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CHRONIC HEART FAILURE AND MODERN METHODS OF ITS TREATMENT

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Annotation: Chronic heart failure (CHF) is a syndrome of various cardiovascular diseases leading to a decrease in the pumping function of the heart (impaired contraction and, to a lesser extent, relaxation), chronic hyperactivation of neurohormonal systems and manifested by shortness of breath, palpitations, increased fatigue, excessive fluid retention in the body and limitation of physical activity .

Keywords: chronic heart failure , natriuretic peptides , left ventricular ejection fraction , diagnosis , drug therapy , non-drug therapy , comorbidity , decompensation , hospitalization quality criteria , recommendations

Chronic heart failure (CHF) is a syndrome of various cardiovascular diseases leading to a decrease in the pumping function of the heart (impaired contraction and, to a lesser extent, relaxation), chronic hyperactivation of neurohormonal systems and manifested by shortness of breath, palpitations, increased fatigue, excessive fluid retention in the body and limitation of physical activity. The chronic form of heart failure , unlike acute, begins asymptomatically and develops gradually. The initial stage of the disease does not have obvious symptoms, and heart problems can only be identified during a medical examination. Over time, the patient develops shortness of breath and rapid heartbeat during physical activity. In later stages, these symptoms persist even at rest.

Epidemiology : CHF is the most common cause of hospitalization in the elderly; five-year survival rate of patients with CHF: less than 50%; in case of severe CHF, half of the patients die within the first year; CHF reduces quality of life by 80%.

Factors in the development of CHF

- Arterial hypertension (AH) is the main cause of chronic heart failure.
- Coronary heart disease (CHD) is in second place among the provoking factors.
- Cardiomyopathies (including those provoked by infectious diseases, diabetes mellitus, hypokalemia , etc.).
- Valvular (mitral, aortic, tricuspid , pulmonary) and congenital heart defects.
- Arrhythmias (tachyarrhythmias , bradyarrhythmias).
- Diseases of the pericardium and endocardium.
- Conduction disturbances (atrioventricular block).
- High load (for example, with anemia).
- Volume overload (eg, renal failure).

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Pathogenesis of CHF .

1. The main trigger of CHF is a decrease in myocardial contractility and a drop in cardiac output , which causes a decrease in the perfusion of a number of organs and activation of compensatory mechanisms (sympathetic-adrenal system, renin-angiotensin - aldosterone system, etc.).

2. Catecholamines (norepinephrine) cause peripheral vasoconstriction of arterioles and venules , increase venous return to the heart and level reduced cardiac output to normal (compensatory reaction). However, further activation of the sympatheticoadrenal system leads to the progression of CHF (catecholamines activate the RAAS, tachycardia worsens heart filling in diastole and other decompensation reactions).

3. Spasm of renal arterioles + renal hypoperfusion due to CHF ⇒activation of the RAAS⇒ hyperproduction angiotensin II (a powerful vasopressor ; potentiates myocardial hypertrophy and remodeling) and aldosterone (increases sodium reabsorption and plasma osmolality , activates the production of ADH, which retains water). An increase in blood volume, on the one hand, normalizes cardiac output (compensation), on the other hand, it potentiates dilatation and damage to the heart (decompensation).

4. In the development of CHF, an important role also belongs to vascular endothelial dysfunction (decreased production of endothelial vasorelaxing factor), hyperproduction of a number of cytokines: IL, TNF- α(impairs the transport of calcium ions into cells, inhibits PVK dehydrogenase , leading to ATP deficiency, triggers apoptosis cardiomyocytes).

Classification of CHF.

1. By origin : due to volume overload, due to pressure overload, primary myocardial

2. According to the cardiac cycle : systolic form, diastolic form, mixed form

3. According to the clinical variant : left ventricular, right ventricular, biventricular (total)

4. According to cardiac output : with low cardiac output, with high cardiac output

Degree of severity of CHF.

1. According to Vasilenko- Strazhesko :

Stage I (initial) – latent HF, manifested only during physical activity (shortness of breath, tachycardia, fatigue).

Stage II (severe) – severe disturbances of hemodynamics, organ function and metabolism

IIA – moderately severe signs of heart failure with hemodynamic impairment in only one circle

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IIB – severe signs of heart failure with hemodynamic disturbances in the large and small circles

Stage III (final, dystrophic) – severe hemodynamic disorders, persistent changes in metabolism and functions of all organs, irreversible changes in the structure of tissues and organs, complete loss of ability to work.

