**STATE UNIVERSITY OF MEDICINE AND PHARMACY**

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**INDIVIDUAL WORKBOOK**

**FACULTY OF DENTISTRY**

**Student: .........................................................**

**Year: .................. Group: ..................**

**Faculty: .........................................................**

**Chișinău, 2025**

**Topic 1: Cell damage. Necrosis. Apoptosis. Cellular dystrophies. Features of cellular injury in the oral cavity**

**Clinical case 1**

Patient A., 55 years old, who suffers from atherosclerosis, was urgently admitted with the following complaints: retrosternal pain radiating to the shoulder blade and left arm (nitroglycerin administration does not alleviate the pain), general weakness, and sweating. Presumptive diagnosis – myocardial infarction.

**Objective:** pallor, cold and moist skin;

**ECG:** ST-segment elevation; pathological Q wave.

**Blood:** elevated total CK (creatine kinase), CK-MB > 190 U/L (Normal < 24 U/L), troponin I, myoglobin, LDH, AST, lactate. Hypercholesterolemia. Hyperkalemia.

**Questions:**

**1. What are the pathogenetic mechanisms of hypoxic injury? (Example of a pathogenetic chain)**

**2. What is the pathogenetic mechanism of hypoxic injury through the cessation of Na⁺/K⁺ pump activity?**

**3. What is the pathogenetic mechanism of hypoxic injury through the cessation of Ca²⁺ pump activity?**

**4. What is the pathogenetic mechanism of hypoxic injury through the activation of anaerobic glycolysis?**

**5. What is the role of oxidative stress in the pathogenesis of cellular hypoxic injury?**

**6. What indicators confirm cardiomyocyte injury in the patient?**

**7. What is the mechanism of hyperkalemia, and what electrophysiological changes are observed in the patient as a result?**

**Clinical case 2**

At the first breastfeeding, the mother noticed that the new-born was suffocating and feeding was impossible. Examination of the new-born revealed a defect of the hard palate in the form of a cavity (“cleft palate”).

**Questions:**

**1. What disorder of the apoptosis process developed in the child during the embryonic period? Definition of apoptosis. Examples of pathologies resulting from excess or deficiency of apoptosis.**

**2. The intrinsic mechanisms of apoptosis. The role of the BCL-2 family proteins, Bax, Bad, Apaf-1.**

**3. The extrinsic mechanisms of apoptosis. The role of TNF, FAS-L.**

**4. What are the criteria for differentiating apoptosis from necrosis.**

**5. What role do mitochondria play in the process of apoptosis? Argue.**

**6. What are the positive and negative signals for the initiation of the apoptosis process?**

**Clinical case 3**

Patient G., 52 years old, was admitted to the hepatology department with a preliminary diagnosis of "hepatitis." From the medical history, for 20 years he has been under the care of a narcologist with the diagnosis of "chronic alcoholism."

Objective - enlarged liver with soft consistency.

Liver biopsy performed for diagnostic purposes: Hematoxylin and eosin staining revealed cytoplasmic vacuolization of hepatocytes, and Sudan staining showed fat droplets.

Biochemical analysis: Moderately elevated AST and ALT, elevated triglycerides.

**Questions:**

**1. What causes contribute to fatty liver disease?**

**2. Through what mechanisms has alcohol contributed to the development of the dystrophy?**

**3. What metabolic processes have been affected and contributed to the accumulation of lipids in hepatocytes?**

**4. How does oxidative stress contribute to the damage of hepatocyte structure and to the intracellular accumulation of lipids?**

**5. What is the pathogenetic mechanism of dystrophies affecting the mitochondria?**

**6. What is the pathogenetic mechanism of dystrophies affecting the functioning of the cell membrane pumps?**

**7. What are the possible consequences of hepatic dystrophy?**

**Topic 2: Disorders of the regenerative process. Atrophy. Hypertrophy. Hyperplasia. Sclerosis. Features of adaptive processes in the oral cavity**

**Clinical case 1**

In patient N., 60 years old, which suffer for 30 years - arterial hypertension, the echocardiographic examination revealed an increase in myocardial mass and thickening of the left ventricular wall.

Microscopic examination: left ventricle - diffuse proliferation of connective tissue; cardiomyocytes enlarged in volume, with a large nucleus. Electron microscopy shows an increase in the number and size of cellular organelles (mitochondria, endoplasmic reticulum, ribosomes, Golgi apparatus).

