular composition. The liver preserves the particular structure, in the particles hepatocytes are located radially, the spaces between trabecules are widened. In centrolobular hepatocytes the cytoplasm and nucleuses are of lighter colour (dystrophic effect) in comparison with the periphery. **Designate**: 1 – trabecules, 2 – cellular lymphocytic infiltration.

Situation Tasks:

1 Three weeks after the appendectomy the patient underwent another operation. A large liver abscess was found. Explain the cause of the liver abscess development.

2 A week after mushroom poisoning the 10-year-old girl died. In section the liver is diminished and red, the capsule is wrinkled. What is the pathologic process in the liver? What is the cause of the girl's death?

3 A country woman produced and sold home-brewed vodka for a long period of time. She died at home after continuous vomiting with blood. What is the most probable cause of the gastric haemorrhage?

4 Two months after blood transfusion, jaundice was found in the patient's body, with liver enlargement, increased level of transaminases and sedimentary test derangement. What is the presumable disease, its etiology and the type of jaundice?

5 The jaundice developed suddenly in the body of a person who had suffered for a long time from calculous cholecystitis. Identify the presumable cause and type of jaundice.

Answers to the Situation Tasks:

1 Pylephlebitis, bacterial embolism, pylephlebitic abscess.

2 Massive hepatic necrosis, hepatic coma.

3 Alcoholic cirrhosis (portal); the haemorrhage comes from varicose veins of stomach and esophagus.

4 Viral hepatitis; hepatitis virus B; hepatic jaundice.

5 Bile duct occlusion; obstructive.

Test Tasks:

1 A 37-year-old woman was operated on ulcerophlegmonous appendicitis. Three days later she suffered from hectic fever, pain in the area of the right hypochondrium, icteritiousness of skin and visible mucous tunics; in her blood the increased levels of conjugated and unconjugated bilirubin were found. What complication of acute destructive appendicitis developed in the given case?

A. Pelvis minor abscesses.
B. Subphrenic space abscesses.
C. Pylephlebitic abscesses.
D. General fibrinopurulent peritonitis.
E. Viral hepatitis.

2 A 6-year-old child was taken to the resuscitation department with signs of hepatorenal syndrome which developed after eating mushrooms. The next day she died. In section the liver was diminished, the capsule – wrinkled, the parenchyma – of cherry-brown colour, with numerous haemorrhages. Histologically one could observe deranged liver particle structure, massive centrolobular hepatocytes necrosis, central veins of particles were preserved but overfilled with blood. What disease such changes are typical for?

A. Parenchymatous hepatitis.
B. Fatty hepatosis.
C. Acute active hepatitis.
D. Massive hepatic necrosis.
E. Persistent hepatitis.

3 For the sake of liver pathology identification, patient C. underwent puncture biopsy of the organ. Histologically one found hydropic degeneration and necrosis of hepatocytes, a

large quantity of Councilman's corpuscles. Electronomicroscopically one found hyaloid hepatocytes and sandy nucleuses. What diagnosis is the most probable?
A. Progressive massive necrosis.
B. Viral hepatitis.
C. Acute alcoholic hepatitis.
D. Persistent alcoholic hepatitis.
E. Hepatosis.

4 At the cholecystectomy on chronic calculous cholecystitis one discovered that the gallbladder was completely filled with faceted stones. What is the type of jaundice in this case?
A. Mechanical.
B. Obstructive.
C. Haemolytic.
D. Parenchymatous.
E. No jaundice.

5 Mushroom poisoning of the patient resulted in jaundice with the signs of hepatic failure. One observed progressing liver diminution. Identify the most probable pathology.
A. Nutmeg cirrhosis.
B. Biliary cirrhosis.
C. Obstructive jaundice.
D. Postnecrotic cirrhosis.
E. Massive hepatic necrosis.

6 The patient has undergone 8-year treatment for liver pathology which developed after viral hepatitis type A (Botkin's disease). At histologic examination of the biopsy material one has found dilated and sclerosed periportal areas, false particles and regeneration nodes, fatty degeneration of hepatocytes. What is the most probable pathology?
A. Portal cirrhosis.

B. Postnecrotic cirrhosis.
C. Biliary cirrhosis.
D. Liver adenoma.
E. Hepatocellular cancer.

7 The autopsy of a woman who had suffered for a long time from calculous cholangitis revealed an enlarged, dense, fine-grained and green in section liver with dilated and bile-filled ducts. What is the most probable cirrhosis form?
A. Primary biliary cirrhosis.
B. Secondary biliary cirrhosis.
C. Portal cirrhosis.
D. Postnecrotic cirrhosis.
E. Mixed cirrhosis.

