CELL INJURY AND NECROSIS

Manual for practical classes in pathomorphology for English-speaking medical students

ДИСТРОФІЯ І НЕКРОЗ

Методичні вказівки до занятті з патоморфології для студентів медичних вузів з англійською мовою навчання

Затверджено вченою радою ХНМУ.
Протокол № 6 від 26.05.2016.

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ХНМУ
2016

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Foreword

Pathomorphology, one of the most important medical subjects is aimed at teaching students understanding material basis and mechanisms of the development of main pathological processes and diseases.

This manual published as separate booklets is devoted to general pathological processes as well as separate nosological forms. It is intended to the English-medium students of the medical and dentistry faculties. It can be used as additional material used both for home and individual work in class. It can also be used to master the relevant terminology and its unified teaching.

The manual is based on the syllabuses in Pathomorphology for Medical Students (2015).

For a practical class of 2 hour duration the following time calculation is recommended:

1. Determining the primary level of the knowledge – 5 min.
2. Independent work of the students – 50 min.
3. Determining the final level of the knowledge – 20 min.
4. Checking the protocols of the practical class and attestation of the students – 15 min.

The suggested Manual allows to organize the teaching process in the proper way.

References:


Lesson

Subject: Intracellular accumulations

Validation of the subject: The knowledge of the present subject is essential for successful understanding of the other chapters in general and specific pathomorphology. In the medical practice the knowledge of parenchymatous degenerations can be useful for diagnosis of cardiovascular, kidney, hepatic and other diseases.
Objectives of the lesson: to study the aetiology, pathogenesis, classification, morphological characteristics, possible outcomes and the role of parenchymatous protein, fat (lipid) and carbohydrate degenerations.

Visual aids

Annotated tables:
– classification of dysproteinoses
– causes and conditions causing degenerative processes
– morphogenesis of degenerative processes
– degenerative processes and diseases of the man
– classification and types of obesity and lipid degenerations
– classification of lipoidoses
– classification of dysfunctional carbohydrate metabolism
– types of glycogenosis

Coloured tables:
– spotty degeneration of the liver, kidney, myocardium
– reaction to fats and carbohydrates

Slides:
– granular degeneration of the liver and kidney
– lipid (fatty) degeneration of the myocardium
– lipid (fatty) degeneration of the liver
– glycogenic infiltration of the epithelium of the renal canaliculi

Macro specimen:
– granular degeneration of the kidney (dull swelling of the kidney)
– cutaneous horn (hyperkeratosis)
– lipid degeneration of the myocardium
– lipid degeneration of the liver
– lipid degeneration of the kidney
– spleen in Gaucher`s disease

Micro specimen:
– # 33 – granular degeneration of the kidney
– # 169 – keratinising type of squamous carcinoma of the skin
– # 44 – fat degeneration of the liver
– # 46 – fat degeneration of the myocardium
– # 152 – glycogen in kidneys

Electronic micrographs
– granular degeneration of the proximal tubules of nephrocytes
– granular degeneration of hepatocytes
– hydropic degeneration of hepatocytes

Questions to control basic knowledge:
1. Do you think granular degeneration is a reversible process?
2. Indicate the most common outcome of hyalin-drop degeneration:
   A. Reverse development.  
   B. Cell necrosis.
3. Name the signs, characterising fat degeneration of the liver:
   - A. Decrease in size.
   - B. Soft texture.
   - C. Increase in size.
   - D. Red colour of the parenchyma.
   - E. Yellowish-ochre colour of the parenchyma.
   - F. Hard texture.

4. Indicate which of the following parenchymatous degenerations belong to:
   1) lipoidosis; 2) glycogenosis:
   - A. Gaucher's disease.
   - B. Pompe's disease.
   - C. Girke's disease.
   - D. Tay-Sach's disease.
   - E. Niemann-Pick disease.
   - F. Anderson's disease.
   Answers: 1) yes; 2) – b; 3) – b, c, e; 4) 1 – a, d, e; 2 – b, c, e.

Stages of individual work in class

Study and describe macro specimen:

**Dull swelling of the kidney degeneration (granular degeneration).** Pay attention to the increase in size of the organ, flabby texture: characterise the type of the sectioned tissue. Pay attention on the state of the capsules.

What is related to the described changes?

