Ministry of Education, Science, Youth and Sports of Ukraine
Sumy State University
Medical Institute

3512 Methodological instructions

for practical classes
on the subject “Internal medicine”
for the students of speciality “Medical practice”

Module 3
MODERN PRACTICE OF INTERNAL MEDICINE

Module 4
EMERGENCIES IN INTERNAL MEDICINE CLINIC

Sumy
Sumy State University
2013

3512 Methodological instructions for practical classes on the subject “Internal medicine”. Module 3. Modern practice of internal medicine. Module 4. Emergencies in internal medicine clinic / compilers: G. A. Fadeeva, V. G. Psareva, L. N. Prystupa, V. F. Orlovskyi, V. V. Laba, N. M. Kyrychenko, O. S. Pogorelova, O. V. Orlovskyi, Y. O. Ataman, N. O. Murenets. – Sumy : Sumy State University, 2013. – 91 p.

Department of internal medicine of postgraduate education

Module 3
MODERN PRACTICE OF INTERNAL MEDICINE

Semantic module 1. Management of patients in cardiology clinic

1. MANAGEMENT OF PATIENS WITH ARTERIAL HYPERTENSION (AH)

Time frame – 6 hours.

Professional motivation. Individuals with a systolic BP of 120 to 139 mm Hg or a diastolic BP of 80 to 89 mm Hg should be considered as prehypertensive and require health-promoting lifestyle modifications to prevent cardiovascular disease. The prevalence of hypertension increases with age and most individuals with hypertension are diagnosed with primary (essential) hypertension. Hypertension occurs in 10–20% of persons aged 25 to 45 years and 30–40% of persons aged 55 to 74 years. Although it is a "silent" disease, these patients usually have no specific symptoms, hypertension is posed as a major risk factor for coronary artery disease (heart attack), cerebrovascular disease (stroke), and renal disease (kidney failure). It is the second most common reason for office visits to physicians in the United States. Analysis of the Framingham study data suggested that individuals aged 40 to 69 years have an increasing risk of stroke or coronary artery disease mortality with every 20 mm Hg increment in systolic blood pressure. On the other hand, a recent Cochrane review revealed that aiming for blood pressure targets lower than 140/90 mm Hg is not beneficial, as it is not proven that this approach will reduce heart attack and stroke.

There are 2 categories of hypertension. Over 90% of all cases of high blood pressure are called *"essential hypertension"*, which has no specific identifiable cause but is due to the body inability to regulate the blood pressure within the normal range – SBP 120–140 mm Hg / DBP 60–85 mm Hg. Onset is usually between ages 30 and 50 years. Essential hypertension is treated with medication, diet, and fluid restriction and is not curable. *"Secondary hypertension"*, on the other hand, is high blood pressure that has an identifiable cause, occurs in a wide age range, is severe, and is abrupt in onset. Secondary hypertension is potentially curable because it is most commonly caused by *stenosis* of the renal arteries. Less often, secondary hypertension can be caused by tumours of the adrenal gland that secrete hormones acting to increase the blood pressure.

Place of carrying out: class-room, wards of the cardiology department, ward of the emergency, department of functional diagnostics.

Study objective: to be able to put provisional diagnosis and assign management.

Basic level:

1. Mechanism of blood pressure regulation.
2. To be able to collect complaints, case history, carry out objective examination. Methods of blood pressure measurement.
3. To interpret instrumental and laboratory data in patients with arterial hypertension.
4. To discover signs inherent to AH.
5. To interpret side effects of antihypertensive drugs. To use diet for AH correction.

Student has to know how to examine patients with cardiovascular disorders.

The main theoretical questions:

1. Definition of arterial hypertension. Essential AH. Secondary AH.
2. Epidemiology and classification of the arterial hypertension.
3. Rick factors for arterial hypertension.
4. Complications in arterial hypertension.
5. Observantional program of the persons with arterial hypertension.
6. Differential diagnosis in AH of different aetiology.
7. General principles of antihypertensive therapy:
   a) recommendations on lifestyle modification;
   b) the general measures employed;
   c) risk factors for an adverse prognosis in hypertension.

8. Recommendations of the European Society of Hypertension for AH treatment.

9. Approach to drug therapy. Antihypertensive “step by step” therapy.

10. Antihypertensive drugs: diuretics, ß-adrenergic blocking agents, angiotensin-convetrting enzyme inhibitors, angiotensin receptor antagonists, calcium channel antagonists, α-adrenergic receptor blockers.

11. Drug combinations.

12. Treatment in AH adjusted to ethnicity, age, pregnancy, concomitant diseases and complications (renal disease, coronary artery disease, diabetes mellitus, obesity).

13. Hypertensive crisis: classification of the hypertensive crises; sequence of clinical events in hypertensive emergencies; treatment of the hypertensive crises.

Assignment for self-assessment

1. What laboratory tests are included to the hypertension management program?

2. At a routine company physical examination, an asymptomatic 46-year-old man is found to have a BP of 150/110 mm Hg, but no other abnormalities are present. What should be done next?

Answers:

1. Urine for protein, blood, and glucose; microscopic urinalysis; serum creatinine and/or blood urea nitrogen; total cholesterol.

2. Obtain repeated BP recording in your office and/or the patient’s home or work site.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald’s heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology
12. https://www.ers-education.org/e-learning/procedure-videos/

2. MANAGEMENT OF PATIENS WITH CARDIALGIA. DIFFERENTIATION BETWEEN THE CORONARY AND NONCORONARY CARDIALGIA

Time frame – 6 hours.

Professional motivation. The prevalence of chest pain or chest discomfort varies in different parts of Europe. In a British study of 7735 men, angina pectoris or a history of possible acute myocardial infarction (AMI) was reported in 14% and a further 24% suffered from atypical chest pain. The underlying cause of chest pain varies depending on whether a patient is seen by a general practitioner, calls the dispatch centre, is treated by the ambulance crew or is seen at the emergency department. Not unexpectedly, chest pain of cardiac origin is less commonly seen by the general practitioner (20%), whereas musculoskeletal disorders are common. A summary of prospective studies in general practices in the Netherlands, in England, and in Iceland shown that most of the episodes were caused by musculoskeletal problems and only about 20% were of cardiac origin. Patients with chest pain without a somatic diagnosis often suffer from psychiatric problems such as anxiety, depression or alcohol abuse.

The ischaemic origin of calls about chest pain is much more frequent at dispatch centres. About 25% of all emergency calls to a dispatch centre are initiated because of chest pain. Among such patients, 40% are reported to have confirmed myocardial ischaemia or infarction, and 66% either confirmed or possible myocardial ischaemia or infarction as the cause of their pain. Patients with acute myocardial infarction who call for an ambulance are different from those who do not. They are older, more likely to be female and have a higher prevalence of previous cardiovascular disease and more severe symptoms. They develop more complications and present a higher risk of cardiac arrest and death. The number and proportion of hospital admissions for chest pain vary. Data from the U.S. showed that 20% of all nonsurgical admissions are for chest pain, in patients with chest pain 17% ultimately met the criteria for cardiac ischaemia and 8% had myocardial infarction. Overall, a similar proportion of men and women seek medical care due to non-ischaemic chest pain. In some subsets such as patients with chest pain due to psychiatric causes there might be an over-representation of women. Patients with non-ischaemic chest pain also have a lower prevalence of various risk indicators, such as a history of previous acute myocardial infarction, angina pectoris, hypertension, and diabetes. Smoking is more frequent in this patient population. There are different types of non-ischaemic causes of chest pain: reflux oesophagitis, oesophageal spasm, pulmonary embolism, spontaneous pneumothorax, aortic dissection, pericarditis, pleuritis, early herpes zoster, peptic ulcer, cholecystitis, pancreatitis.

IHD is the leading cause of mortality in the United States and the rest of the developed world; it is responsible for more than 20% of deaths. In the United States, approximately 1 million persons suffer from MI, and 500,000 coronary deaths occur each year. IHD is the leading cause of death in the United States for both sexes in both white and black populations. The prevalence of IHD increases with age and is higher in men than in women in every age group. The American Heart Association conservatively estimates that more than 6 million persons in the United States experience angina. In addition to posing an increased risk of MI and premature death, chronic stable angina often limits affected persons' capacity for work and other activities, which, in turn, negatively affects their quality of life. The direct and indirect costs of hospitalization, diagnostic procedures, and revascularization related to angina are substantial. Of patients with angina who undergo a coronary revascularization procedure, 30% or more never return to work.

The major modifiable risk factors for IHD are dyslipidemias – in particular, elevated levels of low-density lipoprotein (LDL) cholesterol and low levels of high-density lipoprotein (HDL) cholesterol – as well as hypertension, diabetes mellitus, and cigarette smoking. Other important, but immutable, risk factors are increasing age, family history of premature coronary disease, and male sex. Obesity, physical inactivity, and atherogenic dietary habits also contribute to cardiovascular risk, although it is difficult to distinguish the risks conferred by these risk factors independently of the risks conferred by the major cardiovascular risk factors because of the potential interaction of these factors. Patients with combinations of risk factors may be at particular risk for developing IHD.

Place of carrying out: class-room, wards of the cardiology and rheumatology departments, department of functional diagnostics, X-ray department.

Study objective: to be able to determine extent of examinations to put final diagnosis and assign management.

Basic level:

1. To be able to collect complaints, case history, carry out objective examination.
2. To interpret instrumental (ECG, EchoCG, X-ray) and laboratory data in patients with chest pain.
3. To identify signs from anamnesis and objective data inherent to cardialgia of different origin.
4. To interpret side effects of antianginal agents. To use physiotherapeutic procedures and diet for correction of chest pain related to diseases of musculoskeletal system, oesophagus, abdominal cavity organs.

Student has to be able to:

1. Examine patients with cardiovasculal disorders.
2. Make an algorithm of investigations in patients with cardialgia.
3. Determine approaches to treatment in different aetiology of cardialgia.

The main theoretical questions:

1. Aetiology of cardialgia. The main pathogenetic mechanisms in cardialgia development.
2. Clinical signs of cardialgia depending on aetiology: in coronary diseases, in neurocirculatory dystonia, in pericarditis, myocarditis, valvular disorders, lung and pleural diseases, in oesophageal and abdominal organs pathology, in aortic aneurism.
3. An algorithm for the diagnosis of acute chest pain.
4. Management of patients with different aetiology of cardialgia.
5. The main drugs for treatment of functional disorders of cardiovascular system.
6. The main drugs for treatment of noncoronary myocardium diseases.

Assignment for self-assessment

1. A 44-year-old woman is presenting with prolonged stabbing chest pain on the left from sternum, dizziness, paresthesia, general sweating, sleeplessness. She's sick for a year. The examination reveals emotional lability, a regular heart rate with a systolic murmur above heart apex. Blood pressure is 120/80 mm Hg; pulse is 88 beats/min, regular, respiratory rate is 16 breaths/min. Her lungs are clear. The abdomen is soft without tenderness or distention. The liver spans 10 cm in the midclavicular line with a smooth edge. There is no peripheral oedema, pulse is intact. Her weight is 84 kg, height is 176 cm. She is afebrile. On ECG: sinus rhythm, negative T-waves in V1-V4 leads which disappear (T-waves become positive) after potassium or propranolol test. Suggested diagnosis is:

a) dishormonal cardiomyopathy;
b) IHD: stable angina, FCII;
c) infective myocarditis;
d) rheumocarditis.

2. A 28-year-old man is presenting with chest pain, palpitations, and dyspnoea after adenoviral infection. Examination revealed pale skin, acrocyanosis, a regular weakened heart beats, cardiac borders expanded to the left and to the right. Blood pressure is 90/60 mm Hg; pulse is 92 beats/min, respiratory rate is 20 breaths/min. His lungs are clear. The abdomen is soft without tenderness or distention. The liver spans 10 cm in the midclavicular line with a smooth edge. There is no peripheral oedema. On ECG: decreased voltage of R-waves, PQ 0.22 sec. Suggested diagnosis is:

a) viral myocarditis;
b) infective endocarditis;
c) rheumatic myocarditis;
d) exudative pericarditis;
e) dilated cardiomyopathy.

Answers: 1. a. 2 a.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International

Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology
12. https://www.ers-education.org/e-learning/procedure-videos/

3. MANAGEMENT OF PATIENS WITH ARRHYTHMIAS

Time frame – 6 hours.

Professional motivation. Supraventricular arrhythmias are relatively common, often repetitive, occasionally persistent, and rarely life-threatening. The precipitants of supraventricular arrhythmias vary with age, gender, and associated comorbidity. While supraventricular arrhythmias are a frequent cause of emergency room and primary care physician visits, they are infrequently the primary reason for hospital admission.

The estimated prevalence of paroxysmal supraventricular tachycardia (PSVT) in a 3.5% sample of medical records in the Marshfield (Wisconsin, the USA.). Occurrence rates have been determined for various subtypes of supraventricular arrhythmia after acute myocardial infarction or coronary artery bypass graft surgery and in congestive heart failure (CHF) patients. The incidence rate of supraventricular arrhythmias among patients with CHF is 11.1%; paroxysms are more common in older patients, males, and those with longstanding CHF and radiographic evidence of cardiomegaly.

Age exerts an influence on the occurrence of SVT. The mean age at the time of PSVT onset in the MESA cohort was 57 years (ranging from infancy to more than 90 years old). Among emergency room patients older than 16 years treated with intravenous (IV) adenosine for supraventricular arrhythmias diagnosed by surface electrocardiogram (ECG) criteria, 9% had atrial flutter and 87% had SVT; 70% of these patients (age 51 plus or minus 19 years) reported a history of cardiovascular disease. In the MESA population, compared to those with other cardiovascular disease, "lone" (no cardiac structural disease) PSVT patients without associated structural heart disease were younger (mean age equals 37 vs. 69 years), had faster heart rates (186 vs. 155 beats per minute), and were more likely to present first to an emergency room (69 vs. 30%).

Gender plays a role in the epidemiology of SVT. Female residents in the MESA population had a twofold greater relative risk of PSVT (RR equals 2.0; 95% confidence interval equals 1.0 to 4.2) compared to males. Fifty-eight percent (58%) of symptomatic "lone" PSVT episodes in MESA females without concomitant structural heart disease occurred in the premenopausal age group, as compared to only 9% of episodes in women with cardiovascular disease.

The only reported epidemiologic study of patients with atrial flutter involved a selected sample of individuals treated in the Marshfield Clinic in predominantly white, rural mid-Wisconsin. Over 75% of 58,820 residents and virtually all health events were included in this population database. In approximately 60% of cases, atrial flutter occurred for the first time associated with a specific precipitating event (i.e., major surgery, pneumonia, or acute myocardial infarction). In the remaining patients, atrial flutter was associated with chronic comorbid conditions (i.e., heart failure,

hypertension, and chronic lung disease). Only 1.7% of cases had no structural cardiac disease or precipitating cause (lone atrial flutter). The overall incidence of atrial flutter was 0.088%; 58% of these patients also had AF. Atrial flutter alone was seen in 0.037%. The incidence of atrial flutter increased markedly with age, from 5 per 100000 of those more than 50 years old to 587 per 100 000 over age 80. Atrial flutter is 2.5 times more common in men.

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia, occurring in 1–2% of the general population. Over 6 million Europeans suffer from this arrhythmia, and its prevalence is estimated to at least double in the next 50 years as the population ages. AF confers a 5-fold risk of stroke, and one in five of all strokes is attributed to this arrhythmia. Ischaemic strokes in association with AF are often fatal, and those patients who survive are left more disabled by their stroke and more likely to suffer a recurrence than patients with other causes of stroke. In consequence, the risk of death from AF-related stroke is doubled and the cost of care is increased 1.5-fold. AF may long remain undiagnosed (silent AF), and many patients with AF will never present to hospital. Hence, the "true" prevalence of AF is probably closer to 2% of the population. The prevalence of AF increases with age, from < 0.5% at 40–50 years, to 5–15% at 80 years. Men are more often affected than women. The lifetime risk of developing AF is 25% in those who have reached the age of 40. The prevalence and incidence of AF in non-Caucasian populations is less well studied. The incidence of AF appears to be increasing (13% in the past two decades).

AF is associated with increased rates of death, stroke and other thromboembolic events, heart failure and hospitalizations, degraded quality of life, reduced exercise capacity, and left ventricular (LV) dysfunction. Death rates are doubled by AF, independently of other known predictors of mortality. Only antithrombotic therapy has been shown to reduce AF-related deaths. Stroke in AF is often severe and results in long-term disability or death. Approximately every fifth stroke is due to AF; furthermore, undiagnosed "silent AF" is a likely cause of some "cryptogenic" strokes. Paroxysmal AF carries the same stroke risk as permanent or persistent AF.

Hospitalizations due to AF account for one-third of all admissions for cardiac arrhythmias. Acute coronary syndrome (ACS), aggravation of heart failure, thromboembolic complications, and acute arrhythmia management are the main causes. Cognitive dysfunction, including vascular dementia, may be related to AF. Small observational studies suggest that asymptomatic embolic events may contribute to cognitive dysfunction in AF patients in the absence of an overt stroke.

Quality of life and exercise capacity are impaired in patients with AF. Patients with AF have a significantly poorer quality of life compared with healthy controls, the general population, or patients with coronary heart disease in sinus rhythm. Left ventricular (LV) function is often impaired by the irregular, fast ventricular rate and by loss of atrial contractile function and increased end-diastolic LV filling pressure. Both rate control and maintenance of sinus rhythm can improve LV function in AF patients.

Ventricular arrhythmias include premature ventricular contraction, ventricular tachycardia, and ventricular fibrillation. Both of last are life-threatening arrhythmias most commonly associated with heart attacks. The most serious arrhythmia is ventricular fibrillation, which is an uncontrolled, irregular beat. If cardiopulmonary resuscitation (CPR) can be started, or if electrical energy is used to "shock" the heart back to a normal rhythm, then the heart may not be too damaged. About 220,000 deaths from heart attacks each year are thought to be caused by ventricular fibrillation. People who have heart disease or a history of heart attack have the highest risk of ventricular fibrillation.

A less serious type of ventricular arrhythmia is a premature ventricular contraction (PVC). As the name suggests, the condition happens when the ventricles contract too soon, out of sequence with the normal heartbeat. PVCs generally are not a cause for alarm and often do not need treatment. But if patient has heart disease or a history of ventricular tachycardia, PVCs can cause a more serious arrhythmia.

The incidence of ventricular tachycardia (VT) in the United States is not well quantified because of the clinical overlap of VT with ventricular fibrillation (VF). Examination of sudden death data provides a rough estimate of VT incidence. Most sudden cardiac deaths are caused by VT or VF, at an estimated rate of approximately 300,000 deaths per year in the United States, or about half of the

estimated cardiac mortality in this country. A prospective surveillance study gave a sudden death incidence of 53 per 100,000, accounting for 5.6% of all mortality. This is only a rough estimate of VT incidence, because many patients have nonfatal VT and because arrhythmic sudden deaths may be associated with VF or bradycardia rather than with VT. Ventricular tachycardia (VT) is observed more frequently in men, because ischaemic heart disease is more prevalent among men. In patients with ischaemic cardiomyopathy and nonsustained VT, sudden death mortality rates approach 30% in 2 years.

Place of carrying out: class-room, wards of the cardiology, department of functional diagnostics.

Study objective: to be able to verify supraventricular, ventricular extrasystole and extrasystole from AV-node, to distinguish fibrillation from atrium and ventricles, preexictation syndrome on ECG.

Basic level:

1. To be able to collect complaints, case history, carry out objective examination.
2. To be able to register ECG. ECG classification of the rhythm disorders.
3. Electrophysiology of the heart.
4. To interpret instrumental and laboratory data in patients with arrhythmias.
5. To interpret side effects of antiarrhythmic drugs. Preventive measures against arrhythmia development.

Student has to know:

1. Diseases which are accompanied by arrhythmias.
2. How to make algorithm of investigations in arrhythmias.
3. How to differentiate arrhythmias by clinical signs.

The main theoretical questions:

1. ECG classification of rhythm disorders.
2. Sinus tachycardia, sinus bradycardia. Clinical signs, ECG signs, management.
3. Extrasystoles, clinical signs, ECG signs, management.
4. Supraventricular tachycardias. ECG signs.
5. Management in paroxysmal tachycardias.
6. Ventricular paroxysmal tachycardias. ECG signs.
7. Management in paroxysmal ventricular tachycardias.
8. Atrial fibrillation and flutter. ECG signs. Management.
9. ECG signs of the preexcitation syndromes.
10. Ventricular arrhythmias related to specific pathology (MI, cardiomyopathy, valvular heart diseases). Device therapy.

Assignment for self-assessment

1. 34-year-old patient with sudden onset of fatigue, and palpitation addressed to the doctor's office. On ECG: presence of frequent and regular P waves and QRS complexes. What rhythm disorder has occured and what drug will you select for treatment?

2. A 52-year-old man arrived to the emergency room with irregular tachycardia, ventricular rate of 250/min, blood pressure of 80/60 mm Hg, and prolonged QRS complexes. It is known he has the Wolff-Parkinson-White syndrome. What medicines should be used for immediate management?

3. Negative P waves were registered on ECG in the II and III standard leads, QRS complexes are not changed and go after P waves. Pacemaker is located in:

a) sinus node;
b) AV node;
c) ventricles;
d) inferior part of the atrium;
e) everything is wrong.

Answers:

1. Paroxysmal superventricular tachycardia; verapamil will terminate over 90% of paroxysmal superventricular tachycardia.

2. Wolff-Parkinson-White syndrome is potentially life-threatening when the anomalous atrioventricular connection has a short refractory period and is capable of rapid atrioventricular con-

duction. Very rapid ventricular rates can produce cardiovascular collapse or precipitate ventricular fibrillation. Atrial fibrillation should be terminated immediately with cardioversion.

3. d.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology
12. https://www.ers-education.org/e-learning/procedure-videos/

4. MANAGEMENT OF PATIENS WITH BLOCKS

Time frame – 6 hours.

Professional motivation. The exact incidence of sinus node dysfunction (SND) is unknown. The syndrome occurs in approximately one in 600 cardiac patients older than 65 years. Symptoms of SND almost invariably progress over time. The most dramatic symptom in patients with SND is syncope. About 50% of patients with SND develop tachy-brady syndrome over a lifetime; such patients have higher risk of stroke and death. The survival of patients with SND appears to depend primarily on the severity of underlying cardiac disease and is not significantly changed by pacemaker therapy. However, incidence of sudden death owing directly to SND is extremely low.

No racial preponderance exists. Men and women are affected in equal numbers. SND may occur at any age but is primarily a disease of the elderly, with the average age being about 68 years old. SND in young patients is often related to underlying heart diseases.

Sick sinus syndrome describes abnormalities caused by the malfunction of the heart's natural pacemaker (sinoatrial node) when symptoms such as dizziness or fainting (syncope) are present. Possible complications of sick sinus syndrome include inadequate or inefficient pumping of the heart, heart failure, exercise intolerance, and injuries sustained by fainting spells and falling. Complications may develop from surgery to implant pacemakers, including infection, reaction to medications or anaesthesia, and pacemaker failure. Sick sinus syndrome progresses slowly. No treatment is necessary as long as the individual is not experiencing symptoms. Even with a permanent artificial pacemaker, the long-term prognosis is excellent.

AV blocks occur more frequently in people older than 70 years, especially in those who have structural heart disease. Approximately 5% of patients with heart disease have first-degree AV block, and about 2% have second-degree AV block.

The incidence of AV block increases with age. The incidence of third-degree AV block is highest in people older than 70 years (approximately 5–10% of patients with heart disease). A 60% female preponderance exists in congenital third-degree AV block. For acquired third-degree AV block, a 60% male preponderance exists. No racial proclivity exists in AV blocks.

One study examined first-degree AV block in 2123 patients: it was more prevalent in African-American patients in almost all decades of life (third through 10th). In both groups, first-degree AV block became more common at age 50 years and peaked in the 10th decade for black patients versus the ninth decade for white patients. One study examined 24-hour Holter monitors in 625 asymptomatic, heart-disease-free people, aged 15 to 83 years. Transient type I second-degree AV block was seen in 14 (2.2%) patients, more frequently in patients with resting heart rates of < 60 bpm. First-degree AV block has been associated with about a 2-fold increase in the probability of atrial fibrillation, a 3-fold increase in the probability of pacemaker implantation, and an increase in all-cause mortality.

First-degree AV block can be found in healthy adults. At 20 years of age, the PR interval may exceed 0.20 seconds in 0.5–2% of healthy people. At age 60 years, more than 5% of healthy individuals have PR intervals exceeding 0.20 seconds.

Advanced AV block (usually type II second-degree and third-degree) is usually anatomically infranodal and is seen in advanced His-Purkinje disease. One study examined the prevalence of His-Purkinje disease in the Framingham population. Here, QRS intervals of > 0.12 seconds were significantly associated with coronary heart disease, CHF, AV block, hypertension, left ventricular hypertrophy, and ventricular extrasystoles. QRS intervals > 0.12 seconds were rare before 50 to 60 years of age and were found in 11% of older men and 5% of older women. While intraventricular block does not inevitably lead to AV block, it frequently precedes the development of advanced AV block. Thus, this characterisation of a wide-QRS interval population is likely similar to that of the advanced AV block population.

Mobitz II second-degree AV block (Mobitz II) is rare in healthy individuals, whereas Mobitz I (Wenckebach) second-degree AV block is observed in 1–2% of healthy young people, especially during sleep.

Congenital third-degree AV block is rare, at 1 case per 20,000 births. This form of heart block, in the absence of major structural abnormalities, is associated with maternal antibodies to Ro (SS-A) and La (SS-B) and secondary to maternal lupus. It is most commonly diagnosed between 18 and 24 weeks' gestation and may be first, second, or third degree (complete). Mortality approaches approximately 20%; most surviving children require pacemakers.

Patients treated with permanent pacing to treat AV blocks have an excellent prognosis. Patients with advanced AV blocks who are not treated with permanent pacing remain at high risk of sudden cardiac death.

Although AV block generally is not associated with major morbidity, progressive degrees of AV block carry increasing morbidity and mortality.

The Reykjavik Study, a long-term prospective cardiovascular survey, which included a representative population of 9135 men and 9627 women, 33–79 years old, revealed that Right bundle branch block (RBBB) was found in 126 men and 67 women. The prevalence increased with age, from 0% among men and women 30–39 years of age to 4.1% and 1.6% in men and women, respectively, who were 75–79 years old. In men younger than 60 years RBBB had a significant relationship with hypertension, elevated fasting blood glucose, and increased heart size. In men with RBBB regardless of age, an association was found with cardiomegaly, ischaemic heart disease, arrhythmias, and bradycardia (P<005). A higher mortality from heart disease was found in men with RBBB compared to the control population. RBBB in women younger than 60 years is often associated with hypertension.

Place of carrying out: class-room, wards of the cardiology, department of functional diagnostics.

Study objective: to be able to verify different types of blocks, management of patients.

Basic level:

1. Anatomy and physiology of the conducting system.

2. To be able to collect complaints, case history, carry out an objective examination.
3. To be able to register ECG.
4. To interpret instrumental and laboratory data in patients with blocks.
5. To interpret side effects of drugs which are used in blocks.

Student has to know:

1. Diseases which are accompanied by blocks.
2. How to make algorithm of investigations in blocks.
3. Indications for pacemaker implantation: single-chamber and dual-chamber pacemaker.

The main theoretical questions:

1. Sinoatrial blocks. Causes, ECG signs. Treatment.
2. Intraatrial blocks, ECG signs.
3. Atrioventricular block. ECG signs. Treatment.
4. Adams-Stokes attacks. Diagnosis. Emergency.
5. Bundle branch blocks. Causes. Diagnosis. Classification: right bundle branch block. Block of the left branch of the bundle of His. Anterior/posterior fascicle of the left branch of His bundle.
6. Sinus node dysfunction. The sick sinus syndrome. Causes, ECG signs, treatment.
7. External defibrillation and cardioversion. Temporary/permanent pacemakers.

Assignment for self-assessment

1. A 63-year-old woman was admitted to the coronary care unit with palpitation, weakness and after short period of loss of consciousness. She indicates on impairment of state for about 4 months. Her pulse is 52 per min, arrhythmical, no murmur sounds. On ECG: sinus and irregular rhythm, PQ interval – 0.2 sec, QRS complexes – 0.08, gradual increase R-R interval with subsequent PQRST dropout. What is the reason of such state?

a) sinoatrial block;
b) AV-block, type I;
c) block of 3 fascicles of His bundle;
d) AV-block, type II.

2. The ECG examination of the 32-year-old male shows the PR interval of conducted beats is normal but some P waves are not conducted. What type of block is it? What is the management of this disorder?

3. A type of AV block characterised by progressive lengthening of the PR interval until the P wave fails to conduct is:

a) second degree AV-block: Mobiz type II;
b) second degree AV block: Mobiz type I;
c) first degree AV block;
d) third degree block.

4. Rhythms required permanent pacing in patients with cardiac disorders include:

a) supraventricular tachycardia;
b) second-degree AV block: Mobiz type I;
c) complete heart block;
d) Wolf-Parkinson-White syndrome.

Answers:

1. a.
2. Mobitz type II second degree AV block. A permanent pacemaker is indicated.
3. b. 4. c.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International

Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology
12. https://www.ers-education.org/e-learning/procedure-videos/

5. MANAGEMENT OF PATIENTS WITH STABLE ANGINA, CRITERIA FOR DIAGNOSIS

Time frame – 6 hours.

Professional motivation. Stable angina is the initial manifestation of ischaemic heart disease in approximately 50% of these patients. The prevalence of angina in community studies increases sharply with age in both sexes from 0.1–1% in women aged 45–54 to 10–15% in women aged 65–74 and from 2–5% in men aged 45–54 to 10–20% in men aged 65–74. Therefore, it can be estimated that in most European countries, 20,000–40,000 individuals of the population per million suffer from angina.

Statistics released by the American Heart Association in 2010 indicate that approximately 10.2 million people in the United States suffer from angina pectoris. In 2007 the overall death rate from coronary artery disease was 251.2 per 100,000 people, and coronary artery disease accounted for 33.6% of total deaths from all causes. Though the death rate from coronary artery disease decreased by 27.8% from 1997 to 2007, the over all burden of the disease remains high secondary to high prevalence of risk factors such as smoking, hypertension, diabetes, obesity. People in the Mediterranean region and Eskimos have a lower incidence of coronary artery disease due to higher consumption of canola oil and fish oil, respectively.

In the past, the incidence and prevalence of coronary artery disease were low in developing countries in comparison to developed countries. But with the westernization of developing regions in Middle East, India, and Central and South America, the incidence of CAD is increasing. The prevalence of coronary artery disease among adults in India has risen 4-fold over the last 40 years. By the year 2005, it was the leading cause of death accounting for 29% of the total deaths from all causes. The prevalence of coronary artery disease has been increasing in China with increase in risk factors such as smoking and mean cholesterol levels which have increased from 166 mg/dL to 206 mg/dL over the past 15 years.

According to the Framingham Heart Study, the lifetime risk of developing coronary artery disease at the age of 40 is 49% in men and 32% in women while the risk at 75 years of age is 35% in men and 24% in women. During 26 years of follow-up in the Framingham Heart Study, 80% of women under 75 years of age presented with angina pectoris rather than myocardial infraction. In contrast, only 20% of men presented with angina pectoris as their first manifestation.

Patients with stable angina are at risk of ACS developing: unstable angina, non-ST-elevation MI or ST-elevation MI. Data from the Framingham Heart Study showed that for men and women with an initial clinical presentation of stable angina, the 2-year incidence rates of nonfatal MI and CHD death

were 14.3 and 5.5% in men and 6.2 and 3.8% in women, respectively. More contemporary data regarding prognosis can be gleaned from clinical trials of antianginal therapy and/or revascularization, although these data are biased by the selected nature of the populations studied. From these, estimates for annual mortality rates range from 0.9–1.4% per annum, with an annual incidence of nonfatal MI between 0.5% (INVEST) and 2.6% (TIBET).

LV function is the strongest predictor of survival in patients with chronic stable coronary disease; the next most important factor is the distribution and severity of coronary stenosis. Left main (LM) disease, three-vessel disease, and the proximal involvement of the left anterior descending are common characteristics predicting a poor outcome and increase the risk of ischaemic events. Myocardial revascularization can reduce the risk of death in selected anatomical subgroups, reduce the number of ischaemic episodes (ACIP), and in some instances may improve the LV function in high-risk patients. However, disease progression and the occurrence of acute events may not necessarily be related to the severity of stenosis at coronary arteriography. In all patients, smaller lipid filled plaques are present in addition to those that cause severe stenoses.

The risk of acute events is related to the overall plaque burden and to plaque vulnerability. Although an area of great research interest, our capabilities to identify vulnerable plaque remain limited.

Place of carrying out: class-room, wards of the cardiology and rheumatology departments, department of functional diagnostics.

Study objective: to be able to determine extent of examinations to put final diagnosis and assign management.

Basic level:

1. To know classification and rick factors of the ischaemic heart disease.
2. To be able to collect complaints, case history, carry out objective examination.
3. To interpret instrumental (ECG, 24 hour ECG monitoring (Holter monitor), EchoCG) and laboratory data in patients with chest pain.
4. To identify signs from anamnesis and objective data inherent to stable angina.
5. To interpret side effects of antianginal agents.

Student has to be able to:

1. Examine patients with cardiovascular disorders.
2. Make an algorithm of stable angina management.

The main theoretical questions:

1. Classification of the angina pectoris (functional classes). Diagnostic criteria.
2. Stress and pharmacology testing, evidence for performing and interpretation.
3. Treatment and preventive measurements in stable angina.
4. Importance of ECG, 24 hour ECG monitoring and coronarography in silent form of ischaemia.

Assignment for self-assessment

1. A 53-year-old man complains of pressing chest pain on 100 m walking that lasts about 15 min. The examination reveals a regular heart rate with a reduced intensity S_1 and normal S_2. Blood pressure is 140/90 mm Hg; pulse is 90 beats/min, regular, respiratory rate is 20 breaths/min. His lungs are clear. The abdomen is soft without tenderness or distention. On ECG: sinus rhythm, high and sharp T waves in V2–V4 leads. During the last week pain appears at night and at rest, not stopped after taking 1 tab. of nitroglycerin. What disease can you think of?

a) progressive angina;
b) stable angina, FC4;
c) myocardial infarction;
d) vasospastic angina;
e) stable angina, FC3.

2. A 38-year-old man complains of angina attacks and dyspnoea on excersises. The patient suffers from obstructive form of hypertrophic cardiomyopathy. The examination reveals a regular heart rate. Blood pressure is 145/85 mm Hg; pulse is 80 beats/min. What medicine is contraindicated in this situation?

a) bisoprolol;

b) verapamil;
c) aspirin;
d) nitroglycerin.

3. What ECG sign is typical for myocardial ischaemia?
a) ST elevation less than 1 mm;
b) ST depression less than 1 mm;
c) ST depression more than 1 mm;
d) ST elevation more than 5 mm.

4. What test should be made in inefficiency of 24 hour ECG monitoring for revealing of painless form of ischaemia?
a) pharmacological test with propranolol;
b) pacemaker setting;
c) excersise test.

5. A 58-year-old man complains of dyspnoea on excersises and mild oedema on the legs. He has never complained of chest pain, never used nitrates and other medicines. There are no pathological changes of ST segment and T wave on ECG at rest; in excersise test there is ST segment depression more than 2 mm. What should be suspected?
a) painless form of myocardial ischaemia;
b) vasospastic angina;
c) lung pathology;
d) state of absolute health.

Answers: 1. a. 2. d. 3. c. 4. c. 5. a.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology
12. https://www.ers-education.org/e-learning/procedure-videos/

6. ISCHAEMIC HEART DISEASE. MANAGEMENT OF PATIENTS WITH UNSTABLE ANGINA, CRITERIA FOR DIAGNOSIS

Time frame – 6 hours.

Professional motivation. Unstable angina and non-ST segment elevation myocardial infarction account for about 2.5 million hospital admissions worldwide and are a major cause of mortality and morbidity in Western countries. In the United States, the incidence of unstable angina is increasing, and nearly 1 million hospitalized patients each year have a primary diagnosis of unstable angina. A similar number of unstable angina episodes likely occur outside the hospital and are unrecognized or managed in the outpatient setting. With heightened public awareness, improved survival after myocardial infarction, and aging of the population, this number should continue to rise despite primary and secondary prevention measures.

