**Situation Problem 1**

Patient N., a 32-year-old, complains of intense nose itching, violent sneezing, excessive tearing, watery nasal discharge, sore throat, cough and headache. According to the medical history, these symptoms have appeared every spring for the past three years. The patient’s condition worsens when in the forest or public gardens.

**Examination Findings**: The sclera is hyperaemic, with eyelid erythema and edema, tearing, seromucous nasal discharge, and laboured breathing. Swelling of the nasopharyngeal mucosa with abundant mucus secretion is noted.

**Hemogramm**: Leukocytes - 10.5 × 10⁹/L (N= 4.8–10.8 × 10⁹/L); Lymphocytes - 22% (N= 25–33%); Neutrophils: band - 2% (N=1-5%), segmented - 56% (N=40–70%); Monocytes - 7% (N= 3–7%); Eosinophils - 10% (N= 0–6%)

**Immunogram**: CD4+ T-cells - 1400/mm³ (N= 500–1200/mm³); T-helper cells - 58.5%; B-lymphocytes: 62%; IgA - 2.00 g/L (N= 0.70–3.50 g/L); IgM- 2.1 g/L (N= 0.50–3.0 g/L); IgG - 13.0 mg/dL (N= 7.0–17.0 g/L); Serum IgE level - 500 IU/mL (N= 0–100 IU/mL)

During a **percutaneous test** with plant allergens (flower pollen), a papule surrounded by erythema without induration formed in response to maple pollen.

**Questions:**

1. What type of allergic reaction has developed in the patient? Explain using the medical history, clinical signs, and laboratory data.
2. Deduce the pathogenic chain of active sensitization in Type I (Anaphylactic) allergic reaction. Describe the role of B and T lymphocytes.
3. Which pre-synthesized cellular mediators (and their sources) contribute to the development of erythema, edema, tearing, and mucus secretion?
4. Which de novo synthesized mediators contribute to the clinical signs of an anaphylactic reaction? Outline the synthesis pathway of prostaglandins and leukotrienes.
5. What is the pathogenesis of blood pressure reduction in anaphylactic shock? What are the mechanisms of bronchial obstruction in anaphylactic shock?
6. Interpret the pathogenic increase in the percentage of B and T lymphocytes in an anaphylactic reaction. Explain the prevalence of B lymphocytes in the immunogram.
7. Explain the increase in eosinophil percentage in the complete blood count. List the mediators and their functions synthesized by eosinophils.

**Situation Problem 2**

Patient G, 48 years old, presents with asthenia, vertigo, nausea, and drowsiness. From the medical history: the patient had salmonellosis and was treated with levomycetin for three months.

**Objective:** The skin appears pale, cold, and dry. Respiratory rate is 20 breaths per minute, vesicular breath sounds on auscultation, pulse rate is 86 beats per minute, blood pressure is 110/65 mm Hg.

**In the hemogram**: Erythrocytes - 2.4 × 1012/L (N= 4.0 - 5.2 × 1012/L); Hemoglobin - 110 g/L (N= 120–158 g/L); Hematocrit - 30% (N= 35.4–44.4%); Leukocytes - 10.5 × 10⁹/L (N= 4.8 – 10.8 × 10⁹/L); Lymphocytes - 25% (N= 25–33%); non-segmented neutrophils - 4% (N= 0-5%); Segmented neutrophils - 58% (N= 40–70%); Monocytes - 4% (N= 3-7%); Eosinophils - 12% (N= 0–6%); ESR - 15 mm/hour (N= 2-15 mm/hour)

**Immunogram:** CD4+ - 1400/mm³ (N= 500 to 1200/mm³)**;** CD8+ - 300/mm³ (N= 150 to 1000/mm³)**;** T-helper - 58.5%**;** B lymphocytes - 62%**;** IgA - 1.0 g/L (N= 0.70–3.50 g/L)**;** IgM - 3.4 g/L (N= 0.50–3.0 g/L)**;** IgG - 28.1 g/L (N= 7.0–17.0 g/L)