2. By nyha :

Class I (no restrictions on physical activity) - ordinary (habitual) physical activity does not cause severe fatigue, shortness of breath or palpitations (but there is heart disease!); 6-minute walking distance is 426-550 m.

Class II (mild, slight limitation of physical activity) - satisfactory health at rest, but habitual physical activity causes fatigue, palpitations, shortness of breath or pain; 6-minute walk distance 301-425 m.

Class III (severe, noticeable limitation of physical activity) - satisfactory health at rest, but the load is less than usual leads to the appearance of symptoms; 6-minute walking distance is 151-300 m.

Class IV (complete limitation of physical activity) - inability to perform any physical activity without deteriorating well-being; symptoms of heart failure are present even at rest and intensify with any physical activity; the distance of a 6-minute walk is less than 150 m.

The main clinical manifestations of biventricular CHF:

1. Subjective manifestations: - shortness of breath is the most common and early symptom of CHF; at first it appears only during physical activity, as the disease progresses and at rest; shortness of breath often occurs when lying down and disappears when sitting

- rapid fatigue, severe general and muscle weakness (due to decreased muscle perfusion and oxygen starvation); loss of body weight (due to activation of TNF- αand the development of malabsorption syndrome)

- palpitations (usually due to sinus tachycardia) - initially bother patients during exercise or with a rapid rise in blood pressure, as CHF progresses - and at rest

- attacks of suffocation at night (cardiac asthma) - attacks of severe shortness of breath that occur at night, accompanied by a feeling of lack of air, a feeling of fear of death

- cough - usually dry, appears after or during physical activity (due to venous stagnation in the lungs, swelling of the bronchial mucosa and irritation of cough receptors); in severe cases there may be a wet cough with the release of a large amount of foamy, pink sputum (with the development of pulmonary edema)

- peripheral edema - at first there is a slight pastiness and local swelling in the area of the feet and legs, mainly in the evening, by the morning the swelling disappears; as CHF progresses, edema becomes widespread, localized not only in the area of the feet,

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ankles, legs, but also in the area of the thighs, scrotum, anterior abdominal wall, and lumbar region; extreme degree of edematous syndrome - anasarca - massive, widespread edema with ascites and hydrothorax

- impaired urine output (oliguria , nocturia - predominance of nighttime diuresis over daytime)

- pain, feeling of heaviness and fullness in the right hypochondrium - appear with liver enlargement, caused by stretching of the Glissonian capsule

2. Objectively : a) inspection : - forced sitting or semi-sitting position of patients with their legs down or a horizontal position with the head of the head raised high

- acrocyanosis of the skin and visible mucous membranes, most pronounced in the distal parts of the extremities, on the lips, tip of the nose, ears, subungual spaces, accompanied by cold skin of the extremities, trophic disorders of the skin (dryness, peeling) and nails (fragility, dullness) (due to decreased peripheral tissue perfusion, increased tissue extraction of oxygen, and increased reduced hemoglobin)

- peripheral edema (up to ascites and hydrothorax): located symmetrically, leaving a deep hole after finger pressure, which then gradually smoothes out; the skin in the area of edema is smooth, shiny, soft at first, and with prolonged swelling it becomes dense; at the site of edema, blisters may form, which open and fluid flows out of them, foci of necrosis, skin tears

- swelling and pulsation of the neck veins (with the development of right ventricular failure)

Plesh's symptom (hepatic- jugular test) - when the patient is breathing calmly, pressure is applied with the palm of the hand to the enlarged liver, which causes increased swelling of the neck veins

- atrophy of skeletal muscles (biceps, thenar and hypothenar muscles , temporal and masticatory muscles), loss of body weight, pronounced decrease in subcutaneous fat (“cardiac cachexia”).

b) physical examination : 1) respiratory organs : inspiratory tachypnea ; percussion : dullness at the back in the lower parts of the lungs; Auscultation : crepitus and moist fine bubbling rales against the background of hard or weakened vesicular breathing in the lower parts

2) cardiovascular system : pulse is rapid, low filling and tension, often arrhythmic; Blood pressure is reduced (SBP is greater than DBP); palpation of the apical impulse is diffuse, shifted to the left and down; percussion, the borders of the heart are expanded to the left; auscultation tachycardia and various arrhythmias, often protodiastolic gallop rhythm

3) abdominal organs : bloating (flatulence), palpation - pain in the right hypochondrium; the liver is enlarged, painful on palpation, its surface is smooth, the

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edge is rounded, with large stagnation - systolic pulsation (bulging in systole and decreasing in diastole); ascites.

