ОШСКИЙ
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УНИВЕРСИТЕТ

Кафедра «Естественно-научных дисциплин»

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Фонд тестовых заданий предназначен для контроля знаний студентов направления **лечебное дело** по дисциплине **«Патологическая анатомия»**

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1. INTRODUCTION

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Complications
Variability
The mechanism of death
+The reason of development
The mechanism of development

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Downwards
+Upwards
Without it
By side it
Outside

3. THE SPECIAL STAIN FOR GLYCOGEN AND GAG IS
Sudan III
Picrofuchsin by von Giesone
+PAS-reaction
Perl's reaction
By Ziehl-Nielsen

4. THE TERM "PATHOGENESIS" IN RELATION TO DISEASE MEANS
Complications
Variability
The mechanism of death
The reason of development
+The mechanism of development

5. STUDY OF MICROSECTION IN THE LIGHT MICROSCOPE BEGINS
In immersion oil
In polarized light
+In small magnification
In the large magnification
In electronic field

6. POPULARITY OF IMMUNOHYSTOCHEMICAL METHOD DETERMINES BY
Simplicity
High sensitivity
+High specificity
Availability
Variability

7. THE TERM"THANATOGENESIS" IN RELATION TO DISEASE MEANS
Complications
Variability
+The mechanism of death
The reason of development
The mechanism of development

8. THE SUBJECT OF CYTOLOGY RESEARCH IS
Biopsy material
+Biological fluids and feces
Autopsy material
Tissue sections
Necrotic tissue

9. THE OBJECT INVESTIGATEDS BY PATHOLOGIST ARE
Experimental material
+Postmortem and surgical material
Alive patients
The data of sociological interrogations
Biological liquids and feces

10. THE TERM "PATHOMORPHOSIS" IN RELATION TO DISEAS MEANS
Complications
+Variability
The mechanism of death
The reason of development
The mechanism of development

11. IMMUNOHISTOCHEMICAL RESEARCH IS USED FOR REVEALING OF
Aautoantigens
+Tumorous histogenesis
Genetic diseases
A kind of infection
Autoantibodies

12. THE BASIC PURPOSES OF AUTOPSY IS
To define correctness of treatment
To reveal the reason of death of the patient
+To establish the final diagnosis
To establish biological age of patient
To investigate internal organs

13. THE FOUNDER OF MODERN ANATOMIC PATHOLOGY IS
R.Brhait
+R.Virchov
A.I.Over
E.O.Mukhin
K.Rokitansky

14. THE MAIN METHOD OF BIOPSY EXAMINATION IS
Biochemical
Radiological
Microbiological
+Histological
Ultrasonic

15. THE MAIN LEVEL OF PATHOLOGICAL PROCESS STUDY IS
Subcellular
+Tissue

Cellular
Systemic
Organic

16. PICROFUCHSIN BY VON GIESONE SELECTIVELY REVEALS
Nervous fibers
Smooth muscle cells
Epithelial cells
+Connective tissue and collagen fibers
Fat

17. THE TERM "ETIOLOGY" IN RELATION TO DISEASE MEANS
Complications
Variability
The mechanism of death
+The reason of development
The mechanism of development

18. AUTOPSY IS PERFOMED IN CASE OF DEATH FROM
Gunshot wound
Hangings
Poisonings
+Diseases
Not clear reasons

19. THE SYNONYM OF THE TERM "POSTMORTEM EXAMINATION" IS
Section
Biopsy
+Autopsy
Necropsy
Vivisection

20. THE MOST WIDESPREAD LEVEL OF BIOPSY MATERIAL EXAMINATION IS
Subcellular (ultrastructural)
Macroscopic (anatomic)
+Microscopic (histologic)
Chemical (laboratory)
Surgical

21. SPECIAL STAIN FOR IRON -CONTAINED SUBSTANCES DETECTION IS
Sudan III
Picrofuchsin by von Giesone
PAS-reaction
+Perl's reaction
By Ziehl-Nielsen

22. TOLUIDINE BLUE IS STAIN FOR DETECTION OF
Neural tissue
Cellular nucleus
Basophilic structures
+Metachromatic substances
Muscle tissue

23. SPECIAL COURSE OF ANATOMIC PATHOLOGY STUDIES
Structure of the diagnosis
+Renal diseases
Processes of adaptation
Hemodynamic disorders
Symptoms of diseases

24. INVESTIGATION OF THE SLICE OF ALIFE PATIENT TISSUE WITH THE DIAGNOSTIC PURPOSE IS
Section
+Biopsy
Autopsy
Necropsy
Vivisection

25. SPECIAL STAIN FOR LIPIDS DETECTION IS
+Sudan III
Picrofuchsin by von Giesone
PAS- reaction
Perl's reaction
By Ziehl-Nielsen

26. THE STAIN USUALLY USED FOR PRIMARY HISTOLOGIC EXAMINATION IS
Sudan III
+Hematoxylin and eosin
Picrofuchsin by von Giesone
Alcyan blue
Congo red

27. THE AUTOPSY REVEALES
Medicament treatment
+The reason of death of the patient
Metabolism in an organism
Correctness of the clinical diagnosis
Symptoms of diseases

28. THE STAIN USUALLY USED FOR DETECTION OF AMYLOID IS
Sudan III
Hematoxylin and eosin
Picrofuchsin by von Giesone
Alcyan blue
+Congo red

29. REACTIONS OF METHACHROMASIA PREDISPOSE
Change of size
Change of density
Change of brightness
+Change of color
Decreasing of object

30. STAIN FOR DETECTION OF ACID GLYCOSAMINOGLYCANS IS

Hematoxylin and eosin
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+Alcyan blue
Congo red
Sudan-IV

31. « CONGO RED » IS USED FOR DETECTION OF
Glycogen
DNA
Fat
+Amyloid
Melanin

32. STAINING IN FOCI OF MUCOID DEGENERATION REFERS TO PHENOMENON OF
Fluorescence
Birefringence
+Metachromasia
Dysplasia
Metaplasia

33. THE STAIN FOR REVEALING OF LIPID INCLUSIONS IS
Eosin
+Sudan III
Congo red
Picrofuchsin
Hematoxylin

34. THE STAIN SPECIFIC FOR IRON IS
Hematoxylin and eosin
Sudan III
+Perl's reaction
Congo red
PAS-reaction

35. RESULT OF PERLS' REACTION IS
Nil's blue
+Prussian blue
Congo red
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Cancer pearls

36. THE METHOD OF ULTRASTRUCTURAL INVESTIGATION IS
Light microscopy
Autopsy
Vivisection
+Electronic microscopy
Observation

37. THE LIGHT MICROSCOPY INVESTIGATIONAL LEVEL IS
Organic
+Histologic
Ultrastructural

Organism
Population

38. THE ELECTRONICMICROSCOPY INVESTIGATIONAL LEVEL IS
Organic
Histologic
+Ultrastructural
Organism
Population

39. ONE OF THE GENERAL PATHOLOGIC PROCESSES IS
+Inflammation
Metaplasia
Dysplasia
Petrification
Atrophy

40. ONE OF THE GENERAL PATHOLOGIC PROCESSES IS
+Necrosis
Metaplasia
Dysplasia
Petrification
Atrophy

2. INTRACELLULAR ACCUMULATION

1. STEATOSIS OF LIVER OBSERVES AT
Sports
Flu
Lactation
Pneumonia
+Adiposity

2. INTRACELLULAR ACCUMULATION OF GLYCOGEN IS MARKED AT
Anemia
Adiposity
Alcoholism
+Tesaurismoses
Diabetes

3. THE MORPHOGENETIC MECHANISM OF FATTY DYSTROPHY DEVELOPMENT ON THE PERIPHERY OF HEPATIC LOBULE IS
+Infiltration
Decomposition
Plasmorrhagia
Transformation
Perverted synthesis

4. PARENCHIMATOUSE DYSTROPHIES MAY BE
Water-mineral
Nucleoprotein
Chromoprotein

+Carbohydrate
Mixed

5. THE FIGURATIVE NAME OF THE HEART AT FATTY DYSTROPHY IS
"Bull"
"Goose"
"Hairy"
+"Tiger"
"Porphyry"

6. TYPICAL OUTCOME OF BALOON DYSTROPHY OF CELL IS
Apoptosis
Shrinking of cell
Coagulative necrosis
+Colliquative necrosis
Crush of cell

7. ACCUMULATION OF LIPIDS IN PARENCHYMAL CELLS IS TERMED AS
+Steatosis
Sclerosis
Melanosis
Hyalinosis
Mucinosis

8. HEREDITARY DISEASES OF INTRACELLULAR ACCUMULATION ARE KNOWN AS
System
+Tesauresmoses
Autoimmune
Cerebrovascular
Immunocomplex

9. THE CONSISTENCE OF THE LIVER AT STEATOSIS
+Flabby
Dense
Unchanged
Soft-elastic
Stone-like

10. PROTEINURIA REFLECTS PRESENCE OF ALBUMINOUS DYSTROPHY IN
+Kidney
Liver
Urethra
Urinary bladder
Intestine

11. THE SURFACE OF THE LIVER AT STEATOSIS IS
+Smooth
Rough
Glazy
Granulated
Wrinkled

12. HYDROPIC DYSTROPHY OF HEPATOCYTES IS CHARACTERISTIC OF
Steatosis
+Hepatitis
Fibrosis
Diabetes
Hemochromatosis

13. THE MAIN REASON OF FATTY DYSTROPHY DEVELOPMENT IN MYOCARDIUM IS
+Hypoxia
Arterial hypertension
Glycogenosis
Protein insufficiency
Infection

14. THE SIZE OF THE LIVER AT STEATOSIS
+Increased
Reduced
Unchanged
Renewed
Fragmentized

15. STEATOSIS AS A CONSEQUENCE OF PROTEIN INSUFFICIENCY DEVELOPS IN
Heart
Kidneys
+Liver
Spleen
Brain

16. THE FEATURE OF FATTY DYSTROPHY IN MYOCARDIU IS
Macro-dropletdeposition of lipids
Diffuse character of lipids deposition
+Pulverized accumulation of lipids incell
Accumulation of lipids on pathway of fine veins
Decrease of myocardium contract abilities

17. THE FIGURATIVE NAME OF THE LIVER AT STEATOSIS IS
"Sago liver"
"Beckon"
+"Goose"
"Tiger"
"Icing"

18. HYDROPIC DYSTROPHY OF RENAL TUBULES IS CHARACTERISTIC OF
Steatosis
+Inflammation
Fibrosis
Diabetes
Alcoholism

19. PARENCHIMATOUSE DYSTROPHIES MAY BE
+Protein
Water-mineral

Nucleoproteins
Chromoproteins
Mixed

20. MORPHOGENETIC MECHANISM OF DYSTROPHY DEVELOPMENT IS
Plasmorrhagia
Destruction
+Infiltration
Transmission
Resolution

21. STEATOSIS CAN DEVELOP IN
+Liver
Lungs
Spleen
Vessels
Intestine

22. THE NEPHROTIC SYNDROME IS CHARACTERIZED BY
Hyperproteinemia
Hypoproteinuria
Hematuria
+Massive proteinuria
Hypermelanosis

23. THE MORPHOGENETIC MECHANISM OF FATTY DYSTROPHY DEVELOPMENT IN THE CENTER OF HEPATICLOBULEIS
Infiltration
+Decomposition
Liquefaction
Transformation
Perverted synthesis

24. SEVERE FORM OF FATTY DYSTROPHY IN MYOCARDIUM IS CHARACTERIZED BY
Decreasingof heart chambers
Rusty color of myocardium
Diffuse character of spreading
Accumulation of protein droplets in myocardiocytes
+Flabby consistence of heart

25. FACTOR THAT MAY RESULT IN THE INTRACELLULAR ACCUMULATION OF FAT IS
Decreased amount of fat nutrition
Plasmorrhagia
Extravascular hemolysis
+Overproduction of accumulated material by cells
Increased fat utilization

26. WHAT KIND OF DYSTROPHY IS CHARACTERISTIC FOR “TIGER HEART”
Protein
Balloon
Hydropic
+Fatty

Carbohydrate

27. MORPHOGENETIC MECHANISM OF FATTY DYSTROPHY IN HEPATOCYTES IS
Exudation
+Decomposition
Exposition
Transposition
Perverted synthesis

28. THE PATHOLOGIC PROTEIN SUBSTANCE ACCUMULATING PERICOLAGENIC OR PERIRETICULARY IN VARIOUS TISSUE AND ORGANS IS
Glycogen
Hyaline
Water
+Amyloid
Lipids

29. THE STROMA-VASCULAR DYSTROPHY IS
Lethal damage of cells
Overgrowth of collagen in stroma
Infringement of parenchymal organs function
Convertible damage of connective tissue
+The kind of damage by extracellular accumulation of abnormal quantities of various substances

30. THE SUBSTANCE WHICH HAS RED COLOR BY CONGO RED STAINING IS
Lipid
Hyaline
Water
+Amyloid
Glycogen

31. DUE TO LONG-TERM HYPERTENSION AND DIABETES MELLITUS THE WALLS OF ARTERIOLS BECOME
Sclerozated
Thinned
+Hyalinizated
Ulcerated
Pigmented

32. THE CHARACTERISTIC OF AMYLOID FIBRILS IS
With definite length
Multybranching
Tubular
Lipoprotein
+Composed of paired filaments

33. REVERSABLE PATHOLOGICAL PROCESS IS
+Mucoid swelling
Fibrinoid degeneration
Amyloidosis
Apoptosis
Necrosis

34. THE ORGAN AFFECTED IN BOTH PRIMARY AND SECONDARY AMYLOIDOSIS IS
+Kidneys
Stomach
Uterus
Brain
Lungs

35. THE CELLS ACCUMULATING FAT IN ATHEROSCLEROTIC PLAQUE FORMATION ARE
Fibroblasts
Epithelial cells
+Foam cells
Lymphocytes
Leukocyte

36. THE DEPOSITIONS OF AMYLOID IN RENAL TISSUE IS REVEALED IN
Epithelium of proximal renal tubules
Intimae of blood vessels
Pelvic membrane
+Tubular basement membranes
Calices of the kidneys

37. AT ATHEROSCLEROSIS UNDER INTIMA OF AORTA ACCUMULATE
Apolipids
Cholesterol
Fatty acids
+Cholesterol and its ethers
Triglycerides

38. THE IRREVESIBLE PROCESS OF HIGH WEIGHT MOLECULAR PROTEINS ACCUMULATION IN EXTRCELLULAR MATRIX IS
Hemosiderosis
Glycogenosis
Melanosis
Mucoid changes
+Fibrinoid changes

39. DISTHROPHY IS CHARACTERIZED BY DEPOSITION IN CELL OF
Exogenous substances
Organic acids, nucleic acids
Bacteria bodies
+Lipids, proteins, carbohydrates, pigments
Products of necrosis

40. ACCUMULATION OF ETHERS OF CHOLESTEROL IN MACROPHAGES AT THE CHRONIC CHOLECYSTITIS REFERS TO
Steatosis
Necrosi
+Cholesterolosis
Hyalinosis
Apoptosis

41. CHOLESTEROL ACCUMULATS IN
Apoptosis
Anthracosis
+Atherosclerosis
Necrosis
Glycogenosis

42. CELLS ACCUMULATED CHOLESTEROL AND ITS ESTERS IN ATHEROSCLEROTIC PLAQUE ARE
+Macrophages and smooth muscle cells
Leukocytes and fibroblasts
Lymphocytes and erythrocytes
Fibroblasts and leukocytes
Erythrocytes and lymphocytes

43. ACCUMULATION OF AMYLOID IN RENAL TISSUE OBSERVES IN
Cytoplasm of tubules epithelium
+Arterioles walls
Venues walls
Pelvis
Spaces of Bowmen's capsule

44.THE STROMA-VASCULAR DISTROPHY IS
Lethal damage of cells
Overgrowth of collagen in stroma
Parenchymal organs dysfunction
Reversible damage of connective tissue
+Damage by extracellular congestions of abnormal quantities of various substances

45. THE ORGAN AFFECTED ONLY IN SECONDARY AMYLOIDOSIS IS
Heart
Kidneys
Liver
Striated muscles
+Lungs

46. REACTIVE SYSTEMIC AMYLOIDOSIS MAY OCCUR IN ASSOCIATION WITH
Croupous pneumonia
Flue
+Tuberculosis
Myocardial infarction
Appendicitis

47. THE STAINS FOR DETECTION OF AMYLOID IS
Sudan III
Perl's reaction
Picrofuscin by van Giesone
+Methyl violet
Hematoxylin and eosin

48. LIPIDS ABLE TO COLLECT IN VESSEL WALL ARE
Phospholipids

Triglycerides
+Cholesterol ethers
Triglycerides ethers
Phospholipids ethers

49. THE SUBSTANCE WITH FIBRILLAR STRUCTURE THAT FORMS IN PATHOLOGICAL CONDITIONS IS
Reabsorbing droplets
Russell bodies
Lipids
+Amyloid
Petrificats

50. CHOLESTEROL ACCUMULATION IN CELL OCCURS IN
Hypertension
+Atherosclerosis
Apoptosis
Inflammation
Necrosis

51. PRIMARY ADIPOSITY BY ETHYOLIGY MAY BE
Intestinal
Cardio-vascular
+Cerebral
Hypertrophic
Anemic

52. MACROSCOPICALLY, ORGANS AFFECTED BY AMYLOIDOSIS ARE
+Enlarged, firm, waxy
Diminished, firm, waxy
Enlarged, soft, smooth
Diminished, soft, smooth
Rough, enlarged, firm

53. FOCAL ACCUMULATION OF LIPIDS AT STROMA-VASCULAR DISRTOPHY IS
Steatosis
Sclerosis
Melanosis
Hyalinosis
+Lipomatosis

54. THE MOST COMMON REASON OF DEATH IN SECONDARY AMYLOIDOSIS IS INSUFFICIENCY OF
+Kidneys
Heart
Liver
Lungs
Adrenals

55. HYALINOSI IS THE OUTCOME OF
Fatty dystrophy
Colliquative necrosis

+Mucoid and fibrinoid degeneration
Vacuolar dystrophy
Leucocytes infiltration

56. THE KIND OF STROMA-VASCULAR DYSTROPHY IS
Mucination
Steatosis
+Fibrinoid degeneration
Anthracosis
Beriliosis

57. THE KIND OF STROMA-VASCULAR DYSTROPHY IS
Anthracosis
+Amyloidosis
Mucination
Necrosis
Balloon dystrophy

58. AMYLOIDOSIS CAN BE COMPLICATION OF
Essential hypertension
Flue
Ischemic heart disease
Diphtheria
+Secondary tuberculosis

59. AMYLOIDOSIS CAN BE COMPLICATION OF
Essential hypertension
Flue
Diphtheria
+Chronic pulmonary abscess
Ischemic heart disease

60. THE PIGMENT DEPOSITION CORRECTLY PAIRED WITH THE APPROPRIATE ABNORMAL CLINICAL MANIFESTATIONS IS
Poisoning - fat
Hemolytic anemia - lipofuscin
Idiopathic hemosiderosis - hematin
+Addison’s disease - melanin
Atrophy of hepatic cells – enterochromophinic pigment

61. HEMOSIDERIN IN LUNG IS ACCUMULATED IN
Leukocytes
Lymphocytes
+Macrophages
Fibroblasts
Erythrocytes

62. THE CHARACTERISTIC FEATURE OF HEMOSIDERIN IS
Tyrosine-derived
Golden yellow-to-green
Amorphous
+Hemoglobin-derived

Aggregate of melanin

63.HEART AND LIVER OF PATIENT WITH CACHEXIA MACROSCOPICALLY ARE SEEN AS
+Diminished and brown
Diminished and yellow
Enlarged and brown
Enlarged and yellow
Unchanged

64. INCREASED AMOUNT OF MELANIN IS KNOWN AS
Vacuolization
Vitiligo
+Melanosis
Albinism
Hypomelanosis

65. THE CHARACTERISTIC FEATURE OF MELANIN IS
Soluble substance
+Tyrosine-derived
Hemoglobin-derived
Golden-yellow
Deposits in macrophages

66. THE CHARACTERISTIC FEATURE OF LIPOFUSCIN IS
Most often seen in kidney
+Aging pigment
Yellow-green
Change cellular function
Tyrosine-derived

67. LIPOFUSCIN PIGMENT ACCUMULATION IN CELLS IS THE RESULT OF
Hemosiderosis
Protein accumulation in cytoplasm
Lipid accumulation in cytoplasm
Cellular swelling
+Intracellular lipid peroxidation

68. THE LUNGS IN PULMONARY HEMOSIDEROSIS ARE CHARACTERIZED BY
Only enlarged in size
+Increased in weight with red-brown areas
Diminished with areas of red-brown consolidation
Diminished with fluid exuding from cut surface
Unchanged

69. JAUNDICE OCCURS IN THE FOLLOWING PATHOLOGICAL PROCESSE
Increased hepatocellular excretion
Reduced production of bilirubin
Increased hepatocyte uptake
+Impaired conjugation of bilirubin
Increased bile flow

70. LOCAL DECREASED AMOUNT OF MELANIN IS KNOWN AS

Vacuolization
+Vitiligo
Hyperpigmentation (melanosis)
Albinism
Hyperkeratosis

71. LIPOFUSCIN GRANULES ACCUMULATION IN CELLS ARE SEEN IN
Necrosis
Denervation
+Brown atrophy
Atrophy from pressure
Apoptosis

72. UNCONJUGATED HYPERBILIRUBINEMIA OCCURS IN
Obstruction of bile duct
Increased hepatocyte uptake
Increased hepatocellular excretion
Increased conjugation
+Hepatocyte injury in hepatitis B

73. EXOGENIOUS PIGMENT IS
Lipofuscin
Hemosiderin
+Carbon
Ferritin
Hematin

74. LIPOFUCSIN IN THE LIVER MAY BE FOUND IN
Unchanged cells
Cells with ballooning degeneration
Cells with hyaline droplets
+Cells with regressive changes
Necrotic cells

75. BILIARY DUCTS' OBSTRUCTION MAY LEAD TO
Liver hemosiderosis
Liver steatosis
Ballooning degeneration of hepatocytes
+Cholestasis and jaundice
Hemochromatosis

76. LOCAL HEMOSIDEROSIS IS SEEN IN
Diabetes mellitus
Hereditary increased absorption of dietary iron
+Focus of hemorrhage
Hemolytic anemia
Impaired uptake of iron

77. THE CHARACTERISTIC FEATURE OF BILIRUBIN IS
Prussian blue with Perls’ reaction
Derived from destroyed lymphocytes
Brown color

+Conjugated and unconjugated form
Tyrosine-derived

78. WIDESPREAD DECREASED AMOUNT OF MELANIN IS KNOWN AS
Vacuolization
Vitiligo
Hyperpigmentation (melanosis)
+Albinism
Hyperkeratosis

79. IRON CONTENTED SUBSTANCE AMONG PIGMENTS IS
Bilirubin
Hematoidin
Lipofuscin
+Hematin
Ceroid

80. MORPHOLOGIC CHANGES IN HEMOCHROMATOSIS ARE CHARACTERIZED BY
Deposition of hemosiderin in the skin
Deposition of lipofuscin in skin
+Widespread hemosiderosis of organs
Hyperbilirubinemia
Deposition of hematoidin

81. TO LIPIDOGENIUS PIGMENTS REFER
Hemosiderin
+Ceroid
Pigment of entherochromophin cells
Hemin
Adrenochrom

82. IRON-CONTAINEDPIGMENT IS
+Ferritin
Hematoidin
Bilirubin
Melanin
Ceroid

83. TYROSINOGENIC PIGMENT IS
Hemosiderin
Ceroid
Pigment of vitamin E deficiency
Hemin
+Adrenochrom

84. SUPRAHEPATIC JAUNDICE DEVELOPS AS RESULT OF
Biliary obstruction
Brown atrophy of the liver
Hepatitis B
Intestinal obstruction
+Hemolytic anemia

85. EXAMPLE OF LOCAL HEMOSIDEROSIS IS
Mitral valve in rheumatic mitral stenosis
Left coronary artery involved in atheromatous plaques
+Brain with pigmented cyst in the place of hemorrhage
Lung in areas of old tuberculosis
Hemolytic jaundice

86. STAIN FOR DETECTION OF HEMOSIDERIN IS
Hematoxyline and eosine
+Perls reaction
Congo-red
Sudan-III
Picrofuschin

87. WIDESPREAD MELANOSIS DEVELOPS AT
Albinism
Melanoma
+Addison's disease
Pigmentary nevus
Glomerulopathy

88. AN EXAMPLE OF METASTATIC CALCIFICATION IS
+The kidney in nephrocalcynosis
The mitral valve in rheumatic mitral stenosis
The coronary artery involved by atheromatous plaques
The Gon's focus in lung
Epidermoid cyst

89. STONES IN KIDNEYS MAY BE
Pigmentary
Cholesterol
+Phosphates
Biliary
Vinery

90. BILLE DUCTS' OBSTRUCTION BY GALLSTONES MAY LEAD TO
Liver hemosiderosis
Liver steatosis
Ballooning degeneration of hepatocytes
+Cholestasis and jaundice
Hemochromatosis

91. METASTATIC CALCIFICATION OCCURS IN
+Parathyroid adenoma
Rheumatic heart disease
Atherosclerosis
Phlebothrombosis
Tuberculosis

92. THE KIND OF CALCIFICATION IS
Necrotic
+Dystrophic

Hypertrophic
Atrophic
Proliferative

93. DISEASE THE CAUSE OF METASTATIC CALCIFICATION IS
+Diabetes mellitus
Increased secretion of parathyroid hormone
Destruction of bone tissue
Vitamin D-related disorders
Renal failure

94. METASTATIC CALCIFICATIONNEVER OCCURS IN
Stomach (gastric mucosa)
Kidneys
Lungs
+Liver
Heart, systemic arteries and pulmonary veins

95. DEFICIENCY OF VITAMIN D TENDS TO CAUSE
Hypercalcemia
+Hypocalcemia
Hyperpigmentation
Hypopigmentation
Calcification

96. FACTOR THAT MAY RESULT IN THE INTRACELLULAR ACCUMULATION OF FAT IS
Decreased amount of fat nutrition
Increased mobilization of accumulated material
+Decreased mobilization of accumulated material
Hypoproduction of accumulated material by cells
Petrification

97. FACTOR THAT MAY RESULT IN THE INTRACELLULAR ACCUMULATION OF FAT IS
Decreased amount of fat nutrition
Fat necrosis
Increased mobilization of accumulated material
Hypoproduction of accumulated material by cells
+Irreversible damage of mitochondria

98. ACCUMULATION OF AMYLOID IN RENAL TISSUE OBSERVES IN
Venues walls
Cytoplasm of tubules epithelium
+Glomerules
Pelvis and calices
Bowmen’s capsule spaces

99. ACCUMULATION OF AMYLOID IN RENAL TISSUE CAN’T BE OBSERVED IN
Venues walls
Cytoplasm of tubules epithelium
Spaces of Bowmen’s capsule
+Basement membrane of tubules epithelium
Calices and pelvis

100. ACCUMULATION OF AMYLOID IN RENAL TISSUE CAN'T BE OBSERVED IN
Venues walls
Cytoplasm of tubules epithelium
Bowmen's capsule spaces
Calices and pelvis
+Stroma

3. HEMODINAMIC DISORDERS

1. "ANASARCA" IS THE NAME OF
Lymphostasis
Local swelling
+General edema
Subcutaneous hemorrhage
Puffiness

2. THE METAPHORIC NAME OF LIVER AT CHRONIC VENOUS CONGESTION IS
"Porphyry"
"Sago"
"Grease"
+"Nutmeg"
"Beckon"

3. MECHANISM OF BLEEDING IS
Diathesis
Plasmorrhagia
Hemorrhage
+Diapedesis
Transformation

4. THE EDEMA IS
Increased volume of blood
Increase in volume of lymph
+Accumulation of interstitial fluid
Accumulation of fluid in abdominal cavity
Accumulation of fluid between testis environments

5. ACCUMULATION OF BLOOD IN THE PLEURAL CAVITY IS
Hematoma
Hematuria
Hematemesis
+Hemathorax
Hemangioma

6. IN NEPHROCYTES AT ACUTE VENOUS CONGESTION DEVELOPS
+Dystrophy and necrosis
Hyalinosis and sclerosis
Sclerosis
Amyloidosis
Hemosiderosis

7. COLOR OF LUNG AT CHRONIC VENOUS CONGESTION DETERMINATES BY PRESENCE OF PIGMENT
Hematin
Bilirubin
+Hemosiderin
Hematoidin
Lipofuscin

8. THE NAME OF EDEMATIC FLUID ACCUMULATION IN ABDOMINAL CAVITY IS
+Ascites
Anasarca
Hydrocele
Hydrothorax
Hydronephrosis

9. CHRONIC VENOUS CONGESTION IN SPLEEN LEADS TO
Brown atrophy
Brown induration
+Cyanotic induration
Hemochromatosis
Amyloidosis

10. THE NAME OF FLUID ACCUMULATING IN CAVITIES DUE TO CONGESTION IS
Lymphorrhea
Exudate
+Transudate
Hydrops
Anasarca

11. THE NAME OF CHRONIC VENOUS CONGESTION IN KIDNEY IS
Brown atrophy
Hydronephrosis
Brown induration
+Cyanotic induration
Nephrochirrosis

12. THE STOMACH BLEEDING AS COMPLICATION IS CHARACTERISTIC FOR
Gastritis
+Stomach ulcer
Tesaurismoses
Colitis
Pancreatitis

13. MICROSCOPIC CHARACTERISTIC OF ACUTE PULMONARY CONGESTION IS
Thickened and fibrotic alveolar septa
+Edema fluid in alveoli
Hemosiderosis and fibrosis in alveolar septa
Pneumosclerosis
Carnification

14. MICROSCOPIC CHARACTERISTIC OF CHRONIC PULMONARY CONGESTION IS
Abscesses in lung tissue

Edema in alveolar septa
Edema fluid in alveoli
Carnification
+Hemosiderosis and fibrosis in alveolar septa

15. CHRONIC VENOUS CONGESTION IN LUNG RESULTS IN DEVELOPING
Bleeding
Lipofuscinosis
+Brown induration
Mucoid swelling
Melanosis

16. STASIS DEVELOPMENT IS CHARACTERIZED BY
Loss of fibrin
Damage of vascular wall
+Agglutination of erythrocytes
Leucodiapedesis
Pinocytosis

17. MICROSCOPIC CHARACTERISTIC OF CHRONIC PASSIVE CONGESTION IN LIVERIS
Periphery necrosis of hepatocytes
Capillarisation of sinusoids
Sclerosis of periportal sinusoids
+Centrolobular hemorrhages
Fatty embolism of hepatic arteries

18. ORGANS AT CHRONIC VENOUS CONGESTION
Are reduced in sizes
Have a flabby consistence
+Have a dense consistence
Red color
White color

19. PRINCIPAL CAUSE OF VENOUS HYPEREMIA IS
Reduction of blood inflow
Increase of blood inflow
+Blockade of blood outflow
Increase of blood outflow
Anemia

20. HEMORRHAGE ENCLOSED WITHIN A TISSUE CAUSING ITS DESTRUCTION IS TERMED AS
+Hematoma
Petechia
Purpura
Ecchymosis
Hemothorax

21. MINUTE (1- TO 2-MM) HEMORRHAGES INTO SKIN, MUCOUS MEMBRANES OR SEROSAL SURFACES ARE CALLED
Hematomas
+Petechiae
Purpura

Ecchymoses
Hemothorax

22. MACROSCOPICCHARACTERISTIC OF CHRONIC PASSIVE CONGESTION IN LIVER IS
Red color of liver
Decreasing of size
+Nutmeg liver
White color
Goose liver

23. THE FIGURATIVE NAME OF LIVER AT CHRONIC VENOUS CONGESTION IS
Grease
Sago
Brown
+Nutmeg
Icing

24. GENERAL VENOUS CONGESTION DEVELOPS AT
Compression of superior cava vein
Thrombosis of portal vein
Compression of renal vein by tumor
Polycythemia
+Heart diseases

25. «NUTMEG LIVER» CAN BE CAUSED BY
Brain hyperemia
Spleen hyperemia
Cyanotic induration of kidneys
+Mitral valve stenosis
Acute coronary insufficiency

26. DISSEMINATION OF TUMOR (METASTASIS) IS THE TYPE OF EMBOLISM
Fatty
Bacterial
+Cellular
Thrombus
Purulent

27. ONE OF THE MAIN CONDITION OF THROMBOSIS IS
Change of blood oxigination
+Damage of vascular endothelium
Hereditary thrombastenia
Change of blood flow direction
Emigration of leucocytes

