Colloquium #6

Questions

1) What is modern concept of peptic ulcer disease pathogenesis
2) What protective factors do you know? (6)
3) What enzymes H.Pylory releases?
4) What factors reduce mucus production? (2)
5) What factors enhance mucus production? (5)
6) What is the role of prostaglandins in protection of gastric mucosa? (6)
7) Mechanism of gastro-duodenal inhibition.
8) What aggressive factors do you know? (7)
9) What is the role of alimentary factor in the sores formation in the mucosal lining?
10) What medications (3) lead to the sores formation in the mucosal lining?
11) What is the role of medications in the sores formation in the mucosal lining?
12) What is the role STRESS in the sores formation in the mucosal lining?
13) Principles of treatment of peptic ulcer disease.
14) What is it hepatic failure?
15) What is the mechanism of hemorrhagic syndrome in the case of hepatic failure?
16) What is the mechanism of anemia syndrome in the case of hepatic failure?
17) Level of what vitamins will be reduced in the case of hepatic failure?
18) What consequences can be due to hypersplenism?
19) Pathogenesis of ascites in the case of portal hypertension?
20) Clinical symptoms of portal hypertension?
21) What enzymes will be increased in cholestasis?
22) Symptoms of acholia?
23) Symptoms of cholemia?
24) What types of jaundice do you know?
25) What type of bilirubin will be increased in a) hemolytic jaundice b) mild degree of hepatic jaundice c) severe degree of hepatic jaundice d) mechanical jaundice?
26) What enzymes will be increased in cytolysis?

27) What disorders of carbohydrate metabolism will be in the hepatocellular insufficiency?
28) What disorders of protein metabolism will be in the hepatocellular insufficiency?
29) What disorders of fat metabolism will be in the hepatocellular insufficiency?
30) What disorders of diuresis do you know? (3)
31) What is it polyuria? Causes of polyuria?
32) What is it oliguria? Causes of oliguria?
33) Causes of acute kidney injury
34) Stages of acute kidney injury
35) Stages of chronic kidney disease by Glomerular filtration rate
36) Pathogenesis of renal osteodystrophy
37) Symptoms of Urinary syndrome
38) What is it Proteinuria and its classification
39) What is it hematuria and its classification
40) What is the difference between azotemia and uremia?
41) What clinical symptoms of uremic syndrome do you know?
42) Pathogenesis of nephrotic syndrome
43) Pathogenesis of nephritic syndrome
44) What changes of urine density do you know
45) Pathogenesis of anemia in the case of renal failure
46) Pathogenesis of arterial hypertension in the case of renal failure

COLLOUQIUM #6

Pathophysiology of Digestion.

Case 1.

A 21-year-old man with an asthenic constitution and blood type O (I) (Rh-) was taken to hospital in a poor condition: his consciousness was confused, BP – 60/30 mm Hg, thready pulse with a rate of 120/min, breathing rate – 20/min. Hb – 40 g/l, erythrocyte count – 2.1 x 10^{12}/l; incoercible coffee-ground vomiting, diurnal diuresis – 200 ml. His relatives reported that the man had been following a course of "remedial fasting" without consulting a doctor for the last 2 weeks to treat his chronic bronchitis with a slight asthmatic component. During this period he did not eat anything and only drank fruit juices. Despite all treatment measures the doctors were not able to stop profuse bleeding and save the patient's life. The post mortem examination showed two large "kissing" ulcers of the pyloric region of the stomach.

1. Explain possible pathogenesis of gastric ulcer formation.
2. What are the clinical manifestations of the disease caused by?

Case 2.

Patient D., 42 years old, a locomotive-driver, in March applied to his physician with complaints of intensive dull pains in the epigastrium radiating to the lumbar region and appearing 1.5 – 2 hours after meals and at night which disappeared after consuming a small amount of food. The patient had similar symptoms the previous autumn but he did not go to a doctor and treated himself with a diet. All the winter he did not have any symptoms. In the spring, however, his pains renewed, became more intensive, heartburns appeared. Twice the patient had nausea and vomiting, constipations became more frequent, once he noticed a tarry stool. Over the last 2 months he has lost 2 kg of weight in spite of a good appetite. The patient smokes a lot (up to 2 packs a day), has a cough with sputum (especially in mornings). He

also complains of bad sleep, increased fatigability, irritability, unstable mood, increased sweating.