**Hyperkeratosis «Cutaneus horn»** Pay attention to increased deposition of horny substance in the area of the nail bed. Describe the appearance of the macro specimen. What type of horny degeneration is discussed and what is the cause of this pathology?

**Fatty degeneration of the myocardium (tigers heart).** Pay attention to the organ size, expansion of the chambers, soft texture. Characterise the appearance of the sectioned myocardium, pay attention to the greenish-yellow colour. Describe appearance from the endocardial side.

What is related to the yellowish-white striations from the endocardial side, especially deeply expressed in muscles and trabecules of the heart ventricles?

**Lipid degeneration of the liver (goose’s liver).** Pay attention to the organ size, flabby texture, yellowish-ochre colour of the parenchyma. What is the cause of such changes? What are possible outcomes?

**Large white kidney – lipid degeneration of the kidney.** Pay attention to the organ size, flabby texture, white colour of the parenchyma. How do you characterise the type of the sectioned tissue? What are the causes of such changes? What are possible outcomes?

**Spleen in Gaucher's disease:** Describe the appearance of the macro specimen. Pay attention to the organ enlargement, changes in colour, texture. What are the causes of the changes in the appearance of the organ in Gaucher's disease? Pay attention to the nodular and diffuse nature of cerebrosid deposition.
Study, draw and describe the micro specimen

# 33 – granular degeneration of the kidney (stained with hematoxylin and eosin).

Using low magnification find convoluted tubules, pay attention to the presence of protein fragments coloured pink in their lumens. Using great magnification study the epithelium of the convoluted tubules, pay attention to the swelling of the cytoplasm causing the narrowing of the lumen of tubules, uneven colouring of cytoplasm because of the presence of protein grains, disappearance of some nucleus.

# 169 – keratinising type of squamous carcinoma of the skin;
(stained with hematoxylin and eosin).

Using low and great magnification find the complexes of neoplastic cells growing into the underlying tissues. Pay attention to the presence in the centre of complexes keratinized cells which form so called «cancerous pearls». Name the types of horny degeneration.

# 152 – glycogen in the kidneys (Shabadash reaction).

Using low and great magnification find accumulation of glycogen grains and granules in the lumens and epithelium of the convoluted tubules. The glycogen grains and granules are raspberry coloured. What disease is characterised by such condition in kidneys?

# 44 – fatty degeneration of the liver (stained with hematoxylin and eosin, Sudan III).

Pay attention that fats are accumulated mainly in the peripheral regions of the hepatic lobules. Stained with Sudan III, fat looks like orange drops, however stained with hematoxylin and eosin it looks like emptiness formed at the place of fat location. What fatty hepatic degenerations do you know?

# 46 – fatty degeneration of the myocardium (stained with Sudan III).

Pay attention to the orange colour of drops in cytoplasm of cardiomyocytes. Demonstrative specimen.

Study the electronograms

Granular degeneration of the nephrocytes of the proximal tubules. ×18000.

Pay attention to the swelling and homogeneity of mitochondria, numerous vacuoles in the cytoplasm and to the desquamation of microvilli

Granular degeneration of hepatocytes. ×15000.

Pay attention to the enlargement of the quantity and sizes of the mitochondria, enlargement of canaliculi of endoplasmic reticulum with ribosomes on the membrane.

Ballooning (hydropic) degeneration of hepatocytes. ×18000.

Pay attention to the enlargement of canaliculi of endoplasmic reticulum with forming of vacuoles, filled with flake-like content.
Krok questions:

1. Autopsy of the patient who had been ill with leukemia and died of increasing chronic anemia revealed an enlarged heart, dull, flabby, pale gray myocardium. There were yellow plaques and bands under the endocardium. Which pathologic process is observed in the heart?
   A. Parenchymal fatty degeneration.*  D. Mesenchymal fatty degeneration.
   B. Vacuole degeneration.  E. Functional hypertrophy.
   C. Hyalin-drop degeneration.

2. External examination of a newborn revealed dry dull pale skin with uneven surface and presence of gray scaling plates. Which type of degeneration is this pathology associated with?
   A. Horny.*  D. Fibrinoid swelling.
   B. Hydropic.  E. Mucoid swelling.
   C. Hyalin-drop.

3. Microscopic study of the biopsy material from the female patient who suffers from diabetes mellitus has revealed that the epithelium of narrow and distal segments of the tubules is high with light foamy cytoplasm. Staining with Best’s carmine revealed red grains in the cytoplasm of the epithelium and tubules. Which parenchymatous dystrophy is present?
   A. Protein.  D. Mucous.
   B. Fat.  E. Carbohydrate.*
   C. Hyalin-drop.