28. THE SYNONYM OF THE DIC-SYNDROME
Hemorrhagic diathesis
+Coagulopathy of consumption
Thromboembolic syndrome
Thrombocytopenia syndrome
Antiphospholipid syndrome

29. ONE OF THE BASIC TYPES OF SHOCK IS
Croupous
Lymphatic
Myogenic
+Cardiogenic
Dystrophic

30. SYNDROME FREQUENTLY ASSOCIATED WITH SHOCK IS
Pulmono-coronary
Nephrotic
Thrombohemorrhagic
Thromboembolic
+DIC

31. ONE OF THE BASIC TYPES OF SHOCK IS
Aseptic
Lymphogenic
+Anaphylactic
Hormonal
Thrombolytic

32. THROMBUS IN HEART CHAMBERS MAY BE
+Ball-Like
Occlusive
Circular
Parietal
Progressive

33. HEMORRHAGE ENCLOSED WITHIN A TISSUE CAUSING ITS DESTRUCTION IS TERMED AS
Hemothorax
Petechia
Purpura
Ecchymosis
+Hematoma

34. ONE OF THE BASIC TYPES OF SHOCK IS
Aseptic
+Traumatic
Myogenic
Hormonal
Thrombolytic

35. AT DIC-SYNDROME IN BRAIN MAY BE REVEALED
Necrosis of epithelium
Centers of caseous necrosis
Inflammation
Serous-hemorrhagic exudates
+Microthrombi

36. THE BODY OF THE MIXED THROMBUS ACCORDING TO STRUCTURE AND APPEARANCE IS
White

Red
+Mixed
Hyaloid
Fibrinous

37. ONE OF THE BASIC TYPES OF SHOCK IS
Hormonal
Anaplastic
Lymphogenic
+Septic
Thrombolytic

38. THE HEAD OF THE MIXED THROMBUS ACCORDING TO STRUCTURE AND APPEARANCE IS
+White
Red
Mixed
Hyaloid
Fibrinous

39. SEPTIC SHOCK IS USUALLY CAUSED BY
Viruses
Parasites
Foreign bodies
+Bacteriemia
Micoplasma

40. ONE OF THE STAGES OF DIC-SYNDROME DEVELOPMENT IS
Aggregation of cells
Inflammation
+Coagulopathy of consumption
Activation of coagulation
Reconvalescention

41. TAIL OF THE MIXED THROMBUS ACCORDING TO STRUCTURE AND APPEARANCE IS
White
+Red
Mixed
Hyaloid
Fibrinous

42. INCREASED COAGULABILITY OF BLOOD IS OBSERVED AT
Nephrotic syndrome
Willebrandt disease
Thrombocytopenia purpura
+Last term of pregnancy
Disseminative cancеromatous

43. THROMBUS IS DIFFERED FROM BLOOD CLOT BY
Postmortem appearance
+Attachment to vessel wall
Smooth surface

Soft consistence
Amorphous structure

44. THE RESULT OF MASSIVE FATTY EMBOLISM IS ACUTE INSUFFICIENCY OF
Kidneys
Polyorganic
+Lungs
Heart and vessels
Liver

45. BY DIC-SYNDROME CAN COMPLICATE
Essential hypertension
+Obstetrics sepsis
Gout
Fatty dystrophy of liver
Adiposity

46. FAVORABLE OUTCOME OF THROMBOSIS IS
+Organization
Putrefaction
Destruction
Septic lyses
Retraction

47. THROMBI FORMED AT DIC-SYNDROME MOSTLY ARE
White
Mixed
Red
+Hyaloid
Fibrinous

48. AT FATTY PULMONARY EMBOLISM FATTY DROPS ARE FOUND OUT IN
Veins
Alveolar spaces
Segmental bronchus
+Capillaries of alveolar septum
Pulmonary trunk

49. THE SECOND STAGE OF THE SHOCK IS
Non- progressive
+Progressive
Irreversible
Reversible
Clinical

50. THE REASON OF FATTY EMBOLISM IS
+Fracture of long tubular bones
Fatty dystrophy of myocardium and liver
Subcutaneous injections of oil
Intramuscular injections of oil
Proliferation of bone marrow

51. THE STANE USED FOR DIAGNOSIS OF FATTY PULMONARY EMBOLISM IS
Perls' reaction
+Sudan III
Picrofuchsin by van Giesone
Tolluidin blue
Congo red

52. THE FIRST STAGE OF THE SHOCK IS
+Non- progressive
Progressive
Irreversible
Reversible
Preclinical

53. UNFAVOURABLE OUTCOME OF THROMBOSIS IS
Organization
+Detoughchment of thrombus
Recanalization
Vascularization
Aseptic resolution

54. IN LUNGS AT DIC-SYNDROME MAY BE REVEALED
Necrosis of epithelium
Centers of caseous necrosis
Ischemia and focal necrosis
Serous-hemorrhagic exudates
+Microthrombi in capillary vessels

55. STAGE OF THROMBI MORPHOGENESIS IS
Coagulopathy of consumption
Polymerization of ferretin
Agglutination of plasma
Precipitation blood cells
+Coagulation of fibrinogen

56. WHITE THROMBI ARE FORMED IN
Veins
+Arteries
Cavities aneurysms
Microcirculatory vessels
Heart cavities

57. RESTORATION OF BLOOD FLOW IN VESSEL AFTER THROMBOSIS IS
Reparation
Petrifaction
Occlusion
Resolution
+Recanalization

58. THE PULMONOCORONARY REFLEX INCLUDES SPASM OF
+Pulmonary veins, Bronchial tree, Coronary arteries of heart
Carotids, Branches of pulmonary arteries

Pulmonary veins
Carotids, Branches of pulmonary arteries, Coronary arteries of heart
Aorta, Trachea, Coronary arteries

59. RED THROMBI ARE FORMED IN
+Veins
Arteries
Cavities aneurysms
Microcirculatory vessels
Heart cavities

60. THE MIXED THROMBI ARE FORMED IN
Veins
Arteries
+Cavities aneurysms
Microcirculatory vessels
Lymph vessels

61. OCCLUSIVE THROMBI IN ARTERIES CAN LEAD TO DEVELOPMENT OF
Lymphostasis
+Infarction and gangrene
Thromboembolism of pulmonary arteries
Dystrophies of parenchymal organs
Thrombophlebitis

62. AIR EMBOLISM CAN DEVELOP DUE TO
+Childbirths and abortions
Wounds of abdominal cavity
Fracture of bones
Gangrene of lung
Tamponade of heart

63. MIXED THROMBUS CONSISTS OF
Fibrin, hyaline
Thrombocytes, fibrin, some erythrocytes
Thrombocytes, fibrin, leukocytes
+Thrombocytes, fibrin, leukocytes, erythrocytes
Thrombocytes, fibrin, leukocytes, erythrocytes, hyaline

64. LOW EXTRIMITIES PHLEBOTHROMBOSIS CAN LEAD TO DEVELOPMENT OF
Neoplasia
Infarctions and gangrene
+Massive pulmonary thromboembolism
Dystrophies of parenchymal organs
Rupture of heart

65. REFLEX CONNECTED WITH THANATOGENESIS OF MASSIVE PULMONARY TROMBOEMBOLISM IS
Somatic
+Pulmono-coronary
Viscera-visceral
Adaptive

Basilar

66. PULMONARY THROMBOEMBOLUS MAY ORIGINATE FROM
+Deep leg veins
Superior vena cava
Pelvic arteries
Portal vein
Microcirculation net

67. EMBOLISM CAN BE
Hemolytic
Septic
Mechanical
Parenchimal
+Fatty

68. OPTIMUM OUTCOME OF THROMBOSIS IS
Thrombus taking off
Septic lyses
+Aseptic lyses
Organization
Petrifaction

69. THE POSSIBLE COMPONENT OF EMBOLI IS
Amyloidal masses
Fibrinoid masses
Emphysema bubbles
+Air bubbles
Apoptotic bodies

70. FATTY EMBOLISM DIAGNOSIS IS VERIFICATED BY
Macroscopically
Endoscopically
+Microscopically
Visually
Ultramicroscopically

71. THE GREATEST VALUE AT FATTY EMBOLISM HAS CAPILLARIES OBTURATION OF
Kidneys and liver
+Lung and brain
Liver and spleen
Heart
Brain and bone marrow

72. MECHANISM OF BLEEDING IS
Diathesis
Plasmorrhagia
Hematoma
+Erosion of wall
Hematemasis

73. MECHANISM OF BLEEDING IS

Diathesis
Plasmorrhagia
Hemorrhage
+Rupture of wall
Hematemasis

74. MICROSCOPIC CHARACTERISTIC OF CHRONIC PASSIVE CONGESTION IN LIVER IS
Periphery necrosis of hepatocytes
Capilarisation of sinusoids
+Engorged periportal sinusoids
Centrolobular edema
Cholesterolosis of periportal hepatocytes

75. MICROSCOPIC CHARACTERISTIC OF CHRONIC PASSIVE CONGESTION IN LIVER IS
Periphery necrosis of hepatocytes
Capillarisation of sinusoids
Suppuration of periportal sinusoids
Centrilobular edema
+Fatty dystrophy of periportal hepatocytes

4. CELL DEATH

1. DIGESTION OF CELL BY IMMIGRANT LEUKOCYTES IS TERMED AS
Apoptosis
Autolysis
+Heterolysis
Inflammation
Metaplasia

2. CASEOUS NECROSIS IS CHARACTERISTIC OF
Gas gangrene
Frostbite
Myocardial infarction
+Tuberculosis
Typhoid fever

3. THE VARIANT OF NECROSIS CAN BE FOUND IN TUBERCULOSIS IS
+Caseous
Gangrenous
Liquefactive
Enzymatic fat
Fibrinoid

4. CASEOUS NECROSIS MEETS AT
Diphtheria
Gas gangrene
+Tuberculosis
Infarction of kidney
Myocardial infarction

5. TYPE OF INFARCTION DEPENDING ON MECANISM OF NECROSIS IS
Correct

+Ischemic
Inflammatory
Toxic
Allergic

6. POSSIBLE OUTCOME OF LIQUEFACTIVE NECROSIS CAN BE
Petrifaction
Ossification
+Cyst-formation
Scarring
Mummification

7. VARIANT OF ISCHEMIC NECROSIS CAN BE FOUND IN BRAIN IS
Coagulative
+Liquefactive
Caseous
Gangrenous
Enzymatic fat

8. THE FEATURE DISTINGUISH APOPTOSIS FROM NECROSIS IS
Presence of inflammation nearby the injured cells
Fragmentation of tissue
Formation of necrotic bodies
Affection of large areas of cells
+Programmed cell death

9. THE CAUSES OF INFARCTION INCLUDE
Hemophilia
Thrombocytopenia
Arterial dilation
Local hyperemia
+ Embolic events

10. WHITE INFARCT RESULTS FROM
Venous occlusion
+Arterial occlusion
Suppuration of tissues
Tissues with dual circulation
Congested tissues

11. UNFAVORABLE OUTCOME OF NECROSIS IS
Organization
Encapsulation
Petrifaction
+Purulent putrefaction
Cyst formation

12. MORPHOLOGICAL CHANGES OF APOPTOSIS INCLUDE
+Membrane blebs
Inflammation
Tissue fragmentation
Cell swelling

Cell proliferation

13. STARTING POINT OF APOPTOSIS FOR PROGRAMM CELL DEATH IS
+Activation of endonuclease
Enzymopathy
Accumulation of calcium
Destruction by macrophages
Cell swelling

14. ONE OF THE FOLLOWING IS AN APOPTOSIS INHIBITOR GENE
p53
+Bcl-2
Rb
C-Myc
K-67

15. THE EXAMPLE OF PHISIOLOGIC APOPTOSIS IS
Councilman bodies in liver
Russell bodies
Atrophy of organ following duct obstruction
Tumor necrosis
+Ageing of organism

16. COMMONLY SUDDEN OCCLUSION OF BLOOD SUPPLY RESULTS IN
+Coagulative necrosis
Caseous necrosis
Liquefactive necrosis
Sequester
Fatty necrosis

17. CLOUDY SWELLING IS DUE TO
+Accumulation of water intracellularly
Fat accumulation intracellular
Lysozyme degeneration
Glycogen accumulation intracellularly
Ca accumulation

18. COMMONLY LIQUAFACTIVE NECROSIS OCCURS IN
+Brain
Heart
Liver
Kidney
Spleen

19. FAT NECROSIS IS COMMON IN
Brain
Kidney
Skeleton muscles
+Breast
Heart

20. THE CELLS MOST SENSITIVE TO ISCHEMIA ARE

Skeleton muscles
Glial cells of brain
Renal tubular epithelium
+Cortical neurons
Adipose tissue

21. CELLS MOST SENSITIVE TO HYPOXIA ARE
Myocardial cells
+Neurones
Hepatocytes
Renal tubular epithelial cells
Squamous epithelium

22. ONLY RED INFARCTION IS SEEN IN
Kidney
Spleen
+Small intestine
Heart
Brain

23. WHITE INFARCTION IS SEEN IN
+Kidney
Lung
Brain
Small intestine
Large intestine

24. THE FIRST EFFECT OF ENDOTOXIN IS
+Endothelial damage
Perivascular necrosis
DIC
Shock
Thrombosis

25. MICROSCOPIC FEATURES OF NUCLEAR NECROSIS ARE
Karyopicnosis, karyorrhexis, plasmolysis
+Karyopicnosis, karyorrhexis, karyolysis
Plasmorrhexis, plasmolysis, karyorrhexis
Karyopicnosis, plasmorrhexis, karyorlysis
Karyopicnosis, karyorrhexis, plasmorrhexis

26. ON VISCERAL SURFACE OF SEROUS MEMBRANE IN PLACE OF NECROSIS DEVELOPS
Edema
Hyalinosis
Gangrene
Catarrhal inflammation
+Fibrinous inflammation

27. FAVOURABLE OUTCOMES OF NECROSIS ARE
Mucination, liquefaction
Mummification, desquamation

Suppuration, putrefaction
Progression, inflammation
+Organisation, reparation

28. MACROSCOPIC CHARACTERISTIC OF SPLEEN INFARCTION IS
+Triangular, whitish-grey, dense
Triangular, whitish-grey, soft
Irregular, whitish-grey, dense
Irregular, yellow, soft
Triangular, yellow, dense

29. ISCHEMIA IN MYOCARDIOCYTES LEADS TO DISAPPEARENCE OF
Lipid droplets
Glucose granules
+Glycogen granules
Lipofuscine granules
Cholesteryne crystals

30. THE REASON OF INFARCTION IS
Thrombocytopenia
Dysbolism
Functional intention of organ in sports
Insufficiency of gastric anastomoses
+Thrombosis

31. TYPE OF NECROSIS DEPENDING ON REASON
Atrophic
Macrofocal
Compensatory
Inflammatory
+Vascular

32. THE MOST TYPICAL REASON OF ISCHAMIC BRAIN INFARCTION IS
Thromboembolism
+Thrombosis
Long-duration spasm
Functional intention of organ
Insufficiency of gastric anastomoses

33. MICROSCOPIC FEATURES OF NECROSIS IN CYTOPLASMA ARE
Proteins production
Concentration of plasma proteins
Activation of lysosomes
Centralization of cytoplasm
+Plasmorrhexis, plasmolysis

34. BLACK COLOR OF TISSUE AT GANGRENE DETERMINATES
Hematin deposition
Presence of magnesium
+Presence of iron sulfatis
Calcification
Presence of melanin

35. THE SHAPE OF MYOCARDIAL INFARCTION FOCUS IS
Round
Oval
Triangular
+Irregular
Quadrate

36. THE REASON OF RED INFARCTION DEVELOPMENT IS
Thromboembolism of kidney artery
Low extremity vein thrombosis
Spleen artery embolism
Spasm of coronary artery
+Thrombosis of upper mesenteries artery

37. THE STAGE OF NECROSIS LIKE PROCESS IS
Thrombolytic
Nutritional
Petrification
Cell depletion
+Necrobiosis

38. CELLS THAT ARE PHAGOCYTING APOPTOTIC BODIES
Ring-cells
Erythrocytes
Epitheliocytes
+Macrophages
Amyloidoblasts

39. SEQUESTER IS
Organizated thrombus
+Unresolved part of dead tissue
Vascular necrosis
Necrosis of tissue connected with environment
Suppuration of tissue

40. NOMA IS
Organizated thrombus
Unresolved part of dead tissue
Vascular necrosis
Necrosis of tissue connected with environment
+Wet gangrene of soft tissues

41. GANGRENE IS
Organizated thrombus
Unresolved part of dead tissue
Vascular necrosis
+Necrosis of tissue connected with environment
Suppuration of tissue

42. INFARCTION IS
Organizated thrombus

Unresolved part of dead tissue
+Vascular necrosis
Necrosis of tissue connected with environment
Suppuration of tissue

43. NECROSIS IS
Program cell death
+Death of cells in living organism
Stopping of functions in living organism
Reversibly cell injury
Decreasing of organ in living organism

44. APOPTOSIS IS
+Program cell death
Death of cells in living organism
Stopping of functions in living organism
Reversibly cell injury
Decreasing of organ in living organism

45. THE TYPE OF CELL DEATH IS
Fragmentation
Hydrolysis
+Apoptosis
Mummification
Swelling

46. THE TYPE OF CELL DEATH IS
Fragmentation
Hydrolysis
+Necrosis
Mummification
Swelling

47. MACROSCOPIC CHARACTERISTIC OF BRAIN INFARCTION IS
+Soft consistence
Triangular shape
Basis turned to cortex
Red areole
Green color

48. THE TYPE OF GANGRENE
Soft
Flat
Gas
+Bedsore
Red

49. THE MAIN MORPHOLOGIC FEATURE OF APOPTOSIS IS
Polarization of chromatin
Accumulation of fat
Reproduction of cell
Accumulation of proteins

+Condensation of nuclear chromatin

50. THE SYNONIM OF WHITE INFARCTION IS
Coagulative
Colliquattive
Hemorrhagic
+Ischemic
Gangrene

51. THE CHARACTERISTIC FEATURE OF APOPTOSIS DISTINGUISH IT FROM NECROSIS IS
Presence of inflammation nearby the injured cells
+Fragmentation of nuclear chromatin only
Formation tissue detritus
Affection of large areas of cells
Nonprogrammed cell death

52. THE FEATURE DISTINGUISH APOPTOSIS FROM NECROSIS IS
Presence of inflammation nearby the injured cells
Fragmentation of tissue
+Formation of apoptotic bodies
Affection of large areas of cells
Nonprogrammed cell death

53. THE CAUSES OF INFARCTION INCLUDE ARE
Hemophilia
Local inflammation
Arterial dilation
+Local vasospasm
Thrombocytopenia

54. THE CAUSES OF INFARCTION INCLUDE ARE
Hemophilia
Cholestasis events
+Arterial occlusion
Local inflammation
Thrombocytopenia

55. ONLY RED INFARCTION IS SEEN IN
Kidney
Spleen
+Lung
Heart
Brain

56. WAXY NECROSIS IS DUE TO
Accumulation of water intracellularly
+Fat accumulation intracellularly
Lysenzyme degeneration
Glycogen accumulation intracellularly
Ca accumulation

57. ONLY WHITE INFARCTION IS SEEN IN

Liver
+Spleen
Small intestine
Lung
Brain

58. COMMONLY SEQUESTER OCCURS IN
+Bones
Heart
Liver
Kidney
Spleen

59. WAX NECROSIS IS COMMON IN
Brain
Kidney
+Skeleton muscles
Breast
Heart

60. THE CELLS MOST SENSITIVE TO ISCHEMIA ARE
Skeleton muscles
Glial cells of brain
Renal tubular epithelium
+ Myocardial cells
Adipose tissue

5. INFLAMMATION & IMMUNE SYSTEM PATHOLOGY

1. FLUID THAT COLLECTS DURING ACUTE INFLAMMATION AND THAT HAS PROTEIN CONTENT EXCEEDING 3 G/DL AND SPECIFIC GRAVITY EXCEEDING 1.015 IS
Edema
Effusion
Transudates
Serum
+Exudates

2. PHASE OF ALTERATION AT INFLAMMATION IS CHARACTERIZED BY
Suppuration
Proliferation
Phagocytosis
+Tissue distraction
Reparation

3. PHASE OF PROLIFERATION AT INFLAMMATION IS CHARACTERIZED BY
Damage of tissue
Disorder of blood circulation
+Replication of cells
Phagocytosis
Exudates formation

4. POSITIVE OUTCOME OF INFLAMMATION IS
Incomplete regeneration
+Elimination of microbes and toxins
Sclerosis and strictures formation
Reaction of hypersensitivity to toxins and medicines
Massive replacement by connective tissue

5. INFLAMMATION LOCALIZATED ON SEROUS MEMBRANE IS
Hemorrhagic
Proliferative
Putrefactive
+Fibrinous
Mucinous

6.DIFFERENCE BETWEEN ACUTE AND CHRONIC ABSCESS IS
Shape
Size
Contents
+Structure of walls
Localization

7. NEGATIVE OUTCOME OF INFLAMMATION IS
Elimination of necrotizated cells
Complete regeneration
+Scarring and substitution
Restoration of tissue capasity
Restitution

8. CROUPOUS INFLAMMATION DEVELOPS ON THE MEMBRANES COVERED BY
Mesothelium
+Cylindrical epithelium
Skin
Squamous keratinizing epithelium
Squamous non-keratinizing epithelium

9. INFLAMMATION IS
Hyperplasia of cellular organells
Restoration of the lost structures
Growth of cellular elements
+Exudative-proliferative reaction of tissue to damage
Cellular infiltration in stroma of organs

10. PURULENT INFLAMMATION IN PLEURAL CAVITY IS TERMED AS
Abscess
Apostema
Furuncle
+Empyema
Carbuncle

11. PHASE OF EXUDATION IS BASED ON
Alteration
Proliferation of cells

Change of a blood-flow
+Emigration of cells and phagocytosis
Formation of an inflammatory edema

12. REACTION DEVELOPS IN ORGANISM AS A RESPONSE TO DAMAGE OF TISSUE IS
Amyloidosis
+Inflammation
Regeneration
Phagocytosis
Hyperplasia of cells

13. CHARACTERISTIC OF THE DIPHTHERITIC INFLAMMATION ON TONSILS IS
Pus
Slime (mucus)
Cells elements proliferation
+Fibrinous pellicle
Hematoma

14. STARTING MECHANISM OF INFLAMMATORY REACTION IS
Exudation
+Mediation
Emigration
Phagocytosis
Regeneration

15. SEROSAL INFLAMMATION LOCALIZATED ON SEROUS MEMBRANES IS
Visceral
Lymphoid
Mucoid
+Catarrhal
Fibrinoid

16. CHARACTERISTIC OF LEUCODIAPEDESIS IS
Phagocytosis
Formation of granuloma
Emigration of plasma proteins
Edema
+Intraendotelial migration of leukocytes

17. TYPE OF INFLAMMATION (DEPENDING ON DURATION) IS
+Acute
Simple
Recurrent
Repeated
Complicated

18. THE TYPE OF INFLAMMATION WHICH RESULTS ININCOMPLETE RESTORATION OF ORGANS' STRUCTURE IS
Serous
Catarrhal
Croupous
Diphtheritic

+Proliferative

19. TYPE OF EXUDATIVE INFLAMMATION (DEPENDING ON EXUDATE) IS
Acute
+Purulent
Chronic
Granulematous
Interstitial

20. THE FIGURATIVE NAME OFHEART WITH FIBRINOUS PERICARDITIS IS
"Bull"
"Tiger"
"Goose"
"Glassy"
+"Hairy"

21. FOR CATARRHAL INFLAMMATION IS TYPICAL
Lyses of pellicle
+Presence of mucus in exudates
Proliferation of cell elements
Formation of pellicle on mucous membrane
Putrefaction

22. THE PHASE OF INFLAMMATORY REACTION IS
Pinocytosis
+Proliferation
Elimination
Phagocytosis
Leucodiapedesis

23. TYPICAL OUTCOME OF ACUTE INFLAMMATION IS
Development of malignant tumor
+Complete regeneration
Formation of chronic abscess
Development of cirrhosis
Progressing in various forms of chronic inflammation

24. INFLAMMATION OF SMALL INTESTIN IS TERMED AS
Intestinitis
Colitis
Gastritis
+Enteritis
Proctitis

25. THE TYPE OF PURULENT INFLAMMATION IS
Croupous
+Phlegmonous
Catarrhal
Granulomatous
Diphtheritic

26. ACCUMULATING IN CAVETIES FLUIDAT ACUTE INFLAMMATION IS

Edema
Effusion
Transudate
Serum
+Exudates

27. TYPE OF FIBRINOUS INFLAMMATION IS
Interstitial
+Diphtheric
Catarrhal
Phlegmonous
Granulomatous

28. THE INFLAMMATORY RESPONSE LEADS TO
Dysregeneration
Inactivation of macrophages
Neutralization of leucocytes
Removing of vital tissue
+Isolation of infective tissue

29. PROLIFERATIV PHASE OF INFLAMMATIONIS CHARACTERIZED BY
Damage of tissue
Infringement of blood circulation
+Reproduction of cells
Phagocytosis
Exudates formation

30. LOCALIZATION OF FIBRINOUS INFLAMMATION
+Pericardium
Kidney tissue
Tissue of brain
Skin
Muscle tissue

31. THE FORM OF THE PURULENT INFLAMMATION IS
Catarrh
Granuloma
Vesicle
+Abscess
Emphysema

32. TYPE OF INFLAMMATION AT CROUPOUS PNEUMONIA IS
Purulent
+Fibrinous
Serous
Catarrhal
Granulomatous

33. THE ADHESION OF LEUCOCYTES TO VESSEL WALL BEFORE EMIGRATION IS
+Margination
Diapedesis
Clotting

Congestion
Proliferation

34. THE CHARACTERISTIC OF FIBRINOUS PERICARDITIS IS
Diphtheric type of inflammation
+Develops at uremia
Figuratively refers to «Tiger heart»
Accompanied by conjunctivitis
Leads to myocardial infarction

35. MORPHOLOGICAL MANIFESTATION OF ALTERATION
+Necrosis
Proliferation
Atrophy
Lipofuscinosis
Apoptosis

36. THE MOST TYPICAL OUTCOME OF PRODUCTIVE INFLAMMATION IS
+Sclerosis
Suppuration
Petrification
Ulceration
Metaplasia

37. PURULENT INFLAMMATION IS CHARACTERIZED BY
+Neutrophiles infiltration with tissue lysis
Fibrin deposition
Mucus production
Lymphocyte infiltration
Erythrocyte infiltration

38. TYPE OF INFLAMMATION IS FOUND IN LOBAR PNEUMONIA
Purulent
+Fibrinous
Serous
Catarrhal
Granulomatous

39. SUBTYPE OF PRODUCTIVE INFLAMMATION IS
Serous
Catarrhal
Croupous
+Granulomatous
Diphtheric

40. A LARGE AGGREGATE OF EPITHELIOID CELLS IS SEEN IN
Granulation tissue
Pyogenic granuloma
Granulosa cell tumor
Granulocytosis
+Granuloma

41. GRANULOMA IS:
Focal accumulation of leukocytes
Focal accumulation of slime (mucus)
Focal accumulation of fibrin
+Focal productive inflammatory reaction
Focal hemorrhagic infiltration

42. THE GRANULOMATOUS INFILTRATE IN TERTIARY SYPHILIS IS COMPOSED PREDOMINANTLY OF
Neutrophiles
Monocytes/macrophages
+Plasma cells
Eosinophiles
Lymphocytes

43. SUBTYPE OF INFLAMMATION AT MILIARY LUNG TUBERCULOSIS IS
+Granulomatous
Serous
Fibrinous
Suppurative
Hemorrhagic

44. TYPE OF NECROSIS CAN BE FOUND IN TUBERCULOUS GRANULOMA IS
Coagulative
Liquefactive
+Caseous
Enzymatic fat
Fibrinoid

45. SUBTYPE OF FIBRINOUS INFLAMMATION DEVELOPED ON MUCOUS MEMBRANE OF ORAL CAVITY IS
Phlegmonous
Interstitial
Hemorrhagic
Putrefactive
+Diphtheric

46. THE CELLS ARE INVOLVED IN IMMUNE RESPONSE
+Macrophages
Erythrocytes
Adiposocytes
Thrombocytes
Amyloidoblasts

47. ACCUMULATION OF EPITHELIOID CELLS IN OVARY TISSUE IS CHARACTERISTIC OF
Granulation tissue
Abscess
Granulosa cell tumor
Granulocytosis
+Granulomatous inflammation

48. SUBTYPE OF INFLAMMATION AT LEPROSYIN PARENCHIMAI ORGANS IS

+Granulomatous
Serous
Fibrinous
Suppurative
Hemorrhagic

49. THE MOST IMPORTANT ROLE IN CHRONIC TUBERCULOUS INFLAMMATION PLAYS
+Macrophages
Leukocytes
Eosinophiles
Erythrocytes
Plasma cells

50. TYPICAL SYPHILITIC GRANULOMA IS CHARACTERIZED BY
Platelet infiltrate
Hemorrhagic infiltrate
Lymphocyte absence
Suppuration
+Area of central gummous necrosis

51. FOCUS OF NECROSIS SURROUNDED BYEPITHELIOID CELLS, LYMPHOCYTES AND MACROPHAGES IS TERMED AS:
Papilloma
Condyloma
Melanoma
Lipoma
+Granuloma

52. GRANULOMATOUS INFLAMMATION DEVELOPS IN
Bronchial asthma
Lymphogranulomatosis
Sepsis
Flu
+Tuberculosis

53. TYPICAL TUBERCULOUS GRANULOMA IS CHARACTERIZED BY
+Langhans' giant cells
Leucocytes
Central suppuration
Epithelial cells
Neutraphiles

54. DIAGNOSTIC (GIANT) LEPROSY CEIIS ARE KNOWN AS:
Foam cells
+Virchov’s cells
Epitheliod cells
Langhans' giant cells
Schwann cells

55. PATHOGENESIS OF “HASHIMOTO’S THYROIDITIS” IS ASSOCIATED WITH
Alloantibody

Alloantigens
+Autoantigens
Heteroantibody
Heteroantigens

56. MACROPHAGES IN GRANULOMATOUS INFLAMMATION MAY TRANSFORM INTO
Monocytes
Epithelial cells
+Epithelioid cells
Plasma cells
Lymphocytes

57. HYDATID CYST OF THE LIVER IS AN EXAMPLE OFINFLAMMATIONTYPE
Purulent
Fibrinous
Serous
Catarrhal
+Productive

58. THE MOST COMMON CAUSE OF HIVES (ACUTE ALLERGIC RHINITHIS) IS
+Local anaphylaxis
Immune complex injury
Immunologic tolerance
Genetic immune system deficiency
Genetic deficiency of the complement system

59. DISORDERS OF THE IMMUNE SYSTEMINCLUDE
+Hypersensitivity reactions
Hyalinosis
Heart diseases
Tumors
Fibrinoid degeneration

60. CELLULAR INFILTRATE IN INTERSTICIAL INFLAMMATION IS TERMED AS
Granuloma
Shankar
+Nodulus
Condyloma
Polyp

61. DISEASE RESULTSFROM TYPE III HYPERSENSITIVITY
Myocardial infarction
Pneumonia
+Glomerulonephritis
Posthemorrhagic anemia
Systemic amyloidosis

62. TYPE I HYPERSENSITIVITY (ANAPHYLACTIC TYPE) IS CHARACTERIZED BY
Develops slowly (within days)
Occurs two weeks after
Result of bacteria effect
+Develops rapidly (within minutes)

Chronic current

63. CELLS FORMING GRANULOMA IN TYPE IV HYPERSENSITIVITY REACTION ARE
Neutrophiles
+Macrophages
Erythrocytes
Epithelial cells
Amyloidoblasts

64. DISORDERS OF THE IMMUNE SYSTEM INCLUDE
Sensitive reactions
Hyalinosis
Amyloidosis
+Autoimmune diseases
Ischemic heart disease

65. IMMUNOPATHOLOGIC PROCESS IS
Hyperplasia
Hyperkeratosis
+Hypersensitivity of delayed type
Hypertrophy
Hypercoagulation

66. THE MOST TYPICAL PATHOLOGY OF AIDS PULMONARY SYNDROME IS
Cachexia
Hepatitis
Lymphoadenopathy
+Pneumocystic pneumonia
Enterocolitis

67. TYPICAL OPPORTUNISTIC TUMOR AT CLINICAL PICTURE OF HIV- INFECTION IS
Synovial sarcoma
Osteosarcoma
Jung's sarcoma
+Caposhy's sarcoma
Leiomyosarcoma

