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Contemporary Tendencies of Chronic Rheumatic Heart Disease

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ABSTRACT

Rheumatic fever is a systemic inflammatory disease of the connective tissue, the process of which is located in the cardiovascular system, develops in connection with group A β-hemolytic streptococcal (ABGS) infection, occurs mainly in children (7-15 years) and inclined young people.

V.D. Belyakov's epidemiological analysis shows that ABGS infection appeared at the end of the 20th century and continues to increase as in the previous periods. His analysis made the following prediction: "We are entering the 21st century, and according to the laws of life of streptococcal infection, as in the first half of the twentieth century, it must show its power. In the near future, we will face a highly virulent aggressive ABGS infection, just as powerful as in the early twentieth century. " This prognosis suggests that with the increase in the epidemic "outbreak" of acute respiratory infection (ARI) in the United States and Western Europe, the incidence of ABGS infection is increasing, even fatal.

ARI and chronic rheumatic heart disease (CRHD) are the main cardiovascular diseases for which prevention can be sufficiently effective.

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At present, the prevalence of rheumatic fever has changed significantly. In developed countries, its recurrence rate has decreased, but we think their performance has stabilized at low numbers (Table 1.1).

Table 1.1
Prevalence of rheumatic fever in European countries

Country	Years of inspection	Population (mln.)	Checked age groups	Number per 100 thousand population	Data source
Denmark	1952 1960 1970	4,0 4,5 4,9	All ages	29,5 15,5 10,7	National Health Service
England and Wales	1959 1961	1,2 1,3	1-14 years	15,8 8,8	Health ministry

	1963	1,3		4,7	
Norway	1946 1956 1964	4,0	All ages	72 28 14	Central Bureau of Statistics
Netherland	1959-1963	0,13	All ages	19	
Czechoslovakia	1961 1966 1972	0,1/0,05 0,3/0,07 0,3/0,08	<15/>15 ёш	61,2/42,9 22,8/19,3 8,5/2,6	

The recurrence rate of rheumatic attacks in many European countries and the United States is about 5 per 100,000 population per year. In these countries, rheumatic fever is more sporadic, not epidemic, but this number cannot be said to be small on a national scale. The disease currently occurs as follows: in asymptomatic, and therefore undiagnosed streptococcal infection; in undiagnosed or misdiagnosed respiratory tract streptococcal infection; when streptococcal infection is correctly diagnosed but not treated properly.

Rheumatological fever and rheumatic heart disease (RHD) are a major problem in developing countries in the tropics and subtropics, and are now on par with decades ago in countries in the temperate zone (Table 1.2).

Because group A streptococcus is widespread in all parts of the world, it cannot be eradicated, so rheumatic fever does not go away, but it is clear that it poses a greater or lesser risk to people with streptococcal infection. Therefore, in order to have better control over group A streptococcal infections, further research is needed to further study the pathogenesis of rheumatic fever in more depth.

Already, a very wide range of data on the biological properties of group A streptococci, especially on their cellular structure, has been collected. If most types of streptococcal infection are detected in rheumatic fever, among them types M5, M19 and M24 may be the causative etiological factor. It is these streptococci that are thought to be rheumatoid type or strain. Attempts to prove the possibility of the presence of a rheumatoid component in group A streptococcus have accelerated scientific research in this area and led to the achievement of current knowledge on the subject.

In Tashkent, the incidence of rheumatic fever in women of childbearing age is 29.4 per 1,000, and rheumatic heart disease is 21.2 per 1,000. A.X. Yuldoshev (2000) reports that 2.2% of JRCs occur in rural areas of Andijan region, of which "detected" - 1.65%, "probable" - 0.55%.

The study of streptococcal infections in general has led to the spread of highly invasive streptococcal infection, manifested by sepsis and toxic shock, in the United States and a number of European countries. This life-threatening streptococcal infection usually enters through the skin rather than through the nasopharyngeal ring, however, streptococcal infection strains A have been found to consist of M1 and M3 serotypes in these cases.

Following the emergence of "waves" of rheumatic fever in the United States, serious research has begun on the identification of "rheumatic" strains of streptococcus. In particular, the association of rheumatic fever with separate virulent strains of ABGS has always discussed the issue, especially when rheumatic fever occurs in military units, closed or semi-closed collectives. Although specific serotypes with “rheumatoid potential” have not yet been identified, separate studies in recent decades provide an opportunity to discuss this question.

Analysis of invasive streptococcal infection in the United States from 1985 to 1992 showed that the waves of rheumatic fever and toxic shock syndrome of streptococcal genesis coincided in time and expression. If the period with the highest rate of rheumatic fever was 1985-1987, toxic shock syndrome was observed in 1987-1990.

