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Саҳифаловчи: Хуршид Мирзахмедов

Контакт редакций журналов. www.tadqiqot.uz
ООО Tadqiqot город Ташкент,
улица Амира Темура пр.1, дом-2.
Web: http://www.tadqiqot.uz/; E-mail: info@tadqiqot.uz
Тел: (+998-94) 404-0000

Editorial staff of the journals of www.tadqiqot.uz
Tadqiqot LLC The city of Tashkent,
Amir Temur Street pr.1, House 2.
Web: http://www.tadqiqot.uz/; E-mail: info@tadqiqot.uz
Phone: (+998-94) 404-0000

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МУНДАРИЖА \ СОДЕРЖАНИЕ \ CONTENT

SURGERY

MAXILLOFACIAL SURGERY

БИОМЕДИЦИНА ВА АМАЛИЁТ ЖУРНАЛИ
ЖУРНАЛ БИОМЕДИЦИНЫ И ПРАКТИКИ
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Nargiza Mirza-Bakhtiyarkhonovna Abdurakhmanova
Khalmurad Sadullaevich Akhmedov
Tashkent Medical Academy

REACTIVE ARTHRITIS - A MODERN VIEW OF THE PROBLEM

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ANNOTATION

The review article provides up-to-date information about the etiology, pathogenesis, classification, clinical picture and diagnosis of the disease. The issues of drug treatment of reactive arthritis are covered.

Keywords: reactive arthritis, chlamydia, treatment. Abbreviations: ReA - Reactive arthritis, GC – Glucocorticosteroids.

Наргиза Мирза-Бахтиярхоновна Абдурахманова,
Халмурад Садуллаевич Ахмедов
Ташкентская медицинская академия

РЕАКТИВНЫЙ АРТРИТ- СОВРЕМЕННЫЙ ВЗГЛЯД НА ПРОБЛЕМУ

АННОТАЦИЯ

В обзорной статье приводятся современные сведения об этиологии, патогенезе, классификации, клинической картине и диагностике заболевания. Освещены вопросы медикаментозного лечения реактивного артрита.

Ключевые слова: реактивный артрит, хламидии, лечение.

Наргиза Мирза-Бахтиярхоновна АБДУРАХМАНОВА
Халмурад Садуллаевич АХМЕДОВ
Тошкент тиббиёт академияси

РЕАКТИВ АРТРИТ- МУАММОГА ҚАРАТИЛГАН ЗАМОНАВИЙ НИГОХ

АННОТАЦИЯ

Мазкур мақолада реактив артритнинг этиология, патогенез, классификация, клиник манзара ва диагностикасига оид замонавий маълумотлар берилган. Реактив артритнинг медикаментоз даволаш усуллари, мавжуд муаммолар, хал қилинмаган вазифалар ёритилган.

Калит сўзлар: реактив артрит, хламидия, даволаш

The medical and social significance of chronic arthritis is determined by the constant increase in their occurrence, the tendency to chronization and a steady progressive course with a sharp decrease in the quality of life of patients, high medical and social costs of society (VA. Nasonova, O. M. Folomeeva, 2001). Joint diseases occupy one of the first places among the causes of disability in the population aged 16-72 years and are the main cause of disability in people over 65 years of age [7]. Among chronic inflammatory diseases of the joints, one of the most common is reactive arthritis [8]. Reactive arthritis (ReA) is an immune — inflammatory joint disease that occurs within one month after an intestinal or genitourinary infection, associated with histocompatibility antigens HLA B-27, and is a systemic clinical manifestation of this infection [12]. The incidence of reactive arthritis in the general population is 0.1% [10]. In rheumatological hospitals, the proportion of patients with reactive arthritis is 10%, and chronic forms of the disease are accompanied by a significant violation of the functional activity of the joints due to the development of severe complications, often (up to 42% of cases), leading to disability [21]. An increase in the incidence of ReA, a predominant lesion of young people, frequent chronization of the pathological process, and not always satisfactory treatment results determine the medical and social significance of this problem. In addition, the widespread occurrence, high frequency of resistance to therapy, frequent formation of severe forms, and lack of effective prognostic criteria for early diagnosis of reactive arthritis determine the relevance of studying this problem [1] for a Long time, the term "Reiter's disease" (uretero-oculo-synovial syndrome) was used to determine ReA associated with urogenital infection. In recent years, discussions have been held on the terminology of ReA and Reiter's disease [8].