Disparities in outcomes and the prevalence of risk factors among different ethnic groups have been widely reported. For instance, as a group, blacks exhibit a higher prevalence of atherosclerotic risk factors (e.g., hypertension, diabetes mellitus, smoking), greater left ventricular mass, and decreased peripheral vasodilatory response. Relative to whites, myocardial infarction more frequently results in death in blacks at young ages.

Fewer myocardial events but more cerebral complications have been observed in black patients with unstable angina in randomized clinical trials of heparin versus hirudin (the Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries II [GUSTO II] trial) or eptifibatide versus placebo (the Platelet Glycoprotein IIb/IIIa in Unstable Angina: Receptor Suppression Using Integrilin Therapy [PURSUIT] trial). This may be due to the enhanced fibrinolytic activity and higher prevalence of hypertension in this population.

Women with unstable angina are older and have a higher prevalence of hypertension, diabetes mellitus, congestive heart failure, and family history of coronary artery disease than men. Men tend to have a higher previous incidence of myocardial infarction and revascularization, a higher proportion of positive cardiac enzymes on admission, and higher rates of catheterization and revascularization. However, outcome is related more to the severity of the illness than to sex.

The mean age of presentation with unstable angina is 62 years, with an age range of 23–100 years. To put this in perspective, the mean age is 60 years for patients in clinical trials for myocardial infarction, about 67 years for carotid artery stenosis, and 63 years for congestive heart failure. On average, women with unstable angina are 5 years older than men on presentation, with approximately half of women older than 65 years, as opposed to only about a third of men. Blacks tend to present at a slightly younger age than do people of other races.

The prognosis is substantially worse than for chronic stable angina. In-hospital death and re-infarction affect 5–10%. Despite optimal treatment with anti-ischaemic and antithrombotic drugs, death and recurrent myocardial infarction occur in another 5–10% of patients in the month after an acute episode. Several studies indicate that these patients may have a higher long term risk of death and myocardial infarction than do patients with ST segment elevation.

Patients who present with new ST-segment deviation (1 mm or greater) have a 1-year death or myocardial infarction rate of 11%, compared with a rate of only 6.8% in patients with isolated T-wave inversion.

Place of carrying out: class-room, wards of the cardiology departments, ward of the emergency.

Study objective is to prove diagnosis of unstable angina pectoris, to make differential diagnosis and determine management of patient with unstable angina.

Basic level:

1. Pathogenesis of unstable angina pectoris.
2. Critertia for the diagnosis and differential diagnosis of angina pectoris.
3. Differentiation between coronary cardialgia and noncoronary cardialgia.
4. Primary and secondary prevention of angina pectoris.

Student has to be able to:

1. Find out clinical and laboratory symptoms for stable and unstable angina pectoris and to group them into syndromes.
2. Make clinical diagnosis.
3. Indicate differential programs of the treatment.

The main theoretical questions:

1. Pathogenesis and clinical course of unstable angina pectoris.
2. Standards of unstable angina treatment.
3. Coronarography and 24 hour ECG monitoring for unstable angina diagnosing.
4. Preventive measurements in unstable angina pectoris.

Assignment for self-assessment

1. For several months a 49-year-old physician has had recurrent episodes of severe retrosternal chest pressure; these episodes occur four to five mornings a week and last 15 to 20 minutes. Despite therapy with large dosages of a calcium channel blocking agent these episodes are proceeding. Three months ago a coronary arteriogram showed no fixed obstructions in the coronary arteries, but spasm of the midportion of the right coronary artery occurred during the study and was associated with pain and ST-segment elevation. What is the most appropriate next step?

2. In appearing of acute epigastrical pain or retrosternal burning in middle-aged men investigations should be started from:

a) oesophagogastroscopy;
b) roentgenoscopy of gastrointestinal tract;
c) ECG.

3. A 58-year-old man complains of pressing retrosternal pain with radiation to scapula which goes away in 30 min after taking nitroglycerin under the tongue. Such pain appears 1–2 times per month in the morning. The examination reveals a regular heart rate. Blood pressure is 145/85 mm Hg; pulse is 80 beats/min. In Holter monitoring during attack of pain ST elevation on 5 mm was revealed in V2–V5. On the next day ST was on isoline. What pathology does the patient have?

a) stable angina, FC4;
b) myocardial infarction;
c) acute coronary insufficiency;
d) unstable angina.

4. A 50-year-old man complains of attacks of pressing retrosternal pain up to 3 events successively which appear at early in the morning (about 4–5 a.m.) and go away in 5 min after taking a nitroglycerin under the tongue. The patient is tolerant to physical activity during the daytime. The examination reveals a regular heart rate, normal heart borders. Blood pressure is 120/80 mm Hg; pulse is 76 beats/min. On ECG during attack of pain ST 3 mm elevation was revealed in the I, II, aVL, V4–V6; after attack ECG was normalized. In stress test (exercise test) there were no changes on ECG. What pathology does the patient have?

a) stable angina;
b) vasospastic angina;
c) angina at rest;
d) unstable angina.

Answers:

1. Many reports indicate that nitrates are effective in treatment of patients with coronary arteries spasm, and nitrates should be used in conjunction with calcium-channel blocking agents (calcium antagonist). If the patient described to be symptomatic despite of the usage of adequate dosages of nitrates and calcium antagonist, it would be appropriate to consider either adding the second calcium antagonist that may have a different mechanism of action, or switching to a different calcium antagonist.

2. c. 3. d. 4. b.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.

4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology
12. https://www.ers-education.org/e-learning/procedure-videos/

7. MANAGEMENT OF PATIENTS WITH CARDIOMEGALY (MYOCARDITIS, PERICARDITIS AND CARDIOMYOPATHY). INSTRUMENTAL METHODS FOR DIAGNOSIS, MODERN APPROACHES TO TREATMENT

Time frame – 6 hours.

Professional motivation. The term "cardiomegaly" most commonly refers to an enlarged heart seen on chest X-ray before other tests are performed to diagnose the specific condition causing cardiomegaly. While having an enlarged heart may not always be preventable, it's usually treatable. Treatment for enlarged heart is aimed at correcting the underlying cause. Treatment for an enlarged heart can include medications, medical procedures or surgery.

The diagnosis of myocarditis is diffcult. The first step in diagnosis is to suspect myocarditis. The primary principles of treatment are to make the clinical diagnosis and manage cardiopulmonary emergency promptly. Every effort must be made to confirm the diagnosis of myocarditis by histology, since some cases of specific myocarditis may respond to corticosteroid treatment. In the acute phase, myocarditis management of cardiac pump failure and potentially fatal arrhythmias is the main clinical challenge. The prognosis of myocarditis varies depending on the pathogenesis and type of disease.

Hypertrophic cardiomyopathy (HCM) is a complex and relatively common genetic cardiac disorder (about 1:500 in the general adult population) that has been the subject of intense scrutiny and investigation for over 40 years. Hypertrophic cardiomyopathy affects men and women equally and occurs in many races and countries, although it appears to be under-diagnosed in women, minorities, and under-served populations. Hypertrophic cardiomyopathy is a particularly common cause of sudden cardiac death (SCD) in young people (including trained athletes) and may cause death and disability in patients of all ages, although it is also frequently compatible with normal longevity. Because of its heterogeneous clinical course and expression, HCM frequently presents uncertainty and represents a management dilemma to cardiovascular specialists and other practitioners, particularly those infrequently engaged in the evaluation of patients with this disease.

Dilated cardiomyopathy is much more common than the other major forms of cardiomyopathy (hypertrophic, restrictive, and arrhythmogenic right ventricular cardiomyopathy). The estimated prevalence of dilated cardiomyopathy is 1:2500. This condition is among the most common causes of heart failure. The incidence of dilated cardiomyopathy discovered at autopsy is estimated to be 4.5 cases per 100,000 population per year, whereas the clinical incidence is 2.45 cases per 100,000 population per year. Dilated cardiomyopathy may manifest clinically at a wide range of ages, but this condition most commonly occurs in the third or fourth decade of life. Dilated cardiomyopathy is associated with a survival rate of less than 50% at 10 years. With better supportive care, however, improved 5- and 10-year survival rates have been reported. Peripartum cardiomyopathy may be

reversible in up to 50% of patients but often recurs with subsequent pregnancy.

There is a negative association in dilated cardiomyopathy between survival and frequent ventricular tachyarrhythmias that require antiarrhythmic treatment or automated implantable cardioverter-defibrillator (AICD) placement. During the clinical course of idiopathic dilated cardiomyopathy, a number of clinical and diagnostic measures may be monitored to predict prognosis. The most important and best predictors are the New York Heart Association heart failure functional class, the left ventricular ejection fraction, and the peak oxygen consumption.

Acute pericarditis is more common in adults (typically between 20 to 50 years old) and in men. Epidemiologic data on the incidence of acute pericarditis are lacking, likely because this condition is frequently inapparent clinically, despite its presence in numerous disorders and there are a large number of undiagnosed cases. Lorell noted a diagnosis of acute pericarditis in approximately 1 per 1000 hospital admissions. However, it may account for up to 5% of presentations to emergency departments for chest pain and up to 0.1% of hospital admissions. In addition, acute pericarditis comprises 1% of emergency room visits in patients with ST-segment elevation. In fact, the reported incidence of acute pericardial tamponade is approximately 2% of penetrating trauma; however, this condition is rarely seen in blunt chest trauma. Uremic pericarditis may occur in 6–10% of patients with advanced renal failure before initiation of dialysis. When patients with large effusions are examined, uraemia may account for up to 20% of cases in some series. The widespread availability of dialysis has reduced the incidence of uremic pericarditis.

Pericarditis can range from mild cases that get better on their own to life-threatening cases. The condition can be complicated by significant fluid buildup around the heart and poor heart function. The outcome is good if the disorder is treated promptly. Most people recover in 2 weeks to 3 months. However, pericarditis may come back. Malignant disease is the most common cause of pericardial effusion with tamponade in developed countries. However, tuberculosis should be considered in endemic areas.

Place of carrying out: class-room, wards of the rheumatological department.

Study objective: to improve the skills in determination of the doctors' tactics in cardiomegaly; to develop differential diagnosis of the cardiomegaly caused by myocarditis, pericarditis, and cardiomyopathy; to determine differential approaches to their treatment.

Basic level:

1. To be able to collect complaints, case history, carry out objective examination.
2. To interpret instrumental (ECG, EchoCG, X-ray) and laboratory data in patients with cardiomegaly.
3. To identify signs from anamnesis and objective data inherent to cardiomegaly of different origin.
4. To interpret side effects of agents which are used in cardiomegaly.

Student has to be able to:

1. Find out cardiomegaly using instrumental methods of examination.
2. Make an algorithm of investigations in patients with cardiomegaly.
3. Determine approaches to treatment in different aetiology of cardiomegaly.
4. Differentiate cardiomegaly of various origin.

The main theoretical questions:

1. Definition of the cardiomegaly syndrome.
2. The main causes of cardiomegaly.
3. Approaches for differential diagnosis of cardiomegaly causes.
4. Modern aspects of myocarditis aetiology and pathogenesis. Classification of myocarditis.
5. Criteria for diagnosis of myocarditis. Treatment of myocarditis.
6. Aetiology and pathogenesis of pericarditis. Classification of pericarditis.
7. Criteria for diagnosis and treatment of pericarditis.
8. Classification of cardiomyopathies.
9. Modern aspects of the dilated cardiomyopathy aetiology and pathogenesis.
10. Criteria for diagnosis and management of patients with dilated cardiomyopathy.
11. Modern aspects of hypertrophic cardiomyopathy aetiology and pathogenesis.

12. Classification and treatment of hypertrophic cardiomyopathy.
13. Modern aspects of the restrictive cardiomyopathy aetiology and pathogenesis.
14. Classification and treatment of the restrictive cardiomyopathy.
15. Clinical and ECG signs of arrhythmic and tromboembolic complications.

Assignment for self-assessment

1. What disorders is chronic constrictive pericarditis associated with?
2. What idiopathic cardiomyopathy is related to heredity?
   a) dilated;
   b) hypertrophic;
   c) restrictive;
   d) arrhythmogenic right ventricular dysplasia.
3. What idiopathic cardiomyopathy is accompanied by disturbances of diastolic cardiac function?
   a) dilated;
   b) hypertrophic;
   c) restrictive;
   d) arrhythmogenic right ventricular dysplasia;
   e) hypertrophic, restrictive.
4. What idiopathic cardiomyopathy is accompanied by disturbances of systolic cardiac function?
   a) dilated;
   b) hypertrophic;
   c) restrictive;
   d) arrhythmogenic right ventricular dysplasia.
5. What cause of metabolic cardiomyopathy is the most frequent?
   a) uraemia;
   b) podagra;
   c) electrolytes deficit;
   d) endocrine pathology.
6. A 58-year-old man complains of dyspnoea, weakness, intermission of pulse, oedema on the legs. The examination reveals enlargement of cardiac size in percussion. Blood pressure is 130/80 mm Hg; pulse is 90 beats/min, irregular, respiratory rate is 20 breaths/min. On ECG: tachysystolic variant of atrial fibrilation. On cardiac ultrasound: enlargement of cardiac chambers, EF is 36%. What disease does this patient have?
   a) dilated cardiomyopathy;
   b) hypertrophic cardiomyopathy;
   c) myocarditis;
   d) myocardiodystrophy.

Answers:

1. Rheumatoid arthritis and radiotherapy have been recognized for some years as occasional causes of constrictive pericarditis. Recently, cardiac surgery (usually coronary bypass surgery), including insertion of epicardial pacemaker, has also been associated with later development of constriction.

2. b. 3. e. 4. a. 5. d. 6. a.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.

5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology
12. https://www.ers-education.org/e-learning/procedure-videos/

8. CHRONIC HEART FAILURE (CHF). CLASSIFICATION, CLINICAL FEATURES, MANAGEMENT

Time frame – 6 hours.

Professional motivation. The European Society of Cardiology (ESC) represents countries with a population of >900 million, and there are at least 15 million patients with HF in those 51 countries. The prevalence of asymptomatic ventricular dysfunction is similar, so that HF or asymptomatic ventricular dysfunction is evident in ~4% of the population. The prevalence of HF is between 2 and 3% and rises sharply at ~75 years of age, so the prevalence in 70- to 80-year-old people is between 10 and 20%. In younger age groups HF is more common in men because the most common cause, coronary heart disease, occurs in earlier decades. In the elderly, the prevalence is equal between the sexes. The overall prevalence of HF is increasing because of the aging of the population, the success in prolonging survival in patients suffering coronary events, and the success in postponing coronary events by effective prevention in those at high risk or those who have already survived a first event (secondary prevention). In some countries the age-adjusted mortality from HF is falling at least in part due to modern treatment.

The mean age of patients with HF in the community in developed countries is 75 years. HF with preserved ejection fraction (HFPEF) is more common in the elderly, women, and those with hypertension or diabetes. HF is the cause of 5% of acute hospital admissions, is present in 10% of patients in hospital beds, and accounts for ~2% of national expenditure on health, mostly due to the cost of hospital admissions. Substantial under-reporting is probably due to clinicians' preference for aetiological diagnoses (e.g., aortic stenosis) or the diagnosis of a major comorbidity (e.g., diabetes). Overall 50% of patients are dead in 4 years. 40% of patients admitted to hospital with HF are dead or readmitted within 1 year. Studies show that the accuracy of diagnosis of HF by clinical means alone is often inadequate, particularly in women, the elderly, and the obese. HFPEF (EF >45–50%) is present in half the patients with HF. The prognosis in more recent studies has been shown to be essentially similar to that of systolic HF.

The impact on prognosis of specific treatments in individual patients with HF is often difficult to predict. Conditions associated with a poor prognosis in heart failure are advanced age, hypotension, NYHA functional class III–IV, wide QRS, marked elevation of BNP/NT pro-BNP, hyponatraemia, low LVEF, etc.

Place of carrying out: class-room, wards of the cardiology department.

Study objective is to verify diagnosis of chronic heart failure, to determine management of patients with CHF.

Basic level:

1. To be able to capture complaints, case history, carry out objective examination.
2. To interpret instrumental and laboratory data in patients with CHF.

3. To discover signs inherent in CHF.
4. To interpret side effects of drugs which are used in CHF.

Student has to know:

1. How to examine patients with cardiovascular disorders.
2. Causes of heart failure, functional classes.
3. Criteria for diagnosis of heart failure.
4. How to make program for investigation of patients with CHF.
5. Clinical pharmacology of diuretics, vasodilators, beta-adrenergic receptor blocking agents, digitalis glycosides, angiotensin-converting enzyme inhibitors (ACE); drugs, which improve heart metabolism.

The main theoretical questions:

1. Heart failure classification. Clinical manifestation of heart failure.
2. Diagnostic tests in heart failure.
3. Primary and secondary prophylaxis of heart failure. Prognosis in heart failure.
4. Principles of heart failure management.
5. Therapeutic strategies in systolic and dyastolic dysfunction: vasodilators; ACE; angiotensin II receptor antagonists; nitrates; adrenergic receptor antagonists (alpha-adrenergic receptor antagonists; beta-adrenergic receptor antagonists), calcium channel blockers; digitalis glycosides; diuretics; inotropic agents; phosphodiesterase inhibitors. Indications, contrindications, side effects.
6. Treatment of diastolic heart failure.
7. Surgical treatment of patients with heart failure (intra-aortic balloon counterpulsation, heart transplantation, cardioplasty). Impact of cardiac resynchronization therapy.

Assignment for self-assessment

1. A 39-year-old woman complains of dyspnoea on exertion, chest pain, palpitation. She had previous rheumatic fever twenty years ago. She was treated with acute pharyngitis two weeks ago. Physical examination reveals a low-pitched, rough, and rasping pansystolic murmur, loudest at the base of the heart in the second right intercostal space, weak and regular pulse, HR is 88 beats/min, BP is 150/90 mm Hg. What diagnosis do you suspect? Prescribe the treatment.
2. Initial dose for enalapril in CHFis:
    a) 2.5 mg once a day;
    b) 15 mg bid;
    c) 5 mg bid;
    d) 10 mg bid.
3. Cardiac glycosides have:
    a) sympathicotonic;
    b) vagotonic;
    c) vagolytic;
    d) vagotonic and sympathicoinhibiting.

Answers:

1. Chronic rheumatic cardiac disease. Aortic stenosis. Chronic heart failure II A. Carvedilol 12.5 mg/daily, lisinopril 2.5 mg/daily, nitrosorbid 10 mg every 8 hours, aspirin 100 mg daily, verospiron 25 mg daily, extencillin 2.4 mln units im once in 3weeks.
2. a. 3. d.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P.

Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology
12. https://www.ers-education.org/e-learning/procedure-videos/

9. MANAGEMENT OF PATIENTS WITH CARDIAC MURMURS IN ACQUIRED VALVULAR DISEASES

Time frame – 6 hours.

Professional motivation. Rheumatic fever (RF) and rheumatic heart disease (RHD) remain significant causes of cardiovascular diseases in the world today. Despite a documented decrease in the incidence of acute RF and a similar documented decrease in the prevalence of RHD in industrialized countries during the past 5 decades, this nonsuppurative cardiovascular sequel of group A streptococcal pharyngitis remain medical and public health problems in both industrialized and industrializing countries even at the beginning of the 21st century. The most devastating effects are on children and young adults in their most productive years.

Valvular heart disease is the most serious complication of acute rheumatic fever and infectious endocarditis. Rheumatic fever and infectious endocarditis are widespread in all climatic regions of the world. Last decades data convincingly showed the relationship between incidence of these diseases and economic situation of the country and its preferred expansion in poorly advanced and developing countries. Nowadays the disease is characterized by progressing course with often multiple valvular deformities, lung hypertension, early mortality under worsening heart failure. Rheumatic mitral stenosis in young population is now considered to be rare in developed countries with an incidence of less than 1 per 100,000 population and relates to the decline of rheumatic fever. A similar condition occasionally occurs in association with other diseases such as systemic lupus or infective endocarditis. Consequently, we should remain vigilant and be aware of the presenting symptoms and signs of rheumatic fever and rheumatic heart disease. In asymptomatic patients with mitral stenosis, survival was good up to 10 years, progression being highly variable with sudden deterioration, precipitated by complications, such as atrial fibrillation or embolism, in half of the patients. Symptomatic patients have a poor prognosis.

Patients with acute aortic regurgitation (AR) have a poor prognosis without intervention owing to the significant increase in diastolic LV pressure, leading to poor haemodynamic tolerance. There is little information in the literature on the progression from mild to severe AR. Patients with severe AR and symptoms have a poor prognosis. In asymptomatic patients with severe AR and normal LV function, the number of events during follow-up is low: development of asymptomatic LV dysfunction, <1.3% per year; sudden death, <0.2% per year; and symptoms, LV impairment, or death, 4.3% per year. Age, end-systolic diameter or volume, and EF at rest are predictors of outcome. On multivariate analysis, age and end-systolic diameter, when it is >50 mm, predict a poor outcome.

Aortic stenosis (AS) has become the most frequent type of VHD in Europe and North America. It primarily presents as calcific AS in adults of advanced age (2–7% of the population >65 years). The second most frequent aetiology, which dominates in the younger age group, is congenital, whereas

rheumatic AS has become rare. Calcific AS is a chronic progressive disease. During a long latent period, patients remain asymptomatic. However, it should be emphasized that duration of the asymptomatic phase varies widely among individuals. Sudden cardiac death is a frequent cause of death in symptomatic patients but appears to be rare in the asymptomatic (≤1% per year). Predictors of the progression of AS and, therefore, of poor outcome in asymptomatic patients have recently been identified. They are: 1) clinical (older age, presence of atherosclerotic risk factors); 2) echocardiography (valve calcification, peak aortic jet velocity, LVEF, haemodynamic progression, and increase in gradient with exercise); the combination of a markedly calcified valve with a rapid increase in velocity of ≥0.3 m/s within 1 year has been shown to identify a high-risk group of patients (~80% death or requirement of surgery within 2 years); 3) exercise testing: symptom development on exercise testing in physically active patients, particularly those younger than 70 years, predicts a very high likelihood of symptom development within 12 months.

As soon as symptoms occur, the prognosis is dismal and mortality has been reported to be quite significant even within months of symptom onset, which is often not promptly reported by patients.

Organic mitral regurgitation (MR) covers all aetiologies in which leaflet abnormality is the primary cause of the disease, in opposition to ischaemic and functional MR, in which MR is the consequence of LV disease.

Reduced prevalence of rheumatic fever and increased life span in industrialized countries have progressively changed the distribution of aetiologies. Degenerative MR is the most common aetiology in Europe, whereas ischaemic and functional MR are increasingly frequent. Acute MR is poorly tolerated and carries a poor prognosis in the absence of intervention. In asymptomatic MR, the estimated 5 year rates (±standard error) of death from any cause, death from cardiac causes, and cardiac events (death from cardiac causes, heart failure, or new AF) with medical management were 22±3, 14±3, and 33±3%, respectively. In addition to symptoms, age, atrial fibrillation, degree of MR (particularly ERO), left atrial dilatation, LV dilatation, and low LVEF are all predictors of poor outcome.

Place of carrying out: class-room, wards of the cardiology department, wards of the emergency, department of functional diagnostics.

Study objective: to improve students' skills to make differential diagnosis of systolic and diastolic murmurs, to make diagnosis, to manage patients with valvular disorders.

Basic level:

1. Pathophysiology of systolic and diastolic murmurs.
2. To be able to collect complaints, case history, carry out objective examination.
3. To do percussion and auscultation of the patients for diagnosis of heart murmurs.
4. To interpret instrumental (ECG, EchoCG, X-ray) and laboratory data in patients with valvular disorders.

Student has to know:

1. Differential diagnosis of the systolic murmur in the acquired and congenital valvular diseases.
2. Differential diagnosis of the diastolic murmur in the acquired and congenital valvular diseases.
3. ECG- and Echo findings in the acquired and congenital valvular diseases.
4. X-ray examination in the acquired and congenital valvular diseases.
5. Indications for surgical treatment of the acquired and congenital valvular diseases.

The main theoretical questions:

1. Criteria for diagnosis of aortic stenosis, treatment.
2. Criteria for diagnosis of aortic regurgitation, treatment.
3. Criteria for diagnosis of mitral stenosis, stages of mitral stenosis and choice of treatment depending on stage.
4. Criteria for diagnosis of mitral regurgitation, stages of mitral regurgitation and choice of treatment depending on stage.
5. To make plan of investigations for patient with valvular diseases.
6. Indications for surgical intervention. Valve prosthesis: influence on haemodynamic, signs of dysfunction.

7. Management of pregnant women with valvular disorders.

Assignment for self-assessment

1. A 40-year-old woman presented with palpitation and dyspnoea which increases progressively during the past 3 years. There was chorea and arteritis in childhood. Physical examination: acrocyanosis, PS is 104, irregular, pulse deficit is 22 per minute, a severe systolic murmur and moderate presystolic are heard above heart apex with radiation, the I sound is reduced. On ECG: left ventricular hypertrophy, atrial fibrillation. What is the clinical diagnosis?

2. A 56-year-old man complains of fatigue, dyspnoea on exertion, and palpitations. He has had a murmur since childhood, frequent respiratory infections. Examination reveals intensified right-ventricle beat, a lift at the left sternal border, fixed splitting of S_2, systolic ejection murmur in the pulmonary area (II to IV). Chest X-ray shows right ventricular enlargement and prominent pulmonary arteries. ECG demonstrates atrial fibrillation with a right bundle-branch block. What is the most likely diagnosis?

3. What medicines must be used to decrease pulmonary hypertension?
   a) diuretics and nitrates;
   b) cardiac glycozides;
   c) anticoagulants;
   d) antiarrhythmic drugs;
   e) antibiotics.

4. At what stages of mitral stenosis surgical treatment isn't indicated?
   a) at the I stage;
   b) at the I–II stages;
   c) at the IV–V stages;
   d) at the V stage;
   e) at the I and V stage.

5. At what stages of mitral regurgitation surgical treatment isn't indicated?
   a) at recurrent systemic embolisms despite of anticoagulant treatment;
   b) at the I–II and V stages;
   c) at moderate heart failure with atrial fibrillation, decreased EF or dilated cardiac chambers;
   d) at significant CHF (FC III–IV);
   e) at the I and V stage.

6. What medicines are used for asymptomatic patients with aortic stenosis?
   a) diuretics;
   b) cardiac glycosides;
   c) anticoagulants;
   d) vasodilators;
   e) antibiotics (for prevention of infective endocarditis).

7. A 33-year-old male was seen in the clinic with one year history of worsening exertional dyspnoea and orthopnoea. He had no recollection of any previous symptoms of rheumatic fever. No other past medical illness of note apart from mild asthma.

On clinical examination, he was slim built with blood pressure of 120/80 and a regular pulse. No evidence of peripheral oedema was present. Jugular venous pressure was not raised. Cardiovascular examination revealed a loud first heart sound, opening snap and mid diastolic murmur with presystolic accentuation. Electrocardiograph demonstrated sinus rhythm with right bundle branch block along with evidence of left atrial enlargement.

Transthoracic and transoesophageal echocardiography confirmed severe rheumatic mitral stenosis with thickening and fusion of the commissures, mitral valve area of 0.7–0.9 cm^2, mild mitral regurgitation and left atrial dilatation. There was also marked pulmonary hypertension with a systolic pulmonary artery pressure estimated to be 70 mm Hg.

The clinical and biochemical markers: RBC – $4.0*10^{-12}$/l; Hb – 146 g/l; ESR – 4 mm/h; WBC – $6*10^{-9}$/l; eos. – 2%, stab neutr. – 4%, segmented neutrophils – 59%, lymphocytes – 11%, monocytes – 5%. Total serum protein – 65 g/l, serum urea – 6.7 mmol/l, creatinine – 90 μmol/l, bilirubin –

19 μmol/l, AST – 10 units, fasting plasma glucose – 4.8 mmol/l, rheumatoid factor – 1:32, uric acid – 0.25 mmol/l.

Urinalysis: RBC – 1 to 2 per high-power field, WBC – 3 to 2 per high-power field, specific gravity – 1028. Urinary protein excretion is 0.15 g/day.

Suggest management for such patient.

Answers:

1. Combined mitral valve disorder with predominance of regurgitation.

2. Atrial septal defect. The murmur heard in childhood is often considered "innocent", and symptoms do not appear until adulthood. A left-to-right shunt of blood between the atria causes right ventricular overload and increased pulmonary circulation. These result in the classic findings of a pulmonic systolic ejection murmur, late pulmonic valve closure with wide splitting of S2, and tricuspid flow murmur. Chest X-ray has signs of cardiomegaly and pulmonary overcirculation. Characteristic ECG changes are atrial fibrillation and an incomplete or complete right bundle-branch block.

3. a. 4. e. 5. b. 6. e.

7. Percutaneous balloon mitral valvuloplasty. Interesting feature to note in this case is the marked degree of pulmonary hypertension secondary to mitral stenosis, which is particularly uncommon in this age group, as it has been estimated that progression from mild to severe disabling symptoms usually takes up to 10 years.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology
12. https://www.ers-education.org/e-learning/procedure-videos/

Semantic module 2. Management of patients in rheumatology clinic

10. MANAGEMENT OF PATIENS WITH ARTICULAR SYNDROME

Time frame – 6 hours.

Professional motivation. Arthritis comprises more than 100 different rheumatic diseases and conditions, the most common of which is osteoarthritis. Other frequently occurring forms of arthritis include rheumatoid arthritis, lupus, fibromyalgia, and gout. Common symptoms include pain, aching, stiffness, and swelling in or around the joints. Some forms of arthritis, such as rheumatoid arthritis and

lupus, can affect multiple organs and cause widespread symptoms. Although arthritis is more common among adults aged 65 years or older, people of all ages (including children) can be affected. Nearly two-thirds of people with arthritis are younger than age 65 years. Arthritis is more common among women (24.3%) than men (18.7%) in every age group, and it affects members of all racial and ethnic groups. Arthritis also is more common among adults who are obese than among those who are normal weight or underweight.

Arthritis is the nation's most common cause of disability. Nearly 21 million U.S. adults report activity limitations because of arthritis each year. Among all U.S. adults of working age (18–64 years), 5.3% (6.9 million people) reported that they have arthritis that limits their work. A recent community study estimated that the lifetime risk of developing knee osteoarthritis serious enough to cause painful symptoms is 45%. Risk increases to 57% among people with a past knee injury. Lifetime risk for knee osteoarthritis goes up with increasing weight and rises to 60% among people who are obese.

Many people with rheumatoid arthritis have difficulty carrying out normal activities of daily living, such as standing, walking, dressing, washing, using the toilet, preparing food, and carrying out household chores. The symptoms of rheumatoid arthritis interfere with work for many people. As many as half of those with rheumatoid arthritis are no longer able to work 10–20 years after their condition is diagnosed.

On average, life expectancy is somewhat shorter for people with rheumatoid arthritis than for the general population. This does not mean that everyone with rheumatoid arthritis has a shortened life span. Rheumatoid arthritis itself is not a fatal disease. However, it can be associated with many complications and treatment-related side effects that can contribute to premature death. Although rheumatoid arthritis most often affects the joints, it is a disease of the entire body. It can affect many organs and body systems besides the joints. Therefore, rheumatoid arthritis is referred to as a systemic disease.

Worldwide, about 1% of people are believed to have rheumatoid arthritis. About 75% of these are women. Women are two to three times more likely to develop rheumatoid arthritis than men. Rheumatoid arthritis affects all ages, races, and social and ethnic groups. It is most likely to strike people 35–50 years of age, but it can occur in children, teenagers, and elderly people. Rheumatoid arthritis affects about 5–6% of some Native-American groups, while the rate is very low in some Caribbean peoples of African descent. The rate is about 2–3% in people who have a close relative with rheumatoid arthritis, such as a parent, brother or sister, or child. Although there is no cure for rheumatoid arthritis, the disease can be controlled in most people. Early, aggressive therapy, soon after the initial diagnosis, is optimally targeted to stop or slow down inflammation in the joints can prevent or reduce symptoms, prevent or reduce joint destruction and deformity, and prevent or lessen disability and other complications.

Outcomes are also highly variable. Some people have a relatively mild condition, with little disability or loss of function. Others at the opposite end of the spectrum experience severe disability due to pain and loss of function. Disease that remains persistently active for more than a year is likely to lead to joint deformities and disability. Approximately 40% of people have some degree of disability 10 years after their diagnosis. For most, rheumatoid arthritis is a chronic progressive illness, but about 5–10% of people experience remission without treatment. This is uncommon, however, after the first three to six months.

The early use of the disease modifying antirheumatic drugs (DMARDs) and biologic response modifiers in rheumatoid arthritis has resulted in patients experiencing more profound relief of symptoms and less joint damage and less disability over time.

Overall, the rate of premature death is higher in people with rheumatoid arthritis than in the general population. The most common causes of premature death in people with rheumatoid arthritis are infection, vasculitis, and poor nutrition. Fortunately, the manifestations of severe, long-standing disease, such as nodules, vasculitis, and deforming are becoming less common with optimal treatments.

Reactive arthritis (ReA) typically follows a limited course, where symptoms subsiding in 3–12 months. However, the condition has a tendency to recur. About 15–20% of people with ReA

develop a chronic, and sometimes severe, arthritis or spondylitis. This group of diseases primarily affects the spine (spondylo) and other joints. The complete medical term for group of diseases (ankylosing spondylitis, reactive arthritis (formerly Reiter's syndrome), psoriatic arthritis, Juvenile SpA, enteropathic arthritis (spondylitis/arthritis associated with inflammatory bowel disease), and undifferentiated SpA) is the "seronegative" spondyloarthritides. "Sero" refers to blood (blood serum) and "negative" indicates that there is usually no rheumatoid factor present in the blood.

Most types of SpA begin around the ages of 15–45. Men are more likely to get SpA. Psoriatic arthritis, which affects men and women equally, is the exception.

Most people with spondylitis lead long and productive lives. Certain complications, however, can lead to disability. It is important to be on the lookout for signs and symptoms of the more serious complications.

Up to 30% of people with psoriasis also develop psoriatic arthritis. In most cases psoriasis will precede arthritis, sometimes by many years. When arthritis symptoms occur with psoriasis, it is called psoriatic arthritis (PsA). About 20% of people who develop PsA will eventually have spinal involvement, which is called "psoriatic spondylitis". The inflammation in the spine can lead to complete fusion – as in ankylosing spondylitis – or skip areas where, for example, only the lower back and neck are involved. Those with spinal involvement are most likely to test positive for the HLA-B27 genetic marker.

Place of carrying out: class-room, wards of the rheumatologic department.

Study objective: to improve the skills of differential diagnosis of arthritis in rheumatoid arthritis (RA), osteoarthritis (OA), gout, ankylosing spondylitis (AS), psoriatic arthritis, Reiter's disease, arthritis in systemic lupus erythematosus (SLE), dermatomyositis (DM), systemic scleroderma (SSD).

Basic level:

1. To be able to collect complaints, case history, carry out physical examination in patients with arthritis.
2. To interpret instrumental and laboratory data in patients with arthritis, and sacroileitis.
3. To interpret side effects of agents which are used in rheumatology.

Student has to be able to:

1. Find out joint and spine injury using instrumental methods of examination.
2. Make an algorithm of investigations in patients with arthritis.
3. Determine approaches to treatment in different aetiology of arthritis.

The main theoretical questions:

1. Clinical and roentgenologic semiotics of the joint diseases.
2. Diagnostic criteria of rheumatoid arthritis. Common complications. Management.
3. Diagnostic criteria of osteoarthritis. Management.
4. Diagnostic criteria of ankylosing spondylitis. Management.
5. Diagnostic criteria of reactive arthritis, Reiter's disease. Management.
6. Diagnostic criteria of psoriatic arthritis. Management.
7. Diagnostic criteria of gout. Pathogenic management.
8. DMARDs in autoimmune inflammatory joint diseases (RA, psoriatic arthritis, AS, chronic Reiter's disease).
9. Symptomatic treatment of the joints disorders.
10. Using of nonsteroidal anti-inflammatory drugs (NSAID), corticosteroids, immunosuppressive drugs, physiotherapy and sanatorium-resort therapy. Indications and method of arthrocentesis.
11. Differential diagnosis of arthritis and arthralgia in connective tissue diseases (systemic lupus erythematosus (SLE), systemic sclerosis (SS), dermatomyositis (DM) and polymyositis (PM)).
12. Differential diagnosis of pulmonary involvement in connective tissue diseases.
13. Differential diagnosis of renal involvement in connective tissue diseases.