The above data have made it possible to discuss the issue of the presence of rheumatoid streptococcus. It is well known that such a feature applies only to certain nasal-folk cultures. According to Stollerman (1997), rheumatoid strains are large hyaluronic capsules that form mucoid colonies on the surface of bloody agar

and form very short chains in broth cultures. Another very important feature of rheumatic strains is its high contagiousness, the very rapid transmission of an infectious factor from a patient to a healthy person. A number of studies have suggested that the presence of large molecules of M-protein involved in the pathogenesis of rheumatic fever on the surface of strains is an important indicator of the rheumatogenicity of streptococcus.

Thus, to the great achievements of scientists in the twentieth century, they identified the M-associated protein on the surface of A-streptococcus, consisting of rheumatoid virulent strains, as a I-class epitope different from the non-rheumatic II-class epitope. Subsequently, LGG antibodies were detected in patients with RI with a higher titer than the I class specific epitope. Studies have shown that a microorganism associated with rheumatic fever has symptoms of virulent invasive microbes, but is not strictly specific to the disease. It is also important that the epitopes of the M-protein molecule interact with the heart and other tissues of the body.

Scientists have made significant contributions to the study of the pathogenesis of rheumatic fever. Of particular importance is the discovery of the M5-molecule, which intersects with the epitopes myosin, testicular, brain, sarcolemma membrane. In the M-protein molecule, the proximal location of these epitopes over the special type NH2 epitope is significant. These data strengthened the concept of molecular mimicry, in which the antibody reacts with the autoantigens of the organism against the streptococcal antigen that causes streptococcal infection as the main pathogenetic factor in rheumatic fever. On the other hand, the inductive effect of the autoimmune effect due to the superantigenic property of M-protein is of pathogenetic significance.

Immune (cellular and humoral) reactions are also important in the induction of rheumatic fever. The humoral response to various streptococcal antigens is well known (antistreptolysin-O, antistreptokinase, antistreptogialuronidase, etc.). Antistrepto-lysine-O may be involved in the formation of a circulating immune complex (CIC), which correlates with changes in atrioventricular dissociation and blockade in the ECG. RI also showed the presence of antibodies to IgG cardiolipin (54% of patients) in other humoral immune reactions. High and significant levels of these antibodies have led to the discussion of the hypothesis that neopterin markers in patients with heart disease are valvular injuries.

In the development of rheumatic fever, scientists have made a significant contribution to the development of a new direction of immunopathological reactions, such as activation of IL-1β, rONO-α and neopterins by monocyte / macrophage and T-lymphocytes due to an increase in rIL-2R. These data allowed cellular mediators to discuss the important role, in particular, in the pathogenesis of rheumatic fever.

It is interesting to note that high levels of neopterin and IL-lα are associated with rheumatic fever with heart valve injury during the first attack.

Thus, the pathogenesis of rheumatic fever can be caused by complex immunopathological processes, rheumatoid streptococci, the components of which are activated by cellular and humoral immune responses, autoimmune reactions, including autoantibodies, circulating and fixed immune complexes.

It is important to note that it is group A M3, M5 and M18 types of rheumatoid streptococci that have an immunizing effect and retain the necessary epitopes that cause the immune-mediated pathological process. The results allowed Stollerman to describe rheumatic fever in 1997 as a post-infection complication of pharyngitis caused by group A streptococcus (angina), in which those prone to streptococcus with epitopes similar to human body tissues (joints, heart, brain, skin) is an autoimmune response and cross-reactivity to epitopes.

The development of rheumatic fever after 0.3% sporadic and 3% epidemic nasopharyngeal A-streptococcal infection is assumed that the patient has a specific susceptibility to this infection, a specific predisposition to the formation of rheumatic fever and rheumatic heart disease.

As early as the early twentieth century, A.A. Kisel points out that there is a familial predisposition, a multifactorial model of rheumatism is formed in the eighties, and it is based on a combination of genetic and environmental factors. According to them, familial rheumatism is 6 times higher than in the population.

The discovery of V-lymphocyte alloantigen using monoclonal antibodies D8 / 17 was one of the major advances in understanding the susceptibility to rheumatic fever. N.A. Shostak showed D8 / 17 carriers in patients with carditis and polyarthritis (95.7% and 93.9%, respectively) and a slight decrease in chorea

(75%) in the large clinical material. Family studies reported that this marker was collected in probands mothers (63.6%), children (50%), and sibs (54%).

Thus, the realization of the infection caused by group A streptococcus - familial complex, multifactorial factors and V-cell alloantigen in rheumatic fever is important, and the task of revealing their specific mechanisms is still ahead.

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