**Questions:**

**1. What are the pathogenic mechanisms of hypertrophy under conditions of cardiac overload?**

**2. What are the triggers for genome modification that activate the anabolic process at the cardiomyocyte level?**

**3. What are the common and distinctive signs of physiological and pathological hypertrophy of the myocardium?**

**4. What is the difference between hypertrophy and hyperplasia in the regenerative process?**

**5. What are the types of pathological hypertrophy? Examples.**

**6. What are the types of physiological hypertrophy? Examples.**

**Clinical case 2**

Patient X, 40 years old, was admitted to the gynaecology department with the following complaints: metrorrhagia lasting 8 days, moderate pain in the suprapubic region, and general weakness.  
**Laboratory analysis:** hyperestrogenism.

**Physical examination:** pallor, BP = 90/60, pulse = 105, abdomen bloated, soft, painful in the suprapubic region.

**Ultrasound:** endometrium is heterogeneous in structure, thickened.  
**Histological examination:** complex endometrial hyperplasia without nuclear atypia.

**Questions:**

**1. What is endometrial hyperplasia?**

**2. What factor induces the hyperregenerative process in the endometrium?**

**3. What is the mechanism by which estrogens induce the hyperplasia process?**

**4. Which tissues undergo exclusively hyperplastic processes and why?**

**5. Which tissues undergo exclusively hypertrophic processes and why?**

**6. What is the difference between hyperplasia and metaplasia?**

**Clinical case 3**

Patient R., 45 years old, was admitted to the endocrinology department with the diagnosis of "secondary hypothyroidism," presenting the following complaints: fatigue, drowsiness, memory loss, hair loss, brittle nails, and weight gain.

For diagnostic purposes, a thyroid gland biopsy was performed. Microscopic examination revealed a reduction in the number and size of follicles.

**Questions:**

**1. What typical pathological process in the thyroid gland has developed in this patient? Provide arguments.**

**2. What pathogenic factors contribute to the decrease in the number, size, and function of the thyroid gland in this patient?**

**3. What is the relationship between the intensity of the anabolic and catabolic processes in the pathogenesis of atrophy?**

**4. What types of pathological atrophy do you know? Mechanisms of pathological atrophy. Examples.**

**5. What types of physiological atrophy do you know? Examples.**

**Clinical case 4**

A 55-year-old man with a 20-year history of chronic viral hepatitis C presents to the doctor with symptoms of persistent fatigue, moderate jaundice, and abdominal enlargement.  
**Abdominal ultrasound:** confirms a nodular structure of the liver and ascites.  
**Liver biopsy:** indicates excessive deposition of connective tissue in the liver parenchyma, macronodules, and signs of inflammation.

**Questions:**

**1. What is the typical pathological tissue process in the liver observed in this patient?**

**2. What etiological factors contribute to the development of hepatic cirrhosis?**

**3. What is the role of chronic inflammation in the pathogenesis of sclerosis?**

**4. What are the sources of sclerosis?**

**5. What is collagenogenesis and collagenolysis and what is the balance of these processes (collagenogenesis and collagenolysis) in this patient?**

**6. Which type of macrophages, M1 or M2 (classically or alternatively activated), activate fibroblasts and subsequently lead to excessive extracellular matrix deposition?**

**7. Which cytokines are important fibrogenic factors, and what is their role?**

**8. What conditions would be necessary for physiological regeneration in the liver?**

**Topic 3: Regional blood circulation disorders. Arterial hyperemia. Venous hyperemia. Stasis. Thrombosis. Embolism. Ischemia.**

**Clinical case 1**

Patient L., a 30-year-old woman, presents to the dental office complaining of intense pulsating pain in the area of the lower right molar, associated with a sensation of local warmth and gingival swelling. Upon clinical examination, the gum surrounding the tooth is inflamed, hyperaemic (bright red in colour), and there is increased sensitivity to touch.

**Questions:**

**1. What type of microcirculation disorder is observed in the patient? Justify your answer with the described external signs.**

**2. Pathogenesis of neurogenic arterial hyperemia.**

**3. Pathogenesis of metabolic arterial hyperemia.**

**4. Pathogenesis of humoral arterial hyperemia.**

**5. What are the specific microcirculatory hemodynamic changes associated with arterial hyperemia?**

**6. What are the consequences of arterial hyperemia? List the consequences with positive and negative biological significance.**

**Clinical case 2**

Patient F., 52 years old, underwent surgery for the removal of a femur tumor. During the procedure, the femoral artery was injured. The artery was sutured, and the distal pulse of the artery was restored. After 24 hours, the patient complains of severe pain in the distal region of the operated leg, the pulse in the distal region (at the calf level) is not palpable, the patient cannot move the toes of the operated leg, the skin of the leg is pale, and the local temperature is reduced.