8 Three months after blood transfusion one found jaundice in the patient's body. The liver was enlarged. Puncture biopsy revealed ballooning degeneration of hepatocytes and Councilman's corpuscles. What pathologic process is implied?
A. Viral hepatitis type A.
B. Viral hepatitis type B.
C. Acute massive hepatic necrosis.
D. Chronic aggressive hepatitis.
E. Viral hepatitis type E.

9 Palpation of a person, affected by chronic nonspesific pheumonia for the last five months, showed an enlarged liver. Puncture biopsy revealed hyperaemia of central veins and sinusoids, degeneration of centrolobular hepatocytes and hypertrophy of peripheral ones, copper cells proliferation, continuous basic membrane in sinusoids. Identify the presumable pathologic process in the liver.
A. Nutmeg liver.
B. Nutmeg cirrhosis.

C. Portal cirrhosis.
D. Mixed cirrhosis.
E. Hepatitis.

10 The patient died of hepatorenal insufficiency. In section one found portal liver cirrhosis. Histologically one observed fatty degeneration of hepatocytes and the presence of Mellori's hyaline. What cirrhosis developed in the patient's body, according to the etiologic principle?
A. Viral.
B. Alcoholic.
C. Autoimmune.
D. Hemochromatic.
E. Obstructive.

Answers to the Test Tasks:

1.C; 2.D; 3.B; 4.E; 5.E; 6.A; 7.B; 8.B; 9.A; 10.B.

Illustrations to theme

Figure 1 – Secondary biliary cirrhosis.

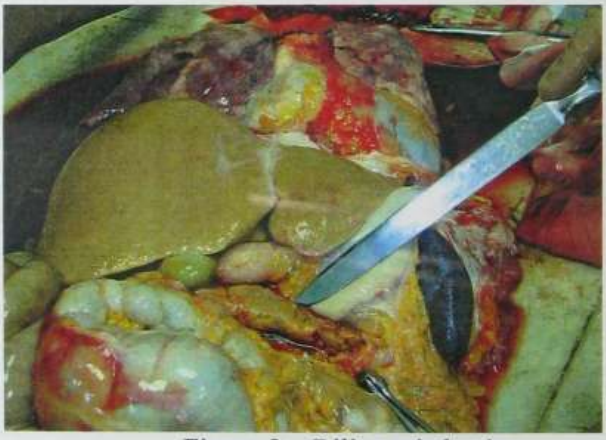


Figure 2 – Biliary cirrhosis.

Figure 3 – Acute viral hepatitis.

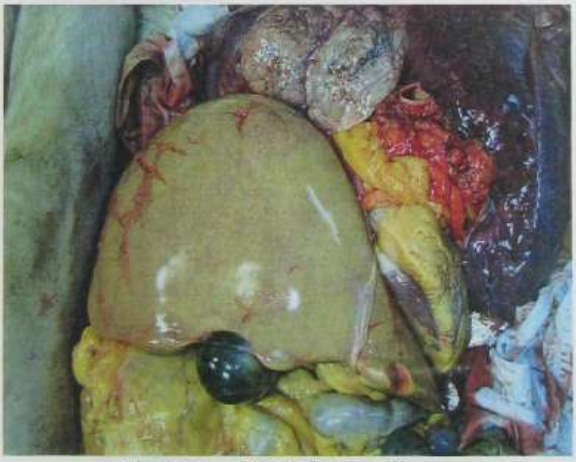


Figure 4 – Fatty hepatosis.

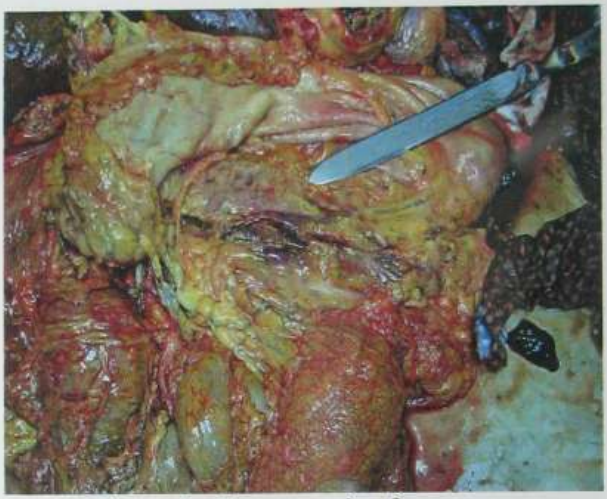


Figure 5 – Fat necrosis of pancreas.