Diagnosis of CHF.

1. ECG : signs of left ventricular hypertrophy: increase in R wave $_{V5,V6,\ I,\ aVL}$, signs of left bundle branch block, increase in the interval of internal deviation (from the beginning of the Q wave to the apex of the R wave) J > 0.05 sec in V5, V6, levogram , displacement of the transition zone in V1/V2, right ventricular hypertrophy: increase in R $_{III,\ aVF\ ,\ V1,\ V2}$; spelling ; displacement of the transition zone in V_4/V_5 ; complete/incomplete blockade of the right bundle branch; increase in the interval of internal deviation J>0.03 sec in V_1 , V_2 ; shift of the ST interval below the isoline, inversion or biphasicity of the T wave in III, aVF , V_1 , V_2 , various rhythm disturbances, etc.

2. Chest X-ray : redistribution of blood flow in favor of the upper lobes of the lungs and an increase in the diameter of blood vessels (a sign of increased pressure in the pulmonary veins); Kerley lines (caused by the presence of fluid in the interlobar fissures and dilation of the lymphatic vessels of the lungs); signs of alveolar pulmonary edema (a shadow spreading from the roots of the lungs), effusion in the pleural cavity, cardiomegaly , etc.

3. Echocardiography (including with stress tests: bicycle ergometry, 6-minute walking, bicycle ergometry, etc.): allows you to determine the size of the heart cavities, myocardial thickness, blood flow in various phases of the cardiac cycle, ejection fraction, etc.

4. Laboratory data are nonspecific: CBC – there may be signs of anemia (due to decreased appetite of patients, impaired iron absorption); OAM – proteinuria, cylindruria (as a manifestation of “congestive kidney”); BAC – decrease in total protein, albumin, prothrombin, increase in bilirubin, ALT and AST , GGTP, LDH (liver dysfunction); fluctuations in electrolytes (the result of pathogenetic processes in heart failure and diuretic therapy); increased levels of creatinine and urea (“stagnant kidney”), etc.

The goals of treatment of patients with CHF: 1) elimination of symptoms of the disease (shortness of breath, palpitations, increased fatigue, fluid retention in the body); 2) slowing down the progression of the disease by protecting target organs (heart, kidneys, brain, blood vessels, muscles); 3) improving quality of life 4) reducing the number of hospitalizations; 5) prolongation of the patient’s life.

1. General activities:

- avoiding alcohol consumption (since ethanol retains water and is a powerful inducer of apoptosis)
- weight loss in obese patients
- correction of hypertension, hyperlipidemia and diabetes

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- limiting salt and liquid intake (up to 1-1.5 l/ day)
- daily weighing to detect hidden edema
- regular moderate physical activity (walking is best)
- avoid taking PAS (cardiodepressive effect), most calcium antagonists (verapamil - cardiodepressive effect, dihydropyridines - activation of the SNS), NSAIDs (retain fluid, increase blood pressure, reduce the activity of ACE inhibitors and β-blockers).

2. Drug therapy for CHF :

a) main drugs – 5 groups, effectiveness has been reliably proven:

1) ACE inhibitors are drugs No. 1 in the treatment of CHF; improve the clinical course of the disease, reduce the risk of death, slow down the progression of the disease and the onset of decompensation.

Principles for prescribing ACE inhibitors: - do not prescribe if the initial blood pressure is less than 90 mm Hg. Art. (in case of initial hypotension, stabilization of blood pressure is necessary before prescribing ACE inhibitors: semi-bed rest, small doses of corticosteroids, digoxin 0.25 mg orally or intravenously and/or dopamine 2-5 mcg/kg/min, albumin intravenously)

- avoid simultaneous administration of β-blockers and vasodilators
- before using ACE inhibitors, avoid large diuresis and excessive dehydration of the patient
- start ACEI dosing with very small doses and very slow titers, the first dose is at night

More often used: enalapril (starting dose 2.5 mg x 1 time / day , optimal 10 mg x 2 times / day , maximum 40 mg / day).