**Questions:**

**1. What type of microcirculatory disorder has developed in the patient? Argument.**

**2. What is the mechanism of paleness and reduced local temperature in the distal region of the operated leg?**

**3. What is the etiological factor of this microcirculatory disorder in this patient?**

**4. What is the pathogenetic mechanism of this microcirculatory disorder?**

**5. What other pathogenetic mechanisms of this pathological process do you know?**

**6. What is the pathogenetic mechanism of the pain in this patient?**

**7. What are the possible consequences of this pathological process?**

**Clinical case 3**

Patient H., 38 years old, was admitted to the trauma department with an open fracture of the left femur with fragment displacement. During the repositioning of the bone fragments, the patient developed shortness of breath, acrocyanosis, and a systolic blood pressure of 40 mm Hg, with diastolic pressure undetectable. After 10 minutes, the pulse in the carotid arteries disappeared, the pupils dilated, and clinical death was confirmed.

**Questions:**

**1. What type of microcirculatory disorder developed in the patient?**

**2. What is the pathophysiological mechanism of this microcirculatory disorder in this patient?**

**3. What is the path of embolus circulation, considering the patient's clinical symptoms that led to death?**

**4. What other types of embolism do you know, based on their origin (give examples)?**

**Clinical case 4**

Patient E., 43 years old, was admitted to the cardiology department with the diagnosis of "right atrioventricular orifice stenosis." She complains of fatigue, leg pain, and edema (which worsens in the evening), and cyanotic discoloration of the lips, ears, and nail beds. Objectively, acrocyanosis, hard edema localized in the calves, and hepatomegaly are observed.

Bio-microscopy of the nail bed microvessels revealed dilated venules with erythrocyte extravasation. Central venous pressure is 15 cm H₂O.

Blood biochemistry: ALAT - 80 UI/L; ASAT - 100 UI/L, Ht - 0.59. Hb - 160 g/L, Er - 5.5 x 10¹²/L.

**Questions:**

**1. What type of microcirculatory disorder do you suspect, considering the cause and clinical manifestations in the patient?**

**2. What compound causes cyanosis and acrocyanosis in venous hyperemia?**

**3. How do you explain the increased activity and levels of ALAT and ASAT in this patient?**

**4. What is the pathogenesis of stasis in right-sided heart failure?**

**5. What are the hemodynamic changes in venous hyperemia?**

**6. What are the metabolic changes in venous hyperemia?**

**7. What is the pathogenic link of hydrostatic edema in right-sided heart failure?**

**8. What is the pathogenic link of hyperosmolar edema in right-sided heart failure?**

**9. What is the pathogenic link of hypo-oncotic edema in right-sided heart failure?**

**10. What is the pathogenic link of membranous edema in right-sided heart failure?**

**Topic 4: The physiopathology of the fluid-coagulant system. Hypo-, hypercoagulation. Clinical Manifestations and Implications in Dental Practice**

**Clinical Case 1**

Patient M., 58 years old, presents to the dentist for a dental implant procedure. From the medical history, the patient suffers from deep vein thrombosis in the lower limbs. She has been smoking for 30 years. It is also known that the patient has thrombophilia (a genetic condition characterized by antithrombin III deficiency), diagnosed in childhood.

General blood analysis: thrombocytosis.

Two weeks ago, she stopped anticoagulant treatment.

The dentist is concerned about the complications that this coagulant-fluid system pathology might cause during the procedure.

**Questions:**

**1. What type of coagulant-fluid system disorder is present in the patient?**

**2. What etiological factors contribute to the dysfunction of the coagulant-fluid system in this patient?**

**3. What components of Virchow's triad are present in this patient?**

**4. What other types of thrombophilia do you know?**

**5. What is the pathogenic mechanism of blood hypercoagulability in cases of arterial hypertension?**

**6. What is the pathogenic mechanism of blood hypercoagulability in cases of genetic antithrombin III deficiency?**

**7. What are the risks in dental practice related to the state of hypercoagulability? What other mechanisms contribute to the state of hypercoagulability?**

**Clinical case 2**

A 60-year-old man presents to the dental office for a tooth extraction. In his medical history, the patient mentions suffering from liver insufficiency. He has also noticed mild bleeding from his gums during tooth brushing.

On clinical examination, slightly inflamed gums were observed, with mild spontaneous bleeding upon touch. The patient has slight bruising on his forearms, suggesting a possible coagulopathy.

Laboratory tests: Prothrombin time (PT) and activated partial thromboplastin time (aPTT) – both are prolonged.

Complete blood count: moderate thrombocytopenia.

The dentist is concerned about the complications that the patient’s fluid-coagulation system pathology could cause.