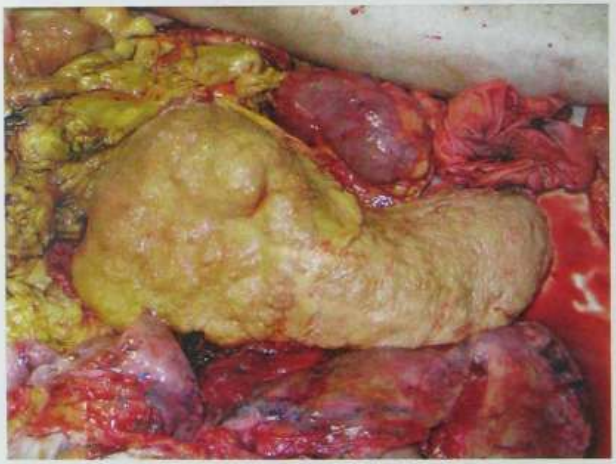


Figure 6 – Mixed cirrhosis.

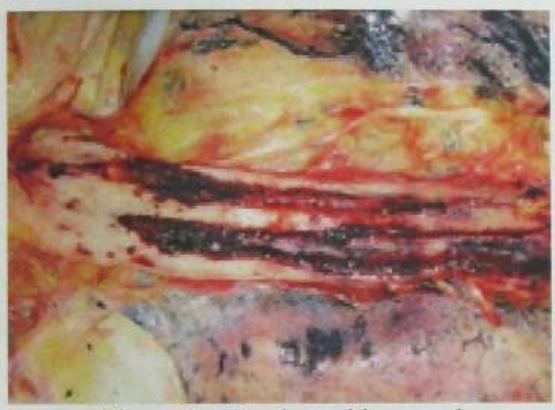


Figure 7 – Esophageal haemorrhage.

Figure 8 – Mesenchymoma of liver in child.

Figure 9 – Circulatory (chronic venous stasis) cirrhosis.

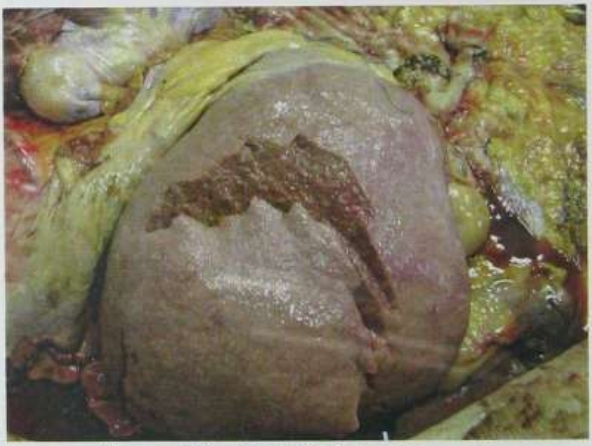


Figure 10 – Portal cirrhosis.

Figure 11 – Calculous cholecystitis and biliary cirrhosis.

Figure 12 – Jaundice peritonitis.

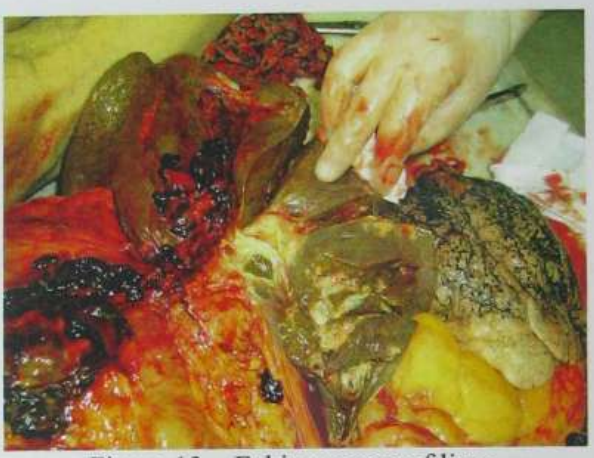


Figure 13 – Echinococcus of liver.

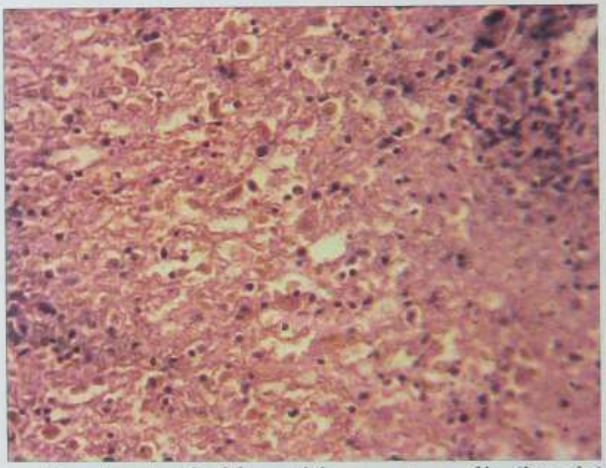


Figure 14 – Acute viral hepatitis - acute cyclic (icteric) form.