**Anti-erythrocytes antibodies were detected in the blood.**

**Questions:**

1. What type of allergic reaction has developed in the patient? Justify based on the medical history, clinical signs, and laboratory data.
2. What does haptens represent? What is their role in the etiology of allergic reactions? Differentiate between a complete allergen and a hapten.
3. Deduce the pathogenetic chain of active sensitization in the type II cytotoxic allergic reaction. What is the role of B and T lymphocytes?
4. What pathogenetic mechanisms contribute to the hemolysis of erythrocytes in this patient?
5. Which components of the complement system induce hemolysis of erythrocytes, and through what mechanisms?
6. Deduce the pathogenetic chain of complement activation via the classical and alternative pathways.
7. Interpret the pathogenetic increase in the percentage of B lymphocytes and T lymphocytes in the cytotoxic reaction. Justify the prevalence of B lymphocytes in the immunogram.

**Situation Problem 3**

Patient D, 9 years old, received 3000 IU of hyperimmune horse antitoxin serum prophylactically due to a leg injury. On the 9th day after the serum was administered, the child developed severe pain and swelling in the brachial and knee joints, generalized skin rashes, and general weakness.

**Objective:** The injection site of the serum is swollen and painful, the morning body temperature is 38.8 °C, and the heart sounds are muffled with blood pressure at 80/50 mm Hg. The child was hospitalized.

**Immunogram:** CD4+ - 1300/mm³ (N= 500 to 1200/mm³), T-helper - 52%, B lymphocytes - 56%, IgA - 1.8 g/L (N= 0.70–3.50 g/L), IgM - 3.2 g/L (N= 0.50–3.0 g/L), IgG - 31.3 g/L (N= 7.0–17.0 g/L).

**The complement fractions in the blood serum are decreased.**

**Questions:**

1. What type of allergic reaction has developed in the patient? Justify your answer based on the medical history, clinical signs, and laboratory test results.
2. Deduce the pathogenic chain of active sensitization in the context of a type III allergic reaction. The role of B and T lymphocytes.
3. What mediators contributed to the injury and inflammation of the brachial and knee joints in the patient?
4. Explain the mechanism of circulation and sedimentation of circulating immune complexes in the context of a type III allergic reaction.
5. What plasma mediators are involved in the pathogenesis of type III allergic reactions? Justify your answer based on the effects of these mediators.
6. Interpret the pathogenic increase in the percentage of B and T lymphocytes in the context of a type III allergic reaction. Justify the prevalence of B lymphocytes in the immunogram.
7. Explain the essence of the decrease in complement system fractions in the blood serum in the context of a type III allergic reaction.

**Situation Problem 4**

Patient R, 18 years old, presented to the family doctor with the following complaints: itching, redness, rashes, and ulcers on the skin in the area of the left arm.

From the medical history: one month ago, she purchased a yellow metal alloy bracelet, and regular wearing of it, over two weeks, caused the aforementioned manifestations.

**Hemogram:** leukocytes - 9.5 × 10^9/L (N = 4.8 – 10.8 × 10^9/L), lymphocytes - 40% (N = 25–33%), bend neutrophils - 3% (N = 0-5%), segmented neutrophils - 46% (N = 40–70%), monocytes - 7% (N = 3-7%), eosinophils - 4% (N = 0–6%).

**Immunogram:** CD4+ - 1600/mm³ (N = 500 to 1200/mm³), CD8+ - 1300/mm³ (N = 150 to 1000/mm³), T-lymphocytes - 68%, B-lymphocytes - 32%. IgA - 1.4 g/L (N = 0.70–3.50 g/L), IgM - 1.8 g/L (N = 0.50–3.0 g/L), IgG - 12.5 g/L (N = 7.0–17.0 g/L).

**Questions:**

1. What type of allergic reaction has developed in the patient? Justify your answer based on the medical history, clinical signs, and laboratory test results.
2. Deduce the pathogenic chain of active sensitization in the context of type IV allergic reaction. What is the pathogenic role of T lymphocytes?
3. What mediators contributed to the injury and inflammation associated with contact dermatitis?
4. What cellular mediators are involved in the pathogenesis of type IV allergic reactions? Justify your answer by discussing the effects of these mediators.
5. Interpret the pathogenic increase in the percentage of T lymphocytes in the context of type IV allergic reaction. Justify the prevalence of T lymphocytes in the immunogram.
6. Explain the functions of CD4+ and CD8+ lymphocytes in the pathogenesis of type IV allergic reaction.
7. What types of cytokines mediate type IV allergic reactions? What are their effects in the context of type IV allergic reaction?