**Questions:**

**1. What type of fluid-coagulation system disorder is present in the patient?**

**2. What etiological factors lead to the dysfunction of the fluid-coagulation system in this patient?**

**3. What is the pathogenic mechanism of coagulopathy in the case of liver insufficiency in this patient?**

**4. What is the pathogenic mechanism of thrombocytopenia in this patient?**

**5. What do prothrombin time (PT) and activated partial thromboplastin time (aPTT) represent?**

**6. What is the difference between thrombocytopenia and thrombocytopathies?**

**7. What are the risks in dental practice related to the state of hypocoagulability? What other mechanisms contribute to the state of hypocoagulability?**

**Topic 5: Inflammation. Etiology. Pathogenesis. Features of inflammation oral cavity.**

**Clinical Case 1**

Patient S., 26 years old, presented to the dentist with acute pain in the premolar region on the right side of the mandible, which intensifies with chewing. Upon inspection of the oral cavity, localized hyperemia was observed in the area of the first premolar, along with swelling of the adjacent tissues and mucosal edema. The pain worsened with slight pressure on the tooth. The submandibular lymph nodes were enlarged and painful upon palpation. The dentist diagnosed “acute periodontitis.”

Blood work: neutrophilic leucocytosis;

Blood biochemistry: fibrinogen - 8g/l (normal 2-4g/l), C-reactive protein – 6mg/l (normal 0.7-2.3mg/l), Amyloid A – elevated levels.

**Questions:**

**1. Name the virulence factors through which microorganisms in the oral cavity can cause lesions and induce a local inflammatory response.**

**2. What mechanisms underlie the transvascular diapedesis of leukocytes, and what is their significance?**

**3. What explains the localized gingival hyperemia in the region of the first premolar?**

**4. Indicate what mechanisms may underlie the localization of the inflammatory focus and reduce the risk of pathogen dissemination.**

**5. What is the pathophysiological mechanism of mucosal edema in this patient?**

**6. List the cellular and humoral chemotactic substances that contribute to the process of phagocytosis in the inflammatory focus.**

**7. List the bacterial chemotactic substances that contribute to leukocyte migration and the process of phagocytosis.**

**8. What are the general changes in the body during inflammation, and by what indicators can they be confirmed in the patient?**

**Clinical Case 2**

A 55-year-old patient presented to the dentist with the following complaints: periodic pain in the upper right maxillary tooth when consuming hard and hot foods, as well as purulent discharge from the right side of the nasal cavity. The patient mentioned that the tooth pain had persisted for a long time, with episodes of sharp pain that intensified during chewing. The tooth had not been treated previously.

**Objective examination:** Facial configuration is unchanged. Regional lymph nodes are not palpable. Mouth opening is free.

**Local examination:** The crown of tooth 1.6 is completely destroyed, and percussion is painful. The gingival mucosa around tooth 1.6 is hyperaemic and edematous.  
 **Intraoral X-ray:** At the apex of the buccal root of tooth 1.6, there is bone tissue destruction with unclear borders, measuring 0.4 x 0.6 cm.

**Questions:**

**1. What is the difference between acute and chronic inflammation?**

**2. What is the role of mesenchymal cells in the evolution of inflammation?**

**3. What is the pathophysiological mechanism of gingival hyperemia in this patient?**

**Topic 6: Allergy. Allergic reactions type I, II, III, IV. Anaphylactic Shock in Dental Practice. Local immunodeficiencies. Autoimmune injuries**

**Clinical Case 1**

Patient B., 43 years old, presented to the dentist with acute pulpitis, the diagnosis being confirmed by dental X-ray. Initially, the dentist administered 3 ml of 2% lidocaine locally in order to perform the pulpitis treatment. After 5 minutes the patient began to complain of: heat sensation in the oral cavity, itching, swelling of the lips and tongue, headache, dizziness, dyspnea, and hypersalivation.  
Objective: the patient is agitated, skin hot, moist, and hyperaemic. Breathing is difficult with a prolonged wheezing-type expiration. BP – 100/60, heart sounds rhythmic but muffled, pulse – 98/min. After 10 minutes, the patient’s condition worsened: he lost consciousness, became cyanotic, had clonic convulsions, BP – 80/50, HR – 110/min, pulse thready.

Hemogram: Hb – 130 g/L, Ht – 48%, erythrocytes – 5.4×10¹²/L, reticulocytes – 1%, leukocytes – 8×10⁹/L, band neutrophils – 4%, segmented neutrophils – 51%, monocytes – 4%, eosinophils – 9%, basophils – 0%, lymphocytes – 25%, ESR – 15 mm/h, platelets – 300,000/mm³. IgE – increased.