68. IN IMMUNE ORGANS AT AIDS OBSERVES
Hyperplasia
Necrosis
+Atrophy
Sclerosis
Plethora

69. THE SUBTYPE OF PRODUCTIVE INFLAMMATION IS
+Interstitial
Serous
Fibrinous
Purulent
Proliferative

70. THE REASON OF CHRONIC INFLAMMATION IS

Acute infection
Facultative infection
+Persisted infection
Respiratory virus
Infarction

71. CHRONIC INFLAMMATION IS CHARACTERIZED BY
+Polyps formation
Deposition of amyloid
Neutraphil infiltration
Fibrinous exudates accumulation
Extravasations of blood.
Complete recovery

72. THE CHARACTERISTIC OUTCOME OF INTERSTITIAL INFLAMMATION IS
Edema
+Sclerosis
Necrosis
Petrification
Putrefaction

73. THE OUTCOME OF INFLAMMATION AROUND PARASITES IS
Aseptic autolysis
Complete recovery
Restitution
Reparation
+Encapsulation

74. TISSUE REACTION IN GRANULOMATOUS INFLAMMATION IS
Alterative
Exudative
+Proliferative
Necrotic
Dystrophic

75. THE PHAGOCYTING CELLS ARE
Erythrocytes
+Macrophages
Amyloidoblasts
Epithelial cells
Thrombocytes

76. CLINICAL CURRENT OF DISEASES BASED ON PROLIPHERATIVE INFLAMMATION IS
Acute
Fulminant
Recurrent
+Subacute
Acutest

77. THE TYPE OF GRANULOMAS DEPENDING ON CELLULAR STRUCTURE IS
+Macrophage cellular
Plasma cellular

Lymphocytic
Erythrocytic
Leukocytic

78. THE TYPE OF GRANULOMAS DEPENDING ON CELLULAR STRUCTURE IS
Lymphocytic
Plasma cellular
+Epithelioid cellular
Erythrocytic
Leukocytic

79. NON INFECTIOUS GRANULOMA OBSERVES AT
Typhoid fiver
Syphilis
Tuberculosis
+Silicosis
Leprosy

80. INFECTIOUS GRANULOMA OBSEVES AT
Anthracnosis
+Leprosy
Silicosis
Around of surgical sutures
Foreign body

81. PURULENT INFLAMMATION IN GALL- BLADDER CAVITY IS TERMED AS
Abscess
Vesicle
Furuncle
+Empyema
Carbuncle

82. FIBRINOUS INFLAMMATION IN PERITONEAL CAVITY IS TERMED AS
Abscess
Ascites
Furuncle
Empyema
+Peritonitis

83. ENCAPSULATED ACCUMULATION OF PUS WITHIN THE TISSUE IS
Ascites
+Abscess
Granuloma
Furuncle
Vesicle

84. WIDESPREAD FLAT PURULENT INFLAMMATION OF SOFT TISSUE IS TERMED AS
Ascites
Abscess
Granuloma
Furuncle
+Phlegmon

85. CONDILOME IS
Malignant tumor
Thrombotic masses
Blood clot
+Result of productive inflammation
Hypertrophic overgrowth

86. POLYP IS
Malignant tumor
+Result of productive inflammation
Thrombotic masses
Blood clot
Hypertrophic overgrowth

87. ABSCESS IS
Accumulation of pus in pleural cavity
Accumulation of pus in abdominal cavity
+Encapsulated accumulation of pus within the tissue
Widespread flat purulent inflammation of soft tissue
Accumulation of pus around foreign body

88. PHLEGMON IS
Accumulation of pus in pleural cavity
Accumulation of pus in abdominal cavity
Encapsulated accumulation of pus within the tissue
+Widespread flat purulent inflammation of soft tissue
Accumulation of pus around foreign body

89. PURULENT INFLAMMATION IN URINARY BLADDER CAVITY IS TERMED AS
Abscess
Pyonephrosis
Furuncle
+Empyema
Carbuncle

90. PURULENT INFLAMMATION OF NAIL BED IS TERMED AS
Abscess
+Paronychia
Furuncle
Empyema
Phlegmon

6. ADAPTATION

1. REVERSIBLE DISORDER IN MATURATION OF CELLS RESULTS IN VARIABILITY OF SIZE, SHAPE ANDPOLARITY IS
Metaplasia
+Dysplasia
Anaplasia
Hyperplasia
Desmoplasia

2. REGENERATION IS
Transformation of one kind of tissue into another
Restoration of tissue instead of lost
Increasing of organ in mass
+Reaction, directed on restoration of structure and function
Process, directed on preservation of kind

3. THE CAUSE OF PATHOLOGIC ATROPHY IS
Intracellular fat accumulation
+ Pressure
Thrombosis
Proliferation
Adaptation

4. HYPERTROPHY IS
Restoration of tissue instead of lost
+Increase in volume of cells, tissue, organs
Reduction of cells, tissue, organ in volume
Transformation of one kind of tissue into another
Replacement by connective tissue

5. COMPENSATARY ENLARGEMENT OF HEART IS TERMED AS
Dilative
Myogenic
Eccentric
+Concentric
Decompensate

6. DYSPLASIA CAN RESULT IN
Aplasia
Hypoplasia
Hyperpigmentation
Calcification
+Neoplasia

7. HYPERPLASIA IS CHARACTERIZED BY
Increase in the size of cells
+Increase in the number of cells
Increase in the number of nuclei
Shrinkage of cells
Atypia of cells

8. HYPOPLASIA IS CHARACTERIZED BY
Decreased volume of cells
Increased number of cells
+Subdevelopment of organ
Progressive cellular proliferation
Agenesis of an organ

9. PROLIFERATION OF ENDOMETRIUM IN PREGNANCY ISVARIANTOF ADAPTATION
Compensatory hyperplasia

Pathologic hyperplasia
+Hormonal hyperplasia
Compensatory hypertrophy
Hormonal hypertrophy

10. TYPE OF METAPLASIA THAT OCCURS IN RESPIRATORY TRACT OF A HABITUALCIGARETTE SMOKER IS
Squamous to columnar epithelial metaplasia
Metaplasia to undifferentiated mesenchymal cells
Squamous to cuboidal epithelial metaplasia
+Columnar to squamous epithelial metaplasia
Intestinal metaplasia

11. COMPENSATION IS
Transition of one kind of tissue into another
Restoration of tissue instead of lost
+ Reaction, directed on restoration of structure and function
Process, directed on preservation of kind in changed conditions
Proliferation of cells

12. HYPERTROPHY AS A PROCESS IS CHARACTERIZED BY
+Increase of cell and organ in size
Shrinkage of cell and organ
Increase in number of cells
Abnormal organization of cells
Variation of cells in size and shape

13. SIMPLE ENDOMETRIAL HYPERPLASIA IS CHARACTERIZED BY
Atypia of glandular cells
+Increased twisted and enlarged endometrial glands
Prevalence of stroma above glands
Narrowed endometrial glands
Incomplete depolarizated glands

14. THE PIGMENT FOUND IN CYTOPLASM OF MUSCLECELLS DUE TO SENILE ATROPHY
+Lipofuscin
Melanin
Hemosiderin
Bilirubin
Ferritin

15.ORGANIZATION IS
Transformation of one kind of tissue into another
Restoration of tissue instead of lost
+Replacement by connective tissue
Increasing of organ in mass
Reduction of cells, tissue, organ in volume

16. EXAMPLE OF ADAPTIVE RESPONS IS
Hypertrophy of breast during lactation
Hypertrophy of skeletal muscles cells in a body-builder
Hypertrophy of uterus during pregnancy

+Hypertrophy of stomach mucosa due to specify of feeding
Hypertrophy of skeletal muscle cells of the patient with immobilized of fracture

17. HYPOPLASIA IS
Restoration of tissue instead of lost
Increase in volume of cells, tissue, organs .
Reduction of cells, tissues, organs in volume
+Congenital subdevelopment of organ
Replacement by connective tissue

18. TYPE OF CONGENITAL SUBDEVELOPMENT OF ORGAN IS
Aplasia
Agenesis
+Hypoplasia
Hypogenesis
Cirrhosis

19. SYSTEMIC FACTOR THAT INFLUENCE ON WOUND HEALING IS
Blood group
Constitution
+Metabolic status
Shoes size
Color of eyes

20. LOCAL FACTOR THAT INFLUENCE ON WOUND HEALING IS
Hormones (glucocorticoids)
Constitution
Structure of body
+Wound infection
Blood group

21. «KELOID» IS
Atrophic scar
Scar with dysplasia
Abnormal organization of cells
Hypotrophic scar
+Hypertrophic scar

22. EXAMPLE OF ORGANIZATION IS
Hyperplasia of stomach mucosa
Reduction of organ in size in living organism
Deposits of calcium salts duo to hypercalcemia
Occlusion of artery by thrombus
+ Replacement of thrombus by connective tissue

23. HYDRONEPHROSIS IS CHARACTERIZED BY
+Dilatation of the renal pelvis
Thickening of the renal parenchyma
Activation of urine production
Narrowing the renal calyces
Progressive proliferation of cells

24. CHARACTERISTIC VOLUME-OVERLOADED (ECCENTRIC) CARDIAC HYPERTROPHY
Reduced left cavity diameter
Shot and thick papillary muscle
Diffuse muscle hypertrophy
Diffuse diminished wall thickness
+ Dilated left cavity diameter

25. METAPLASIA IS CHARACTERIZED BY
Reversible increase in the size of cells
+Irreversible changes in which one adult cell type is replaced by another adult cell type
Reversible changes in which one adult cell type is replaced by another adult cell type
Reversible abnormal organization of atypical cells
Irreversible abnormal organization of atypical cells

26. METAPLASIA IS
Reversible increase in the size of cells
+Irreversible transformation of one adult cell type into another adult cell type
Reversible changes in which one adult cell type is replaced by another adult cell type
Reversible abnormal organization of atypical cells
Irreversible abnormal organization of atypical cells

27. DYSPLASIA IS CHARACTERIZED BY
Reversible increase in the size of cells
Irreversible changes in which one adult cell type is replaced by another adult cell type
+Reversible changes in which adult cell type is replaced by young dedifferentiated cells
Reversible abnormal organization of atypical cells
Irreversible abnormal organization of atypical cells

28. DYSPLASIA IS
Reversible increase in the size of cells
Irreversible changes in which one adult cell type is replaced by another adult cell type
Reversible abnormal organization of atypical cells
Irreversible abnormal organization of atypical cells
+Reversible disorder of cellular differentiation

29. APLASIA IS
Subdevelopment of organ
Decreasing of size in living organism
Replacement of functional parenchyma on connective tissue
+Congenital defect of organ development
Decreasing of cells in number

30. CONGENITAL ABSENCE OF ORGAN IS
+Aplasia
Atrophy
Hypoplasia
Hypogenesis
Cirrhosis

7. NEOPLASIA

1. THE TERM OF BENIGN TUMOR IS CONSTRUCTED BY COMBINING THE WORD DESIGNATING THE TUMOR CELL ORIGIN PLUS ENDING
-sarcoma
-carcinoma
+-oma
-itis
-osis (-asis)

2. BENIGN TUMOR ARISING FROM FIBROBLASTIC CELLS IS NAMED
Chondroma
Adenoma
Osteoma
+Fibroma
Papilloma

3. DIFFERECE BETWEEN “CANCER IN SITU” AND INVASIVE CANCER
+Penetration of basement membrane
Number of mitotic cells
Metastasis
Nuclear plemorphism
Dedifferentiation of cells

4. MALIGNANT TUMOR ARISING FROM THE MESENCHIMAL TISSUE IS
+Sarcoma
Adenocarcinoma
Papilloma
Cystadenoma
Polyp

5. PAPILLOMA IS
A senile wart
+Epithelial benign tumor
Epithelial malignant tumor
Vascular benign tumor
Inflammatory growth of skin

6. CANCER IS MALIGNANT TUMOUR DEVELOPING FROM
Mesenchymal tissue
Muscular tissue
+Epithelial tissue
Nervous tissue
Vascular tissue

7. HISTOLOGICAL ATTRIBUTE OF CANCER IN SITU (CARCINOMA IN SITU) IS
Invasive growth
Metastases
+Intraepithelial malignant growth
Hemorrhages in tumor tissue
Focus of necrosis in tumor

8. FOLLICULAR CANCER OF THYROID GLAND DEVELOPS FROM
+A-cells

B-cells
C-cells
Mast cells
Reticular cells

9. SARCOMA IS
Tumor-like formation
Benign tumor from mesenchymal tissue
+Malignant tumor from mesenchymal tissue
Focal hyperplasia of epithelium
Malignant tumor from epithelial tissue

10. THE FIRST HEMATOGENIOUS METASTASES OF FEMUR SARCOMA APPEAR IN
Liver
Kidneys
Spleen
+Lungs
Organs of pelvis

11. THE EARLIEST KIND OF CANCER METASTATIC SPREADING IS
Hematogenic
+Lymphogenic
Perineural
Periductal
Implantational

12. THE DIFFERENTIATED CRITERIA BETWEEN BENIGN AND MALIGNANT TUMORS IS
Localization
Size
+Rate and character of the growth
Presence of metaplasia
Character of growth according to the lumen of organ

13. DUE TO MALIGNANT TUMORSIN TISSUES SECONDARY ACCUMULATS
Hyaline
Lipofuscin
+Amyloid
Hemosiderin
Melanin

14. DIAGNOSTIC CELLS FOR HODGKIN'S DISEASE ARE
Pirogov’ cells
+Reed-Berezovsky-Shternberg’ cells
Langhans’ cells
LE cells
Virchov’s cells

15. DIAGNOSTIC CELLS FOR LYMPHOGRANULOMATOSIS ARE
Eosinophils
Epithelioid cells
+Reed-Berezovsky-Shternberg’s cells
Reticular cells

Virchov’s "spheres"

16. FILADELFIAL CHROMOSOME IS CHARACTERISTIC FOR
Myeloma disease
Lymphogranulomatosis
Berkytt’s lymphoma
+Myeloleukemia
Lympholeukemia

17. LEUCEMIC INFILTRATE IS
The center of inflammation
+The center of leukemic cells growth
The center of extramedullaryhematopoiesis
The center of inflammatory proliferation
The center of leucocyte infiltration

18. PROGENITOR CELL OF HEMATOPOIETIC TISSUE IS
Reticular cell
+Stem cell
Lymphoblast
Myeloblast
Erythroblast

19. HISTOLOGICAL SUBTYPE OF HODGKIN'S DISEASE IS
+Nodular sclerosis
Nodular necrosis
Leucocyte predominance
Nodular amyloidosis
Leucocytes depletion

20. HISTOLOGICAL SUBTYPE OF HODGKIN'S DISEASE IS
Nodular necrosis
Leucocyte predominance
+Lymphocyte predominance
Nodular amyloidosis
Aleukemic

21. PRIMARY TUMOR DAMAGE OF BONE MARROW IS NAMED AS
Leukemic reaction
Leukemoid reaction
Lymphomatosis
+Leukemia
Metastatic tumor

22. PATHOHISTOLOGICAL TYPE OF LYMPHOGRANULOMATOSIS IS
With predominance of leucocytes
With leucopenia
+Mixed cellular
Round cellular
Necrotic

23. TYPE OF LEUKEMIA DEPENDING ON CHARACTER OF CURRENT

+Acute, chronic
Acute, subacute
Acute, relapsing
Acute, recurrent
Acutest, acute

24. TYPE OF LEUKEMIA DEPENDING ON DEGREE OF CELL DIFFERENTIATION
+Blastic, cellular
Adult cellular, young cellular
Mature cellular, immature cellular
Small cellular, large cellular
Fast cellular, slow cellular

25. ONE OF TYPES OF LYMPHOMAS NAMED AS
Franclin's disease
Valdenstrem's disease
Mieloma disease
+Hodgkin's disease
Addisson' disease

26. POSTHEMORRHAGIC ANEMIA IS
Local ischemia
Type of systemic hemoblastoses
+General ischemia
Type of regional hemoblastoses
General hyperemia

27. SIGNIFICANT ENLARGEMENT OF ORGANS AT LEUKEMIAS IS A RESULT OF
Hyperemia
Sclerosis
Necrosis
+Leukemic infiltration
Inflammatory infiltration

28. "PORPHYRY" SPLEEN OBSERVES AT
Anemia
Sepsis
+Hodgkin's disease
Reticulosarcoma
Hypertension

29. BONE MARROW AT ACUTE MYELOBLASTIC LEUKEMIA IS
+Greenish-grey color
"Current jelly"-like
Bright-red
Rusty
Brownish-black

30. MAIN DIFFERENCE BETWEEN HYPOPLASTIC ANDHYPOREGENERATIVE ANEMIAIS
Different amount of reticulocytes
+Atrophy of bone marrow
Blast cells proliferation in bone marrow

Megakaryocytic dysplasia
Hemosiderosis

31. THE DIFFERENTIATED CRITERIA BETWEEN BENIGN AND MALIGNANT TUMORS IS
+Maturity
Localization
Size
Presence of metaplasia
Character of growth according to the lumen of organ

32. THE DIFFERENTIATED CRITERIA BETWEEN BENIGN AND MALIGNANT TUMORS IS
Localization
+Metastases
Size
Presence of metaplasia
Character of growth according to the lumen of organ

33. THE DIFFERENTIATED CRITERIA BETWEEN BENIGN AND MALIGNANT TUMORS IS
Localization
+Anaplasia
Size
Presence of metaplasia
Character of growth according to the lumen of organ

34. HISTOLOGICAL SUBTYPE OF HODGKIN'S DISEASE IS
Nodular necrosis
Leucocyte predominance
+Mixed –cellular
Nodular amyloidosis
Aleukemic

35. BONE MARROW AT ACUTE MYELOBLASTIC LEUKEMIA IS TERMED AS
+Pyoid
Dermoid
Hemorrhoid
Fibrinoid
Rusty

36. THE EARLIEST HEMATOGENIC METASTASES OF GIT–LOCALISATED CANCER SPREADING CAN BE FOUNED IN
Brain
Lymph nodes
Brest
+Liver
Peritoneum

37. THE DIFFERENTIATED CRITERIA BETWEEN BENIGN AND MALIGNANT TUMORS IS
Localization
Size
+Metastasizing
Presence of metaplasia
Character of growth according to the lumen of organ

38. “CANCER” IS
Signature of malignant tumors from mesenchymal tissue
Signature of benign tumors from mesenchymal tissue
+ Signature of malignant tumors from epithelial tissue
Signature of benign tumors from epithelial tissue
Signature of malignant tumors from nervous tissue

39. “BLASTOMA” IS EDITION FOR THE NAME OF
Malignant tumors from mesenchymal tissue
Benign tumors from mesenchymal tissue
Malignant tumors from epithelial tissue
Benign tumors from epithelial tissue
+Malignant tumors from nervous tissue

40. THE DIFFERENTIATED CRITERIA BETWEEN BENIGN AND MALIGNANT TUMORS IS
Localization
Size
+ Presence of cellular atypia
Presence of metaplasia
Character of growth according to the lumen of organ

8. CARDIO-VASCULAR DISEASES

1. GENERAL PATHOLOGICAL PROCESS IN DECOMPENSATED MYOCARDIUM AT HEART DEFECTS IS
Atrophy
+Fatty dystrophy
Calcification
Hemosiderosis
Necrosis

2. ABACTERIAL ACUTE WARTY ENDOCARDITIS IS CHARACTERIZED BY
Parietal endocardium involvement
+Mitral and/or aortic valve involvement
Small bacterial verrucae
Verrucae on papillary muscles
Ulceration of valve surface

3. COMPLICATION OF ACUTE WARTY ENDOCARDITIS OF MITRAL VALVE CAN BE
Infarction of lung
Thromboembolism of lung arteries
Abscess of brain
+Infarction of kidney
Embolic nephritis

4. RHEUMATIC HEART DISEASE IS CHARACTERIZED BY CHANGES OF MITRAL VALVE
Abscess formation
Thinned and elongated chordate muscles
«Dog mouth» appearance of the mitral valve
Undamaged valve shatters
+Thickened and deformed valve shatters

5. IN SYSTEMIC HYPERTENSION WITHIN ARTERIOLAR WALLS DEPOSETS
Amyloid
Glycogen
Cholesterol or cholesterolesters
Lipoprotein
+Hyaline

6. CHANGES IN KIDNEY DEVELOPING DUE TO HYPERTENSIVE DISEASE
Secondary - reduced kidney
+Primarily - reduced kidney
Far's nephrosclerosis
Kimmelstyl-Wilson's syndrome
Polycistosis

7. RISK FACTOR FOR ENDOTHELIAL INJURY IN ATHEROSCLEROSIS MAY BE
Immune reactions.
Hypertrophy
Sports
Hypercalcemia
+Hyperlipidemia

8. THE COMPLICATION OF ATHEROMATOUS PLAQUE FORMATION IS
+Thrombus formation
Stone formation
Plaque malformation
Inflammation
Metastatic calcification

9. BROWN ATROPHY OF HEART CHARACTERIZED BY
Increased size of heart
Twisting of heart
Increased subepicardial fat
Flabby yellow-colored myocardium
+ Flabby brown-colored myocardium

10. LEFT-SIDED HEART FAILURE IS MOST OFTEN CAUSED BY
+Ischemic heart disease
Brown atrophy of the myocardium
Pulmonary edema
Hepatitis
Anasarca

11. MYOCARDIAL INFARCTION FULL ORGANIZATION IS
2 weeks
2 month
3 weeks
+1 months
3 months

12. MORPHOLOGICAL AND CLINICAL EFFECTS OF PURE (ISOLATED) RIGHT-SIDED HEART FAILURE

+Cerebral hematoma
Congestive hepatomegaly
Ascites
Pleural and pericardial effusions
Anasarca

13. ACUTE RHEUMATIC CARDITIS IS CHARACTERIZED BY
Brown atrophy of heart
+Aschoff bodies in the myocardium
Goose liver
Cardiosclerosis
“Tiger heart”

14. ETIOLOGY AND PATHOGENESIS OF RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE ARE CHARACTERIZED BY
Decreased serum protein levels
Absence of serum titers of antibodies to streptolysin and hyaluronidase
Unsterile tissue lesions
Acute streptococcal infection
+Initial attack of disease some weeks after streptococcal infection

15. HEART LESIONS IN ACUTE RHEUMATIC FEVER ARE TERMED
Tuberculoma
+Aschoff bodies
Foreign bodies
Gum
Mitral stenosis

16. MICROSCOPICACHARACTERISTIC OF CHRONIC RHEUMATIC HEART DISEASE IS
Acute purulent inflammation
Devascularization of shutters
+Diffuse fibrosis of shutters
Destruction of myocardiumt architecture
Apoptotic bodies in connective tissue

17. POSSIBLE OUTCOME OF RHEUMATIC MYOCARDITIS IS
Heart defect
+Cardiosclerosis
Brown atrophy of heart
Obliteration of pericardium cavity
Adiposity of heart

18. THE FIGURATIVE NAME OF HEART AT RHEUMATIC PERICARDITIS IS
Pulmonary heart
"Bull" heart
"Tiger" heart
+"Hairy" heart
Stone heart

19. ENDOCARDITIS AT RHEUMATISM OCCURS AS
Polipous-ulcer
+Diffuse [Talalaev's] valvulitis

Acute ulceral
Calcificated
Dystrophic

20. CHARACTERISTIC MORPHOLOGICAL CHANGE AT NODULAR PERIARTERIITIS ISArteriolosclerosis
Atherosclerosis
Fibrinoid necrosis
+Destructive – proliferative vasculitis
Amyloidosis

21. AT ATHEROSCLEROSIS PREDOMINANTLY SUFFERED
Veins
Small arteries
Arterioles
+Aorta
Capillary net

22. THE DIAGNOSIS OF ESSENTIAL HYPERTENSION IS GIVEN ONLY IF
Presence of risk factors
Presence of arteriolosclerotic nephrocirrhosis
+Absence of organic diseases, explained hypertension
Presence of true features of connection with psychoemoutional stress
Coronary insufficiency development

23. DECOMPENSATION OF HYPERTROPHITED HEART IS APPEARED AS
+Myogenic dilation of cavities
Tonogenic dilation of cavities
Rheumatic myocarditis
Fibrinouse pericarditis
Acute warty endocarditis

24. THE ESSENTIAL HYPERTENSION IS
+Hypertension – primary appearance of disease
Hypertension – complication of other disease
Hypertension – secondary appearance of other disease
Hypertension on background of atherosclerosis
Hypertension on background of heart defect

25. BRAIN INFARCTION CAN COMPLICATED BY
Pneumonia
+Edema and dislocation of brain
Pulmonary thromboembolism
Thrombocytopenia
Anencephalia

26. CHRONIC ISCHEMIA OF RENAL TISSUE DUE TO ATHEROSCLEROSIS LEADS TO:
Hypertrophy
Sclerosis of capsule
Infarction
Gangrene
+Atrophy of parenchyma

27. CHANGES IN ARTERIOLS DUE TO ESSENTIAL HYPERTENSION ARE
Amyloidosis
Liposclerosis, calcynosis
Atheromatosis, ulceration
Atherosclerosis, elastofibrosis
+Infiltration by plasma, hyalinosis

28. THE MOST OFTEN OUTCOME OF ISCHEMIC INFARCTION IN BRAIN IS
Complete regeneration
Scarring
+Cyst formation
Hydrocephalus
Purulent lepthomeningitis

29. ATHEROSCLEROSIS OF RENAL ARTERIES CAN RESULT IN
+Infarctions
Amyloidosis
Embolic purulent nephritis
Hydronephrosis
Calcinosis

30. MORPHOLOGICAL CHANGES OF ARTERIOLS IN HYPERTENSIVE STROKE IS
Liposclerosis
Elastofibrosis
+Plasmatic infiltration, fibrinoid necrosis
Hyalinosis, sclerosis
Atherosclerosis, calcinosis

31. THE IMMEDIATE CAUSE OF ATHEROSCLEROTIC LOW EXTREMITY GANGRENE IS
Limphostasis
+Occlusion of arteries
Thrombosis of deep veins
Rupture of veracious-expended veins
Endarteritis

32. ON THE WALL OF ABDOMINAL AORTAAT ATHEROSCLEROSIS CAN BE SEEN
Concrements
Amyloidosis
Vesicles
+Fatty stains and strips, fibrous plaques
Polyps

33. IN BRAIN TISSUE DUE TO HYPERTENSIVE STROKE CAN BE SEEN
Cyst
Glial scar
Atrophy of cortex
+Diapedetic hemorrhages
Cellular proliferation

34. CLINICAL-MORPHOLOGIC TYPES OF ESSENTIAL HYPERTENSION BY CHARACTER OF CURRENT ARE

Cardiac, brain
Mesenterial, pulmonary
Hepatic, aortal
Low extremity
+Benign, malignant

35. CEREBRAL FORM OF ESSENTIAL HYPERTENSION IS CHARACTERIZED BY
Atrophy of cortex
Hydrocephalus
Encephalitis
+Brain hemorrhage
Meningitis

36. ATHEROSCLEROSIS IS CHARACTERIZED BY
Progressive desorganisation of connective tissue
Hyalinosis of vessels
Damage of microcirculative blood supply
Adge-prowided sclerosis of vessels walls
+Damage of elastic and muscle-elastic type arteries as a result of fat and protein metabolism disorders

37. CHARACTERISTIC OUTCOME OFRENAL FORM AT HYPERTENSION IS
+Primary reduced kidney
Amyloido-reduced kidney
Secondary reduced kidney
Hydronephrosis
Glomerulonephritis

38. DURATION OF MYOCARDIAL INFARCTION PRENECROTIC STAGE IS
Two hours
1 month
10 days
2 days
+18 hours

39. ONE OF THE STAGES OF MYOCARDIAL INFARCTION DEVELOPMENT IS
Dyscirculatory
Transitional
Dystrophyc
+Necrotic
Reconvalescence

40. ACUTE TRANSMURAL MYOCARDIAL INFARCTION MAY RESULT IN
Idiopathic pericarditis
Concentric left ventricle hypertrophy
Thrombosis of low extremity vein
+Acute aneurysm
Atherosclerosis of aorta

41. THE MOST FREQUENT REASON OF SUBARACHNOID HEMORRHAGE IS
+Rupture of brain artery aneurism
Rupture of atherosclerotic plaque
Closed crania-cerebral trauma

Blood disease
Vasculitis

42. THE RISK FACTORS OF ISCHEMIC HEART DISEASE DEVELOPMENT ARE
Arterial hypotension and hypodinamia
Diathesis and diarrhea
Anemia and shock
Usual sweets and much sugar eating
+Smoking and atherosclerosis

43. CHRONIC ISCHEMIC HEART DISEASE RESULTS IN
+Cardiosclerosis
Myocardial infarction
Tamponade of heart
Thrombophlebitis of low extremity
Avitaminosis

44. THE MAIN REASON OF DEATH AT SUBARACHNOID HEMORRHAGE IS
+Dislocation of brain
Destruction of vegetative centers of regulation
Destruction of sensomotoric centers
Hypothalamic-pituitary disorders
Anemia and shock

45. EXCEPT HEART DAMAGE RHEUMATISM IS CHARACTERIZED BY
+Damage of joints
Caseouse necrosis
Pancreatitis
Pyelonephritis
Gettington'schorea

46. THE MOST OFTENCOMBINATIVE CONGENITAL HEART DEFECT IS
The common arterial trunk
Three-chambered heart
Aortal stenosis
Transposition of main vessels
+Fallouts' tetrad

47. RHEUMATISM MOST COMMONLY AFFECTS
Aortal valve
+Mitral valve
Tricuspid valve
Pulmonary artery valve
Peripheric venous valves

48. ENDOCARDITIS AT SYSTEMIC LUPUS ERYTHEMATOSUS IS
Acute ulceral
Bacterial subacute
Septic
+Abacterial warty
Polypous-ulceral

49. AORTAL VALVE STENOSIS RESULTS IN
Pulmonary hypertension
+Hypertrophy of left ventricle
Hypertrophy of right ventricle
Myocardial infarction
Heart aneurysm

50. RHEUMATIC DAMAGE OF JOINTS CHARACTERIZED BY
+Acute serous-fibrinouse synovitis
Productive synovitis
Purulent destructive arthritis
Ankylosis
Pathological fractures

51. THE CHARACTERISTIC FEATURE OF RELAPSING-WARTY ENDOCARDITIS IS
Sclerosis of shutters and there ulceration
Perivascular cardiosclerosis
+Sclerosis of shutters with thrombotic warty-like depositions
Petrifaction and hyalinosis of shutters
Perforation of shutters

52. AT RHEUMATOID ARTHRITIS IN JOINTS DEVELOPS
Acute serous-fibrinouse synovitis
+Productive non-purulent synovitis
Purulent destructive arthritis
Amyloidosis
Acute ulceral synovitis

53. SKIN CHANGES DUE TO SYSTEMIC SCLERODERMA ARE CHARACTERIZED BY
+Hyalinosis
Petrifaction
Dermatitis
Suppuration
Coagulation

54. THE MOST COMMON DAMAGE OF KIDNEY AT LUPUS ERYTHEMATOSUS IS
Amyloidosis
Nephrolithiasis
Pyelonephritis
+Glomerulonephritis
Papillary necrosis

55. PROVIDING FACTOR IN RHEUMATISM DEVELOPMENT IS
Staphylococcus Aureus
+β-Hemolytic Streptococcus
Streptococcus Type B
EscherichiaColi
Clostridia Perpfringens

56. RHEUMATOID ARTHRITIS USUALLY RESULTS IN
Idiopathic primary amyloidosis
Chronic gastric ulcer

Immunodeficiency syndrome
+Ankylosis
Obliterate thrombangitis

57. THE DISEASE REFERS TO COLLAGENOSES
Tuberculosis
+Scleroderma
Atherosclerosis
Amyloidosis
Arthrosis

58. THE DIFFERENTIAL FEATURE OF ACUTE WARTY ENDOCARDITIS FROM RELAPSING WARTY IS
Presence of endothelial damage
Degree of connective tissue disorganization
+Fibrosis of valve
Progression
Presence of thrombotic warty-like depositions

59. POSSIBLE COMPLICATION OF RELAPSING WARTY ENDOCARDITIS IS
Purulent meningitis
Mediastinitis
+Infarction of spleen
Thrombosis of veins
Pulmonary infarction

60. THE FEATURE OF ACTIVITY OF RHEUMATIC PROCESS IS
Aortal valve insufficiency
Aortal valve stenosis
Blood regurgitation
Shorting and thickening of chords
+Perivascular cardiosclerosis

61. THE OUTCOME OF VALVULAR ENDOCARDITIS IS
Sclerotic plaques
Diffuse cardiosclerosis
Hydrocephalus
+Heart defect
Macrofocal cardiosclerosis

