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CLINICAL AND DIAGNOSTIC PARAMETERS OF RHEUMATIC HEART DISEASE AND FEATURES OF THE COURSE.

Azizbek F.Valijonov
5th year student of the medical faculty of the Tashkent Medical Academy. Tashkent, Uzbekistan, AzizbekValijonov97@gmail.com;
Nigora A.Akhmedova
Candidate of Medical Sciences, Associate Professor of the Department of Internal Diseases and Endocrinology No. 2, Tashkent Medical Academy. Tashkent, Uzbekistan; nigora.ahmedova1963@gmail.com;
Shirina A.Valijonova
5th year student of the medical faculty of the Tashkent Medical Academy. Tashkent, Uzbekistan, misirova-1998@mail.ru;

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Abstract:

In the next 15-20 years, the clinical picture of RF (rheumatic fever) has changed significantly. Most authors note an extremely low incidence of severe rheumatic carditis, a decrease in mortality and relapses of the disease, a transition to a monocindromic form of the disease, low-symptomatic and latent variants of the course, etc. American researchers note that due to the low prevalence of the disease, most doctors still do not know about complex and changing clinical picture of the disease.

Keywords: Rheumatic fever, chorea, carditis, acute heart failure, cardiac rheumatic fever, chronic heart failure

INTRODUCTION: one of the leading clinical syndromes of RF is acute or subacute polyarthritis, characterized by complete regression of inflammatory changes in all affected joints within 2-3 weeks, and under the influence of modern anti-inflammatory therapy, this period can be reduced to several hours or days. The frequency of polyarthritis ranges from 60 to 100% according to different authors. The most commonly injured are the ankle and knee (90%), forearm and elbow (61%), less often the shoulder (44%), hip (31%), hip, acromial (6%) joints. Atypical manifestations of the articular syndrome are less common: monoarthritis, small joints of the palms of the hands and feet, the cervical spine, and a relapse of arthritis occurs during treatment.

As for primary rheumatic heart disease, opinions differ here. According to most authors, the incidence of primary rheumatic heart disease is very high and ranges from 88.5 to 100%. Meanwhile, according to other authors, at the first attack of rheumatism it is not so high - 50-69% and even very low - 3-38%. Such a discrepancy is difficult to explain only by the polymorphism of clinical symptoms and course variants in different regions and countries of the worldIt is known that with pronounced polyarthritis, the clinical signs of rheumatic heart disease may be little known. Therefore, an unusual interpretation of both the symptoms of carditis and the disease as a whole is not excluded. However, the American Heart Association's information on the correct application of the restored Jones criteria (1944) accurately describes the damage to the heart in primary rheumatic heart disease. It was noted above that at the first attack of rheumatic heart disease, overdiagnosis of RF or a hypodiagnostic state is possible. On the other hand, the WHO research team believes that patients (especially in developing countries) first visit a doctor without reliable anamnestic, clinical and laboratory data that meet the Jones criteria, which have been revised for acute rheumatic heart disease. For greater adequacy of the criteria, it is necessary that in clinical experience it was established that in these cases the diagnosis of RF in such patients can be considered justified only if cardiomyopathy and myopericarditis of other etiologies are excluded, as well as circulatory failure as a result of stagnation in valvular heart disease. Therefore, it should be noted the importance of revolutionary research methods for the early diagnosis of rheumatic heart disease (phonocardiography, echocardiography, Doppler echocardiography).

Rheumatic lesion of the nervous system - chorea minor - occurs mainly in children, less often in

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adolescents, in 6-31% of cases. However, the Brazilian authors showed that the incidence of chorea was quite high and amounted to 44.8%. Rheumatic chorea can only be diagnosed with the exclusion of other diseases of the nervous system, when other criteria for the disease (RF) are not met. Some authors believe that erythema annulare and rheumatoid arthritis have reduced their diagnostic value to a minimum due to declining detection in the last 2-3 decades.

Other authors point out that the noted syndromes (especially Holtz-Simon erythema) are not so rare in case of polysyndromic clinical picture of RF and retain their diagnostic value. So, annular erythema is 4-17%, rheumatic nodes - 1-9%. Nonspecific clinical and laboratory syndromes (fever, arthralgia, acute phase manifestations of inflammation, prolongation of the R-Q interval on the ECG), related to the diagnostic criteria of "small" Joneses, are now also quite common in acute RF. However, the diagnostic value of the listed indicators is preserved only if there is at least 1 "major" criterion.