**Questions:**

**1. What type of allergic reaction did this patient develop and what is the presumed allergen? What is the general pathogenesis of this type of hypersensitivity?**

**2. What type of antibodies are formed as a result of sensitization in the immunological stage of this type of hypersensitivity? Where are the antibodies located?**

**3. What does the pathochemical stage of this type of hypersensitivity involve? Which cellular and plasma mediators of anaphylactic allergic reaction trigger the reaction in this patient? Sources of cellular mediators.**

**4. What effects of mediators developed in this patient during the pathophysiological stage? Explain. Describe the physiopathological mechanism of cardiovascular manifestations in this patient.**

**5. Explain the physiopathological mechanisms of respiratory manifestations in this patient.**

**Clinical Case 2**

Patient A., 60 years old, presented to the dentist with the following complaints: ulcerative erosions on the oral mucosa, persistent oral pain for about 2 months, difficulty chewing and brushing. No improvement was observed after the use of oral antiseptics and antifungal agents.  
Objective: On examination of the oropharyngeal cavity, extensive ulcerative-erosive lesions with irregular borders were observed on the hard palate and gingival mucosa, paraesthesia, the mucosal area detached easily upon light touch. Desquamative gingivitis.

**Direct immunofluorescence** – IgG deposits in intercellular spaces – “fishnet” pattern. Serological test – anti-desmoglein-3 antibodies. Preliminary diagnosis – Pemphigus vulgaris.

**Questions:**

**1. What type of allergic reaction did this patient develop? What is an autoimmune allergic reaction? What is the presumed allergen?**

**2. What type of antibodies are formed as a result of sensitization in the immunological stage of this type of hypersensitivity? Where are the antibodies located?**

**3. What does the pathochemical stage of this type of hypersensitivity involve? Which mediators of cytotoxic–cytolytic allergic reaction trigger the allergic reaction in this patient?**

**4. Explain the physiopathological mechanism of oropharyngeal manifestations in this patient.**

**Clinical Case 3**

Patient, 24 years old, returned to the dentist one month after placement of a fixed orthodontic appliance “Wipla” (chrome-nickel). The patient complained of burning sensation in the oral cavity, metallic or altered taste, gingival swelling and erosions causing pain during chewing.  
Objective: on the dental arches, erythematous erosive areas were observed. Gingival mucosa hyperaemic and swollen.

**Questions:**

**1. What type of allergic reaction developed in this patient after placement of the orthodontic appliance and what is the presumed allergen?**

**2. Pathogenesis of delayed hypersensitivity reaction. Which cells are involved in the mechanism of type IV allergic reaction?**

**3. What does the pathochemical stage of this type of hypersensitivity involve? Which mediators of delayed hypersensitivity allergic reaction are involved in the development of oral lesions?**

**4. Explain the physiopathological mechanism of oropharyngeal manifestations in this patient (gingival swelling and erosions, etc.).**

**Topic 7: Physiopathology of red and white blood. Anemias, Leukocytosis. Leukoses. Clinical manifestations in oral cavity**

**Clinical case 1**

A 47-year-old patient presents to the dental clinic with complaints of persistent pain in the oral cavity, burning sensations on the tongue and difficulty swallowing. The patient also complains of the following complaints: asthenia, irritability, unsteady gait, headache, dizziness, paraesthesia. From the patient's history - one year ago he underwent gastric resection.

Objective: pale integument, bright red tongue (Hunter's glossitis) with fissures.

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| --- | --- | --- |
| **CBC** | **VALUES** | **REFERENCE RANGES** |
| **Hematocrit** | 35 | **Men**39-49%  **Women** 35-45% |
| **Hemoglobin** | 11,7 | **Men** 13,6-17,5 g/dL  **Women** 12,0-15,5 g/dL |
| **Red blood cells (RBC)** | 3,6 | 4,7-6,1 million/mm3 |
| **Reticulocyte count** | 0,3 | 0,5-1,5% |
| **MCV** | 114 | 80 -100 fL |
| **MCH** | 38 | 26 – 34 pg |
| **MCHC** | 33 | 31 -36 g/dL |
| **White blood cell (WBC) count,** | 4,6 | 4,800–9,000/cumm |
| **Neutrophil count** | 70 | 60 -62% |
| Segmented mature neutrophils | 65 | 40-60% |
| Non-segmented neutrophils (band) | 5 | 1-6% |
| Metamyelocytes | 0 | 0% |
| Myelocytes | 0 | 0% |
| **Basophil count** | 0 | 0- 1,0%  10 -120/cu mm |
| **Eosinophil count** | 2 | 1-4%  4- -500 cu mm |
| **Lymphocyte count** | 25 | 25-35%  800 -3,500/cu mm |
| **Monocyte count** | 3 | 3-7%  200-800/cu mm |
| **Platelet count** | 143 | 150,000-450,000/cu mm |
| **Morphological changes of blood cells** | Anisocytosis, poikilocytosis, Giant neutrophils with hypersegmented nuclei, erythrocytes with Cabot rings and Jolly inclusions |  |