Study questions to control the knowledge:

1) Define the term "Lesion". Name the types of lesions.
2) Name the morphologic mechanism of degenerations.
3) Give the classification of degenerations.
4) Name the types of dysproteinoses. What macro-, micro- and electron-microscopic changes take place in the organs during dysproteinoses.
5) Outcome and functional significance of different kinds of dysproteinoses.
6) Name the hereditary degenerations related to amino acid metabolism disturbance.
7) What are the causes and mechanisms of fat degeneration.
8) Characterise the appearance of the heart, liver and kidneys in fat degeneration.
9) Which histochemical methods (reactions) will help to trace fat in the tissues.
10) Outcome and functional significance of parenchymatous fat degenerations.
11) Name systemic lipoidoses.
12) Give the modern classification of carbohydrate degeneration. State the histochemical reactions, necessary to reveal the presence of carbohydrates in tissues.
13) What are manifestations of carbohydrate dysfunction in diabetes mellitus.
14) Name glycogenoses.
15) Give the characteristics of carbohydrate degeneration related to disturbed metabolism of glycoprotein.
Terminology

Lesion, alteration, degeneration, decomposition, transformation, infiltration, hyperkeratosis, ichthyosis, leukoplakia, enzymopathy; grained, hyalin-drop, hydropic, balloon, horny degenerations, coagulation and colliquation necrosis, "tiger's" heart, "goose's" liver, lipidoses, glycogenosis.

Practical habits and skills

Students are be able to definite different types of parenchymatous degenerations, to differentiate the types of degeneration on the basis of investigation of macro- and micro- specimen. Use the knowledge for diagnosis of parenchymatous degenerations in clinics.

Revise the word-building elements:

de – lack of
dys – bad, painful, difficult
-ogenesis – development
-osis – abnormal condition
-trophy – development, norishment
gen – produce
patho – disease
lip – fat
glyco – sugar
glycogeno – glycogen
leuko – white
necro – death
muco – mucus

Lesson

Subject: Extracellular accumulations

Validation of the subject: studying the subject is necessary for understanding of the other subjects of general pathology and also as a guide to study the pathological anatomy of diseases (infectious, allergic, rheumatic as well as hypertension, atherosclerosis, kidney diseases, endocrinopathy). The knowledge of the cause and pathogenesis of mesenchymal degeneration is very important for understanding of clinical disciplines when studying them and also for medical practice.

Objective of the lesson: to study the aetiology, pathogenesis, classification, morphological changes in the connective tissues; possible outcomes and significance of mesenchymal degeneration in organ dysfunction.

Visual aids

Annotated tables:
– connective tissue structure;
– morphogenesis of mesenchymal degeneration;
– morphogenesis of amyloidosis;
– classification of amyloidosis.

Coloured tables:
– hyalinosis;
– amyloidosis;
– fat metabolism dysfunction;
– specific microscopic staining for amyloid.

Slides:
– "sago spleen";
– hyalinosis of spleen artery;
– arteriosclerotic nephrosclerosis;
– obesity of the heart.

Macro specimen
– "sago spleen";
– sebaceous (waxy) spleen;
– kidney amyloidosis;
– glased spleen (sugar-icing spleen);
– hyalinosis of scars;
– general obesity – adipose tissue;
– fat capsule of the kidney;
– obesity of the heart.

Micro specimen
– # 32 – mucoid swelling of the aorta wall in atherosclerosis;
– # 36 – hyalinosis of the spleen arteriole;
– # 38 – amyloidosis of the spleen (sago spleen);
– # 42 – amyloidosis of the liver;
– # 43 – obesity of the heart.

Questions to control basic knowledge:

1. Is amyloidosis a type of carbohydrate degeneration?
2. Name the types of mesenchymal protein degeneration:
   A. Mucoid swelling.  D. Amyloidosis.
   B. Turbid swelling.  E. Hyaline-drop degeneration.
   C. Hyalnosis.       F. Fibrinoid swelling.

3. What structures are changed in mucoid swelling:
   A. Collagenous fibres.  C. Main substance of connective tissue.
   B. Hepatocytes.       D. Epithelium of convoluted tubules.

