**Clinical case 1**

**Patient’s Chief Complaints**

Provided by wife: “My husband’s very confused and he has been acting strangely. This morning, he couldn’t answer my questions and seemed not to recognize me. He stopped drinking four years ago, but now it seems to be getting worse.”

**The patient S.** is a 46 years male with a history of chronic alcoholism. He was admitted to the hospital from the outpatient clinic with abdominal swelling and confusion. He has unintentionally gained 15 lbs during the past four weeks. According to his wife, the patient has not been sleeping well for several weeks, has been feeling very lethargic for the past three days.

Before becoming confusing the patient complains of abdominal pain, itching, nausea, vomiting, hematemesis, gum bleeding, loss of appetite, weakness, diarrhea and dry mouth.

**Medical history**

• Cirrhosis diagnosed 4 years ago with ultrasound and liver biopsy (micronodular cirrhosis)

• uncontrolled ascites

• two episodes of upper GI hemorrhages from esophageal varices (2 years ago)

• E. coli-induced bacterial peritonitis 3 years ago

• No history to suggest cardiac or gallbladder disease

• No previous diagnosis of viral or autoimmune hepatitis

**On physical examination**: The patient is restless, mildly jaundiced, and disoriented to time, place, and people.

• BP 110/65, Ps- 83, regular (supine)

• BP 95/60, Ps- 106, regular (standing); ECG – Sinus tachycardia. Low amplitude of T wave, U wave.

• Breathing rate - 27/min

**Skin.** Dry, warm with reduced turgor, hyperkeratosis, scratching. Jaundice. Spider nevi on chest. Palmar erythema. Ecchymoses on lower extremities. Ginecomastia.

**Abdomen** is moderately distended, firm, and slightly tender. Prominent veins observed around umbilicus.

**Diuresis** is reduced. Urine is dark.

**Feces fade**, presence of lipids.

**Laboratory Blood Test Results**

|  |  |  |
| --- | --- | --- |
| **CBC** | **VALUES** | **REFERENCE RANGES** |
| **Hematocrit** | 36 | **Males** 39-49%  **Females** 35-45% |
| **Hemoglobin** | 11,8 | **Males** 13,6-17,5 g/dL  **Females** 12,0-15,5 g/dL |
| **Red blood cells (RBC)** | 3,7 | 4,7-6,1 million/cu mm |
| **MCV** | 71 | 80 -100 fL |
| **MCH** | 19 | 26 – 34 pg |
| **MCHC** | 25 | 31 -36 g/dL |
| **Basophil count** | 0,5 | 0- 1,0%  10 -120/cu mm |
| **Eosinophil count** | 3 | 1-4%  4- -500 cu mm |
| **Lymphocyte count** | 26 | 25-35%  800 -3,500/cu mm |
| **Monocyte count** | 5 | 3-7%  200-800/cu mm |
| **Thrombocytes** | 86,000 | 150,000 – 450,000/cu mm |