**Questions:**

**1. What pathology of the erythrocyte system is present in this patient and what is the etiologic factor? Argue by the changes in the hemogram.**

**2. What is the mechanism of vitamin B12 malabsorption in this patient?**

**3. The values of the parameters MCV and MCH are plotted in the hemogram. What do these parameters indicate in this patient?**

**4. What is the pathogenetic mechanism of Hunter’s glossitis (bright red tongue)? (presented as a pathogenetic chain)**

**5. What is the pathogenetic mechanism of the neurologic signs present in this patient?**

**Clinical case 2**

The 37-year-old patient presented to the dentist with the following complaints: for several months she has been suffering from bleeding gums, especially after brushing. She also complained of extreme tiredness and weakness. She reports a diet low in red meat and occasional heavy menstrual periods, but no other chronic health problems.

Objectively: pronounced pallor, labial commissures and taste paresthesias, pale mucosa of the oral cavity with greenish-gray tinge, fragile nails and hair.

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| --- | --- | --- |
| **CBC** | **VALUES** | **REFERENCE RANGES** |
| **Hematocrit** | **32** | **Males 39-49%**  **Females 35-45%** |
| **Hemoglobin** | **9,0** | **Males 13,6-17,5 g/dL**  **Females 12,0-15,5 g/dL** |
| **Red blood cells (RBC)** | **4,0** | **4,7-6,1 mln/mm3** |
| **Reticulocyte count** | **0,5** | **0,5-1,5%** |
| **MCV** | **74** | **80 -100 fL** |
| **MCH** | **22** | **26 – 34 pg** |
| **MCHC** | **28** | **31 -36 g/dL** |
| **White blood cell (WBC) count,** | **5,7** | **4,800–9,000/mm3** |
| **Neutrophil count** | **60** | **60 -62%** |
| **Basophil count** | **0,5** | **0- 1,0%**  **10 -120/ mm3** |
| **Eosinophil count** | **3** | **1-4%**  **4- -500 mm3** |
| **Lymphocyte count** | **32** | **25-35%**  **800 -3,500/ mm3** |
| **Monocyte count** | **5** | **3-7%**  **200-800/ mm3** |
| **Platelet count** | **258** | **150,000-450,000/ mm3** |
| **Morphological changes of blood cells** | **Anisocytosis, poikilocytosis, anulocytosis.** |  |

**Questions:**

**1. What type of pathologic process of the erythrocyte system is present in the patient? Argue by the changes in the hemogram.**

**2. Describe the mechanism of iron absorption in the body.**

**3. Explain the changes of MCH, MCV and MCHC in the hemogram.**

**4. What is the pathogenetic mechanism of clinical signs such as brittle nails and hair, labial commissures and taste paresthesias?**

**5. What are anisocytosis, poikilocytosis, and anulocytosis, and what is the mechanism of these morphologic changes?**

**Clinical case 3**

A 45-year-old patient presents to the dentist's office complaining of severe pain in the right mandibular area and gingival swelling in the molar region. The symptoms started a few days ago and gradually worsened, the patient also had mild fever, generalized fatigue and facial discomfort.

On oral examination: gingival edema, accentuated erythema and purulent discharge around the affected molar was noted.

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| **CBC** | **VALUES** | **REFERENCE RANGES** |
| **Hematocrit** | 45 | **Males** 39-49%  **Females** 35-45% |
| **Hemoglobin** | 15,1 | **Males** 13,6-17,5 g/dL  **Females** 12,0-15,5 g/dL |
| **Red blood cells (RBC)** | 5,6 | 4,7-6,1 million/cu mm |
| **Reticulocyte count** | 1,3 | 0,5-1,5% |
| **MCV** | 97 | 80 -100 fL |
| **MCH** | 27 | 26 – 34 pg |
| **MCHC** | 34 | 31 -36 g/dL |
| **White blood cell (WBC)** | 13,7 | 4,800–9,000/cumm |
| **Neutrophil count** | 80 | 60 -62% |
| Segmented mature neutrophils | 55 | 40-60% |
| Non-segmented neutrophils (band) | 20 | 1-6% |
| Metamyelocytes | 5 | 0% |
| Myelocytes | 0 | 0% |
| **Basophil count** | 0 | 0- 1,0%  10 -120/cu mm |
| **Eosinophil count** | 2 | 1-4%  4- -500 cu mm |
| **Lymphocyte count** | 15 | 25-35%  800 -3,500/cu mm |
| **Monocyte count** | 3 | 3-7%  200-800/cu mm |
| **Platelet count** | 357 | 150,000-450,000/cu mm |
| **Morphological changes of blood cells** |  |  |

**Questions:**

**1. What changes in the white blood count are seen in the patient? Argue your answer.**

**2. What is the pathogenetic mechanism of this pathologic process of the leukocyte system?**

**3. Explain what type of nuclear deviation is present in the patient’s CBC?**

**4. In what type of pathology is eosinophilic and basophilic leukocytosis present?**

**5. In what type of pathology is absolute and relative lymphocytic leukocytosis present?**

**Clinical case 4**

A 34-year-old female patient presents to the dental clinic with complaints of severe oral pain, difficulty swallowing and a persistent sore throat. She reports that her symptoms started about 2 weeks ago and gradually worsened after treatment with penicillin antibiotics.