Assignment for self-assessment

1. Pain and swelling of the right knee has developed in 24-year-old man, following enterocolitis epidemic in building team. Arthritis is accompanied by the considerable limitation of the motion range, cutting pain in the eyes, pain in mouth mucous membrane during eating hot and strong food. Patient

has severe synovitis of the right knee. Laboratory tests: an elevated erythrocyte sedimentation rate (ESR). Consultation of the ophtalmologist – acute conjunctivitis. Questions: A. What is the provisional diagnosis? B. What is an additional plan of examination? C. What pathological forms should you make differentiation between?

2. A 54-year-old woman complains of night pain in knees, starting pain, limitation of the motion range. General state became worse a week after cooling. She has been ill through 5 years. Polyarthralgias started when the menstruation stopped. She didn't take any treatment.

Objectively: General state is satisfactory. BP – 130/75 mm Hg, pulse – 78 beats per minute. Cardiovascular and respiratory systems are without any pathology. Abdomen is soft, liver isn't palpated. Knees are deformated, skin and local temperature are normal, the movement of knees is limited by 15°. Blood analysis: L – 6.4×10^9/l, ESR – 11 mm/hour, CRP - -, sialic acid – 0.180. X-ray examination of joint – unequal loss of joint space, osteophytes. What is the provisional diagnosis?

3. A 22-year-old man complains of low back pain and stiffness that is worse on arising and improves with exercise. On examination, he is found to have limited mobility of the sacroiliac joints and lumbar spine. X-ray examination shows bilateral sacroiliitis. A serum test for histocompatibility antigen HLA-B27 is positive. What diagnosis do you suspect? Prescribe the main groups of drugs.

Answers:

1. A. Reiter's syndrome, epidemic form. B. Consultation of urinologist, urethra cytologie examination for finding chlamydies, proteins and its fractions, X-ray examination of knees and sacroiliac joints, synovial fluid aspiration with analysis. C. Peripheric form of ankylosing spondilitis, Psoriatic arthropathy, gonococcal arthritis, and tuberculous arthritis.

2. Oseoarthritis.

3. Ankylosing spondilitis. Disease-modifying drugs, nonsteroidal anti-inflammatory drugs, if it is necessary – corticosteroids, treatment of the muscular spasm.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology
12. https://www.ers-education.org/e-learning/procedure-videos/

11. MANAGEMENT OF PATIENTS WITH HAEMORRHAGIC SYNDROME

Time frame – 6 hours.

Professional motivation. Haemorrhagic syndrome can be caused by different pathogenetic mechanisms. The first group deals with the diseases related to changes in the number and functions of thrombocytes (thrombocytopenia and thrombocytopathy). The second group comprises diseases the bleeding of which is caused by blood coagulation disorders as a result of a hereditary or acquired deficiency of procoagulants or increased content of anticoagulants (haemophilia, dysprothrombinemia, hypo- and afibrinogenemia). The third group comprises diseases the bleeding of which is caused by damage to the vascular wall (Schonlein-Henoch disease, Rendu-Osler disease, etc.). Knowledge of pathogenesis of a certain disease allows to select the only correct way of patient management.

Many disorders can cause diffuse alveolar haemorrhage but autoimmune disorders are the most common (e.g., systemic vasculitides, Goodpasture's syndrome, antiphospholipid antibody syndrome); apart from autoimmune disorders they include pulmonary infections (e.g., invasive aspergillosis, hantavirus infection), toxic exposures (e.g., trimellitic anhydride, isocyanates, crack cocaine, certain pesticides), drug reactions (e.g., propylthiouracil, amiodarone, methotrexate, montelukast, infliximab), cardiac disorders (e.g., mitral stenosis), idiopathic pulmonary haemosiderosis.

Pulmonary-renal syndrome is defined as the combination of diffuse alveolar haemorrhage (DAH) and glomerulonephritis. Haemoptysis is the most common clinical manifestation of DAH. However, 30–35% of patients may have DAH without evidence of haemoptysis. Breathlessness, cough and low-grade fever may also be present. In about 50% of cases of DAH, patients suffer from acute respiratory failure requiring mechanical ventilation. The most common renal manifestation of pulmonary-renal syndrome is haematuria, proteinuria and active urinary sediment. If left untreated, patients can progress to end-stage renal failure, requiring haemodialysis.

Several types of immunologic injury as well as other nonimmunologic mechanisms such as antiglomerular basement membrane (anti-GBM) antibodies, antineutrophil cytoplasm antibodies (ANCA), immunocomplexes and thrombotic microangiopathy are involved in the syndrome's pathogenesis. The underlying pulmonary lesion in the majority of cases of pulmonary-renal syndrome is small-vessel vasculitis, characterized by a destructive inflammatory process that involves arterioles, venules and alveolar capillaries (necrotic pulmonary capillaritis). The term "Goodpasture's syndrome" is used for the clinical entity of DAH and rapidly progressive glomerulonephritis associated with anti-GBM antibodies. Goodpasture's syndrome is extremely rare (one case per 1,000,000 population per year). The disease predominantly affects Caucasians of every age but mostly those in the second to third decades and the fifth to sixth decades of life, with a slight predominance of males. Although rare, this syndrome is responsible for about 20% of acute renal failure cases due to rapidly progressive glomerulonephritis. Circulating ANCA autoantibodies are detected in the majority of patients presenting with pulmonary-renal syndrome. ANCA do not confirm a specific entity but practically lead the differential diagnosis to three major systemic vasculitides syndromes: Wegener's granulomatosis, microscopic polyangiitis and Churg–Strauss syndrome. Wegener's disease (granulomatosis) is characterized by the triad of systemic necrotizing vasculitis, necrotizing granulomatous inflammation of the upper and lower respiratory tract, and necrotizing glomerulonephritis. The incidence of the disease is estimated up to 8.5/million (range 5.2–12.9/million) with a male-to-female ratio of 1:1. The disease usually involves Caucasians (80–97%) with a mean age at the time of diagnosis of 40–55 years, although persons of every age may be affected. The lungs are involved in 90% of cases. In a small percentage of patients, a limited form of the disease that spares the kidney has been described. Microscopic polyangiitis is a systemic small-vessel vasculitis manifested by pauci-immune necrotic glomerulonephritis (80–100% of patients), pulmonary capillaritis (10–30%), skin lesions and arthralgias.

Churg-Strauss syndrome (CSS) is a systemic disease, typically presenting with an initial asthma/sinusitis phase, followed by eosinophilia and vasculitis. In CSS, renal involvement is milder compared with Wegener's disease, Goodpasture's syndrome and microscopic polyangitis. The incidence of CSS is difficult to determine, but limited published data suggest that in the general population the incidence is on the order of 1 to 3 cases/millions persons per year, whereas in asthmatics it is about 60 cases/million/yr. The disease is associated with peripheral eosinophilia as well as eosinophilic infiltration of tissues. P-ANCA (perinuclear ANCA) is reported to be positive in up to

70% of cases.

Pulmonary-renal syndrome in ANCA-negative systemic vasculitis is very rare and has been described only occasionally in Behçet's disease, Henoch-Schönlein purpura, IgA nephropathy, and mixed cryoglobulinaemia. In Henoch-Schönlein purpura, acute capillaritis and DAH involve deposition of IgA immuno-complexes along the pulmonary alveoli.

Patients can require mechanical ventilation and even die as a result of haemorrhage-associated respiratory failure. Recurrent alveolar haemorrhage causes pulmonary haemosiderosis and fibrosis, both of which develop when ferritin aggregates within alveoli and exerts toxic effects. COPD occurs in some patients with recurrent diffuse alveolar haemorrhage secondary to microscopic polyarteritis.

Place of carrying out: class-room, wards of the rheumatologic department.

Study objective: to improve the skills in making a differential diagnosis of haemorrhagic syndrome in rheumatological diseases, to determine a specific management strategy.

Basic level:

1. To be able to collect complaints, case history, carry out physical examination in patients with haemorrhagic syndrome.
2. To interpret instrumental and laboratory data in patients with haemorrhagic syndrome.

Student has to be able to:

1. Make an algorithm of investigations in patients with haemorrhagic syndrome.
2. Determine approaches to treatment considering different aetiopathogenetic factors of haemorrhagic syndrome.
3. Aetiology and pathophysiology of systemic vasculitis.
4. Criteria for systemic vasculitis.

The main theoretical questions:

1. Differential diagnosis of the skin lesion in connective tissue diseases.
2. Differential diagnosis of the pulmonary involvement in systemic vasculitis.
3. Differential diagnosis of the renal involvement in systemic vasculitis.
4. Differential diagnosis of the cardiovascular involvement in connective tissue diseases.
5. Specific methods of research in systemic vasculitis.
6. Criteria for diagnosis of systemic vasculitis. Autoimmune markers. Changes on angiography.
7. Principles of differential diagnosis in systemic vasculitis.
8. Differential diagnosis of vasculitis and disseminated intravascular coagulation syndrome.
9. Differential diagnosis of polyarteritis nodosa and Takayasu's disease.
10. Clinical manifestation of pulmonary-renal syndrome, evaluation and treatment of the critically ill patient.
11. Principles of systemic vasculitis management.

Real-life situations to be solved:

1. A 18-year-old woman had respiratory viral disease a month before and was treated by analgin, paracetamol, biseptol. Pain and swelling of knee joints, haemorrhagic rash on the anterior surface of shins appeared. She withheld medicines but in 3 weeks subfebrile temperature appeared and she restarted taking paracetamol. She was complaining of pain and swelling of knee joints, haemorrhagic rash (purpura) over the crus, hips, rumps, high temperature (38.2 °C), headache. With suspicion on meningitis patient was admitted to the infectious department. In 2 days colicy pain in abdomen and bloody diarrhoea appeared.

Objectively: general state of the patient is serious, skin of the face is pale, multiple conjugating haemorrhagic rash. The temperature is 38.2. BP – 100/65 mm Hg, pulse is 104 beats per minute. Cardiac activity is rhythmic, weakening of the first sound, no murmurs, vesicular respiration in lungs, painful abdomen during palpation. The patient has difficulty motions, swelling knee and ankle joints.

Laboratory examinations: RBC – $2.0*10^{-12}$/l; Hb – 76 g/l; ESR – 44 mm/h; WBC – $26*10^{-9}$/l; eos. – 2%, stab neutr. – 14%, segmented neutrophils – 59%, lymphocytes – 11%, monocytes – 5%. Total serum protein – 65 g/l, serum urea – 6.7 mmol/l, creatinine – 90 µmol/l, bilirubin – 19 µmol/l, AST – 10units, fasting plasma glucose – 4.8 mmol/l, rheumatoid factor – 1:32, CRP – ++, uric acid – 0.25 mmol/l.

Urinalysis: RBC – 45 to 50 per high-power field, WBC – 3 to 4 per high-power field, specific gravity – 1028, red cell casts are detected. Urinary protein excretion is 2.5 g/day. Occult blood test in stool was positive. Abdominal ultrasound is normal. What is the presumptive diagnosis? Suggest additional investigations. What is the treatment protocol? What is prognosis determined by?

2. A 58-year-old white man presented with a six-month history of arthralgia that affected his shoulders, hands, ankles and feet, associated with early morning stiffness and intermittent joint swelling. Two weeks before he had developed ulceration on his elbow and tongue. He also reported weight loss, fever with profuse sweating, haemoptysis, nasal stuffiness, and a six-month history of intermittent deafness. On examination, he had an ulcer over his right elbow measuring 2 × 3 cm and two small ulcers over the left side of his tongue. The second, third, and fourth metacarpophalangeal joints of his right hand and his left ankle were swollen. Urinalysis showed a trace of protein and blood. What is your differential diagnosis? What investigations would you perform to establish the diagnosis?

3. A 28-year-old man presented with a 2-week history of arthralgia that affected his hands and ankles, associated with intermittent joint swelling. A month before he had developed erythema on his face and ulceration on his tongue. He also reported weight loss, loss of vision, fever, nasal stuffiness, periodical epistaxis. On examination, he had pale skin, nasal crusting, small ulcers on his tongue. His ankles were swollen. There is dullness above the inferior lobe of the right lung, moist rales. Heart sounds are weak, systolic murmur above apex, pulse is 96/min, rythmical, BP – 190/110. From the history: 4 months ago patient finished the treatment with antibiotics (from 3 different groups) due to infective endocarditis.

Urinanalyse showed a trace of protein and blood, the 24-hour urinary protein was slightly raised at 0.55 g in 24 hours. Blood tests showed anaemia RBC – $2.7*10^{-12}$/l; Hb – 80 g/l and raised inflammatory markers: ESR of 59 mm/h, C-reactive protein of 84 mg/l. On the entering day haemoptysis appeared. After additional investigations there was revealed shadowing at both lung bases (infiltrates) on the chest X-ray more marked on the right, immunological test showed a positive antineutrophil cytoplasmic antibody with a titre of 1:1280. Blood cultures were negative. Transthoracic echocardiography confirmed moderate mitral regurgitation without vegetations, left atrial dilatation. There was also mild pulmonary hypertension with a systolic pulmonary artery pressure estimated to be 30 mm Hg.

Further investigations to assess the extent of involvement included a high-resolution computed tomogram of the thorax, in which the lungs had a patchy ground glass appearance. In 3 days oedema on the legs and the level of proteinuria increased to 1.4 g in 24 hours, RBC – $2,5*10^{-12}$/l; Hb – 78 g/l; ESR – 60 mm/h; WBC – $16*10^{-9}$/l; eos. – 2%, stab neutr. – 14%, segmented neutrophils – 59%, lymphocytes – 11%, monocytes – 8%. Total serum protein – 45 g/l, serum urea – 9.7 mmol/l, creatinine – 190 µmol/l, bilirubin – 19 µmol/l, AST=20 units. Suggest additional investigations. What is your diagnosis? What is the treatment protocol?

Answers to the self-assessment

1. Presumptive diagnosis: haemorrhagic vasculitis with skin, articular, renal and abdominal syndromes; complicated by gastrointestinal bleeding.

Additional investigations: ECG, bleeding time, clotting time, thrombocytes' count, FGDS, consultations of neurologist and ophthalmologist. Antineutrophil cytoplasmic antibody (ANCA) levels, biopsy of involved organ or tissue, such as skin, sinuses, lung, nerve, and kidney. The biopsy elucidates the pattern of blood vessel inflammation.

Corticosteroid therapy is initiated with prednisone in a dose of 0.5 to 1.5 mg/kg per day.

2. The differential diagnoses are systemic vasculitis with multisystem involvement; infections (HIV, hepatitis B or C); endocarditis; rheumatoid arthritis; and malignancy. Blood tests, immunological tests, skin ulcer biopsy and nasal biopsy, chest radiograph and computed tomography.

3. A diagnosis of Wegener's granulomatosis, a form of necrotising vasculitis, was made on the basis of clinical presentation, the presence of classical antineutrophil cytoplasmic antibody, which is 99% specific for the diagnosis of primary systemic vasculitis (about 90% of patients with active Wegener's granulomatosis are positive for this antigen). It is preferable to have tissue biopsies. Diagnosis is confirmed with a tissue biopsy at a site where disease is active. Nasopharyngeal biopsies are preferable because they are relatively non-invasive compared with a lung or kidney biopsy, which

are at times the only options. In admission we can suspect SLE, so additional investigations should be immunological.

Treatment depends on the extent of involvement. In cases of widespread systemic involvement, and especially if the lungs or kidneys are involved, steroids and immunosuppressive therapy are needed. Cyclophosphamide is typically used to induce remission, and other immunosuppressive agents are considered once the disease is under control. If a diagnosis is in doubt, treatment should not be delayed because Wegener's granulomatosis has a high mortality if left untreated. With the use of cytotoxic drugs 8 year survival is reported to be 80%. So, remission can be induced with pulse-therapy (bolus i.v. methylprednisolone (10 mg/kg) initially 3 days and cyclophosphamide (15 mg/kg) on the 2nd day), and subsequently with oral high-dosage prednisolone (1 mg/kg daily) and continuous oral cyclophosphamide (2 mg/kg daily). Doses of cyclophosphamide should be reduced in the elderly and those with renal impairment. The dose of oral prednisolone is rapidly reduced once remission has occurred. Cyclophosphamide is usually continued for 6–12 months in total. Antihypertensive medicines, antiagregants.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology
12. https://www.ers-education.org/e-learning/procedure-videos/

Semantic module 3. Management of patients in gastroenterology clinic

12. MANAGEMENT OF PATIENTS WITH STOMACH DYSPEPSIA

Time frame – 6 hours.

Professional motivation. Dyspepsia is extremely prevalent, affecting up to 80% of the population at some time, and very often no abnormality is discovered during investigation, especially in younger patients. Patients with "alarm" symptoms, those over 55 years old with new dyspepsia and younger patients unresponsive to empirical treatment require prompt investigation to exclude serious gastrointestinal disease.

Gastro-oesophageal reflux disease is the most common gastrointestinal diagnosis recorded during visits to outpatient clinics. In the United States, it is estimated that 14 to 20% of adults are affected, although such percentages are at best approximations, given that the disease has a nebulous definition

and that such estimates are based on the prevalence of self-reported chronic heartburn. A current definition of the disorder is "a condition which develops when the reflux of stomach contents causes troublesome symptoms (i.e., at least two heartburn episodes per week) and/or complications". Several extra-oesophageal manifestations of the disease are well recognized, including laryngitis and cough. With respect to the oesophagus, the spectrum of injury includes oesophagitis, stricture, the development of columnar metaplasia in place of the normal squamous epithelium (Barrett's oesophagus), and adenocarcinoma. The rising incidence of oesophageal adenocarcinoma is of particular concern, an epidemiologic trend strongly linked to the increasing incidence of this condition. There were about 8000 incident cases of oesophageal adenocarcinoma in the United States in 2004, which represents an increase by a factor of 2 to 6 in disease burden during the past 20 years.

Oesophagitis occurs when excessive reflux of acid and pepsin results in necrosis of surface layers of oesophageal mucosa, causing erosions and ulcers. Impaired clearance of the refluxed gastric juice from the oesophagus also contributes to damage in many patients. Whereas some gastro-oesophageal reflux is normal (and relates to the ability to belch), several factors may predispose patients to pathologic reflux, including hiatus hernia, lower oesophageal sphincter hypotension, loss of oesophageal peristaltic function, abdominal obesity, increased compliance of the hiatal canal, gastric hypersecretory states, delayed gastric emptying, and overeating. Often multiple risk factors are present.

A consistent paradox in gastro-oesophageal reflux disease is the imperfect correspondence between symptoms attributed to the condition and endoscopic features of the disease. In a population-based endoscopy study in which 1000 northern Europeans were randomly sampled, the prevalence of Barrett's oesophagus was 1.6%, and that of oesophagitis was 15.5%. However, only 40% of subjects who were found to have Barrett's oesophagus and one third of those who were found to have oesophagitis reported having reflux symptoms. Conversely, two thirds of patients reporting reflux symptoms had no oesophagitis. Furthermore, although gastro-oesophageal reflux is the most common cause of heartburn, other disorders (e.g., achalasia and eosinophilic oesophagitis) may also cause or contribute to heartburn.

Barrett's oesophagus columnar lined oesophagus (CLO) is a premalignant glandular metaplasia of the lower oesophagus, in which the normal squamous lining is replaced by columnar mucosa composed of a cellular mosaic containing areas of intestinal metaplasia. It occurs as an adaptive response to chronic gastro-oesophageal reflux and is found in 10% of patients undergoing gastroscopy for reflux symptoms. Community-based epidemiological and autopsy studies suggest the true prevalence up to 20 times greater as the condition is often asymptomatic until first discovered when the patient presents with oesophageal cancer. CLO principally occurs in Western Caucasian males and is rare in other racial groups. It is the major risk factor for oesophageal adenocarcinoma, with a lifetime cancer risk of around 10%. Cancer incidence is estimated at 1 in 200 patient years (0.5% per year). The absolute risk is low, however, and more than 95% of patients with CLO die of causes other than oesophageal cancer. The epidemiology and aetiology of CLO are poorly understood. The prevalence is increasing, and it is more common in men (especially white) and those over 50 years of age. It is weakly associated with smoking but not alcohol. Recent studies suggest that cancer risk is related to the severity and duration of reflux rather than the presence of CLO, but this remains to be proven.

In the industrialised world the prevalence of *H. pylori* infection in the general population rises steadily with age, and in the UK approximately 50% of those over the age of 50 years are infected. In many parts of the underdeveloped world infection is much more common and is often acquired in childhood. Up to 90% of the population are infected by adult life in some countries. The vast majority of colonised people remain healthy and asymptomatic and only a minority develop clinical disease. Around 90% of duodenal ulcer patients and 70% of gastric ulcer patients are infected with *H. pylori*; the remaining 30% of gastric ulcers are due to NSAIDs. Although the prevalence of peptic ulcer is decreasing in many Western communities, it still affects approximately 10% of all adults at some time in their lives. The male to female ratio for duodenal ulcer varies from 5:1 to 2:1, whilst that for gastric ulcer is 2:1 or less.

Place of carrying out: class-room, wards of gastroenterology.

Study objective: to be able to determine extent of examinations, put final diagnosis, and assign

management in patients with stomach dyspepsia.

Basic level:

1. Anatomy and physiology, endoscopic peculiarities of alimentary tracts.
2. The main clinical syndromes in the alimentary tract disorders.
3. To make physical examination of patients with disorders of gastrointestinal system.

Student has to know:

1. Criteria for diagnosis and treatment in gastro-oesophageal reflux disease (GERD).
2. Interpretation of laboratory and instrumental investigations (gastric and duodenal juice, X-ray examination of the gastrointestinal system).
3. Rome III diagnostic criteria for functional gastrointestinal disorders.
4. How to make an algorithm of investigations in patients with stomach dyspepsia.
5. Diagnostic and management possibilities of endoscopy with biopsy in gastroenterology.
6. *H. pylori*-associated gastritis. Peptic ulcer disease.

The main theoretical questions:

1. Criteria for functional dyspepsia.
2. "Alarm" symptoms of dyspepsia.
3. Differential programs of stomach dyspepsia treatment.
4. Factors associated with the development of gastro-oesophageal reflux disease. Features of hiatus hernia.
5. Criteria for diagnosis and treatment of gastro-oesophageal reflux disease (GERD). Complications of GERD. Lifestyle modifications.
6. Indications for 24-hours pH-metry of oesophagus, evaluation of results.
7. Diagnostic tests for *H. pylori* infection.
8. Peptic ulcer disease: clinical signs, investigations, management (*H. pylori* eradication, the first-line and second-line therapy). Indications for surgery. Complications of peptic ulcer disease.

Assignment for self-assessment

1. Diagnostic criteria for autoimmune atrophic gastritis are all mentioned, except:
   a) achlorhydria induces G-cell (gastrin producing) hyperplasia, which leads to hypergastrinemia;
   b) pernicious anaemia may develop in longstanding cases;
   c) anti-parietal cell and anti-intrinsic factor antibodies;
   d) low prevalence of Helicobacter pylori;
   e) high prevalence of Helicobacter pylori.

2. A 58-year-old man complaints were as follows: inability to swallow any solids, due to this, the patient was on a liquid diet, reflux after eating food, weight loss. He reported a 2-month history of progressively worsening dysphagia with solids only and weight loss of 10 kg over a period of 3 months. He denied cough, regurgitation, hoarseness, palpitations, and dyspnoea. Past medical history was significant for hypertension for 5 years which had been treated with valsartan and hydrochlorothiazide. He denied any history of cardiovascular problems or arrhythmias. He quit smoking 7 years before and denied drinking alcohol. There was no other significant medical, family or social history. Physical examination revealed: pale skin and mucus with yellow hue, weight – 51 kg, height – 172 cm. He is afebrile. BP is 120/75 mm Hg; pulse is 100 beats/min, regular, respiratory rate is 18 breaths/min.

The thyroid gland is normal to palpation. Normal chest conformity. Peripheral lymph nodes are not enlarged. There is vesicular breathing at auscultation of lungs. Tactile fremitus is normal. The heart apex is nondisplaced. There is no gallop or murmur. The abdomen is soft without tenderness or distention. The liver spans 12 cm in the midclavicular line with a smooth edge. There is no oedema on the legs. Distal pulses are equal.

Blood testing: RBC – $2.8*10^{-12}$/l; Hb – 86 g/l; ESR – 10 mm/h; WBC – $8*10^{-9}$/l; eos. – 2%, stab – 6%, neutrophils – 69%, lymphocytes – 15%, monocytes – 5%. Total protein – 63 g/l, albumin 36 g/l, urea – 8.7 mmol/l, creatinine – 100 μmol/l, bilirubin – 19 μmol/l, fasting glucose – 3.8 mmol/l, cholesterol – 5 mmol/l, triglycerides – 1.8 mmol/l. Urine chemistry: normal.

Chest X-ray: no infiltrates. Oesophagography revealed an irregular stenosing lesion accompanied by low elevation with a major axis of 40 mm in the lower thoracic oesophagus.

What investigations have diagnostic meaning? What pathological process is more probable? What does blood test indicate on?

3. Broncho-oesophageal syndrome in GERD is caused by:
   a) regurgitation of stomach contents to the airways;
   b) overweight;
   c) dysphagia;
   d) oesophagus spasm.

Answers:

1. e.

2. Oesophageal endoscopy and biopsy with histopathological examination. Diagnosis of type 4 oesophageal cancer. (Oesophageal endoscopy showed a stenosing lesion, which bled easily, low elevation and irregular erosion in the lower thoracic oesophagus. A biopsy and histopathological examination revealed either highly or moderately differentiated squamous cell cancer). Myelotoxic anaemia.

3. a.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368с.
6. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
7. https://reference.medscape.com/guide/medical-calculators

13. MANAGEMENT OF PATIENTS WITH ABDOMINAL PAIN

Time frame – 6 hours.

Professional motivation. Functional gastrointestinal disorders are extremely common. Irritable bowel syndrome (IBS) is a functional bowel disorder in which abdominal pain is associated with defecation or a change in bowel habit with features of disordered defecation and distension. Approximately 20% of the general population fulfil diagnostic criteria for IBS but only 10% of these consult their doctors because of gastrointestinal symptoms. Nevertheless, IBS is the most common cause of gastrointestinal referral and accounts for frequent absenteeism from work and impaired quality of life. Young women are most often affected. There is wide overlap with non-ulcer dyspepsia, chronic fatigue syndrome, dysmenorrhoea and urinary frequency.

In Western countries gallstones are common and occur in 7% of males and 15% of females aged 18–65 years, with an overall prevalence of 11%. In those under 40 years there is a 3:1 female preponderance, whereas in the elderly the sex ratio is about equal. Gallstones are common in North America, Europe and Australia, and are less frequent in India, the Far East and Africa. In developed countries the incidence of symptomatic gallstones appears to be increasing and they occur at an earlier age. Stones in the common bile duct (choledocholithiasis) occur in 10–15% of patients with gallstones. These stones account for more than 80% of common bile duct stones; they migrate from the gallbladder, and are similar in appearance and chemical composition to the stones found elsewhere. Primary bile duct stones may develop infrequently within the common bile duct many years after a

cholecystectomy or represent the accumulation of biliary sludge consequent upon dysfunction of the sphincter of Oddi. In Far Eastern countries, where bile duct infection is common, primary common bile duct stones are thought to follow bacterial infection secondary to parasitic infections with *Clonorchis sinensis*, *Ascaris lumbricoides* or *Fasciola hepatica*. Common bile duct stones can cause partial or complete bile duct obstruction and may be complicated by cholangitis due to secondary bacterial infection, septicaemia, liver abscess, and biliary stricture.

Place of carrying out: class-room, wards of the gastroenterology.

Stuydy objective is to improve students' skills in putting final diagnosis and assigning management of patients with abdominal pain.

Basic level:

1. To make physical examination of patients with disorders of gastrointestinal system.
2. The main syndromes and symptoms of the diseases of stomach and duodenum.
3. Mechanisms of the abdominal pain.
4. To evaluate data of the laboratory and instrumental investigations.

Student has to know:

1. Diseases which are accompanied by abdominal pain.
2. Criteria for diagnosis of gastritis.
3. Criteria for diagnosis of ulcer disease.
4. Treatment of gastritis and ulcer disease.
5. Complications of ulcer disease and its treatment.
6. Indications to the surgical treatment of ulcer disease.
7. Interpretation of laboratory and instrumental investigations (gastric and duodenal juice, X-ray examination of the gastrointestinal system).
8. How to make an algorithm of investigations in patients with abdominal pain.

The main theoretical questions:

1. Criteria for gastritis diagnosis.
2. Zollinger-Ellison syndrome.
3. Invasive and non-invasive methods for H. pylori diagnostics.
4. Criteria for diagnosis of the stomach tumours.
5. Peptic ulcer disease: indications for surgery. Complications of peptic ulcer disease and treatment.
6. Management of patients with gastritis, ulcer diseases.
7. Chronic pancreatitis: clinical features, investigations, complications, management.
8. Pancreatic carcinoma: clinical features, investigations, management.
9. Functional bowel disorders: criteria for irritable bowel syndrome. Management.
10. Ischaemic gut injury as a result of arterial occlusion: chronic mesenteric ischaemia.
11. Gallstones and choledocholithiasis: clinical features, investigations, complications, management.
12. Biliary motor disorder ("biliary dyskinesia").

Assignment for self-assessment

1. A 29 year old man was treated with the first line medicines for 10 days (according to Maastricht II consensus) due to ulcer disease. Now he continues treatment with omeprazol but 2 weeks ago persistant and rising pain appeared. He underwent appendectomy at the age of 22, which was uncomplicated. His father has a duodenum ulcer.

On physical examination BP is 110/70 mm Hg, heart rate is 104 beats/min and respiratory rate is 18 breaths/min. He is afebrile. The chest is clear to auscultation and percussion. The heart is regular without extra sounds or murmurs. The abdomen is painful in epigastria, isn't tender when touched. The liver and spleen are not palpable. The liver spans 11 cm in the midclavicular line with a smooth edge.

Blood testing: RBC – $3.9*10^{-12}$/l; Hb – 136 g/l; ESR – 8 mm/h; thrombocytes – $250*10^{-9}$/l; WBC – $9*10^{-9}$/l; eos. – 2%, neutrophils – 69%, lymphocytes – 32%, monocytes – 5%. Total protein – 63 g/l, albumins – 60%, globulins – 40% (α-globulins 10%, β-globulins 11%, γ-globulins 17%), urea – 6.7 mmol/l, creatinine – 70 μmol/l, bilirubin – 19 μmol/l, fasting glucose – 4.8 mmol/l, cholesterol –

4 mmol/l, AST – 37 U/L, ALT – 46 U/L, γ-GT – 57 U/L, sodium – 125 meq/L, prothrombin time – 80%, alkaline phosphatase – 46 U/L.

Chest radiographic findings, ECG are normal.

What complication caused impairment in patient's condition?

2. The main complaints in ulcerative colitis are: a) abdominal pain, frequent, small-volume fluid stools or constipation; b) heartburn, nausea; c) eructation; d) elevation of BP.

3. Signs of Crohn's disease are: a) fasting night pain in epigastria, relieving-pain vomiting; b) abdominal pain mostly in the morning, fluid stools with mucus; c) colicy abdominal pain that relieve after defecation and passage of flatus; d) pain is often associated with diarrhoea which is watery and does not contain blood or

4. mucus, weight loss.

5. Signs of chronic cholecystitis complicated by cholangitis are: a) epigastric pain, vomiting; b) heartburn, hypersalivation; c) anorexia; d) diarrhoea; e) rigors, right-hypochondrium pain.

Answers:

1. Penetration.

2. a. 3. d. 4. e.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
6. Mayerle, J. Chronic Pancreatitis [Електронний ресурс] / J. Mayerle, G. Beyer. — 2017.
7. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
8. https://reference.medscape.com/guide/medical-calculators

14. MANAGEMENT OF PATIENTS WITH JAUNDICE

Time frame – 6 hours.

Professional motivation. Chronic hepatitis B affects about 300 million people around the world; infection is associated with cirrhosis and primary hepatocellular carcinoma. Chronic carrier rates of the virus following infection vary from 10–20% in Asia, Africa, the Middle East and the Pacific Islands, where most infections are acquired in infancy, to 2% in Europe and North America.

Prognosis varies depending on the cause of the hepatitis. The overall mortality of acute viral hepatitis is about 0.5% in otherwise well patients under 40 years of age, but mortality reaches about 3% in patients over 60 years and may be much higher in patients with other serious diseases, such as chronic liver disease, carcinoma or lymphoma. Prognosis in hepatitis B: full recovery occurs in 90–95% of adults following acute HBV infection. The remaining 5–10% develop a chronic infection which usually continues for life, although later recovery occurs occasionally. Infection passing from mother to child at birth leads to chronic infection in the child in 95% of cases and recovery is rare. Recovery from acute HBV infection occurs within 6 months and is characterised by the appearance of antibody to viral antigens. Persistence of HBeAg beyond this time indicates chronic infection. Combined HBV and HDV infection causes more aggressive disease. Most patients with chronic hepatitis B are asymptomatic and develop complications such as cirrhosis and hepatocellular carcinoma only after many years. Cirrhosis develops in 15–20% of patients with chronic HBV, over 5–

20 years. This proportion is higher in those infected in childhood.

HCV caused over 90% of post-transfusion hepatitis before serological tests allowed the screening of blood donors, and accounted for the high incidence of chronic hepatitis in patients with haemophilia. Parenteral drug users continue to be at high risk of HCV infection. Chronic infection occurs in about 70–80% of patients and this is usually life-long. Most never suffer from acute illness. Chronic HCV usually remains asymptomatic for years and is not associated with an early increase in mortality. However, many patients eventually develop cirrhosis and some progress to hepatocellular carcinoma. Approximately 20% of chronically infected patients will develop cirrhosis after 20 years of infection, and around 50% after 30 years. This is more likely if patients are misusing alcohol as well. Once cirrhosis is present, 2–5% per year will develop hepatocellular carcinoma.

Place of carrying out: class-room, wards of the gastroenterology.

Study objective is to improve students' skills in differential diagnosis and treatment of patients with jaundice.

Basic level:

1. Bilirubin metabolism.
2. The main clinical syndromes in liver disorders.
3. To be able to collect complaints, case history, carry out objective examination of patients with liver disorders.
4. To interpret instrumental and laboratory data in patients with jaundice.

Student has to know:

1. How to put provisional and final diagnosis and assign management in jaundice.
2. Differential diagnosis in jaundice of different origin.

The main theoretical questions:

1. Jaundice causes. Examples of conditions with increased breakdown of red blood cells.
2. Congenital nonhaemolytic hyperbilirubinaemia. Criteria for diagnosis and treatment of Gilbert's syndrome.
3. Clinical and biochemical criteria for different types of jaundice.
4. Algorithm of investigations in patients with jaundice.
5. Hepatitis B, C, D: clinical features, investigations, complications.
6. Indications for interferon therapy in viral hepatitis. Criteria of effective therapy by interferons in hepatitis. Side effects of interferon therapy.
7. Autoimmune hepatitis: clinical features, investigations, complications, management.
8. Sclerosing cholangitis: clinical features, investigations, management.
9. Gallstones and choledocholithiasis: clinical features, investigations.

Assignment for self-assessment

1. A 32 year old man is noted to have fatigue at the end of a busy working week, yellow sclera. He underwent appendectomy at the age of 22, which was uncomplicated. Risk factors for chronic hepatitis are absent, except that he insufflated cocaine a few occasions during his college years.

On physical examination the patient looks generally well, except jaundice. Blood pressure is 132/92 mm Hg, heart rate is 84 beats/min, respiratory rate is 14 breaths/min. He is afebrile. The neck is supple without lymphadenopathy or thyromegaly. The chest is clear to auscultation and percussion with no gynecomastia or spider telangiectasias. The heart is regular without extra sounds or murmurs. The abdomen is soft without tenderness or distention. The left lobe of liver is not palpable nor is there splenomegaly. The liver spans 8 cm in the midclavicular line with a smooth edge. There is no abdominal collateral circulation, umbilical hernia, or bruit. There are no signs of ascites, other stigmata of chronic liver disease. The extremities show no clubbing, cyanosis, oedema, nor is there palmar erythema. Neurologic examination is within normal limits without asterixis. The skin has no stigmata of chronic liver disease.