62. THE COMON COMPLICATION OF RELAPSING-WARTY ENDOCARDITIS IS
+Infarctions in organs of systemic circulation
Obesity
Abscess of brain
Ulcer of stomach
Pulmonary thromboembolism

63. ABACTERIAL ACUTE WARTY ENDOCARDITIS IS CHARACTERIZED BY
Parietal endocardium involvement
+Small (1 to 5 mm), sterile warty
Small bacterial warty
Warty on papillary muscles

Ulceration of valve surface

64. ABACTERIAL ACUTE WARTY ENDOCARDITIS IS CHARACTERIZED BY
Parietal endocardium involvement
Small bacterial warty
+Warty along the line of valve closure
Warty on papillary muscles
Ulceration of valve surface

65. ABACTERIAL ACUTE WARTY ENDOCARDITIS IS CHARACTERIZED BY
Parietal endocardium involvement
Small bacterial warty
Warty on papillary muscles
Ulceration of valve surface
+Connective tissue disorganization

66. RHEUMATIC HEART DISEASE IS CHARACTERIZED BY CHANGES IN MITRAL VALVE
Abscess formation
+"Jacket loop" appearance of mitral valve
Thinned and elongatedpapillarymuscles
«Dog mouth» appearance of the mitral valve
Undamaged valve shatters

67. RHEUMATIC HEART DISEASE IS CHARACTERIZED BY
Abscess formation
+Thickened and shortened papillary muscles
Thinned and elongated papillary muscles
«Dog mouth» appearance of the mitral valve
Undamaged valve shatters

68. RHEUMATIC HEART DISEASE IS CHARACTERIZED BY CHANGES IN MITRAL VALVE
Abscess formation
Kidney calcification
+«Fish mouth» appearance of the mitral valve
Thinned and elongated papillary muscles
Undamaged valve shatters

69. RISK FACTOR FOR ENDOTHELIAL INJURY IN ATHEROSCLEROSIS MAY BE
Immune reactions.
Hypertrophy
Sports
+Genetic defects
Hyperbillirubinemia

70. BROWN ATROPHY OF HEART CHARACTERIZED BY
Increased size of heart
Twisting of heart
Obesity of heart
Flabby yellow myocardium
+Decreased subepicardial fat

9. PULMONARY DISEASES

1. HEART INJURY AT PULMONARY FIBROSIS AND EMPHYSEMA OF LUNGS RESULTS IN
Atrophy of myocardium
Adiposity
Hypertrophy of left ventricle
+Hypertrophy of right ventricle
Myocardial infarction

2. ONE OF POSSIBLE COMPLICATIONS OF BRONCHOPNEUMONIA IS
Lung infarction
Pneumothorax
Milliary tuberculosis
+Abscess formation
Renal failure

3. THE COMMONPREDISPOSAL FACTOR OF BACTERIAL PNEUMONIA DEVELOPMENT IS
Bacterial urinary tract infection
Usual sweets and sugar eating
Teeth diseases
Congestive heart failure
+Viral respiratory tract infections
Drug abuse

4. THE ABNORMAL DILATION OF BRONCHIAL TUBES IS THE CHARACTERISTIC OF
Emphysema
Lung abscess
+Bronchiectasis
Bronchial asthma
Bronchitis

5. CARNIFICATION OF LUNG FOR CROUPOUSE PNEUMONIA IS
Outcome
+Complication
Appearance
Reason
Background

6. NAME TYPES OF BRONCHOECTASES
Pneumoniogenic
Obstructive
+Sackular
Infective
Dystrophic

7. THE MOST OFTEN TYPE OF ACUTE BRONCHITIS IS
Obstructive
Deformative
+Catarrhal
Pulpous
Fibrinous

8. THE MOST COMMON REASON OF LUNG EMPHYSEMA IS
Lung abscess
+Chronic bronchitis
Focal pneumonia
Tracheitis
Laringitis

9. ONE OF POSSIBLE PULMONARY COMPLICATION AT CROUPOUSE PNEUMONIA IS
+Lung abscess
Lung infarction
Mediastenitis
Lung cancer
Tuberculosis

10. ACUTE ABSCESS BECOMES CHRONIC THROUGHOUT
2 weeks
+1 month
2 years
8 month
2 hours

11. BROWN INDURATION OF LUNG IS THE RESULT OF
Inflammation
Atelectasis
Necrosis
+Congestion
Bronchiectasis

12. THE CHARACTER OF INFLAMMATION AT BRONCHOPNEUMONIA DEPENDS ON
Volume of destruction
Age of patient
Type of pathogenic agent
+Mechanism of inflammation
Localization of process

13.EXTRAPULMONARY COMPLICATION OF CHRONIC ABSCESS IN LUNG IS
+Amyloidosis
Hyalinosis
Lipidosis
Hyperglycemia
Thrombosis

14. AT PNEUMOFIBROSIS AND EMPHYSEMA OF LUNGS IN THE HEART DEVELOPS
Atrophy
Adiposity
Hypertrophy of left ventricle wall
+Hypertrophy of right ventricle wall
Myocardial infarction

15. EXUDATES IN GREY HEPATISATION STAGE OF CROUPOUS PNEUMONIA CONTENTS
Edematous fluid and bacteria
Fresh erythrocytes and fibrin

+Leucocytes and fibrin
Granulation tissue
Lymphocytes and plasma cells

16. LUNG ABSCESS AT CROUPOUSE PNEUMONIA MORE OFTEN FORMS IN STAGE
Inflow
Red hepatization
Grey hepatization
+Resolution
Incubational

17. USUALLY THE INFLOW STAGE OF CROUPOUS PNEUMONIA IS LASTING
5 day
+1-2 day
4-6 day
9-11 day
21 days

18. DISEASE RESULTS IN CHRONIC OBSTRUCTIVE LUNG EMPHYSEMA DEVELOPMENT
+Chronic bronchitis
Tracheitis
Focal pneumonia
Lung abscess
Croupous pneumonia

19. THE SYNONYM OF FOCAL PNEUMONIA IS
Croupouse pneumonia
Pleuropneumonia
+Bronchopneumonia
Interstitial pneumonia
Caseous pneumonia

20. MINOR PULMONARY THROMBOEMBOLISM RESULTS IN
Sudden death from pulmonocoronary reflex
Myocardial infarction
Cardiogenic shock
+Hemorrhagic pulmonary infarction
DIC-syndrome

21. THE SYNONYM OF CROUPOUSE PNEUMONIA IS
+Lobar
Lobular
Hemorrhagic
Focal
Bronchopneumonia

22. CARNIFICATION OF LUNGS IS
Inflammatory infiltration of interstitial tissue
Persistence of pulmonary exudates in alveoli
Deposition of pigment in intraalveolar septs
Formation of hyaline membranes on alveolar walls
+Organization of unresolved exudates

23. THE CHARACTER OF PLEURISY AT CROUPOUSE PNEUMONIA IS
Serous
Purulent
Granulomatous
+Fibrinous
Hemorrhagic

24. THE MOST COMMON ETHYOLOGICAL FACTORS OF BRONCHOPNEUMONIA ARE
+Bacteria, viral-bacterial association
Mycoplasma, Chlamydia
Viral
Fungi
Protozoa

25. PULMONARY COMPLICATIONS OF CROUPOUS PNEUMONIA ARE
+Abscess, cornification
Chronic bronchitis, emphysema
Bronchiectasis
Pneumothorax, empyema
Brain abscess

26. MORPHOLOGICAL CHANGES IN LUNGS DUE TO CHRONIC VENOUS CONGESTION
Hemorrhagic infarction and petrifaction
Acute edema
Antracosis and necrosis
+Hemosiderosis and diffuse pneumosclerosis
Carnification

27. MORPHOLOGICAL CHANGES IN LUNGS AT ASPIRATION PNEUMONIA ARE
Infarction and petrifaction
Hemorrhage
+Necrosis and suppuration
Serous inflammation
Edema

28. TYPE OF FOCAL PNEUMONIA DEPENDING ON SPREADING OF PROCESS
Hypostatic
Pneumococcal
Aspiration
+Polysegmentary
Bronchopneumonia

29. MACROSCOPICAL APPEARANCE OF LUNGS AT EMPHYSEMA
Increased, dense, pail
+Increased, soft, pail
Increased, soft, hyperemia
Decreased, soft, honeycomb appearance
Decreased, dense, brown

30. MASSIVE PULMONARY THROMBOEMBOLISM RESULTS IN

+Sudden death from pulmonocoronary reflex
Myocardial infarction
Shock
Hemorrhagic pulmonary infarction
DIC

31. STAPHYLOCOCCAL BRONCHOPNEUMONIA IS CHARACTERIZED BY
Fibrinous exudates
Hemorrhagic exudates
+Purulent exudates, necrosis
Lymphoid infiltrations
Granulomatous inflammation

32. THE COMPOSITION OF HYALINE MEMBRANE IN LUNGS AT ACUE RESPIRATORY DISTRESS SINDROM OF FETUS
Albumins and complement
+Fibrin
Precipitated surfactant
Mucus
Plasma

33. IN GREY HEPATIZATION STAGE OF CROUPOUS PNEUMONIA
White blood cells fill the alveoli
Red Blood cells fill the alveoli
Organisms fill the alveoli
+Accumulation of fibrin fills the alveoli
Accumulation of pus fills the alveoli

34. GOODPASTURE'S SYNDROME IS CHARACTERISED BY
+Necrotizing hemorrhagic interstitial pneumonitis
Alveolitis
Patchy consolidation
Pulmonary edema
Purulent bronchitis

35. FAVORABLE OUTCOME OF LOBAR PNEUMONIA IS
Consolidation of exudate
+Resolution of exudate
Abscess formation
Empyema of pleura
Emphysema

36. BROWN INDURATION OF LUNG IS RESULT OF
Silicosis
Berilliosis
Asbestosis
Bronchiectasis
+Hemosiderosis

37. MORPHOLOGIC TYPE OF BRONCHIECTASES IS
+ Varicose

Bullous
Obstructed
Irregular
Panacinary

38. COMMONEST TYPE OF EMPHYSEMA IS
+Bullous
Obstructed
Irregular
Panacinary
Sackular

39. SOLID AIRLESS LUNG DUE TO FIBRINOUS EXUDATES ACCUMULATION IN ALVEOLI
Chronic bronchitis
Bronchial asthma
Bronchiectasis
+Lobar pneumonia
Pneumonitis

40. CHARACTERISTIC OF PULMONARY INFARCTION IS
+Mostly red infarct with hemorrhagic exudate
Mostly white infarct with hemorrhagic areola
Occurs commonly with fat embolism
Often from aortal aneurism
Irregular shape

41. INVESTIGATION OF SPUTUM IN ASTHMA MAY SHOW
Numerous neutrophils
Accumulation of erythrocytes
Foreign body cells
+Curschmann's spirals
Viral bodies

42. MOST COMMON LUNG MALFORMATION
Hypoplasia of lung
+Congenital cyst
Vascular anomalies
Lobar sequestration
Fragmentation of lung

43. THE MOST COMMON SITE OF SMALL CELL LUNG CARCINOMA METASTASATIONIS
Brain
+Adrenal
Liver
Bones
Kidny

44. CHARACTERISTIC FEATURE OF VIRAL PNEUMONIAS IS
+Intra-alveolar tree types exudate accumulation
Intra-alveolar proteinaceous exudate
Hyaline membrane lining alveoli
Fibrotic septa

Fibrinous exudate

45. EMPHYSEMAIS
Inflammation of bronchi
+Overloading f alveolar sack by air
Pathological expansion of terminal bronchiole
Occlusion of respiratory bronchiole
Hyperventilation of lung

46. COMMONEST TYPE OF LUNG CANCER IN NON-SMOKERS IS
Squamous cell carcinoma
+Adenocarcinoma
Small cell carcinoma
Large cell carcinoma
Chorioepithelioma

47. COMPLICATION OF BRONCHIECTASIS IS
Chorionepithelioma
+Amyloidosis
Peritonitis
Myocardial infarction
Thirotoxicosis

48. STAGES OF LOBAR PNEUMONIA WITH CLASSICAL FIBRINOUS INFLAMMATION
Hyperemia
Red hepaization
+Grey hepatization
Resolution
Inflow

49. IN PRIMARY ATYPICAL PNEUMONIA INFLAMMATORY CELLS ACCUMULATE IN
Alveolar lumen
Bronchioles
+Alveolar wall
Pleural space
Interstictium

50. REID'S INDEX IS USED IN DIAGNOSIS OF
+Chronic bronchitis
Bronchiectasis
Bronchial asthma
Pneumonia
Pleuritis

51. SMOKING CAUSES
Bronchiectasis
+Chronic bronchitis
Empyema
Theratoma
Ankyloses

52. HYALINE MEMBRANES IN THE LUNG IS SEEN IN
+Respiratory distress syndrome
Pulmonary edema
Pneumococcal Pneumonia
Acute viral hepatitis
Emphysema

53. LATE COMPLICATION OF BRONCHOPULMONARY DYSPLASIA IS
Peritonitis
Hepatitis
+Decreased functional residual capacity
Atrophy of kidney
Hypotrophy of heart

54. MYCOPLASMA INFECTION SIMULATES
Pneumococcal pneumonia
+Viral pneumonia
Hypersensitivity pneumonia
Aspiration pneumonia
Empyema of pleura

55. NORMAL AMOUNT OF PLEURAL FLUID IS
5 ml
+15 ml
50ml
100ml
1L

56. PREDISPOSING FACTORS OF LUNG ABSCESS IS
Adequate treatment of pneumonia
Rhinitis
+Endobronchial obstruction
High immunity
Blood circulation

57. AN INFLAMMATORY STAGE OF PNEUMONIA IS
+ Grey hepatisation
Congestion
Resolution
Organization
Scarring

58. LUNG CANCER COMMONLY METASTASISES TO
Kidney
Extremity
Pericardium
+Bones
Stomach

59. FOCAL PNEUMONIA IS CHARACTERIZED BY
Damage of lung lobe
Involving of pleura in process

+Presence of bronchitis, bronchiolitis
Caseouse necrosis of exudates
Pulmonary tissue necrosis

60. ONE OF POSSIBLE PULMONARY COMPLICATION AT CROUPOUSE PNEUMONIA IS
+Lung abscess
Lung infarction
Mediastenitis
Lung cancer
Tuberculosis

61. MACROSCOPICAL APPEARANCE OF LUNGS AT BRONCHOECTASIS
Increased, dense, pail
+Increased, dense,honeycomb appearance
Increased, soft, hyperemia
Decreased, soft, pail
Decreased, soft, brown

62. COMMONEST TYPE OF LUNG CANCER IN SMOKERS IS
+Squamous cell carcinoma
Adenocarcinoma
Small cell carcinoma
Large cell carcinoma
Chorioepithelioma

63. COMPLICATION OF BRONCHIECTASIS IS
Chorioepithelioma
Peritonitis
+Lung abscess
Myocardial infarction
Thyrotoxicosis

64. COMPLICATION OF BRONCHIECTASIS IS
Chorioepithelioma
Peritonitis
Myocardial infarction
+Cor pulmonale
Thyrotoxicosis

65. COMPLICATION OF BRONCHIECTASIS IS
Chorioepithelioma
Peritonitis
Myocardial infarction
Thyrotoxicosis
+Obstructive emphysema

66. SMOKING CAUSES
Bronchiectasis
Empyema
Theratoma
Ankyloses

+Emphysema

67. SMOKING CAUSES
Bronchiectasis
Empyema
Theratoma
Ankyloses
+Bronchogenic cancer

68. SMOKING CAUSES
Bronchiectasis
Empyema
Theratoma
Ankyloses
+Antracosis

69. ACCUMULATION OF FLUID IN THORACIC CAVITY IS TERMED AS
Hydropericardium
Hydrocele
Hemothorax
+Hydrothorax
Ascites
70. MORPHOLOGIC FEATURE OF BRONCHIAL WALL IN BRONCHOPNEUMONIA IS
Endobronchitis
Mesobronchitis
Perybronchitis
+Panbronchitis
Mesoarteritis

10. GIT DISEASES

1. MICROSCOPICAL FEATURE OF ACUTE APPENDICITIS IS
+Diffuse neutrophil infiltration
Epithelioid cell infiltration
Granuloma formation
Polypus overgrowth
Hydrocele

2. ONE OF CELLTYPE IN GLANDS OFSTOMACH
Squamose
Plasmatic
Giant
+Parietal
Chordal

3. CHRONIC INFECTION OF THE GASTRIC MUCOSA IS ASSOCIATED WITH
Escherichia coli
+Helicobacter pylori
Campilobacter jejuni
Enterococcus falcium
Staphillacoccus aureus

4. ACUTE GASTRITIS IS COMMONLY ASSOCIATED WITH

Delayed gastric emptying
Headache
Hypersaivation
+Systemic infections
Obesity

5. THE MOST TYPICAL CHANGES OF VESSELS IN CHRONIC ULCER REMISSION ARE
+Sclerosis of walls
Hyperemia
Anemia
Thinning of vessels walls
Lipoidosis

6.MACROSCOPIC CHARACTERISTIC OF APPENDIX IN ACUTE APPENDICITIS IS
Firm and indurate
+Enlarged with thickened walls
Hydrocele
Mucocele
Multychamber

7. MORE OFTEN PRIMARY MALIGNANT TUMOR OF ESOPHAGUS IS
Adenocarcinoma
+Squamous cancer
Undifferentiated cancer
Malignant melanoma
Leiomyosarcoma

8. LARGE ULCER WITH DIRTY YELLOW-GREEN BOTTOM AND ROUGH EDGES ON MUCOUS MEMBRANE OF RECTUM IS
+Cancer of rectum
Amebiasis
Cholera
Iersiniosis
Salmonellosis

9. KRUKENBERG'S TUMOR IS
Teratoblastoma
Bilateral ovary cancer with solid structure
+Metastasis of stomach cancer to ovary
Metastasis of stomach cancer to supraclavicular lymph node
Tumour of kidney

10. IN STOMACH PRECANCEROUS CONDITION IS
Melory-Vayss' syndrome
Catarrhal gastritis
Squamous metaplasia of epithelium
+Chronic atrophic gastritis with dysplasia
Erosive gastritis

11. THE PIGMENT FORMATING IN THE BOTTOM OF ULCER IS
Hemomelanin
Hemosiderin

+Hydrochlorid hematin
Porphyrin
Hemochromatin

12. GENERAL FACTORS OF ULCERAL DISEASE PATHOGENESIS ARE
Vascular, necrotic
+Neuro-humoral, infection
Toxic, traumatic
Chemical, physical
Exogenic, endogenic

13. STOMAC CANCER MORE OFTEN GIVES HEMATOGENIOUS METASTASES IN
+Liver
Ovary
Adrenals
Regional lymph nods
Pararectal adiposal tissue

14. THE DESTRUCTIVE TYPE OF APPENDICITIS IS
Simple
Superficial
+Phlegmonous
Catarrhal
Serous

15. THE MOST OFTEN LOCALISATION OF LARGE INTESTINE CANCER IS
Caecum
Ascending part
+Recto-sigmoid
Colon
Transvers part

16. CHANGES IN MUSCLE LAYER OF STOMACH WALL AT CHRONIC ULCER IS
+Replacement by connective tissue
Dystrophy
Atrophy
Inflammation
Petrifaction

17. POSSIBLE COMPLICATION OF ULCERAL DISEASE OF STOMACH IS
Duodenitis
+Antral stenosis
Hepatitis
Sepsis
Meningitis

18. “VIRCHOV’S METASTASIES” OF STOMACH CANCER ARE
Lymph nodes of curvature minor
+Supraclavicular lymph nods
Ovary
Perirectal lymph nods
Mesenteric lymph nods

19. THE MOST COMON HYSTOLOGICAL TYPE OF STOMACH CANCER IS
+Adenocarcinoma
Signet ring cell carcinoma
Squamous cellular carcinoma
Undifferentiated cancer
Skyrr (fibrous cancer)

20. POSSIBLE COMPLICATIONS OF DESTRUCTIVE APPENDICITIS ARE
Hydrocele
Empyema of pleura
+Perforation and peritonitis
Gangrene of small intestine
Mummification

21. ETIOLOGICAL FACTOR OF ESOPHAGITIS IS
Chronic gastritis
+Reflux-gastritis
Chemical reactions in stomach
Stenosis of aorta
Urination

22. CHRONIC TYPE OF APPENDICITIS DUE TO ACCUMULATION OF MUCOSAL SECRET IS TERMED AS:
Cyst
Hydrocele
+Mucocele
Pneumocele
Varicocele

23. THE DIFFERENCE BETWEEN EROSION AND ACUTE ULCER OF STOMACH IS
Bottom sclerosis
+Deepness of necrosis
Inflammatory reaction
Hyperplasia of glands in margins
Epithelial metaplasia

24. THE COMPLICATION DEVELOPING AT ACUTE PERIOD OF CHRONIC STOMACH ULCER IS:
Deformation of stomach
Pylorostenosis
Malignesation
+Erosive bleeding
Polyp formation

25. THE ENTEROLISATION OF STOMACH MUCOSA IS
Hyperplasia of epithelium
Aplasia of epithelium
+Metaplasia of epithelium
Hypertrophy of epithelium
Atrophy of epithelium

26. THE ULCERAL-DESTRUCTIVE COMPLICATION OF CHRONIC STOMACH ULCERIS
Gastritis
+Pylorostenosis
Perforation
Malignesation
Penetration

27. FACTOR PROVIDED APPENDICITIS DEVELOPMENT IS
+Enterogenic autoinfection
Thrombosis of mesenteric arteries
Hemodynamic disorders
Splenomegaly
Epithelial metaplasia

28. “SHNIZLER’S METASTASIES” OF STOMACH CANCER ARE FOUND OUT IN
Lymph nodes of curvature minor
Ovary
Supraclavicular lymph nodes
+Pararectal lymph nodes
Parabronchial lymph nodes

29. THE TYPES OF ESOPHAGEAL DIVERTICULI ARE
+True, muscle
True, false
Exophitic, endophitic
Cylindrical, saccular
Tonogenic, myogenic

30. PRECANCEROUS DISEASE OF RECTUM IS
Hemorrhoid
Chronic stomach ulcer
Coprolythiasis
+Adenomatous polyps
Shigellosis

31. COLORECTAL CARCINOMA IS ASSOCIATED WITH
High fiber& low fat intake
High fat& high fiber intake
+Low fiber& high fat intake
Smoked fish
Salting fish

32. PREMALIGNANT CONDITION OF THE GIT IS
+Familial polyposis
Ileocecal tuberculosis
Shigellosis
Salmonellosis
Actynomycosis

33. COMMONEST VARIETY OF STOMACH CARCINOMA IS
Squamous carcinoma
+Adenocarcinoma

Colloid carcinoma
Mucoid carcinoma
Fibrinous carcinoma

34. GREATER RISK OF STOMACH CARCINOMA IS ASSOCIATED WITH
Old age
+Intestinal metaplasia
Acute gastritis
Vomiting
Dyspepsia

35. THE TYPES OF ESOPHAGEAL DIVERTICULI ARE
Exophitic, endophitic
+Single, plural
True, false
Cylindrical, saccular
Tonogenic, myogenic

36. CHRONIC GASTIRITIS IS CAUSED BY
+H. Pylori
Contaminated food
Hot water
Spices
Fatty food

37. CHRONIC GASTIRITIS IS CAUSED BY
Contaminated food
Hot water
Spices
+Overuse of salicylates
Fatty food

38. “BECKON” SPLEEN IS SEEN IN
Alcoholic hepatitis
Chronic active hepatitis
Focal amyloidosis
+Diffuse amyloidosis
Atherosclerosis

39. PEPTIC ULCER MORE OFTEN OCCURS IN
Upper part of esophagus
Middle part of esophagus
First part of jejunum
+Lesser curvature of stomach
Lower end of ileuum

40. MOST COMMON TYPE OF GASTRIC POLYP IS
+Hyperplastic polyp
Hamartomatous polyp
Malignant polyp
Familial polyposis
Inflammatory polyps

41. ALMOST NEVER MALIGNEZATING STOMACH POLYPS ARE
Tubular adenoma
Villous adenoma
Multiple polyposis
+Hyperplastic polyps
Dysplastic polyps

42. SECRETORY DIARRHEA IS CAUSED BY
Pancreatic exocrine deficiency
Esophageal polyp
+Gastrinoma
Lipoma
Dehydration

43. PATHOGENETIC TYPES OF CHRONIC GASTRITIS
Primary, secondary, reflux-gastritis
Exogenic, endogenic
Deep, superficial, atrophic
+Type A, type B, type C
Antral, cardial, pyloric

44. CHRONIC INFECTION OF THE GASTRIC MUCOSA IS ASSOCIATED WITH
Escherichia coli
+Helicobacter pylori
Campilobacter jejuni
Enterococcus falcium
Escherichia aurescens

45. CHRONIC GASTRITIS MAY BE CHARACTERIZEDBY
+Lympho-plasmocyte infiltration
Suppurative inflammation
Leucocytic-necrotic infiltration
Intestinal dysplasia
Atrophy of pancreas

46. MORPHOLOGICCHARACTERISTIC OF ACUTE CATARRHAL GASTRITIS IS
Atrophy of the mucosa
General venous congestion
+Moderate edema of the lamina propria
Lympho-plasmocytary infiltration
Metaplasia of gastric epithelium

47. THE MAJOR ETIOLOGIC ASSOCIATIONOF CHRONIC GASTRITIS IS
Ischemia and shock
+Chronic Helicobacter pylori infection
Obesity
Hereditary factors
Constitutional factors

48. SPECIAL FORMOF GASTRITISIS

Interstitial gastritis
Metaplastic gastritis
Dysplastic gastritis
Neutrophilic gastritis
+Eosinophilic gastritis

49.CLINICAL SYNDROME ASSOCIATED WITH GASTRIC, DUODENAL AND JEJUNAL PEPTIC GASTRIN-INDUCED ULCERS IS
Horner's
Nephrotic
DIC
+Zollinger-Ellison
Malabsorption

50. PREDISPOSING CONDITION FOR ISCHEMIC BOWEL DISEASE IS
Helicobacter pylori infection
Arteriolar dilation
Arterial hypertension syndrome
+Portal hypertension syndrome
Venous twisting

51.GIANT CEREBRIFORM ENLARGEMENT OF THE GASTRIC MUCOSA IN MENETRIERDISEASE IS CAUSED BY
Atrophy of the mucosa
Acute inflammation
+Hyperplasia of the mucosal epithelial cells
Interstitial metaplasia
Fibrosis

52. COMPLICATIONS OF DUODENAL PEPTIC ULCER INCLUDE
+ Perforational bleeding
Malabsorption
Fragmentation
Obesity
Diabetes

53. THE MOST COMMON LOCALIZATION OF GASTRIC PEPTIC ULCER IS
Greater curvature-
+Lesser curvature
Duodenal-pyloric ring
Anterior wall of the gastric corpus
Posterior wall of the gastric corpus

54. MACROSCOPIC CHARACTERISTIC OF CLASSICAL PEPTIC ULCER IS
Quadrate deep defect
Superficial defect
Tumor-like red
Superficial defect with exudation
+Punched-out defect with elevated margins

55. THE MOST COMMON PATHOLOGYOF GASTRIC MUCOSA ASSOCIATED WITH PEPTIC ULCER IS

Cancerous ulcer
Hypertrophic gastropathy
Gastric dilatation
Menetrier disease
+Chronic gastritis

56. COMPLICATION OF CHRONIC PEPTIC ULCER IS
+Malignization
Caseation
Pleuritis
Enterocolitis
Hemorrhoy

57. MICROSCOPICAL FEATURE OF ACUTE APPENDICITIS IS
Epithelioid cell infiltration
+Abscesses formation
Granuloma formation
Polypus overgrowth
Hydrocele

58. MICROSCOPICAL FEATURE OF ACUTE APPENDICITIS IS
Epithelioid cell infiltration
+Ulceration of the mucosa
Granuloma formation
Polypus overgrowth
Hydrocele

59. MICROSCOPICAL FEATURE OF ACUTE APPENDICITIS IS
Epithelioid cell infiltration
Granuloma formation
Polypus overgrowth
Hydrocele
+Foci of hemorrhages and edema

60. MACROSCOPIC CHARACTERISTIC OF APPENDIX IN ACUTE APPENDICITIS IS
Firm and indurate
Hydrocele
Mucocele
Multychamber
+Swollen with pus inside

61. MACROSCOPIC CHARACTERISTIC OF APPENDIX IN ACUTE APPENDICITIS IS
Firm and indurate
Hydrocele
Mucocele
Multychamber
+Hyperemic with engorged vessels

62. MACROSCOPIC CHARACTERISTIC OF APPENDIX IN ACUTE APPENDICITIS IS
Firm and indurate
+ Thickened fibrin covered red serosa
Hydrocele

Mucocele
Multychamber

63. POSSIBLE COMPLICATIONS OF DESTRUCTIVE APPENDICITIS ARE
Mummification
+Empyema of appendix and periappendicitis
Hydrocele
Empyema of pleura
Gangrene of small intestine

64. POSSIBLE COMPLICATIONS OF DESTRUCTIVE APPENDICITIS ARE
+Self-amputation of appendix
Hydrocele
Empyema of pleura
Gangrene of small intestine
Mummification

65. ETIOLOGICAL FACTOR OF ESOPHAGITIS IS
Chronic gastritis
Chemical reactions in stomach
+Chemical burn
Stenosis of aorta
Urination

66. ETIOLOGICAL FACTOR OF ESOPHAGITIS IS
Chronic gastritis
Chemical reactions in stomach
Stenosis of aorta
+ Stenosis of esophagus
Urination

67. ETIOLOGICAL FACTOR OF ESOPHAGITIS IS
Chronic gastritis
Chemical reactions in stomach
Stenosis of aorta
+Uremia
Urination

68. PRECANCEROUS DISEASE OF RECTUM IS
Hemorrhoid
Chronic stomach ulcer
Coprolythiasis
+Black acanthosis
Shigellosis

69. CHRONIC GASTRITIS MAY BE CHARACTERIZED BY
Leucocyte infiltration
Suppurative inflammation
+Intestinal metaplasia and atrophy of mucoca

70. FACTORS PROVIDED APPENDICITIS DEVELOPMENT ARE
Thrombosis of mesenteric arteries

Hemodynamic disorders
Splenomegaly
+ Hemodynamic disorders of appendix wall
Epithelial metaplasia

71. COMPLICATION OF CHRONIC PEPTIC ULCER IS
Caseation
+Perforation
Obliteration
Blood congestion
Fragmentation

72. COMPLICATION OF CHRONIC PEPTIC ULCER IS
+ Bleeding
Malformation
Caseation
Metastasizing
Coagulation

73. COMPLICATION OF CHRONIC PEPTIC ULCER IS
Malformation
Caseation
+Penetration
Coagulation
Fragmentation

74. MORPHOLOGICCHARACTERISTIC OF ACUTE CATARRHAL GASTRITIS IS
Atrophy of the mucosa
+Vascular congestion of the lamina propria with neutrophil infiltration
General venous congestion
Lympho-plasmocytary infiltration
Metaplasia of gastric epithelium

75. MORPHOLOGICCHARACTERISTIC OF ACUTE CATARRHAL GASTRITIS IS
Atrophy of the mucosa
+ Abundant mucus on the gastric epithelium
General venous congestion
Lympho-plasmocytary infiltration
Metaplasia of gastric epithelium

76. THE MAJOR ETIOLOGIC ASSOCIATIONOF CHRONIC GASTRITIS IS
Ischemia and shock
+ Autoimmune factors
Obesity
Hereditary factors
Constitutional factors

77. THE MAJOR ETIOLOGIC ASSOCIATIONOF CHRONIC GASTRITIS IS
Ischemia and shock
+ Toxic factors
Hereditary factors
Constitutional factors

Obesity

78. THE MAJOR ETIOLOGIC ASSOCIATIONOF CHRONIC GASTRITIS IS
Ischemia and shock
Hereditary factors
Constitutional factors
+Bile reflux
Reflux-esophagitis

79. SPECIAL FORM OF GASTRITISIS
Neutrophylic gastritis
Metaplastic gastritis
+Lymphocytic gastritis
Caseous gastritis
Interstitial gastritis

80. SPECIAL FORMS OF GASTRITIS ARE ALL, EXCEPT:
+Granulomatous gastritis
Caseous gastritis
Interstitial gastritis
Neutrophylic gastritis
Metaplastic gastritis

11. LIVER PATHOLOGY

1. THE COMMON OUTCOME OF ACUTE VIRAL HEPATITIS "A" IS
Postnecrotic cirrhosis of the liver
Carrier state formation
Portal cirrhosis
Chronic hepatitis
+Recovery