On oral examination: hyperemic, swollen and inflamed buccal mucosa, multiple ulcerations on tongue, buccal mucosa and oropharynx.

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| --- | --- | --- |
| **CBC** | **VALUES** | **REFERENCE RANGES** |
| **Hematocrit** | 45 | **Males** 39-49%  **Females** 35-45% |
| **Hemoglobin** | 14,1 | **Males** 13,6-17,5 g/dL  **Females** 12,0-15,5 g/dL |
| **Red blood cells (RBC)** | 5,6 | 4,7-6,1 million/cu mm |
| **Reticulocyte count** | 1,3 | 0,5-1,5% |
| **MCV** | 97 | 80 -100 fL |
| **MCH** | 27 | 26 – 34 pg |
| **MCHC** | 34 | 31 -36 g/dL |
| **White blood cell (WBC) count,** | 2,0 | 4,800–9,000/cumm |
| **Neutrophil count** | 18 | 60 -62% |
| Segmented mature neutrophils | 18 | 40-60% |
| Non-segmented neutrophils (band) | 0 | 1-6% |
| Metamyelocytes | 0 | 0% |
| Myelocytes | 0 | 0% |
| **Basophil count** | 0 | 0- 1,0%  10 -120/cu mm |
| **Eosinophil count** | 0 | 1-4%  4- -500 cu mm |
| **Lymphocyte count** | 75 | 25-35%  800 -3,500/cu mm |
| **Monocyte count** | 7 | 3-7%  200-800/cu mm |
| **Platelet count** | 357 | 150,000-450,000/cu mm |
| **Morphological changes of blood cells** |  |  |

**Questions:**

**1. What changes in the CBC of white blood are seen in the patient? Argument.**

**2. What is the pathogenetic mechanism of this pathologic process of the leukocyte system?**

**3. Explain the pathogenetic mechanism of multiple ulcerations on the tongue, buccal mucosa and oropharynx.**

**4. Explain the pathogenetic mechanism of the clinical signs: hyperaemic, swollen and inflamed buccal mucosa.**

**Topic 8: Physiopathology of the cardiovascular system. Physiopathology of the respiratory system. Clinical manifestations in oral cavity.**

**Clinical Case 1**

Patient R., 48 years old, presented to the dentist with the following complaints: moderate pain when chewing and sensitivity to cold and heat in the upper right molar (tooth 16). During the intraoral examination, a deep cavity was observed on the occlusal surface of molar 16.

**Medical History:** The patient has liver cirrhosis. He does not consume alcohol. He has been under the supervision of a cardiologist for several years.

**Objective Findings:** Generalized edema, acrocyanosis, presence of fluid in the abdominal cavity, dyspnea, fatigue, pale skin, dilation of superficial abdominal wall veins, hepatomegaly, and splenomegaly.

The family doctor gave a preliminary diagnosis of decompensated heart failure, and the patient was referred to a specialized cardiology center where the diagnosis was confirmed as heart failure with tricuspid valve stenosis due to rheumatic disease.

Considering the patient's medical history, the dental treatment should be planned so as not to interfere with his cardiovascular condition and treatment.

**Questions:**

**1. Which compartment of the heart is predominantly affected in this patient? Justify your answer based on the presence of characteristic symptoms.**

**2. What type of cardiac overload develops in cases of valve stenosis? Describe the changes in intracardiac hemodynamics with right atrium overload due to resistance.**

**3. What type of hypertrophy develops in these patients? Describe the pathogenic mechanism.**

**4. What is the mechanism of cardiac function exhaustion in hypertrophy?**

**5. Where does venous congestion develop in this patient, and by what mechanisms do generalized edema occur?**

**6. Describe the mechanism of fluid accumulation in the abdominal cavity as well as the dilation of superficial veins of the abdominal wall, hepatomegaly, and splenomegaly.**

**Clinical Case 2**

Patient T., 55 years old, presented to the dentist for tooth prosthetics.

**Medical History:** The patient has had essential arterial hypertension for 15 years. He does not consume alcohol. During the procedure, he experienced headaches, ringing in the ears, blurred vision, and was referred to a specialized cardiology center for further evaluation.