2) β- adrenergic blockers (BABs) – with long-term administration, they reduce the risk of decompensation and reliably prolong the life of patients (more than ACE inhibitors!), lead to an increase in ejection fraction and pumping function of the heart, inhibit and cause regression of pathological myocardial remodeling , reduce electrical instability, indirectly reduce activity RAAS. NB! A two-phase effect of β- blockers on the myocardium in patients with CHF is characteristic : in the first 2 weeks of treatment, CO may decrease and the course of CHF even worsens somewhat, then, as a result of a decrease in tachycardia and oxygen consumption by the myocardium, hibernating (sleeping) cardiomyocytes restore their activity and CO begins to grow.

Requirements for β-AB therapy :

- cannot be started if the patient has an unstable condition (if administration of diuretics or drugs with inotropic action is required)
- the initial period of therapy should take from 2 to 6 weeks, you should start with small doses (1/8 of the treatment dose), titrating the daily doses
- prescribed for life and preferably in addition to ACE inhibitors

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Use: metoprolol -SR (initial dose 5-12.5 mg/ day , optimal – up to 100 mg/ day); bisoprolol (initial dose 1.25 mg/ day , optimal – up to 10 mg/ day); carvedilol (initial dose - 3.125 mg/ day , optimal - up to 50 mg/ day - the most optimal, is non-cardioselective β- α_1 - adrenergic blocker, antioxidant)

3) diuretics - indicated only for clinical signs and symptoms of fluid retention in the body (i.e., congestive heart failure), mainly together with ACE inhibitors; the criterion for a sufficient dose is a decrease in body weight by 0.5-1 kg/ day ; loop diuretics increase sodium excretion by 20-25% and the excretion of free water, thiazide diuretics increase sodium excretion by 5-10%, but do not increase the clearance of free water. Use: thiazide diuretics (hydrochlorothiazide orally in the morning 25-75 mg), if their effectiveness is insufficient - loop diuretics (furosemide orally in the morning 20-500 mg)

4) cardiac glycosides (only digoxin 0.125 mg 1-2 times a day) - indicated in the presence of atrial fibrillation, in case of sinus rhythm - the fourth drug (after ACE inhibitors, beta blockers, diuretics); use in patients with sinus rhythms in low doses does not improve the prognosis and does not slow down the progression of CHF, but improves the quality of life; It is not advisable to prescribe in patients with heart failure with impaired LV diastolic filling, high-output heart failure, and cor pulmonale.

5) spironolactone orally 25-50 mg once in the morning or in 2 doses in the morning - reduces the risk of overall mortality by 30%, is used

b) additional drugs – drugs, the effectiveness and safety of which require clarification: 1) AT $_{II}$ **antagonists** – used for intolerance to ACE inhibitors (valsartan orally at an initial dose of 40 mg 2 times a day, gradually increasing to a maximum of 160 mg 2 times a day, losartan , irbesartan)

2) **cardioprotectors** - used in short courses to enhance the contractility of the heart (mildronate - limits the transport of long-chain fatty acids across mitochondrial membranes, while short-chain fatty acids can freely penetrate and oxidize; trimetazidine / preductal orally 20 mg 3 times / day - inhibits beta in mitochondria - oxidation of all fatty acids, which promotes the accumulation of activated fatty acids in mitochondria).

c) auxiliary drugs: 1) peripheral vasodilators (nitrates) - only with concomitant angina and pulmonary edema

2) calcium channel blockers (amlodipine only) - “on top” of ACE inhibitors with severe valvular regurgitation , high arterial and/or pulmonary hypertension

3) antiarrhythmics (group III only) – only for life-threatening arrhythmias

4) GCS (prednisolone, methylprednisolone) – for persistent hypotension and as a “therapy of despair” when other drugs are ineffective

5) non-glycoside inotropic stimulants (dopamine , dobutamine) - in short courses during exacerbation and CHF with persistent hypotension

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6) acetylsalicylic acid - used by patients after myocardial infarction

7) indirect anticoagulants (only warfarin) - for heart dilatation, intracardiac thrombus, atrial fibrillation, after operations on the heart valves.

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