2. DUE TO CHRONIC CHOLESTASIS COLOR OF THE LIVER BECOMES
Grayish
Yellowish
+Greenish
Brown
Red

3. THE TOXIC DYSTROPHY OF THE LIVER CAN DEVELOP AT
Leukemia
+Gestational toxicosis
Dysentery
Cardiac insufficiency
Typhoid fever

4.CLINICAL FORM OF VIRAL HEPATITIS RESULTING IN LIVER NECROSIS IS
Anicteric
Acute cyclic
Chronic
Cholestatic
+Fulminant

5. THE CLINICAL-MORPHOLOGICAL FORMS OF ACUTE VIRAL HEPATITIS ARE:
Hepatomegalic, splenomegalic
+Cyclic icteric, anicteric
Dystrophic, hypertrophyc
Hypoproteinemic, hyperproteinemic
Obstructive, hydropic

6. FATTY HEPATOSIS MOST COMMONLY IS A RESULT OF
Glycogenosis
Viral hepatitis
Essential hypertension
Lung cancer
+Chronic alcoholism

7. IN STAGE OF YELLOW DYSTROPHY THE LIVER IS
Red, reduced
+Yellow, increased
Red, increased
Brown, sclerotic
Diffuse hemorrhages in liver tissue

8. MACROSCOPIC CHARACTERISTIC OF ALCOHOLIC (PORTAL) CIRRHOSIS OF LIVER IS
Macronodular surface of liver
+Micronodular surface of liver
Expansion of bilious channels
Nerrow fibrous fields between lobules
Smooth yellow surface

9. HISTOLOGIC STAIN FOR REVEALING OF LIVER CIRRHOSIS IS
Sudan III
+Picrofuchsin by von Giesone
Shiff-reaction
Perl's reaction
By Ziehl-Nielsen

10.POSSIBLE OUTCOME OF ACUTE VIRAL HEPATITIS IS
Nutmeg liver
Blue atrophy of liver
Glycogenosis of liver
+Cirrhosis of liver
Hemosiderosis of liver

11.THE SYNDROME OF PORTAL HYPERTENSION IS CHARACTERIZED BY
Pulmonary embolism
Syndrome of compression of superior vena cava
+Ascites, expansion of esophagus veins, splenomegaly
Ischemia of mesenteric vessels
Thrombosis of mesenteric vessels

12.THE FIGURATIVE NAME OF LIVER AT STEATOSIS IS
+"Goose"

"Tiger"
"Grease"
"Sago"
“Nutmeg”

13.OCCURRENCE OF INFLAMMATORY INFILTRATE IN THE LIVER MEANS
Regeneration
Cirrhosis
Hepatoma
Hepatosis
+Hepatitis

14.THE MOST OFTEN REASON OF TOXIC DYSTROPHY OF THE LIVER IS
Brain hemorrhage
+Poisoning
Stomach ulcer
Diabetes
Hypertension

15.MICROSCOPIC CHARACTERISTICS OF POSTNECROTIC LIVER CIRRHOSIS ARE
+False lobules, wide fibrous layers
Hemosiderosis, hemochmatosis
Amyloidosis, hyalinosis
Diffuse hemorrhages in liver tissue
Dystrophy, necrosis of hepatocytes

16. VIRAL HEPATITIS “A” IS CHARACTERIZED BY
Development of cirrhosis of liver
Malignant current
Parenteral way of transmission
+Low mortality, lifelong immunity
High mortality, absence of immunity

17.NUTMEG LIVER DEVELOPS AT
Chronic alcoholism
Hepatitis;
Cancer of liver
+The general venous congestion
Hydatid cyst

18.VIRAL HEPATITIS “B” IS CHARACTERIZED BY
The fine sizes of virus with defective RNA
+Long persistence virus in host organism
Oral-fecal way of transmission
Transmissive pathway
Complete recovery

19. FATTY HEPATOSIS DEVELOPS AT
Appendicitis
+Diabetes
Chollangitis
Hypertension

Chronic pyelonephritis

20.THE MOST OFTEN REASON OF DEATH IN CIRRHOSIS OF LIVER IS
Acute cardiac insufficiency
+Bleeding from esophagus varicose veins
Pneumonia
Respiratory-cardiac insufficiency
Cachexy

21. VIRAL HEPATITIS “C” IS CHARACTERIZED BY
Low frequency of development of liver cirrhosis
Transmissive pathway
+High frequency of progressing to chronic
Long-life effective immunity
Oral-fecal way of transmission

22.THE MASSIVE BLEEDING IS OBSERVED AT
Viral hepatitis
Billiary cirrhosis
+Nutmeg cirrhosis
Gallstones
Abscess of liver

23.IN AN OUTCOME OF PROGRESSING MASSIVE LIVER NECROSIS DEVELOPS
+Postnecrotic cirrhosis
Portal cirrhosis
Mixed cirrhosis
Biliary cirrhosis
Nutmeg cirrhosis

24.VIRAL HEPATITIS “D” IS CHARACTERIZED BY
Following with hepato-cellular carcinoma
High frequency of progressing to chronic
Meets at housewifes
Oral-fecal way of transmission
+Transforms hepatitis B in fulminant form

25.YELLOW COLOR OF SKIN, SCLERA, SEROUS AND MUCOUS MEMBRANES AS A RESULT OF INCREASED LEVEL OF BILIRUBIN IN BLOOD IS:
Melanosis
Vitiligo
+Jaundice
Cyanosis
Albinism

26.PERIPHERAL EDEMA IS OBSERVED AT
Focal cirrhosis of liver
Postnecrotic cirrhosis of liver
Biliary cirrhosis of liver
+Nutmeg cirrhosis of liver
Cryptogenic cirrhosis of liver

27. IN CIRRHOSIS OF LIVER IS SEEN
Fatty infiltration
+Loss of normal architecture
Regeneration of hepatocytes
New complications of lobules
Smooth surface of liver

28.POSTNECROTIC LIVER CIRRHOSIS IS A RESULT OF
+Toxic dystrophy of liver
Acute hepatitis “A”
Cholecystolythiasis
The cyclic form of viral hepatitis
Anicteric forms of viral hepatitis

29.SECONDARY BILIARY CIRRHOSIS OF LIVER DEVELOPS IN OUTCOME OF
+Cholelythiasis with chronic cholestasis
Toxic dystrophy of liver
Non purulent cholangitis
Acute viral hepatitis “A”
Chronic persistent hepatitis

30. MACROSCOPIC CHARACTERISTIC OF VIRAL (POSTNECROTIC) CIRRHOSIS OF LIVE IS
+Macronodular surface of liver
Micronodular surface of liver
Narrow fibrous band between lobules
Nutmeg liver
Smooth surface of a liver

31. THE FORM OF LIVER CIRRHOSIS WITH ESPECIALLY HIGH RISK OF HEPATO-CELLULAR CARCINOMA DEVELOPMENT IS
Alcohol
+After hepatitis C
Primary biliary
Secondary biliary
Dyscirculatory

32. LARGE LIPID DROPLETS(MACROVESICULAR STEATOSIS) MAY OBSERV IN HEPATOCYTES AT
Hepatitis B
Herpes II infection
Thrombosis
+Obesity
Diabetes insipitus

33. CIRRHOSIS OF LIVER IS CHARACTERIZED BY
Cellular atypia
Regenerative foci in liver
Proliferation of hepatocytes
Restoration of liver tissue architecture
+False lobules with fibrosis

34. HISTOLOGICAL ATTRIBUTE OF VIRAL HEPATITIS “B” IS

Steatosis of hepatocytes
Plethora of the central veins
+Matte – glassy hepatocytes
Huge multinuclear hepatocytes
Light Kraevsky' cells

35. ONE OF THE BASIC HISTOLOGICAL ATTRIBUTES OF VIRAL HEPATITIS IS
+Caunsilman's corpuscles
Giant mitochondrion
Granulomatous inflammation
Pericellular fibrous
Sclerosis

36. MORPHOLOGIC FEATURE OF LIVER CIRRHOSIS IS
Hemosiderin granules in liver cells
+Parenchymal nodular architecture
Keeping of main liver architecture
Average vascular architecture
Replication of liver cells

37. MICRONODULAR CIRRHOSIS IS SEEN IN
+Alcoholic cirrhosis
Wilson's disease
Budd-Chyary syndrome
Post necrotic cirrhosis
Cholecystolythiasis

38. PATHOLOGICAL CHANGE OF LIVER CELLS IN ACUTE VIRAL HEPATITIS IS
Fibrinoid necrosis
+Ballooning degeneration
Fibrinoid degeneration
Mucoid degeneration
Hyalinosis

39. GALL- STONES IN HEMOLYTIC ANEMIA ARE
+Pigmentary
Mixed
Cholesterol
Phosphates
Urates

40. A PERSON IS LABELLED AS HEPATITIS CARRIER IF HBS AG IS POSITIVE AFTER
2 weeks
2 months
4 months
+6 months
12 month

41. IN INDIA ACUTE HEPATITIS "A" MOSTLY AFFECTS
Elderly diabetics
+Children between ages 3 and 12 years
Pregnant women in 3rd trimester

New born infants
Male population

42. MALLORY HYALINE IS FOUND IN
Chronic active hepatitis
Pleurites
Peritonitis
+Alcoholic cirrhosis
Secondary biliary cirrhosis

43. MALLORY HYALINE BODIES ARE PRESENT IN
Secondary biliary cirrhosis
Pleuritis
Peritonitis
+Indian childhood cirrhosis
Chronic active hepatitis

44. LARGE GIANT CELLS ARE FOUND IN
Alcoholic hepatitis
+Neonatal hepatitis
Serum hepatitis
Amoebic hepatitis
Peritonitis

45. MACRONODULAR CIRRHOSIS OCCURS IN
+Postnecrotic
Willsons disease
Cryptogenic
Alcohol
Toxic

46. PERIPORTAL FATTY INFILTRATION OF LIVER IS SEEN WITH
Alcoholism
Viral hepatitis
+Malnutrition
Tetracycline
Toxic

47. IN CIRRHOSIS OF LIVER IS SEEN
Fatty infiltration
Normal architecture
+ Loss of inter cellular connective tissue matrix
Replication of hepatocytes
New threads formation

48. HBV IS ASSOCIATED WITH
Cholangio carcinoma
Acute hepatitis
Stomach adenjcarcinjma
+Chronic persistent hepatitis
Pancreocirrhosis

49. THE FECAL-ORALLY TRANSMITTED IS SEEN IN
+Hepatitis A
Hepatitis B
Hepatitis C
Hepatitis D
Hepatitis F

50. NUTMEG LIVER IS APPEARANCE OF LIVER IN
Cirrhosis of liver
Hepatoma
Secondary carcinomatous deposit in liver
+Chronic passive congestion in liver
Fatty dystrophy

51. ENCEPHALOPATHY OF PREGNANT LADY CAN CAUSE HEPATITIS
+Acute fatty liver of pregnancy
Fulminant from of HBV
HVA
Fatty dystrophy of liver
Cirrhosis

52. HYPOGONADISM IN CIRRHOSIS IS DUE TO
Increased testosterone
Decreased estrogen due to decreased catabolism
Decreased peripheral conversion of androgens into estrogen
+Direct effect of alcohol on testes
Fatty hepatosis

53. HISTOPATHOLOGIC FEATURE OF BILE DUCT OBSTRUCTION IS
Billirubinemia
Pneumofibrosis
Proliferation of hepatocytes
+Cholestasis
Hypoproteinenia

54. NECROSIS IS SEEN IN ANOXIA OF LIVER
+Centrilobular
In the periphery
Around the hepatic vein
Around the bile duct
Around the artery

55. FEATURE OF ALCOHOLIC LIVER DISEASE
Hyalinosis of capsule
Petrification
Replication of hepatocytes
Hemangioma
+Mallory bodies seen

56. INCREASED LIVER ATTENUATION WITH INTRACELLULAR INFILTRATION IS FEATURE OF

+Fatty liver
Amyloidosis
Hemochromatosis
Hemosiderosis
Sago liver

57. MACRONUDULAR CIRRHOSIS IF ONCE NODULE DIAMETER IS MORE THEN
1 mm
5 mm
+1 sm
4 sm
5 sm

58. VIRAL HEPATITIS “B” IS CHARACTERIZED BY
The fine sizes of virus with defective RNA
+Transplacental pathway of transmission
Oral-fecal way of transmission
Transmissive pathway
Complete recovery

59. VIRAL HEPATITIS “B” IS CHARACTERIZED BY
The fine sizes of virus with defective RNA
+Carrier state formation
Oral-fecal way of transmission
Transmissive pathway
Complete recovery

60. VIRAL HEPATITIS “A” IS CHARACTERIZED BY
Development of cirrhosis of liver
Malignant current
+Benign current
Parenteral way of transmission
High mortality, absence of immunity

61. VIRAL HEPATITIS “A” IS CHARACTERIZED BY
Development of cirrhosis of liver
Malignant current
Parenteral way of transmission
+Oral-fecal way of transmission
High mortality, absence of immunity

62. VIRAL HEPATITIS “B” IS CHARACTERIZED BY
The fine sizes of virus with defective RNA
+Parenteral way of transmission
Oral-fecal way of transmission
Transmissive pathway
Complete recovery

63. PATHOLOGICAL CHANGE OF LIVER CELLS IN ACUTE VIRAL HEPATITIS IS
Fibrinoid necrosis
+ Fatty change
Fibrinoid degeneration

Mucoid degeneration
Hyalinosis

64. HISTOPATHOLOGIC FEATURE OF BILE DUCT OBSTRUCTION IS
Petrification
+Bile lakes
Pneumofibrosis
Hematoma
Regeneration of liver

65. HISTOPATHOLOGIC FEATURE OF BILE DUCT OBSTRUCTION IS
+Portal fibrosis
Petrification
Pneumofibrosis
Hematoma
Regeneration of liver

12. KIDNEYS PATHOLOGY

1. MICROSCOPIC CHARACTERISTIC OF ACUTE POSTSTREPTOCOCCAL GLOMERULONEPHRITIS
Presence of small podocytes processes
Intact membranous
Extracapillar hyaline formation
Amyloid deposition on membrane
+Intracapillary productive glomerulonephritis

2. CHRONIC PYELONEPHRITIS CAN BE CAUSED BY
Transfusion of incompatible blood
Poisoning by quicksilver
Smoking
+Stone in renal pelvis
Obesity

3. MICROSCOPIC FEATURE FOR DIFFERENTIATION CHRONIC PYELONEPHRITIS FROM INTERSTITIAL NEPHRITIS IS
Presence a lot of macrophages in infiltrate
Line-radial scarring
Fibrosis of intersticium
“Thyrioidisation” of kidney
+Sclerosis and mononuclear infiltration of pelvis and calices

4. THE COMPLICATION OF ACUTE PYELONEPHRITIS IS
+Papillonecrosis
Glomerulosclerosis
Hemosiderosis of kidney
Cyanotic induration
Ischemic infarction

5. TO TUBULOPATHIES REFERES
+Acute renal failure

Pylorostenosis
Glomerulonephritis
Chjiecystitis
Embolic nephritis

6. THE OUTCOME OF CHRONIC LONG-DURATING GLOMERULOPATHIES IS
Arteriolosclerosis
Dilatation and obstruction of tubules
+Nephrosclerosis
Hydronephrosis
Nephrolythiasis

7. THE MAIN CONDITION OF EPITHELIUM COMPLETE REGENERATION AT NECROTIC NEPHROSIS IS
Saving of single glomuli
+Undamaged of basal membrane
Evident lympho- plasmocytic infiltration
Presence of fibroblasts in stroma
Medium edema of stroma

8. ACUTE RENAL FAILURE CAN DEVELOP AT
Extravascular hemolysis
Gastric adenoma
Obstruction of bile tract
+ Shock
Atrophy of pancreas

9. GLOMERULONEPHRITIS IS DEFINED AS
+Infectious-allergic inflammation of renal glomeruli
Infectious inflammation of interstitial tissue, pelvis and calices of kidneys
Congenital defect with prevalence of canalicular epithelium damage
Dystrophy and necrosis of tubular epithelium
Infectious-allergic inflammation of renal tubules

10. MACROSCOPICAL APPEARANCE OF KIDNEYS AT ACUTE GLOMERULONEPHRITIS ISPrimary reduced kidney
Big bacon kidney
Big white kidney
+Big motley kidney
Big waxy kidney

11. THE OUTCOME OF CHRONIC LONG-DURATING STROMAL DISEASES OF KIDNEY IS
Arteriolosclerosis
Amyloidosis
Hydronephrosis
+Nephrosclerosis
Pyonephrosis

12. THE MAIN MORPHOLOGICAL FEATURE OF ACUTE GLOMERULONEPHRITIS IS
+Interstitial infiltration by leucocytes
Dystrophic changes of tubular epithelium

Hypoemia of juxtamedullary area of kidney
Protein cylinders in tubules
Amyloidosis

13. SECONDARY-REDUCED KIDNEY IS DEVELOPED IN OUTCOME OF
Essential hypertension
+Chronic glomerulonephritis
Diabetes mellitus
Kidney infarction
Acute glomerulonephritis

14. ACUTE GLOMERULONEPHRITIS IS APPEARED AS
+Intracapillary productive
Mesangial
Mesangial proliferative
Extramedullary
Intramedullary

15. THE MOST OFTEN VARIANT OF KIDNEY'S AMYLOIDOSIS IS
Primary
Senile
+Secondary
Local
Hereditary

16. CHARACTERISTIC OF EXTRACAPILLARY GLOMERULONEPHRITIS IS
Inflammation of vessel loops and mesangium
Isolated inflammation of glomerular capsule
Inflammation of glomerular capsule and glomeruli
+Inflammation of vessel loops with spreading on glomerular capsule
Tubular necrosis

17. THE OUTCOME OF ACUTE GLOMERULONEPHRITIS IS
Amyloidosis
Chronic renal failure
Secondary-reduced kidney
Complete convalescence
+Progressing to chronic

18. THE ACUTE DIFFUSE GLOMERULONEPHRITIS DEVELOPS ON BACKGROUND OF
+Streptococcal infection
Staphylococcal infection
Viral infection
Pneumococcal infection
Toxoplasmosis

19. THE MORPHOLOGIC SUBSTRATE OF SUBACUTE EXTRACAPILLARY GLOMERULONEPHRITIS IS
Timorous growth
Proliferation of vascular endothelium
Deposition of amyloid in glomeruli
+Formation of fibroepithelial "demi lunes" in glomeruli

Accumulation of purulent exudates in glomeruli

20. AT ACUTE RENAL FAILURE IN KIDNEY IS MARKED
Plethora of cortex
+Ischemia of cortex
Ischemia of medullary layer
Plethora of medullary layer
Amyloid deposition

21. SUBACUTE GLOMERULONEPHRITIS IS APPEARED AS
Intracapillary productive
Mesangiocapillary
Mesangioproliferative
+Extracapillary productive
Intracapillary exudative

22. THE MAIN MORPHOLOGIC CHANGES AT ACUTE RENAL FAILURE ARE SEEN IN
Glomeruli
+Tubules
Vessels
Stroma
Capsule

23. PROGRESSION OF INTRA- AND EXTRACAPILLARY CHRONIC GLOMERULONEPHRITIS IS CONNECTED WITH
Acute inflammatory process in glomeruli
Reorganization of kidney tissue
Pyonephrosis
Increased proliferation of endothelial cells and mesangium
+Progressive sclerosis due to deposition of plasma proteins

24. THE STAGE OF NECROTIC NEPHROSIS IS
Latent
+Oligoanuria
Proteinuria
Edematous-hypotonic
Clinical appearance

25. THE MAIN MORPHOLOGICAL FEATURE OF ACUTE PYELONEPHRITIS IS
+Interstitial infiltration by leucocytes
Dystrophic changes of tubular epithelium
Hyperemia of juxtamedullary area of kidney
Hyalinosis of glomeruli
Amyloidosis of glomeruli

26. THE MOST CHARACTERISTIC MORPHOLOGIC FEATURE OF ACUTE GLOMERULONEPHRITIS IS
+Proliferation of glomerular cells
Fibrinoid necrosis
Evident thickness of capillary basal membrane
Necrosis of capillary loops
Hyalinosis of tubular epitheliem

27. PRIMARY-REDUCED KIDNEY DEVELOPS IN OUTCOME OF
+Essential hypertension
Chronic glomerulonephritis
Diabetes insipidus
Chronic pyelonephritis
Acute glomerulonephritis

28.CHARACTERISTIC OUTCOME FOR AMYLOIDOSIS IS
Reconvalescention
+Chronic renal failure
Malignisation
Formation of chronic pulmonary heart
Acute renal failure

29. DEPENDING ON CHARACTER OF EXUDATES EXTRACAPILLARY GLOMERULONEPHRITIS CAN BE
Putrefactive
Purulent
+Hemorrhagic
Mucinous
Fibrozating

30. THE TYPE OF GLOMERULONEPHRITIS DEPENDING ON DURATION IS
Active
Persistent
+Subacute
Fulminant
Aggressive

31. THE MAIN COMPLICATION OF ACUTE PYELONEPHRITIS IS
Pylephlebitic abscess of liver
Infarction of kidney
Amyloidosis of kidney
Peritonitis
+Pyonephrosis

32. BIG BACON KIDNEY IS RESULT OF
Necrosis of tubular epithelium
Proliferation of mesangium
+Diffuse deposition of amyloid
Proliferation of podocytes and nephrothelium
Subendothelial deposition of electron-dense sediments

33. CHARACTERISTIC OF NEPHRITIC SYNDROME IS
Hypolipidemia
Hyperproteinemia
+Proteinuria
Dehydratation
Hematuria

34. GLOMERULAR INJURY CAUSED BY CIRCULATING IMMUNE COMPLEXES OCCURS IN

Lung cancer
Sepsis
DIC syndrome
Hepatitis A
+Systemic lupus erythematosus

35. HYALINOSIS OF GLOMERULAR APPARATUS USUALLY REVEALS AT
Chronic pyelonephritis
Chronic gastritis
+Arteriolonephrosclerosis
Acute pyelonephritis
Acute hepatitis

36. GLOMERULONEPHRITIS REFERS TO
Type I hypersensitivity reaction
Type IV hypersensitivity reaction
+Type III hypersensitivity reaction
Type II hypersensitivity reaction
Immediate type hypersensitivity reaction

37. A FEATURE OF BENIGN HYPERTENSION IN KIDNEY IS
Fibrinoid necrosis
Cellular replication
Nephrolythiasis
+Hyaline arteriosclerosis
Amyloidosis

38. THE WORST PROGNOSIS FOR RENAL CELL CARCINOMA IS CONNECTED WITH
+Vascular invasion
Associated with hypercalcemia
Presence of hematuria
Size more than 5 cm
Stone formation

39. BILATERAL SYMMETRICAL REDUCED SMALL-GRANULATED KIDNEYS ARE SEEN IN
Nephrosclerosis due to atherosclerosis of renal arteries reduced
+Chronic glomerulonephritis
Final stage of renal disease
Chronic pyelonephritis
Acute pyelonephritis

40. CYLINDRICAL DILATATION OF RENAL TUBULES IS SEEN IN
+Polycystic disease of kidney
Medullary cystic disease
Wilms tumor
Lipoid nephrosis
Amyloidosis

41. LIPID CASTS ARE SEEN IN
Acute tubular necrosis
+Nephrotic syndrome
Cytomegalic infection disease

Amyloidosis
Atherosclerosis

42. BENIGN HYPERTENSION IS ASSOCIATED WITH
+Hyaline arteriolosclerosis
Mucoid necrosis
Basal ganglia fibrosis
Perivascular inflammation
Petrification

43. THICKENING OF BASEMENT MEMBRANE OF GLOMERULI IS SEEN IN
IgA nephropathy
+Membranous proliferative glomerulonephritis
Lipoid nephrosis
Post streptococcal glomerulonephritis
Acute pyelonephritis

44. SUB-EPITHELIAL HUMPS OF MEMBRANE ARE CHARACTERISTIC OF
Minimal change glomerulonephritis
Membranous glomerulonephritis
Membranous proliferative glomerulonephritis
+Post-streptococcal glomerulonephritis
Acute pyelonephritis

45. FOCAL GLOMERULONEPHRITIS IS CAUSED BY
Post streptococcalimmune complex
+Infective endocarditis
B-hemolytic streptococcus group A
Croupous pneumonia
Nephrolythiasis

46. COMPLICATION OF UREMIA IS
+Pericarditis
Panbronchitis
Bronchiectasis
Emphysema
Pancreatitis

47. COMPLICATION OF UREMIA IS
Panbronchitis
Bronchiectasis
Emphysema
+Plevritis
Pancreatitis

48. MASSIVE PROTEINURIA MAY BE ASSOCIATED WITH
Polycystic kidneys
Vicarious hypertrophy
+Amyloidosis
Hydrops of pregnant
Doubling of kidney

49. UNILATERAL SMOOTH REDUCED KIDNEY AND HYPERTENSION IS SEEN IN
+Stenosis of renal artery
Chronic glomerulonephritis
Renal cell carcinoma
Pyelonephritis
Polycystic kidneys

50. CRESCENTS ARE DERIVED FROM
+Epithelial cells + fibrin + macrophage
Mesangium + fibrin + macrophage
Tubule + mesangiaum + fibrin
Mesangiaum + fibrin
Tubule + macrophages

51. DISEASE THAT RECURS AFTER TRANSPLANTATION OF KIDNEY IS
Pyelonephritis
+Membranous proliferative glomerulonephritis
Systemic lupus erythematosus
Mesangial nephritis
Polycystic kidneys

52. CAUSE OF NEPHROCALCINOSIS IS
Glomerulonephritis
Hypoparathyroidism
Amyloidosis of kidney
+Hypercalcemia
Pyelonephritis

53. BILATERALLY ENLARGED KIDNEYS ARE SEEN IN
Chronic glomerulonephritis
Chronic pyelonephritis
Benign nephrosclerosis
+Amyloidosis
Neephrocirrhosis

54. NEPHROTIC SYNDROME IS CHARACTERIZED BY
Haematuria
Hyperproteinemia
Dehydration
+Proteinuria
Thrombosis

55. THE FACTOR MAY CAUS ACUTE PYELONEPHRITIS IS
Pregnancy
Nephrolithiasis
Catheterization of the bladder
Prostatic hypertrophy
+Septicemia

56. MICROSCOPIC CHARACTERISTIC OF ACUTE POSTSTREPTOCOCCAL GLOMERULONEPHRITIS
+Losing of small podocytes processes

Presence of small podocytes processes
Intact membranes
Extracapillar hyaline formation
Amyloid deposition on membraneIntracapillary exudative glomerulonephritis

57. MICROSCOPIC CHARACTERISTIC OF ACUTE POSTSTREPTOCOCCAL GLOMERULONEPHRITIS
Presence of small podocytes processes
Intact membranes
Extracapillar hyaline formation
Amyloid deposition on membrane
+Membranous transformation

58. MICROSCOPIC CHARACTERISTIC OF ACUTE POSTSTREPTOCOCCAL GLOMERULONEPHRITIS
Presence of small podocytes processes
Intact membranous
Extracapillary hyaline formation
Amyloid deposition on membrane
+Extracapillary productive glomerulonephritis (semi-loons formation)

59. ACUTE RENAL FAILURE CAN DEVELOP AT
+Intravascular hemolysis
Extravascular hemolysis
Gastric adenoma
Obstruction of bile tract
Atrophy of pancreas

60. ACUTE RENAL FAILURE CAN DEVELOP AT
+Obstruction of urinary tract
Extravascular hemolysis
Gastric adenoma
Obstruction of bile tract
Atrophy of pancreas

61. NEPHROTIC SYNDROME IS CHARACTERIZED BY
Extravascular hemolysis
Hyperproteinemia
Dehydration
Thrombosis
+Edema

62. NEPHROTIC SYNDROME IS CHARACTERIZED BY
Haematuria
Hyperproteinemia
Dehydration
Thrombosis
+Lipiduria

63. NEPHROTIC SYNDROME IS CHARACTERIZED BY
Haematuria
Hyperproteinemia

Dehydration
+Cylindruria
Thrombosis

64. CAUSE OF NEPHROCALCINOSIS IS
+Hyperparathyroidism
Amyloidosis of kidney
Hypocalcemia
Glomerulonephritis
Pyelonephritis

65. CAUSE OF NEPHROCALCINOSIS IS
Hypoparathyroidism
+Tuberculosis of kidney
Hypocalcemia
Glomerulonephritis
Pyelonephritis

13. ENDOCRINOLOGY

1. THE COMMON REASON OF ADDISON'S DISEASE AT ADRENALS DYSFUNCTION IS
Amyloidosis
Tuberculosis
Hypoplasia
Hyperplasia
+Tumor

2. GOITER IS
+Increasing of thyroid gland in size
Increasing of parathyroid glands
Decreasing of thyroid gland
Increasing of thymus
Increasing of thyroid gland in number

3. LOOSING OF TEETH MEANES AVITAMINOSIS
A
B1
B6
+C
D

4. THE PATIENTS WITH DIFFUSE TOXIC GOITER CAN DIE FROM
+Heart failure
Acute adrenal failure
Liver failure
Adiposity
Respiratory insufficiency

5. ITCENCO-KUSHING'S DISEASE IS CHARACTERIZED BY
Diffuse adiposity
+Hyperplasia of adrenal's cortex
Hypoplasia of adrenal's cortex

Hypotonia
Hyperfunction of ovary

6. HYPOVITAMINOSIS PP IS CHARACTERIZED BY
Blood coagulation disorder
+Hyperkeratosis and atrophy of skin
Loosing of teeth
Osteoporosis
Hemeralopia

7. THE TYPES OF THYROIDITIS DEPENDING ON CURRENT ARE
Acute, chronic
Primary, secondary
Acute, relapsing
+Acute, subacute, chronic
Simple, progressive, regressive

8. DUE TO NECROTIC FOCI IN HYPOPHYSIS DEVELOPS
Acromegaly
+Cerebro-hypophisal cachexia (Simmond's disease)
Gigantism
Nannism
Adipose-genital dystrophy

9. THE MORPHOLOGICAL APPEARANCE OF DIABETIC MICROANGIOPATHY IS
Atherosclerosis
Lipoidosis
+Hyalinosis
Thrombosis
Lipomatosis

10. THE CHARACTERISTIC CHANGES IN PANCREAS AT DIABETES ARE
+Lipomatosis and sclerosis
Fibrinoid necrosis
Hyalinosis of stroma and fibrosis
Suppuration
Amyloidosis

11. THE THYROID GLAND FUNCTION AT THYROTOXIC GOITER IS
+Increased
Unchanged
Decreased
Absent
Reduced

12. ADENOMA FROM EOSYNOPHILIC CELLS OF FRONT HYPOPHISAL PART IN ADULTS RESULTS IN
Gigantism
Diabetes insipid
+Acromegaly
Nanism
Obesity

13. THE POSSIBLE REASON OF DEATH AT DIABETES MELLITUS IS
Cancer of lung
Cachexia
Asphyxia
+Sepsis
Obesity

14. ADENOMA FROM BASOPHILIC CELLS OF ANTERIOR HYPOPHISAL PART RESULTS IN DEVELOPMENT OF
+Itsenko-Kushing disease
Diabetes incipit
Acromegaly
Adipose-genital dystrophy
Gigantism

15. CHILDREN WITH GOITER ARE SUFFERED FROM
Gigantism
+Cretinism
Acromegaly
Nannism
Diabetes mellitus

16. THE REASON OF ENDEMIC GOITER DEVELOPMENT IS
+Iodine deficiency
Iodine prevalence
Potassium prevalence
Hypercalcemia
Hypermelanosis

17. AT DIABETES MELLITUS THE CHANGES IN KIDNEY GLOMERULI ARE
+Hyalinosis and sclerosis
Dystrophy and necrosis
Atrophy
Hypertrophy
Hypotrophy

18. THE MAIN BIOCHEMICAL APPEARANCE OF HYPERPARATHYROIDISM IS
+Hypercalciuria and hyperphosphaturia
Increased level of sialic acid
Increased amount of proteins in urine
Uremia
Hyperuricemia
19. DUE TO NECROTIC FOCI IN HYPOPHYSIS OF CHILDREN DEVELOPS
Acromegaly
Cerebro-hypophisal (Simmonds's disease)
Gigantism
+Nanism
Addison's disease

20. THE DIABETES MELLITUS DEVELOPMENT IS CONNECTED WITH FUNCTIONAL DISORDER OF

Stromal elements
+Langerhans islands
Ductal calls
Blood vessels
Pancreatic capsule