**BIOCHEMICAL BLOOD TESTS**

|  |  |  |
| --- | --- | --- |
| **Protein total** | 4,1 | 6,0 – 8,0 g/dL |
| **Albumin** | 2,2 | 3,4 – 4,7 g/dL |
| **Globulin** | 5,7 | 2.6 - 4.6g/dL |
| **Fibrinogen** | 98 | 160 – 450 mgd/L |
| **Prothrombin time** | 20,2 | 11,0 -13,5 sec |
| **Glucose, *serum fasting*** | 46 | 60 – 110 mg/dL |
| **Glucose, *2 hours postprandial*** | 197 | < 150 mg/dL |
| **Triglyceride** | 145 | <165 mg/dL |
| **Cholesterol** | 109 | Desirable: < 200 mg/dL  Borderline: 200–239 mg/dL  High risk: >240 mg/dL |
| **Blood urea nitrogen (BUN)** | 5,8 | 8 – 20 mg/dL |
| **Creatinine** | 0,4 | 0,6-1,2 mg/dL |
| **Bilirubin total** | 3,8 | 0,1 – 1,2 mg/dL |
| **Direct or conjugated bilirubin** | 2,4 | 0,1 - 0,5 mg/dL |
| **Indirect or unconjugated bilirubin** | 1,4 | 0,1 – 0,7 mg/dL |
| **Alanine aminotransferase (ALT)** | 209 | 7-56 IU/L |
| **Aspartate aminotransferase (AST)** | 107 | 0 – 35 IU/Ll |
| **Ammonia (NH3)** | 250 | 18 – 60 µg/dL |
| **Erythropoietin** | 3,5 | 5,4-31,0 UI/L3 |
| **Bicarbonate** | 30 | 21 – 28 mEq/L |
| **Lactic acid** | 2,8 | < 2,0 mmol/L |
| **Ketone bodies** | 2,2 | < 1mg/dl |
| **Ca++** | 1,7 | 2,1-2,6 mmol/L |
| **Na+** | 156 | 135-145 mEq/L |
| **K+** | 3,3 | 3,5 – 5,5 mEq/L |
| **Ferritin** | 2,9 | Males 16 – 300 ng/mL  Females 4 – 161 ng/mL |
| **Iron (Fe2+)** | 35 | 50 – 175 µg/L |
| **Iron-binding capacity (TIBC)** | 478 | 250 -460 µg/dL |
| **Transferrin saturation** | 5 | 30 - 50% |
| **Folic acid** | 103 | 165 -760 ng/mL |
| **B12 vitamin** | 98 | 140 -820 pg/mL |
| **Vitamin A** | 21 | 30 – 65 mg/dL |
| **Vitamin E** | 0,3 | 0,5 – 0,7 mg/dL |
| **Vitamin D, 1,25OH** | 16 | 20 -76 pg/mL |

**Arterial Blood Gases**

|  |  |  |
| --- | --- | --- |
| **Parameter** | **Value** | **Reference ranges** |
| **pH** | 7,32 | 7,35-7,45 |
| **PaO2** | 78 | 98 mmHg |
| **PaCO2** | 32 | 35-40 mmHg |
| **SaO2** | 85 | >95% |

1. What is etiology of this disease? Pathogenetic mechanism?
2. Explain the hemodynamic changes in the patient?
3. Explain the respiratory changes in the patient?
4. Explain the cutaneous changes in the patient?
5. What biochemical tests shows impairment of liver function? What are liver-specific tests?
6. What is glycemic dyshomeostasis in the patient? Pathogenetic mechanism.
7. What are the changes in protein profile in the blood? Mechanisms? What other changes in protein metabolism can develop in chronic liver failure? Consequences
8. What are the changes in lipid profile in the blood? Mechanisms? Consequences
9. Explain the hematological changes in the patient. Pathogenetic mechanisms?
10. Is the patient anemic at this time and, if so, is the anemia normocytic, microcytic, or macrocytic?
11. What is the pathogeny of anemic syndrome in this patient?
12. What types of anemia can develop in patients with chronic liver failure? Pathogenetic mechanisms.
13. What are the hydro-electrolytic disturbances in the patient? Mechanisms?
14. Is there any evidence that this patient is at high risk for osteoporosis? Why
15. What are the acid-base disorders in the patient? Mechanisms.
16. What are the pathogenetic mechanisms of ascites in the patient?
17. Identify two abnormal laboratory tests that are consistent with ascites?
18. Explain the changes of diuresis?
19. Which laboratory test strongly suggests that the patient has developed hepatic encephalopathy?
20. What is the pathogenetic mechanism of hepatic encephalopathy?
21. Explain the pathophysiology of liver fibrosis?
22. Explain the pathophysiology of changes in bilirubin metabolism in this patient.

**Clinical case 2**

Patient A, 39 years old, admitted to the therapy department with the following complaints: headache, decreased work capacity, chest pain, nausea, polydipsia, itching, edema localized in the face, periorbital region. History of frequent angina.