4. Classify for 1 – protein; 2 – fatty; 3 – carbohydrate mesenchymal degeneration:
   B. General obesity.    E. Hyalinosis.
   C. Amyloidosis.        F. Mucus degeneration.

Answers: 1) no; 2) a, c, d, f; 3) a, c; 4) 1 – a, c, d, e; 2 – b; 3 – f.
Stages of individual work in class

Study and describe macro specimen:

Hyalinosis of the spleen capsule (glased spleen, sugar-icing spleen). Describe spleen capsule, colour, texture, outlook. Characterise the nature of capsule changing. Give the definition of the process, indicate previous condition. Characterise the level of reversibility.

Amyloidosis of the spleen (sago spleen). Describe the dimensions of the organ, its texture, colour, appearance on the section. Indicate the nature of process, localisation of amyloid. Define the process, indicate the steps of morphogenesis. Name specific microscopic staining for amyloid

Amyloidosis of the spleen (sebaceous or waxy spleen). Describe the dimensions of the organ, its texture, colour, appearance on the section. Indicate localisation of amyloid. Indicate the difference between <<sebaceous>> and <<sago>> spleen.

Amyloidosis of the kidney. Describe the dimensions of the organ, its texture, the width of cortical layer, the appearance of surface on the section. Name the diseases which can outcome to amyloidosis of kidney. What is the result of it?

Obesity of the hart. Determine the dimensions of the organ. Pay attention to the quality of fat under the epicardium. Pay attention the growth of fatty tissue in the heart wall on the section, more developed in the right portions. Characterise the type of dysfunctional fatty metabolism. Name the aetiology and mechanisms of development of the general obesity, its significance, outcome.

Atherosclerosis of aorta. Characterise the appearance, colour of the aortic intima. Define the nature of the changes, explain the mechanism of development, significance for the organism.

Study, draw and describe the micro specimen.

# 32 – mucoid swelling of the aorta wall (stained with toluidine blue). Pay attention to the difference in colour of unchanged regions and those with mucoid swelling. What are the substances accumulated in the region with mucoid swelling? What are their properties? Name the diseases and conditions which are accompanied by mucoid swelling. Name the outcomes of mucoid swelling.

# 36 – hyalinosis of the spleen arteriole (stained with hematoxylin and eosin). Using low magnification find the central arteries of the spleen follicle. Using great magnification study the width of the vessel wall and its lumen, the condition of internal and external tunics. Explain the mechanisms of development of hyalinosis of the spleen arteriole. Determine the outcome and significance.

# 42 – amyloidosis of the liver (stained with Congo red). Describe the localisation of the amyloid, its appearance. Characterise the significance and outcome of liver amyloidosis

# 38 – amyloidosis of the spleen (sago spleen) (stained with hematoxylin and eosin, Congo red). Pay attention to the colour and localisation of amyloid in definite structures of the spleen and to the conditions of cellular elements of the pulp.
# 43 – obesity of the heart (stained with hematoxylin and eosin). Describe the level of development of subepicardial fat, conditions of the adjacent muscular fibers. Name specific microscopic staining. Significance of obesity for the organism.

**Study the electronograms: amyloidosis of kidney.**
Pay attention to localisation and structure of amyloid mass in glomerular filter, to the width of basal membrane.

**Questions to control the knowledge:**

1. Name morphogenetic mechanisms of development of mesenchymal degenerations.
2. Name the main causes of fibrinoid changing.
3. As outcome of which pathological processes can hyalinosis develop? Enumerate the types of vascular hyalinosis.
4. Classification of amyloidosis.
5. Stages of amyloid morphogenesis. Theories of amyloid pathogenesis.
6. The causes and types of obesity.

**Krok questions:**

1. Microscopy of the kidneys from a man died of systemic lupus erythematosus revealed sclerosed glomeruli, the lumens of the small arteries and arterioles are narrow, the median membrane is thin, homogeneous, eosinophilic masses are present in the subendothelial space. Immunologically these masses contain immune complexes and fibrin. Which substance is present in the subendothelial space?
   - A. Fat-Protein detritus.
   - B. Simple hyalin.
   - C. Lipohyalin.
   - D. Complex hyalin.*
   - E. Amyloid.