Examination data: The patient has an asthenic constitution, low body mass; hand type is radial, foot type is intermediate, blood type – O(I). The skin is pale with marked nasolabial folds. The tongue is white and coated, filiform and fungiform papillae are hypertrophic. The abdomen is painful and resistant on palpation, especially in the epigastric region. Mendel's syndrome is positive (tenderness in the epigastrium on percussion). BP – 110/60 mm Hg; heart rate – 60/min; Hb – 105 g/l; erythrocytes – 4.5 x 10^{12}/l; leukocytes – 9 x 10^{9}/l. Urine test is normal. Feces analysis showed creatorrhea and steatorrhea, fecal occult blood test is positive. Basal and stimulated HCl secretion is higher than the norm. Coefficient of gastric juice aggression approaches 1. X-ray examination detected a "niche" symptom in the duodenal bulb area.

1. Make a conclusion about the nature of the patient's main disease. Justify your conclusion.
2. Explain the etiology and pathogenesis of the disease.
3. What additional investigations would you carry out?
4. Point out the principles of pathogenetic therapy for this disease.

Case 3

Patient N., 52 years old, a gastroenterologist, often performs endoscopic investigations. Recently she has begun to note gnawing pains in the left epigastric region radiating to the xiphisternum region and left part of the chest. She believed the pains to be caused by angina pectoris and took validol, but unsuccessfully. The patient has paid attention to the fact that pains appear 30 minutes – 1 hour after meals. She has also noted bitter eructation and meteorism, unstable stools, frequent diarrhea. She sleeps badly, her working ability has decreased. She gets tired quickly and has become irritable; her mood is often bad, sometimes depressive.

Examination data: normosthenic body type, subcutaneous adipose layer is within the norm. The tongue is coated and white, the papillae are flattened. Bad smell

from the mouth (halitosis); on palpation – tenderness in the left epigastric region. Blood pressure – 140/80 mm Hg, heart rate – 70/min; breathing rate – 16. Hemoglobin – 115 g/l, leukocytes – 8 x 10^9/l. Urine and feces tests are normal. Basal and stimulated secretion of gastric juice is sharply decreased. A test for urease presence in the gastric juice is positive. Proteolytic activity of the gastric juice is increased. Fucose and N-acetylneuraminic acid (NANA) content in the gastric juice is decreased. X-ray examination reveals a "niche" symptom in the upper part of the cardial region of the stomach.

1. Make a conclusion about the nature of the patient's main disease. Justify your conclusion.
2. Explain possible pathogenesis of the disease and the mechanism of the symptoms.
3. Point out the principles of pathogenetic therapy.
4. What complications can this disease lead to?

Pathophysiology of the Liver.

Case 1.

Patient Sh., a 48-year-old nurse at a TB dispensary, had been having general weakness, ache in the muscles and joints of the extremities, skin itching, continuous nausea (once she vomited) and a decreased appetite for a week before hospitalization. For four days she had been also having a fever (37.5 – 37.7˚C) and taking an anti-flu medicine on her doctor's advice. She was admitted to a hepatology center after having developed jaundice. Her condition was of moderate severity, but persistent skin itching, bad sleep and headaches added to the previous symptoms.

Physical examination findings: marked jaundice of the skin, sclera and mucosa; single hemorrhages on the skin; coated white tongue. The liver is 3 cm lower than

the costal arch, soft, tender on palpation and percussion. The spleen is not enlarged.

Blood test: Hb -120 g/l, erythrocytes – 4.5 x 10^{12}/l, leukocytes – 4.7 x 10^{9}/l, erythrocyte sedimentation rate (ESR) – 27 mm/h. AlAT activity - 4 times as high as normal, increased alkaline phosphatase (AP) activity. Total bilirubin – 156.9 mCmol/l, bilirubin index – 81%. "Australian" antigen and increased IgG are detected. Prothrombin index - 73%; decreased content of proaccelerin and proconvertin; decreased albumin/ globulin coefficient. Fasting glucose level ranges from 2 to 4.5 mmol/l.

The jaundice and itching persisted for 45 days. The patient was discharged after two months of treatment with AlAT twice as high as normal.

