**Clinical case 1**

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)**

**Clinical case 2**

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?**

**Clinical case 3**

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.**

**Clinical case 4**

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?**