Particular importance is attached to the careful conduct of laboratory studies aimed at confirming streptococcal infection, which precedes the development of RI. In this sense, serological studies should be given paramount importance in detecting antibodies to group A streptococcus. Most scientists point out the need to identify several types of anti-streptococcal antibodies. Based on this, in RF, the titer of antistreptolysin-o increases only by 80%, if, in the absence of such, its 3 types (antistreptolysin-o, antideoxyribonuclease-b, antistreptohyaluronidase) are investigated, then the accuracy increases to 95-97It should be borne in mind that the level of antibodies to streptococcus, as a rule, rises in the early stages of acute rheumatic fever (ARF) and may be low if several months have passed between the rheumatic fever and the examination. This condition is usually seen more often when there are no other symptoms other than chorea. Such a "regularity" is also found in patients who do not have "big" criteria, except for carditis.

It should be noted that in patients with RF or cardiac rheumatic fever (CRF) (if salicylates or corticosteroids have not been taken for at least 2 months), confirmed by a history according to the WHO expert committee, a preliminary approximate diagnosis of RF in combination with one "large" criterion or only with an increased or increased titer of antibodies against streptococcus. A definitive diagnosis of RF can only be made if it denies intercurrent disease or a complication of the disease (including infective endocarditis).

In subsequent years, the structure of rheumatic fevers also changed somewhat. Most authors point to a decrease in the number of isolated heart defects and an increase in the number of concomitant and combined valve injuries. Part of this pattern can be explained by the fact that the treatment, as well as the diagnosis of the disease itself, has not yet improved. Benevolenskaya M.M. The Brzezovskis believe that there has been some overdiagnosis of CRF in the past, primarily due to an insufficient amount of mitral valve, which is interpreted as a rather complex condition. On the other hand, CRF "contributed" to the reported changes in patients' life expectancy. According to generalized data, the mitral valve occupies the first place in terms of the number of injuries (85-90%). Aortic defects, both separate and combined (with other valve damage), occur in 11-15%. Combined and articular malformations of the mitral valve account for 60-85%, of the aortic valve - 6-11% within the injury of the common valve. Combined mitral-aortic defects occur in 25-35%. The use of new generation devices, namely Doppler echocardiography, made it possible to identify the criteria for rheumatic endocarditis Doppler echocardiography, which can be successfully applied in any age group. Based on this, rheumatic mitral valve endocarditis includes the following features: hump-shaped thickening of the anterior mitral plate, hypokinesia of the posterior mitral plate, mitral regurgitation, correcting the dome-shaped curvature of the anterior mitral plate. Ultrasound in M-mode can detect diastolic flutter of the mitral valves of small amplitude in 50% of children with aortic valvulitis. In some patients, thickening of the EXO signal of the aortic valve is detected. ExoKG showed limited thickening of the aortic valve margins, transient valve prolapse, and aortic regurgitation. One of the important criteria confirming the presence of primary rheumatic heart disease in a patient is the positive dynamics of clinical and paraclinical parameters under the influence of antirheumatic therapy. RF is characterized by a variety of clinical symptoms and a variable course, therefore, since the time of large studies by Sokolsky and Buyo, the diagnostic criteria for this disease have not ceased to improve. Their excellent interpretation was given by the outstanding pediatrician A. A. Kisel in 1940, calling it the absolute symptom complex of the disease. It includes polyarthritis, heart damage, chorea, erythema annulare, and rheumatic nodules. Some time later, the criteria for determining RF were formulated by the American scientist T.D. Jones in 1944. Important additions to the diagnostic criteria were made by A.I. Nesterev in 1963-1973. The criteria for rheumatism, identified by A.A. Kisel, T.D. Jones and A.I. Nesterev, have been successfully used by pediatricians and therapists for many years.

MATERIALS AND METHODS: In foreign countries, for the diagnosis of RF, the criteria proposed by WHO (1989) are used.

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The fact that rheumatic heart disease remains difficult to diagnose even in modern conditions is associated with the following conditions:

- streptococcal genesis of the transferred nasopharyngeal infection is not always clear;
- the titer of antistreptococcal antibodies may be low;
- Throat shots often fail;
- the initial manifestation of carditis is less pronounced;
- signs of systemic inflammation will not be sufficiently pronounced.