**Situation Problem 5**

Patient B., 32 years old, was brought to the emergency medicine ward with the following complaints: confusion, general weakness, sweating, tachycardia, palpitations, excessive feeling of hunger and episodes of loss of consciousness.

The patient reported a constant sensation of vertigo for the last 2-3 months, which improved after taking the sweetened drinks. From past history the patient did not suffer from any chronic disease or previous surgeries. The patient is a non-smoker and does not consume alcohol.

*Objective:* cold, moist skin. BP - 140/90 mm Hg; pulse - 112 per minute; FCC - 100 b/min; FR - 20/min.

Laboratory data: glucose - 40 mg/dl; serum insulin - 50.8 μU/ml (6 - 35 μU/ml), C-peptide - 10.6 ng/ml (0.9-4 ng/ml); Na+ - 160 mEq/l; K+ - 3.0 mEq/l.

On CT scan was found a tumor in the pancreas - insulinoma.

**Questions:**

1. **What change in carbohydrate metabolism is seen in this patient and what is the pathogenetic mechanism? Argument answer.**
2. **What is the mechanism of tachycardia in this patient (replay by pathogenetic chain)?**
3. **What is the mechanism of the elevated blood pressure in this patient (replayed by pathogenetic chain)?**
4. **How do glucagon and epinephrine compensate for hypoglycemia?**
5. **How does cortisol compensate for hypoglycemia?**
6. **What explains the electrolyte changes in this patient?**
7. **Insulin is an anabolic hormone; how do carbohydrate metabolic processes change with insulin hypersecretion?** **(Glycolysis, glycogenolysis, glycogenogenesis, gluconeogenesis) (indicate by increase or decrease arrows)**

**Situation Problem 6**

Patient A., 13 years old, was brought in by her parents with the following complaints general weakness, vomiting, obtundation, deep and noisy breathing.

According to the parents, a few months ago they noticed a decrease in the child's body mass, although he was eating quite frequently, intense thirst, frequent urination. Following further investigations, the endocrinologist diagnosed type 1 diabetes mellitus.

 *Objective:* cold, clammy skin. BP - 90/60 mm Hg; RF - 30/min; FCC - 100 b/min; pulse - 110/min; acetone odor.

Laboratory data: Glucose - 200 mg/dL; Na+ - 125 mEq/L; K+ - 5.9 mEq/L; Bicarbonates - 10 mEq/L; Urea - 18 mmol/L; Creatinine -140 mmol/L; Hb - 14 g/dL (12.0-15.5 g/dL); Ht - 49% (35-45%); ketone bodies - +++; osmolarity - 330 mOsm/l.

Urine: glucose - +++, ketone bodies - +++

Blood gas: pH - 7.2; PaO2 - 107 mm Hg; PaCO2 - 20 mm Hg.

**Questions:**

1. **What is the mechanism of hyperglycemia in this patient? Argument answer.**
2. **What is the mechanism of polyuria? (****Replay by pathogenetic chain)**
3. **What is the mechanism of polydipsia? (Replay by pathogenetic chain)**
4. **How do carbohydrate metabolic processes change in insulin hyposecretion? (Glycolysis, glycogenolysis, glycogenogenesis, gluconeogenesis) (indicate by increase or decrease arrows)**
5. **Which laboratory and blood gas data indicate an acid-base imbalance and which imbalance is present in this patient?**
6. **What is the mechanism of diabetic ketoacidosis?**
7. **What is the type of breathing and how does it work?**
8. **What are the mechanisms of reduced lipogenesis and enhanced peripheral lipolysis?**

**Situation Problem 7**

Patient P., 49 years old, suffering from chronic alcoholism, was admitted with the following complaints: general weakness, lack of appetite, vomiting, diarrhea, edema, epistaxis and gingival bleeding.