**Objective:** pale, dry skin, decreased turgor. BP - 190/100 mm Hg. Blood: Hb - 90 g/L, erythrocytes - 3.2x10^12/L, leukocytes - 10.2x10^9/L, pH - 7.3, plasma osmolality > 290 mOsm/kg H2O. Total protein 50 g/L (65-85 g/L). Diuresis - 500 ml/24 h, nocturia. Zimnitsky test - urine density in all samples - 1010-1012. Creatinine clearance - 40 ml/min (norm 120 ml/min).

**Blood:** urea concentration - 17 mmol/L, creatinine - 50 mg/% (N - 0.5-1.2).

**Urine:** pink color (meat washing water), protein – 1,9 g/L with molecular mass > 70,000 (selectivity index - IgG/transferrin ratio > 0.1), leukocytes 2-3 per HPF, modified erythrocytes - many per HPF. Cylinders: hyaline - 2-4 per HPF, erythrocyte - 2-4 per HPF.

**Antistreptolysin O titer** - increased.

**Renal biopsy**: diffuse glomerular permeability, cellular infiltration with neutrophils and monocytes; endothelial and mesangial cell proliferation, interstitial edema in tubules, blood cells.

**Immunofluorescent investigations**: IgG and C3 deposits in mesangium and basement membrane.

**Diagnosis of post-streptococcal glomerulonephritis was established.**

**Questions:**

1. Considering the clinical symptoms and laboratory data, in which syndrome are they included? List the characteristic symptoms of the syndrome.
2. What is pathogenetic mechanism of this renal disorder?
3. What is the mechanism of pale skin?
4. What are changes of diuresis? Explain.
5. What changes are attested in urine of patient? Explain modification.
6. What type of syndrome is present – nephritic or nephrotic in this patient? Argument answer.
7. What is the pathogenesis of edema in this patient?

**Clinical case 3**

Patient J, 46 years old, suffers from lipoid nephrosis. Admitted to the therapeutic department with the following complaints: pronounced edema, weakness, low appetite.

**Objective:** pale, pasty skin, ascites, heart rate - 90 per minute, heart enlarged, heart sounds muffled. **Blood:** albumins - 15 g/L, dysproteinemia, hyperlipidemia, hypercholesterolemia, decreased antithrombin III, transferrin, gamma-globulins.

**Urine:** protein - 20 g/L with molecular mass < 70,000, selectivity index < 0.1. Cylinders - hyaline, waxy, epithelial, granular - up to 10 per HPF.

**Questions:**

1. Considering the clinical symptoms and laboratory data, in which syndrome are they included? List the characteristic symptoms of the syndrome.
2. What is pathogenetic mechanism of this renal disorder?
3. What is the pathogenesis of albuminuria in this syndrome?
4. What is the pathogenesis of hyperlipidemia, hypercholesterolemia?
5. What is the pathogenesis of pronounced edema in this patient?
6. What is the pathogenesis of decreased antithrombin III, transferrin, gamma-globulins and what are the consequences?
7. What changes are attested in urine of patient? Explain modification.

**Clinical case 4**

Patient K, 48 years old, is being treated in the therapeutic department with a diagnosis of chronic glomerulonephritis. The patient's condition has worsened dynamically: complaints of headache, dyspnea, nausea, vomiting, diarrhea, itching, hypersalivation, polydipsia, bone pain.

**Objective:** patient is apathetic, skin dry, gray-earthy color with hemorrhagic eruptions. BP 210-120, HR - 100, ECG - left ventricular hypertrophy, conduction disturbances, extrasystole, deep rare noisy breathing, signs of pulmonary congestion.