In the 21st century, there is an urgent need to revise the classification and presentation of the disease in accordance with internationally accepted terminology (3 table).

Classification of rheumatic fever (2003)

Clinical Options	Clinical view		Exodus	QAE stages SVT* NYHA**	
	Main	Additional			
Acute rheumatic fever. Recurrent rheumatic fever.	Carditis. Arthritis. Chorea. Annular erythema. Rheumatic diseases.	Feverish arthralgias. abdominal syndrome. serositis.	Recovery SYURK: - pure heart. - with heart disease.	0 I Pa Ib III	0 I II III IV

Note: * - according to the classification * - N.D.Strajesko and V.X. Vasilenko
functional class NYHA;

- peripheral fibrosis of the valve layers after inflammation, which can be determined using - ExoKG

In the case of newly diagnosed heart disease, if possible, other causes that form it (infective endocarditis, primary antiphospholipid syndrome, degenerative valvular calcification, etc.)

The term acute rheumatic fever (ARF) (rather than rheumatism) is more appropriate because it draws the physician's attention to the diagnosis of group A beta-hemolytic streptococcus (AGBS) infection, thus emphasizing the need for acute antibiotic use. period for eradication (primary prevention) and prevention of recurrent attacks (secondary prevention). As for the term "primary rheumatic carditis (rheumatism)", clinical experience has expanded this concept to include any initially diagnosed heart defects, both with mitral valve prolapse, a manifestation of hypermobility syndrome, and with large and medium ones, used to refer to arthralgia caused by instability in the joints. In addition, the last 20 years of dynamic echocardiography studies have shown that valvulitis is considered the main component of rheumatic heart disease and can occur together with myopericarditis, manifesting as organic heart murmurs. The defeat of the heart in the form of myopericarditis without valvulitis, as observed, is generally not characteristic of measles, but is diagnosed in comparison with carditis of a different etiology (mainly viral). On the other hand, the clinical symptoms of carditis may be mild in migratory polyarthritis, which is clearly expressed in acute rheumatic fever, or in chorea minor. Therefore, the diagnostic significance of the use of Doppler echocardiography increases, since it is possible to see the anatomical structure of the heart and the state of intracardiac blood flow, including mitral and aortic insufficiency, the first sign of valvulitis. In particular, an outbreak of acute rheumatic fever in the United States showed valvular heart disease in 47% of patients with rheumatoid arthritis and in 57% "isolated" chorea on echocardiography. In view of the above, modified Kisel-Jones diagnostic criteria for acute rheumatic fever were included in the ExoKG surveys (Table 4). In connection with the predominant form of joint damage - migratory oligoarthritis and less often - monoarthritis of large joints, it is necessary to distinguish them from arthritis of other genesis, including post-streptococcal arthritis. Post-streptococcal arthritis has a distinct clinical and epidemiological characteristic: a short latent period after ABGS of tonsillitis/pharyngitis; persistent, usually symmetrical damage to large joints; absence of carditis and chorea, slow response to non-steroidal anti-inflammatory therapy; after treatment, complete regression of the disease without radiological changes in 2-4 months from the onset of the disease. The problem of differential diagnosis of "isolated" (that is, in the absence of other diagnostic criteria) rheumatic chorea in subsequent years was exacerbated by the recently interpreted PANDAS syndrome (childhood autoimmune neuropsychiatric disorders associated with group A streptococcal infection), pediatricians and pediatric psychoneurologists. This syndrome is characterized by: obsessive-compulsive disorders (obsessive thoughts + obsessive situations (unpleasant));

1) the onset of the disease in the prepubertal period (up to 12 years);
2) acute onset and undulating course;
3) chronological affiliation to ABGS, confirmed by a recent microbiological study (isolation of the pathogen in a throat swab and / or an increase in serological asl-o and anti-DNA-ase titer);
4) neurological disorders (choreic hyperkinesis)

Table4
Kiesel-Jones criteria for the diagnosis of acute rheumatic fever (modified by Rra, 2003)

Main criteria	Minor Criteria	Signs of a previous A-streptococcal infection

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Carditis. Polyarthritis. Chorea. Annular erythema. Subcutaneous rheumatic nodules.	Clinical: • arthralgia. • fever. Laboratory: • upper phase reagents: • ECHT (erythrocyte sedimentation rate) • C-reactive protein. Functional: Prolongation of the PR interval on the ECG • • Mitral and/or aortic regurgitation on Doppler echocardiography.	A positive culture of A-streptococcus isolated from the throat, or a rapid test for A-streptococcus antigen. Elevated or increasing titer of anti-streptococcal antibodies (baseline, anti-DNA-ase V)