Blood testing: RBC – $3.9*10^{-12}$/l; Hb – 136 g/l; ESR – 8 mm/h; thrombocytes – $250*10^{-9}$/l; WBC – $6*10^{-9}$/l; eos. – 2%, neutrophils – 69%, lymphocytes – 32%, monocytes – 5%. Total protein – 63 g/l, albumins – 60%, globulins – 40% (α-globulins 10%, β-globulins 11%, γ-globulins 19%), urea – 6.7 mmol/l, creatinine – 70 μmol/l, bilirubin – 39 μmol/l, fasting glucose – 4.8 mmol/l, cholesterol –

6 mmol/l, AST – 87 U/L, ALT – 166U/L, γ-GT – 87 U/L, sodium – 105 meq/L, prothrombin time – 42.8%, alkaline phosphatase – 96 U/L, fibrinogen – 2 g/l.

Hepatitis serology: anti-HAV total – positive, anti-HAV IgM – negative, anti-HBc – positive, HBsAg – negative, anti-HBs – positive, anti-HCV – positive, HCV RNA – positive. Chest radiographic findings, ECG are normal. Abdominal ultrasound revealed a mildly echogenic liver with normal contour, portal vein diameter is 10 mm, spleen vein diameter is 5 mm. No gallstones are seen.
What is the most likely clinical diagnosis? What treatment does this patient need?

2. Mild jaundice, best seen by examining the sclerae in natural light, is usually detectable when serum bilirubin reaches: a) 25 μmol/l; b) 35 μmol/l; c) 60 μmol/l; d) 100 μmol/l.

3. Jaundice in pancreatic cancer differs from jaundice in gallstones by mentioned signs except: a) development without previous pain attack; b) fast increasing of bilirubin; c) positive Courvoisier-Terrier syndrome; d) persistant and intensive jaundice; e) appears in elder patients.

Answers:

1. The elevation of serum alanine aminotransferase indicates hepatocellular injury as opposed to cholestasis. Serologic testing reveals previous exposure to hepatitis A, hepatitis B, and hepatitis C. Hepatitis A infection does not become chronic. Hepatitis B serologic pattern is diagnostics of previous exposure, but not chronic infection. Hepatitis C antibody positivity with an exposure risk and elevation of aminotransferases makes the diagnosis of chronic HCV infection a near certainty.

The quantification of HCV RNA has become an important part of the therapy of patients chronically infected with HCV, although it is important to note that there is no correlation between viral load and disease severity. Liver biopsy is recommended before treatment to assess the grade and stage of disease and to exclude other forms of liver disease or complications (such as concurrent alcoholic liver disease, medication-induced liver injury, and iron overload).

The patient was noted to be viremic with HCV viral load of 650,000 IU/ml, genotype 1a. Liver biopsy revealed portal inflammatory cells with periportal hepatic fibrosis (grade 2 stage 2). After detailed discussion with the patient regarding the risks and benefits of interferon-based therapy, it was decided to proceed with administration of peginterferon and ribavirin.

2. b. 3. b.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
6. Mayerle, J. Chronic Pancreatitis [Електронний ресурс] / J. Mayerle, G. Beyer. — 2017.
7. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
8. https://reference.medscape.com/guide/medical-calculators

15. MANAGEMENT OF PATIENTS WITH HEPATOMEGALY

Time frame – 6 hours.

Professional motivation. Hepatic cirrhosis can occur at any age and often causes prolonged morbidity. It frequently manifests itself in younger adults and is an important cause of premature death. Any condition leading to persistent or recurrent hepatocyte death may lead to hepatic cirrhosis, e.g., viral hepatitis and alcohol. Prolonged biliary damage or obstruction, as can occur in primary

biliary cirrhosis, sclerosing cholangitis and post-surgical biliary strictures, will also result in cirrhosis. Persistent blockage of the venous return from the liver, e.g., veno-occlusive disease and Budd-Chiari syndrome, will eventually result in liver cirrhosis. Worldwide, the most common causes of cirrhosis are viral hepatitis and prolonged excessive alcohol consumption. The overall prognosis in cirrhosis is poor.

Many patients present with advanced disease and/or serious complications that carry a high mortality. Overall, only 25% of patients survive 5 years from diagnosis but, where liver function is good, 50% survive for 5 years and 25% for up to 10 years. The prognosis is more favourable where the underlying cause of the cirrhosis can be corrected, as in alcohol misuse, haemochromatosis and Wilson's disease. Laboratory tests give only a rough guide to prognosis in individual patients. Deteriorating liver function, as evidenced by jaundice, ascites or encephalopathy, indicates a poor prognosis unless a treatable cause such as infection is found. The course of cirrhosis is uncertain, as unforeseen complications such as variceal bleeding may lead to death unexpectedly.

Fatty liver is a common and generally benign condition. The majority of obese patients (60–90%) and up to 50% of type II diabetics have fatty liver. The outlook for most patients with steatosis is excellent, although a few deaths have been reported. In patients with alcoholic steatosis, the severity of the fatty change can predict the eventual progression to cirrhosis. Previously, the prognosis of patients with acute fatty liver of pregnancy was considered poor. However, milder forms of this condition are now more frequently recognised.

Place of carrying out: class-room, wards of the gastroenterology.

Study objective is to improve students' skills in putting final diagnosis and assigning management of patients with hepatomegaly.

Basic level:

1. Examination of the patients with hepatomegaly.
2. Evaluation of the laboratory and instrumental investigations data.
3. Classification of hepatitis and cirrhosis.

Student has to know:

1. Diseases for which hepatomegaly is inherent and how to put presumptive diagnosis.
2. Investigations in patients with suspected liver disease (the liver function tests, biochemical and coagulation tests, liver biopsy).
3. How to make differential diagnosis in hepatomegaly.
4. How to indicate the treatment for patients with hepatomegaly.

The main theoretical questions:

1. Definition and causes of hepatolienal syndrome.
2. Plan of investigations of hepatolienal syndrome.
3. Criteria for diagnosis of fatty liver.
4. Criteria for diagnosis of drugs-toxic hepatitis.
5. Classification of chronic hepatitis. Criteria for diagnosis of chronic autoimmune hepatitis.
6. Classification of cirrhosis. Criteria for diagnosis of liver cirrhosis.
7. Criteria for diagnosis of alcoholic cirrhosis.
8. Treatment of chronic hepatitis, and cirrhosis, their complications.

Assignment for self-assessment

1. Splenomegaly and ascitis are observed in: a) primary biliary cirrhosis; b) portal liver cirrhosis; c) Wilson's disease; d) portal hypertensive syndrome of different origin.

2. A 47 year old female patient was referred with a complaint of pruritus which developed 2 months before.

Medical history: she has ulcerative colitis (complaints of the bloody defecation with mucus for 6–7 times a day) and she uses mesalazine tablet with 3 g/day; the bloody diarrhoea with mucus had been regressed. A pruritus was begun about 2 months ago which become refractory and she was referred to the clinic. She did not have any connective tissue disease. She also did not have a story of drug use that will affect the hepatobiliary system except mesalazine. She is a non-smoker, non-alcoholic.

On the physical examination: she is fully conscious and oriented. Patient is of average build. Pulse is 72/minute regular, blood pressure is 130/90 mmHg, her skin and sclera's are in a mild icteric appearance, and the liver is exceeding the costa border about 2 cm. There was not any pathological finding on examination of the other systems.

Laboratory examinations: RBC – $3.5*10^{-12}$/l; Hb – 116 g/l; ESR – 28 mm/h; thrombocytes – $250*10^{-9}$/l; WBC – $6*10^{-9}$/l; eos. – 2%, neutrophils – 69%, lymphocytes – 32%, monocytes – 5%. Total protein – 63 g/l, albumins – 60%, globulins – 40% (α-globulins 10%, β-globulins 11%, γ-globulins 19%), urea – 6.7 mmol/l, creatinine – 70 μmol/l, bilirubin – 39 μmol/l, fasting glucose – 4.8 mmol/l, cholesterol – 6 mmol/l, AST – 67 U/L (normal: 0–41 U/l), ALT – 106 U/L (normal: 0–40 U/L), γ-GT – 87 U/L, sodium – 105 meq/L, prothrombin time – 42.8%, alkaline phosphatase – 396 U/L (normal: 30–91 U/L), GGT–124 U/L (normal: 0–61 U/L), fibrinogen – 2 g/l. CRP – 2.08 mg/dL. Viral hepatitis panel is negative.

Immunological tests: anti-nuclear antibody (ANA), antismooth muscle antibody (ASMA), antiliver and antikidney microsome antibody (LKM), p-ANCA are negative. Antimitochondrial antibodies (AMA) are positive with a value >1/160 and anti-M2 antibody (AMA-M2) is also positive. Thyroid function tests are normal.

Abdominal ultrasonography: hepatomegaly. Magnetic resonance cholangiopancreatography: gallbladder, common bile duct and intrahepatic bile ducts were evaluated as normal.

Total colonoscopy: ulcerative colitis was detected in the descending colon, sigmoid colon and mild to moderate with rectal involvement.

Biopsy was taken from the sigmoid colon and rectum and crypts: cryptic micro abscesses and crypt distortion are observed. Liver biopsy: diffuse inflammation and the portal areas were infiltrated by the lymphocytes and histiocytes.

Upper gastrointestinal endoscopy revealed that the oesophageal mucosa and lumen are normal, no oesophageal varices.

What is your clinical diagnosis? Management?

3. Bad prognostic signs in liver cirrhosis are:
   a) increasing plasma bilirubin;
   b) hypoalbuminemia or an albumin concentration < 30 g/l;
   c) marked hyponatraemia (< 120 mmol/l not due to diuretic therapy);
   d) prolonged prothrombin time;
   e) all of the above signs are correct.

Answers:

1. d.

2. Primary biliary cirrhosis (PBC), stage 1. Ulcerative colitis.

Ursodeoxycholic acid is introduced as 15 mg/kg/day besides mesalazine therapy (on the follow-up conducted a month later, her complaint of pruritus was regressed and the outcome of liver function test reached normal ranges).

Ulcerative colitis is a disease which may have extraintestinal complications. Wide range variations of the liver diseases may be seen in the patients with ulcerative colitis. Hepatobiliary disease, primary sclerosing cholangitis is the most seen concomitant disease by 5% in the patients with ulcerative colitis. PBC is another autoimmune disease similar to primary sclerosing cholangitis characterized by itching, and hyperbilirubinemia. AMA is found in approximately 95% of the patients with a negative value of 5%. PBC generally affects the middle-aged females more than males. The prevalence of PBS in the patients with UC is 30 times higher than in the general population. The best test used in the differential diagnosis is AMA. AMA may be always positive in PBC, while it is always negative in primary sclerosing cholangitis.

3. e.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed.

— Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
6. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
7. https://reference.medscape.com/guide/medical-calculators

16. MANAGEMENT OF PATIENTS WITH PORTAL HYPERTENSION

Time frame – 6 hours.

Professional motivation. Population-based prevalence data for portal hypertension are not available, but portal hypertension is a frequent manifestation of liver cirrhosis. Extrahepatic portal vein obstruction is frequently the cause of portal hypertension in childhood and adolescence, while cirrhosis causes 90% or more of portal hypertension in adults in Western countries. Schistosomiasis is the most common cause of portal hypertension worldwide but it is infrequent outside endemic areas. Variceal haemorrhage is the most common complication associated with portal hypertension. Almost 90% of patients with cirrhosis develop varices, and approximately 30% of varices bleed. The first episode of variceal haemorrhage is estimated to carry a mortality rate of 30–50%.

Place of carrying out: class-room, wards of the gastroenterology.

Study objective is to to be able to put final diagnosis in portal hypertensive syndrome and assign management.

Basic level:

1. Collateral circulation in portal hypertension.
2. To examine patients with liver diseases.
3. To determine symptoms, to group them into the syndromes, and to select the leading syndrome.
4. To evaluate data of the laboratory and instrumental investigations in portal hypertensive syndrome.

Student has to know:

1. Clinical signs in portal hypertension.
2. Investigational methods for the diagnosis of portal hypertension.
3. How to make differential diagnosis, clinical diagnosis due to modern classification.
4. How to indicate the treatment for patients with portal hypertensive syndrome.

The main theoretical questions:

1. Types of portal hypertension.
2. Methods for pressure evaluation in portal vein.
3. Differential diagnosis in portal hypertension.
4. Complications of portal hypertension.
5. Treatment of patients with portal hypertension. Pharmacological reduction of portal pressure.

Assignment for self-assessment

1. Methods of portal hypertension diagnostics are the mentioned besides: a) splenomanometry; b) hepatomanometry; c) splenoportography; d) oesophagography; e) FGDS.

2. What is the pressure in splenomanometry in moderate portal hypertension?

a) 200–300 mm; b) 120–150 mm; c) 350–500 mm; d) 50–80 mm.

3. Portal hypertension develops in: a) liver veins thrombosis; b) liver arteries thrombosis; c) splenic vein thrombosis; d) mesenteric arteries thrombosis; e) splenic infarction.

4. What advantages of endoscopic ligation of bleeding oesophageal varices do you know comparing to endoscopic sclerotherapy?

5. A 57-year-old man was admitted with a three day history of weakness, anorexia, unsteady gait. He reported smoking 1 pack per day and drinking alcohol for the past 10 years. Over the previous 6 months, the patient had been unemployed and reported increased alcohol consumption.

On physical examination, the patient was noted to have marked ascites with a prominent fluid wave and bulging flanks, bilateral pitting oedema above the knees, pallor, spider teleangiectasia on the arms, palmer erythema, jaundice. His pulmonary and cardiovascular examinations were unremarkable. The abdomen is distended. The liver spans 17 cm in the midclavicular line with a smooth and dense edge. Blood pressure –130/80 mmHg, pulse rate – 69 beats/min. No endocrine changes.

Blood testing: RBC – $2.9*10^{-12}$/l; Hb – 96 g/l; ESR – 8 mm/h; thrombocytes – $120*10^{-9}$/l; WBC – $6*10^{-9}$/l; eos. – 2%, neutrophils – 69%, lymphocytes – 32%, monocytes – 5%. Total protein – 50 g/l, albumins – 40%, globulins – 60% (α-globulins 10%, β-globulins 11%, γ-globulins 39%), urea – 6.7 mmol/l, creatinine – 70 μmol/l, bilirubin – 59 μmol/l, fasting glucose – 4.8 mmol/l, cholesterol – 6 mmol/l, AST – 107 U/L, ALT – 66 U/L, γ-GT – 97 U/L, sodium – 105 meq/L, prothrombin time – 42.8%, fibrinogen – 2 g/l.

Chest radiographic findings, ECG are normal.

Abdominal ultrasound revealed a nodular liver surface, massive splenomegaly, portal vein diameter is 15 mm, moderate ascities. Upper gastrointestinal endoscopy showed grade III oesophageal varices and severe portal gastropathy.

What syndromes does this patient have? What additional diagnostic examinations are indicated?

What is the most likely clinical diagnosis?

Answers:

1. d. 2. a. 3. a.

4. Associated with lower complication rate, lower mortality rate, fewer number of treatments for varices eradication.

5. Portal hypertensive syndrome, hepatic encephalopathy, anaemic syndrome.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
6. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
7. https://reference.medscape.com/guide/medical-calculators

Semantic module 4. Management of patients in pulmonology clinic

17. MANAGEMENT OF PATIENTS WITH BRONCHOOBSTRUCTIVE SYNDROME

Time frame – 6 hours.

Professional motivation. According to WHO estimates, 65 million people have moderate to severe chronic obstructive pulmonary disease (COPD). More than 3 million people died of COPD in 2005, which corresponds to 5% of all deaths globally. Most of the information available on COPD prevalence, morbidity and mortality comes from high-income countries. Even in those countries, accurate epidemiologic data on COPD are difficult and expensive to collect. It is known that almost 90% of COPD deaths occur in low- and middle-income countries. At one time, COPD was more common in men, but because of increased tobacco use among women in high-income countries and the

higher risk of exposure to indoor air pollution (such as biomass fuel used for cooking and heating) in low-income countries, the disease now affects men and women almost equally.

In 2002 COPD was the fifth leading cause of death. Total deaths from COPD are projected to increase by more than 30% in the next 10 years unless urgent action is taken to reduce the underlying risk factors, especially tobacco use. Estimates show that COPD becomes in 2030 the third leading cause of death worldwide.

According to WHO estimates, 235 million people suffer from asthma. There is evidence that its prevalence has increased considerably over the past 20 years, especially in children. Asthma is the most common chronic disease among children. Unfortunately the prevalence of asthma symptoms in children varies from 1 to more than 30 percent in different populations and is increasing in most countries, especially among young children. Fortunately asthma can be effectively treated and most patients can achieve good control of their disease. Asthma is not just a public health problem for high income countries: it occurs in all countries regardless of level of development. Over 80% of asthma deaths occur in low and lower-middle income countries. Asthma is under-diagnosed and under-treated, creating a substantial burden to individuals and families and possibly restricting individuals' activities for a lifetime.

Bronchiectasis shares many clinical features with chronic obstructive pulmonary disease (COPD), including inflamed and easily collapsible airways, obstruction to airflow, and frequent office visits and hospitalizations. The prevalence of bronchiectasis is unknown, due to the lack of standardised medical care and poor healthcare access in underdeveloped countries. Helpful information is provided by independent analyses from two large and different United States databases: an estimated 110,000 individuals have bronchiectasis in the United States, the prevalence of bronchiectasis increases with age, bronchiectasis is more common in women.

In the UK, incidence is estimated at 1.06 to 1.3 per 100,000 population. A study in New Zealand estimated the incidence there to be high in children, with a rate of 3.7 per 100,000; incidence varied with ethnicity, with the highest rate being 17.8 per 100,000 in Pacific children. In general, it is estimated that the incidence has decreased over the past several decades due to implementation of vaccination programmes and the development of more potent antibiotics. Infection is the most common cause of bronchiectasis in underdeveloped countries.

Factors that affect mortality in patients with moderate to severe bronchiectasis include advanced age, St George's Respiratory Questionnaire activity score, Pseudomonas aeruginosa infection, total lung capacity (TLC), and residual volume divided by TLC.

Prognosis for people with bronchiectasis depends on how well infections and other complications are prevented or controlled. People with co-existing conditions, such as chronic bronchitis or emphysema, and people who have complications, such as pulmonary hypertension or cor pulmonale, tend to have a worse prognosis.

Study objective: to improve the practical skills in evaluation of the most important signs of ventilation abnormalities, to do the differentiation between lung diseases which accompany bronchial obstruction. To prescribe the treatment according to aetiology, peculiarities, and intensity of bronchial disorders and allergic status.

Basic level:

1. Examination of patient with lung disease.
2. Laboratory and instrumental data in patients with pathology of respiratory system.
3. Interpretation of sputum analysis, spirography, peakflowmetry, X-ray examination, chest tomography.
4. Symptoms and differential diagnosis of lung diseases.
5. Treatment of bronchial obstruction.

Student has to know:

1. Diagnostic algorithm in bronchoobstructive syndrome.
2. How to make clinical diagnosis.
3. How to indicate differential programs of the treatment.
4. How to interpret side effects of corticosteroids, broncholytics (short- and long-acting).

The main theoretical questions:

1. Aetiology and pathogenesis of bronchial obstruction.
2. Clinical signs of syndromes: bronchial spasm (paroxysmal and steady), bronchial inflammation (diffuse and local), delay of sputum, obturation and compression of bronchus, trachea and bronchial narrowing, bronchial drainage disturbances, mucous hypersecretion, hypersensitivity of bronchus.
3. Classification and clinical signs in BA. Diagnostic significance of peakflowmetry.
4. Classification and clinical signs in COPD. Spirometry values.
5. Classification and clinical signs in bronchiectasis.
6. Differential diagnosis between BA, COPD, and bronchiectasis.
7. Management of patients with BA depending on levels of asthma control. Daily dosages for inhaled glucocorticosteroids. Indication for systemic steroid treatment.
8. Management of asthma exacerbation. Side effects of inhaled steroids.
9. Management of patients with COPD depending on severity. Indication for inhaled steroids.
10. Definition of bacterial exacerbation of COPD. Management.
11. Management of patients with bronchiectasis.

Assignment for self-assessment

1. Diagnostic criteria for COPD are:
   a) FEV1<90% predicted, FEV1/FVC<80%; b) FEV1<80% predicted, FEV1/FVC<70%;
   c) FEV1<70% predicted, FEV1/FVC<60%; d) FEV1<60% predicted, FEV1/FVC<50%;
2. A 21-years-old patient works at the pharmacy, complains of dry cough, recurrent attacks of dyspnoea for 2 months, on weekends symptoms decrease, t – 37.0 °C. Current attack of dyspnoea was stopped by salbutamol. BP is 126/80 mm Hg, heart rate is 92 beats/minute and regular, respiratory rate is 18 breaths/minute. Examination reveals mildly oedematous nasal mucosa with no discharge. Cardiac examination shows regular rate and rhythm without murmur. On pulmonary auscultation prolonged expiration and bilateral wheezing are heard over all parts of the lungs. X-ray shows no abnormalities. What is the presumptive diagnosis?
   a) bronchial asthma;
   b) COPD;
   c) medicamentous disease;
   d) spontaneous pneumothorax.

Answers:

1. b. 2. a.

REFERENCES

1. Global Initiative for Asthma. http://www.ginasthma.org
2. http://www.goldcopd.org
3. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
4. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
5. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
6. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
7. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
8. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
9. https://reference.medscape.com/guide/medical-calculators
11. https://www.ers-education.org/e-learning/procedure-videos/

18. DIFFERENTIAL DIAGNOSIS AND MANAGEMENT OF PATIENTS WITH PULMONARY INFILTRATES

Time frame – 6 hours.

Professional motivation. Pulmonary infiltrates frequently develop in pneumonia, infiltrative tuberculosis, pulmonary infarction, lung cancer, acute eosinophilic pneumonia, and Churg-Strauss syndrome.

The clinical approach to persistent pulmonary infiltrate requires evaluating several factors, including host factors (age, comorbidities, immunodeficiency), the severity of symptoms, and the possibility of a non-infectious aetiology. The history and clinical examination, augmented by laboratory evaluation and radiographic techniques, can narrow the differential diagnosis.

In 2010, 8.8 million people fell ill with tuberculosis (TB) and 1.4 million died from TB. Over 95% of TB deaths occur in low- and middle-income countries, and it is among the top three causes of death for women aged 15 to 44. TB is a leading killer of people living with HIV causing one quarter of all deaths. In the absence of major complications, short-course therapy using four drugs initially is curative. Occasionally, patients die of overwhelming infection (usually miliary disease or bronchopneumonia) and some patients succumb to the later complications of tuberculosis (e.g., cor pulmonale). A few patients die unexpectedly soon after commencing therapy and it is possible that some of these individuals have subclinical hypoadrenalism that is unmasked by a rifampicin-induced increase in steroid metabolism. In HIV-associated tuberculosis, mortality is increased but mainly as a result of superimposed bacterial infection.

Pulmonary embolism (PE) affects an estimated 117 people per 100,000 person years, resulting in about 350,000 cases yearly, and causes up to 85,000 deaths/yr. PE affects mainly adults.

An estimated 10% of patients with PE die within 1 h. Of those who survive the first hour, only about 30% are diagnosed and receive treatment; > 95% of these patients survive. Thus, most patients with PE are never diagnosed; it is in such patients that most mortality from PE occurs. The best prospects for reducing mortality lie in improving diagnosis, not in improving treatment. Patients with chronic thromboembolic disease represent a tiny fraction of patients with PE who survive.

At the end of the 20th century, lung cancer had become one of the leading causes of preventable death. The 5-year relative survival rate for lung cancer for the period of 1995 to 2001 was 15.7%, reflecting a steady but slow improvement from 12.5% from 1974 to 1976. The 5-year relative survival rate varies markedly depending on the stage at diagnosis, from 49 to 16 to 2% for local, regional, and distant stage disease, respectively. Stage at diagnosis accounts for the most marked variation in prognosis, but patient characteristics associated with poorer survival also include being older, male, and African American.

Study objective: to study the differential diagnosis in cases of pulmonary infiltrates using laboratory and instrumental investigations.

Basic level:

1. Anatomy, physiology, and pathophysiology of respiratory system.
2. Aetiology, pathogenesis, classification, and diagnostic criteria for pneumonia.
3. Diagnostic criteria for tuberculosis.
4. Diagnostic criteria for lung cancer.
5. Diagnostic criteria for lung atelectasis and middle lobe syndrome.
6. X-ray examination of lungs.
7. Pharmacodynamics, pharmacokinetics, indications and contraindications to antibacterial drugs administration. Principles of antibacterial therapy.

Student has to know:

1. Diagnostic algorithm in pulmonary infiltrates detection.
2. How to make clinical diagnosis.
3. How to indicate differential programs of the treatment.

The main theoretical questions:

1. Differential diagnostics of pulmomycoses (systemic candidosis, histoplasmosis, coccidiomycosis,

actinomycosis).

2. Differntial diagnosis in case of lung atelectasis and middle lobe syndrome.
3. Differential diagnostics of tuberculosis and pneumonia.
4. Differential diagnostics of lung cancer and pneumonia.
5. Differential diagnosis between pneumonia and pulmonary infarction.
6. Diagnostic criteria for eosinophilic pulmonary infiltration, sarcoidosis. Management.
7. Diagnostic algorithm in the most frequent pulmonary dissemination (disseminated (miliary) tuberculosis, pneumonia, pulmonary sarcoidosis, collagenoses).

Real-life situation to be solved:

1. A 35 year old patient had sudden onset of fever, high temperature (40 °C), headache, dry cough. He cought a cold during fishing. On examination: body temperature is 39.9 °C, hyperemia of the cheeks, a light cyanosis of the lips, the skin is pale, humid, rapid, breathing is shallow – 32/min. On the 3rd day from the onset of disease the patient had haemoptysis. The expansion of the right lung is limited, there is dullness above the inferior lobe of the right lung, brochial breathing, moist rales on percusion. Heart sounds are weak, tachycardia – 96/min, rythmical, BP – 110/60. Other systems without any changes. Laboratory findings: RBC – $3.0*10^{-12}$/l; Hb – 116 g/l; ESR – 28 mm/h; WBC – $16*10^{-9}$/l; eos. – 2%, stab – 6%, neutrophils – 69%, lymphocytes – 15%, monocytes – 5%. Total protein – 73 g/l, albumin 46 g/l, urea – 6.7 mmol/l, creatinine – 90 µmol/l, bilirubin – 19 µmol/l, fasting glucose – 4.8 mmol/l, cholesterol – 5 mmol/l, triglycerides – 1.8 mmol/l. The X-ray findings: confluent opacities in inferior lobe.

What is the diagnosis? What complications of this disease do you know? What diseases should you make differentiation between? Treatment.

2. A 26 year old patient F. complains of moist cough. Two weeks ago there was dry cough and high temperature (38.2 °C). On X-ray: infiltrate in the upper lobe (under the clavicular) of the left lung. After 10 days of antibioticotherapy X-ray investigation was performed: shadow of the upper left lobe resolved but oval infiltration with illegible edge of 2×2 cm appeared in the right lower lobe. In percution: clear pulmonary sound, on auscultation: vesicular breathing. Laboratory findings: RBC – $3.5*10^{-12}$/l; Hb – 126 g/l; ESR – 18 mm/h; WBC – 10×10^{-9}/l; eos. – 20%, stab – 2%, neutrophils – 69%, lymphocytes – 25%, monocytes – 5%. Total protein – 73 g/l, albumin 46 g/l, urea – 6.6 mmol/l, creatinine – 98 µmol/l, bilirubin – 19 µmol/l, fasting glucose – 4.8 mmol/l. Sputum analysis: no mycobacteria.

What is the possible diagnosis? What diseases should you make differentiation between? Additional investigations. Treatment?

Answers:

1. Pneumonia. Pulmonary failure, myocarditis, pleuritis. Tuberculosis, lung infarction. Antibiotics, broncholytics.
2. Eosinophilic infiltration. Pneumonia, tuberculosis. Chest CT. Corticosteroids.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
6. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
7. https://reference.medscape.com/guide/medical-calculators
8. https://www.ers-education.org/e-learning/procedure-videos/

19. FEVER OF UNDETERMINED ORIGIN. CRITERIA FOR RHEUMATIC FEVER, DIFFERENTIAL DIAGNOSIS

Time frame – 6 hours.

Professional motivation. Fever of undetermined origin (FUO) presents one of the most challenging and perplexing problems in clinical medicine. Such fevers may persist for weeks or months in the absence of characteristic clinical findings or clues. Ultimately, most such obscure fevers prove to be caused by common diseases presenting in an atypical fashion rather than by rare and exotic illnesses.

The relative frequency of the aetiologic categories responsible for FUO have been relatively stable over the past 5 decades. Noninvasive testing provides the diagnosis in 42% of patients, with serologies and lysis centrifugation blood cultures being the most useful noninvasive techniques. Computed tomography-guided percutaneous biopsies are the most useful invasive procedures. Only 9% of patients remain undiagnosed. With an increasing population of immunosuppressed and chronically ill patients, the relative importance of the disorders that cause FUO may change. As a result, it may be prudent to consider so-called classic FUO separately from FUO in patients with HIV infection, neutropenia, or nosocomial fevers. In the initial evaluation of FUO, the possibility of infection should be carefully considered.

The two major systemic infections to consider in the evaluation of FUO are tuberculosis (usually disseminated but sometimes confined predominantly to the liver and spleen) and infective endocarditis. Most FUO cases caused by miliary tuberculosis arise in elderly patients in which, dissemination has followed activation of quiescent foci. Often, in cases caused by miliary tuberculosis, the intermediate-strength (5 tuberculin units) purified protein derivative skin test is negative, and miliary pulmonary lesions are not present on the chest X-ray. Anaemia, leukopenia, or, rarely, a leukemoid reaction caused by bone marrow involvement may be evident; bone marrow biopsy is a very helpful diagnostic test in patients in whom miliary tuberculosis is suspected. An isolated elevation of the serum alkaline phosphatase level may indicate miliary involvement of the liver by tuberculosis, other infection, or neoplasm. The histologic findings on liver biopsy often suggest the diagnosis, and a portion of the specimen should always be cultured for the presence of tubercle bacilli.

Infective endocarditis, usually subacute, is also an important diagnostic consideration. Most patients with subacute bacterial endocarditis have a heart murmur. In about 5% of cases, however, particularly in the elderly, the murmur may be absent or may be considered functional. Blood cultures would be expected to provide the diagnosis in a patient with subacute bacterial endocarditis, particularly because only 5% of patients with endocarditis have negative blood cultures. The leading cause of negative blood cultures in patients with endocarditis is the prior administration of antibiotics. It is therefore very important that a number of blood cultures be obtained, including some as long as 5 to 10 days after antibiotics have been withdrawn. Other causes of culture-negative endocarditis that should be considered in patients with FUO include infection with fastidious bacteria, chlamydial infection, and Q fever.

Viral infections are usually self-limited and do not produce fevers that last longer than 3 weeks. Important exceptions to this generalization are Epstein-Barr virus (EBV) and cytomegalovirus (CMV) infections, which may occasionally present as FUO (often with some mononucleosis-like features) in otherwise healthy individuals. More frequently, CMV infection develops in patients who have received multiple blood transfusions or who have undergone organ transplantation; CMV is the cause of 50% of all febrile episodes in renal transplant recipients.

More common types of localized infection that present as FUO include hepatic abscess, subphrenic abscess, and subhepatic and pericholecystic abscess. Liver abscesses are often occult; the physician should look for a history that includes symptoms of biliary tract disease, recent blunt abdominal trauma, or travel, which might suggest the diagnosis of amebiasis. Hepatomegaly may be absent initially. The serum alkaline phosphatase level is usually elevated even when the abscess is solitary. Serologic tests for amebiasis are positive in patients with amebic liver abscess. Localized infection in

the urinary tract is an important consideration in a patient with FUO; perinephric abscess and renal carbuncle are best diagnosed by ultrasonography or CT. Many other localized infections occasionally present as FUO; occult dental infections are one such example and illustrate the need for thoroughness in the evaluation of patients with obscure fevers.

Lymphoma, particularly Hodgkin disease, is the most common neoplastic cause of obscure fever. Lymphoma may be difficult to diagnose when the principal site of involvement is the retroperitoneal nodes, but abdominal CT scans greatly facilitate this diagnosis; a skin biopsy may help identify intravascular lymphoma as the cause of an FUO. The development of fever in a patient who has myeloma or chronic lymphocytic leukemia is usually caused by superimposed infection and not by the neoplastic process; in some patients, however, the febrile course appears to be caused by the malignancy itself. Occasionally, a patient with the preleukemia syndrome will present with fever and atypical blood and bone marrow changes, suggesting myeloid metaplasia or a leukemoid response. Only after some months the haematologic picture can be established as leukemia.

Solid tumours can also be associated with fever; hypernephroma is the leading example. As many as 10% of patients with colorectal carcinoma present with fever; either extension of the tumour through the bowel wall, producing a paracolonic abscess, or necrosis and abscess formation in a polypoid intraluminal lesion may be the underlying mechanism. Metastatic cancer may be responsible for continuing fever; hepatic involvement is not necessary for fever to occur. Occasionally, a neuroblastoma involving bone or soft tissues or a pheochromocytoma may have a febrile course. Fevers caused by malignant disease often respond to therapy with NSAIDs; fevers caused by infections may be less likely to respond completely to these agents, but this distinction is not sufficient as a diagnostic test.

A variety of connective tissue disorders and vasculitis may produce prolonged fevers before the development of articular or other characteristic manifestations. In the elderly, polymyalgia rheumatica and the closely related disorder giant cell arteritis (temporal arteritis) are the most common connective tissue disorders presenting as FUO.

As many as 15% of cases of giant cell arteritis present as FUO, and in some patients, the vasculitis itself remains occult. Similarly, virtually all patients with adult-onset Still disease are febrile, and systemic symptoms such as fever and weakness may antedate by weeks or months the evolution of the more characteristic clinical manifestations of adult juvenile rheumatoid arthritis. In other patients, involvement of the paranasal sinuses and mastoid or the rapid excavation of a pulmonary lesion suggests Wegener granulomatosis. Many other connective tissue diseases, ranging from classic vasculitides such as systemic lupus erythematosus to uncommon disorders such as relapsing polychondritis, can also present as FUO.

Granulomatous diseases of noninfectious origin may be responsible for FUO. Prolonged fever is uncommon in sarcoidosis, but when it does occur, prominent hilar adenopathy, ocular involvement, erythema nodosum, and hepatic granulomas are usually also present. Biopsy of involved lymph nodes, muscle, or liver usually shows noncaseating granulomas. In addition to sarcoidosis, there are about 40 diseases that may be associated with hepatic granulomas. Treatable infectious granulomatous diseases (e.g., tuberculosis, brucellosis, histoplasmosis, and cat-scratch disease) must be ruled out by cultures, skin tests, serologic tests, and special stains of tissue biopsy specimens. Beneficial results have been achieved in such cases by giving corticosteroids after excluding the other specific granulomatous diseases; methotrexate also appears to be helpful.

Inflammatory bowel disease. Bowel symptoms are prominent in almost all patients with idiopathic ulcerative colitis, granulomatous colitis, or regional enteritis, and the diagnosis is obvious in such febrile patients. Occasionally, however, bowel symptoms may not be marked or may be of such long duration that they become accepted as the norm. In this setting, FUO may be the presenting complaint in a patient with inflammatory bowel disease.

Fever is occasionally observed in cases of cirrhosis. Attention should first be directed to possible complicating infections – such as spontaneous bacterial peritonitis, enterogenous bacteremias, or tuberculosis – titis and may account for low-grade fever.

Drug fever frequently occurs in the absence of other manifestations of hypersensitivity, such as rash

and eosinophilia. Antimicrobial agents (e.g., β-lactams, sulfonamides, nitrofurantoin, and isoniazid), antihypertensives (e.g., hydralazine and methyldopa), anticonvulsants (e.g., phenytoin), and allopurinol are among the most common offenders, but many other drugs have been implicated. In most instances, the diagnosis of drug fever is considered within the first several weeks of onset of FUO, and any recently administered drugs are discontinued. Several drugs, however, such as phenytoin, methyldopa, and isoniazid, may not produce drug fever until weeks or months after their initial use. Intramuscular injections of analgesics can pro duce FUO, which may or may not be accompanied by the presence of a sterile abscess or other gross evidence of tissue injury.