**Complaints:** Fatigue, headache, dyspnea, blurred vision.

**Objective Findings:** Red skin, plethoric appearance. Blood pressure – 210/100 mm Hg.

**Questions:**

**1. Which compartment of the heart is predominantly affected in the patient with hypertensive disease? Justify your answer.**

**2. What type of cardiac overload develops in cases of arterial hypertension? Describe the changes in intracardiac hemodynamics with left ventricular overload due to resistance.**

**3. Describe the changes in intracardiac hemodynamics with left ventricular overload due to volume (preload).**

**4. What type of hypertrophy develops in patients with arterial hypertension, and which compartment of the heart is predominantly affected? Describe the pathogenic mechanism.**

**5. What is the mechanism of cardiac function exhaustion in concentric hypertrophy?**

**6. Where does venous congestion develop in this patient, and by what mechanisms do venous congestion in the lungs and pulmonary edema occur?**

**Topic 9: Physiopathology of the digestive system. Physiopathology of the liver.**

**Clinical case 1**

Patient D., 50 years old, complains of persistent epigastric pain, more intense after meals, associated with nausea and occasional vomiting. He also reported a weight loss of about 6 kg in the last 2 months. He suffers from constipation. The patient has a history of regular use of non-steroidal anti-inflammatory drugs (NSAIDs) for chronic back pain.

**Objective:** epigastric tenderness

**Upper GI endoscopy** demonstrates an ulcerative lesion on the anterior wall of the gastric antrum.

**Questions:**

**1. What are the pathogenetic mechanisms that contributed to the gastric ulcerogenesis against the background of chronic nonsteroidal anti-inflammatory drug use?**

**2. What aggressive etiologic factors may contribute to gastric and duodenal ulcerogenesis?**

**3. List and explain the protective mechanisms of the gastric mucosa that oppose aggressive actions.**

**4. Which pathogenetic mechanisms contribute to gastric hyperacidity?**

**5. How is the motor, evacuation, absorptive, and reservoir function of the stomach altered under gastric hyperacidity?**

**6. How does digestion and intestinal motility change under conditions of gastric hyperacidity?**

**7. What factors and mechanisms contribute to gastric hypoacidity?**

**8. How does gastric motor, evacuation, absorptive, and reservoir functions of the stomach change under conditions of gastric hypoacidity?**

**9. How does digestion and intestinal motility change under conditions of gastric hypoacidity?**

**10. What consequences may occur in the oral cavity in gastric hyperacidity?**

**11. What changes may occur in the oral cavity as a consequence of gastric hypoacidity?**

**Clinical case 2**

Patient A., 60 years old was admitted to the hepatology ward. **Objective:** patient underweight; visibly jaundiced skin and mucous membranes; moderate ascites.

**History:** 5 years of treatment for chronic hepatitis, because during a routine check-up he was found to have antibodies against hepatitis B virus, HBS- positive antigen and increased levels of ASAT, ALAT.

**Biochemical examination**: total bilirubin -45 µmol/l (N-3,4-22), conjugated bilirubin 25 µmol/l ( N-0 -5,1), free bilirubin 20 µmol/l (N-3,4-17);prothrombin- 1.0mcM/l (N -1,4-2,1);fibrinogen -2,0 µmol/l (N-4-10); ALAT-180Ul/l (N 7-55); ASAT- 120 UL/l (N- 11-47); G-Glutamyltransferase 100 Ul/l (20-76); protein (total fraction) -55 g/l (N-65-85); serum albumin 20g/l (N-36-50); albumin/globulin ratio 0.3(N -0.64); plasma ammonia - 60 mmol/l (N-19-43);

**Urine:** colour brownish-brown; conjugated bilirubin (++), urobilin (+++), stercobilin (+).

**Questions:**

**1. What type of jaundice developed in the patient against the background of chronic viral hepatitis? Argue your answer based on clinical symptoms and laboratory findings.**

**2. Which tests indicate the presence of cytolytic syndrome in the patient? Please explain.**

**3. Which tests are indicative of altered liver protein synthetic function? Please explain.**

**4. What consequences may occur in the patient as a result of hypoalbuminemia?**

**5. Which tests show disturbed ammoniogenesis function of the liver? Argue? What may be the consequences of disturbance of this function?**

**6. Describe the disturbance of bile pigment metabolism and circulation in parenchymal jaundice. Argue on the basis of clinical case data.**

**7. Describe the disturbance of bile pigment metabolism and circulation in mechanical jaundice. What changes occur in the urine and feces?**

**8. Describe the disturbance of bile pigment metabolism and circulation in mechanical jaundice. What changes occur in the urine and feces?**

**9. How does intestinal digestion change in complete bile duct obstruction?**