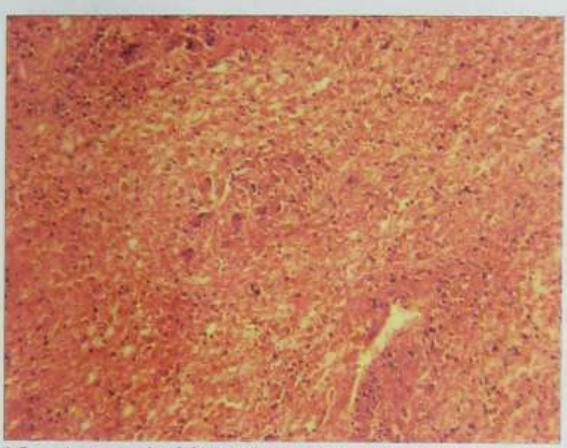


Figure 15 – Acute viral hepatitis - necrotic (malignant) form.

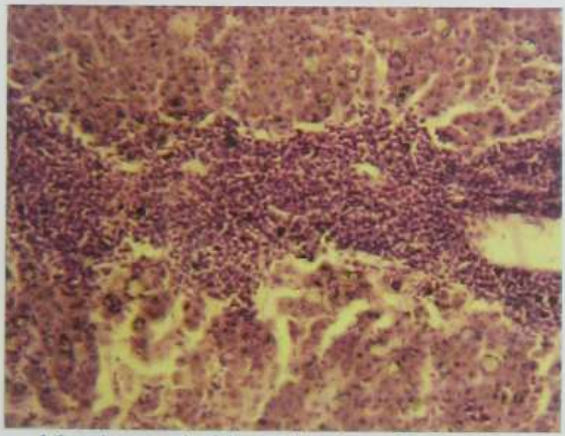


Figure 16 – Acute viral hepatitis- lympho- and macrophage infiltration of portal tracts.

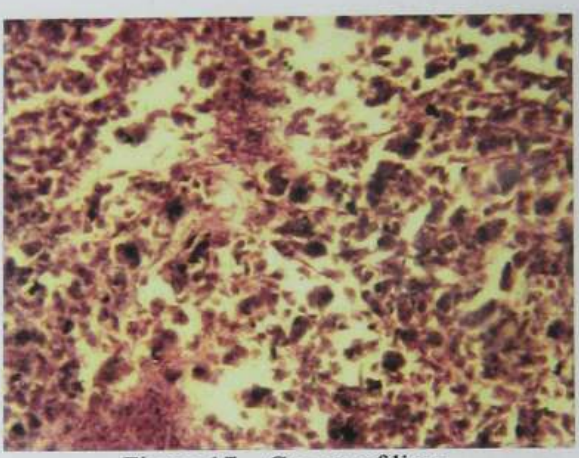


Figure 17 – Cancer of liver.

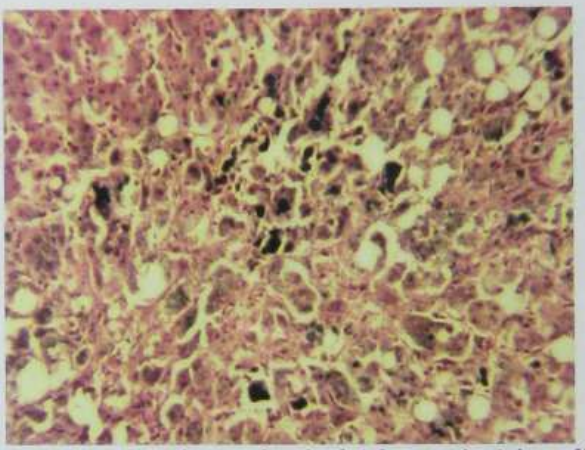


Figure 18 – Liver in mechanical (obstructive) jaundice.

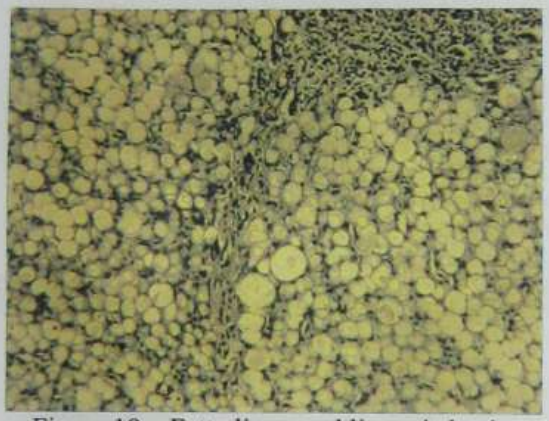


Figure 19 – Fatty liver and liver cirrhosis.

CONTENTS

Content module 6

Навчальне видання

Романюк Анатолій Миколайович,
Карпенко Людмила Іванівна

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