Whipple disease is a multisystem infection caused by the gram-positive actinomycete *Tropheryma whippelii*. Patients may present with a prolonged febrile illness in association with weight loss, arthralgias, and weakness. The use of special tissue culture and immunodiagnostic tests in diagnosis are being studied; a polymerase chain reaction test for the causative organism is highly sensitive and specific, but false positive reactions have been reported.

CNS lesions are decidedly uncommon causes of FUO, except in very obtunded patients with extensive brain damage. Endocrinologic abnormalities, such as subacute thyroiditis, or metabolic disorders, such as hypertriglyceridemia, hypercholester olemia, or glycosphingolipid storage disease (Fabry's disease), may occasionally present as FUO. Many other disorders as diverse as pernicious anaemia and xanthogranulomatous pyelo nephritis have been identified as rare causes of FUO.

Fever is extremely common in patients infected with HIV; typically, the diagnostic challenge is not in determining the source of fever but in deciding which of several potentially pyrogenic processes is most important. However, patients infected with HIV can also present with prolonged, diagnostically obscure fevers. Most often, such patients have advanced AIDS, with $CD4^+$ T cell counts of less than $100/mm^3$. Infections account for more than 75% of FUO cases in such patients; in one series, the more common diagnoses were disseminated *Mycobacterium avium* complex (31%), *Pneumocystis carinii* pneumonia (13%), CMV infection (11%), disseminated histoplasmosis (7%), and lymphoma (7%). Other infectious aetiologies are toxoplasmosis, cryptococcosis, salmonellosis, and varicella-zoster virus infections. In Europe, leishmaniasis is also an important cause of FUO in patients with AIDS. Among noninfectious aetiologies, lymphoma and drug fever are most prominent. HIV itself is an uncommon cause of FUO.

In 10% to 15% of patients with FUO, a detailed workup fails to reveal the diagnosis. In about half these cases, the fever resolves spontaneously. Reevaluation of the patient some weeks or even months later may provide the diagnosis. The prognosis of patients with undiagnosed FUO is surprisingly good; few require empirical corticosteroid therapy, and many can be managed symptomatically with NSAIDs.

Place of carrying out: class-room, wards of cardiology and pulmonology department.

Study objective: to improve students' skills to make diagnosis of fever of unknown origin.

Basic level:

1. To be able to collect complaints, case history, carry out objective examination.
2. To interpret instrumental (ECG, EchoCG, X-ray) and laboratory data in patients with fever of unknown origin.
3. Modern aspects of aetiology and pathogenesis of rheumatic fever. Classification of rheumatic fever.
4. Criteria for rheumatic fever, infective endocarditis.
5. Criteria for AIDS, SLE, tuberculosis (other granulomatous diseases), etc.

Student has to be able to:

1. To make an algorithm of investigations in patients with fever of unknown origin.
2. To determine approaches to treatment of fever of unknown origin.
3. To find out clinical and laboratory symptoms in patients with rheumatic fever, to unite them into syndromes.

The main theoretical questions:

1. Criteria for fever of unknown origin. Common etiologies of fever of unknown origin (infections, malignancies, autoimmune conditions, miscellaneous).
2. Diagnostic significance of blood cultures, serologic tests, functional blood tests, skin tests, radiologic examinations, biopsy specimens.

3. Differential diagnosis of rheumatic fever and myocarditis.
4. Differential diagnosis of rheumatic fever and infective endocarditis.
5. Differential diagnosis of rheumatic polyarthritis and rheumatoid arthritis.
6. Differential approaches to treatment.
7. Prevention of recurrent rheumatic fever.
8. Aetiological and pathogenetic treatment of rheumatic fever.
9. Symptomatic treatment of rheumatic fever.
10. Aetiological and pathogenetic treatment of infective endocarditis.
11. Indications to operative treatment of infective endocarditis.

Assignment for self-assessment

1. A 34-year-old man was admitted to the emergency room with the recent onset of fever (38–39 °C), dyspnoea, palpitation. A high-pitched, diastolic murmur, heard best in the third intercostal space along the left sternal border. Infective endocarditis is suspected. What sign would be found on echocardiography?

2. Doctor didn't prescribe antipyretic for patient with flu proceeding from protecting influence of high temterature. What mechanism of protective influence in fever do you know?

a) direct negative influence of fever on infecting agent;
b) activation of erythrogenesis;
c) activation of lysozyme production;
d) activation of interferon production;
e) activation of antibody formation.

3. A 19 year old woman was admitted to the hospital with history of high grade swinging temperature up to 38.8 °C. She had had a sore throat, which lasted for a few days, accompanied by fever, rigors, and myalgia. Her general practitioner prescribed amoxicillin, and she subsequently developed a macular rash on her wrists, back, and legs associated with the fever spikes. The symptoms were persistent over three weeks, prompting referral to the department. She hadn't travelled to anywhere recently. She had no history of recreational drug use or sexual contact and she was not taking any regular medication.

On initial examination she was tachycardic (100 beats/min) and febrile (37.5 °C) with a BP of 108/68 mm Hg. Her oral cavity and cardiovascular, respiratory, abdominal, and nervous system examinations were normal. There was no lymphadenopathy. Her right knee was tender but she had no swollen joints.

Laboratory findings: RBC – $3.0*10^{-12}$/l; Hb – 116 g/l; ESR – 28 mm/h; WBC – $14*10^{-9}$/l; eos. – 2%, stab – 6%, neutrophils – 79%, lymphocytes – 15%, monocytes – 5%. Total protein – 73 g/l, albumin 46 g/l, urea – 6.7 mmol/l, creatinine – 90 μmol/l, bilirubin – 19 μmol/l, AlT = 64 IU/l, γ glutamyltransferase – 227 (normally 12–43) IU/l, alkaline phosphatase 127 (30–115) IU/l, CRP = 326 mg/l, normal antistreptolysin O titres, negative blood film for malarial parasites, negative results for mononucleosis spot test, IgM for Epstein-Barr virus, cytomegalovirus polymerase chain reaction, hepatitis B surface antigen, and serology for hepatitis C, chlamydia, autoantibody screen negative (RF, ANA, double stranded DNA, extractable nuclear antigen, antineutrophil cytoplasmic antibodies).

Chest radiography and abdominal ultrasonography showed no abnormality. Blood and throat was sent for culture. Throat swab culture, fungal blood cultures, serial bacterial blood cultures, and malarial films gave negative results. Transthoracic echocardiograms appeared normal. Three weeks after admission she continued to have spiking temperatures of up to 40 °C. She also described a simultaneous erythematous rash, which was not raised and was most pronounced on the dorsal aspect of her legs. She thought that the rash was similar to her previous rash but not as prominent. She later developed a 1 cm firm, mobile lymph node in the right anterior triangle of her neck. Fine needle aspiration gave negative results and she had a biopsy. The lymph node biopsy specimen was initially reported to be consistent with reactive inflammation.

What differential diagnoses would you consider? What further investigations should be carried out?

Answers:

1. Vegetations are recognized substantially more frequently in two-dimensional echocardiography – show valvular destruction and vegetations.

2. d.

3. The differential diagnoses after initial investigations include malignancy (particularly lymphoma), sarcoidosis, Still's disease, connective tissue disease, vasculitis, and infective causes including tuberculosis, fungal infection, endocarditis and HIV. She had no relevant risk factors for HIV, but sexual histories can be initially unreliable, particularly if taken in the presence of relatives. Computed tomography of the abdomen and pelvis, transoesophageal echocardiograms, HIV IgM antibody, polymerase chain reaction (for detecting HIV DNA) should be carried out.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
6. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
7. https://reference.medscape.com/guide/medical-calculators
8. https://www.ers-education.org/e-learning/procedure-videos/

20. MANAGEMENT OF PATIENTS WITH PLEURAL EFFUSION

Time frame – 6 hours.

Professional motivation. Approximately 1.5 million pleural effusions are diagnosed in the United States each year. Heart failure is responsible for approximately one third of all pleural effusions and the leading cause of pleural effusion in the US is also CHF, with an estimated annual incidence of 500,000. Pneumonia is second with an incidence of 300,000. Approximately 40% of the hospitalised patients with pneumonia have an associated parapneumonic effusion. Malignancy is the third leading cause overall, with an estimated incidence of 200,000; however, it is the second most common cause of effusion in patients >50 years of age. Pulmonary embolus, viral disease, coronary artery bypass surgery, and cirrhosis are also common causes of effusion. Small pleural effusions are present in up to 40% of patients with pulmonary embolism. Of all patients with cirrhosis, 5% have an associated pleural effusion. TB is an important cause of pleural effusion in the developing world and should also be considered in travellers returning from endemic areas and in immunocompromised people.

The prognosis in pleural effusion varies in accordance with the condition's underlying aetiology. However, patients who seek medical care earlier in the course of their disease and those who obtain prompt diagnosis and treatment have a substantially lower rate of complications than do patients who do not.

Morbidity and mortality of pleural effusions are directly related to cause, stage of disease at the time of presentation, and biochemical findings in the pleural fluid. Morbidity and mortality rates in patients with pneumonia and pleural effusions are higher than those in patients with pneumonia alone. Parapneumonic effusions, when recognized and treated promptly, typically resolve without significant sequelae. However, untreated or inappropriately treated parapneumonic effusions may lead to empyema, constrictive fibrosis, and sepsis.

Development of malignant pleural effusion is associated with a very poor prognosis, with median survival of 4 months and mean survival of less than 1 year. The most common associated malignancy in

men is lung cancer, and the most common associated malignancy in women is breast cancer. Median survival ranges from 3–12 months, depending on the malignancy. Effusions from cancers that are more responsive to chemotherapy, such as lymphoma or breast cancer, are more likely to be associated with prolonged survival, compared with those from lung cancer or mesothelioma.

Cellular and biochemical findings in the fluid may also be indicators of prognosis. For example, a lower pleural fluid pH is often associated with a higher tumour burden and a worse prognosis.

Place of carrying out: class-room, wards of the cardiology and rheumatology departments, X-ray department.

Study objective: to be able to determine extent of examinations to put final diagnosis and assign management.

Basic level:

1. To be able to collect complaints, case history, carry out objective examination.
2. To interpret instrumental (X-ray, CT) and laboratory data in patients with pleural effusion.
3. To identify signs from objective data inherent to pleural effusion.

Student has to know:

1. How to examine patients with pleural effusion.
2. How to make an algorithm of investigations in patients with pleural effusion.
3. How to determine approaches to treatment in different aetiology of pleural effusion.

The main theoretical questions:

1. Differential diagnosis of pleural effusion of different aetiology.
2. Infectious pleuritis, aceptic pleuritis: pathogenesis.
3. X-ray examination in pulmonology: indications, methodology, interpretation of obtained data.
4. Investigation of sputum: methodology of collection, interpretation of obtained data.
5. Techniques of pleural punction, indications, interpretation of obtained data of pleural effusion.

Assignment for self-assessment

1. The most frequent causes of non-infectious pleuritis are:
   a) diffuse connective tissue diseases;
   b) chest trauma;
   c) pulmonary infarction in pulmonary embolism;
   d) malignancy;
   e) acute pancreatitis, myocardial infarction;
   f) all written above.

2. Specific gravity of transudate, protein content and leukocytes count in 1 mkl of transudate in laboratory investigation are:
   a) <1005 g/l, <15g/l, <500;
   b) <1010 g/l, <20g/l, <750;
   c) <1015 g/l, <30g/l, <1000;
   d) <1020 g/l, <35g/l, <1500;
   e) <1025 g/l, <40g/l, <1500.

3. Patient F., 60 years old, with clinical symptoms of pleuritis was performed pleural punction. Obtained haemorrhagic fluid examination revealed a specific gravity of more than 1.030, protein – 3.5 g/dL; fluid LDH to serum LDH ratio 0.75; atypical cells.

What kind of fluid was obtained? What are the most frequent causes of exudative pleuritis?

4. A patient with low-grade fever and weight loss has poor excursion on the right side of the chest with decreased fremitus, flatness to percussion, and decreased breath sounds all on the right. The trachea is deviated to the left. The most likely diagnosis is:
   a) pneumothorax;
   b) pleural effusion;
   c) consolidated pneumonia;
   d) atelectasis.

Answers:

1. f. 2. c.

3. Exudative fluid. Tuberculosis, cancer, nonspecific inflammation.
4. b.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
6. Mayerle, J. Chronic Pancreatitis [Електронний ресурс] / J. Mayerle, G. Beyer. — 2017.
7. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
8. https://reference.medscape.com/guide/medical-calculators
9. https://www.ers-education.org/e-learning/procedure-videos/

21. CRITERIA FOR DIAGNOSIS AND DIFFERENTIAL PROGRAMS OF TREATMENT IN COMMUNITY-ACQUIRED PNEUMONIA

Time frame – 6 hours.

Professional motivation. Community-acquired pneumonia (CAP) is a common and potentially serious illness. It is associated with considerable morbidity and mortality, particularly in elderly patients and those with significant comorbidities. The overall rate of CAP in adults is approximately 5.16 to 6.11 cases per 1000 persons per year; the rate of CAP increases with increasing age. There is seasonal variation, with more cases occurring during the winter months. The rates of pneumonia are higher for men than for women and for black persons compared with Caucasians. The aetiology of CAP varies by geographic region; however, Streptococcus pneumoniae is the most common cause of pneumonia worldwide.

In 2003, the age-adjusted death rate caused by influenza and pneumonia was 20.3 per 100,000 persons. Estimates of the incidence of community-acquired pneumonia range from 4 million to 5 million cases per year, with about 25% requiring hospitalization. In 2005, pneumonia and influenza combined was the eighth most common cause of death in the United States and the seventh most common cause of death in Canada. There were over 60,000 deaths due to pneumonia in the United States. Mortality is highest for CAP patients who require hospitalization, with a 30-day mortality rate of up to 23 percent in such patients. All-cause mortality in patients with CAP is as high as 28% within one year.

Study objective: to verify diagnosis of community-aquired pneumonia (CAP), to determine management of patients with community-aquired pneumonia.

Basic level:

1. Anatomy, physiology, and pathophysiology of respiratory system.
2. To be able to collect complaints, case history, carry out objective examination.
3. To interpret instrumental and laboratory data in patients with CAP.
4. To interpret side effects of antibacterial drugs. Indications and contraindications.

Student has to know:

1. How to put provisional and final diagnosis and assign management in CAP.
2. How to differentiate CAP with TB, cancer.

The main theoretical questions:

1. Classification of pneumonia.
2. Symptoms, laboratory and instrumental data in CAP.

3. Signs of severe pneumonia.

4. The symptoms, laboratory and instrumental data of pneumonia in immunocompromised patients. Management.

5. Complications of pneumonia.

6. Management of pregnant women with pneumonia.

7. Antibiotics: groups, indications depending on aetiology of pneumonia.

Real-life situation to be solved:

1. A 50 year old man presented with a 3 days history of fever and a productive cough. There was dyspnoea and patient addressed his complaints to a doctor. He doesn't smoke. There was no history of past surgical procedures, any drug or environmental allergies.

Objectively: he has a temperature of 37.6 °C, respiratiory rate is 20 breaths/min; heart rate of 96, regular; blood pressure of 135/80. He has bronchial breath sounds at the right lung base, impairment of the percussion note and intensified tactile fremitus. The heart is regular without extra sounds or murmurs. The abdomen is soft without tenderness or distention. The liver spans 10 cm in the midclavicular line with a smooth edge. The extremities show no oedema.

Laboratory examinations: RBC – $4.2*10^{-12}$/l; Hb – 140 g/l; ESR – 22 mm/h; WBC – $11*10^{-9}$/l; eosinophils – 1%, stab neutrophils – 5%, segmented neutrophils – 79%, lymphocytes – 11%, monocytes – 5%. Total serum protein – 65 g/l, serum urea – 5.7 mmol/l, creatinine – 110 μmol/l, bilirubin – 18 μmol/l, fasting plasma glucose – 5.3 mmol/l. Chest X-ray shows a right lobe infiltrate (homogeneous opacity).

What is your differential diagnosis? What is your final diagnosis? How would you treat this patient? How can you estimate the adequacy of therapy?

2. Patient M, 46 years old, complains of dry cough, dyspnoea after physical exertion during 3 month. On chest X-ray: diffuse lung fibrosis and bilateral mediastinal adenopathy. Differential diagnosis? What examinations are necessary?

1. A 35 year old man presented with a 5 days history of fever, muscular pain and dry cough. Sore throat and running nose started 2 weeks ago. He used amoxicillin 2 g/day but there was no effect. There was no history of past surgical procedures, any drug or environmental allergies.

Objectively: he has a temperature of 37.6 °C, respirations 20 breaths/min; heart rate of 96, regular; BP is 135/80. He has harsh breathing above lungs, percussion and tactile fremitus is normal. The heart is regular without extra sounds or murmurs. The abdomen is soft without tenderness or distention. The liver spans 12 cm in the midclavicular line with a smooth edge. The extremities show no oedema.

Laboratory examinations: RBC – $4.2*10^{-12}$/l; Hb – 140 g/l; ESR – 24 mm/h; WBC– $8*10^{-9}$/l; eosinophils – 1%, stab neutrophils – 5%, segmented neutrophils – 79%, lymphocytes – 19%, monocytes – 5%. Total serum protein – 65 g/l, serum urea – 5.7 mmol/l, creatinine – 110 μmol/l, bilirubin – 18 μmol/l, fasting plasma glucose – 5.3 mmol/l. Chest X-ray shows low-intensity homogeneous opacities in inferior lobe of right lung.

What is your presumptive diagnosis? How would you treat this patient?

Answers:

1. TB, lung cancer. CAP. Antibiotics, expectorants, bronchodilatators, oxygenotherapy. In 72 hours we have to check effectiveness of treatment using clinical signs, laboratory criteria.

2. Sarcoidosis, limphoma, mediastinal or lung cancer, lymphogranulomathosis. CT, sputum smear and microorganisms sensitivity, biopsy.

3. Mycoplasma pneumonia. Macrolides.

REFERENCES

1. Management of Community-Acquired Pneumonia in Adults //www.pulmonology.co.za/guidelines/CAP%202007.pdf

2. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.

3. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.

4. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
5. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
6. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
7. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
8. https://reference.medscape.com/guide/medical-calculators
9. https://www.ers-education.org/e-learning/procedure-videos/

22. CRITERIA OF DIAGNOSIS AND TREATMENT IN CASE OF HOSPITAL-ACQUIRED PNEUMONIA

Time frame – 6 hours.

Professional motivation. Hospital-acquired pneumonia (HAP) is the second most common nosocomial infection with a crude overall rate of 6.1 per 1000 discharges. It is estimated to occur in 250,000 persons per year, representing about 15% to 18% of all nosocomial infections. By comparison, the infection rate for nosocomial urinary tract infection, the most common hospital-acquired infection, is 11 per 1000 discharges. The incidence of HAP varies depending on the hospital environment.

A Canadian descriptive study of non-ICU HAP in a tertiary care hospital showed a mean (± SD) age of 63±17 years of which 55 (65%) were male. The majority of HAP cases (81%) were acquired on surgical wards.

The incidence of HAP is greater among patients in the ICU. Generally, approximately 30% of HAP occurs in critical care settings.

The incidence of ventilator-associated pneumonia (VAP) from the National Nosocomial Infections Surveillance (NNIS) data is 7.6 cases per 1000 ventilator-days. The risk for VAP peaks at day 5 of mechanical ventilation. NNIS data showed that the incidence of VAP was highest for trauma ICUs (15.2 per 1000 ventilator-days). The overall prevalence of VAP was 9.3%. In a Canadian cohort study of 1014 patients ventilated for 48 h or greater, 177 (17.4%) developed VAP. The median duration from ICU admission to the onset of VAP in this study was seven days. Acute respiratory distress syndrome (ARDS) carries an increased risk for VAP.

HAP has been shown to have the highest mortality rate of all nosocomial infections. In one study, the crude case-fatality rate was 30%, rising to 33% in cases attributable to an initial episode of HAP. In a Canadian study of non-ICU HAP, overall mortality rate was 20%, with a direct attributable mortality of 14%. The mortality rate from HAP varied from 7% in patients on general wards to as high as 62% in patients in bone marrow transplant units.

Death from bacteremic HAP occurred in 20% of patients within one week of their first positive blood culture, and *Pseudomonas aeruginosa* bacteremia was associated with the highest mortality rate (45%). The mortality rate in this study was similar for both the ICU (22.2%) and non-ICU patients (17.6%).

The mortality rate for VAP ranges from 24% to 50%, and can reach as high as 76% in specific settings or when lung infection is caused by high-risk pathogens. The attributable mortality of VAP in a Canadian study showed an increase in risk of death (absolute risk increase: 5.8%).

The costs of HAP and VAP are substantial and have been attributed to longer stays in hospital and greater hospital expenditures when compared with patients without HAP.

Major points. The incidence of HAP and VAP together is between five and 10 cases per 1000 hospital admissions, depending on the case definition used and the study population. Together, HAP and VAP are the second most common cause of hospital-acquired infection and are associated with a higher mortality than any other nosocomial infection. Patients with late-onset HAP or VAP have a similar rate of mortality to those with early-onset disease. Approximately 30% of HAP occurs in the

ICU setting where the majority of cases (greater than 85%) occur in patients on mechanical ventilation.

Study objective: to diagnose and prescribe the treatment in case of HAP.

Basic level:

1. To be able to collect complaints, case history, carry out objective examination.
2. To interpret instrumental and laboratory data in patients with HAP.
3. To interpret side effects of antibacterial drugs. Indications and contraindications.
4. Symptoms of hospital pneumonia.

Student has to know:

1. How to put provisional and final diagnosis.
2. How to assign management in HAP.

The main theoretical questions:

1. Definition of HAP. Microbial aetiology. Ventilator-associated pneumonia.
2. Risk factors for HAP or VAP.
3. Clinical manifestations of HAP and VAP. Diagnostic approaches. Diagnostic algorithms.
4. Antibiotic treatment in case of HAP. Nonantimicrobial therapeutic interventions.
5. Major points and recommendations for prevention and risk reduction of HAP and VAP.
6. Pneumonia in immunocompromised patients.

Real-life situation to be solved:

A 50 year old man complains of 4 days history of fever, dry cough, dyspnoea. 5 days ago patient admitted to the surgical department due to acute pancreatitis. He smokes. There was no history of past surgical procedures, any drug or environmental allergies.

Objectively: he has a temperature of 37.6 °C, respiratiory rate is 24 breaths/min; heart rate of 96, regular; blood pressure of 135/80. He has bronchial breath sounds at the right lung base, crepitation, impairment of the percussion note and intensified tactile fremitus. The heart is regular without extra sounds or murmurs. The abdomen is soft without tenderness or distention. The liver spans 10 cm in the midclavicular line with a smooth edge. The extremities show no oedema.

Laboratory examinations: RBC – $4.2*10^{-12}$/l; Hb – 140 g/l; ESR – 22 mm/h; WBC– $11*10^{-9}$/l; eosinophils – 1%, stab neutrophils – 5%, segmented neutrophils – 79%, lymphocytes – 11%, monocytes – 5%. Total serum protein – 65 g/l, serum urea – 5.7 mmol/l, creatinine – 110 µmol/l, bilirubin – 18 µmol/l, fasting plasma glucose – 5.3 mmol/l. Chest X-ray shows a right lobe infiltrate (homogeneous opacity).

What is your clinical diagnosis? How would you treat this patient? What aetiology of pneumonia is more probable?

Answer: hospital-acquired (nosocomial) pneumonia. Patient belongs to group 1 (according to treatment algorithm for HAP). Empirical treatment with iv/oral antibiotic (monotherapy) for 7–8 days, broncholytics. Staphylococci, gram-negative bacilli, *P. aeruginosa*.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
6. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
7. https://reference.medscape.com/guide/medical-calculators
8. https://www.ers-education.org/e-learning/procedure-videos/

23. MANAGEMENT OF PATIENTS WITH CHRONIC COMPLICATIONS IN TYPE II DIABETES MELLITUS (DM)

Time frame – 6 hours.

Professional motivation. IDF (International Diabetes Federation) predicts that the number of people with diabetes will rise from 194 million today to more than 333 million by 2025 .Type 1 and type 2 diabetes are chronic, life-long conditions that require careful monitoring and control. Without proper management they can lead to very high blood sugar levels which can result in long-term damage to various organs and tissues: affects the heart and blood vessels and may cause fatal complications such as coronary heart disease (leading to heart attack) and stroke. Cardiovascular disease is the major cause of death in people with diabetes, accounting in most populations for 50% or more of all diabetes fatalities, and much disability. Diabetic nephropathy can result in total kidney failure and the need for dialysis or kidney transplant. Diabetes is an increasingly important cause of renal failure, and indeed has now become the single most common cause of end stage renal disease, i.e., that which requires either dialysis or kidney transplantation. Diabetic neuropathy can ultimately lead to ulceration and amputation of the toes, feet and lower limbs. Loss of feeling is a particular risk because it can allow foot injuries to escape notice and treatment, leading to major infections and amputation. Diabetic retinopathy is characterised by damage of the retina of the eye which can lead to vision loss.

Type 2 diabetes accounts for at least 90% of all cases of diabetes. The diagnosis of type 2 diabetes usually occurs after the age of 40 but can occur earlier, especially in populations with high diabetes prevalence. Type 2 diabetes can remain undetected for many years and the diagnosis is often made from associated complications or incidentally through an abnormal blood or urine glucose test. It is often, but not always, associated with obesity, which itself can cause insulin resistance and lead to elevated blood glucose levels.

Good diabetes control means keeping your blood sugar levels as close to normal as possible. This can be achieved by a combination of the following: physical activity (a goal of at least 30 minutes of moderate physical activity per day (e.g., brisk walking, swimming, cycling, dancing) on most days of the week); body weight (weight loss improves insulin resistance, blood glucose and high lipid levels in the short term, and reduces blood pressure. It is important to reach and maintain a healthy weight); healthy eating (avoiding foods high in sugars and saturated fats, and limiting alcohol consumption); avoid tobacco; monitoring for complications (includes regular foot and eye checks, controlling blood pressure and blood glucose, and assessing risks for cardiovascular and kidney disease). At present, type 1 diabetes cannot be prevented. The environmental triggers that are thought to generate the process that results in the destruction of the body's insulin-producing cells are still under investigation. Type 2 diabetes, however, can be prevented in many cases by maintaining a healthy weight and being physically active. IDF recommends that all people at high risk of developing type 2 diabetes be identified through opportunistic self-screening. People at high risk can be easily identified through a simple questionnaire to assess risk factors such as age, waist circumference, family history, cardiovascular history and gestational history. Once identified, people at high risk of diabetes should have their plasma glucose levels measured by a health professional to detect Impaired Fasting Glucose or Impaired Glucose Tolerance, both of which indicate an increased risk of type 2 diabetes. Prevention efforts should target those at risk in order to delay or avoid the onset of type 2 diabetes. Each year, 3.2 million people around the world die from complications associated with diabetes.

Study objective: to improve the skills of clinical examination of patients with diabetes mellitus, to establish the clinical diagnosis of type 2 DM and its complications.

Basic level:

1. Regulation of glucose level in blood. Pathophysiology of hyperglycemia.
2. To be able to collect complaints, case history, carry out physical examination in patients with

type 2 DM.

3. To interpret clinical, laboratory and instrumental data in chronic complications of type 2 DM.
4. To find out data inherent to chronic complications in type 2 DM.
5. To interpret side effects of oral glucose-lowering drugs, insulinotherapy.

Student has to know:

1. Risk factors that have been associated with type 2 diabetes.
2. Criteria for diagnosis of DM. The oral glucose tolerance test: indications, normal response, impaired glucose tolerance, diabetes.
3. Treatment (lifestyle interventions, oral glucose-lowering drugs, insulinotherapy).
4. What are the chronic complications of diabetes?

The main theoretical questions:

1. Diabetes mellitus: classification, aetiology, pathogenesis.
2. Laboratory findings in diabetes mellitus patients. Diagnostic significance of glycated haemoglobin (HbA_1) measurement, C-peptide, ketonuria.
3. Diagnostic criteria of insulin dependant and insulin independent diabetes mellitus.
4. Classification and diagnostic criteria for diabetic nephropathy. Treatment.
5. Classification and diagnostic criteria for diabetic micro- and macroangiopathy.
6. Classification and diagnostic criteria for diabetic rethinopathy.
7. Diabetic foot: diagnostic criteria and treatment.
8. What does good diabetes control mean? Self-monitoring of blood glucose.
9. Primary prevention for development of type 2 DM.
10. Treatment of insulin-dependent diabetes mellitus. Complications in insulinotherapy.

Assignment for self-assessment:

1. The Somogyi effect is:
   a) episode of night-time hypoglycemia resulting in high blood sugar levels;
   b) period of high blood sugar in the morning (7–9 a.m.);
   c) night hyperglycemia related to deficit of prolong insulin;
   d) resistance to insulin.

2. A 26-year-old man presents with headache, weakness, facial and crus oedema. His current medication includes insulin 34 units/daily. He has a history of diabetes for the past 6 years.

The examination reveals oedematous face, legs, pale skin, heart rate is regular, 100/min. Blood pressure is 180/100 mm Hg; respiratory rate is 20 breaths/min. His lungs are clear. The abdomen is soft without tenderness or distention. The liver spans 10 cm in the midclavicular line with a smooth edge. His weight is 58 kg, height is 176 cm. He is afebrile.

Blood testing: RBC – $3.4*10^{-12}$/l; Hb – 104 g/l; ESR – 10 mm/h; WBC – $7.2*10^{-9}$/l. Total protein – 63 g/l, urea – 6.2 mmol/l, creatinine – 98 μmol/l, bilirubin – 19 μmol/l, glucose profile – 12–7.8–10 mmol/l, cholesterol – 5 mmol/l, triglycerides – 1.5 mmol/l. Haemoglobin A_1c – 7.9%. Urine chemistry: protein 2.5 g/l.

Chest X-ray: no infiltrates.

What complication has been developed in this patient?

   a) diabetic nephropathy;
   b) kidney amyloidosis;
   c) chronic pyelonephritis;
   d) nephrotic syndrome;
   e) glomerulonephritis.

What are the main directions in treatment?

1. A 60-year-old man presented with dry mouth, paresthesia of the crus, dyspnoea on exertion that is accompanied by chest pain. Pain with radiation to neck or chest tightness lasts about 15 min. A rest or taking a pill under the tongue makes the symptoms go away.

The patient suffered from anterior wall MI 10 months ago. His current medications include ramipril 5 mg twice a day, aspirin 100 mg, metformin 500 mg bid. His social history is positive for occasional alcohol and negative for smoking. He has a history of hypertension and diabetes for the past 4 years.

The examination reveals a regular heart rate with a reduced intensity S_1 and normal S_2. Blood pressure is 160/90 mm Hg; pulse is 82 beats/min, regular, respiratory rate is 20 breaths/min. His lungs are clear. The abdomen is soft without tenderness or distention. The liver spans 10 cm in the midclavicular line with a smooth edge. There is no peripheral oedema and the pulses are intact. His weight is 88 kg, height is 176 cm.

Blood testing: RBC – $4.4*10^{-12}$/l; Hb – 134 g/l; ESR – 10 mm/h; WBC – $7.2*10^{-9}$/l; eos. – 1%, stab neutr. – 4%, segmented neutrophils – 59%, lymphocytes – 15%, monocytes – 5%. Total protein – 63 g/l, urea – 6.2 mmol/l, creatinine – 98 μmol/l, bilirubin – 19 μmol/l, fasting glucose – 7.8 mmol/l, cholesterol – 7 mmol/l, triglycerides – 2.5 mmol/l. Haemoglobin A_1c – 7.9%. Potassum 5.0 μmol/l, GFR – 80 ml/min. Urine chemistry: protein 1.5 g/l. Chest X-ray: no infiltrates, mild cardiomegaly.

What can be done to slow diabetes complications?

Answers:

1. a. 2. a, treatment: compensation of diabetes mellitus, angioprotectors use, ACE-inhibitor, insulinotherapy in adequate dose.

3. Intensive control of elevated levels of blood sugar in patients with type 1 or type 2 diabetes decreases the complications of nephropathy, neuropathy, retinopathy, and may reduce the occurrence and severity of large blood vessel diseases. Aggressive control with intensive therapy means achieving fasting glucose levels between 70–120 mg/dl; glucose levels of less than 160 mg/dl after meals; and a near normal haemoglobin A1 levels.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
6. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
7. https://reference.medscape.com/guide/medical-calculators

24. MANAGEMENT OF PATIENTS WITH ARTERIAL HYPERTENSION IN ENDOCRINE DISEASES

Time frame – 6 hours.

Professional motivation. Approximately 5% of patients with hypertension have specific causes. The case history, examination, and routine laboratory tests may identify such patients. Endocrine hypertension accounts for approx. 3% of the secondary forms of hypertension and is a term assigned to states in which hormonal derangements result in clinically significant hypertension. The most common causes of endocrine hypertension are excess production of mineralocorticoids (i.e., primary hyperaldosteronism – 0.3–1.5%), catecholamines (pheochromocytoma – 0.1–0.3%), thyroid hormone, and glucocorticoids (Cushing's syndrome). One important question in this regard is when to screen for secondary causes. The clinician should carefully screen for other cardinal signs and symptoms of Cushing's syndrome, hyper- or hypothyroidism, acromegaly, or pheochromocytoma. Hypertension in young patients and refractory hypertension (characterized by poorly controlled blood pressure on > 3 antihypertensive drugs) or those previously well controlled who become refractory to treatment should alert the physician to screen for secondary causes.

The importance of endocrine mediated hypertension resides in the fact that in most cases, the cause is clear and can be traced to the actions of a hormone, often produced in excess by a tumour such as an

aldosteronoma in a patient with hypertension due to primary aldosteronism. More importantly, once the diagnosis is made, a disease-specific targeted antihypertensive therapy can be implemented, and in some cases, surgical intervention may result in complete cure, obviating the need for life-long antihypertensive treatment.

Pheochromocytomas are uncommon; they are probably found in less than 0.1% of all patients with hypertension and in approximately two individuals per million population. In about 50% of patients with pheochromocytoma, hypertension is sustained but the blood pressure shows marked fluctuations, with peak pressures during symptomatic paroxysms. During a hypertensive episode, the systolic blood pressure can rise to as high as 300 mm Hg. In about one-third of cases, hypertension is truly intermittent. In some cases, hypertension is absent.

It now appears that up to 5–15% of patients in whom primary (essential) hypertension is diagnosed actually have primary hyperaldosteronism, with most having normal serum potassium levels.

Study objective: to improve the skills in management of patients with arterial hypertension in endocrine diseases.

Basic level:

1. Anatomy and physiology of suprarenal gland.
2. Pathogenesis of the main clinical symptoms in acromegaly.
3. Pathogenesis of the main clinical symptoms in hyperaldosteronism (Conn's syndrome).
4. Pathogenesis of the main clinical symptoms in Cushing's syndrome.
5. Pathogenesis of the main clinical symptoms in hypo- or hyperthyroidism.
6. To interpret clinical, laboratory, and instrumental data in endocrine hypertension.
7. To find out data inherent to endocrine hypertension.

Student has to know:

1. How to make program for investigation when evaluating a patient with suspected endocrine-related hypertension.

2. Differential diagnosis of endocrine hypertension (pheochromocytoma, acromegaly, Cushing's syndrome, diabetes mellitus).

The main theoretical questions:

1. Definition of secondary AH. Laboratory and instrumental methods in secondary AH.
2. Diagnosis and treatment of AH in acromegaly, hyperaldosteronism, Cushing's syndrome, hypo- or hyperthyroidism, pheochromocytoma.
3. Clinical findings, diagnosis, and emergency care in Addison's crisis.
4. Clinical findings, diagnosis, and emergency care in pheochromocytoma crisis.

Assignment for self-assessment

1. The triad headache, palpitations, and sweating in a hypertensive patient was found to have a sensitivity of 91% and specificity of 94% for:

a) pheochromocytoma;
b) hyperaldosteronism;
c) Cushing's syndrome;
d) acromegaly.