1. What type of jaundice did the patient have? What are possible causes of its development?
2. Justify your conclusion.
3. Explain the mechanism of the symptoms and changes in the laboratory data.
4. What syndromes are observed in this patient?
5. What changes can be found in the patient's urine?

Case 2.

Patient S., a 32-year-old woman, was admitted to hospital with complaints of acute weakness, dizziness, dyspnea at rest, pain in the heart area and palpitations, headache.

Examination findings: the skin is pale with a lemon-yellow tint, the sclera – icteric, the spleen is slightly enlarged and painful on palpation. BP -140/80 mm Hg, HR – 90 – 100/min.

Blood test: Hb – 40g/l, erythrocytes – 1.5 x 10^{12}/l, reticulocytes – 28%, platelets – 240 x 10^{9}/l, leukocytes – 14.5 x 10^{9}/l. Coombs test is positive. Total bilirubin – 80 mCmol/l, bilirubin index – 20%, serum iron – 45 mCmol/l, total protein level and albumin/globulin index are normal, activity of alanine-aminotransferase (AlAT), aspartate-aminotransferase (AsAT), alkaline phosphatase (AP) and gamma-

glutamyltranspeptidase (GGT) – within the norm; the level of LDG is increased, prothrombin index – 95%, ESR – 38 mm/h.

The feces are hypercholic, the urine – dark. The patient is known to have been taking sulfonamide medications for a long time.

1. What type of jaundice does the patient have? Justify your conclusion.
2. Explain the pathogenesis of jaundice in this patient.
3. Name the main features distinguishing this type of jaundice from the others.
4. Explain the mechanism of the symptoms and changes in the laboratory data.

Case 3.

Patient T., a 55-year-old man, was admitted to hospital with complaints of jaundice of the skin and sclera, skin itching, feeling of heaviness in the right infracostal and epigastric area, weakness, dark urine. He became ill three days ago, when he suddenly felt a severe pain in the right infracostal and epigastric area radiating to the right shoulder and lumbar region. The attack had lasted for about two hours and was stopped with an intravenous administration of spasmolytic drugs. A similar attack occurred two days after the first one and was accompanied by nausea, vomit, chills, and temperature rise (up to 38˚C). The next day jaundice, skin itching and colorless feces appeared.

Physical examination findings: The condition is satisfactory, the skin and the sclera are icteric with a greenish tint. BP – 100/60 mm Hg, HR – 54/min, the tongue is moist, the abdomen – soft and tender in the right infracostal area. The liver and gall bladder are not palpated.

Blood test: Hb – 130 g/l, erythrocytes – 4.3 x 10^{12}/ l, reticulocytes – 0.5%, leukocytes – 11.5 x 10^{9}/ l, ESR – 20 mm/h. Total bilirubin is 149 mCmol/l, conjugated bilirubin (CB) – 97 mCmol/l. Activity of alkaline phosphatase (AP) and gamma-glutamiltranspeptidase (GGT) is elevated. AlAT and AsAT are normal. Albumin/globulin index is unchanged; prothrombin index – 95%.

The feces are colorless and contain free fats and fatty acids. The urine is dark and foamy when shaken.

1. What type of jaundice does the patient have? Justify your opinion.
2. Explain the pathogenesis of jaundice in this patient.
3. Make a differential diagnostics with other types of jaundice.
4. Explain the mechanism of the symptoms and changes in the laboratory data.
5. What tactics should a physician choose?

Case 4.

Patient D., 23 years old, applied to an out-patient department because of icteric sclera and dark urine. She had become ill five days before: she was feeling general weakness, nausea, loss of appetite; for three days she had been running a temperature (37.8˚C) with chills. A month before her younger sister had had jaundice.

Examination findings: jaundice of the skin and sclera is not strongly marked. The urine has a color of strong tea, when it is shaken orange foam appears. The feces are of light color. The liver is 1.5 cm below the edge of the costal arch, soft and tender on palpation. The spleen is slightly enlarged.