2. Microscopy of the internal organs of the patient who had suffered from rheumatism and died of cardiac decompensation showed that the bands of collagen fibers of the organs were saturated with plasma proteins, were homogenous, eosinophilic, picrinophilic when stained according to vas Gieson, PAS-positive, pironinophilic at Brachet’s reaction and argyrophilic at impregnation with silver salts. Which pathological process in the connective tissue is most probable?
   - A. Mucoid swelling.
   - B. Fibrinoid swelling.*
   - C. Fibrinoid necrosis.
   - D. Hyalinosis.
   - E. Amyloidosis.

3. In 53 year-old patient suffered from bronchoectatic disease and hemoptysis, the edema of face and waist have appeared. The protein (33 mg/l) was found in urine. Pulmonary hemorrhage was the cause of patient’s death. In autopsy: enlargement of kidneys was found; the kidneys were densed with lardaceous surface of section. Histologically: the deposition of homogenous eosinophilic masses colored with Congo red and given of metachromasia with methyl violet color in glomeruli and canals were found. What pathological process took place in the patient?
   - A. Amyloidosis.*
   - B. Grainish degeneration.
   - C. Fatty degeneration.
   - D. Mucoid degeneration.
   - E. Hyalinosis.
Terminology
Histion, plasmatic saturation, mucoid swelling, fibrinoid swelling, hyalinosis, amyloidosis, metachromasia, plasmorrhagia, keloid, glazed (sugar-coating) spleen, sago-like spleen, sebaceous spleen, obesity, cachexia.

Practical habits and skills
On the basis of the gained knowledge the students are to diagnose the mesenchymal degenerations and to be able to interpret them from the clinico-anatomical point of view.

Revise the word-building elements:
meso – middle
-osis – abnormal condition
-oid – resembling
-rrhagia – bursting forth
-emia – blood condition
-sclerosis – hardening
muco – mucus
fibrino – fibrin
plasmo – plasma
athero – plaque
nepho – kidney
uro – urine

Lesson
Subject: Pigments pathology (Mixed degenerations).
Chromo- and nucleoprotein metabolism disturbance

Validation of the subject: the knowledge of mixed degenerations, manifesting themselves in the metabolism of chromo- and nucleoprotein in the stroma and parenchyma is essential to study many diseases of the lungs, liver, kidneys, as well as those of the blood, endocrine system ect. Mixed degeneration can be hereditary or acquired. General knowledge about dysmetabolism of compound proteins is important for understanding the pathogenesis of haemolytic disease, malaria and other diseases.

Objective of the lessons: the students are to be able to differentiate mixed dystrophy from the other pathological processes. Compound proteins (chromo-, nucleo- and lipoproteids) play an important role in the life of the organism therefore it is necessary to study the conditions accompanied by accumulation of pigments which are normally produced or appear in pathological conditions. During the classes, it will be necessary to give definition of mixed degenerations, name their types, study the aetiology, pathogenesis, classification, morphological changes and also the possible outcomes and the importance for the organism.
Visual aids

Annotated tables:
– metabolic dysfunction in tissues;
– pigments and dysfunctional pigments metabolism;
– chromoproteins;
– haemosiderosis.

Coloured tablets:
– metabolic dysfunction of nucleoproteins;
– brown induration of the lungs.

Slides:
– brown induration of the lungs;
– liver in posthepatic jaundice.

Macro specimen:
– brown induration of the lungs;
– haemosiderosis of the spleen;
– liver in posthepatic jaundice;
– spleen hemomelanosis;
– melanoblastoma of the skin;
– liver and pancreas in hemochromatosis.

Micro specimen:
– # 2–3 – brown induration of the lungs;
– # 67 – liver in posthepatic jaundice;
– # 65 – kidney hemosiderosis;
– # 63 – spleen hemomelanosis;
– # 170 – melanoblastoma of the skin.

Electronograms:
– Brown induration of the lungs. Siderophag is a cell of heart Defect;
– Deposition of lipofuscin in the myocardium;
– Molecules of ferritin in the grains of hemosiderin.

Questions to control basic knowledge:

1. Does haemosiderin contain iron (Fe)?
2. Indicate which of the following diseases and procedures cause to general hemosiderosis:
3. In which disease can we notice widespread melanosis:
4. Which of the pigments are lipogenic:
5. Name, which hemoglobin pigments are produced in normal (i) and pathological (ii) states:
   A. Bilirubin.  
   B. Hematin.  
   C. Hemosiderin.  
   D. Hematoidin.  
   E. Ferritin.  
   F. Porfirin.
   Answers: 1) yes, 2) a, c, d, e, 3) d, 4) b, d, e, f, h, 5) i) a, c, e; ii) b, d, f.