2. There are several mechanisms of blood pressure elevation in Cushing's syndrome with the exception of:

a) increased hepatic production of angiotensinogen and cardiac output by glucocorticoids;
b) reduced production of prostaglandins via inhibition of phospholipase A;
c) increased insulin resistance;
d) oversaturation of 11beta-HSD activity with increased mineralocorticoid effect through stimulation of the mineralocorticoid receptor;
e) increased peripheral resistance.

3. Screening studies for Cushing's syndrome include: a) measuring 24-h urinary free cortisol excretion on at least 2 occasions; b) performing 1 mg dexamethasone suppression test; c) checking a midnight salivary cortisol and diurnal rhythm of cortisol secretion; d) stress excersise test.

4. A 37-year-old women was admitted to the hospital with such symptoms: rounded "moon" feaces

with a plethoric appearance; truncal obesity with prominent supraclavicular and dorsal cervical fat pads; the distal extremities and fingers are usually quite slender; muscle wasting and weakness are present. The skin is thin and atrophic, with poor wound healing and easy bruising. Purple striae appear on the abdomen. Blood pressure is 165/100 mm Hg. Menstrual irregularities and hypertrichosis.

What is your primary diagnosis? Management.

5. A 40-year-old man with paroxysmal hypertension was admitted to the hospital. Tachycardia attack, tachypnoea, flushing, cold and clammy skin, severe headache, palpitation, nausea, vomiting, visual disturbances, dyspnoea, paresthesias occurred. Paroxysmal attacks are provoked by palpation of the abdomen or after emotional trauma.

What is your primary diagnosis? Management.

6. The best screening tests for primary hyperaldosteronism involve determinations of: a) plasma aldosterone concentration (normal: 1–16 ng/dL) and plasma renin activity (normal: 1–2.5 ng/mL/h); b) calculation of the plasma aldosterone/renin ratio (normal: < 25); c) plasma prostaglandins concentration.

Answers:

1. a. 2. e. 3. a, b, c.

4. Cushing's syndrome. Adrenal inhibitors such as metyrapone, mitotane, and aminoglutethimide may be used to control severe metabolic disturbances resulting from hyperfunction of the adrenal cortex. Probably surgery.

5. Pheochromocytoma. Treatment includes surgical removal of the tumour. It is usually possible to delay surgery until the patient is in optimum physical condition by the use of combination of a- and b-adrenergic blocking agents.

6. a, b.

REFERENCES

1. The diagnosis of Cushing's syndrome: an endocrine society clinical practice guideline http://www.endo-society.org/guidelines/final/upload/Cushings_Guideline. pdf
2. Case detection, diagnosis, and treatment of patients with primary aldosteronism: an endocrine society clinical practice guideline http://www.endo-society.org/ guidelines/ final/upload/Final-Standalone-PA-Guideline.pdf
3. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
4. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
5. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
6. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
7. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
8. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
9. https://reference.medscape.com/guide/medical-calculators

25. MANAGEMENT OF PATIENTS WITH METABOLIC SYNDROME

Time frame – 6 hours.

Professional motivation. Many people are unaware that they have metabolic syndrome (MS), even though the American Heart Association estimates that approximately 20–30% of the population in industrialized countries have metabolic syndrome – between 58 and 73 million men and women. Metabolic syndrome is present in about 5% of people with normal body weight, 22% of those who are overweight and 60% of those considered obese. Adults who continue to gain five or more pounds per year raise their risk of developing metabolic syndrome by up to 45%. Metabolic syndrome increases

the risk of type 2 diabetes (the common type of diabetes) anywhere from 9–30 times over the normal population. As to the risk of heart disease, studies vary, but the metabolic syndrome appears to increase the risk 2–4 times that of the normal population. Adult population with metabolic syndrome twice as likely to die from and three times as likely to have a heart attack or stroke compared with people without the syndrome.

Metabolic syndrome is associated with fat accumulation in the liver (fatty liver), resulting in inflammation. The kidneys can also be affected, as there is an association with microalbuminuria – the leaking of protein into the urine, a subtle but clear indication of kidney damage. Other problems associated with metabolic syndrome include obstructive sleep apnoea, polycystic ovary syndrome, increased risk of dementia with aging, and cognitive decline in the elderly.

Study objective: to be able to find out criteria of metabolic syndrome and determine management.

Basic level:

1. Measuring weight in relation to height: body mass index (BMI).
2. Criteria for the MS.

Student has to know:

1. The new International Diabetes Federation (IDF) definition of metabolic syndrome.
2. What is the pathogenesis of the MS?
3. How is central obesity measured?
4. How to interpret clinical, laboratory, and instrumental data in patients with MS.

The main theoretical questions:

1. How is metabolic syndrome defined?
2. What diagnostic tests should be done to diagnose MS? Indications, methodology and interpretation of glucose-tolerant test.
3. How is metabolic syndrome treated?
4. How can cardiovascular risk be prevented and treated?

Assignment for self-assessment:

1. Metabolic syndrome is diagnosed if a person has central obesity and any 2 of the following are mentioned except (according to the new IDF definition):

a) raised fasting plasma glucose ≥ 100 mg/dL (5.6 mmol/L) or previously diagnosed type 2 diabetes;

b) serum triglycerides 150 mg/dl (1.7 mmol/L) or above;

c) HDL cholesterol 40 mg/dl (1.03 mmol/L) or lower in men and 50 mg/dl (1.29 mmol/L) or lower in women;

d) systolic BP ≥ 130 or diastolic BP ≥ 85 mm Hg or treatment of previously diagnosed hypertension; e) raised creatinine level ≥ 115.6 µmol/L.

2. Central obesity defined as:

a) a waist circumference over 94 cm in men and over 80 cm in women;

b) a waist circumference over 84 cm in men and over 70 cm in women;

c) a waist circumference over 104 cm in men and over 100 cm in women.

3. A 56-year-old man had ST-elevated MI. He has no history of diabetes. The examination reveals that heart rate is regular, 90/min. Blood pressure is 150/90 mm Hg; respiratory rate is 16 breaths/min. His lungs are clear. The abdomen is soft without tenderness or distention. His weight is 58 kg, height is 176 cm.

Blood testing: RBC – $3.4*10^{-12}$/l; Hb – 134 g/l; ESR – 10 mm/h; WBC – $7.2*10^{-9}$/l. Total protein – 63 g/l, urea – 6.2 mmol/l, creatinine – 98 µmol/l, bilirubin – 19 µmol/l, fasting glucose – 4.8 mmol/l, cholesterol – 5 mmol/l, triglycerides – 1.5 mmol/l, uric acid – 233 µmol/l. Chest X-ray: no infiltrates.

Determine the grade of obesity. What risk factor should be removed in secondary prevention?

a) hyperglycemia; b) hyperuricemia; c) hypercholesterolemia; d) obesity; e) hypertension.

Answers:

1. e. 2. a. 3. grade I, d.

REFERENCES

1. http://www.idf.org/

2. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
3. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
4. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
5. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
6. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
7. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
8. https://reference.medscape.com/guide/medical-calculators

Semantic module 6. Management of patients in nephrology clinic

26. MANAGEMENT OF PATIENTS WITH URINE SEDIMENT CHANGES

Time frame – 6 hours.

Professional motivation. Proteinuria is a common finding in adults in primary care practice. Proteinuria is defined as urinary protein excretion of greater than 150 mg per day. An algorithmic approach can be used to differentiate benign causes of proteinuria from rarer, more serious disorders. Benign causes include fever, intense activity or exercise, dehydration, emotional stress, and acute illness. More serious causes include glomerulonephritis and multiple myeloma. Alkaline, dilute or concentrated urine; gross haematuria; and the presence of mucus, semen or white blood cells can cause a dipstick urinalysis to be falsely positive for protein. Of the three pathophysiologic mechanisms (glomerular, tubular, and overflow) that produce proteinuria, glomerular malfunction is the most common and usually corresponds to the urinary protein excretion of more than 2 g per 24 hours.

When a quantitative measurement of urinary protein is needed, most physicians prefer a 24-hour urine specimen. However, the urine protein-to-creatinine ratio performed on a random specimen has many advantages over the 24-hour collection, primarily convenience and possibly accuracy. Most patients evaluated for proteinuria have a benign cause. Proteinuria on initial dipstick urinalysis testing is found in as much as 17% of selected populations. Although a wide variety of conditions, ranging from benign to lethal, can cause proteinuria, fewer than 2 percent of patients whose urine dipstick test is positive for protein have serious and treatable urinary tract disorders.

Place of carrying out: class-room, wards of nephrology.

Study objective is to improve students' skills in the diagnosis of urine sediment changes and different forms of renal disorders, the management of patients with urine sediment.

Basic level:

1. Aetiology, pathogenesis of renal disorders with urine sediment changes.
2. Clinical, laboratory methods of nephritic syndromes diagnosis (estimation of urine sediment changes).

Student has to know:

1. How to make physical examination of patients with renal disease.
2. How to interpret results of the physical, laboratory and instrumental data with following differential diagnosis.
3. Who is at risk for proteinuria?
4. Peculiarities of the urine sediment changes in different forms of the renal disorders.
5. How to make program for investigation when evaluating a patient with suspected renal disorder.

The main theoretical questions:

1. Peculiarities of the urine sediment changes in acute and chronic glomerulonephritis,

pyelonephritis, tuberculosis, amyloidosis, renal tumour, nephropathy of different origin.

2. What diseases are accompanied by macrohaematuria, isolated haematuria?

3. What diseases are accompanied by proteinuria, leukocyturia?

4. Involvement of the kidneys in the connective tissue diseases: Rheumatoid arthritis. Systemic lupus erythematosus. Systemic sclerosis. Sjogren's syndrome. Polyarteritis nodosa.

5. How is proteinuria (haematuria) treated?

Assignment for self-assessment

1. A female college student complains of dysuria and pollakiuria, nausea, vomiting, fever. Urinalysis reveals 28 to 30 WBCs per high-power field and numerous gram-negative bacteria. What diagnosis do you suspect?

2. Nephritic syndrome is characterized by the mentioned signs except:

a) proteinuria less than 3.5 g/24-hours;
b) erythrocyturia (more than 2000/ml in Nechiporenko test);
c) leukocyturia (more than 4000/ml in Nechiporenko test);
d) erythrocyturia (more than 3000/ml in Nechiporenko test);
e) casts.

3. A 24-year-old farmer has had acute tonsillitis. In 2 weeks he noticed facial oedema on waking, elevated temperature of 37.7 °C, weakness. BP is 130/75mm. Urinalysis reveals: specific gravity – 1026, protein – 1.66 g/l, 28 to 30 RBCs per high-power field and numerous casts. What diagnosis do you suspect?

a) acute glomerulonephritis, nephrotic syndrome;
b) acute glomerulonephritis, isolated urinary syndrome;
c) chronic glomerulonephritis, exacerbation;
d) acute pyelonephritis;
e) chronic pyelonephritis, exacerbation.

Answers:

1. Acute pyelonephritis.

2. d. 3. b.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
6. Mayerle, J. Chronic Pancreatitis [Електронний ресурс] / J. Mayerle, G. Beyer. — 2017.
7. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
8. https://reference.medscape.com/guide/medical-calculators
9. https://kdigo.org/guidelines/

27. MANAGEMENT OF PATIENTS WITH OEDEMATOUS SYNDROME

Time frame – 6 hours.

Professional motivation. Six factors can contribute to the formation of oedema: increased hydrostatic pressure; reduced oncotic pressure within blood vessels; increased tissue oncotic pressure; increased blood vessel wall permeability, e.g., inflammation; obstruction of fluid clearance via the

lymphatic system; changes in the water-retaining properties of the tissues themselves. Raised hydrostatic pressure often reflects retention of water and sodium by the kidney.

Swelling of the the ankles is a common complaint in general practice, affecting up to 30% of the population. It is important to understand that while the oedema itself can be physically limiting, ascertaining the underlying cause is important so that treatment can be targeted specifically to the condition causing oedema.

Generalized oedema can occur in a variety of disorders, including heart failure, cirrhosis (where ascites is usually most prominent), the nephrotic syndrome, and renal failure; when massive, the excess fluid accumulation is called anasarca. Oedematous patients generally respond to the combination of dietary sodium restriction and diuretic therapy, usually with a loop diuretic. Some patients, however, are resistant to this regimen. A variety of factors can account for persistent fluid retention, including inadequate diuretic dose, excess sodium intake, delayed intestinal absorption of oral diuretics, decreased diuretic excretion into the urine, and increased sodium reabsorption at sites in the nephron other than those inhibited by the diuretic. In addition, nonsteroidal antiinflammatory drugs should be discontinued, if possible, since diminished synthesis of vasodilator and natriuretic prostaglandins can impair diuretic responsiveness.

Starting of syndromes of oedema and ascitis means the unfavourable streaming of disease, transferring in a stage of decompensation of cardiovascular, cardiopulmonary, liver, and renal failure. Clinical signs of oedema and ascitis syndromes at various diseases are the same, that results in some difficulties for proper diagnosis and adequate therapy. Therefore knowledge of mechanisms of oedema and ascitis and differential diagnostics is important in practical activity of the doctor.

Place of carrying out: class-room, wards of nephrology.

Study objective is determination of patients' management with oedematous syndrome.

Basic level:

1. To be able to collect complaints, case history, carry out objective examination of patients with renal diseases.
2. To interpret instrumental and laboratory data in patients with renal diseases.
3. Determination of free fluid in abdominal cavity and to distinguish ascites from other disorders with increased abdomen.

Student has to know:

1. Diseases which are accompanied by oedema.
2. How to make program for investigation when evaluating a patient with oedema.
3. How to differentiate oedema and ascites of various origin.

The main theoretical questions:

1. Aetiology and mechanisms of oedema.
2. Diagnostic algorithm in oedematous syndrome.
3. Laboratory investigations of the renal function: routine urine analysis, urinary examination according to Zimnitsky, and Nechyporenko. Valuation of the daily proteinuria, renal excretion.
4. Instrumental investigations in oedematous syndrome. Renal biopsy.
5. Treatment of oedema in cardiac failure, renal failure.
6. Treatment of oedematous syndrome in liver cirrhosis.
7. Treatment of oedematous syndrome in venous and lymphatic congestion.

Assignment for self-assessment

1. A 44-year-old man is referred to you for evaluation and treatment of recurrent renal colic and passage of renal stones. What kind of examination will you choose?

2. A 30-year-old man complains of headache, high BP, oedema on different parts of the body. He has been sick for 7 years. BP is elevating for the past 4 years. He is noted on heamaturia periodically. Physical examination reveals tachycardia 96/min, BP is 190/130mm Hg. Heart sounds are weak, rhythmical, accentuated S_2 above aorta. Blood testing: RBC – $3.4*10^{-12}$/l; Hb – 114 g/l; ESR – 10 mm/h; WBC – $7.2*10^{-9}$/l; eos. – 1%, stab neutr. – 4%, segmented neutrophils – 69%, lymphocytes – 19%, monocytes – 5%. Total protein – 63 g/l, urea – 6.2 mmol/l, creatinine – 198 μmol/l, bilirubin – 19 μmol/l, fasting glucose – 4.8 mmol/l, cholesterol – 7 mmol/l, triglycerides – 2.5 mmol/l. GFR

80 ml/min. Urinalysis reveals: specific gravity is 1015, protein – 2.6 g/l, 12 to 14 RBCs per high-power field, 4–6 WBCs per high-power field and numerous casts. Urine chemistry: protein – 3.5 g/l. What is the most likely diagnosis?

a) chronic glomerulonephritis;
b) kidney amyloidosis;
c) kidney stones;
d) chronic pyelonephritis.

3. Effectiveness of diuretics in chronic heart failure can be decreased in therapy by:

a) diclofenac;
b) ibuprofen;
c) prednisolon;
d) everything is right.

4. What diuretic has ototoxic action?

a) verospiron;
b) hypothiazide;
c) furosemide;
d) triamteren.

5. A 40-year-old man was admitted at the nephrological department with diagnosis – chronic glomerulonephritis, nephrotic syndrome, hypertensive stage. Objectively: oedema on different parts of the body, pleural effusion and ascites. What pathogenic treatment should be prescribed in the first place?

a) antibiotics;
b) antiaggregants;
c) prednisolon;
d) antisensitizer;
e) diuretics.

Answers:

1. Intravenous pyelography.
2. b. 3. d. 4. c. 5. c.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
6. Mayerle, J. Chronic Pancreatitis [Електронний ресурс] / J. Mayerle, G. Beyer. — 2017.
7. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
8. https://reference.medscape.com/guide/medical-calculators
9. https://kdigo.org/guidelines/

28. MANAGEMENT OF PATIENTS WITH CHRONIC RENAL FAILURE

Time frame – 6 hours.

Professional motivation. Patients with chronic kidney disease stages 1–3 are generally asymptomatic; clinically manifestations typically appear in stages 4–5. Early diagnosis and treatment of the underlying cause and/or institution of secondary preventive measures is imperative in patients

with chronic kidney disease. These may delay, or possibly halt, progression.

The social and economic consequences of CRF are considerable. In the UK 85–95 new patients per million of the adult population are accepted for long-term dialysis treatment each year; the availability of dialysis and transplantation has transformed the outlook for such patients. The incidence of CRF is much higher in some other countries due to differences in regional and racial incidences of disease as well as because of differences in medical practice.

Dialysis and transplantation can be considered as highly effective forms of treatment, with a 5-year survival of approximately 80% for home haemodialysis, 80% following renal transplantation, 60% for hospital haemodialysis and 50% for continuous ambulatory peritoneal dialysis. These figures are not directly comparable because of patient selection-many older patients and those with systemic diseases such as diabetes mellitus are treated by CAPD. They also conceal a very large increase in death rates from certain causes, but particularly vascular disease, in comparison with an age-matched population. However, they indicate how the prognosis of end-stage renal disease is now much better than that of many other potentially fatal diseases.

Place of carrying out: class-room, wards of nephrology.

Study objective is to improve students' skills in chronic renal failure (CRF) diagnosis using clinical, laboratory, and instrumental signs and to determine management.

Basic level:

1. Pathogenesis of leading clinical syndromes of CRF.
2. To be able to collect complaints, case history, carry out objective examination of patients with CRF.
3. To interpret instrumental and laboratory data in patients with CRF.

Student has to know:

1. How to discover signs inherent to CRF.
2. How to determine the stage of the CRF.
3. How to make program for investigation of patients with CRF.
4. Management of patients with CRF.

The main theoretical questions:

1. Aetiology and pathophysiology of CRF.
2. Classification of CRF. Stages of chronic kidney disease.
3. Peculiarities of clinical manifestations of CRF due to diseases. Potassium and calcium disorders. Haematological abnormalities.
4. Treatment of CRF: principles of the diet. Correction of electrolytes abnormalities.
5. Treatment of hypertensive syndrome, anaemia, infectious complications.
6. Renal replacement therapy in CRF. Indications to haemodialysis, continuous ambulatory peritoneal dialysis, and transplantation. Immunosuppressive treatment after transplantation. Contraindications to haemodialysis.
7. Renal involvement in systemic disorders (diabetes mellitus, systemic vasculitis, SLE).
8. Adult polycystic kidney disease.

Assignment for self-assessment

1. A 50-year-old man with end-stage of CRF due to chronic glomerulonephritis is maintained on long-term haemodialysis three times each week. He has came to the dialysis unit with a 4.5 kg weight gain since his last dialysis 2 days ago. He is moderately short of breath and his blood pressure is 195/125 mm Hg. What is the most appropriate management of his hypertension at this point?

2. Pulmonary haemorrhage has appeared in patient with progressive nephritis and CRF. What is the most likely diagnosis?

a) SLE with renal involvement;
b) bronchiectasis;
c) pulmonary oedema;
d) pulmonary infarction;
e) Goodpasture's syndrome.

3. Indications to haemodialysis in CRF:
   a) glomerular filtration < 5 ml/min.;
   b) stable decrease of daily urine less than 700 ml;
   c) hypercreatinaemia up to 1100–1300 μmol;
   d) symptoms of pericarditis, encephalopathy, neuropathy;
   e) all mentioned above.
4. The earliest symptom of CRF is:
   a) high blood presure;
   b) hypostenuria;
   c) oedema;
   d) polyuria;
   e) anaemia.

Answers:

1. Hypertension in the end-stage of renal failure is usually related to an increase in extracellular fluid volume. The best management is the expeditious removal of fluid by ultrafiltration dialysis. Such treatment usually results in normalization of the blood pressure and relief of circulatory congestion.

2. e. 3. e. 4. a.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
6. Mayerle, J. Chronic Pancreatitis [Електронний ресурс] / J. Mayerle, G. Beyer. — 2017.
7. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
8. https://reference.medscape.com/guide/medical-calculators
9. https://kdigo.org/guidelines/

29. MANAGEMENT OF PATIENTS WITH RENAL ARTERIAL HYPERTENSION

Time frame – 6 hours.

Professional motivation. The most common cause of secondary hypertension is renal artery stenosis. The most common cause of narrowing is atherosclerosis. Blockage of the renal arteries causes the kidney to increase production of the hormone renin. Increased levels of renin in the body cause a cascade of events to occur that result in peripheral vasoconstriction and fluid retention, causing an increase in blood pressure. Renovascular hypertension is particularly dangerous to the heart because of the direct toxic effects of the “renin-angiotensin system” on the heart muscle. In addition, as the renal arteries become narrow, the kidney is deprived of its normal blood flow, which can lead to kidney failure.

Overall, about two thirds of cases of renovascular hypertension are caused by atherosclerosis and one third by fibromuscular dysplasia. Atherosclerosis is more common among men > 50 and affects mainly the proximal one third of the renal artery. Fibromuscular dysplasia is more common among younger patients (usually women) and usually affects the distal two third of the main renal artery and the branches of the renal arteries. Rarer causes include emboli, trauma, inadvertent ligation during surgery, and extrinsic compression of the renal pedicle by tumours.

Renovascular hypertension is characterized by high cardiac output and high peripheral resistance.

Renovascular hypertension is usually asymptomatic. A systolic-diastolic bruit in the epigastrium, usually transmitted to one or both upper quadrants and sometimes to the back, is almost pathognomonic, but it is present in only about 50% of patients with fibromuscular dysplasia and is rare in patients with renal atherosclerosis.

Renovascular hypertension should be suspected if diastolic hypertension develops abruptly in a patient < 30 or > 50; if new or previously stable hypertension rapidly worsens within 6 mo; or if hypertension is initially very severe, associated with worsening renal function, or highly refractory to drug treatment.

Prompt diagnosis and timely intervention done by a skilled vascular surgeon can significantly decrease target organ damage and potentially cure high blood pressure due to renal artery disease.

Place of carrying out: class-room, wards of nephrology.

Study objective is to assign patients' management with renal arterial hypertension.

Basic level:

1. Student should be able to collect complaints, case history, carry out objective examination of patients with arterial hypertension.
2. To interpret instrumental and laboratory data in patients with arterial hypertension.
3. Mechanism of action, pharmacology/pharmacokinetics, side effects of antihypertensive drugs.

Student has to know:

1. Diseases which are accompanied by secondary arterial hypertension.
2. How to make program for investigation when evaluating a patient with arterial hypertension.
3. How to differentiate diseases with arterial hypertension.
4. How to put presumptive diagnosis, to prescribe dietary, medical treatment for the patients with renal hypertension.

The main theoretical questions:

1. Clinical, laboratory, and instrumental diagnostic methods for revealing renal hypertension.
2. Pathogenesis of renal hypertension.
3. Classification of renal hypertension.
4. Clinical manifestation of renal hypertension.
5. Complications in renal hypertension and their pathogenesis.
6. Management of patients with renal hypertension.

Assignment for self-assessment

1. The "gold standard" (the definitive test) in diagnosis of renal artery stenosis is:
   a) arteriography;
   b) duplex ultrasonography;
   c) radionuclide imaging.
2. Renovascular hypertension should be suspected if:
   a) diastolic hypertension develops abruptly in a patient < 30 or > 50;
   b) new or previously stable hypertension rapidly worsens within 6 months;
   c) hypertension is initially very severe, associated with worsening renal function;
   d) hypertension is highly refractory to drug treatment;
   f) all of the above signs are correct.
3. A class of drugs that cause vasodilation and are used to treat hypertension and heart failure:
   a) ACE inhibitors;
   b) glycosides;
   c) potassium and sodium channel blockers (amiodarone).
4. Adverse effect of all ACE inhibitors:
   a) retention of potassium;
   b) retention of sodium;
   c) hypokalaemia;
   d) retention of catecholamine.
5. ACE inhibitors should be used with caution in patients with:

a) aortic valve stenosis or cardiac outflow obstruction;
b) congestive heart failure;
c) prevention of nephropathy in diabetes mellitus.

6. The most common cause of renal artery stenosis is:
a) atherosclerosis;
b) increased production of the hormone rennin;
c) extrinsic compression of the renal pedicle by tumours.

Answers:

1. a. 2. f. 3. a. 4. a. 5. a. 6. a.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
6. Mayerle, J. Chronic Pancreatitis [Електронний ресурс] / J. Mayerle, G. Beyer. — 2017.
7. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
8. https://reference.medscape.com/guide/medical-calculators
9. https://kdigo.org/guidelines/

30. MANAGEMENT OF NEPHROTIC SYNDROME

Time frame – 6 hours.

Professional motivation. Nephrotic syndrome is caused by different disorders that damage the kidneys. This damage leads to the release of too much protein in the urine. These include kidney diseases such as minimal-change nephropathy, focal glomerulosclerosis, and membranous nephropathy. Nephrotic syndrome can also result from systemic diseases that affect other organs in addition to the kidneys, such as diabetes, amyloidosis, and lupus erythematosus.

Nephrotic syndrome may affect adults and children of both sexes and of any race. It may occur in typical form, or in association with nephritic syndrome. About 2 in every 10,000 people experience nephrotic syndrome. Nephrotic syndrome prevalence is difficult to establish in adults because the condition is usually a result of an underlying disease.

The main complications of nephrotic syndrome are hypovolaemia, infection (due to leakage of immunoglobulins, encapsulated bacteria such as *Haemophilus influenzae* and *Streptococcus pneumoniae* can cause infection) and thrombosis (due to leak of anti-thrombin 3, which helps prevent thrombosis. This often occurs in the renal veins. Treatment is with oral anticoagulants (not heparin as heparin acts via anti-thrombin 3 which is lost in the proteinuria so it will be ineffective.) Hypercoagulopathy due to extravasation of fluid from the blood vessels (oedema) is also a risk for venous thrombosis.

Treatment for nephrotic syndrome and its complications appear to have reduced the morbidity and mortality once associated with the syndrome. From the therapeutic perspective, nephrotic syndrome may be classified as steroid sensitive, steroid resistant, steroid dependent, or frequently relapsing. Currently, the prognosis for patients with primary nephrotic syndrome depends on its cause. Poor patient response to steroid therapy may predict a poor outcome. Only approximately 20% of patients with focal glomerulosclerosis undergo remission of proteinuria; an additional 10% improve but remain proteinuric. Many patients experience frequent relapses, become steroid-dependent, or become steroid-

resistant. End-stage renal disease develops in 25–30% of patients with focal segmental glomerulosclerosis by 5 years and in 30–40% of these patients by 10 years.

Nephritic syndrome is a collection of signs associated with disorders affecting the kidneys, more specifically glomerular disorders. It is characterized by having small pores in the podocytes of the glomerulus, large enough to permit proteins and red blood cells to pass into the urine. By contrast, nephrotic syndrome is characterized by only proteins (proteinuria) moving into the urine. Both nephritic syndrome and nephrotic syndrome result in hypoalbuminemia. The outlook depends on the disease that is causing nephritis. When the condition improves, symptoms of fluid retention (such as swelling and cough) and high blood pressure may go away in 1 or 2 weeks. However, urine tests may take months to return to normal.

Children tend to do better than adults and usually recover completely. Only rarely do they develop complications or progress to chronic glomerulonephritis and chronic kidney disease. Adults do not recover as well or as quickly as children. Although it is unusual for the disease to return, at least one-third of adults whose disease does return will develop end-stage kidney disease and may need dialysis or kidney transplant.

Place of carrying out: class-room, wards of the nephrology.

Study objective is to assign patients' management with nephrotic syndrome.

Basic level:

1. Student should be able to collect complaints, case history, carry out objective examination of patients with nephrotic syndrome.
2. To interpret instrumental and laboratory data in patients with nephrotic syndrome.
3. Mechanism of action, pharmacology/pharmacokinetics, side effects of glucocorticoids.
4. Mechanism of action, pharmacology/pharmacokinetics, side effects of anticoagulants.
5. Mechanism of action, pharmacology/pharmacokinetics, side effects of antiaggregants.

Student has to know:

1. Diseases which are accompanied by nephrotic syndrome.
2. How to make program for investigation when evaluating a patient with nephrotic syndrome.
3. How to differentiate disease with nephrotic syndrome.
4. How to put presumptive diagnosis. To prescribe dietary, medical, and physiotherapeutic treatment of the nephrotic syndrome due to the form of the disease.

The main theoretical questions:

1. Clinical, laboratory, and instrumental diagnostic methods for revealing nephrotic syndrome.
2. Pathogenesis of the nephrotic proteinuria, hypoalbuminemia, hyperlipidemia.
3. Classification of the nephrotic syndrome.
4. Clinical manifestation of nephrotic syndrome and pathogenesis of nephrotic oedema.
5. Complications in nephrotic syndrome and their pathogenesis.
6. Management of patients with nephrotic syndrome.
7. Treatment of the immune-inflammatory renal diseases (glomerulonephritis): dietotherapy. Diuretics. Glucocorticoids. Immunosuppressive therapy. Heparinotherapy. Platelet aggregation inhibitor. Angiotensin-converting enzyme (ACE) inhibitors. Physiotherapy and phytotherapy.
8. Preventive and sanatorium-resort therapy in the renal disorders.

Assignment for self-assessment

1. A 49-year-old man has been ill with chronic osteomyelitis for 10 years after crus fracture. Nephrotic syndrome appeared 3 years ago. He died of uraemia. During dissection: dense and white kidneys, with scars in the cortical layer, greasy glitter on cross-section. What pathology had developed?

a) chronic glomerulonephritis;
b) primary amyloidosis;
c) secondary amyloidosis;
d) chronic pyelonephritis.

2. A 46-year-old woman was admitted to the nephrological department with oedema on crus, face, high BP (160/100 mm Hg). Urine chemistry: protein 3.8 g/l. What is the most appropriate pathogenic

treatment for this patient?

a) antibiotics;
b) corticosteroids;
c) diuretics;
d) Ca-channel blockers;
e) NSAID.

Answers:

1. c. 2. b.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
6. Mayerle, J. Chronic Pancreatitis [Електронний ресурс] / J. Mayerle, G. Beyer. — 2017.
7. https://empendium.com/mcmtextbook/ 2
8. https://www.mdcalc.com/
9. https://reference.medscape.com/guide/medical-calculators
10. https://kdigo.org/guidelines/

31. MANAGEMENT OF PATIENTS WITH ANEMIA

Time frame – 6 hours.

Professional motivation. Anaemia is a serious global public health problem that particularly affects young children and pregnant women. WHO estimates that 42% of children less than 5 years of age and 40% of pregnant women worldwide are anemic. Anemia contributes to increased morbidity and mortality, decreased work productivity, and impaired neurological development.

Many diseases are associated with anemia through multiple mechanisms, including disease-specific effects on blood loss, hemolysis or erythropoiesis, through the effects of inflammation on iron metabolism. The presence of an inappropriately low reticulocyte count for the degree of anemia is used clinically to indicate conditions due to nutritional deficiencies, decreased erythropoietin levels, aplastic anemia, or inherited bone marrow failure syndromes.

Place of carrying out: class-room, wards at the hematology department.

Study objective is to assign patients' management with anemia.

Basic level:

To understand the process of formation of blood cells

To understand normal iron metabolism, how iron defi ciency may arise and how to investigate it

To understand the pathophysiology and typical laboratory features of the anaemia of chronic disease

To understand the normal metabolism of vitamin B12 and folic acid, and to appreciate how megaloblastic anaemia may arise

To understand the pathophysiology of sickle cell anaemia, the mechanisms by which the thalassaemias arise

Student has to know:

Causes of microcytic, normocytic and macrocytic anaemia

Mechanisms by which anaemia may arise

Morphological classification of anaemia.

Difference between intravascular and extravascular haemolysis, and to recognise the laboratory features of each

The role of autoantibodies in the production of haemolytic anaemias and to know the types of disease with which they are associated

Clinical features and laboratory findings indicative of haemoysis.

To appreciate the clinical presentations and complications of thalassaemia

The main theoretical questions:

1. Diagnostic and differential diagnostic criteria of iron deficiency and B12 deficiency anemia.
2. Instrumental and laboratory methods of examination of iron deficiency and B12 deficiency anemia.
3. Diagnostic criteria and management of patients with anaemia of chronic disease. Differential diagnosis of microcytic anemias.
4. The management of patients, non-pharmacological and pharmacological treatment. Indications for blood transfusion. Evidence-based treatment standards.
5. Diagnostic criteria and management of patients with acquired hemolytic anemia.
6. Diagnostic criteria and management of patients with aplastic anemia.
7. Primary and secondary prevention.
8. Prognosis and working-capacity.
9. Management of pregnant women with anemia.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
6. Villatoro, V. A Laboratory Guide to Clinical Hematology [Електронний ресурс] / V. Villatoro, M. To. — 1st ed. — University of Alberta, 2018. — 302 p.

Topic 29. Management of patient with leukemoid reaction and leukemia.

Time frame – 6 hours.

Professional motivation. Leukemoid reaction denotes pronounced neutrophilia (>40,000 cells/ml) in acute inflammatory reaction that may be mistaken for leukemia, especially chronic myeloid leukemia.

Leukemias are a group of malignant disorders that present with increased numbers of leucocytes in the blood and/or bone marrow. Leukemia types vary in pathogenesis, origin, incidence, and prognosis. Among acute leukemia types, acute lymphoblastic leukemia (ALL) is frequently diagnosed in children and young adults, with incidence peaks between 2 and 5 years of age, whereas acute myeloid leukemia (AML) is the most common acute type in adults. Although the risk factors contributing to leukemia have been extensively investigated, the current understanding of leukemia tumorigenesis remains limited. Previous studies have reported that exposure to ionizing radiation, herbicides and pesticides, and radon is associated with an increased risk of leukemia.

The geographic distribution of leukemia burden is patterned by country-level development, with age-standardized incidence, and mortality higher in more developed countries. Survival from AML varies substantially by age, with dramatic declines observed for older patients. For those diagnosed before age 65, overall 5-yr relative survival is 45.6%, compared to 7.1% for those diagnosed at age 65 or older. CML has a moderate prognosis, with 5-yr relative survival at 68.7%. Survival from CML underwent drastic improvements after the introduction of the first tyrosine kinase inhibitor (TKI), imatinib mesylate (Gleevec), in 2001. Prior to this, the prognosis was poor, with overall relative survival <50% for patients within 3 yr of diagnosis.

Place of carrying out: class-room, wards at the hematology department.

Study objective is to assign patients' management with leukemia.

Basic level: To understand the process of formation of blood cells

To understand the meaning of the terms leucopenia, neutropenia, leucocytosis, lymphopenia and lymphocytosis.

The definition, main causes, classification of leukemia.

Palpation of peripheral lymph nodes;

Inspection of abdomen, superficial palpation of abdomen, deep sliding palpation of abdomen, percussion of liver and spleen.

Flow cytometry: a technique for identification of cells in suspension

Student has to know:

the identification of mean clinical syndromes in acute and chronic leukemias;

the development of treatment plan of patients with acute and chronic leukemias;

the interpretation of laboratory findings in case of leukemia;

the clarification of differential diagnosis;

the drugs prescription to patients with leukemia.

The main theoretical questions:

1. Differential diagnosis of leukemia and leukemoid reaction.
2. Diagnostic criteria and management of patients with acute lymphoblastic leukemia, acute myeloblastic leukemia.
3. Principles of differentiated evidence-based treatment.
4. Management of patients with chronic myeloid leukemia, chronic lymphocytic leukemia.
5. Bone marrow transplantation. Supportive therapy.
6. Primary and secondary prevention. Evaluation of prognosis and working-capacity.
7. Complications on the background of treatment of hematological diseases.
8. Management of a patient with tumor lysis syndrome, anemia caused by chemotherapy, with febrile neutropenia.
9. Management of a patient with extravasation at the site of chemotherapy.
10. Management of a patient with dyspeptic disorders on the background of chemotherapy.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
6. Villatoro, V. A Laboratory Guide to Clinical Hematology [Електронний ресурс] / V.