Reiter's disease is a systemic disease, inflammatory joint damage associated with sexually acquired chlamydia trachomatis infection, and is combined with urethritis and prostatitis in men, cervicitis and salpingitis in women, as well as eye and skin damage. If there is a lesion of the urogenital system, joints, eyes and skin, we speak of the Reiter tetrad, if there is no skin lesion, we speak of the Reiter triad. This disease was described by the military doctor Rance Reuter in 1916. There is a sporadic form, or Reiter's disease, associated with a sexually acquired infection, and endemic (Reiter's syndrome), associated with a trigger infection acquired in a non-sexual way (post-dysentery, post-enterocolitic) [21]

There are 2 main forms of ReA – urogenital and postenterocolitic (enterogenic). In addition, there are 2 variants of the course – sporadic and epidemic. The sporadic variant usually observed in urinogenous the form ReA and epidemic – when potentialities. This variant occurs in closed groups (for example, in youth camps or army units), usually occurs in the summer and is associated with violation of sanitary conditions.

Etiology. Etiological factors (triggers) of ReA are: Chlamydia trachomatis, Yersinia enterocolitica, Yersinia pseudotuberculosis, Campylobacter jejune, Salmonella enteriditis, Salmonella tiphimuri - and Shigella flexneri.Arthritis, the development of which is associated with streptococcal, borrelious, brucellosis, and viral infection, does not belong to Rea [15, 24].

When proving a trigger infection, the diagnosis must have an etiological characteristic. For example, when a chlamydia infection is detected, arthritis is called chlamydia-induced ReA. Chlamydia trachomatis, which is a pathogenic, obligate intracellular gram-negative bacteria, causes chlamydia-induced arthritis [23].

The main role in the **pathogenesis** of ReA is played by immunopathological processes associated with the development of a hyperimmune response to an infectious agent located inside the joint or extraarticularly. Trigger factors (for example, chlamydia and Yersinia) can initiate a cytotoxic T-cell response, which leads to proliferation and activation of CD8+ T-lymphocytes, leading to damage to the synovial membrane and, consequently, the development of arthritis [2,7].

There is a pathogenetic hypothesis of “antigenic mimicry” of bacteria that share antigenic determinants with the HLA system, which provides a cross-reaction of the resulting antibodies not only with foreign, but also with their own antigens. The role of the HLA-B27 antigen in the development of ReA is also explained in the theory of "arthritogenic peptide", the essence of which

is that HLA-B27 presents an arthritis-inducing peptide (a component of the cell wall of trigger microorganisms) to cytotoxic T-lymphocytes from the CD8+ population, triggering an immuno-inflammatory response [28].

In chlamydia-induced arthritis, the penetration of chlamydia into the human body occurs during sexual contact, which leads to urethritis, prostatitis, vesiculitis in men, as well as endocervicitis, urethritis and salpingitis in women. The primary focus of chlamydia infection can be proctitis and pharyngitis (with sexual perversions). A non-sexual pathway is possible, such as pool conjunctivitis. Infection of newborns can occur from a sick mother in utero or during childbirth [27]. Further development of events depends on the genetic predisposition of a particular individual (the presence or absence of HLA-B27), the state of non-specific protection factors (complement activity, bactericidal ability of blood serum, etc.). if it is impossible to control the spread of infection, infectious antigenemia occurs. These processes form the basis of the first stage of the disease - infectious and toxic. In this stage, the formation of antibodies, circulating immune complexes, etc. The effectiveness of anti-chlamydia antibiotics during this period is maximum. If the infection persists, the primary focus is a source of constant antigenic stimulation (autosensitization) and an immune response occurs not only to bacterial, but also to its own modified antigens. The second stage begins - immune inflammation. The production of antibodies and circulating immune complexes increases, anti-tissue antibodies appear (an autoimmune reaction occurs). Anti-chlamydia antibiotics are ineffective during this period. The inflammatory process is mainly caused by immunopathological processes [26].