**Stages of individual work in class**

**Study and describe macro specimen:**

*Brown induration of the lungs.* Determine the colour and texture of the specimen under section. In which diseases can we find such pathologies? What reactions do you know which can help to trace iron containing pigments?

*Spleen hemosiderosis.* Describe the appearance of the organ, its colour, size. In which diseases can we find general hemosiderosis?

*Spleen hemomelanosis.* Describe the appearance of the organ, its colour and size. In which disease can we find spleen hemomelanosis?

*Liver in posthepatic jaundice.* Describe the appearance of the organ, its colour, the condition of bile ducts. Name disease the which can be characterised by mechanical jaundice. What is the outcome of bile stasis?

*Melanoblastoma of the skin.* What is the appearance of the skin? Which pigment is responsible for such skin colour? Which group of pigments is it? How is it classified according to the spread of the process?

**Study, draw and describe the micro specimen**

#2–3 *brown induration of the lungs* (stained with hematoxylin and eosin, Prussian blue reaction). Name the organ, determine where hemosiderin is accumulated. What is its colour in Prussian blue reaction? What changes are found in alveoli’s walls? What is the possible colour of sputum? What is the outcome of this pathology?

#67 *liver in posthepatic jaundice* (stained with hematoxylin and eosin). Describe the condition of the bile ducts, capillaries, hepatocytes in posthepatic jaundice, the outcome. When does mechanical jaundice develop most frequently?

Study electronograms: brown induration of the lungs. Find siderophag which is cell of heart defect.

**Questions to control the knowledge:**

1. What is hemosiderosis? Name its types.
3. Name types of jaundice depending on the way of its development.
4. Which pathology is caused by bile stasis?
5. Where is hemomelanin accumulated and in what disease? What is the colour of the organs?
6. In what diseases does porphyria develop?
8. Give the examples of general and acquired melanosis.
9. What conditions and diseases cause to increased quantity of lipofuscin?
10. Name end products of nucleic acid decomposition.

Krok questions:
1. Gastroscopy revealed an ulcer with dense borders and black-brown bed in the gastric mucosa. Microscopy revealed black-brown pigment on the necrotic layer in the ulcer bed. Which pigment is it?
   A. Hydrochloric hematin.*
   B. Porphyrin.
   C. Bilirubin.
   D. Ferritin.
   E. Hemosiderin.
2. An 80-year-old man dies from complications of Alzheimer disease. In autopsy, his heart is small (250 gm) and dark brown on sectioning. Microscopically, there is light brown perinuclear pigment with H&E staining of the cardiac muscle fibers. Which of the following substances is most likely increased in the myocardial fibers to produce this appearance of his heart?
   A. Lipochrome from "wear and tear".*
   B. Hemosiderin resulting from iron overload.
   C. Glycogen resulting from a storage disease.
   D. Cholesterol as a consequence of atherosclerosis.
   E. Calcium deposition following necrosis.
3. In patient with jaundice the following data were established: in serum the increasing of bilirubin because of the unconjugated form; in faeces and urine increasing of stercobilin; the level of conjugated (direct) bilirubin in serum is normal. What type of jaundice takes place?
   A. Hemolytic jaundice.*
   B. Parenchymatous (hepatic) jaundice.
   C. Gilbert’s disease.
   D. Jaundice of newborns.
   E. Mechanical (posthepatic) jaundice.

Terminology
Hemosiderin, hemosiderosis, ferritin, ferritinemia, bilirubin, bilirubinemia, hematoidin, hematin, porphyrin, sideroblast, brown induration of lungs; hemolytic, obstructive and parenchymatous jaundice, hemomelanin, melanin, melanosis, large intestine melanosis, nevus, melanoma, leukoderma, vitiligo, lipofuscin, gout, urolithiasis, uric acid infarct of the newborn.

Practical habits and skills
Students are to be able to define metabolic dysfunction for hemosiderin and melanin, to differentiate different types of jaundices. To use knowledge for diagnosis of the parenchymatous degenerations in clinics.
Lesson

Subject: Disturbances in electrolyte (mineral) metabolism.