Comment: The presence of 2 major criteria or 1 major and 2 minor criteria in combination with documented previous group streptococcal infection indicates a high probability of acute rheumatic fever.
Special cases:
1. Isolated ("pure") chorea - with the exclusion of other causes.
2. Late (recent) carditis - prolonged (> 2 months) clinical and instrumental signs of valvulitis - with the exclusion of other causes.
It is noteworthy that in such patients, adequate and continuous (until the symptoms disappear) antibacterial therapy against streptococci (penicillins or oral cephalosporins) leads to a complete regression of neuropsychiatric symptoms.
Despite the fact that the frequency of erythema nodosum and rheumatic nodules is significantly reduced in the pediatric population and practically does not occur in adolescents and older patients, these signs remain highly characteristic of acute rheumatic fever, therefore, while maintaining their diagnostic value, carditis, arthritis as the main criterion and stands with chorea. In acute rheumatic fever, clinical and laboratory syndromes that are currently included in the "small" Kizel-Jones diagnostic criteria are quite common. However, the diagnostic value of these indicators is retained only in the presence of at least 1 major clinical criterion for chorea.

RESULTS: In the diagnosis and differential diagnosis of acute rheumatic fever, it is often difficult to interpret evidence that ABGS infection of the throat is the cause of the disease. The main cases of a more correct interpretation of the results obtained include:
A positive ABGS culture taken from the throat of patients can be both evidence of an active infection and confirmation of asymptomatic carriage of this microbe.
1. A negative microbiological test and a negative rapid antigen test do not rule out active ABGS infection.
2. The diagnostic value of testing immunity to streptococci increases with the simultaneous detection of 2 types of antibodies (antistreptolysin-o, antideoxyribonuclease-V), but we should not forget that the above antibodies can also be observed after an infection caused by β-hemolytic streptococcus.
3. With late carditis or "isolated" chorea, the titer of antistreptococcal antibodies may be normal.
In patients with a rheumatic history, a second attack is treated as a new episode of urticaria, and not a recurrence of the first. In this situation (especially when the diagnosis of carditis against the background of established rheumatic heart disease is very difficult), the diagnosis of acute rheumatic fever is made only when one "major" or only "small" criterion is combined with an increased or increasing titer of antistreptococcal antibodies. The final diagnosis can be made only with the exclusion of intercurrent diseases associated with heart defects and their complications (primarily infective endocarditis).
Chronic heart failure (CHF) is determined based on the classifications of V.Kh. Vasilenko and N.D. Strajesko and NYHA. According to modern neurohumoral theory, the main cause of the development of CHF syndrome is hyperactivation of the renin-angiotensin-aldosterone (RAAS) and sympathoadrenal systems. That is, if the main cause of CHF is the action of an excess of neurohormones, then the blockade of their effects with AAFI should theoretically lead to a 100% positive clinical outcome. In real life, AAFI can reduce the risk of death in CHF patients by only 23% on average. In recent years, many clinical facts have appeared that it is difficult to explain CHF only by an increase in the activity of neurohormones. Along with neurohormones, some other (including immune) mechanisms play a central role in the pathogenesis of the disease. The participation of the immune system in the pathogenesis of CHF may seem incomprehensible only at first glance: it is well known that the body's immune defense "works" not only with the aggression of an infection, but also in response to any stressful effect, including ischemia, hemodynamic overload, shows a reaction during intoxication and etc. Cytokines play an important role in the new model of CHF. Cytokines are low molecular weight protein mediators that provide intercellular communication. Depending on their functions, they are divided into pro-inflammatory and anti-inflammatory cytokines. The first group consists of interleukin (yl) IL-1, IL-2, IL-6, IL-8, IL-12, tumor necrosis factor-a (it-α), interferons. The second group includes IL-4, IL-10, IL-11, IL-7. Cytokines control the amplitude and duration of the inflammatory and immune response, so they are produced and secreted transiently and have a short half-life. They have a very