***Objective:*** cachexia, pale and dry skin with ecchymosis, generalized edema, hepatomegaly. Body mass index - 16.5 (norm 18.5 - 24)

**Laboratory data:** Plasma protein - 40 g/dL, albumin - 2.5 g/dL (norm 3.4-4.7 g/dL); transferrin - 1.0 (norm 2.0-3.6 g/L); Glucose - 60 mg/dL; Na+ - 155 mEq/L; K+ - 2.9 mEq/L; Creatinine -0.3 mg/dL (norm 0.6-1.2 mg/dL); Hb - 11.5 g/dL (13.6-17.5 g/dL); ALAT - 85 IU/L (norm 7-56 IU/L); ASAT - 55 IU/L (norm 0-35 IU/L); polyuria.

MRI (nuclear magnetic resonance) - liver steatosis was detected.

**Questions:**

1. **What is the mechanism of protein maldigestion and malabsorbtion?**
2. **What is the pathogenetic mechanism of fatty liver dystrophy due to hypoproteinemia?**
3. **What is the mechanism of generalized edema in this patient? (Explain by pathogenetic chain)**
4. **The patient with hypoproteinemia shows clinical signs suggestive of hemorrhagic syndrome** (**epistaxis, gingival bleeding and ecchymoses on the skin surface). What is the pathogenetic mechanism of hemorrhagic syndrome?**
5. **How does the immune status change in patients with hypoproteinemia? Argument.**

**Situation Problem 8**

 Patient X, a 60 years old man, addresses to the family doctor with complaints of periodic pain in the precordial region.

 **From the anamnesis**: The job involves constant emotional stress, working as a lawyer and consumes a lot of animal fat. 2 years ago he suffered a myocardial infarction. For 1 year he is taking statins.

 **Objective**: Mass=115 kg, Height=170 cm. BP=150/105 mmHg. Ps=90.

 Paraclinically, the patient's lipid profile was of interest:

|  |  |  |
| --- | --- | --- |
| Walk | Patient value | Standard |
| Total cholesterol | 450 | <200 mg/dl |
| HDL-cholesterol | 25 | >40 mg/dl |
| LDL-cholesterol | 300 | <100 mg/dl |
| Triglyceride | 400 | <150 mg/dl |

**It has been established:** Absolute coronary insufficiency was established on the basis of atherosclerosis of the coronary arteries. Secondary hypercholesterolemia. Obesity gr. II.

**Questions:**

1. **What changes in lipid metabolism are seen in the patient? Argue from the data of the problem.**
2. **What are the types of hyperlipidemias? Pathogenesis of transport hyperlipidemia.**
3. **What are the types of hyperlipidemias? Pathogenesis of retention hyperlipidemia.**
4. **Which lipoprotein fractions are atherogenic? Argue from the data of the problem.**
5. **What is the role of emotional stress in the pathogenesis of atherogenesis?**

**Situation Problem 9**

Patient, M, 54 years old was admitted with complaints of abdominal pain, diarrhea for 4 days, dizziness, muscle weakness. On objective examination dry skin, reduced cutaneous turgor, dry mucosa of the oral cavity, dental impressions on the sides of the tongue. Diuresis for 24 hours approximately 500 ml. Blood pressure reduced - 75/ 45 mmHg, FCC - 118/min.

**Blood biochemical analysis:** serum sodium - 143 mEq/L (N: 135 - 145 mEq/L), serum potassium - 4.9 mEq/L (N: 3.5 - 5.5 mEqL), serum osmolarity 305 mOsm/L.

**Questions:**

**1.** **What water dyshomeostasis is present in this patient and what is the pathogenetic mechanism?**

**2.** **How is the extracellular water compartment affected in this water dyshomeostasis?**

**3.** **How is the intracellular water compartment affected in this water dyshomeostasis?**

**4.** **How is the intravascular water compartment affected in this water dyshomeostasis?** **What clinical manifestations are reflective of this change?**

**5.** **What changes in the general blood analysis (erythrocytes, hemoglobin, hematocrit) are expected to be present in this hydric dyshomeostasis?**

**6.** **What compensatory reactions are triggered in this hydrate dyshomeostasis?**

**7.** **What are the pathogenetic mechanisms that explain the reduction in diuresis in this patient?**

**Situation Problem 10**

The patient, M, 64 years old was hospitalized with complaints of headache, tinnitus, muscle weakness, heart palpitations.