Features of the pathogenesis of the disease largely depend on the individual predisposition of the patient. So, if a patient has HLA type B-27 due to the predominant activation of CD8+ - T-lymphocytes, the process usually turns into a chronic form, severe lesions of the spine and iliosacral joint appear, in many ways similar to those that occur in patients with ankylosing spondylitis. Patients with Th1 type of immune response (CD4+-t-lymphocyte activation prevails) most often develop peripheral arthritis [15].

The clinical picture of ReA has some features. Arthritis is usually asymmetric, involving the joints of the lower extremities (ankle, knee, and foot joints), but other peripheral joints may also be affected. Usually a small number of joints are affected, and oligoarthritis occurs. Often, the sacroiliac joints (sacroiliitis) and the spine (spondylitis) are involved in the pathological process. Very characteristic intesity most frequently involved areas of the heels (achillodynia, achillitis, achillobursitis, plantar fasciitis), tendovaginitis, dactylitis, "sausage-shaped" fingers occur. Due to damage to the joints of the feet, inflammation of the ligamentous apparatus of the feet, flat feet gradually develop [15].

It is possible to develop systemic manifestations (aortitis, myocarditis, pericarditis, conduction disorders, glomerulonephritis, pleurisy, polyneuritis, etc.) [7].

The clinical picture of Reiter's disease is diverse and varies depending on the duration of the disease. After sexual intercourse, in which there was an infection with chlamydia infection, and damage to the urogenital system, usually 2-3 weeks pass. In men there is a urethritis and prostatitis, in women - cervicitis, adnexitis. However, in some cases, the pathology of the genitourinary organs is asymptomatic or poorly symptomatic, often not noticed by patients. Joint syndrome develops within 1-6 weeks after the onset of urethritis. Simultaneously with the joint syndrome or after it, eye pathology develops (conjunctivitis, iridocyclitis). The classic Reiter triad is formed (damage to the genitourinary system, joints, and eyes). With the development of skin manifestations ("psoriasiform" rashes, "blennorrheic" keratodermia), the Reiter tetrad is formed [19

The joint syndrome in this disease is dominant. Its severity determines the course and severity of the disease. The options for destruction of various joints: transient arthralgia, synovitis, erosive arthritis, osteoarthritis, ankylosis of the joints. Arthritis can occur acutely, accompanied by severe pain and General symptoms (fever, chills, weakness). There are subacute variants of joint syndrome with moderate exudative changes in the joints. The joints of the lower extremities (knee, ankle), and the joints of the feet are more often affected. There are no "exception" joints for Reiter's disease, therefore, any joint can be involved in the pathological process. Usually, joint damage is asymmetric,

and the joints are consistently involved in the inflammatory process. Often defined as the "ladder" symptom-gradual involvement of joints from the bottom up, as well as the "spiral" symptom-ascending involvement of dissimilar joints. At the onset of the disease, mono - and oligo - arthritis is more common, with further progression, polyarthritis develops. Arthritis is accompanied by exudative processes, synovitis occurs, swelling of the joint area, and soft tissue swelling are observed. Simultaneously with arthritis, various periarticular processes and enthesopathies occur, which increase pain. Almost one in five patients with Reiter's disease are affected by hip joints, develop coxitis, leading to significant functional disorders. Often inflamed muscles (myositis), tendons (tendinitis, tendovaginitis). In Reiter's disease, there is a pronounced muscle atrophy, which is not associated with immobilization of the limbs due to joint inflammation, but is the result of neuro-trophic disorders [27].

A mandatory clinical sign of the disease is urethritis. Its manifestations can be violent (dysuria, abnormal discharge from the urethra), but more often urethritis is asymptomatic or low-symptomatic, and therefore patients do not receive treatment.they pay attention to it.

Almost every patient with chlamydia-induced arthritis under targeted urological examination reveals chronic prostatitis, which in most cases is asymptomatic. Much less frequently, patients develop these lesions of the urinary organs like epididymitis, Cabernet will cooperit etc.