**Blood:** Hb - 80 g/L, erythrocytes - 4.0x10^12/L, creatinine - 6 mg/%, urea - 22 mmol/L, pH - 7.25, BE - -11 mmol/L, hyperkalemia, hypermagnesemia, hypocalcemia, hyponatremia, hypochloremia. Diuresis - 300 ml/24 h, isosthenuria, non-selective proteinuria, leukocyturia, hematuria.

**Questions:**

1. What is the pathogenesis of hyponatremia and hyperkalemia in chronic kidney disease?
2. What is the pathogenesis of hyperazotemia in chronic kidney disease?
3. What is the pathogenesis of bone pain in this patient?
4. What are the stages of chronic kidney disease?
5. What is the pathogenesis of changes attested on ECG, as conduction disturbances and extrasystoles?
6. What is the pathogenesis of deep rare noisy breathing, signs of pulmonary congestion?
7. What acid-base imbalance is attested in this patient?
8. What changes of diuresis is attested in this patient? Explain.

**Clinical case 5**

Patient X, 60 years old aged, reports the following **complaints** to the family doctor**:**

- The patient cannot maintain adequate glycemic control instead of using the maximum dose of metformin and sulfonylurea derivatives,

- The patient, being on antihypertensive treatment continues to experience frequent hypertensive episodes for the last 3 months

- Weight gain +4 kg within 2 months

- Burning sensation starting with the fingers and toes and spreading throughout the limbs.

**Anamnesis:** he is the chief of the village, has a stressful job. He doesn't follow the diet; he prefers fatty grilled meat and he faces the stress usually with a glass of wine. He has been diagnosed with diabetes for 8 years, and 2 years ago he suffered a myocardial infarction, and 1 a year ago he performed laser eye surgery. Frequent urinary infections on the background of erectile dysfunction.

**Objective: BP:** 170/100 mmHg, Ps: 68 -/min, Weight: 115 kg, Waist 182 cm,

**Paraclinical:** fasting blood sugar 182 mg/dl, total cholesterol= 520 (N<200 mg/dl), HDL=25 (N >40 mg/dl), LDL= 210 (<100mg/dl), TG 290 (N<150mg/dl), glycosylated Hb=11% (N=4.8-5.6%), serum sodium-160 mEq/l, potassium=3.1 mEq/l.

**Diagnosis:** DM type 2 complicated with diabetic macroangiopathy (atherosclerosis of the coronary arteries) and microangiopathy (proliferative diabetic retinopathy/diabetic peripheral neuropathy). HTA gr. II very high additional risk. Obesity gr. II. Dyslipidemia.

**Questions:**

1. Definition of insulin resistance. List the risk factors that predispose the concrete patient to type 2 DM.

2. Explain the pathogenesis of insulin resistance in the event of genetic defects occurring at the level of the insulin receptor and intracellular signaling pathways.

3. List 3 pathogenetic mechanisms by which obesity induces insulin resistance.

4. Describe the pathogenetic mechanism of insulin resistance related to increase in non-esterified fatty acids in the concrete patient with DM type 2.

5. Definition of lipotoxicity.

6. What are DAG and ceramides. The cause of their increase in the DM patient. Specify by which mechanism they induce insulin resistance.

7. Describe the role of adipokines in the development of insulin resistance.

8. It is known that obesity induces a pro-inflammatory state in the patient with DM type 2, explain why.

9. What is the impact of pro-inflammatory cytokines on the appearance of insulin resistance in the patient.

10. The patient is obese. It is known that adipose tissue can be a source of pro-inflammatory cytokines. List them and specify what are their effects.

11. What is glucotoxicity?

12. What type of hyperlipidemia is found in the specific patient.

13. How do we explain the increased values ​​of LDL and TG in the patient's blood.

14. What is glycosylated Hb and what is the mechanism of its formation.

15. AGEs are involved in the development of diabetic microangiopathy in the concrete patient. Give examples of AGEs.

16. AGEs are involved in the development of diabetic microangiopathy in the concrete patient. Where are the receptors for AGEs located?