**On objective examination** icteric and dry skin, reduced cutaneous turgor, dry mucosa of the oral cavity, dental impressions on the sides of the tongue. Diuresis for 24 hours approximately 3500 ml. Blood pressure reduced - 60/ 40 mmHg, FCC - 128/min.

**Blood electrolytes:** Serum sodium - 123 mEq/L (N: 135 - 145 mEq/L), serum potassium - 6.4 mEq/L (N: 3.5 - 5.5 mEqL), Serum osmolarity 265 mOsm/L.

**From the anamnesis**, the patient is known to have ovarian tumor, and at the last examination, about 10 months ago, multiple metastases in the adrenal cortex were detected.

**Questions:**

**1.** **What water dyshomeostasis is present in this patient and what is the pathogenetic mechanism?**

**2.** **How is the extracellular hydric compartment affected in this hydric dyshomeostasis?**

**3.** **How is the intracellular water compartment affected in this water dyshomeostasis?**

**4.** **How is the intravascular water compartment affected in this water dyshomeostasis?** **What clinical manifestations are reflective of this change?**

**5.** **What changes in the general blood analysis (erythrocytes, hemoglobin, hematocrit) are expected to be present in this hydric dyshomeostasis?**

**6.** **How can the presence of hyperkalemia in this water dyshomeostasis be explained?**

**7.** **What sodium dyshomeostasis is present in this patient and explain the pathogenetic mechanism?**

**8.** **What compensatory reactions are triggered in this hydrate dyshomeostasis?**

**Situation Problem 11**

Patient P, 72 years old was hospitalized in an unconscious state. According to his son he was found at home by neighbours. He is presumed to have been in this unconscious state for approximately 72 hours.

**On objective examination** dry skin, reduced cutaneous turgor, dry mucosa of the oral cavity, dental impressions on the sides of the tongue. Blood pressure low - 60/40 mmHg, C.B.C. 125/min. Respiration rate 36/min.

**Blood Electrolytes:** Serum Sodium - 163 mEq/L (N: 135 - 145 mEq/L), Serum Potassium - 4.4 mEq/L (N: 3.5 - 5.5 mEqL), Serum Osmolarity 365 mOsm/L.

**Questions:**

**1.** **What water dyshomeostasis is present in this patient and what is the pathogenetic mechanism?**

**2.** **How is the extracellular water compartment affected in this water dyshomeostasis?**

**3.** **How is the intracellular water compartment affected in this water dyshomeostasis?**

**4.** **How is the intravascular water compartment affected in this water dyshomeostasis?** **What clinical manifestations are reflective of this change?**

**5.** **What changes in the general blood analysis (erythrocytes, hemoglobin, hematocrit) are expected to be present in this hydric dyshomeostasis?**

**6.** **What sodium dyshomeostasis is present in this patient and explain the pathogenetic mechanism?**

**7.** **What compensatory reactions are triggered in this hydrate dyshomeostasis?**

**Situation Problem 12**

The patient, D, 54 years old was admitted with complaints of headache, generalized peripheral edema. On objective examination edema in both legs and on the sides of the abdomen. Diuresis for 24 hours about 800 ml. Blood pressure - 180/115 mmHg, CBC - 67/min.

**Blood electrolytes:** Serum sodium - 158 mEq/L (N: 135 - 145 mEq/L), serum potassium - 3.2 mEq/L (N: 3.5 - 5.5 mEqL), serum osmolarity 365 mOsm/L, pH = 7.3 (N: 7.35 - 7.45)

From the history, the patient is known to have active secreting tumor in the glomerular area of the left adrenal gland.