Eye pathology most often occurs in one of three ways: conjunctivitis, keratitis, or uveitis. The clinical course of conjunctivitis depends on the stage of the disease. When conjunctivitis occurs in the infectious stage, the inflammatory process is usually two-sided, characterized by complaints of a feeling of pain in the eyes, photophobia, lacrimation, proceeds benign, and is characterized by spontaneous self-healing. In the autoimmune stage, a chronic inflammatory process of the conjunctiva develops, which is poorly symptomatic, and is often chronicled [28].

At the initial stage of the disease, visceral manifestations may cause myocarditis and pericarditis. Often there is aortitis and endocarditis of the semilunar aortic valves, which leads to the development of aortic insufficiency. Possible lung damage with the development of pneumonia, pleurisy, kidney damage such as glomerulonephritis, hepatopathy and hepatomegaly, damage to the Central and peripheral nervous system. The long-term course of the disease leads to the development of systemic amyloidosis [24].

According to the duration of symptoms of musculoskeletal system damage, ReA is divided into acute ReA -with a duration of up to 6 months and chronic ReA - with a duration of more than 6 months.

There is also the following classification of variants of the course of Reiter's disease [6]: acute course-damage to the musculoskeletal system passes completely within 6 months, at the same time laboratory parameters are normalized. Prolonged course - the duration of joint syndrome and laboratory changes from 7 to 12 months. Chronic course - the duration of damage to the musculoskeletal system and laboratory disorders for more than a year.

The classification criteria adopted at the 4th International workshop in Berlin (Workshop Report, 1999) are used for the diagnosis of ReA. They are divided into large and small.

ReA classification criteria [14]:

1. Large criteria:

1.1. arthritis (2 criteria out of 3):

- asymmetric,
- mono-or oligoarthritis,
- arthritis of the joints of the lower extremities.

1.2. previous clinically expressed infection:

- urethritis/cervicitis (dysuria) preceding arthritis for up to 8 weeks,
- enteritis that precedes arthritis for up to 6 weeks.

2. Small criteria:

2.1. laboratory confirmation of the trigger of the infection.

A reliable diagnosis of ReA is made if both large criteria and small criteria are present. ReA is considered probable if there is a first large criterion and a small criterion, as well as if there are only large criteria.

Diagnostics. In 2003, at the European Congress of rheumatologists, the European working group for the study of seronegatous arthritis provided a list of five infectious agents related to ReA triggers (Ch. trahomatis,Y, enterocoliijca, Y. pseudotuberculosis, S. enteritidis, Sh. Flexneri and C. jejuni.), arthritis developing after viral, bacterial and spirochettose pathogens recommended to be classified as "arthritis associated with with infections (post-infectious), except septic arthritis [6]

Criteria for the diagnosis of reactive arthritis. For the diagnosis of reactive arthritis the criteria of B. Amor are used for a long time.

1. Aseptic arthritis with any following features:

a) monoartrit, asimmetrichnyi oligoarthritis

b) oligoarthritis with pain in the spine and in the sacroiliac joints, pain in heel; oligoarthritis with affection of the joints of the finger or toe in the form of "sausages"

C) Detection of pseudovascular inflammation without hyperplasia of synovial cells in biopsy of synovial membrane

d) Dysuria preceding the arthritis, less than a month

d) Diarrea, preceding the arthritis, less than a month

f) Conjunctivitis that accompanies arthritis or precedes its onset in less than a month

g) Characteristic damage of the skin and mucous membranes: keratoderma, aphthus in the oral cavity, icarcinaric balanitis

Detection of HLA-B27 antigen or cases of reactive arthritis, ankylosing spondylitis, or seronegative oligoarthritis of immediate relatives. Detection by bacteriological or serological methods of one of the microorganisms responsible for the development of reactive arthritis.

Laboratory diagnostics:

General blood analysis: there are no specific changes. Possible increase in ESR, leukocytosis, anemia, thrombocytosis.

Biochemical blood test: increased content of C-reactive protein, fibrin. The rheumatoid factor is not determined in the diagnostic titer.