17. AGEs are involved in the development of diabetic microangiopathy in the concrete patient. List their effects.

18. The given patient has a history of myocardial infarction caused by atherosclerosis of the coronary arteries. What is the mechanism of atherosclerosis in the patient with DM type 2?

19. One of the mechanisms of diabetic neuropathy appearance is the activation of the polyol pathway. Explain through the pathogenetic chain how neuron injury occurs when this pathway is activated.

20. Microvascular complications in the patient can also been explained by the activation of the protein kinase C pathway. List the resulting effects on the vascular endothelium.

21. Determine the pathogenetic chain of erectile dysfunction in the patient.

22. Describe the pathogenetic mechanism of recurrent urinary infections in the patient.

23. Explain the pathogenetic mechanism of hypernatremia in the indicated patient.

24. List two pathogenetic factors that control the exacerbation of metabolic acidosis in the patient with type 2 DM.

25.Give other one option of treatment in this concrete patient, that would help in maintaining of BP and body weight.

**Clinical case 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?**
2. **In DZ a triad of signs is specific - polyuria, polydipsia and polyphagia, which are also present in this patient. What is the mechanism of polyuria? (****Replay by pathogenetic chain)**
3. **In DZ there is a specific triad of signs - polyuria, polydipsia and polyphagia, which are also present in this patient. 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. **What is the mechanism of loss of body mass in this patient? (Replay by pathogenetic chain).**
6. **Which laboratory and blood gas data indicate an acid-base imbalance and which imbalance is present in this patient?**
7. **What is the mechanism of diabetic ketoacidosis?**
8. **What is the type of breathing and how does it work?**
9. **What are the mechanisms of reduced lipogenesis and enhanced peripheral lipolysis?**
10. **Which parameter in the laboratory data indicates dehydration of the patient? And which type of dehydration?**
11. **What Na+ ion imbalance is seen in the patient? What is the mechanism of this imbalance?**
12. **What K+ ion imbalance is seen in the patient? What is the mechanism of this imbalance?**

**Clinical case 7**

The patient was admitted to the emergency department with continuous abdominal pain that began spontaneously four hours prior. The pain has progressively intensified, accompanied by fever. She reports experiencing tremors, excessive sweating, a rapid heartbeat, weakness, and severe exhaustion. Additionally, she fainted this morning when getting out of bed.

**Medical History:** The patient has a history of pulmonary tuberculosis. Two years ago, ovarian TB was confirmed following a diagnostic laparoscopy conducted to investigate infertility in the couple. Over the past two years, she has felt chronically unwell, citing fatigue, mood swings between irritability and depression (which she attributes to the absence of a child in the family), occasional diarrhea (without fever), frequent episodes of faintness, polyuria, and nocturia. Weight loss is noted (18 kg over two years). Six months ago, an FGDS exam revealed atrophic gastritis. Her condition worsens under stress.

**Physical Examination**: Height: 172 cm; Weight: 60 kg; Blood Pressure: 90/45 mmHg at admission; Heart Rate: 105 bpm; Respiratory Rate: 22 bpm; Skin: bronzed appearance

**Laboratory Results**

1. **Complete Blood Count**
   * Hemoglobin (Hb): 85 g/L
   * Red Blood Cells (RBC): 2.1 x 10⁹/L
   * White Blood Cells (WBC): 14 x 10⁹/L
   * Hematocrit (Ht): 52%
2. **Urinalysis**
   * Urine: clear
   * Density: 1030
   * Proteinuria: absent
   * Bacteriuria: absent
   * Leukocyturia: absent