21. DEPENDING ON FUNCTION OF THYROID GLAND THE GOITER CLASSIFIED ON
Fulminant
Wavy
Chronic
Intracanalicular
+Euthyroid

22. THE TYPE OF GOITER BY APPEARANCE IS
Dissiminative
Follicular
Arteriolar
Progressive
+Nodular

23. THE MORPHOLOGIC APPEARANCE OF DIABETIC MACROANGYOPATHY IS
Plasmorrhagia
+Atherosclerosis
Vasculitis
Calcinosis
Hyalinosis

24. THE CRANIOTABES DEVELOPMENT IS CHARACTERISTIC OF AVITAMINOSIS
A
B1
B6
C
+D

25. THE REASON OF SPORADIC GOITER IS
Iodine deficiency
Iodine prevalence
Potassium prevalence
Potassium deficiency
Action of goitergenic factors

26. THE MORPHOLOGIC APPEARANCE OF DIABETIC MICROANGIOPATHY IS
+Hyalinosis
Atherosclerosis
Vasculitis
Calcinosis
Necrosis

27. THE REASON OF PARATHYROIDISM IS
Endocrine glands dysfunction
+Adenoma of parathyroid gland
Hypoplasia of parathyroid apparatus

Thyroid gland adenoma
Thyrotoxicosis

28. HYPOVITAMINOSIS D IS CHARACTERIZED BY
Hemorrhagic syndrome
+Rickets
Keratomalacia
Disorder of hemopoiesis
Hyperkeratosis

29. HYPOVITAMINOSIS B_{12} AND FOLIC ACID IS CHARACTERIZED BY
Thrombohemorrhagic syndrome
Rickets
Keratomalacia
+Disorder of hemopoiesis and hemosiderosis
Hyperkeratosis and melanosis

30. HYPOVITAMINOSIS A IS CHARACTERIZED BY
Hemorrhagic syndrome
Rickets
+Keratosis
Disorder of hemopoiesis
Hemeralopia (day-blindness)

31. PHEOCHROMOCYTOMA ARISES FROM
Adrenal cortex
+Adrenal medulla
Adrenal capsule
Kidney parenchyma
Kidney stroma

32. COMMONEST CAUSE OF HYPERCALCEMIA IS
Parathyroid hypoplasia
Thyroid hyperplasia
+Parathyroid adenoma
Thyroid carcinoma
Hypoplasia of parathyroid apparatus

33. SERUM ANTIBODIES IN HASHIMOTO'S DISEASE ARE MAINLY AGAINST
Thyroid follicles
Thyroxin
+Thyroglobulin
Iodine
Stromal elements

34. MEDULLARY CARCINOMA OF THYROID IS ASSOCIATED WITH INCREASING
+Calcitonin
Thyroglobulin
T3
T4
Thyrothropin

35. DIABETES MELLITUS IS ASSOCIATED WITH
Urate nephropathy
Hyperuricemia
Hypouricemia
+Diffuse glomerulosclerosis
Kidney hypertrophy

36. CONDITION PREDISPOSE TO UROLITHIASISIS
+Gout
Sickle cell nephropathy
Hypoparathyroidism
Glomerulonephritis
Hematuria

37. THE MOST OFTEN CHANGES OF PANCREAS AT DIABETES
+Atrophy and sclerosis
Hypertrophy
Hyperplasia
Purulent inflammation
Necrosis

38. INSULIN-DEPENDENT DIABETES MELLITUS IS CHARACTERIZED BY
+Decreased blood insulin level
Normal blood insulin level
Hereditary character
Alkalosis
Normal or ncreased blood insulin level

39. THE CAUSES OF MORBIDITY AND DEATH FROM DIABETES ARE THE LATER COMPLICATIONS DEVELOPING IN
Liver
Brain
+Kidneys
Lymph vessels
Spleen

40. DIABETIC NEPHROPATHY CAN LEAD TO
Hydronephrosis
+Nephrosclerosis
Hematuria
Thromboembolism
Pylephlebitis

41. DEVELOPMENT OF DIABETES IS CONNECTED WITH DISORDER OF CELLS FUNCTION
Alpha
+Betta
Sigma
Gamma
Delta

42. THE CLINICAL MANIFESTATIONSOF HYPERTHYROIDISM IS

+Hypertrophy of myocardium.
Skin striate
Anasarca
Ptyalism
Gigantism

43. FUNCTION OF THYROID GLAND AT THYROTOXIC GOITE
+Increased
Unchanged
Decreased
Absent
Perverted

44. LONG EXISTENCE OF ENDEMIC GOITER AT ADULTS RESULTS IN
Gigantism
+Carcinoma
Cushing’s syndrome
Simond’s disease
Addison’s disease

45. POSTERIOR LOBE OF HYPOPHYSIS INJURY RESULTS IN
Itcenco-Kushing’s disease
+Diabetes insipid
Acromegaly
Myxedema
Adiposogenital dystrophy

46. SECONDARY ENDOCRINE HYPERTENSION IS FOUND IN
Addison's disease.
DIC - syndrome
Adenoma of kidney
+Pheochromocytoma
Crush syndrome

47. THE SKIN PIGMENTATION IN BRONZE DIABETES IS DUE TO ACCUMULATION OF
Hemosiderin
Lipofuscin
Melanin
+Both melanine &hemosiderin
Adrenochrom

48. THE POSSIBLE REASON OF DEATH AT DIABETES MELLITUS IS
+Uremia
Cancer of lung
Cachexia
Asphyxia
Obesity

49. SECONDARY ENDOCRINE HYPERTENSION IS FOUND IN
Addison's disease
DIC - syndrome
Adenoma of kidney

Crush syndrome .
+Cushing's syndrome

50. DEPENDING ON FUNCTION OF THYROID GLAND THE GOITER CLASSIFIED ON
Acute
Wavy
Chronic
Intracanalicular
+ Hyperthyroid

14. FEMALE GENITAL TRACT PATHOLOGY. PATHOLOGY OF PREGNANT.

1.MACROSCOPICAL CHARACTERISTIC OF UTERUS AT SEPTIC ENDOMETRITIS IS
Decreased, flabby
Petechial hemorrhages
Diphtheria pellicle on serous environment
Endometrial veins occluded by thrombi
+Suppuration of endometrium

2.THE MOST PROBABLE REASON OF SPONTANEOUS ABORTIONS IS
Cyst of corpus lutein in ovary
Aplasia of ovary
+Transferred earlier acute purulent endometritis
Impassability of fallopian tubs
Atherosclerosis

3.BIOLOGICAL FACTOR WITH GREATEST TERATOGENIOUS EFFECT IS
Bacteria
+Viruses
Parasites
Fungi
Rickettsia

4.AT UTEROGENOUS SEPSIS PRIMARY METASTATIC ABSCESSESAPPEAR IN
Liver
+Lung
Ovary
Brain
Kidney

5.CONDITION FOR ECTOPIC PREGNANCY DEVELOPMENT IS
Hyperplasia of tubs
Tumors of ovary
Hepatomegaly
+Salpingooforitis
Bronchiectasis

6.HISTOLOGICALCHARACTERISTIC OF GLANDULAR ENDOMETRIAL HYPERPLASIA IS
Distinct division of endometrium on compact and sponges layers
The expressed polymorphism of endometrial glandular epithelium

+Hyperplastic condition of uterus mucous membrane with attributes of glandular epithelium hyperactivity
Papillary proliferation of glandular epithelium
Suppuration of endometrium

7. THE DIAGNOSIS OF ATYPICAL ENDOMETRIAL HYPERPLASIA IS BASED ON
Expressed atrophy of glands in combination with increased proliferative activity of glandular epithelium in parts of glands
+Expressed proliferation of glands with change of their figure (" gland-into-gland ") and occurrence of papillary structures
Tumor polymorphism in single epithelial cells
Hormonal proliferation of glandular epithelium with thickness of endometrium
Cyclic desquamation of epithelium

8.DEVELOPMENT OF LACTATION MASTITIS IS PROMOTED BY
Viral infection
Increasing of immune protection of organism
Subinvolution of uterus
+Lactostasis
Teeth diseases

9.CAMBIAL FUNCTION IN CERVICAL PART OF UTERUS CERVIX IS CARRIED OUT BY
Stromal cells
Epithelial cells
+Reserve cells
Lymphocytes
Erythrocytes

10.HYDATID (VESICULAR) MOLE IS THE FORM OF
Toxicosis of pregnancy
+Trophoblastic diseases
Noncarrying pregnancy syndrome
Ectopic pregnancy
Abnormality of development

11. DISORDERS OF ESTROGENIC HORMONES SECRETION CAUSE IN ENDOMETRIUM
Secretory transformations of glandular epithelium
Focal plasma cellular perivascular infiltration
+Structure of endometrium according to proliferative phase
Atrophy of endometrium
Hypotrophy of endometrium

12. MACROSCOPICALLY UTERUS BODY CANCER CAN LOOK LIKE
Mushroom-like
Souse-like
Sausage-like
Flat growth
+Endophytic growth

13. CURETTAGE OF UTERINE CAVITY AT ECTOPIC PREGNANCY IS CHARACTERIZED BY PRESENCE OF

Normal endomertium in proliferative phase
Decidual tissue and chorionic villi
+Decidual tissue and absence of chorionic villi
Normal endomertium in secretion phase
Atrophic endometrium

14. MOST FREQUENTLY ENDOMETRIOSIS OF UTERUS CERVIX MEETS AFTER
+Abortions
Diathermic coagulation of uterus cervix
Gysterosalpingographia
Endometritis
Uterus duplex

15. VARIANT OF LEIOMYOMA DEPENDING ON LOCALIZATION IN UTERUS WALL IS
Nodular
Subtotal
+Submucosal
Transmural
Diffuse

16.SOURCE OF INFECTION AT POSTNATAL MASTITIS IS
Microbial flora from pharynx and nose of newborn
Focus of infection in child organism
Increased immunity of mother
+Infringement of sanitary-and-epidemiologic order
Alimentary factors

17.TRUE EROSION OF UTERUS CERVIX IS CHARACTERIZED AS
Increasing of cellular elements differentiation with the tendency to keratinization of squamous epithelium
+Destruction of epithelium with inflammatory infiltration of subjunctive tissue
Proliferation of reserve cells
Presence of endometrial glands in ectocervix
Atrophy of cervical epithelium

18.THE KRUKENBERG'S TUMOR IS
Tumor from stroma of sexual band
+Metastasis of stomach cancer in ovary
Metastasis of uterus cancer in ovary
Metastasis of lung cancer
Metastasis of stomach cancer in the liver

19.MORPHOLOGICAL FEATURES OF VULVACONDILOMA ARE
Papillomatosis, acantosis and hyperkeratosis
+Papillomatosis, acantosis, pararkeratosis and inflammation of stroma
Dysplasia of epithelium with hyperkeratosis
Metaplasia of epithelium
Hyperkeratosis, parakeratosis

20. THE MOST COMMON MORPHOLOGICAL VARIANT OF BREAST CANCER IS
+Invasive canalicular cancer
Invasive lobular cancer

Medullary cancer
Colloid cancer
Noninvasive canalicular cancer

21. MAJORITY OF ECTOPIC PREGNANCY CASES APPEAR IN
Ovary
Cervix
+Tubs
Abdominal
Thorax

22. THE MOST COMMON HISTOLOGICAL VARIANT OF ENDOMETRIAL CANCER IS
+Adenocarcinoma
Squamous cancer
Transition-cellular cancer
Light-cellular cancer
Choriocarcinoma

23.ENDOMETRIOSIS IS
Dishormonal hyperplasia of ectopic endometrium
+Presence of endometrial glands in abnormal location outside of uterus cavity
Benign growth of tissue morphologically and functionally similar to endometrium
Inflammation of endometrium
Atrophy of endometrium

24.TYPE OF TROPHOBLASTIC DISEASES IS
Endometrial hyperplasia
Tubulopathy
Glomerulopathy
Adenocarcinoma
+Choriocarcinoma

25. HISTOLOGIC FEATURES OF CHORIONEPITHELIOMA ARE
Presence of chorionic villi and growth of throphoblast
+Absence of chorionic villi and proliferation of throphoblast
Presence of Aryas-Stell's reaction in endometrial glands
Absence of endometrial decidual reaction
Presence of endometrial decidual reaction

26. BENIGN DYSPLASIA OF BREAST IS
+Mastopathy
Intracanalicular fibroadenoma
Pericanalicular fibroadenoma
Paget disease
Cystadenoma of breast

27.TYPE OF TROPHOBLASTIC DISEASES IS
Endometrial hyperplasia
Tubulopathy
Glomerulopathy
Teratoma
+Grapes (vesicular) mole

28.MACROSCOPICALLY VESICULAR MOLE LOOKS AS
Cyst cavity
Dense polycystic node
+Grapes-like congestions of numerous babbles
Spongy structure formation
Soft-elastic ball-like formation

29.DYSPLASIA OF UTERUS CERVIX MUCOSA IS
Presence of glandular structures in ectocervix
+Increased proliferation of ectocervix cellular elements without their tendencies to maturation
Increased differentiation of cellular elements with tendency of squamousepitheliumto keratinization
Replacement of squamoused epithelium on cylindrical
Regenerative proliferation

30.MACROSCOPICALLOOKING OF UTERUS CERVIX CANCER IS
Mushroom-like
Souse-like
Polyp on wide basis
Dark-red colored spongy tissue
+Exsophytic growth

31.ATTRIBUTE OF UTERINE PREGNANCY IN CURETTAGE IS
Division of endometrium on compact and spongy layers
Presence of great number of vessels
+Presence of trophoblast and decidual reaction
Absence chorionic villi
Vesicular transformation of trophoblast

32.INFLAMMATORY DISEASE OF UTERUS MUCOSA IS TERMED AS
Ectropion
+Endometritis
Salpingoophoritis
Glandular hyperplasia of endometrium
Fibroadenoma

33.CHARACTERISTIC OF DISHORMONAL CONDITIONS OF UTERUS MUCOSAIS
Presence of structures from one of menstrual cycle phases according to must observed in norm
Massive round-cellular infiltration of stroma
Neutrophil infiltration of stroma
Decidual reaction and trophoblast elements
+Attributes of hypertrophy and cystic changes of endometrial glands

34. THE MOST OFTEN REASON OF ENDOMETRITIS DEVELOPMENT IS
Disorder of blood circulation in uterus
Hormonal disorders
Disregeneration
Tumor growth
+Entry of infection in uterine cavity

35. THE CHARACTERISTIC OF SIMPLE GLANDULAR ENDOMETRIAL HYPERPLASIA IS
Division of endometrium on compact and spongiest layers

Expressed polymorphism of glandular epithelium
+Uterus mucous membrane hyperplasia with corkscrew- twisted glands
Presence of papillary proliferation in glandular epithelium
Presence of decidual reaction

36. MORPHOLOGIC CHARACTERISTIC OF VESICULAR MOLE IS
+Presence of much avascular vesicular villi
Absence of chorion villi
Proliferation of endometrial basal layer
Hyperplasia of endometrial glands
Atrophy of uterus mucosa

37. SIMPLE LEUCOPLACIA OF UTERUS CERVIX IS CHARACTERIZED BY
Presence of immature forms of epithelium
+The tendency of epithelium to hyperkeratosis
Presence of glandular structures
Presence of papillary structures
Presence ofcysts filled with slime

38. CONDITION LEADING TO ENDOMETRIAL HYPERPLASIA IS
Endometriosis
Uterus duplex
Uterus retroposition
+Polycystic ovarian disease
Antibiotic therapy

39.TRUE STATEMENT ABOUT LEIOMYOMA IS
Known as organospecific tumor
+Regress or calcify after castration or menopause
Couse of dishormonal condition
Result of dysontogenesis
Is found in 100% of reproductive women

40. ATTRIBUTE OF UTERINE PREGNANCY IN UTERINE CAVITY SCRAPE IS
+Decidual reaction
Presence a lot of vessels
Presence invasive trophoblast
Absence of chorion villi
Hyperplasia of endometrial basal layer

15. VIRAL & CHILDREN DISEASES

1. TO THE GROUP OF ARVI IS REFEREED
Chicken pox
Meningococcal infection
Shigellosis
+Flue
Measles

2. MORPHOLOGICAL CHANGES IN LUNGS AT HEAVY TOXIC TYPES OF FLUE IS
Foci of caseouse necrosis
Foci of purulent inflammation and panbronchitis

+Massive diapedeses hemorrhages
Vasculitis
Granulomatouse inflammation

3. RESPIRATORY SYNCYTIAL INFECTION AFFECTING
+Upper respiratory tract
Low respiratory tract
Urinary tract
Gastro-intestinal tract
Bile tract

4. THE MACROSCOPIC DAMAGE OF BRAIN AT VIRAL INFECTIONS IS
+Edema, swelling and hemorrhages
Cyst with rusty walls
Hydrocephalus
Dystrophy and necrosis
Purulent meningeal infiltration

5. THE MICROSCOPIC CHANGES IN LUNGS AT CROUPOUS PNEUMONIA ARE
Serous exudates in lumens of alveoli
Purulent exudates with formation of micro-abscesses
Athelectases
Hemosiderosis of septi
+Fibrinous-purulent exudates in alveolar space

6. THE MAIN PATHWAY OF FLUE TRANSMISSION IS
Alimentary
Parenteral
+Aero-droplet
Genital
Transmissive

7. THE VARIANT OF HERPES INFECTION OF CNS IS
Diffuse purulent meningitis
Purulent focal meningitis
+Acute necrotizing encephalitis
Fibrinous encephalitis
Purulent encephalitis

8. THE SYNONYM OF ACUTE INTERSTITIAL PNEUMONIA IS
Desquamate pneumonia
Acute bronchiolitis
+Acute fibrozating alveolitis
Obstructive bronchiolitis with carnificating pneumonia
Respiratory distress-syndrome of adults

9. THE PNEUMONIA CAUSED BY STAPHILLOCOCCUS AS A RULE IS
Catarrhal
Croupous
Serous-hemorrhagic
+Purulent
Interstitial

10. CHARACTER OF INFLAMMATION IN TRACHEA AND BRONCHI IN MODERATE TYPE OF FLUE IS
Catarrhal
+Serous-hemorrhagic
Purulent-hemorrhagic
Croupous
Diphtheric

11. PULMONARY COMPLICATIONS DEVELOPMENT AT HEAVY FLUECONNECT WITH
Specific pneumotropic of virus
+Connection of bacterial flora
Evident vasoparalitic action of virus
Athelectasis and respiratory insufficiency
Bronchial obstruction

12. THE MOST CHARACTERISTIC APPEARANCE OF TYPHUS FEVER IS
Enteritis
Colitis
+Myositis
Vasculitis
Bronchitis

13. VARIANT OF HEAVY FLUE IS
+Toxic
Neuropathic
With complications on heart
Hyperergic
Nephropathic

14. THE FAVORITE LOCALIZATION OF TYPHUS FEVER GRANULOMAS IS
+Liver
Spleen
CNS
Bone marrow
Kidney

15. CHARACTERISTIC OF LIGHT FLU
+Serous laringo-tracheitis
Serous-hemorrhagic pneumonia
Purulent panbronchitis
Productive pneumonitis
Serous nasopharingitis

16. THE FAVORIT PATHWAY OF HEPATITIS B VIRUS TRANSMISSION IS
Alimentary
Genital
Aerogenic
+Parenteral
Transmissive

17. TRANSMISSION OF TYPHUS IS PROVIDING BY

Ticks bite
+Louse feces
Mosquitoes bite
Fly bite
Animal's bite

18. THE SEVERE TOXIC FLUE IS CHARACTERIZED BY DEVELOPMENT OF
Pneumosclerosis
+Plural hemorrhages
Brain abscess
Spleen infarction
Fibrinouse pericarditis

19. TO GROUP OF ACUTE RESPIRATORY VIRAL INFECTIONS REFERES
Rubeola
Measles
+Adeno-viral infection
Typhus
Meningococcal infection

20. THE TYPE OF INFLAMMATION IN TRACHEA AT MODERATE FLUE IS
+Catarrhal
Serous-hemorrhagic
Purulent-hemorrhagic
Croupous
Diphtheric

21. WAY OF HIV TRANSMISSION IS
Alimentary
+Transplacentary
Urogenic
Aerogenic
Transmissive

22. TYFUS GRANULOMA BY AUTHOR IS NAMED AS
Virchov's
Miculitch
Berezovsky
+Popov's
Pirogov's

23. THE PATHWAY OF HAV IS
+Alimentary
Parenteral
Aerogenic
Genital
Transmissive

24. SECONDARY BACTERIAL SUPERINFECTION AT FLU IS LEAD TO
Inflammation become hemorrhagic
Development of extrapulmonary complications
Development of purulent meningitis and encephalitis

+Inflammation becomes purulent with massive destruction of pulmonary tissue
Development of hemorrhages and hemorrhagic infarctions

25. PROBABLE DIAGNOSIS AT THE PATIENT WITH LYMPHOPENIA AND PNEUMOCYSTIC PNEUMONIA IS
Sogren diseas
+Heavy combined immunodeficiency (HIV)
Gudpascher syndrome
Isolated deficiency IgA
AIDS

26. TO DNA HEPATITIS VIRUS REFERES
HAV
+HBV
HCV
HDV
HEV

27. HIV IS ASSOSIATED WITH
Rhinitis
+Pneumocystis pneumonia
Tracheobronchitis
Meningoencephalitis
Struma

28. BIG MOTLEY LUNG IS TYPICAL FOR
Viral hepatitis
+Flue
Herpes
Viral parotitis
Viral papillomatosis

29. MOST TYPICAL CHANGE IN TRACHEA AND LARGE BRONCHI AT TOXIC FLU IS
+Serous-hemorrhagic inflammation
Necrotic process
Granulematous inflammation
Purulent inflammation
Fibrinous inflammation

30. THE PATHWAY OF RABIES IS
Alimentary
+Sec animal bite
Aerogenic
Genital
Insect bite

31. INFLAMMATION OF RESPIRATORY TRACT MUCOSA AT UNCOMPLICATED MEASLES HAS CHARACTER OF
Purulent
Necrotic
Fibrinous
+Catarrhal

Hemorrhegic

32. MEASLES EXANTEMA IS FINISHED BY
Macrolamellar peeling
+Scaly peeling
Focal hyperpigmentation
Focal leicoderma
Complete disappearance

33. USUAL WAY OF INFECTION AT MEASLES IS:
Alimentary
Parenteral
+Air - drop
Hematogenic
Transmissive

34. AT MEASLES BELSKY -KOPLIC-FYLATOV'S SPOTS ARE FOUND OUT ON
Palms and stops
Extensor surfaces of forearm
Tongue
+Internal surface of cheeks
Head

35. BRONCHIAL INFLAMMATION AT COMPLICATED MEASLES HAS CHARACTER OF
Granulematous
+Purulent-necroyic
Catarrhal
Serous
Hemorrhagic

36. CHARACTER OF EXUDATES AT MENINGOCOCCAL MENINGITIS IS
Putrefactive
Hemorrhagic
Fibrinous
+Purulent
Fibrinous-hemorrhagic

37. USUAL COMPLICATION OF MENINGOCOCCAL MENINGITIS IS
Cyst of brain
Tumor of brain
Hemorrhagic infarction of brain
+Hydrocephalus
Glial scar

38. AT MENINGOCOCCAL MENINDITIS TYPICAL INFLAMMATION IS
Hemorrhagic
Catarrhal
Productive
+Purulent
Granulomatous

39. AT DIPHTHERIA INFLAMMATION HAS CHARACTER OF

Purulent
+Fibrinous
Productive
Hemarrhagic
Putrefactive

40. THE MOST SENSITIVE TO DIPHTERIC TOXIN ARE
+Adrenal glands
Lungs
Liver
Spleen
Intestine

41. RASH AT SCARLET FEVER IS
Macromacular
+Punctate
Roseola-papular
Vesicular
Hemorrhagic

42. TYPICAL LOCALIZATION OF LOCAL CHANGES AT SCARLET FEVER IS
+Mucosa of oral cavity
Skin
Conjunctive of an eye
Mucous of genital tracts
Mucous of esophagus

43. SCARLET FEVER IS CAUSED BY
Diplococcus
Escherichia coli
+Streptococcus of type A
Hemolytic streptococcus type B
Staphilococcus

44. IN REGIONAL LYMPHATIC NODES AT SCARLET FEVER DEVELOPS
+Necrosis
Anemia
Sclerosis
Hypoplasia
Atrophy
45. AT MEASLES BELSKY -KOPLIC-FYLATOV'S SPOTS ARE FOUND OUT ON
Extensor surfaces of forearm
Tongue
Internal surface of larynx
Head
+Oral cavity vestibular mucosa

46. AT MEASLES BELSKY -KOPLIC-FYLATOV'S SPOTS ARE FOUND OUT ON
Palms and stops
Tongue
+Oral mucosa opposite to premolars
Head

Tonsils

47. RASH AT MEASLES IS
+Macromacular
Punctate
Roseola-papular
Vesicular
Hemorrhagic

48. RASH AT MENINGOCOCCAL INFECTION IS
Macromacular
Punctate
Roseola-papular
Vesicular
+Hemorrhagic

49. RASH AT CHICKEN POX IS
Macromacular
Punctate
Roseola-papular
+Vesicular
Hemorrhagic

50. CLINICAL-MORPHOLOGICAL FORM OF MENINGOCOCCAL INFECTION IS
Laringo-tracheitis
+Naso-pharingitis
Tracheo-bronchitis
Gastro-enteritis
Entero-colitis

16. BACTERIAL INFECTIONS

1. FOR TYPHOID FEVER THE MOST TYPICAL IS
Fibrinous colitis
Diphteric inflammation of intestine
Ulceral colitis
+Medullary swelling of Peyer's patches with ulceration in ileum
Catarrhal enterocolitis

2. THE TERM "DYSENTERY" REFERS TO DIARRHEA ASSOCIATED WITH
"Rice-water" stools
Melena
Abdominal angina
+Abdominal cramping
Formatting stool

3. SHIGELLAE CAUSE IN ORGANISM
Acute tubular necrosis
Profuse diarrhea
Medullary swelling of Peyer's patches
+Damage of endothelial cells in the colon
Necrotic ulcers

4. THE INVASIVE PROPERTIE OF BACTERIA IN INTESTINAL INFECTION IS
Synthesize biologic active substance
+Adhere to the mucosal epithelial cells
Replicate epithelial cells
Elaborate vitamins
Protect mucosal epithelial cells

5. CHARACTERISTIC MORPHOLOGIC ATTRIBUTE OF CHOLERA IS
Follicular colitis
Medullary swelling of ileum group follicles
+Serous-hemorrhagic enteritis, gastritis
Fibrinous colitis
Ulceral colitis with purulent exudates

6. THE COMPLICATION OF BACTERIAL ENTEROCOLITIS SEPTIC FORM IS
Intestinal stenosis
Massive fluid accumulation (hydration)
Duplication of the intestinal mucosal barrier
Protection of the intestinal wall
+Generalization of infection (sepsis)

7. TYPHOID ULCER IS
Endocardial subacute ulcer
Unperforated ulcer
Undistracted ulcer
Stomach chronic ulcer
+Ulceration of the Peyer's patches

8. ULCERATION OF PEYER'S PATCHES OCCURS IN
Amoebiasis
Crohn's disease
+Salmonella
Clostridium
Shigella

9. THE COMPLICATION OF BACTERIAL ENTEROCOLITIS SEPTIC FORM IS
Intestinal stenosis
Massive fluid accumulation (hydration)
Duplication of the intestinal mucosal barrier
Protection of the intestinal wall
+ Destruction of the intestinal mucosal barrier

10. CHANGES IN PEYER'S PATCHES OF SMALL INTESTINE AT TYPHOID FEVER ARE
Fibrinous enteritis
Mucoid degeneration
+Medullary swelling
Murder swelling
Ulceral enteritis

11. THE TERM “DYSENTERY” REFERS TO DIARRHEA ASSOCIATED WITH

“Rice-water” stools
+Tenesmus
Melena
Formatting stool
Coprolythos

12. THE TERM “DYSENTERY” REFERS TO DIARRHEA ASSOCIATED WITH
“Rice-water” stools
Ascites
Melena
Formatting stool
+Stools containing pus and mucus

13. THE COMPLICATION OF SEPTIC FORM OF BACTERIAL ENTEROCOLITIS IS
Intestinal stenosis
+Massive fluid loss (dehydration)
Massive fluid accumulation (hydration)
Duplication of the intestinal mucosal barrier
Protection of the intestinal wall

14.THE TERM “DYSENTERY” REFERS TO DIARRHEA ASSOCIATED WITH
Melena
Formatting stool
Abdominal angina
“Rice-water” stools
+Ulceral colitis with purulent exudates

15. SHIGELLAE CAUSE IN ORGANISM
+Catarrhal-hemorrhagic colitis
Acute tubular necrosis
“Rice-water” stools
Necrosis of Peyer’s patches
Profuse diarrhea

16.SHIGELLAE CAUSE IN ORGANISM
Hemorrhagic enteritis
Damage and necrosis of lymphoid follicles
Acute tubular necrosis
+Stools containing blood, pus and mucus
“Rice-water” stools

17. THE INVASIVE PROPERTIE OF BACTERIA IN INTESTINAL INFECTION IS
Synthesize biologic active substance
+Replicate in the mucosal epithelial cells
Replicate epithelial cells
Elaborate vitamins
Protect mucosal epithelial cells

18. THE INVASIVE PROPERTIE OF BACTERIA IN INTESTINAL INFECTION IS
Replicate epithelial cells
Elaborate vitamins
Protect mucosal epithelial cells

Synthesize biologic active substance
+Elaborate enterotoxins

19. THE INVASIVE PROPERTIE OF BACTERIA IN INTESTINAL INFECTION IS
Replicate epithelial cells
Elaborate vitamins
Protect mucosal epithelial cells
Synthesize biologic active substance
+Invade mucosal epithelial cells

20. THE COMPLICATION OF SEPTIC FORM OF BACTERIAL ENTEROCOLITIS IS
Protection of the intestinal wall
Duplication of the intestinal mucosal barrier
+Perforation of the intestinal wall
Intestinal stenosis
Massive fluid accumulation (hydration)

17. TUBERCULOSIS. SYPHILIS.

1. MORPHOLOGIC APPEARANCE OF PRIMARY TUBERCULOSIS
Caseous pneumonia
+Primary tuberculous complex
Caseous lymphadenitis
Primary cavern
Primary affect

2. HUTCHINSON'S TRIAD IS CHARACTERISTIC FOR
Sepsis
Scarlet fevers
+Syphilis
Typhus
AIDS

3. INFECTION AGENT OF SYPHILIS IS
Shigella
Lamblia
Listeria
Corynebacterium
+Treponema

4. THE BASIC COMPONENT OF PRIMARY INFECTION COMPLEX AT SYPHILIS IS
Bubo
+Chancre
Vesicle
Carbuncle
Phlyctena

5. THE MOST OFTEN COMPLICATION OF SYPHILITIC MESAORTITIS IS
Ulceration
Sclerosis
+Aneurysm
Petrification

Plasmorrhagia

6. SYPHILITIC MESAORTITIS DEVELOPS IN
Primary syphilis
Secondary
+Tertiary
Early congenital
Late congenital

7. GHONS' FOCUS IS
The center of fibrosis
The center of caseous necrosis
The center of hemorrhage
+The center of petrificated primary affect
The center of encapsulated pneumonia

8. PRIMARY TUBERCULOSIS IS A RESULT OF
+Specific infection
Reinfection
Hematogenic dissemination
Toxicoinfection
Chronic nonspecific infection

9. TUBERCULOMA IS FORM OF
Primary pulmonary tuberculosis
Primary with hematogenic dissemination
Hematogenic pulmonary tuberculosis
Hematogenic tuberculosis of bones and joints
+Secondary pulmonary tuberculosis

10. TUBERCULOSIS IS CAUSED BY
Pneumococcus
Corynebacterium
+Mycobacterium
Blue pus bacillus
Streptococcus

11. FORM OF TUBERCULOSIS WITH MULTIPLE GRANULOMAS IN LUNGSIS
Tuberculous pneumonia
Brown induration of the lungs
+Miliary tuberculosis
Cavitary tuberculosis
Tuberculoma

12. TUBERCULIN TEST POSITIVISM INDICATES
Good humoral immunity
Mycobacterial infection
+Good cell-mediated immunity
Nothing
Immunodepression

13. ROUTE OF INFECTION IN TUBERCULAR PYELONEPHRITIS

Ascending
Descending
+Haemotogenic
Airogenic
Intracanalicular

14. PRIMARY COMPLEX AT TUBERCULOSIS MAY DIRECTLY TRANSFORM INTO
Cavitary tuberculosis
Progressive thrombosis
Pulmonary edema
+Fibro-petrificated scars
Latent mycoplasma infection