Necrosis

Validation of the subject: The knowledge of disturbances in electrolyte (mineral) metabolism is essential for understanding general changes occurring in general pathological processes (necrosis, organisation, tumours) and for studying a group of diseases (metabolic disturbances, those of cardiovascular systems, liver, kidney, endocrine system, musculoskeletal system). The knowledge of necrosis is important to understand the materials of the general course (disturbances in circulatory system, inflammation, immunopathological and compensatory-adaptational processes, tumours) as well as the outcome of many diseases, to study clinical subjects and for clinical anatomical analysis.

Objectives of the lesson: to understand the significance of disturbances in electrolyte (mineral) metabolism, the mechanism causing the processes, the causes, morphological and functional significance of the disturbances of calcium (Ca), potassium (K), iron (Fe) metabolism. The students are to study aetiology, pathogenesis, appearance and microscopic changes in necrosis, clinicomorphological forms, the outcome, functional significance of necrosis. The students of the paediatric faculty are to know the peculiarities of mineral metabolism and necrosis in children. For the dentist, the knowledge of calcium and fluorine (F) metabolism is essential.
Visual aids

Annotated tables:
– disturbances in electrolyte (mineral) metabolism;
– morphogenesis of tissue calcification;
– formation of the stones;
– necrosis.

Colour tables:
– metastatic calcification;
– petrifcates;
– stones in kidney and gallbladder;
– gangrene of the foot;
– macroscopic changes in necrosis;
– microscopic changes in necrosis.

Slides:
– necrosis of the kidney epithelium;
– tubercular caseous lymphadenitis.

Macro specimen:
– calcificated fibromyoma of the uterus;
– femur in parathyroid osteodystrophia;
– stones in gallbladder;
– stones in kidney;
– gangrene of the foot;
– anaemic infarction of the spleen;
– haemorrhagic infarction of the lung.

Micro specimen:
– # 55 calcified capsule of thyroid gland;
– # 73 necrosis of the kidney epithelium;
– # 75 tubercular caseous lymphadenitis.

Electronograms:
– calcified metastases in the myocardium;
– cell necrosis (karyoplcnosis).

Questions to control basic knowledge:

1. Do you think vascular necrosis is a kind of indirect necrosis?
2. Name the organs which take part in regulation of calcium metabolism:
   A. Liver.
   B. Parathyroid glands.
   C. Spleen.
   D. Heart.
   E. Thyroid gland.
   F. Lungs.
3. Name in which organs we often notice deposition of calcium salts during metastatic calcification:
   A. Skin.
   B. Lungs.
   C. Stomach.
   D. Spleen.
   E. Kidneys.
   F. Heart.
   G. Arteries.
4. Name the local causes of stone formation:
   A. Secretion disturbances.  C. Secretion stasis.
   B. Metabolic disturbances.  D. Inflammation.

5. Classify the groups of the following necroses: (i) according to etiological signs (ii) according to morphological signs:
   A. Vascular.  D. Ceraceous (waxy).  G. Colliquative
   B. Caseous.  E. Traumatic.
   C. Toxic.  F. Gangrene

   Answers 1. Yes 2. b, e; 3. b, c, e, f, g; 4. a, c, d; 5. (i) a, c, e, (ii) b, d, f, g.

Stages of individual work in class

Study and describe the macro specimen

1. Calcified fibromyoma of the uterus. Describe the shape, size, texture and colour of the calcified area under section. State the possible changes in the tumour, which caused deposition of calcium salts. What mechanism caused this type of calcinosis?

2. Femur in parathyroid osteodystrophy. Describe the states of the soft and compact core (part) of the bone. What changes occur in the bone tissue? What mechanism caused this type of calcinosis and what are the possible organs where we can find deposition of calcium salts.

3. Gallstones. Determine the size of the gallbladder. What has filled its cavity? Describe the colour of the stones, the size and the surface. Name the stones of the bile duct according to their chemical composition.

4. Renal calculi (stones). The appearance of the stones, their shape and surface. Pay attention to the changes in renal tissues. What is the possible outcome of renal stones?

5. Gangrene of the foot. Pay attention to the changes in the colour (causes and pathogenesis), texture. Give the definition of the process and indicate the border of the unchanged tissue and name it.

6. Anaemic infarction of the spleen. Find the location, shape, colour and size of the area of necrosis. What type of necrosis is it and what are the causes and mechanisms which caused its development?

Study, draw and describe the micro specimen.