Blood test: Hb – 140 g/l, erythrocytes – 4.5 x 10^{12}/l, leukocytes – 5.0 x 10^{9}/l, ESR – 9 mm/h. AlAT activity is increased. Total bilirubin is 66 mCmol/l, bilirubin index – 73%. Thymol test is strongly positive, the content of IgM is increased, "Australian" antigen (HbsAg) is absent. Prothrombin index is 85%. The content of urea, ammonium and amino acids is normal. Fasting glucose level – 3- 4 mmol/l.

The icteric period lasted 4 days. The patient was discharged on the 30th day of the disease in a satisfactory condition with normal AlAT.

1. What type of jaundice did the patient have? Justify your conclusion.
2. Explain the pathogenesis of jaundice in this patient.
3. Explain the mechanism of the symptoms and changes in the laboratory data.
4. Assess the functional state of the liver using the data of the laboratory tests.

Case 5

Patient B., 38 years old, two years ago had a cold after which fever and the symptoms of polyneuritis developed (she could not hold a spoon); the patient lost weight. On examination she was found to have anemia, thrombocytopenia, increased ESR, enlarged liver. A diagnosis "nodular periarteriitis" was made, and the patient was treated with glucocorticoids. Half a year after the treatment with prednisolone had been discontinued, she developed weakness, pains in the right infracostal area and in the epigastrium, dyspepsia, meteorism, skin itching, subfebrile temperature. Physical examination showed jaundice of the sclera, vascular "spiders" on the chest and shoulders, hyperemia of the face and palms. The liver projected 9 cm out of the costal arch, the spleen was near the edge of the costal arch. Some symptoms of polyradiculoneuritis were still present.

Blood test: Hb – 80g/l; leukocytes – 10 x 10^9/l; ESR – 57 mm/h; total protein – 67 g/l, A/G coefficient is decreased. Conjugated bilirubin – 40 mCmol/l, bilirubin index – 70%. AlAT activity 4 times as high as normal; there is a two-fold increase in AP and GGT. Scanning of the liver revealed its marked enlargement with a diffuse- focal decrease in the accumulation of the preparation in all parts.

The patient's relatives reported that she had been abusing alcohol for 10 years. A considerable improvement of the patient's condition was achieved by administering *Essentiale* intravenously and orally.

1. What liver pathology can be most likely suspected in this patient?
2. What symptoms and syndromes can be found in this pathology?
3. Explain their mechanism.
4. Determine the type of jaundice.

Pathophysiology of the Kidneys.

Case 1.

Patient B., 39 years old, was admitted to an internal disease department with complaints of persistent headaches, impaired vision, fatigability, pains in the heart,

nausea, continuous thirst, skin itching, face edemata in the morning. The patient is known to have frequent anginas (tonsillitis). He has been having these symptoms for more than a year.

Examination findings: The patient is pale, the skin is dry. The heart boundaries are extended to the left. BP is 190/100 mm Hg, breathing rate – 25/min.

Blood test: Hb – 90 g/l; erythrocytes – 3.2 x 10^{12} /l; leukocytes – 6.2 x 10^{9}/l, plasma osmolality is > 290 mOsm/l; blood pH – 7.3. Diurnal diuresis is 4 liters, nocturia. In Zimnitsky test the relative urine density in all portions is 1010 – 1012, concentration index – 1.2; creatinine clearance - 40 ml/min; blood urea – 17 mmol/l, creatinine – 0.5 mmol/l.

Urine test: protein 1 – 1.92 g/l (molecular mass >70, 000); leached erythrocytes – 5 – 6 in the vision field, casts – 2 – 4 in the vision field. Selectivity index (ratio of IgG and transferrin) is > 0.1.

1. Assess the functional state of the renal glomerules and renal tubules. Justify your conclusion.
2. Make a preliminary conclusion about the patient's renal pathology and determine the stage of the disease.
3. Explain the etiology and pathogenesis of this disease.
4. Explain the pathogenesis of the main renal and extrarenal syndromes.
5. List the principles of pathogenetic therapy for this patient.

Case 2

Patient K., 28 years old, was admitted to hospital 3 weeks after having tonsilitis, with complaints of dull pain in the loin, headache, frequent urination, face edemata (especially in the morning), dyspnea, thirst.

Examination findings: The patient is pale, with edemata on the legs and especially on the face. BP is 180/100 mm Hg; HR – 100; on ECG – signs of diffuse lesion of the myocardium.