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low concentration (picogram) effect when they bind to high affinity receptors on the surface of target cells. In most cases, cytokines secreted by cells have a local (autocrine, i.e., on their producer cells and paracrine, i.e., on other target cells) endocrine effect to a very small extent. The action of various cytokines on each other and on the cell can be synergistic, antagonistic or additive (complementary). In CHF, there is an imbalance between pro- and anti-inflammatory cytokines. Pro-inflammatory cytokines (IL-1, IL-3, IL-6, IL-8, o'no-α), cellular receptors for cytokines and their soluble forms (sono-α-P55, sono-α-P75, sL-2P and etc.), adhesion molecules (superfamily of lg immunoglobulins, integrins and selectins), chemoattractants, apoptosis receptors, growth mediators, neopterin. The immune system produces the synthesis of anti-inflammatory cytokines - lymphocytes, monocytes, macrophages, as well as endotheliocytes, smooth muscle cells. Ono-α is a low molecular weight peptide, first isolated in 1975 from the blood serum of patients with malignant tumors, as a substance that ensures the destruction of tumor cells. Ono-α has partially overlapping synergistic and additive activities, plays a very important role in the regulation of the immune response and tissue homeostasis at physiological concentrations, and at high concentrations has a pathological endocrine-like effect. There is evidence that the increase in the concentration of It-α is observed more in non-ischemic than in ischemic CHF. The correct association of Ono-α with heart failure syndrome was demonstrated by Levin et al. in 1990, and they were the first to show that Ono-α levels were higher in patients with severe heart failure than in healthy individuals. several times higher. In the next series of studies, it was shown that there is a correlation between it-α, il-1β and il-μ with the activity of clinical signs of CHF. The hemodynamic and clinical effects of these inflammatory cytokines in heart failure include: negative inotropic effect; remodeling of the heart (irreversible dilatation of chambers and hypertrophy of cardiomyocytes (CMC); endothelium-dependent dilatation of arterioles and increased apoptosis of CMC and peripheral muscle cells. It is assumed that the negative inotropic effects of cytokines may be the cause of hemodynamic symptoms characteristic of CHF - low cardiac output and high cardiac output in combination with dysregulation of peripheral arteriole tone - hypotension characteristic of end-stage heart failure. Endothelium-dependent relaxation of arterioles, an important compensatory mechanism, causes clinical symptoms of RBE, such as increased exercise tolerance in peripheral muscles and reduced skeletal muscle strength and endurance. But the most important in the formation of CHF syndrome is the result of the "long-term" action of pro-inflammatory cytokines, which manifests itself in the gradual breakdown of the extracellular collagen matrix of the myocardium, dilatation of the heart chambers and CMC hypertrophy. A number of studies have shown that these changes underlying the phenomenon of cardiac remodeling are irreversible, and CMC induces a cytokine-induced increase in apoptosis, the onset and progression of CHF, and a poor prognosis for these patients. The Ro'no-α receptor (Ro'no-α-P) is the most unconscious prognostic predictor of unrelated soluble receptors in patients with CHF and surpasses all prognostic markers in its accuracy and specificity, there is even evidence that the stroke-derived fraction exceeds FS CHF and maximum respiratory oxygen consumption (Vo2max). The main reason for the activation of immunity when the inflammatory process is inactive in CHF patients remains unclear. There are 3 hypotheses explaining the causes and mechanisms of the increase in the amount of cytokines (mainly Ono-α) in heart failure:

1) the hypothesis of cytokine production by the myocardium, based on this hypothesis, KMS produce cytokines, and their amount is directly related to the level of myocardial wall tension and stress ("diastolic stress");
2) extramyocardial production of cytokines as a result of myocardial damage and reduced blood flow in the heart is stimulated by tissue hypoxia and excess free radicals. In turn, an excess of cytokines disrupts the mechanism of endothelium-dependent relaxation of peripheral vessels;
3) according to the bacterial endotoxin hypothesis (first shown by Anker, 2002), endotoxins enter the body through the swollen intestinal wall.

Intestinal venous stasis is inevitable with myocardial damage and a decrease in cardiac output, which leads to an increase in the permeability of the intestinal wall for bacteria or their toxins, their passage into the blood and to the SD14 receptor of immunocompetent cells, interaction with, initiates the synthesis of ono-α and other cytokines.