# 55 – calcified capsule of the thyroid gland (stained with hematoxylin and eosin). Pay attention to the staining with hematoxylin of the large focus of the sclerotised and hyalinated capsule of the thyroid gland. Which type of calcinosis is it?

# 73 – Necrosis of the renal epithelium (stained with hematoxylin and eosin). Determine the microscopic changes which characterise necrosis within the proximal and distal tubules of the kidney. Determine cell swelling, absence of nucleus, homogenic nature of the cytoplasm, the narrowing of the tubules lumen and the presence of eosinophilic grains in it.

# 75 – Tubercular caseous lymphadenitis (stained with hematoxylin and eosin). Demonstrational specimen
Study the electronograms
1. Calcified metastases in myocardium. Determine the organelles in which calcium salts are deposited.
2. Cell necrosis, karyopicnosis. Pay attention to the decrease in the size of the nucleus, high density of the karyoplasm; nucleolus can not be differentiated.

Questions to control the knowledge:
1. Name the importance of microelements for the organism.
2. In which processes do calcium salts takes place?
3. By what is calcium metabolism regulated in the organism?
4. Name the types of calcification according to their mechanisms.
5. In which organs does petrification often occur?
6. In which organs does deposition of calcium salts occur during metastatic calcification?
7. Name the diseases, which are accompanied by hyper- and hypo-kalemia.
9. Name the general and local causes of stone formation.
10. Name the types of cholelithiasis according to the chemical components.
11. Name the types of uric stones according to theirs components.
12. Outcomes of urolithiasis.
13. Define the term necrosis.
14. State the stages of necrosis.
15. Describe the changes in micro and macro specimen in necrosis.
16. Types of necrosis according to the mechanisms and the aetiology.
17. Clinico-morphological forms of necrosis.
18. Define the term "gangrene". Its types.
19. The outcome of necrosis.
20. The importance of necrosis for the organism.

Krok questions:
1. Autopsy of a woman who had suffered from rheumatism with combined mitral defect showed that the cusps of the mitral valve are thickened, adhere to each other, stone-like. Which pathological process is responsible for the stone-like density of the heart valves?
   A. Metastatic calcification.
   B. Metabolic calcification.
   C. Hyalinosis.
   D. Amyloidosis.
   E. Dystrophic calcification.*
2. Fragment of dead tissue, which can’t be autolized, replaced by connective tissue and which is localized among alive tissue is named…
   A. Sequestrum.*
   B. Dry gangrene.
   C. Wet gangrene.
   D. Infarction.
   E. Caseous necrosis.
3. Call a kind of an infarct according to macroscopic signs, which is characteristic in myocardium.

A. White with a hemorrhagic halo.*  C. Anemic.  E. Red.

**Terminology**

Osteoporosis, osteomalacia, calcinosis, petrification, ossification, microlith, macrolith, lithopedion, caprolith, sialolith, broncholith, arteriolith, phlebolith, cholelithiasis, nephrolithiasis, hypercalciemia, gout, calcifilaxia, necrosis, paraneerosis, necrobiosis, autolysis, karyopcinosis, karyorhexis, karyolysis, plasmorhexis, plasmolysis, cytology, myomalacia, encephalomalacia, mummification, infarct, transplantation, gangrene, line of demarcation, organisation, encapsulation.

**Practical habits and skills**

The students are to be able to diagnose necrosis and diseases according to the morphological signs, which caused the disturbances in electrolyte metabolism.

**Revise the word-building elements:**

hyper – increased
micro – small
macro – large

-ogenesis – formation, development
-oma – tumor
-osis – pathological condition
-emia – blood condition
-lysis – break down
-malacia – softening
-rhexis – rupture
-lith – stone
 necro – death
 morpho – shape
 osteo – bone
 calcio – calcium
 sialo – saliva
 broncho – bronchus
 arterio – artery
 phlebo – vein
 chole – bile
 nephro – kidney
 pancreo – pancreas
 bio – life
 auto – self
 karyo – cell
 cyto – cell
Навчальне видання

ДИСТРОФІЇ І НЕКРОЗ

Методичні вказівки до заняття з патоморфології для студентів медичних вузів з англійською мовою навчання

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Свідоцтво про внесення суб’єкта видавничої справи до Державного реєстру видавництв, виготовників і розповсюджувачів видавничої продукції серії ДК № 3242 від 18.07.2008 р.
CELL INJURY AND NECROSIS

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