Blood test: Hb – 100 g/l; ESR – 26 mm/h; BUN (blood urea nitrogen) – 70-85 mmol/l; increased level of globulin fractions in the blood. Diurnal diuresis is 1litre;

relative urine density – 1029; concentration index – 3.3. The urine is cloudy, looks like “meat slops” and contains large quantities of erythrocytes as “shadows”; leukocytes - 6-8 in the vision field, hyaline casts – 3 – 4 in the vision field; protein – 1.5 – 2 g/l (molecular mass < 70, 000). Creatinine clearance is less than 50% of the norm. In the needle bioptate of the kidneys deposits of immune complexes are detected.

1. What disease are these clinical features and laboratory findings typical of? Justify your conclusion.
2. Explain the etiology and pathogenesis of this nephropathy.
3. Assess the filtration and concentration function of the kidneys.
4. Explain the mechanism of the patient’s symptoms and syndromes.

Case 3.

Patient N., a 20- year-old woman, was admitted to hospital with signs of sepsis in an extremely severe condition.

Examination findings: The patient is pale with a cyanotic tint and jaundice of the sclera; the body temperature is 36 – 37˚C in the morning and 41 – 42˚C in the evening. She has chills, confused consciousness, nausea, vomit (with the smell of ammonium), edemata all over the body, convulsions and meningeal symptoms. The heart rate is 110/min; the heart boundaries are extended to the left; the tones are muffled; there is a pericardial friction murmur. ECG shows decreased voltage, extrasystoles. The patient has Kussmaul breathing and unstable blood pressure. Diurnal diuresis is about 100 ml, the urine is dark and cloudy; hemoglobinuria, nonselective proteinuria (up to 5 g/l), leukocyturia, large amounts of casts. Concentration index is 0.9.

Blood test: Hb – 60 g/l; pH – 7.0; BUN – 290 mmol/l; creatinine – 0.9 mmol/l; urea – 29 mmol/l; potassium– 7 mmol/l.

1. Assess the functional state of the kidneys.

2. What nephropathy are these clinical features and laboratory findings typical of? Determine the form and stage of this nephropathy. Justify your conclusion.
3. What renal and extrarenal syndromes are characteristic of this pathology? Explain their pathogenesis.
4. Point out the principles of treatment of this pathology.

Case 4

A 46-year-old woman suffering from lipoid nephrosis was admitted to an internal disease department with complaints of marked edemata, weakness, bad appetite.

Examination findings: The patient is extremely pale, edematous, having ascites. The heart sounds are muffled. HR is 96; the heart boundaries are extended.

Blood test: albumins – 15g/l; dysproteinemia, hyperlipidemia, hypercholesterolemia.

In the urine: protein – 20g/l daily (protein molecular mass > 70, 000); hyaline, waxy and granular casts – up to 10 in the vision field.

1. What is this syndrome called?
2. Explain the mechanism of the edemata, presence of casts in the urine and proteinuria.
3. What type of proteinuria is typical of this syndrome? Justify your answer.
4. State the most likely cause of antithrombin III, transferrin and γ-globulin deficiency in this patient. What are the consequences of this condition?

Case 6.

Patient D., a 12-year-old girl, was admitted to hospital with complaints of headache, chills, temperature rise to 39 – 40˚C, pains in the loin and abdomen, frequent and painful urination.

Examination findings: the tongue is dry and coated; BP – 100/60 mm Hg; kidney area is painful on palpation.

In the blood: Hb – 120 g/l; neutrophil leukocytosis with the left shift, BUN (blood urea nitrogen) – 11 mmol/l, ESR – 35 mm/h.

In the urine: protein < 1 g/l (albumins and β- microglobulins); erythrocytes – 3-5 in the vision field (of irregular form < 15%); leukocytes – 25-30 in the vision field; bacteriuria > 150, 000/ml. Diurnal diuresis is 2 – 2.5 liters, hyposthenuria. Creatinine clearance – 100 ml/min.

1. What renal disease are these clinical features and laboratory findings typical of?
2. Assess the function of the renal glomerules and tubules in this patient.
3. Explain the etiology and pathogenesis of this disease and the mechanism of the symptoms.
4. List the principles of therapy of the disease.