The main drugs in the treatment of CHF, and among them primarily AAFI and α-blockers, have an immunomodulatory effect. In 1993, one of the first E.L. Nasonov, M.Yu. Samsonov demonstrated the immunomodulatory effect of AAFI - captopril in monocyte culture. The anticytokine effect of AAFI is primarily associated with a mediated decrease in the synthesis of angiotensin II, which stimulates the formation of α, while the effect of β-blockers is associated with a decrease in the synthesis of norepinephrine.

CONCLUSION: Thus, the "cytokine" model of the pathogenesis of CHF does not challenge the neurohumoral theory, but expands our understanding

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of the development of CHF. The involvement of inflammatory mediators in the disease scheme expands the "base of the therapeutic approach" and expands the prospects for improving the effectiveness of the treatment of patients with CHF.

LITERATURE:

1. Насонов Е.Л., Александрова Е.Н. Современные стандарты лабораторной диагностики ревматических заболеваний: Клин. рекомендации. М.: БиоХимМак, 2006, 71с.
2. Клиническая ревматология (руководство для пактических врачей).Под ред. проф. В.И. Мазурова. СПб.-ООО "Издательство Фолиант", 2001, 416с
3. Клинические рекомендации. Ревматология.Под ред. Е.Л. Насонова. – М.: ГЭОТАР Медиа, 2005, 288с.
4. Маршалкина Н.А., Косунова Е.Н. Влияние вируса простого герпеса на течение и клинические проявления ревматизма. Тезисы конфер. "Актуальные вопросы практической и теоритической ревматологии", Звенигород, 24-26 мая, 2004, 108 с.
5. Белов Б.С. Клинико-диагностические аспекты современного ревматизма. Тер. архив, №5. – 2007. – С.147-148.
6. Homer C., Shulman S.T. Clinical aspects of acute rheumatic fever (review). J. Rheumatol. - 2001. - Vol. 18. - Suppl. 29. - P. 6-13.
7. YoungE.J.,WeingartenN.M., BaughnR.E.etal. Studies on the pathogenesis of the Jarisch-Herxheimer reaction. Infect. Dis., 146: 606, 2002.
8. WollheimF.A. Enteropathic arthritis. In: Kelley W.N., Harris E.D., Ruddy S., Sledge C.B. Textbook of Rheumatology, 4th ed. Philadelphia, W.B. Saunders, 2003, 985-997.
9. Анохин В.Н., Стулова М.А., Новиков Ю.И., Прохорова И.А. Ревматологические заболевания сегодня. Ревматология.2007. № 4. С. 20-29.
10. Букуп К. Клиническое исследование костей, суставов и мышц: пер. с англ. М.: Мед. лит., 2007. – 320с.
11. Сторожаков Г.И., Кузмина Н.Н., Воронина Н.М. Возможности эхокардиографической диагностики ревматического валвулита и пролапса митрального клапана.Вопр. ревмат. - 2001. - № 1. - С. 18-21.
12. Букуп К. Клиническое исследование костей, суставов и мышц: пер. с англ. М.: Мед. лит., 2007. – 320с.
13. Назарова К.Х., Ширанов И.О. «Цитокиновая» модель патогенеза хронической сердечной недостаточности. Бюлл. Ассоц. Врачей Узбекистана. – Т., 2005. - №3. – С.19-22.
14. Павликова Е.П., Мерай И.А. Клиническое значение IL-6 и FNO-α при ИБС. Кардиология. 2003; 8:68-71.
15. Олбинская Л.И., Игнатенко С.Б. Роль системы цитокинов в патогенезе хронической сердечной недостаточности. Тер.арх. 2001; 12: 82-84.
16. Палеев Н.Р., Палеев Ф.Н. Цитокины и их роль в патогенезе заболеваний сердца. Клиническая медицина, 2004. - №5. – С.4-15.
17. Хохлов А.Л., Спешилова С.А., Кондарева Е.А. Прогноз ифармакоэпидемиологические аспекты сердечной недостаточности при хронической ревматической болезни сердца. Проблемы стандартизации в здравоохранении, 2005. - №6. – С.20-27.
18. Гуревич М.А. Место ингибиторов АПФ в лечении сердечной недостаточности. Клиническая медицина, 2004. - №2. – С.4-8.
19. Насонова В.А., Кузмина Н.Н., Белов Б.С. Классификация и номенклатура ревматической лихорадки (лекция). Науч.-практическая ревматология, №2. – 2004. – С.48-51.