15. PRIMARY COMPLEX AT TUBERCULOSIS MAY DIRECTLY TRANSFORM INTO
Cavitary tuberculosis
Progressive thrombosis
+Progressive primary tuberculosis
Pulmonary edema
Latent mycoplasma infection

16. TARGET-ORGAN FOR MILIARY EXTRAPULMONARY TUBERCULOSIS SEEDING IS
Uterus
Eyes
Mucous membranes
+Kidneys
Skin
17. CALCIFIED FOCUS (FIBROPETRIFICATED SCAR) FORMING IN THE LUNG AFTER SECONDARY TUBERCULOSIS INFECTION IS TERMED AS
Keloid
Granuloma
Ghon’s focus
Aschoff-Pule focus
+Simon focus

18. THE MOST COMMON SITES OF SKELETAL TUBERCULOSIS INVOLVEMENT ARE
+Thoracic vertebrae
Skull bones
Neck vertebrae
Palmary bones
Foot bones

19. FORM OF TUBERCULOSIS MAY AFFECT THE INTESTINE IS
Secondary
Cavitary flbrocaseous
Dormant
+Primary
Miliary

20. PATHOLOGY OF VASA VASORUM AT SYPHILITIC MESAORTITIS TERMS AS
Migratory thrombophlebitis
+Productive vasculitis (obliterative endarteritis)
Thromboangitis obliterance

Necrotising arteriolitis
Thrombotic microangiophathy

21. THE TYPE OF IMMUNE RESPONSE INITIATED AT TUBERCULOSIS IS
Type I
+Type IV
Immediate type
Type II
Type III

22. THE TYPE OF NECROSIS IN CENTER OF TUBERCULOUS GRANULOMA IS
Coagulative
Liquefactive
+Caseous
Enzymatic fat
Fibrinoid

23. MILIARY TUBERCULOSIS IS ASSOCIATED WITH
Reinfection
Lung caseation
Lymph node caseation
Primary infection
Primary hematogenic tuberculosis

24. SECONDARY TUBERCULOSIS IS CHARACTERIZED BY
Ghon's thread in the lung
+Caseous necrosis and cavities in lungs
Ghon's focus
Henatogenic dissemination
Dystrophic calcification in primary affect

25. TUBERCULOUS SPONDILITIS WITH INVOLVEMENT OF INTERVERTEBRAL DISCS AND SOFT TISSUES WITH COLD ABSCESSES FORMATION IS KNOWN AS
Paget's disease
+Pott disease
Ghon's complex
Reinfection focus
Dormant disease

26. CASEATING DESTRUCTIVE SECONDARY TUBERCULOSIS INCLUDES
Miliary extrapulmonary lesions
Miliary pulmonary lesions
Lung hematogenic dissemination
+Lung caseation (tuberculoma)
Extrapulmonary caseation

27. SYNONIM OF SYPHILITIC GRANULOMA IS
Fibroma
+Gumma
Tuberculoma
Leproma
Hepatoma

28. MEDIAL DESTRUCTION OF AORTA AT TERTIARY SYPHILIS MAY LEAD TO
+Aneurismal dilation of aorta
Marfan's syndrome
Atherosclerotic aneurism
Takayasu arthritis
Giant cell arteritis

29. CHARACTERISTIC FEATURE OF TUBERCULOSIS MYCOBACTERIUMIS
+ Aerobic
Anaerobic
Pili-forming
Spore-forming
Motile

30. THE CAVITY AT CAVITARY TUBERCULOSIS IS CHARACTERIZED BY
Filled with purulent exudate
Localized in the low part of the lung
Lined by yellow-green pus
Walled by pyogenicmembrane
+Drained by bronchus

31. FORM OF TUBERCULOSIS MAY AFFECT THE LIVER IS
Secondary
Cavitary fibrocaseous
Dormant
Primary complex
+Miliary

32. POSSIBLE CAUSE OF TUBERCULOS INFLAMMATION CHRONICITY IS
Complete phagocytosis
Removing of certain microorganisms
Irresistance of etiologic agent
+Prolonged exposure to toxic agents
Acute expose of toxic agent

33. ON GROSS INSPECTION SYPHILITIC GUMMA IS CHARACTERIZED BY
Red-brown
Irregular defined
Soft
+Solitary,tumor-like
Multiply spots

34. CHARACTERISTIC TYPE OF INFLAMMATION IN MILIARY LUNG TUBERCULOSIS IS
Hemorrhagic
Fibrinous
+Granulomatous
Serous
Purulent

35. SEVERE DESTRUCTION OF VERTEBRAE AT TUBERCULOUS SPONDILITIS MAY RESULT IN

Drainage tract (sequester) formation
Open fractures
+Scoliotic deformations
Spiral deformities
Muscular defects

36. MACROSCOPIC CHARACTERISTIC OF TUBERCULOMA IS
Intraparenchymal single mass,several millimeters in diameter
Greyish-white, irregular-circumscribed
Well-circumscribed, brownish-red
Interstitial microfocal lesion
+Intraparenchymal single mass,several centimeters in diameter

37. TYPICAL SYPHILITIC GRANULOMA IS CHARACTERIZED BY
Neutrophil infiltrate
Area of central suppuration
Giant foreign-body cells presence
Area of central caseous necrosis
+Area of central gummous necrosis

38. HEALED LESIONS IN PRIMARY TUBERCULOSIS INCLUDE
Assmann's focus
+Ghon’s thread
Simon focus
Fibrocaseous cavitation
Dormant infection

39. MAIN PATHOLOGIC CONDITION FOR GROWTH AND MULTIPLICATION OF THE TUBERCULOUSE BACILLI IS
Lymphatic drainage obstruction
Progressive hypoxia
Increased blood perfusion
+Increased oxygen tension
Sludging of blood in alveolar capillaries

40. COMPLICATION OF TUBERCULOUS OSTEOMYELITIS IS
Sinus tract formation
+Cold abscess formation
Rheumatoid arthritis
Ghon’s thread in the lung
Ankyloses

41. TUBERCULOUS SALPINGITIS CAN BE FOUND IN TUBERCULOSIS
Secondary
Cavitary fibrocaseous
Dormant disease
+Miliary
Primary

42. GUMMOUS INFILTRATE AT TERTIARY SYPHILIS CAN BE FOUND IN
Aorta
Testes

+Liver
Bones and joints
Skin and subcutaneous tissue

43. TARGET-ORGAN FOR MILIARY EXTRAPULMONARY TUBERCULOSIS SEEDING IS
Uterus
Eyes
Mucous membranes
Skin
+Liver

44. TARGET-ORGAN FOR MILIARY EXTRAPULMONARY TUBERCULOSIS SEEDING IS
Uterus
Eyes
Mucous membranes
+ Bone marrow
Skin

45. TARGET-ORGAN FOR MILIARY EXTRAPULMONARY TUBERCULOSIS SEEDING IS
Uterus
Eyes
Mucous membranes
+Spleen
Skin

46. SECONDARY TUBERCULOSIS IS CHARACTERIZED BY
Ghon’s thread
Henatogenic dissemination
Dystrophic calcification in primary affect
Primary focus in the lung
+Reinfection focus

47. SECONDARY TUBERCULOSIS IS CHARACTERIZED BY
Primary focus in the lung
Ghon’s thread
Henatogenic dissemination
Dystrophic calcification in primary affect
+Reactivation of dormant disease

48. SECONDARY TUBERCULOSIS IS CHARACTERIZED BY
Primary focus in the lung
+Dystrophic calcified Simon foci
Ghon’s thread
Henatogenic dissemination
Dystrophic calcification in primary affect

49. CASEATING DESTRUCTIVE SECONDARY TUBERCULOSIS INCLUDES
Miliary extrapulmonary lesions
Miliary pulmonary lesions
Lung hematogenic dissemination
+Lung cavitation
Extrapulmonary caseation

50. CHARACTERISTIC FEATURE OF TUBERCULOSIS MYCOBACTERIUM IS
Anaerobic
Pili-forming
Spore-forming
Motile
+Red colored in acid-fast staining

51. CHARACTERISTIC OF THE CAVITY AT CAVITARY TUBERCULOSIS IS
Filled with purulent exudate
Localized in the low part of the lung
+Localized in the apex of the lung
Lined by yellow-green pus
Walled by necrotic tissue

52. CHARACTERISTIC OF THE CAVITY AT CAVITARY TUBERCULOSIS IS
Filled by purulent exudate
Localized in the low part of the lung
Lined by yellow-green pus
+Lined by yellow-grey caseous material
Walled by necrotic tissue

53. CHARACTERISTIC OF THE CAVITY AT CAVITARY TUBERCULOSIS IS
Filled by purulent exudate
Localized in the low part of the lung
Lined by yellow-green pus
Walled by necrotic tissue
+Walled by thick fibrous tissue

54. POSSIBLE CAUSE OF TUBERCULOS INFLAMMATION CHRONICITY IS
+Incomplete phagocytosis
Complete phagocytosis
Removing of certain microorganisms
Irresistance of etiologic agent
Acute expose of toxic agent

55. POSSIBLE CAUSE OF TUBERCULOS INFLAMMATION CHRONICITY IS
Complete phagocytosis
Removing of certain microorganisms
Irresistance of etiologic agent
+ Persistence of certain microorganisms
Acute expose of toxic agent

56. POSSIBLE CAUSE OF TUBERCULOS INFLAMMATION CHRONICITY IS
Complete phagocytosis
Removing of certain microorganisms
Iresistance of etiologic agent
+ Resistance of etiologic agent
Acute expose of toxic agent

57. ON GROSS INSPECTION SYPHILITIC GUMMA IS CHARACTERIZED BY
Red-brown

Irregular defined
Soft, warty-like
Multiply spots
+Hard, tumor-like

58. SEVERE DESTRUCTION OF VERTEBRAE AT TUBERCULOUS SPONDILITIS MAY RESULT IN
Drainage tract (sequester) formation
+Kyphotic deformities
Spiral deformities
Open fractures
Muscular defects

59. SEVERE DESTRUCTION OF VERTEBRAE AT TUBERCULOUS SPONDILITIS MAY RESULT IN
Drainage tract (sequester) formation
Open fractures
+ Permanent compression fractures
Spiral deformities
Muscular defects

60. TYPICAL SYPHILITIC GRANULOMA IS CHARACTERIZED BY
Neutrophil infiltrate
+Lymphocyte-plasma cell infiltrate
Giant foreign-body cells presence
Area of central caseous necrosis
Area of central suppuration

18. SEPSIS

1. TYPE OF SEPSIS THAT CHARACTERIZED BY ABSCESS DEVELOPMENT IS
Septicemia
+Septicopiemia
Chroniosepsis
Septic endocarditis
Cryptogenic sepsis

2. EMBOLIC PYOGENIC NEPHRITIS IS MOST COMMONLY CAUSED BY
Thromboembolism
Viral embolism
Foreign body embolism
Metastatic calcification
+Bacterial embolism

3. COMMON HISTOLOGICAL FINDINGS IN PYOGENIC LEPTOMENINGITIS INCLUDE
Purulent abscesses in the cerebral tissue
Erythrocytes depositionin the subarachnoid space
Spasm of blood vessels
+Neutrophil infiltration of the meninges
Shrinking of tissues

4. AMYLOIDOSIS CAN DEVELOP AS RESULT OF

Septicemia
Septicopyemia
+Chronic abscess
Bacterial enteritis
Infective colitis

5. SEPTICOPYEMIA IS CHARACTERIZED BY THE PRESENCE OF INFLAMMATION
+Purulent
Fibrinous
Granulomatous
Serous
Catarrhal

6. SEPSIS DIFFERS FROM OTHER INFECTIONS BY
Prove immunity
Infectivity
Cyclicity
Specificity of infection agent
+Polyetiology

7. REVEALED ON AUTOPSY PLURAL ABSCESSES IN ORGANS ARE TERMED AS
Cold abscess
Phlegmon
Purulent leakage
Septicemia
+Septicopyemia

8. CLINICAL-MORPHOLOGICAL FORM OF SEPSIS IS
Purulent
Toxic
Fungoid
+Septicemia
Viral

9.SEPSIS IS NEVER CAUSED BY
Bacteria
+Viruses
Fungi
Protozoa
Chlamydeous

10. RING ABSCESSES IN THE MYOCARDIUM ARE THE CHARACTERISTIC FEATURE OF
+Bacterial endocarditis
Nonbacterial thrombotic endocarditis
Libman-Sacks endocarditis
Rheumatic endocarditis
Syphilis

11. THE MOST COMMON DEATH REASON OF PATIENTS WITH SUBACUTE ENDOCARDITIS IS
Amyloidosis
+Chronic heart failure

Brown atrophy of the myocardium
Infarct of the kidney
Cachexia

12. SEPTIC SHOCK IS CAUSED BY
Virus
Foreign body
+Bacteria
Toxins
Immune complex

13. GRAYISH-WHITE ROUND AREAS ON THE CUT SURFACEOF KIDNEY AT EMBOLICNEPHRITIS ARE
+Pyemic abscesses
Hemorrhages
Fat droplets
Petechia
Focal atrophy

14. FORM OF SEPSIS WITH PYOGENIC LEPTOMENIGITIS IS
Septicemia
+Septicopyemia
Chronic abscess
Bacterial endocarditis
Infective nonbacterial thrombotic endocarditis

15. TYPE OF INFLAMMATION IN ORGANS AND TISSUES AT SEPTICOPYEMIA IS
+Purulent
Fibrinous
Granulomatous
Serous
Catarrhal

16. MACROSCOPIC APPEARANCES OF KIDNEY AT EMBOLIC PYOGENIC NEPHRITIS IS
+Enlarged, soft with small purulent foci
Enlarged, firm with large hemorrhages
Diminished, firm with small purulent foci
Diminished, soft with large hemorrhages
Diminished with granular surface

17.CARDIAC COMPLICATION IN BACTERIAL ENDOCARDITIS IS
Brown atrophy of the heart
Hemopericardium
Aortal coarctation
+Myocardial ring abscess
Hydropericardium

18. PATHOLOGIC PROCESS IN MYOCARDIUM AT BACTERIAL ENDOCARDITIS IS
Brown atrophy
Heart amyloidosis
Hemosiderosis

+Left-sided heart hypertrophy
Right-sided heart hypertrophy

19. PATHOLOGIC PROCESS IN LYMPHOID ORGANS AT SEPTICEMIA IS
Hypoplasia
+Hyperplasia
Infarction
Acute inflammation
Hemosiderosis

20. FIRST PYEMIC METASTASES AT SEPTICOPYEMIA CAN BE FOUND IN
Lymph nodes
Spleen
+Lungs
Heart
Skin

21. RENAL PATHOLOGYAT PATIENTS WITH SUBACUTEBACENDOCARDITIS IS
+Glomerulonephritis
Kidney amyloidosis
Pyelonephritis
Hemosiderosis
Lipoid nephrosis

22. PATHOLOGIC PROCESS DEVELOPING IN PARENCHYMA OF ORGANS DUE TO CHRONIC INFLAMMATION IS
Hypertrophy
Calcinosis
Hemosiderosis
Hyalinosis
+Atrophy

23. PATHOLOGIC PROCESS IN STROMA OF ORGANS AT SEPTICEMIA IS
+Interstitial inflammation
Hyalinosis
Necrosis
Apoptosis
Hyperplasia

24. PREDOMINANT CELLS IN PYEMIC ABSCESSES AT EMBOLIC PYOGENIC NEPHRITIS
Macrophages
Lymphocytes
+Neutrophils
Plasma cells
Erythrocytes

25. PATHOLOGIC PROCESS IN BLOOD VESSELS AT INFECTIVE ENDOCARDITIS IS
Hemosiderosis
+Vasculitis
Hyalinosis
Sclerosis
Amyloidosis

26. THE PATHOLOGIC PROCESS THAT CAN BE FOUND IN SPLEEN AT SEPSIS IS
Hemochromatosis
Infarction
Cyanotic induration
Atrophy
+Hyperplasia

27. COMMON HISTOLOGICAL FINDINGS IN PYOGENIC LEPTOMENINGITIS INCLUDE
Purulent abscesses in the cerebral tissue
Erythrocytes depositionin the subarachnoid space
+Neutrophii depositionin the subarachnoidal space
Spasm of blood vessels
Shrinking of tissues

28. COMMON HISTOLOGICAL FINDINGS IN PYOGENIC LEPTOMENINGITIS INCLUDE
Purulent abscesses in the cerebral tissue
Erythrocytes depositionin the subarachnoid space
Spasm of blood vessels
Shrinking of tissues
+Edema of the cerebral tissue

29. SEPTIC SHOCK IS CAUSED BY
Virus
Foreign body
+Streptococcus
Endotoxins
Immune complex

30.CARDIAC COMPLICATION OF BACTERIAL ENDOCARDITIS IS
Brown atrophy of the heart
Hemopericardium
Aortal coarctation
Hydropericardium
+Valvular insufficiency

31.CARDIAC COMPLICATIONS OF BACTERIAL ENDOCARDITIS INCLUDE
Brown atrophy of the heart
Hemopericardium
Aortal coarctation
Hydropericardium
+Valvular stenosis

32.CARDIAC COMPLICATIONS OF BACTERIAL ENDOCARDITIS INCLUDE
+Fibrinous pericarditis
Brown atrophy of the heart
Hemopericardium
Aortal coarctation
Hydropericardium

33. THE MECHANISM OF SEPTICOPYEMIA DEVELOPMENT IS
Thrombosis

Calcification
Necrosis
+Metastasion
Compensation

34. THE MECHANISM OF SEPTICEMIA DEVELOPMENT IS
Thrombosis
Calcification
+Intoxication
Metastasion
Compensation

35. THE MECHANISM OF SEPTICOPYEMIA DEVELOPMENT IS
Thrombosis
Metastatic calcification
Bleeding
+Embolism
Compensation

EXAMINATIONAL TASKS FOR FOREIGN STUDENTS

Task 1. A child was admitted to the hospital with weakness, fever and nasal hemorrhage. The examinations revealed: sharp elevation of lymphocytes (about several tens of thousands) and numerous lymphoblasts; enlarged lymph nodes of mediastinum; hepatosplenomegaly.
1. What disease had this patient?
2. Name the morphological subtype of the disease.
3. Name the typical complications of the disease.
4. Explain pathogenesis of hepatospleno-megaly.
5. What histological changes in the liver and spleen tissue must be revealing?

Task 2. 50 years old patient has noted augmentation of lymph nodes. He com-plains of weakness, periodical fever. In peripheral blood test excess amount of myelo-cytes and promyelocytes was revealed. Physical examination revealed enlargement of spleen and liver.
1. Name the disease.
2. What morphological variant of the dis-ease depending on histogenesis?
3. Explain pathogenesis of enlargement of spleen and liver.
4. How excess enlargement of spleen and liver is termed?
5. What outcome of the disease is possible in this case?

Task 3. 50 years old patient has noted augmentation of lymph nodes group at the left of neck. He complains of weakness, loss of body weight, dermal itch, fever. In peripheral blood test: ESR - 40 mm/hour, there are no changes in the leukocytes. The biopsy of lymph node revealed proliferation of atypical reticular cells among lymphoid elements, huge multinuclear cells (diagnostic cells of Berezovsky-Shternberg-Rid) and foci of necrosis and sclerosis.
1. Name the disease.
2. What is the morphological variant of the disease basing on results of histological research?
3. What morphological features are the hallmarks the disease?
4. How does the spleen look like at this disease?
5. Give its name.

Task 4. The patient suffering from stomach ulcer died suddenly. In autopsy his skin was pale, in the lumen of stomach and intestine 3,5 liters of blood were revealed.

1. What complication of the stomach ulcer took place in the case?
2. What general pathological process did develop in its result?
3. Name its kind depending on pathogenesis.
4. Describe gross changes of inner organs (size, color, density).
5. What general acute hemodinamic disorder caused the death of the patient?

Task 5. The man of 32 years after sharp supercooling felt weakness, dyspnea, and pains in the right half of thorax at a brith; body temperature is 39C. Blunted sound, absence of breath in low part of right lung, pleural murmur was revealed at examination. Treatment was without effect; patient died a week later after beginning of disease from pulmonary-coronary insufficiency. Autopsy revealed: enlarged heavy dense low lobe of right lung with imposing of fibrin on pleura, on cut section the whole lobe is air-less and grey; there is round cavity filled with pus at 9-10 segments.
1. What disease developed at the patient?
2. What pleural murmur was connected with?
3. What stage of disease took place on section?
4. What microscopical changes are characteristic for this disease?
5. What pulmonary complication developed at the patient?

Task 6. The man of 51 years arrived in clinic with complaints to dispnea, cough with plentiful sputum. Disease began 30 years ago, all this time he smoked much. X-ray examination of lung is found out: emphysema, saccular and cylindrical bronchial expansions; borders of heart are expanded due to right ventricle; fingers look like “drum-type sticks”. Symptoms of increasing renal failure appeared in hospital and patient died from uremia.
1. What pulmonary disease took place at the patient?
2. To what group of pulmonary diseases it refer?
3. What figurative name of lung with this pathology?
4. What changes of heart are revealed on section?
5. What pathological process complicated pulmonary disease and was the reason of renal failure?

Task 7. The patient has arrived in clinic with sharp pains in abdomen, weakness. He lost consciousness in few minutes after hospitalization, pulse is threadlike. Approximately 1500 ml of blood revealed in abdominal cavity during operation, abdominal part of aorta protruded, its wall in this place was thinned.
1. Name abnormal dilation of aorta.
2. What dangerous complication of it occurred?
3. What disease predisposed to such changes in aorta wall?
4. Give the definition of this disease.
5. What other local complication of the disease can take place in aorta?

Task 8. The patient with severe pains in abdomen died suddenly. Autopsy revealed expanded loops of small intestine with dark red to black wall. The mesenteric vessels are rigid, thickened, occluded by dark red solid masses.
1. What general pathological process developed in intestinal wall?
2. What vascular disease predisposed to it?
3. Name the clinico-morphological form of this disease.
4. What local hemodynamic complication was immediatereason of changes in intestinal wall?
5. What type of shock caused the death of the patient?

Task 9. The patient suffered from essential hypertension with manifestation of chronic renal failure has died. The autopsy revealed reduced kidneys with fine-granular surface. At microscopic examination in many organs the changes of arterioles are found: walls are thickened, lumen is narrowed, intima infiltrated with homogeneous pink masses.

1. Name the clinico-morphological form of the disease.
2. What stages of it?
3 What dystrophy developed in vessels walls?
4. Name the general pathological process developed in the kidneys.
5. What pathological changes in other parenchimal organs are possible in this stage?

Task 10. The 70-years old patient complained of pain in right foot. The soft tissues of the 1-st finger became black, mummified, shrinkages.
1. What general pathologic process developed in the low extremity?
2. What clinico-morphological pattern of it?
3. What vascular disease caused these changes?
4. Name its clinico-morphological form.
5. What complications are possible in this case?

Task 11. Patient with severe form of atherosclerosis of coronary arteries died in two days after onset of retrosternal pain attack. Myocardial infarction is diagnosed at ECG recording.
1. What stage of myocardial infarction was diagnosed?
2. Give the definition of myocardial infarction.
3. What is the reason of death at this stage infarction of?
4. What immediate cause of myocardial infarction is possible?
5. Describe macroscopical changes in myocardium.

Task 12. Unconsciousness patient of 70 years old with stroke and left-side paralysis was admitted in clinic. Dyscirculatory ischemic infringements of brain and severe atherosclerosis are in anamnesis.
1. What changes can be found out in brain tissue on section?
2. What disease was the cause of these changes?
3. Explain the mining of term “stroke”
4. List two types of it: a), b).

Task 13. A 69-year-old man died from the chronic heart failure. 15 years ago he hadmyocardial infarction.
1. What general hemodynamic process developed in his organs and tissues due to chronic heart failure?
2. Describe the liver on gross inspection.
3. How this is liver called?
4. Name the form of chronic ischemic heart disease at that patient.
5. What morphological changes are characteristic for it?

Task 14.A 66-year-old woman suffering from hypertensive vascular disease (HVD) about 10 years died from intracerebral hemorrhage.
1. What clinico-morphological form of HVD took place in this case?
2. Describe the brain on gross inspection.
3. What is the possible cause of death?
4. What material is deposited in arteriolar walls in the systemic hypertension?
5. What pathological process can be found in the kidneys in this disease?

Task 15. A 70-year-old man with left-sided heart failure and severe pulmonary hypertension died from chronic cardiac and lung insufficiency.
1. Describe the lung on gross inspection.
2. How is this lung termed?
3. Explain the mechanism of the development of the changes.

4. What changes can be found in different body cavities and in subcutaneous tissue?
5. Give definition of chronic ischemic heart disease

Task 16. The 50 years old man suffered from rheumatism since the childhood, has arrived to clinic with disorder of blood circulation. He died at the background of progressing heart failure. Autopsy investigation revealed mitral valve damage: the atrium-ventricular foramen narrowed to 1 cm, shutters are thickened; sclerosed, warty-like thrombotic masses are displaced on shutters closing edges.
1. What diagnosis is probable?
2. What its kind has developed at the patient as a result of rheumatism according to character of pathological process?
3. Give the definition of rheumatism
4. What microscopical changes can be revealed in the valve endocardium?
5. How termed irreversible stenosis of the valve in this case?

Task 17. The girl of 12 years died from quickly progressing rheumatism with the expressed allergic reactions. Streptococcal tonsilitis is in anamnesis 2 month ago. Histological examination of autopsy material revealed diffuse inflammatory infiltration of whole heart wall.
1. What clinico-morphological form of rheumatism took place?
2. Explain pathogenesis of the disease.
3. How the inflammation of heart in this case need termed?
4. Describe the heart at opening of pericardium cavity?
5. What is the possible cause of death in this case?

Task 18. The patient had transmural myocardial infarction some years ago. Chronic heart aneurysm with chronic heart failure followed.
1. Explain the pathogenesis of heart aneurysm.
2. Is it true or false?
3. What hemodynamic disorders can complicate the heart aneurysm: a), b) ?
4. What pigment accumulation is the hallmark of chronic heart failure?

Task 19. The patient after aortal-coronary shunting surgery suffered with progressing chronic heart failure. Autopsy revealed enlargement of heart. Pericardial cavity obliterated completely, myocardium is flabby, yellowish brown.
1. What is the probable diagnosis?
2. What is the figurative name of heart at this pathology?
3. Explain pathogenesis of deposition of calcium salts.
4. What general pathological process in myocardium followed?
5. What special stain needs to use for detection of such change?

Task 20. The patient suffered from chronic stomach ulcer. Sudden severe pain in epigastrium occurred with irradiation in shoulder. Cold sweat and paleness of skin appeared. Sharp intention of abdominal wall muscles is marked at palpation.
1. What complication of stomach ulcer developed at the patient?
2. Explain the pathogenesis of sharp intention of abdominal wall muscles?
3. What period of the disease took place (acute attack or remission)?
4. What histological findings can be seen in the bottom of ulcer in this period?
5. Name ulceral-neoplastic complication of chronic stomach ulcer?

Task 21. During investigation of gastroscopy biopsy material in mucous and submucosal layers of stomach wall invasive growth of atypical epithelial cells was revealed. Some large cells with pale pink cytoplasm and constricted nucleus (signet ring cells) were reveled between tumor cells.

1. What tumor was diagnosed?
2. Where can be found first limphogenous metastases of stomach cancer?
3. What is Krukenberg's tumor?
4. What is Virchov's metastasis?
5. What complication of stomach cancer can cause by secondary necrotic changes in the tumor?

Task 22. Pains in right iliac area, nausea, vomiting suddenly appeared at the patient. Pains in right area under ribs body temperature of 39 degrees joined for the second day. Operation was done. Enlarged and thickened appendix with plethoric serous membrane covered by fibrinous-purulent sedimentation was removed during operation.
1. What morphological type of acute appendicitis took place?
2. List microscopic changes of appendix.
3. What are possible complications of acute destructive appendicitis? a), b)
4. What are the two main reasons of appendicitis?

Task 23. During microscopical investigation of removed appendix was reveled infiltration of all layers of its wall by leucocytes with superficial and deep mucosal defects.
1. What type of acute appendicitis took place?
2. How did this appendix look like?
3. What complication of this type of appendicitis is lead to peritonitis development?
4. What complication can develop if inflammation will spread to branches of portal vein?
5. To development of what complications is lead spreading of purulent process on surrounding tissue and caecum?

Task 24. A 67-years man complained on intermitted abdominal pain, weight loss and melena. He had died 1,5 month later. Whitish-grey dense tumor with mixed (exso-endophitic) growth was reveled in colon on autopsy.
1. Give the diagnosis.
2. What histological type of tumor can be suspected at this patient?
3. Where first hematogenous metastases can be found?
4. What local complication of tumor had developed at this patient?
5. What general complication occurred at this patient?

Task 25. The symptoms of acute liver failure had been developed at the 25-year woman after poisoning by mushrooms. Investigation revealed progressive decreasing of liver.
1. What general pathological process had developed in patient's liver?
2. What diagnosis can be at this case?
3. What is the stage of this disease?
4. What can be reveled in liver tissue during histological investigation?
5. What possible outcomes of this disease?

Task 26. Cirrhosis of liver was diagnosed at the patient in gastroenterologic department. It is known from anamnesis, that five years ago he had transferred the heavy form of viral hepatitis B. Laparoscopy reviled reduction of liver in sizes its surface is irregularly nodular, character of surface is caused by presence of nodes with 5cm in diameter.
1. What morphological (macroscopical) variant of liver cirrhosis found out at the patient?
2. What morphogenetic type of cirrhosis?
3. What microscopic features of this cirrhosis?
4. What special stain needs to use for detection of cirrosis?
5. Name the possible reasons of death at cirrhosis of liver.

Task 27. 64-years woman during last 6 month felled weakness, losing of weight on10 kg. Large dense rough liver with multiple nodes, iron-deficiency anemia was reveled at examination. In the colon was found dense large tumor circularly grows in wall with ulceration in center.

1. What tumor was at this patient?
2. Is it benign or malignant?
3. Explain the pathogenesis of changes in the liver?
4. What pigment can deposit in the liver cells due to progressing of tumor and cachexia?
5. Give the definition of cachexia.

Task 28. The patient, suffering from gallstones, had pains in right below the ribs, the jaundice was developed.

1. Explain the pathogenesis of jaundice?
2. Name morphologic kind of jaundice?
3. What is the chemical composition of gallstones?
4. Name the most common complication of gallstones.
5. List etiological factors of gallstones.

Task 29. The patient was ill acutely after overcooling. Hypertension, hematuria, edema of face and phenomena of renal failure are marked. Patient died 6 month later the beginning of the disease. On section: kidneys are increased in size, flabby; cortical layer yellow - grey with red mottled, pyramids are dark red on cut surface.

1. What disease was at the patient?
2. Give the name to found out macroscopic changes in kidneys?
3. What changes in kidneys were found out at microscopic investigation?
4. Name the histological pattern of this disease.
5. What direct reason of patient death?

Task 30. Following changes were revealed on section of the patient, suffering from chronic glomerulonephritis during 12 years: kidneys are sharply reduced in sizes, dense, their surface fine-grained; fibrinous inflammation of serous and mucous membranes; dystrophic changes in myocardium and liver, edema of brain.

1. Name the found out macroscopic changes of kidneys.
2. Give the microscopic characteristic of these changes.
3. What complication occurred in the end of disease?
4. Explain pathogenesis of fibrinous inflammation in mucous and serous membranes.
5. Name other, most often diseases of kidneys with same complication.

Task 31. Increasing of thyroid gland at the patient living in mountain area leaded to breathing disorders, difficult gulp, and expansion of subcutaneous veins of neck frontal surface. Thyroid hormone amount did not change.

1. What disease of thyroid gland developed at the patient?
2. Name its pattern depending on functionof thyroid.
3. What the reason and pathogenesis of this diseases?
4. What macroscopic changes of thyroid gland are characteristic for this disease?
5. What changes of glands are found out at microscopic investigation?

Task 32. The patient complains of increased appetite, thirst, poliuria, dryness and itch of integuments, often-purulent diseases. Hyperglycemia and glycosuria is marked.

1. Name this disease.
2. What endocrine gland is damaged by pathological process?
3. What macroscopic changes developed in it?
4. What microscopic changes are in it?

5. What changes take place in kidneys?

Task 33. 70-yars woman suffered from decompensate diabetes mellitus II type had died from ischemic infarction of brain. In anamnesis periodic hyperglycemia and glucosuria was. Obesity is 45%. Autopsy revealed severe metabolic changes in the vessels, heart, liver and kidneys.