**3.** **Biochemical blood analysis**

|  |  |  |
| --- | --- | --- |
| **Parameter** | **Patients result** | **Norm** |
| Na⁺ | 122 | 135-145 mEq/l |
| K⁺ | 6 | 3,5-5,5 mEq/l |
| Mg⁺ | 2,8 | 1,6-2,6 mg/dl |
| pH | 7,32 | 7,35-7,45 |
| Osmolaritate sînge | 275 | 285- 295 mosmol/l |
| ALAT | 32 | <35 U/L |
| ASAT | 30 | <35 U/L |
| Bilirubina totala | 1,1 | <1,2 mg/dl |
| Bilirubina liberă | 0,75 | <1 mg/dl |
| Ureea | 47 | <43 mg/dl |
| Creatinina | 2,4 | <1 mg/dl |

**4. Endocrine Markers:**  
Cortisol (7:00-10:00) = 50 (172-497 nmol/L)  
ACTH (7:00-10:00) = 120 (7.2-63.3 pg/mL)  
Aldosterone = 0.5 (1.76-23.2 ng/dL)  
Aldosterone/Renin Ratio = 6 (Normal < 3.7)

Considering the acute abdomen, persistent pain unresponsive to antispasmodics, and dynamic increase in leucocytosis, an emergency diagnostic laparoscopy was performed after 24 hours, converting to laparotomy.

Intraoperatively, necrosis of the small intestine was found, and intestinal resection with side-to-end anastomosis was performed.

**Diagnosis:** Primary adrenal insufficiency (Addison's Disease), Mesenteric artery thrombosis.

**Questions:**

1. Based on symptoms and objective data, list the specific pathological changes of Addison's Disease.
2. List paraclinical criteria suggestive of Addison's Disease.
3. Explain the pathophysiological mechanism of hypotension in Addison's Disease.
4. Explain the pathophysiological mechanism of tachycardia in the patient.
5. Describe the pathophysiological chain of hypoglycemia in a patient with adrenal insufficiency.
6. Specify the pathogenic factors contributing to the patient’s weight loss over the past two years.
7. Describe the compensatory mechanisms in hypoglycemia induced by hypocortisolism.
8. How is the patient’s bronzed skin explained pathophysiological?
9. Explain the pathophysiological mechanism of diarrhea in the patient.
10. Why is creatinine elevated in a patient with hipocorticism?
11. What is the primary pathogenic factor that induced mesenteric artery thrombosis in the patient?
12. Why does blood pressure remain low despite sympathetic nervous system activation (due to hypoglycemia)?
13. Name the treatment of choice for managing an Addisonian crisis (hypoglycemia, hyponatremia, acidosis, hypotension).
14. Cortisol deficiency inhibits lipolysis. How do we explain the activation of lipolysis in hipocorticism?

**Clinical case 8**

**Patient X**, a 42-year-old man, consulted his general practitioner due to a skin and soft tissue injury and fever. The patient's history reveals he is a forest worker, and two days prior, he lost control of a chainsaw at work, cutting his leg. He took ibuprofen and dexalgin and treated the wound with hydrogen peroxide. However, the wound became infected, and by evening, he developed a fever. The general practitioner was surprised by the physical appearance of the patient, whom he hadn’t seen in two years: the patient had gained 18 kg, with fat distributed primarily around the trunk and face. Purple stretch marks had appeared on his abdomen, along with white, depigmented, itchy spots on his chest and back, and pronounced acne on his face. The patient complained of muscle weakness in his arms and legs, and of two fractures in the lower limbs within the past year when lifting weights, making it increasingly difficult for him to work in the forest.

The patient was admitted to the trauma hospital, where he underwent surgery requiring repeated cleaning and drainage of the post-operative wound, which healed very slowly. Upon discharge, the general practitioner contacted him to come to the health center for further investigations, which included:

**Hormonal and Blood Tests**:

* + **Cortisol** (7:00-10:00) = 900 (172-497 nmol/L)
  + **ACTH** (7:00-10:00) = 120 (7.2-63.3 pg/mL)
  + **K⁺** = 2.9 (3.5-5.5 mEq/L)
  + **Fasting Blood Glucose** = 145 mg/dL (70-126 mg/dL)
  + **BP** = 165/100 mmHg, Pulse = 98 /min

1. **Ultrasound** - Bilateral enlargement of adrenal glands.
2. **Brain MRI** - Detected a 1.5 cm diameter pituitary adenoma.