Villatoro, M. To. — 1st ed. — University of Alberta, 2018. — 302 p.
7. https://www.esmo.org/guidelines/haematological-malignancies

Topic 30. Management of patient with polycythemia.

Time frame – 6 hours.

Professional motivation. Polycytemia Vera (PV) is a Philadelphia chromosome–negative myeloproliferative neoplasm as myelofibrosis and essential thrombocythemia. PV is characterized by clonal stem-cell proliferation of red blood cells, white blood cells, and platelets. Increased RBC mass results in hyperviscosity of the blood, increased risk for thrombosis, and a shortened life expectancy. Effective management of PV is essential, given the risk for morbidity and mortality, complexity associated with diagnosis and treatment, and overall impact on patients' quality of life. The incidence of PV is higher among men than among women in all races and ethnicities. Approximately 96% of patients with PV have a mutation of the Janus kinase 2 (*JAK2*) gene. *JAK2* is involved in intracellular signaling in PV progenitor cells, a process that occurs after exposure to cytokines to which these cells are hypersensitive.

Place of carrying out: class-room, wards at the hematology department.

Study objective is to assign patients' management with polycytemia.

Basic level: To understand the process of formation of blood cells

Assessment of clinical and laboratory features of PV.

Student has to know: Clinical symptoms and acute events associated with PV (thrombotic complications, secondary cancer)

That a single mutation in *JAK2* gives rise to at least 3 different disease phenotypes—PV, myelofibrosis and essential thrombocythemia

Diagnostic algorithm for PV.

The main theoretical questions:

1. Diagnostic criteria for polycythemia (WHO, 2016). JAK2 mutation screening
2. Differential diagnosis between secondary polycythemia and PV.
3. Differential diagnosis of PV with chronic myelogenous leukemia, essential thrombocythemia, osteomyelofibrosis.
4. Evidence-based treatment of PV. Indications for phlebotomy. Cytoreductive therapy.
5. Prognosis (survival and risk of transformation to myelofibrosis and acute myeloid leukaemia). Working-capacity.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
6. Villatoro, V. A Laboratory Guide to Clinical Hematology [Електронний ресурс] / V. Villatoro, M. To. — 1st ed. — University of Alberta, 2018. — 302 p.

Topic 31. Management of patient with lymphadenopathy.

Time frame – 6 hours.

Professional motivation. Lymphomas are more common in males and in older people. Marked variations in racial incidence, histology and immunological subtypes occur around the world. The

aetiology of lymphomas is generally unknown, although certain types are associated with specific infectious agents and chronic antigen stimulation. Autoimmune disorders such rheumatoid arthritis and Hashimoto's thyroiditis are associated with a higher incidence of lymphoma. About 60% of lymphomas involve the lymph nodes; the remaining 40% may involve almost any organ of the body.

Place of carrying out: class-room, wards at the hematology department.

Study objective is to assign patients' management with lymphadenopathy.

Basic level: To have a basic understanding of the structure of lymph nodes

To understand the term 'lymphoma' and the difference between Hodgkin lymphoma and non-Hodgkin lymphoma

To understand the basis for the classification of lymphoma

Inspection of abdomen, superficial palpation of abdomen, deep sliding palpation of abdomen, percussion of liver and spleen.

Student has to know: To understand the difference between high- and low-grade lymphoma.

Clinical manifestation of lymphomas.

To be familiar with the principles of staging and to have an awareness of diagnostic techniques used in lymphoma

The main theoretical questions:

1. The main causes of lymphadenopathy.
2. Differential diagnosis of Hodgkin's and non-Hodgkin's lymphoma, lymph node enlargement in the case of other diseases (tuberculosis, sarcoidosis, metastasis of malignant tumors, SLE, etc.).
3. The evidence-based treatment strategy, non-pharmacological and pharmacological treatment.
4. Primary and secondary prevention.
5. Prognosis and working capacity.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
6. Villatoro, V. A Laboratory Guide to Clinical Hematology [Електронний ресурс] / V. Villatoro, M. To. — 1st ed. — University of Alberta, 2018. — 302 p.
7. https://www.esmo.org/guidelines/haematological-malignancies

Topic 32. Management of patient with purpura.

Time frame – 6 hours.

Professional motivation. The incidence of thrombotic thrombocytopenic purpura is about 4–6 per million people per year. Idiopathic thrombotic thrombocytopenic purpura occurs more often in women and African-American people, while the secondary forms do not show this distribution. Pregnant women and women in the postpartum period accounted for a notable portion (12–31%) of the cases in some studies; thrombotic thrombocytopenic purpura affects approximately 1 in 25,000 pregnancies.

The mortality rate is approximately 95% for untreated cases, but the prognosis is reasonably favourable (80–90% survival) for patients with idiopathic thrombotic thrombocytopenic purpura diagnosed and treated early with plasmapheresis.

Place of carrying out: class-room, wards at the hematology department.

Study objective is to assign patients' management with purpura.

Basic level: To understand the process of formation of blood cells

To know the function of platelets and the relationship between the platelet count in peripheral blood and the extent of abnormal bleeding

To know the main sequence of events in the coagulation pathways

To understand normal fibrinolysis and the principles of fibrinolytic therapy

To know the principles underlying the prothrombin time (PT), activated partial thromboplastin time (APTT) and thrombin time (TT)

To know the effects of vitamin K deficiency and liver disease on the clotting mechanisms

Student has to know:

About the diseases associated with (i) a failure of platelet production and (ii) a shortened platelet lifespan, especially immune thrombocytopenic purpura (ITP) To know the principles of investigation of a patient suspected of having a haemostatic defect

To know the mode of inheritance, clinical presentation, method of diagnosis and principles of treatment of haemophilia A (factor FVIII deficiency), haemophilia B (factor IX deficiency) and von Willebrand disease

To know the alterations in the haemostatic and fibrinolytic mechanisms associated with disseminated intravascular coagulation (DIC) and the causes of DIC

The principles of anticoagulant therapy with unfractionated heparin, low molecular weight heparin and warfarin and to know about the laboratory control of such therapy

The natural anticoagulant mechanisms in blood and some of the prothrombotic states (thrombophilia)

The main theoretical questions:

1. Causes of thrombocytopenia (reduced platelet production, shortened platelet survival, increased splenic pooling).
2. Diagnostic criteria and evidence-based treatment of ITP.
3. Secondary autoimmune thrombocytopenic purpura.
4. Prognosis and working capacity.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
6. Villatoro, V. A Laboratory Guide to Clinical Hematology [Електронний ресурс] / V. Villatoro, M. To. — 1st ed. — University of Alberta, 2018. — 302 p.

Module 4
EMERGENCIES IN INTERNAL MEDICINE CLINIC

Semantic module 1. Emergencies in cardiology

1. EMERGENCY TREATMENT OF THE HYPERTENSIVE CRISES

Time frame – 6 hours.

Professional motivation. 60 million US inhabitants suffer from hypertension. The vast majority of these patients have essential hypertension. Moreover, a large number of affected individuals are unaware of their hypertension. Three quarters of those affected do not have their BP well controlled. Fewer than 1% of these patients will develop one or multiple episodes of hypertensive crises. The incidence of hypertensive crises is higher among African Americans and the elderly. The majority of patients presenting with hypertensive crises have previously received a diagnosis of hypertension, and many have been prescribed antihypertensive therapy with inadequate BP control. The incidence of postoperative hypertensive crises varies depending on the population examined, being reported in 4 to 35% of patients shortly after the surgical procedure.

Place of carrying out: class-room, wards of the cardiology and rheumatology departments, department of functional diagnostics.

Study objective is to improve students' skill to do clinical examination of the patients with acute coronary syndrome, to find out clinical and laboratory manifestations, to group them into syndromes, to make the diagnosis, and indicate the treatment.

Basic level: examination of patients with hypertension.

Student has to know:

1. Definition of the hypertensive crises.
2. Variants of the hypertensive crises.
3. How to give the emergency to the patients with hypertensive crises and to determine future tactics of management.

The main theoretical questions:

1. Criteria for hypertensive crises.
2. Classification of hypertensive crises.
3. The clinical manifestations of hypertensive crises
4. Differential programs of treatment depending on type of hypertensive crises.
5. Antihypertensive drugs used in hypertensive crises: dose, side effects.
6. Treatment of hypertensive crises in special situations: acute aortic dissection, acute pulmonary oedema, acute myocardial ischaemia, acute renal failure.

Assignment for self-assessment

A 58-year-old woman presented to the emergency department complaining of worsening occipital headache and confusion. She reported experiencing numbness and weakness involving the right side of her body as well as blurry vision over the past 12 hours. Her past medical history: hypertension, bilateral renal artery stenosis, hyperlipidemia.

On arrival, her blood pressure was 200/130 mm Hg. On physical examination, she was confused. Papilloedema was seen on fundoscopic examination. She had mild motor weakness in the right upper extremity. Laboratory examinations revealed the following: serum creatinine was 2.5 mg/dL (baseline creatinine – 1.5 mg/dL). Electrocardiogram revealed left ventricular hypertrophy by voltage criteria and nonspecific ST-T wave abnormalities in the lateral leads. Computed tomography scan of the head without contrast revealed diffuse bilateral white matter changes consistent with hypertensive encephalopathy. Suggest emergency tactic.

Answer: hospitalization to the intensive care unit, intravenous nitroprusside. Blood pressure control and neurologic symptoms control.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology

2. MANAGEMENT OF PATIENTS WITH ACUTE CORONARY SYNDROME

Time frame – 6 hours.

Professional motivation. Patients with chest pain represent a very substantial proportion of all acute medical hospitalizations in Europe. Distinguishing patients with acute coronary syndromes (ACS) within the very large proportion with suspected cardiac pain are a diagnostic challenge, especially in individuals without clear symptoms or electrocardiographic features. Despite modern treatment, the rates of death, MI, and readmission of patients with ACS remain high.

Registry data consistently show that non-ST-elevation acute coronary syndrome (NSTE-ACS) is more frequent than ST-elevation acute coronary syndrome (STE-ACS). The annual incidence is ~3 per 1000 inhabitants, but varies between countries. Hospital mortality is higher in patients with STEMI than among those with NSTE-ACS (7% vs. 3–5%, respectively), but at 6 months the mortality rates are very similar in both conditions (12% and 13%, respectively). Long-term follow-up showed that death rates were higher among patients with NSTE-ACS than with STE-ACS, with a twofold difference at 4 years. This difference in mid- and long-term evolution may be due to different patient profiles, since NSTE-ACS patients tend to be older, with more comorbidities, especially diabetes and renal failure.

Many deaths occur in the very first hours after STEMI due to ventricular fibrillation. The implementation of an organization to cope with out-of-hospital cardiac arrest is pivotal to provide prompt cardiopulmonary resuscitation, early defibrillation if needed, and effective advanced cardiac life support.

Place of carrying out: class-room, wards of the cardiology and rheumatology departments, department of functional diagnostics.

Study objective is to improve students' skill to do clinical examination of the patients with acute coronary syndrome, to find out clinical and laboratory manifestations, to make the diagnosis, and indicate the treatment.

Basic level:

1. Definition of the acute coronary syndrome.
2. Examination of patients with acute coronary syndrome.

3. Electrocardiographic detection of myocardial infarction.
4. Biomarker evaluation in myocardial infarction.

Student has to know:

1. Variants of the clinical course of the myocardial infarction.
2. Interpretation of laboratory methods of examination for myocardial infarction.
3. Differential programs of the treatment of the acute coronary syndrome.
4. Differential treatment for the following complications of the myocardial infarction (cardiac insufficiency (collapse, pulmonary oedema, cardiogenic shock), pericarditis (epistenocardiac, Dresler's syndrome), mechanical causes of heart failure (myocardial rupture, ventricular septal defect due to perforation of the ventricular septum, mitral regurgitation, acute aneurysm, cardiac tamponade), thromboembolism, reinfarction, recurrent chest discomfort, arrhythmias, clinical death).
5. Anti-ischaemic agents (b-blockers, nitrates, calcium channel blockers, other antianginal drugs like ivabradine), antiplatelet agents, anticoagulants.

The main theoretical questions:

1. Definition of acute coronary syndrome.
2. Pathogenesis of acute coronary syndrome.
3. Non-ST-elevation acute coronary syndrome.
4. Criteria for the diagnosis and differential diagnosis of the unstable angina.
5. Differential diagnoses of mimic non-ST-elevation acute coronary syndrome: cardiac and noncardiac conditions that can mimic non-ST-elevation acute coronary syndrome.
6. Non-invasive imaging techniques (echocardiography, cardiac magnetic resonance imaging) and invasive imaging techniques (coronary angiography).
7. Long-term management: secondary prevention of myocardial infarction.
8. ST-elevation myocardial infarction: prehospital or early in-hospital care (restoring coronary flow and myocardial tissue reperfusion: percutaneous coronary interventions, fibrinolytic therapy, antithrombotic treatment).
9. Doses, side effects, and contraindications to fibrinolytic therapy.
10. Heart failure: Killip's classification, clinical features, treatment.
11. Cardiogenic shock: clinical features, treatment.
12. Mechanical complications: cardiac rupture and mitral regurgitation.
13. Arrhythmias and conduction disturbances in the acute phase. Management.
14. Management of specific types of infarction: right ventricular infarction, myocardial infarction in diabetic patients, patients with renal dysfunction.
15. Long-term medical treatment after STEMI.
16. Differential diagnosis of the myocardial infarction.
17. Coronary revascularization: indications. Coronary artery bypass surgery.

Assignment for self-assessment

1. A 65-year-old male patient on aspirin, nitrates and bisoprolol, being followed for chronic stable angina, presents to the hospital with a history of 2 to 3 episodes of more severe and long-lasting pressing chest pain each day over the past 3 days. His ECG and cardiac enzymes are normal. What is the most likely diagnosis?

a) myocardial infarction;
b) unstable angina;
c) stomach ulcer;
d) stable angina.

The best course of action of the following is to:

a) admit the patient and begin intravenous digoxin;
b) admit the patient and begin intravenous heparin;
c) admit the patient and give thrombolytic therapy;
d) admit the patient for observation with no change in medication;
e) discharge the patient from the hospital with increases in nitrates and bisoprolol.

2. A 61-year-old man is admitted to the CCU (coronary care unit) with crushing chest pain and the

changes in II, III and aVF leads. He is agitated, pale, and diaphoretic. Peripheral pulse is weak, and systolic BP is 90 mm Hg. Neck veins are distended, Kussmaul's sign is present, but no murmur. What is the most likely cause of hypotension?

3. You are examining a patient with the main complaint of relatively sudden onset of shortness of breath and weakness but no chest pain. ECG shows nonspecific ST-T changes. You would be particularly attuned to the possibility of painless, or silent, myocardial infarction in the:

a) advanced coronary artery disease patient with unstable angina on multiple medications;
b) elderly diabetic;
c) premenopausal female;
d) inferior MI patient;
e) MI patient with PVCs.

Answers:

1. b), b).

2. Right ventricular infarction.

3. b), the classic presentation of acute myocardial infarction involves heavy substernal chest pain or pressure. However, 15 to 20% of infarctions may be painless, with the greatest incidence in diabetics and the elderly. Dyspnoea or weakness may initially predominate in these patients. Other presentations include altered mental status, the appearance of an arrhythmia or hypotension. Diabetics are likely to have abnormal or absent pain response to myocardial ischaemia due to generalized autonomic nervous system dysfunction. The other choices have no specific link to greater likelihood of silent MI.

REFERENCES

1. 1 Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology

3. ACUTE LEFT VENTRICLE INSUFFICIENCY. RESUSCITATION MANAGEMENT OF THE CARDIAC ARREST

Time frame – 6 hours.

Professional motivation. ACS is the most frequent cause of acute new-onset heart failure. In-hospital mortality is especially high in patients with evidence of cardiogenic shock (from 40 to 60%). In contrast, patients with acute hypertensive heart failure have low in-hospital mortality, with patients

usually discharged alive and frequently asymptomatic. Registries indicate that almost half of the patients hospitalized with acute heart failure are rehospitalized at least once within 12 months. Estimates of the combined outcome of death or rehospitalizations within 60 days of admission vary from 30 to 50%.

Patients who have suffered from cardiorespiratory arrest who present with asystole as the initial rhythm have a poor prognosis. Survival from cardiorespiratory arrest for patients who present with ventricular fibrillation or pulseless ventricular tachycardia can approach 45–50%. The key intervention in improving survival is timely defibrillation.

Place of carrying out: class-room, wards of the cardiology and rheumatology departments.

Study objective is to improve students' skill to distinguish clinical symptoms of acute heart failure, to determine management strategy of emergency medicine and resuscitation.

Basic level:

1. To be able to collect complaints, case history, carry out objective examination of the patients with acute heart failure.
2. To interpret instrumental (ECG, EchoCG, X-ray) and laboratory data in patients with acute heart failure.

Student has to know:

1. Criteria for diagnosis of acute heart failure.
2. Management of acute heart failure.

The main theoretical questions:

1. Definition of acute left ventricular failure.
2. Clinical classification of acute heart failure.
3. Causes of acute left ventricular failure.
4. Indications and dosing of diuretics in acute heart failure. Potential adverse effects of loop diuretics.
5. Indications and dosing of i.v.vasodilators in acute heart failure.
6. Inotropic agents: indications for inotropic therapy. Dosing of positive inotropic agents in acute heart failure.
7. Sudden cardiac arrest: symptoms and emergency treatment. Prevention.

Assignment for self-assessment

At the grocery store you see an elderly lady slump to the floor. Coming to her to aid, your first step in adult basic life support (cardiopulmonary resuscitation) should be the following:

a) check for a carotid pulse;
b) assess breathing;
c) establish an airway;
d) determine responsiveness;
e) determine chest compression.

Answers:

d, one cannot automatically assume initially that an individual has had a cardiac or respiratory arrest. Therefore, first determine responsiveness by tapping or gently shaking the victim and shouting, "Are you ok?" Then shout or phone for help, then position the victim and yourself. Follow this with the ABCDs (establishing the Airway, assessing Breathing, assessing Circulation, and managing any need for Defibrillation).

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P.

Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology

4. DIAGNOSIS CRITERIA AND EMERGENCY TREATMENT OF PULMONARY EMBOLISM

Time frame – 6 hours.

Professional motivation. Pulmonary embolism is a common complication of hospitalization and contributes to 5 to 10% of deaths in hospitalized patients, making it one of the leading causes of preventable hospital deaths. Despite it being an enormous health problem, the true incidence of pulmonary embolism is uncertain.

The diagnosis of venous thrombi and pulmonary emboli can be difficult and requires specialized imaging techniques that are not available in all hospitals or healthcare settings.

In the United States, the estimated incidence of diagnosed pulmonary embolism is 71 to 117 per 100,000 person-years, but the true incidence is likely to be much more than this rate because studies show that for every case of diagnosed, nonfatal pulmonary embolism, there are 2.5 cases of fatal pulmonary embolism diagnosed only after death. Other studies have estimated that more than one million people in the United States are affected by pulmonary embolism per year, with 100,000 to 200,000 of these events being fatal.

Over half of all diagnosed cases of pulmonary embolism in the United States occur in patients in hospitals or nursing homes. One recent report estimated that more than 12 million patients (31% of patients discharged from hospitals in the United States) are at risk of pulmonary embolism.

Pulmonary embolism has earned the reputation of a silent killer because less than half of patients who die of pulmonary embolism were diagnosed with the problem prior to death.

Pulmonary embolism occurs in approximately 10% of patients with acute DVT. Most patients (up to 75%) are asymptomatic.

Place of carrying out: class-room, wards of the pulmonology.

Study objective is to be able to identify symptoms of pulmonary embolism and assign management.

Basic level:

1. Examination of patients with pulmonary embolism.
2. To determine symptoms of pulmonary embolism.
3. To evaluate data of the laboratory investigations of pulmonary embolism.

Student has to know:

1. Clinical signs in pulmonary embolism.
2. Investigational methods for the diagnosis of pulmonary embolism.
3. Markers of right ventricular dysfunction.

The main theoretical questions:

1. Risk factors for pulmonary embolism.
2. What are the symptoms of pulmonary embolism?

3. Health and lifestyle management to prevent pulmonary embolism.
4. Clot elimination by means of embolectomy or dissolution by IV thrombolytic therapy.
5. Anticoagulation: unfractionated heparin, low molecular weight heparin, long-term anticoagulation by warfarin. Dosage, complication of warfarin/heparin treatment, duration of treatment.

Assignment for self-assessment

A 35-year-old woman was hospitalized for premature labor at 37 weeks of gestation. Fetal distress was identified, and an emergency cesarean section resulted in the delivery of a healthy baby. Two days later, the mother reported "crampy" pain in her right leg and was prescribed pain medications. 4 days after delivery, she developed sudden shortness of breath and rapid heart rate. What is your presumptive diagnosis? What risk factors for development of this state do you know? What investigations should be performed? Management.

Answer: pulmonary embolism is suspected. Intravenous anticoagulant heparin is indicated. Additional investigations: computed tomography scan with a contrast dye injected into the vein to outline the pulmonary arteries, a lower extremity ultrasound. This patient's risk factors for pulmonary thromboembolism include immobility, the high levels of estrogens associated with pregnancy, and the tissue injury associated with surgery. Anticoagulant treatment should be administered for at least 3 months after delivery.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology

5. EMERGENCY IN THE PAROXYSMAL ARRHYTHMIA AND BLOCKS

Time frame – 6 hours.

Professional motivation. The estimated prevalence of paroxysmal supraventricular tachycardia (PSVT) in a 3.5% sample of medical records in the Marshfield (Wisconsin, the USA). Occurrence rates have been determined for various subtypes of supraventricular arrhythmia after acute myocardial infarction or coronary artery bypass graft surgery and in congestive heart failure (CHF) patients. The incidence rate of supraventricular arrhythmias among patients with CHF is 11.1%; paroxysms are more common in older patients, males, and those with longstanding CHF and radiographic evidence of cardiomegaly.

The only reported epidemiologic study of patients with atrial flutter involved a selected sample of individuals treated in the Marshfield Clinic in predominantly white, rural mid-Wisconsin. Over 75% of the 58,820 residents and virtually all health events were included in this population database. In approximately 60% of cases, atrial flutter occurred for the first time associated with a specific precipitating event (i.e., major surgery, pneumonia, or acute myocardial infarction). In the remaining patients, atrial flutter was associated with chronic comorbid conditions (i.e., heart failure, hypertension, and chronic lung disease). Only 1.7% of cases had no structural cardiac disease or precipitating cause (lone atrial flutter). The overall incidence of atrial flutter was 0.088%; 58% of these patients also had AF. Atrial flutter alone was seen in 0.037%. The incidence of atrial flutter increased markedly with age, from 5 per 100000 of those more than 50 years old to 587 per 100 000 over age 80. Atrial flutter is 2.5 times more common in men.

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia, occurring in 1–2% of the general population. Over 6 million Europeans suffer from this arrhythmia, and its prevalence is estimated to at least double in the next 50 years as the population ages. AF confers a 5-fold risk of stroke, and one in five of all strokes is attributed to this arrhythmia. Ischaemic strokes in association with AF are often fatal, and those patients who survive are left more disabled by their stroke and more likely to suffer a recurrence than patients with other causes of stroke. In consequence, the risk of death from AF-related stroke is doubled and the cost of care is increased 1.5-fold. AF may long remain undiagnosed (silent AF), and many patients with AF will never present to hospital. Hence, the “true” prevalence of AF is probably closer to 2% of the population. The prevalence of AF increases with age, from <0.5% at 40–50 years, to 5–15% at 80 years. Men are more often affected than women. The lifetime risk of developing AF is ~25% in those who have reached the age of 40. The prevalence and incidence of AF in non-Caucasian populations is less well studied. The incidence of AF appears to be increasing (13% in the past two decades).

AF is associated with increased rates of death, stroke and other thromboembolic events, heart failure and hospitalizations, degraded quality of life, reduced exercise capacity, and left ventricular (LV) dysfunction. Death rates are doubled by AF, independently of other known predictors of mortality. Only antithrombotic therapy has been shown to reduce AF-related deaths. Stroke in AF is often severe and results in long-term disability or death. Approximately every fifth stroke is due to AF; further-more, undiagnosed “silent AF” is a likely cause of some “cryptogenic” strokes. Paroxysmal AF carries the same stroke risk as permanent or persistent AF.

Hospitalizations due to AF account for one-third of all admissions for cardiac arrhythmias. Acute coronary syndrome (ACS), aggravation of heart failure, thromboembolic complications, and acute arrhythmia management are the main causes. Quality of life and exercise capacity are impaired in patients with AF.

Patients with AF have a significantly poorer quality of life compared with healthy controls, the general population, or patients with coronary heart disease in sinus rhythm. Left ventricular (LV) function is often impaired by the irregular, fast ventricular rate and by loss of atrial contractile function and increased end-diastolic LV filling pressure. Both rate control and maintenance of sinus rhythm can improve LV function in AF patients.

Ventricular arrhythmias include premature ventricular contraction, ventricular tachycardia and ventricular fibrillation. Both of last are life-threatening arrhythmias most commonly associated with heart attacks. The most serious arrhythmia is ventricular fibrillation, which is an uncontrolled, irregular beat. If cardiopulmonary resuscitation (CPR) can be started, or if electrical energy is used to “shock” the heart back to a normal rhythm, then the heart may not be too damaged. About 220,000 deaths from heart attacks each year are thought to be caused by ventricular fibrillation. People who have heart disease or a history of heart attack have the highest risk of ventricular fibrillation.

A less serious type of ventricular arrhythmia is a premature ventricular contraction (PVC). PVCs generally are not a cause for alarm and often do not need treatment. But if patient have heart disease or a history of ventricular tachycardia, PVCs can cause a more serious arrhythmia.

The incidence of ventricular tachycardia (VT) in the United States is not well quantified because of the clinical overlap of VT with ventricular fibrillation (VF). Examination of sudden death data

provides a rough estimate of VT incidence. Most sudden cardiac deaths are caused by VT or VF, at an estimated rate of approximately 300,000 deaths per year in the United States, or about half of the estimated cardiac mortality in this country. A prospective surveillance study gave a sudden death incidence of 53 per 100,000, accounting for 5.6% of all mortality. This is only a rough estimate of VT incidence, because many patients have nonfatal VT and because arrhythmic sudden deaths may be associated with VF or bradycardia rather than with VT. Ventricular tachycardia (VT) is observed more frequently in men, because ischaemic heart disease is more prevalent among men. In patients with ischaemic cardiomyopathy and nonsustained VT, sudden death mortality rates approach 30% in 2 years.

The exact incidence of sinus node dysfunction (SND) is unknown. The syndrome occurs in approximately one in 600 cardiac patients older than 65 years. Symptoms of SND almost invariably progress over time. The most dramatic symptom in patients with SND is syncope. About 50% of patients with SND develop tachy-brady syndrome over a lifetime; such patients have higher risk of stroke and death. The survival of patients with SND appears to depend primarily on the severity of underlying cardiac disease and is not significantly changed by pacemaker therapy. However, incidence of sudden death owing directly to SND is extremely low.

Possible complications of sick sinus syndrome include inadequate or inefficient pumping of the heart, heart failure, exercise intolerance, and injuries sustained by fainting spells and falling. Complications may develop from surgery to implant pacemakers, including infection, reaction to medications or anesthesia, and pacemaker failure.Sick sinus syndrome progresses slowly. No treatment is necessary as long as the individual is not experiencing symptoms. Even with a permanent artificial pacemaker, the long-term prognosis is excellent.

AV blocks occur more frequently in people older than 70 years, especially in those who have structural heart disease. Approximately 5% of patients with heart disease have first-degree AV block, and about 2% have second-degree AV block.

One study examined 24-hour Holter monitors in 625 asymptomatic, heart-disease-free people, aged 15 to 83 years. Transient type I second-degree AV block was seen in 14 (2.2%) patients, more frequently in patients with resting heart rates of <60 bpm. First-degree AV block has been associated with about a 2-fold increase in the probability of atrial fibrillation, a 3-fold increase in the probability of pacemaker implantation, and an increase in all-cause mortality.

First-degree AV block can be found in healthy adults. At 20 years of age, the PR interval may exceed 0.20 seconds in 0.5–2% of healthy people. At age 60 years, more than 5% of healthy individuals have PR intervals exceeding 0.20 seconds.

Advanced AV block (usually type II second-degree and third-degree) is usually anatomically infranodal and is seen in advanced His-Purkinje disease. One study examined the prevalence of His-Purkinje disease in the Framingham population. Here, QRS intervals of >0.12 seconds were significantly associated with coronary heart disease, CHF, AV block, hypertension, left ventricular hypertrophy, and ventricular extrasystoles. QRS intervals >0.12 seconds were rare before 50 to 60 years of age and were found in 11% of older men and 5% of older women. While intraventricular block does not inevitably lead to AV block, it frequently precedes the development of advanced AV block. Thus, this characterisation of a wide-QRS interval population is likely similar to that of the advanced AV block population.

Mobitz II second-degree AV block (Mobitz II) is rare in healthy individuals, whereas Mobitz I (Wenckebach) second-degree AV block is observed in 1–2% of healthy young people, especially during sleep.

Patients treated with permanent pacing to treat AV blocks have an excellent prognosis. Patients with advanced AV blocks who are not treated with permanent pacing remain at high risk of sudden cardiac death.

Although AV block generally is not associated with major morbidity, progressive degrees of AV block carry increasing morbidity and mortality.

Place of carrying out: class-room, wards of the cardiology and rheumatology departments, department of functional diagnostics.

Study objective is to improve students' skill to do clinical examination of the patients with tachyarrhythmias and bradycardia, indicate the treatment.

Basic level:

1. Clinical and ECG signs of extrasystoles, paroxysmal tachycardias, fibrillation, disturbances in conduction.
2. Examination of patients with tachyarrhythmias and bradycardia.
3. Drugs and doses for pharmacological conversion of (recent-onset) atrial fibrillation.

Student has to know:

1. Anti-arrhythmic medications: adenosine, amiodaron, calcium channel blockers: verapamil and diltiazem, lidocaine, sotalol, procainamide. Side effects, doses.
2. Management algorithm in bradycardia.
3. Management algorithm in tachyarrhythmias.

The main theoretical questions:

1. Classification of tachyarrhythmias.
2. Regular narrow-complex tachycardia: ECG signs, therapeutic choice.
3. Wide- (broad-) complex tachycardia: ECG signs, therapeutic choice.
4. Irregular tachycardias: atrial fibrillation and flutter. ECG signs, therapeutic choice.
5. Pharmacological cardioversion of recent-onset atrial fibrillation. Choice of rate and rhythm control strategies.
6. Management of patients with preexcited tachycardias (associated with or mediated by an accessory pathway).
7. Ventricular tachycardia, ventricular fibrillation: ECG signs, emergency.
8. Electrical defibrillation or synchronized cardioversion: indications, initial dose for cardioversion of atrial fibrillation, atrial flutter and other SVTs.
9. Radiofrequency catheter ablation.
10. Sick sinus syndrome, AV-blocks: ECG signs. Algorithm in management of different types of AV-blocks. Morgani-Adams syndrome.

Assignment for self-assessment

1. What medicines may accelerate arrhythmia via accessory pathway:
   a) digoxin, verapamil;
   b) amiodarone, heparin;
   c) amiodarone, sotalol.
2. In treatment with warfarin we have to check:
   a) INR (target 2–3);
   b) bleeding time (target up to 5 min);
   c) clotting time (target up to 6 min);
   d) INR (target 1–2);
   e) bleeding time (target up to 10 min).
3. The first-line drug for acute symptomatic bradycardia:
   a) hydrocortisone;
   b) dextrose;
   c) atropine;
   d) amiodarone.
4. The recommended atropine dose for symptomatic bradycardia is:
   a) 0.5 mg IV every 3 to 5 minutes to a maximum total dose of 3 mg;
   b) 1.5 mg IV every 3 to 5 minutes to a maximum total dose of 5 mg;
   c) 2.5 mg IV every 3 to 5 minutes to a maximum total dose of 5 mg;
   d) 5 mg IV every 3 to 5 minutes to a maximum total dose of 10 mg.
5. Synchronized cardioversion is recommended to treat the following states, except:
   a) unstable SVT due to reentry;
   b) unstable atrial fibrillation;
   c) unstable atrial flutter;

d) unstable monomorphic (regular) VT;
e) unstable angina.

Answers:
1. a. 2. a. 3. c. 4. a. 5. e.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Integrative Cardiology [Електронний ресурс]: A New Therapeutic Vision / edited by Massimo Fioranelli. – 1st ed. 2017. – Cham: Springer International Publishing, 2017. – XII, 276 p.
6. Cardiology Procedures [Електронний ресурс]: A Clinical Primer / edited by Robert C. Hendel, Carey Kimmelstiel. – 1st ed. 2017. – London: Springer London, 2017. – XI, 347 p
7. Braunwald's heart disease: A textbook of cardiovascular medicine, 2-Volume Set 10th Edition. International edition. // by Douglas L. Mann, Douglas P. Zipes, Peter Libby, Robert O. Bonow. – Publisher: Elsevier, 2015. – 2136 pages.
8. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.
9. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
10. https://reference.medscape.com/guide/medical-calculators
11. https://www.escardio.org/Education/E-Learning/basic-tools-in-general-cardiology

Semantic module 2. Emergencies in rheumatology

6. MANAGEMENT OF PATIENTS WITH LOW BACK PAIN

Time frame – 6 hours.

Professional motivation. The prevalence of low back pain increases markedly with age, and many of the disorders are affected by lifestyle factors, such as obesity and certain types of physical activity. Although the economic and public health effects of back disorders and especially low back pain are enormous, epidemiologic research into the problem is in a formative stage, especially compared with cardiovascular conditions and cancer. As a result of the increasing number of older people throughout the world, the burden on the individual and society as a whole is expected to increase dramatically. While not a disease, back pain is a major cause of disability, especially in areas where compensation systems take it into cognizance. Approximately 90% cases of back pain have no identifiable cause and are designated as nonspecific. A variety of diagnostic labels have been used by health care professionals.

In the US population, the third National Health and Nutrition Examination Survey (NHANES III) (1988–1994) estimated that the 12-month period prevalence of back pain episodes lasting for at least 1 month was 17.8%. In the adult Greek population, the 1-month prevalence of back pain has been estimated as 32%. A direct comparison of back pain between the United Kingdom and Germany not only showed differences in prevalence between the two countries (22% compared with 44.9% in women) but also demonstrated marked differences in the prevalence of current back pain within each country or region.

Over a lifetime 80% of people have lower back pain, with 26% of American adults reporting pain of at least one day in duration every three months. 41% of adults aged between 26 and 44 years reported

having back pain in the previous 6 months. Most people with acute lower back pain recover completely over a few weeks regardless of treatment. 60% of people recover after seven weeks, regardless of the treatments they receive. Consistent with these statistics, a recent study found that almost 30% of patients did not recover from the presenting episode of low back pain within a year. For those patients whose low back pain continues on to chronicity, it is rarely self limiting, as fewer than 10% of those patients whose low back pain becomes chronic report no pain five years later.

Place of carrying out: class-room, wards of the rheumatologic department.

Study objective: to improve the skills of differential diagnosis of states which are accompanied by low back pain, to determine management of such patients.

Basic level:

1. To be able to collect complaints, case history, carry out physical examination in patients with low back pain.
2. To interpret instrumental and laboratory data in patients with low back pain.

Student has to know:

1. How to find out joint and spine injury using instrumental methods of examination.
2. What diseases are accompanied by low back pain.
3. How to make an algorithm of investigations in patients with low back pain.
4. How to determine approaches to treatment in different aetiology of low back pain.

The main theoretical questions:

1. Diagnostic criteria of ankylosing spondylitis. Management.
2. Diagnostic criteria of reactive arthritis, Reiter's disease. Management.
3. Diagnostic criteria of psoriatic arthritis. Management.
4. Diagnostic criteria of gout. Pathogenic management.
5. Symptomatic treatment of the disorders of the joints.
6. Using of nonsteroidal anti-inflammatory drugs (NSAID), corticosteroids, muscle relaxants in treatment of dorsalgia, side effects and advantages. Physiotherapy and sanatorium-resort therapy.
7. Diagnostic differences of pain in inflammatory and degenerative joint diseases.
8. Diagnosis and treatment of radicular syndrome.
9. Diagnosis and treatment of kidney stones.