1. Is this type of diabetes insulin dependent?
2. Pathology of which organ is this disease connected with?
3. Name complication of the disease in the vascular walls.
4. What general pathological process was reveled in the liver?
5. What is the main morphogenetic mechanism of its development?

Task 34. A 41-year-old woman with repeated metrorrhagia was admitted to the hospital for a routine hysterectomy, which revealed enlarged irregular uterus. Macroscopic examination of surgical material showed multiple, round-shaped, dense-elastic, pale-pink tumors with fibrous structure and well defined borders in the myometrium. Histological examination revealed well differentiated tumor tissue consisting fascicles of collagen fibers.

1. What is probable diagnosis?
2. Is it benign or malignant neoplasm?
3. Name two histological variants of this tumor depending on predominance of cells or fibers.
4. What are possible sites of its localization in the uterine wall? - a), b), c).
5. What special stain must be used for detection of its origin?

Task 35.A 30-year-old woman was admitted to the hospital in three years afterdelivery. On examination of cervix irregular-shaped focus of bright red color was found out on a background of a pale mucous membrane. On histologic examination a growth of cylindrical epithelium was founded.

1. What pathological process (disease) takes place?
2. What is the disease depending on pathogenesis?
3. Is it reversible?
4. What general pathologic process underlies such changes of epithelium?
5. What disease can develop on its background?

Task 36. Pulmonary bleeding occurred in the young woman. Abortion is in anamnesis 6 month ago. X-ray examination revealed multiple tumor-like centers of consolidation in the lungs. Overgrowth of atypical cells cyto- and sincytiotrophoblast are found at histological examination of diagnostic scrape from uterus cavity.

1. Name this tumor?
2. Is it benign or malignant?
3. Is it organ specific?
4. Describe macroscopic changes in uterus cavity.
5. How were connected changes in lung with this tumor?

Task 37. A focus consolidation occurred in the left breast of 38-years old woman. The sectoral resection of breast was made. Surgical material examination revealed dense-elastic tumor node 3 cm in diameter surrounded with capsule. On cut section tumor tissue is whitish-gray with dilated lumens of ducts. Histological investigation revealed slit-like glandular and tubular structures, compressed by overgrowth of connective tissue predominated above parenchyma.

1. What is the probable diagnosis?
2. Give the morphological variant of this tumor depending on connective tissue overgrowth?
3. Is it benign or malignant?
4. Is this tumor organospecific?
5. What category of breast diseases is it refereed to?

Task 38. Patient complained on fever, temperature up to 40C, headache, cough, and expressed dyspnea arrived in clinic during flu epidemic. Moist wheeze were listened in lungs. In spite of treatment, patient died at phenomena of pulmonary-cardiac insufficiency three days later.

1. What disease was the reason of patient's death?
2. What form of disease?
3. What kind of inflammation was found out at autopsy in trachea?
4. Describe appearance of lungs?
5. How named the lung with such appearance figuratively?

Task 39. The child attending kinder garden had high temperature up to 38,5C, cold, conjunctivitis and cough. There is macular rash on skin, whitish branny appearance on mucous membrane of cheeks in oral cavity. Dyspnea and moist wheeze in lungs developed on the fourth day. The difficulty of breath has suddenly appeared. The child died from phenomena of asphyxia.

1. Give the name of the disease.
2. What is its etiology?
3. Name the appearances on mucous membrane of cheeks?
4. What process in lungs was complicated this disease?
5. What complication was the reason of child death?

Task 40.A 26-year-old man complained of massive diarrhea about liters of dilute "rice-water" stool containing flecks of mucus.

1. What disease did the patient suffer from?
2. What are the mechanisms and cause of the disease?
3. Name the stages of the disease.
4. Describe the intestine on gross inspection.
5. Name the possible complications of the disease.

Task 41. Typhoid feverwas diagnosed in 56-year-old man.

1. What is the cause of the disease?
2. What type of interaction between microorganism and epithelium of intestine in this disease?
3. What department of intestine is commonly affected?
4. Name the gross changes in the intestine in the 1-st stage of the disease.
5. Name the possible complications of the disease in second week from the onset.

Task 42.A 30-year-old man was diagnosed to have acute infection with bloody diarrhea, tenesmus, and fever. The phenomena of paraproctitis are found out. Later the pains in a waist show up, pyuria, has appeared, the body temperature has sharply increased.

1. What bacterial infection occurred in the patient?
2. Where is entry of infection?
3. What form of colitis took place?
4. What is cause of the paraproctitis?
5. What type of interaction between microorganism and epithelium of intestine in this disease?

Task 43.A 41-year-old man was admitted to the hospital with symptoms of acute infectious disease. The physical examination revealed dirty-white films on the tonsils and larynx.

1. What is the most likely diagnosis in this case?
2. What etiology of the disease?
3. What general pathologic process can be found in the trachea?
4. Describe gross appearance of the tonsils.
5. What fatal complication is possible in this disease?

Task 44.A 5-year-old child was admitted to the hospital with symptoms of acute infectious disease and dot erythematous skin rash over the trunk and face. The examination revealed severe necrotic tonsillitis.
1. What is the diagnosis?
2. What is its etiology?
3. Name the variant of this disease depending severity of course.
4. Describe the tonsils on gross inspection.
5. List possible local complications of necrotic tonsillitis.

Task 45. Increasing of the temperature up to 40C, cloudiness of consciousness, presence of plural haemorrhages on skin is marked at the patient three days after criminal abortion. She died insecond day after occurrence of these symptoms.
1. What did clinical-morphological form of sepsis take place?
2. What was a kind of sepsis depending on character of entry of infection?
3. Characterize morphology of local changes.
4. What general pathologic process can be found out: in parenchyma of organs?
5. What general pathologic process can be found out n hemopoeitic and lymph tissues?

Task 46. The patient arrived in clinic for draining of abscess on buttock which is formed after intramuscular injection. The temperature remained 39C, dyspnea and cloudiness of consciousness appeared after draining of abscess. The patient died at phenomena of acute heart failure.
1. What did clinical-morphological form of sepsis develop at the patient?
2. What was a kind of sepsis depending on character of entry of infection agent?
3. What macroscopical changes in connection with widespread of infection can be found in lungs, heart, brain?
4. What general pathologic process must be in spleen?
5. How termed the spleen with such changes?

Task 47.A 5 years-old child after transferred measles had marked weakness, hyperhidrosis, and high temperature. The skin is pale. Tuberculin test is sharply positive. X-ray examination of thorax revealed the round-shaped consolidations in IX segment of right lung under the pleura and near root of lung. The primary lung tuberculosis had diagnosed.
1. What morphological manifestation of primary tuberculous complex in the lungs.
2. Name its elements.
3. What tissue reaction prevails?
4. Name favorable outcome of primary tuberculosis.
5. What the pathways of primary tuberculosis spreading?

Task 48.A 25 years-old man who had transferred primary tuberculosis in the childhood, the signs of thoracic vertebrae deformation have appeared and began to accrue. The tuberculous spondylitis has diagnosed. Later 4 years a hunch is developed.
1. Name the clinico-morphologic form of tuberculosis?
2. Explain pathogenesis of spondylitis?
3. Describe gross changes in the vertebrae characteristic for tubercular infection.
4. Where the primary lesion settles down: in marrow, in a bone tissue or in the bones and joints?
5. What pathologic changes must be revealed in the entry of infection?

Task 49. A 32-year-old woman was admitted to the hospital because of the productive cough with high temperature. A peripheral round shaped subpleural shadow was found on radiological examination. Following further examination, mycobacterium tuberculosis was cultured. The miliary tuberculosis was diagnosed.
1. What are the histological findings in the lungs of the patient?

2. What is the pathogenesis of tuberculous granuloma?
3. What cell transformations can be found in tuberculous granuloma?
4. What are the causes of caseous necrosis in tuberculous granuloma?
5. What favorable outcome of the disease is possible?

Task 50.A 53 years-old man suffered from cavitary fibrocaseous tuberculosis complicated by pleura empyema. Later 6 years the progressing renal insufficiency is associated oneself with lung process. The patient died at the phenomena of uremia.
1. What kind of tuberculosis is it: primary, hematogenous or secondary?
2. Describe the changes in the lungs characteristic for this stage of tuberculosis.
3. What special stain needs to use for detection of etiologic agent in microsections?
4. What process had developed in the kidneys?
5. What complication determined the development of uremia?

Task 51.A 30 years-old man had transferred primary syphilis 7 years ago. He had marked retrosternal pains as stenocardia. Acute infringement of coronary blood was diagnosed.
1. What period of syphilitic process is it?
2. What is the clinic-morphologic form of it?
3. What tissue reaction is typical for this period?
4. Where is localized inflammatory process?
5. What is its morphological picture?

Task 52. The liver biopsy of the 25-year-old man revealed multiple gummous granulomas.
1. What disease had this patient?
2. What stage of it?
3. What is the cause of the disease?
4. What type of inflammation is characteristic for it?
5. Describe gross appearance of the liver.

MACROPREPARATES

1. 1 «Acute warty endocarditis of mitral valve»
2. 6 «Polipous-ulcerosal endocarditis of aortal valves»
3. 9 «Fibroplastic endocarditis, mitral valve stenosis»
4. 16 «Chronic aneurysm of heart»
5. 18 «Fibrinous pericarditis»
6. 21 «Hypertrophy of the heart»
7. 26 «Brown atrophy of myocardium»
8. 28 «Gangrene of the small intestine»
9. 31 «Aneurysm of the arch of aorta at syphilis»
10. 32 « Pulmonary thromboembolism»
11. 35 «Aneurysm of aorta with thrombosis »
12. 48 «Subarachnoid hemorrhage»
13. 50 «White (ischemic) infarcts of spleen»
14. 53 «Red (hemorrhagic) pulmonary infarct»
15. 70 «Bullous emphysema of lung»
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17. 84 «Compound congenital heart disease»
18. 90 «Hypertrophic gastritis»
19. 97 «Phlegmonous appendicitis»
20. 98 «Chronic stomach ulcer»

21. 104 «Fatty dystrophy of liver»
22. 110«Nutmeg liver»
23. 115«Cirrhosis of the liver»
24. 116 “Uterus cancer “
25. 118 «An esophageal varices with rupture of the vessels wall»
26. 125«Tubal pregnancy»
27. 131 “Saucer-like stomach cancer”
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34. 191«Embolic purulent interstitial nephritis»
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38. 223«Subacute glomerulonephritis»
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42. 238«Purulent leptomeningitis»
43. 240 «Hyperplasia of spleen at sepsis»
44. 242 “Primary pulmonary tubercular complex with miliary generalization”
45. 245 «Caseous necrosis of lymph node at the tuberculosis»
46. 248 “Adrenals adenoma”
47. 252 “Caseous pneumonia”
48. 254 “Fibrous-cavernous tuberculosis of lungs”
49. 259 «Hydatid cyst of the liver»
50. 269«Diffuse goiter»
51. 280«Hydrocephalus»
52. 282«Splenomegalia at chronic myeloleukemia»
53. 289 “Tuberculosis of a kidney”
54. 294«Bile-cystolithiasis»
55. 306 «Lymph nodes in chronic lymphocytic leukemia»
56. 311 «Big motley lung at flu»
57. 313 “Chorionepithelioma”
58. 319 «Gangrene of foot fingers»
59. 350 «Varicose veins with phlebothrombosis»
60. 363«Hydatidiform mole»
61. 364 «Hyaline change of spleen capsule» («icing spleen»)
62. 372 «Amyloidosis of kidney» («big bacon kidney»)
63. 378«Teratoma»
64. 418 “True croup at diphtheria”
65. 420«Croupous pneumonia»
66. 421«Chronic lung abscess»
67. 422 «Recurrent myocardial infarction with acute aneurism and its thrombosis»
68. 439 “Spleen at lymphogranulomatosis”
69. 457«Ascending pyelonephritis»
70. 500 «Measles rash», «Rash at scarlet fever»
71. 520«Pigmented cyst of brain»

MICROPREPARATES

1. 2.«Croupous pneumonia» (hematoxylin and eosin)
2. 8. «Chronic stomach ulcer» (hematoxylin and eosin)
3. 9. «Mucous cancer» (hematoxylin and eosine)
4. 14. «Fatty dystrophy of liver» (Sudan - III)
5. 15. «Amyloidosis of kidney» (Congo - red)
6. 16.«Caseous necrosis of lymph node at the tuberculosis» (hematoxylin and eosin)
7. 18. «Septic myocarditis» (hematoxylin and eosin)
8. 20.«Granulation tissue» (hematoxylin and eosin)
9. 23. «Hemosiderin in the locus of hemorrhage» (reaction of Perls)
10. 25. “Squamous skin cancer” (hematoxylin and eosin),
11. 27. “Stomach adenocarcinoma”(hematoxylin and eosin),
12. 35. «Abscesses of kidney at septicopiemia» (hematoxylin and eosin)
13. 36. “Syphilitic mesaortitis” (hematoxylin and eosin)
14. 38. «Hypertrophy of the myocardium» (hematoxylin and eosin)
15. 39. «Purulent leptomeningitis» (hematoxylin and eosin)
16. 58.«Leiomyoma» (picrofuscin by Von Hyzone)
17. 61.«Ischemic renal infarct» (hematoxylin and eosin)
18. 62. «Hemorrhagic pulmonary infarct» (hematoxylin and eosin)
19. 71. «Intracerebral hematoma» (hematoxylin and eosin)
20. 75.«Fat embolism of the lung» (Sudan - III)
21. 80. «Glandular hyperplasia of the endometrium» (hematoxylin and eosin)
22. 81. “Lymphatic node at lymphogranulomatosis” (hematoxylin and eosin)
23. 87. “Papillary thyroid cancer “ (hematoxylin and eosin)
24. 88. «Actinomycosis» (hematoxylin and eosin)
25. 89. «Cardiosclerosis» (picrofuscin by Von Hyzone)
26. 90.«Kidney in acute myeloblastic leukemia» (hematoxylin and eosin)
27. 94. «Acute myocardial infarction» (hematoxyline and eosin)
28. 97. «Brown induration of the lungs» (Prussian blue)
29. 100. «Multilobular cirrhosis of liver» (picrofucsin on van Hyson)
30. 103.«Nutmeg liver» (stained by hematoxylin and eosin)
31. 109. «Focal influenzal pneumonia» (hematoxylin and eosin)
32. 110. «Mixed thrombus in vein» (hematoxylin and eosin)
33. 113. «Miliary tuberculosis of lung» (hematoxylin and eosin)
34. 117. «Colloid goiter» (hematoxylin and eosin)
35. 126. «Melanoma of skin» (hematoxylin and eosine)
36. 127. "Bronchopneumonia" (hematoxylin and eosin)
37. 133. «Lung emphysema» (hematoxylin and eosin)
38. 135. «Hyaline changes of pleura» (hematoxylin and eosine)
39. 136. «Petrifications in lung (Ghon focus)» (hematoxylin and eosine)
40. 141. “Papilloma of skin “(hematoxylin and eosin)
41. 150. «Hydatidiform mole» (hematoxylin and eosin)
42. 153. «Atherosclerosis of arteria» (Sudan - III)
43. 159. «Tubal pregnancy» (hematoxylin and eosin)
44. 163.“Mammary gland fibroadenoma”(hematoxylin and eosin)
45. 165. «Fibrinous pericarditis» (hematoxylin and eosin)
46. 178. «Cavernous hemangioma of the liver» (hematoxylin and eosin)
47. 182. «Ulceral enteritis in salmonellosis» (hematoxylin and eosin)
48. 183. “Chorionepithelioma” (hematoxylin and eosin)
49. 187. «Atrophy of pancreas at diabetes» (hematoxylin and eosin)
50. 198. «Phlegmonous appendicitis» (hematoxylin and eosin)

51. 203. «Extracapillary serous glomerulonephritis» (hematoxylin and eosin)
52. 205. «Septic polypous-ulcerous endocarditis» (hematoxylin and eosin)

ELECTRONOGRAMMS

1. Baloon dystrophy of hepatocyte (atlas fig. 8)
2. Fibrinoid degeneration of collagenic fibers (fig. 25)
3. Amyloidosis of glomerulus (fig. 38)
4. Ischemic myocardium (fig. 74)
5. Inflammation, emigration of segmentonuclear leukocytes (fig. 98)
6. Hypertrophy of the myocardium (the stage of compensation) (fig. 166)
7. Hypertrophy of the myocardium (the stage of decompensation) (fig. 166)
8. Ultrastructural atypia of tumor cell (fig. 175)
9. Membranous glomerulonephritis (fig. 320)
10. Croupous pneumonia, fibrin resorbtion (fig. 280)

ANSWERS ON TASKS

Task №1

1. Acute leukemia.
2. Lymphoblast leukemia.
3. Septic complications.
4. Unchecked increasing of neoplastic cells in hematopoietic organs, there hematogenic spreading in other organs with infiltration of them.
5. Infiltration of tissue by lymphoblasts, diapedetic hemorrhgias, venous congestion.

Task №2

1. Chronic leukemia.
2. Myelocytic leukemia.
3. Unchecked increasing of neoplastic cells in hematopoietic organs, there hematogenic spreading in other organs with infiltration of them, what resulted in edema, infarctions and congestion.
4. Hepato-, splenomegalia.
5. Slow progression with blast crisis, transformation in acute leukemia.

Task №3

1. Lymphogranulematosis.
2. Mixed-cellular variant.
3. Diagnostic giant multinuclear cells of Berezovsky-Shtermberg-Rid.
4. Spleen is enlarget, consistence is dense, change of foci: dark red, grey and whitish-yellow, reminds porphyry.
5. Porphyric spleen, diffuse waxy spleen.

Task №4

1. Bleeding.
2. Anaemia.
3. Posthemorrhagic.
4. Size-diminished, color-pail, density-flabby.
5. Hemorrhagic shock.

Task №5

1. Croupouse (fibrinouse) pneumonia.
2. Fibrinouse pleuritis.
3. III stage, grey hepatisation.
4. Diffuse loading of alveoli with fibrinose exudates (croupouse inflammation), Konn's bridges.
5. Abscess of lung.

Task №6

1. Bronchoectatic disease.
2. Chronic Non-specific pulmonary diseases (CNPD).
3. Honey-comb lung.
4. Hypertrophy of heart (cor bovinum).
5. Amyloidosis.

Task №7

1. Aneurism.
2. Rupture of aneurism.
3. Atherosclerosis.
4. Atherosclerosis is the chronic disease arising as a result of fatty and protein methabolism abnormality, characterized by injury of muscle and muscle-elastic types arteries as focal deposition of lipids, proteins and reactive overgrowth of connective tissue in its intima.
5. Thrombosis.

Task №8

1. Necrosis.
2. Atherosclerosis.
3. Atherosclerosis of mesenteric vessels.
4. Thrombosis.
5. Pain shock.

Task №9

1. Renal type.
2. III stage, changes of organs in connection with arterieschanges and intraorganic blood circulation disorder.
3. Hylinosis.
4. Cirrhosis.
5. Infarctions, gangrene, hemorrhages, hematoms, cyst formation.

Task №10

1. Gangrene.
2. Wet gangrene.
3. Atherosclerosis.
4. Atherosclerosis of low extrimity.
5. Mutilation (selfamputation).

Task №11

1. II stage (necrosis).
2. Coronary disease of heart is caused by acute absolute or relative insufficiency of coronary blood supply.
3. Caused by early complications:acute cardiac failure, cardiogenic shock, asystoly, ventricular fibrillation, rupture of heart.
4. Occlusion of coronary arteryby atherosclerotic plaque.

5. Yellowis irregular flabby focus of necrosis.

Task №12

1. Intracerebral hemorrhage.
2. Atherosclerosis of brain arteries.
3. Insult (formation of brain hematoma).
4. a) ischemic,
b) hemorrhagic.

Task №13

1. General venouse congestion.
2. On cut cection liver has motly painting: small black points(hemorrhages) on yellow background (fatty dystrophy).
3. Nutmeg liver.
4. Macrofocal cardiosclerosis.
5. Organisation of necrosis, replacement of myocardium on connective tissue.

Task №14

1. Cerebral form.
2. Intracerebral hematoma in place of ganglions.
3. Distruction of vital centers.
4. Hyaline.
5. Nephrocirrhosis.

Task №15

1. Lung is enlarged, consistence is dense, color - brown.
2. Brown induration of lung.
3. Increased pressure in small circle of blood supply (pulmonary hypertension) causes increasing of vessel wall permeability and exudation of blood.
4. Anasarca and accumulation of fluid (edema) in all cavities of body (hydrothorax, hydropericardium, ascitis,hydrocele).
5. Chronic IHD is disease caused by absolute or relative chronic coronary insufficiency.

Task №16

1. Rheumacarditis.
2. Relapsing warty endocarditis of mitral valve with atrioventricular stenosis.
3. Rheumatism is the group of diseases characterized by connective tissue injury, caused by infringement of an organism immune homeostasis.
4. Shutters of valve are thickened, submitted by hyalinizated tissue. Fresh centers of connective tissue disorganization (mucoid swelling and fibrinoid degeneration) are visible on background of sclerosis. Covering endothelium is destroyed with warty-like fresh thrombotic mass imposing.Endocardium diffusely infiltrated by lymphocytes and macrophages.
5. Acquired heart defect, stenosis – “fish mouth”.

Task №17

1. Cardiovascular form (rheumatic carditis).
2. Reaction of hypersensitivity III type (immune-complex), autoimmunization with damage of vessels walls and systemic progression of disorganization of connective tissue in certain organs.
3. Pancarditis.
4. “Hairy heart”(fibrinouse pericarditis), strings of fibrin can be seen on surface of pericardium.
5. Acute coronary insufficiency.

Task №18

1. Protrusion of heart wall on the background of macrofocal cardiosclerosis.
2. True.
3. a) Rupture of aneurism,
   b) thrombosis.
4. Lipofuscine.

Task №19

1. Pancarditis with fibrinouse pericarditis (Hairy heart) complicated by adhesive pericarditis and cavity obliteration.
2. “Stone heart”.
3. Surface of pericardium become rough due to fibrinouse exudates imposing and fibrin strings are the matrix for calcium salts deposition (dystrophic calcification) and cavity obliteration.
4. Diffuse microfocal cardiosclerosis (in outcome of interstitial inflammation).
5. Picrofuchsine (by von Giesone).

Task №20

1. Perforation of stomach wall with ulceral bleeding.
2. Outcome of blod in abdominal cavity with peritonitis development.
3. Acute attack.
4. Ulceral defect extends on mucosa and muscle layers with destruction of muscle fibers. Four layer can be seen in the bottom of ulcer: fibrinous-purulent exudates, fibrinoid necrosis, granulation tissue and cicatricle tissue.
5. Malignesation, transformation of chronic ulcer in ulcer-cancer (malignant neoplasm).

Task №21

1. Ring-cell carcinoma of stomach.
2. In mesenteric lymph nods and omentum (regional lymph nods).
3. Metastasis of stomach cancer in ovary.
4. Retrograde lymphogenic metastases of stomach cancer in supra-clavicles lymph nods.
5. Erosive bleeding.

Task №22

1. Phlegmanouse appendicitis.
2. The wall of appendix is thickened, all layers are diffusely infiltrated by polymorphonuclear leucocytes, serous membrane covered by fibrinous exudates (reactive inflammation).
3. a) perforation→peritonitis; b) gangrene of appendix→mutilation.
4. Development of sclerosis, atrophic changers and immune difficiency..

Task №23

1. Ulceral-phlegmanouse appendicitis.
2. Appendix is increased in size, serouse membrane is dim, red (due to plethora), covered by fibrinouse exudate.
3. Perforation in place of ulceral defect.
4. Pilephlebitic liver abscess.
5. Typhlitis and perityphlitis.

Task №24

1. Colon cancer.
2. Adenocarcinoma.
3. First hematogenous metastases can be found in liver.
4. Necrosos and ulceration of tumor.

5. Erosive bleeding.

Task №25

1. Acute hepatosis of liver.
2.Toxic dystrophy of liver .
3. The stage of "red atrophy".
4. Various (from dusty to lager) droplets of fat colored by Sudan III in orange.
5. Progressive massive necrosis of liver→hepato-rhenal insufficiency, cirrhosis of liver.

Task №26

1. Macronodular cirrhosis of liver.
2. Postnecrotic cirrhosis.
3. Parenchima of liver is submitted by various oval false lobules, where central vein is absent, hepatic beams are destroyed, pulled together hepatic triads are visible among wide areas of connective tissue .
4. Picrofuchsin by von Giesone.
5. Hepatic coma, hepato-renal insufficiency.

Task №27

1. Circular colon cancer.
2. Malignant.
3. Cirrhosis of liver due to cancerous cachexia connect with intoxication and anemia.
4. Lipofuscin.
5. Cachexia is the general atrtophy (diffuse reduction of organs in size in alive organism).

Task №28

1. Mechanical jaundice (obturative) due to occlusion of bile tract by concrement.
2. Subhepatic.
3. Phosphates, cholesteric, pigmentary and mixed.
4. Obturation of common bile duct.
5. General (disbalance between buffer systems, alkaline ph) and local (inflammation, narrowing of lumen, scarring, infringement of passage, high viscosity of secret and etc.).

Task №29

1. Acute glomerulonephritis.
2. Big motley kidney.
3. Observed marked capillary hyperemia, lumens of glomulei are increased, filled with serous exudates looking like semy-lunes.
4. Exudative exstracapillary glomerulonephritis.
5. Acute renal failure.

Task №30

1. Secondary reduced kidney (arteriolosclerotic nephrocirrhosis).
2. Overgrowth of connective tissue is seen on background of renal tissue.
3. Acute renal failure.
4. Uremia.
5. Renal form of hypertensive disease, nephropathy at diabetes.

Task №31

1. Goiter.
2. Euthyroid.
3. Iodine deficiency in water and food (endemia – natural absence of iodine in mountain area).

4. Thyroid gland is diffusely increased in size in all anatomical parts, homogeneous. Morphological features of structure are determined at histological investigation.
5. Follicles of thyroid gland are enlarged in size, overloading with dense colloid, some of them ruptured forming cysts. There walls are thinned, covered by atrophic and flat epithelium (atrophy from hydrostatic pressure). Interlobular overgrowth of connective tissue is observed.

Task №32

1. Diabetes.
2. Pancreas.
3. Gland is reduced in size, dense, overgrowth of adiposal and fibrosal tissue (lipomatosis and sclerosis) is marked in it.
4. Langergans's isles are reduced in size (atrophy), some of them vicariously hypertrophied, overgrowth of adiposal (lipomatosis) and connective tissue (sclerosis) is marked, vessels walls are hyalinizated.
5. Nephropathy →nephrocirrhosis (primary reduced kidney.

Task №33

1. No (insulinindependent).
2. Pancreas.
3. Hyalinosis of vessels (stroma-vascular protein distrophy.
4. Fatty dystrophy of liver ("Goose liver").
5. Transformation, decomposition (infiltration).

Task №34

1. Fibromyoma of uterus.
2. Benign.
3. Fibromyoma (hard, predominance of collagen fibers), myofibroma (soft, predominance of muscle fibers).
4. a) submucosal, b) intramural, c) subserosal.
5. Picrofuchsin.

Task №35

1. Endocervicosis (pseudoerosion (false erosion) of uterus cervix).
2. Dishormonal disease.
3. Yes (after adequate treatment).
4. Metaplasia of epithelium.
5. Uterus cervix cancer.

Task №36

1. Chorionepithelioma (chorioncarcinoma).
2. Malignant.
3. Yes (organospecific).
4. Uterus cavity contains dark red tumoral node with rough surface and spongiform structure which ingrowths in myometrium. Plural foci of hemorrhage are visible in it.
5. Tumor has given metastases in lungs.

Task №37

1. Fibroadenoma of brest .
2. Intracanalicular variant.
3. Benign.
4. Yes, organospecific.
5. Dishormonal diseases.

Task №38

1. Pneumonia at flue.
2. Severe toxic form.
3. Necrotic tracheitis.
4. Lung is enlarged in size. Plural centers of acinary or lobulary pneumonia with ability to abscess formation (whitish yellow) and centers of hemorrhages (dark red) are visible in pulmonary tissue.
5. “Big motley lung”.

Task №39

1. Measels.
2. Viral diseas.
3. Enanthema (Belsky-Copplik-Filatov’s spot).
4. Viral pneumonia.
5. False croup.

Task №40

1. Cholera.
2. Vibrio cholerae, vibrio El-Tor.
3. 1) Cholera enteritis, 2) Cholera gastroenteritis, 3) Algid period.
4. Loops of small intestine are swollen, serous membrane is dry with necrosis of enterocytes, lumen contains colorless liquor looking like “rice-water”.
5. Exicosis (dryness of the body), reduction of spleen, necrotic and necrobiotic changes in liver, brain, myocardium, acute renal failure (necrosis of renal tubular epithelium).

Task №41

1. Salmonella typhi, Salmonella paratyphi.
2. Transenterocytary relation.
3. Large intestinum.
4. In stage of medullary swelling Peyer’s patches are increased, jut out above mucosa, forming convolutions and bulgings similar to surface of brain.
5. Stage of necrosis of Peyer’s patches can complicated by perforation with peritonitis development, severe intoxication, sepsis, intestinal bleeding.

Task №42

1. Dysentery (shigellosis).
2. Colon.
3. Purulent colitis.
4. Spreading of infection on surrounded tissue.
5. Intraenterocytary.

Task №43

1. Diphtheria.
2. Bacterial infection, corynebacteria diphtheriae (Leffler’s bacillus).
3. Fibrinous inflammation (croupous and diphtheritic).
4. Tonsils are enlarged in size, red, soft, edematic, covered by thick whitish-grey pellicle which is closely related to the surface of epithelium, difficultly removed with deep ulceral defects after removing.
5. True croup (mechanical asphyxia, occlusion of trachea by diphtheria pellicle).

Task №44

1. Scarlet fever.

2. Bacterial infection, ß-hemolitic streptococcus group A (Streptococcus pyogenes).
3. Toxic-allergic.
4. Tonsills are enlarged in size, swollen, dark red. There are ulceral defects with necrotic masses inside on there surface.
5. Gangrene of tonsils, retrotonsilar abscess, phlegmone of neck, otitis, antritis.

Task №45

1. Septicemia (acutest, fulminant form).
2. Obstetrical (uterogenic).
3. Uterus enlarged in size, swelled, edematic, dark red with plural petechial hemorrhages.
4. Dystrophic changers.
5. Hyperplasia.

Task №46

1. Septicopiemia (acute form).
2. Surgical sepsis.
3. Organs enlarged in size, flabby, edematic with plural foci of purulent inflammation.
4. Hyperplasia and hypertrophy.
5. Splenomegalia.

Task №47

1. Tuberculous granulema.
2. Primary affect (Ghon's focus), lymphangitis, lymphadenitis.
3. Productive-necrotic.
4. Intention of primary affect, encapsulation, fibrosis (scar formation), petrification, ossification.
5. Growth of primary affect, lymphogenic, hematogenic, mixed.

Task №48

1. Extrapulmonary tuberculosis of bones (primary tuberculous spondilitis).
2. Lymphogenic spreading of infection from primary affect.
3. Plural foci of caseous necrosis with destruction of bodies in vertebral bones.
4. In bone marrow.
5. Primary tuberculous complex.

Task №49

1. Lungs are enlarged with numerous whitish-yellow foci 1-2 mm with dense necrotic masses looking like cottage cheese (casious necrosis) in all fields of lungs.
2. Productive tissue reaction, hematogenic spreading from primary affect.
3. Transformations of macrophages in large cells: mononuclear epithelioid cells and giant Pirogov-Langhans's cells.
4. destruction of tissue by productive inflammation (fibrinoid necrosis).
5. Scarring of granulemas.

Task №50

1. Secondary.
2. Extensive irregular shaped cavities with thick and dense walls are seen in tissue of lung. Cavities contain yellowish-grey breaking caseous mass. Large bronchi are open in the cavities. Peribronchial and privascular sclerosis are visible on the background of emphysema.
3. Ziehl-Nielsen.
4. Amyloidosis of kidney.
5. Acute renal failure.

Task №51

1. Tertiary syphilis.
2. Cardiovascular syphilis.
3. Productive-necrotic reaction.
4. In ascending aorta, thoracic aorta, aortal valves, coronary arteries. (Fibrosis of aortal and arterial walls →aneurisms formation, narrowing of lumens.)
5. Cellular infiltrates represented by lymphocytes, plasma-cells, fibroblasts and singular Langhan's giant cells are observed in mesangium by pathway of vasa vasorum.

Task №52

1. Syphilis.
2. Tertiary gummous syphilis.
3. Treponema pallidum.
4. Productive granulomatous inflammation.
5. Liver is enlarged, with whitish-grey foci of liquafactive necrosis surrounded sclerosis on background of fatty dystrophy.