With these results, he was referred to an endocrinologist to confirm the diagnosis and determine the treatment plan.

**Questions:**

1. Establish the patient’s diagnosis.
2. Explain the pathophysiological reason for the prolonged post-operative period in this patient.
3. Explain the pathophysiological mechanism behind the patient's increased susceptibility to infections.
4. Explain, from a pathophysiological perspective, why the patient’s post-operative wound healing is delayed.
5. List the differentiating criteria between Cushing’s Disease and Cushing’s Syndrome.
6. What is the pathophysiological mechanism of hyperglycemia in the patient?
7. Explain the pathophysiological mechanism behind the preferential fat distribution that leads to moon face, buffalo hump, and truncal obesity in the patient.
8. What are the pathophysiological mechanisms of chronic hypertension in this patient?
9. Identify the pathophysiological mechanisms for the patient’s bone fractures.
10. Explain the pathophysiological basis for the appearance of depigmented and pruritic spots on the patient’s skin.

**Clinical case 9**

Women, 28 years old, Mrs. Adams is approached to the family doctor with the following complaints: fatigue, intolerance to heat, weight loss, pain in the bones, cardiac palpitations, irritability, amenorrhea, events that occurred the last six months. Her hand is shaking and she is not able to perform some procedures in the beauty salon where she works. She is worried also about frequent diarrhea. her menstruation appeared 3 months after birth (2 years ago), but suddenly disappeared 4 months ago. Pregnancy test was done- negative result.

Objective data: Weight 45 kg, Waist 168 cm, BP= 140/60mmHg, HR-118, RR-22. Soft, worm, with excessive sweating skin. Exophthalmos and staring gaze. On palpation thyroid gland is enlarged by 2 times. Pronounced tendon reflexes and fine tremor when stretching the arms.

|  |  |  |
| --- | --- | --- |
| Test | Result | Normal range |
| leucocytes | 3.1/mm³ | 3.5-5.5/mm³ |
| Ht | 41 % | 37-48 % |
| Urea | 56 mg/dl | <43 mg/dl |
| BUN | 26 mg/dl | <20 mg/dl |
| Alkaline phosphatase | 130 U/L | <104 U/l |
| Calcium serum ions | 13 mg/dl | 8.6-10.2 |
| T4 | 241 nmol/l | 66-181 |
| T3 | 4.6 nmol/l | 1.3-3.1 |
| TSH | 0.05 microUI/ml | 0.27-4.20 |
| Capture of radioactive iodine | 88% after 24 hours | 10-25% after 24 hours |
| Total HCG | negative | + during pregnancy |

Because of higher level T 4 and T3, instead of low TSH, TGIs was performed and found increased. Also, VLDL< LDL and total cholesterol plasma concentration are diminished. Fasting plasma glucose- 140 m/dl

Grave's Disease was established.

**Questions:**

1. Explain the pathogenetic mechanism of goiter in the concrete patient.
2. What is the pathogenetic mechanism of tachycardia in the patient,
3. Haw is explained hyperventilation in the patient.
4. What is the pathogenetic mechanism of hypercalcemia in the patient.
5. What is the pathogenetic mechanism of BP disorders in the patient.
6. Explain amenorrhea from pathogenetic point of view in this patient?
7. What are pathogenetic factors leading to weight loss in the patient?
8. It is known that lipolysis is activated in case of hyperthyroidism, why the amounts of VLDL, LDL and cholesterol remain low in the plasma?
9. Ht is determined as increased in the value, how is expected to be the amount of RBC? Why?
10. What kind of exophthalmos is detected in the patient? False or true? Pathogenetic mechanism.
11. Write the pathogenetic chain leading to hyperglycemia in the patient.
12. What is election treatment option in hypertension?
13. What elective treatment is given in grave's disease?