Assignment for self-assessment

1. Radiographic data in osteochondrosis:
   a) vertebral sclerosis;
   b) osteophytes;
   c) decrease of intervertebral space height;
   d) lumbar lordosis straightening;
   e) everything listed above.

2. A 22-year-old man complains of low back pain and stiffness that is worse on arising and improves with exercise. On examination, he is found to have limited mobility of the sacroiliac joints and lumbar spine. X-ray examination shows bilateral sacroileitis. A serum test for histocompatibility antigen HLA-B27 is positive.

What diagnosis do you suspect? Prescribe the main groups of drugs.

Answers:

1. e.

2. Ankylosing spondilitis. Disease-modifying drugs, nonsteroidal anti-inflammatory drugs, if it is necessary – corticosteroids, treatment of the muscular spasm.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International

Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.
6. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
7. https://reference.medscape.com/guide/medical-calculators

Semantic module 3. Emergencies in pulmonology and allergology

8. EMERGENCY TREATMENT OF SEVERE ASTHMA ATTACK EMERGENCY TREATMENT OF ANAPHYLACTIC REACTIONS

Time frame – 6 hours.

Professional motivation. Inadequate control of asthma leads to much morbidity and poor quality of life. Complications mostly relate to acute exacerbations: pneumonia, pneumothorax, pneumomediastinum, respiratory failure and arrest, death. Individuals continue to die from asthma (approximately 1,300 deaths in the UK from asthma in 2005). A common feature of deaths from asthma is that the patient and/or the medical staff have underestimated the severity of the attack.

Professional motivation. The American College of Allergy, Asthma and Immunology Epidemiology of Anaphylaxis Working group summarised the findings from a number of important international epidemiological studies and concluded that the overall frequency of episodes of anaphylaxis using current data lies between 30 and 950 cases per 100,000 persons per year.

The same group provided data indicating a lifetime prevalence of between 50 and 2000 episodes per 100,000 persons or 0.05–2.0%. More recent UK primary care data concur, indicating a lifetime age-standardised prevalence of a recorded diagnosis of anaphylaxis of 75.5 per 100,000 in 2005.13 Calculations based on these data indicate that approximately 1 in 1,333 of the English population have experienced anaphylaxis at some point in their lives.

The overall prognosis of anaphylaxis is good, with a case fatality ratio of less than 1% reported in most population-based studies. Risk of death is, however, increased in those with preexisting asthma, particularly if asthma is poorly controlled or in those asthmatics who fail to use, or delay treatment with, adrenaline. There are approximately 20 anaphylaxis deaths reported each year in the UK, although this may be a substantial under-estimate.

Place of carrying out: class-room, wards of the pulmonology.

Study objective is to be able to identify severe asthma attack and assign the first aid and management. To identify signs of anaphylaxis and assign management.

Basic level:

1. Aetiology and pathogenesis of bronchial obstruction.
2. Interpretation of sputum analysis, spirography, peakflowmetry, X-ray examination, chest tomography.
3. Pathophysiology of anaphylactic reactions.
4. To examine patients with allergic reactions.
5. To determine symptoms of anaphylaxis.

Student has to know:

1. Clinical signs of severe asthma attack.
2. Investigational methods for the diagnosis of severe asthma attack.
3. How to make differential diagnosis, clinical diagnosis.
4. How to indicate the treatment for patients with severe asthma attack.
5. Clinical signs of anaphylaxis, Quincke’s oedema.
6. How to indicate the treatment for patients with anaphylaxis.

The main theoretical questions:

1. What causes a severe asthma attack?

2. What are the symptoms of a severe asthma attack?
3. Does wheezing indicate a severe asthma attack?
4. How is a severe asthma attack diagnosed?
5. How is a severe asthma attack treated?
6. Definition of anaphylaxis. Triggers for anaphylactic reactions.
7. Criteria for anaphylaxis.
8. General principles of allergic diseases therapy.
9. Emergency treatment of patients with anaphylaxis.

Assignment for self-assessment

Task1. A 27-year-old woman had been suffering from severe asthma since she was a teenager. Her symptoms had worsened over the previous three days, after she'd caught a cold. Now she was using salbutamol every hour with no relief. She delayed calling her primary doctor's office in the hope that her symptoms would get better.

She frequently suffered from cough, wheezing and chest tightness. She awakened with nighttime symptoms two to three times per week. She was using high dose inhaled steroids daily to decrease airway inflammation and also using inhaled salbutamol. She had a history of multiple hospitalizations for asthma, including one admission to the intensive care unit two years earlier. At that time, she had required mechanical ventilation (machine-assisted breathing) for several days.

The patient was markedly short of breath and could only speak in short sentences. She had a fever of 37 degrees, was breathing rapidly, and her heart rate was 150/minute. She was no longer wheezing and doctor could barely hear any breath sounds at all. Her lips were a little blue. Oxygen saturation is only 84%.

What treatment should be given? Who is at risk for the development of status asthmaticus? Can status asthmaticus be prevented?

Answer:

Nebulized albuterol (salbutamol) administered by face mask, oxygen by nasal cannula and high doses of intravenous steroids.

Although patients with mild asthma will occasionally have episodes of status, this dangerous condition occurs mostly in patients with very severe disease and in those who have had previous severe attacks. The best way to decrease the possibility of having a severe asthma attack is to take the medications regularly as prescribed.

Task 2. A 60-year-old female with acute pancreatitis was given 50 mg of ranitidine as a slow intravenous bolus for epigastric discomfort. She had myocardial infarction in the past. She was allergic to metronidazole. She had no family history of drug allergies. Few minutes after the injection, the patient complained of itching at the injection site that spread to involve the entire upper limb. She also complained of swelling of her tongue and difficulty in breathing. Within minutes her level of consciousness deteriorated and she became comatose. The initial examination revealed the following features: a grossly oedematous face, neck and extremities, a grossly swollen tongue, congested conjunctiva, cyanosis, diffuse rhonchi over both lung fields. Suggest treatment for this patient.

Answer:

Immediate administration of intramuscular adrenaline, intravenous hydrocortisone and high flow oxygen. Commence cardiopulmonary resuscitation in case of cardiorespiratory arrest.

REFERENCES

1. Global strategy for asthma http://www.ginasthma.org/
2. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
3. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
4. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.

5. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
6. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368с.
7. https://empendium.com/mcmtextbook/ 2 https://www.mdcalc.com/
8. https://reference.medscape.com/guide/medical-calculators
9. https://www.ers-education.org/e-learning/procedure-videos/
10. https://www.resus.org.uk/library/additional-guidance/guidance-anaphylaxis/emergency-treatment

Semantic module 4. Emergencies in gastroenterology

9. DIAGNOSTIC CRITERIA AND EMERGENCY TREATMENT OF ACUTE ABDOMINAL PAIN

Time frame – 6 hours.

Professional motivation. Acute pain in abdominal part is the symptom of dangerous state. Therefore well-timed diagnosis and correct choice of medical tactics is necessary for emergency care. Acute abdominal pain accounts for approximately 50% of all urgent admissions to general surgical units. Cancer is a more common cause of acute pain in those over 70 years than in those under 50 years. Older people with vague abdominal symptoms should therefore be carefully assessed and serious pathology should be excluded.

Acute pancreatitis accounts for 3% of all cases of abdominal pain admitted to hospital. It affects 2–28 per 100,000 of the population and may be increasing in incidence. Despite recent advances in management, the mortality has remained unchanged at 10–15%. About 80% of all cases are mild with a mortality less than 5%; 98% of deaths occur in 20% of severe cases. One-third occur within the first week, usually from multi-organ failure. After this time the majority of deaths result from sepsis, especially that complicating infected necrosis.

In ischaemic gut injury the key steps in treatment are resuscitation, correction of cardiac disease, and intravenous antibiotic therapy, followed by laparotomy. If this is done early enough, embolectomy and vascular reconstruction may salvage some small bowel. In these rare cases a “second look” laparotomy is undertaken 24 hours later and further necrotic bowel resected. The results of therapy are dependent upon early intervention; patients treated at a late stage have a 75% mortality rate. Survivors often have nutritional failure from short bowel syndrome and require intensive nutritional support, sometimes including home parenteral nutrition.

Place of carrying out: class-room, wards of the gastroenterology.

Study objective is to improve students’ skills in differential diagnosis and treatment of acute abdominal pain.

Basic level of knowledge and skills:

1. Mechanisms of pain originating in case of abdominal organs diseases.
2. Procedure of examination of the patients with an acute abdominal pain.

Student has to know:

1. Clinical signs of the bowel diseases.
2. Investigational methods used in case of acute abdominal pain.
3. Mechanisms of action of the drugs which are used in the treatment of acute abdominal pain.
4. How to evaluate data of the laboratory and instrumental investigations.
5. Diagnostic algorithm in acute abdominal pain.

The main theoretical questions:

1. Diagnostic criteria for acute pancreatitis. Complications, management.
2. Criteria for diagnosis of acute small bowel ischaemia.
3. Criteria for diagnosis of peptic ulcer disease. Complications of peptic ulcer disease and its treatment.
4. Criteria for diagnosis of gallstones and choledocholithiasis; complications, management.

5. Criteria for diagnosis of aortic aneurism.
6. Criteria for diagnosis of ureteric colic.
7. Determination of medical tactics at acute abdominal pain.

Assignment for self-assessment

A 42-year-old man was admitted to the hospital due to the presence of acute pain in the epigastric region without any irradiation. The pain is intermittent, can be decreased after meal or a glass of milk; the patient feels the most severe pain at night and in the early morning. The patient has also nausea and sometimes vomiting after which may feel better. The patient is also constipated. This pain started 1 week ago after the stressful situation. The patient didn't have such a pain before. During this period of time the patient didn't go to the doctor, he took aAlmagel without the effect. His father suffers from gastritis, his grandmother died from stomach cancer.

Complete blood count: Hb – 110 g/l, er. – 3.2×109/l, leuc. – 8.4×1012/l, ESR – 11mm/hour; biochemical analysis: protein – 62 g/l, alb. – 60%, glob. – 40%, creatini – 76 μmol/l, urea – 6.2 mmol/l, ALT – 21 U/l, AST – 18 U/l.

Gastroscopy: redness of the stomach mucosa, ulcer 0.5×0.6 cm in the diameter in the anterior wall of the duodenum bulb.

Make the diagnosis; name the main aetiology factor. Name other laboratory and instrumental findings to confirm the diagnosis. Which complications may have the patient? Name treatment strategy.

Answers:

1. Peptic ulcer disease; H. pylory infection.
2. Invasive or noninvasive tests for the detecting of H. pylory; pH-monitoring; coprologycal investigation.
3. Bleeding, perforation, penetration, gastric outlet obstruction.
4. Anti H. pylory therapy (triple or quadriple), PPI after this therapy.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.

Semantic module 5. Emergencies in endocrinology

11. DIAGNOSTIC CRITERIA AND EMERGENCY TREATMENT OF HYPOGLYCEMIA MANAGEMENT OF HYPERGLYCEMIC CRISES IN PATIENTS WITH DIABETES

Time frame – 6 hours.

Professional motivation. Hypoglycemia is a common side effect of insulin therapy in diabetes, particularly in people with type 1 diabetes. Mild (self-treated) episodes occur frequently (1–2 episodes/week), while severe hypoglycemia, defined as any episode requiring external help, affects up to 30% of people with type 1 diabetes annually (1–5), with an incidence ranging from 1.0 to 1.6 episodes per patient per year in unselected northern European populations. In contrast, the rate of severe hypoglycemia in people with type 2 diabetes treated with insulin is reported to be low, but these have been recorded in people with a short duration of insulin therapy.

Prolonged hypoglycemia may result in serious neurologic complications and death.

Professional motivation. Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) are two of the most serious acute complications of diabetes. These hyperglycemic emergencies continue to be important causes of morbidity and mortality among patients with diabetes in spite of

major advances in the understanding of their pathogenesis and more uniform agreement about their diagnosis and treatment. The annual incidence rate for DKA estimated from population-based studies ranges from 4.6 to 8 episodes per 1,000 patients with diabetes, and in more recent epidemiological studies in the U.S., it was estimated that hospitalizations for DKA during the past two decades are increasing. Currently, DKA appears in 4–9% of all hospital discharge summaries among patients with diabetes. It is estimated that the rate of hospital admissions due to HHS is lower than the rate due to DKA and accounts for <1% of all primary diabetic admissions. Mortality rates, which are <5% in DKA and 15% in HHS, increase substantially with aging and the presence of concomitant life-threatening illness. It is important to note that up to 20% of patients may present in the emergency room with either DKA or HHS without a previous diagnosis of diabetes. In the African-American population, DKA has been increasingly noted in newly diagnosed obese type 2 diabetic patients. Therefore, the concept that the presence of DKA in type 2 diabetes is a rare occurrence is incorrect.

Place of carrying out: class-room, wards of the endocrinology.

Study objective is to be able to identify symptoms of hypoglycemia and assign management. To identify symptoms of DKA and assign management.

Basic level:

1. Examination of patients with diabetes.
2. To determine symptoms of hypoglycemia.
3. To evaluate data of the laboratory investigations of hypoglycemia.
4. Pathogenesis of DKA: carbohydrate metabolism, lipid and ketone metabolism, water and electrolyte metabolism.
5. Examination of patients with DKA (case history and physical examination).

Student has to know:

1. Clinical signs of hypoglycemia.
2. Investigational methods for the diagnosis of hypoglycemia.
3. How to indicate the treatment for patients with hypoglycemia.
4. Clinical signs of hyperglycemia.
5. How to indicate the treatment for patients with hyperglycemia.

The main theoretical questions:

1. What causes hypoglycemia?
2. What are the symptoms of hypoglycemia?
3. Health and lifestyle management to prevent hypoglycemia.
4. Differential approach to the first aid in hypoglycemia.
5. Clinical signs of diabetic ketoacidosis (the triad of DKA).
6. Diagnostic criteria for DKA, HHS.
7. Management of patient with DKA and HHS: replacement of fluid and electrolytes, insulin therapy.

Assignment for self-assessment

1. The hypoglycemia protocol says to start treatment when the fasting blood glucose is less than:
   a) 70 mg/dl regardless symptoms or not;
   b) 80 mg/dl regardless symptoms or not;
   c) 90 mg/dl regardless symptoms or not.

2. A 26 year old man with a history of type I diabetes is admitted to the emergency because he is sleepy and desoriented. His fasting blood glucose is 44 mg/dl. Doctor tried to give a cup of juice but the patient is not swallowing well. What should be done?

3. A 20 year old man with a history of type I diabetes is admitted with pyrexia, drowsiness, and fast deep breathing. You suspect DKA. What is the initial treatment priority?
   a) IV drip of 5% dextrose solution;
   b) IV drip of 0.9% saline;
   c) 10 units of subcutaneous short-acting insulin;
   d) insulin via infusion pump;

e) potassium supplementation.

Answers:

1. a.
2. Glucagon im, check fasting blood glucose in 20 minutes.

3. b, patient with DKA tend to be significantly dehydrated due to osmotic diuresis and reduced fluid intake. In adults this is usually at least 3.5 litres. A treatment plan in DKA would be: check blood gas, capillary glucose; give IV drip of 0.9% saline 15–30 ml/kg/h for first 2 hours then at reduced rate; 10 units of subcutaneous short acting insulin followed by infusion at a rate of 2–6 units/h; monitor potassium and capillary blood glucose.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.

Semantic module 6. Emergencies in haematology

12. DIAGNOSTIC CRITERIA AND EMERGENCY TREATMENT OF THROMBOTIC THROMBOCYTOPENIC PURPURA

Time frame – 6 hours.

Professional motivation. The incidence of thrombotic thrombocytopenic purpura is about 4–6 per million people per year. Idiopathic thrombotic thrombocytopenic purpura occurs more often in women and African-American people, while the secondary forms do not show this distribution. Pregnant women and women in the postpartum period accounted for a notable portion (12–31%) of the cases in some studies; thrombotic thrombocytopenic purpura affects approximately 1 in 25,000 pregnancies.

The mortality rate is approximately 95% for untreated cases, but the prognosis is reasonably favourable (80–90% survival) for patients with idiopathic thrombotic thrombocytopenic purpura diagnosed and treated early with plasmapheresis.

Place of carrying out: class-room, wards of the haematology departments.

Study objective is to improve students' skill to do clinical examination of the patients with thrombotic thrombocytopenic purpura, to find out clinical and laboratory manifestations, to indicate the treatment.

Basic level:

1. Examination of patients with thrombocytopenic purpura.
2. To determine symptoms of thrombocytopenic purpura.

Student has to know:

1. Clinical signs in thrombocytopenic purpura.
2. Investigational methods for the diagnosis of thrombocytopenic purpura.
3. How to indicate the treatment for patients with thrombocytopenic purpura.

The main theoretical questions:

1. Causes and pathogenesis of thrombocytopenic purpura.
2. Diagnostic criteria for thrombocytopenic purpura.
3. Management of patient with thrombocytopenic purpura.

Assignment for self-assessment

A 44-year-old woman has complications on skin haemorrhages which appear spontaneously, menorrhagia, general weakness, dizziness. She suffers from these diseases since childhood.

On examination, she was normotensive (BP 120/90). She has pale skin, haemorrhages on the anterior surface of the trunk, enternal surface of the extremities. Lymph nodes of the neck are enlarged. Her lungs are clear. Cardiac examination shows regular rate and rhythm without murmur. The abdomen is soft without tenderness or distention. The liver spans 9 cm in the midclavicular line with a smooth edge. Spleen is palpable. Kidneys are not palpable. Pasternatsky's sign is positive in both sides. Blood testing: RBC – 1.9×10^{-12}/l; Hb – 86 g/l; ESR – 16 mm/h; WBC – $8*10^{-9}$/l; eos. – 2%, neutrophils – 69%, lymphocytes – 15%, monocytes – 5%, platelet 20,000/μL of large size, with otherwise normal morphology. Total protein – 73 g/l, urea – 6.7 mmol/l, creatinine – 80 μmol/l, bilirubin – 11.8 μmol/l, fasting glucose – 5.0 mmol/L. Urinalysis: RBC – 1–3, WBC – 1–2, specific gravity – 1012, protein 0.03 g/L. Chest radiographic findings, ECG are normal. Ultrasound shows splenomegaly.

Questions: what is the most likely clinical diagnosis? What additional diagnostic studies are indicated? What is differential diagnosis? What treatment does this patient need?

Answer: thrombocytopenic purpura. Myelogramm. Haemoblastosis, B12-deficit, haemolytic, aplastic anaemia, haemorrhagic vasculitis, autoimmune trombocytopenia due to SLE. Glucocorticosteroids, cytostatics, splenectomy.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.

13. DIAGNOSTIC CRITERIA AND EMERGENCY TREATMENT OF AGUTE THROMBOSIS

Time frame – 6 hours.

Professional motivation. Deep venous thrombosis (DVT) and pulmonary embolism (PE) are manifestations of a single disease entity, namely, venous thromboembolism (VTE).

DVT is one of the most prevalent medical problems today, with an annual incidence of 80 cases per 100,000. Each year in the United States, more than 200,000 people develop venous thrombosis; of those, 50,000 cases are complicated by PE. Lower-extremity DVT is the most common venous thrombosis, with a prevalence of 1 case per 1000 population. In addition, it is the underlying source of 90% of acute PEs, which cause 25,000 deaths per year in the United States. The goals of pharmacotherapy for DVT are to reduce morbidity, prevent postthrombotic syndrome (PTS), and prevent PE.

Most small thrombi in the lower extremities tend to resolve spontaneously after surgery. In about 15% of cases, however, these thrombi may extend into the proximal femoral venous system of the leg. Untreated proximal thrombi represent a significant source of clinically significant pulmonary emboli.

Age has been well studied as an independent risk factor for venous thrombosis development. Although a 30-fold increase in incidence is noted from age 30 to age 80, the effect appears to be multifactorial, with more thrombogenic risk factors occurring in the elderly than in those younger than 40 years. Venous stasis, as seen in immobilized patients and paralyzed limbs, also contributes to the development of venous thrombosis. Autopsy studies parallel the duration of bed rest to the incidence of venous thrombosis, with 15% of patients in those studies dying within 7 days of bedrest to greater

than 80% in those dying after 12 weeks. Within stroke patients, DVT is found in 53% of paralyzed limbs, compared with only 7% on the nonaffected side. From a demographic viewpoint, Asian and Hispanic populations have a lower risk of VTE, whereas whites and blacks have a higher risk (2.5–4 times higher).

Acute thromboembolic occlusion of the superior mesenteric artery (SMA) is a condition with a serious prognosis. Acute mesenteric ischaemia (AMI) is an uncommon occurrence and represents 0.1% of hospital admissions. Despite considerable advances in medical diagnosis and treatments over the past 4 decades, mesenteric vascular occlusion still has a poor prognosis, with an in-hospital mortality rate of 59 to 93%. The high rate of mortality can be explained by the nonspecific signs and symptoms that characterize AMI.

Place of carrying out: class-room, wards of the pulmonology, rheumatology.

Study objective is to be able to identify symptoms of acute thrombosis and assign management.

Basic level:

1. Three factors that are critically important in the development of venous thrombosis (Virchow's triad).
2. Coagulation pathway. Anticoagulant mechanisms for prevention inadvertent activation of the clotting process.
3. Examination of patients with acute thrombosis.
4. To determine symptoms of acute thrombosis.

Student has to know:

1. Clinical signs in venous thromboembolism.
2. How to evaluate data of the laboratory investigations in acute thrombosis.
3. Investigational methods for the diagnosis of venous thromboembolism.
4. How to indicate the treatment for patients with venous thromboembolism.

The main theoretical questions:

1. Factors that contribute to DVT.
2. Lower-extremity deep venous thrombosis.
3. Upper-extremity deep venous thrombosis (2 forms of upper-extremity DVT are effort-induced thrombosis (Paget-von Schrötter's syndrome) and secondary thrombosis).
4. Common risk factors for DVT.
5. Approaches for confirming the diagnosis of DVT (Wells' score for probability of DVT, venous ultrasonography, venography, magnetic resonance imaging, biomarkers).
6. Acute thromboembolic occlusion of the superior mesenteric artery.
7. Budd-Chiari syndrome.
8. The first aid in venous thromboembolism.
9. The primary objectives for the treatment of deep venous thrombosis. How should the patient with thrombosis be managed? Initial therapy. Long-term therapy. Thrombolytic therapy.
10. Complications of anticoagulant therapy.

Assignment for self-assessment

A 22-year-old woman was admitted to the hospital with complaints of abdominal "bloating".

The patient had been in excellent health until 4 weeks earlier, when she started taking estradiol tablets. 4 days later, she began to have bloating and discontinued the medication; an ultrasonographic examination of the abdomen was normal. The patient was not sexually active; vaginal examination 3 months earlier had been normal. Her menses were normal; her last menstrual period had occurred 23 days before admission. She took no medications. Previous summer she had traveled to Israel. She drank alcohol in moderation and did not smoke. The family history obtained at that hospital included renal-vein thrombosis in the patient's father and deep venous thrombosis in a paternal aunt.

There was no recent history of anorexia, nausea, vomiting, chills, fever, lymphadenopathy, back or bone pain, arthralgia, pruritus, menstrual changes, abdominal trauma, risk factors for infection with the human immunodeficiency virus. She gained 3.6 kg over a period of three months and found abdominal distention.

The physical examination was normal except for abdominal distention, ascites and palpable liver edge 2 cm below the right costal margin; the spleen was not felt. No icterus or lymphadenopathy was found, and the rectal examination was normal.

Haematologic laboratory tests: complete blood count is normal. Test for fibrin-split products was positive (titre of more than 40 and less than 66 μg/ml (normal value <10; value usually considered abnormal >40). The conjugated bilirubin concentration was 9 μmol/l, total bilirubin – 26 μmol/l, γ-GT – 59 U/l (normal range – 7 to 33), urea – 6 mol/l, creatinine – 90 μmol/l, glucose – 4.8 mol/l, total protein – 68 g/l, albumin – 60%, globulin – 40%, aspartate aminotransferase – 38 units, alanine aminotransferase – 42 units, anticardiolipin IgG and IgM antibodies are normal, slightly prolonged prothrombin time, slightly prolonged activated partial-thromboplastin time, fibrinogen – 5 g/l.

Films of the abdomen showed distended loops of bowel. CT scan of the abdomen confirmed the presence of ascites; the liver and spleen were enlarged. Abdominal paracentesis yielded 3 liters of ascitic fluid that contained 50 white cells/mm^3, all of which were lymphocytes; the total protein concentration was 3.9 g/dl. Microscopical examination of the fluid revealed no microorganisms, and cytologic examination showed no malignant-tumour cells. One day later, the patient had increasing abdominal pain and low-grade fever.

On the second hospital day, the temperature was 38 °C, the pulse was 112, the respirations were 18, the blood pressure was 110/70 mm Hg. Physical examination revealed abdominal distention; diffuse tenderness, which was most marked in the right upper quadrant; palpable liver edge – 3 cm below the costal margin. There was + peripheral oedema. No signs of deep venous thrombosis were detected. No prominent venous pattern was observed in the abdominal wall, and the spleen was not felt.

An electrocardiogram revealed no abnormalities. Radiographs of the chest revealed a right pleural effusion and a probable left pleural effusion. Ultrasonographic examination of the abdomen showed ascites and heterogeneous echogenicity of the liver texture with areas of increased echogenicity. The portal, superior mesenteric, and splenic veins were patent, with hepatopetal flow. The hepatic artery was patent, and the middle and left hepatic veins were small but patent. Cardiac ultrasonographic study showed a moderate pericardial effusion, without evidence of cardiac constriction. Hepatic venographic examination revealed a stenotic middle hepatic vein with intraluminal filling defects consistent with nonocclusive thrombi; the hepatic-vein tributaries were obliterated. The mean hepatic-vein wedge pressure was 38 mm Hg; the right free hepatic-vein pressure was 15/6 mm Hg, with a mean pressure of 9 mm Hg. The portal vein was not opacified during wedged injection of the hepatic vein.

On the eighth hospital day, an ultrasonographic examination of the portal vein showed that it was widely patent, with normal hepatopetal flow. The hepatic artery appeared normal, with normal Doppler-signal characteristics.

What is the clinical diagnosis?

What is the mechanism of ascites formation?

What diseases should be differentiated in this patient if she has pleural effusions?

What factors are responsible for hepatic-vein thrombosis?

What additional investigations should be suggested?

Treatment.

Answers: the clinical course of this woman's illness with the rapid development of liver disease over a period of a few weeks is typical of the Budd-Chiari syndrome, but occasionally the symptoms extend over a period of months to years. Selective hepatic venographic findings indicate on the Budd-Chiari syndrome – thrombosis of the hepatic venous circulation. The results of liver-function tests are usually not markedly abnormal, as was the case in this patient.

The high mean hepatic wedge pressure indicated the presence of portal hypertension, which resulted from increased resistance to hepatic blood flow. Portal hypertension leads to the extravasation of fluid from plasma into the peritoneal cavity. Another factor contributing to the formation of ascites is the weeping of hepatic lymph from the surface of the liver as a result of the obstruction of hepatic sinusoids and lymphatics.

The differential diagnosis of pleural effusions includes collagen-vascular, cancer and infectious diseases, but the normal sedimentation rate makes these diagnoses untenable in this case. Since this

woman had one site of thrombosis in the hepatic veins, it is tempting to postulate that the pleural effusions resulted from pulmonary emboli with infarction. The absence of respiratory tract symptoms and pleuritic chest pain, however, makes it more likely that the effusions were due to the hepatic dysfunction.

The factors responsible for hepatic-vein thrombosis are divided into 2 groups. The first group includes acquired diseases (polycythemia vera, paroxysmal nocturnal haemoglobinuria, neoplasms), those involving predisposing factors (such as the postoperative or postpartum state or use of oral contraceptives), and acquired abnormalities (such as the lupus anticoagulant or antiphospholipid syndrome). Two haematologic diseases that account for approximately 20% of cases of the Budd-Chiari syndrome are polycythemia vera and paroxysmal nocturnal haemoglobinuria. Neoplasms (hepatocellular carcinoma, renal-cell carcinoma, adrenal carcinoma), infections (amebic abscesses) account for about 10% of the cases. Among the predisposing factors, use of oral contraceptives and pregnancy are associated with about 20% of the cases, and trauma with 2%. The second group of patients includes those with hereditary or biologic defects that confer a predisposition to thrombosis.

The absence of a high titre of anticardiolipin antibodies is evidence against the diagnosis of the antiphospholipid syndrome. With a base-line prolongation of the prothrombin and partial-thromboplastin times due to liver disease or vitamin K deficiency in this patient, it would be very difficult to interpret the results of the clotting assays that are used to detect the presence of a lupus anticoagulant. The moderate elevation in the level of fibrin-split products probably resulted from endogenous fibrinolysis of hepatic-vein thrombi rather than from a process such as disseminated intravascular coagulation.

Congenital deficiencies of antithrombin III, protein C, protein S are associated with an increased risk of venous thrombosis and have been reported in patients with the Budd-Chiari syndrome. These deficiencies are usually inherited as autosomal dominant disorders. In rare cases, hereditary abnormalities in fibrinogen are also associated with a thrombotic diathesis. The diagnostic yield of a laboratory evaluation is increased somewhat if the patient has the first thrombotic episode at a young age, recurrent thromboembolic events, and family history of thrombosis.

An additional mechanism underlying familial thrombotic disease: plasma of patients with thrombotic disorders had a poor anticoagulant response to activated protein C in an assay of the activated partial-thromboplastin time. Protein C is a key element in the regulation of haemostasis and it is produced by liver. It circulates in plasma as an inactive precursor that is activated when thrombin binds to the thrombomodulin receptor on vascular endothelial cells. Once generated, activated protein C inactivates two activated cofactors of the coagulation cascade, factor VIIIa and factor Va, by limited proteolysis, limiting the conversion of factor X to factor Xa and that of prothrombin to thrombin. Protein S is a cofactor for activated protein C in the protein C pathway.

Resistance to activated protein C has emerged as the most frequent abnormality in patients with idiopathic thrombosis and can be diagnosed in 20 to 50% of such patients. These patients have a molecular defect in factor V that substitutes a glutamine for an arginine at amino acid 506 (factor V Leiden), the site at which activated protein C cleaves factor Va. This alteration in the sequence makes the mutant factor Va molecule biochemically resistant to activated protein C. Most patients with resistance to activated protein C are heterozygous for factor V Leiden, but a number of homozygous patients have also been identified. Homozygotes, as expected, are at higher risk for thrombosis than heterozygotes, as are patients with both heterozygous resistance to activated protein C and another prothrombotic defect.

Test for factor V Leiden can be performed by amplifying a DNA fragment containing the factor V mutation site by the polymerase chain reaction in peripheral-blood mononuclear cells. Use of oral contraceptives and pregnancy, including the postpartum state, are important triggers for thrombotic events in patients with resistance to activated protein C or other hereditary thrombotic disorders. The risk of venous thrombosis among those who use oral contraceptives and have resistance to activated protein C increases by about 35 times. We can think of Budd-Chiari syndrome, with a hereditary defect conferring a predisposition to thrombosis.

The aim of additional investigations is to identify a hypercoagulable state: total and free protein S levels, antithrombin III, resistance to activated protein C, a PCR test to detect the factor V Leiden mutation; values for protein C.

The patient should be treated with warfarin. If the patient is not treated, fibrosis and portal hypertension develop rapidly.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. USMLE Step 2 CK: Internal Medicine [Текст]: Lecture Notes / Editors J.J. Lieber, F.P. Noto. — New York: Kaplan, 2019. — 503 p.
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – K.: Медицина, 2018. – 368c.

Semantic module 7. Emergencies in nephrology

14. DIAGNOSTIC CRITERIA AND EMERGENCY TREATMENT OF ACUTE RENAL FAILURE

Time frame – 6 hours.

Professional motivation. Acute renal failure can present in all medical settings but is predominantly acquired in hospitals. The condition develops in 5% of all hospitalized patients, and approximately 0.5% of hospitalized patients require dialysis.

Acute renal failure occurs in approximately 19% of patients with moderate sepsis, 23% – with severe sepsis, and 51% – with septic shock when blood cultures are positive.

Over the past 40 years, the survival rate for acute renal failure has not improved, primarily because affected patients are now older and have more comorbid conditions. Infection accounts for 75% of deaths in patients with acute renal failure, and cardiorespiratory complications are the second most common cause of death. Depending on the severity of renal failure, the mortality rate can range from 7% to as high as 80%. The combination of acute renal failure and sepsis is associated with a 70% mortality, as compared with a 45% mortality among patients with acute renal failure alone.

Place of carrying out: class-room, wards of the nephrology department.

Study objective: to improve the skills in the diagnosis of acute renal failure, to determine management of such patients.

Basic level:

1. To be able to collect complaints, case history, carry out physical examination in patients with acute renal failure.
2. To interpret instrumental and laboratory data in patients with acute renal failure.

Student has to know:

1. Causes and clinical signs of acute renal failure.
2. Biochemical abnormalities, changes in fluid and electrolyte balance of acute renal failure.
3. Mechanisms of the development of the pulmonary oedema, anaemia.
4. Emergency resuscitation in hyperkalaemia, hypovolemia, pulmonary oedema.

The main theoretical questions:

1. Causes and pathogenesis of acute renal failure.
2. Diagnostic criteria of acute renal failure.
3. Blood and urine studies to distinguish prerenal from intrinsic acute renal failure.
4. Management of patients depending on the underlying cause of the acute renal failure.

5. Control of blood pressure in acute renal failure due to accelerated hypertension.
6. Prognosis for patients with acute renal failure.

Assignment for self-assessment

1. What is the cause of acute renal failure classified as “postrenal”?
   a) failure on the basis of inadequate perfusion;
   b) failure on the basis of parenchymal pathology;
   c) failure secondary to obstruction;
   d) everything listed above.

2. A 50-year-old man is hospitalized for acute myocardial infarction. He has decreased cardiac output with hypotension requiring multiple pressor agents. His urine output drops over the next 3 days. His serum urea nitrogen increases to 59 mg/dL, with creatinine of 2.9 mg/dL. Urinalysis reveals no protein or glucose, a trace blood, and numerous hyaline casts. What is the name of this condition? What is the cause? Management.

Answers:

1. c.

2. Prerenal factors (severe hypotension, low cardiac output – cardiogenic shock due to MI) was initial factors of acute renal failure. This patient has oliguria-anuria stage now. Our management must be start from elimination of aetiologic factors (shock), correction of blood pressure. Cessation of any diuresis stimulation, limitation water intake.

REFERENCES

1. Goldman-Cecil medicine [Текст]. V.1 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1489 p.
2. Goldman-Cecil medicine [Текст]. V.2 / L. Goldman, L. Schafer, M. Crow etc. —25-th ed. — Saunders: Saunders Elsevier, 2016. — 1129 p.
3. Internal Medicine [Електронний ресурс]: An Illustrated Radiological Guide / J.A. Al-Tubaikh; by Jarrah Ali Al-Tubaikh. — 2nd ed. 2017. — Cham: Springer International Publishing, 2017. — XVI, 592 p.
4. https://kdigo.org/guidelines/
5. Internal Medicine: Critical Care: textbook (III-IV a.l.)/ O.Ya. Babak, O.M. Bilovol, N.M. Zhelezniakova et al.; edited by O.Ya. Babak, O.M. Bilovol. – К.: Медицина, 2018. – 368с.

Навчальне видання

3512 Методичні вказівки
до практичних занять
з дисципліни “Внутрішня медицина”
Модуль 3. Сучасна практика внутрішньої медицини
Модуль 4. Невідкладні стани у клініці внутрішньої медицини
для студентів спеціальності “Лікувальна справа”
денної форми навчання
(Англійською мовою)

Відповідальний за випуск Л. Н. Приступа
Редактор М. В. Буката
Комп’ютерне верстання Ю. С. Коваленко

Формат 60х84/8. Ум. друк. арк. 10,7. Обл.-вид. арк. 10,92.

Видавець і виготовлювач
Сумський державний університет,
вул. Римського-Корсакова, 2, м. Суми, 40007
Свідоцтво суб’єкта видавничої справи ДК № 3062 від 17.12.2007.