**Questions:**

1. **What water dyshomeostasis is present in this patient and what is the pathogenetic mechanism?**

2. **How is the extracellular hydric compartment affected in this hydric dyshomeostasis?**

3. **How is the intracellular water compartment affected in this water dyshomeostasis?**

4. **How is the intravascular water compartment affected in this water dyshomeostasis?** **What clinical manifestations are reflective of this change?**

5. **What changes in the general blood analysis (erythrocytes, hemoglobin, hematocrit) are expected to be present in this hydric dyshomeostasis?**

6. **How can the presence of hypokalemia in this hydric dyshomeostasis be explained?**

7. **What sodium dyshomeostasis is present in this patient and explain the pathogenetic mechanism?**

8. **What are the pathophysiologic mechanisms of edema associated with this hydric dyshomeostasis?**

9. **What compensatory reactions are triggered in this hydric dyshomeostasis?**

**Situation Problem 13**

The patient, G, 64 years old, was admitted with complaints of headache, generalized peripheral edema, muscle weakness, heart palpitations.

**On objective examination** jaundiced skin, edema on both legs and sides of the abdomen. Diuresis for 24 hours about 500 ml. Blood pressure - 175/110 mmHg, CBC - 68/min.

**Blood electrolytes:** Serum sodium - 128 mEq/L (N: 135 - 145 mEq/L), serum potassium - 5.9 mEq/L (N: 3.5 - 5.5 mEqL), serum osmolarity 265 mOsm/L.

From history, the patient is known to have ADH-secreting bronchial carcinoma.

**Questions:**

1. **What water dyshomeostasis is present in this patient and what is the pathogenetic mechanism?**

2. **How is the extracellular hydric compartment affected in this hydric dyshomeostasis?**

3. **How is the intracellular water compartment affected in this water dyshomeostasis?**

4. **How is the intravascular water compartment affected in this water dyshomeostasis?** **What clinical manifestations are reflective of this change?**

5. **What changes in the general blood analysis (erythrocytes, hemoglobin, hematocrit) are expected to be present in this hydric dyshomeostasis?**

6. **How can the presence of hyperkalemia in this hydric dyshomeostasis be explained?**

7. **What sodium dyshomeostasis is present in this patient and explain the pathogenetic mechanism?**

8. **What are the pathophysiologic mechanisms of edema associated with this hydric dyshomeostasis?**

9. **What compensatory reactions are triggered in this hydric dyshomeostasis?**

**Situation Problem 14**

Patient B, 56 years old, known to have diabetes mellitus, insulin-dependent, was admitted urgently with the following manifestations: confusion, feeling nauseous and vomiting, dizziness.

**On objective examination**: deep and accelerated breathing, low blood pressure, hot, sweaty skin.

**Blood biochemistry reveals**: Glucose - 206 mg/dL (norm 60 - 110 mg/dL), free fatty acids - 2.3 mmol/L (norm 0 - 0.70 mmol/L), Sodium - 158 mEqL (norm 135 - 145 mEq/L), Potassium - 6.1 mEq/L (norm 3.5 - 5,5 mEq/L), Calcium - 2,9 mmol/L (norm 2,1 - 2,6 mmol/L), Chloride - 90 mmol/L (norm 98 - 106 mmol/L), ketone bodies 3,8 mg/dL (norm below 1,0 mg/dL), lactic acid 0,6 mmol/L (norm < 2,0 mmol/L).

**Analysis of acid-base balance** reveals: pH - 7.31 (norm 7.35 - 7.45), Plasma bicarbonate - 18 mEq/L (norm 24 - 26 mEqL), PaCO2 - 32 mmol/L (norm 35 - 40 mmol/L), SaO2 - 85 %.

**Questions:**

1. What acid-base dyshomeostasis developed in the patient and what is the pathogenetic mechanism?

2. Which biochemical pathogenetic links are involved in the development of ketoacidosis in insulin deficiency?

3. Explain the pH changes in the described clinical situation?

4. Explain the bicarbonate changes in the clinical situation described?

5. Reveal the pathogenetic mechanisms underlying the development of hyperpnea (frequent and accelerated breathing) in the acid-base dyshomeostasis present in the patient?

6. List the clinical and biochemical changes that reveal the presence of compensatory reactions in the given patient?

7. By which pathogenetic mechanisms can the hypernatremia in this patient be explained?

8. By which pathogenetic mechanisms can hyperkalemia be explained in this patient?

9. By which pathogenetic mechanisms can hyperkalemia be explained in this patient?

10. By which pathogenetic mechanisms can hypocapnia be explained in this patient? What is the biological significance of this compensatory reaction?

11. By which pathogenetic mechanisms can hypochloraemia be explained in this patient?

12. How can you explain the change in SaO2 in the given clinical situation?

**Situation Problem 15**

Patient B, 36 years old, was urgently hospitalized in deep coma after a benzodiazepine overdose.

**On objective examination**: shortness of breath (FR - 6/min), blood pressure 85/40 mmHg, tachycardia (FCC - 130/min).

**Blood biochemical analysis** reveals: Glucose - 106 mg/dL (norm 60 - 110 mg/dL) Sodium - 158 mEq/L (norm 135 - 145 mEq/L), Potassium - 6.1 mEq/L (norm 3.5 - 5.5 mEq/L), Calcium - 2.8 mmol/L (norm 2,1 - 2,6 mmol/L), Chloride - 90 mmol/L (norm 98 - 106 mmol/L), Ketone bodies 0,8 mg/dL (norm < 1,0 mg/dL), Lactic acid 0,9 mmol/L (norm < 2,0 mmol/L).

**Analysis of acid-base balance** reveals: pH - 7, 30 (norm 7, 35 - 7,45), Plasma bicarbonate - 32 mEq/L (norm 24 - 26 mEq/L), PaCO2 - 52 mmol/L (norm 35 - 40 mmol/L), SaO2 - 75 %.

**Questions:**

1. What acid-base dyshomeostasis developed in the patient and what is the pathogenetic mechanism?
2. Explain the pH changes in the described clinical situation?
3. Explain the serum bicarbonate changes in the described clinical situation?
4. Explain the mechanisms by which the kidney is involved in compensating for the acid-base dyshomeostasis present in this patient
5. By which pathogenetic mechanisms can the hypernatremia in this patient be explained?
6. By which pathogenetic mechanisms can hyperkalemia be explained in this patient?
7. By which pathogenetic mechanisms can hyperkalemia be explained in this patient?
8. By which pathogenetic mechanisms can the hypochloraemia in this patient be explained?
9. What clinical manifestations may be triggered by increased plasma CO2 (PaCO2) in the presence of this acid-base dyshomeostasis?
10. Explain the pathogenetic mechanisms underlying the decrease in blood pressure value in acid-base dyshomeostasis present in the patient
11. This acid-base dyshomeostasis is associated with osmolarity disturbances. What osmolarity disturbances may be present and how do they manifest?
12. Which biochemical or blood gas parameter allows us to differentiate respiratory acidosis from metabolic acidosis?

**Situation Problem 16**

Patient A, 67 years old, was hospitalized with the following complaints: nausea and vomiting for 4 days, headache, dizziness, muscle cramps in the lower limbs.

**On objective examination**: low blood pressure, dry complexion, reduced skin turgor.

**Blood biochemical analysis** revealed: Glucose - 106 mg/dL (norm 60 - 110 mg/dL), Sodium - 128 mEq/L (norm 135 - 145 mEq/L), Potassium - 3.1 mEq/L (norm 3.5 - 5.5 mEq/L), Calcium - 1.9 mmol/L (norm 2.1 - 2.6 mmol/L), Chloride - 118 mmol/L (norm 98 - 106 mmol/L),

**Acid-base balance analysis** reveals: pH - 7,55 (norm 7,35- 7,45), Plasma bicarbonate - 32 mEqL (norm 24 - 26 mEqL), PaCO2 - 46 mmol/L (norm 35 - 40 mmol/L).

**Questions:**

1. What acid-base dyshomeostasis developed in the patient and what is the pathogenetic mechanism?

2. Explain the pH changes in the described clinical situation?

3. By which pathogenetic mechanisms can the hyponatremia in this patient be explained?

4. By which pathogenetic mechanisms can hypokalemia be explained in this patient?

5. By which pathogenetic mechanisms can hypocalcemia be explained in this patient?

6. By which pathogenetic mechanisms can hypercapnia be explained in this patient? What is the biological significance of this change?

7. By which pathogenetic mechanisms can hyperchloremia be explained in this patient?

8. 11. This acid-base dyshomeostasis is associated with osmolarity disturbances. What osmolarity dysregulations may be present and how do they manifest?

9. Which biochemical or blood gas parameter allows us to differentiate between metabolic and respiratory alkalosis?