General urinalysis: signs of an inflammatory process (leukocyturia, proteinuria) are characteristic. To detect urethritis, it is advisable to perform a three-step urine sample, while pathological changes are most clearly detected in the first portion.

HLA-B27 antigen detection: HLA system antigens are determined using a complement-dependent lymphocytotoxic test (Terasaki method) or polymerase chain reaction. The presence of the HLA-B27 antigen is found in 60% of patients with ReA and in 80-95% of patients with Reiter's disease.

The study of synovial fluid: it is characterized by inflammatory changes of the synovial fluid leukocytosis and neutrophilia, low viscosity. It is necessary to examine the crystals of uric acid, as well as the seeding of the synovial fluid in the nutrient medium to exclude septic arthritis.

Identification of a trigger microorganism: the most evidence-based identification of a trigger infection is by the culture method. Indirect signs of evidence of infection are immunological methods (determination of antibodies to infectious agents or their antigens), as well as amplification of fragments of nucleic acids at least two different methods, one of which is PCR. To prove the role of enterobacteria in the etiology of ReA, it is necessary to perform fecal culture and serological reactions (determination of the level of antibodies in the blood serum) [2].

Detection of chlamydia infection is carried out in the following ways [11]:

1. Microscopy is performed using polychrome aniline dyes.

2. Direct immunofluorescence analysis.The sensitivity of the method is about 80%, the disadvantages of the method are the subjectivity of evaluating the results of the study, as well as the possibility of obtaining false - positive and false-negative results.

3. Enzyme Immunoassay is based on the detection of specific antibodies in the blood serum, in the secret of the prostate gland. In the acute process, class M immunoglobulins are produced, and

these antibodies can be detected within the first week of the onset of the disease, as well as in the first days when the chronic process worsens. Then the number of class g immunoglobulins gradually increases (approximately within 15-20 days). When reinfection (re-infection) or reactivation (activation of own infection) occurs, titers of class g immunoglobulins increase.

4. Polymerase chain reaction is based on the amplification of a fragment of microbial DNA using DNA polymerase.

5. The Culture method is performed using cells that are sensitive to chlamydia: McCoy, Hela-229, VNK-21, etc. The sensitivity of the method is about 80%, the specificity is 100%.

Thus, to detect chlamydia, it is necessary to use at least 2 methods of its diagnosis, one of which is a polymerase chain reaction. The "gold" standard for the diagnosis of chlamydia is the culture method [17].

Instrumental diagnostics. All patients undergo x-ray examination of the peripheral joints, spine and sacroilliac joints. In the acute process, radiological signs of damage to the articular structures are usually absent. With severe synovitis of the peripheral joints, the expansion of the joint gap is determined. It is very likely to detect edema of the soft tissues of the joint (periarthritis), subentesial osteitis, and with long-term enthesitis, subentesial erosions may occur (erosion at the site of attachment to the bones of ligaments and tendons). In the chronic process, subchondral sclerosis, periostitis, and bone proliferation are formed. It is not excluded in ReA and bone erosive process, which often occurs in small joints of the feet. The formation of osteophytes, calcaneal spurs (osteophytes on the posterior or lower surface of the calcaneal bones), vertebral syndesmophytosis, and paraspinal calcifications (ossifications) is characteristic [12].

Treatment. The main principles of treatment of reactive arthritis of any etiology can be attributed to the following main components: antibacterial therapy, pathogenetic therapy of arthritis, including anti-inflammatory therapy aimed at suppressing the inflammatory process; therapy that controls the course of the disease in its chronic, disabling course, the use of basic means, methods of local therapy [7].

In addition to the main methods of treating ReA, there is auxiliary therapy: extracorporeal methods; physiotherapy, treatment of extra-articular manifestations.

The experiments of the positive effect of antibacterial drugs on the course of reactive arthritis are being described in literature.[3]. Antimicrobial therapy is performed in accordance with the detected pathogen before the infection is eradicated. These results can be taken into account, but no conclusions should be drawn. After treatment, monitoring of cure is mandatory. When treating sexually transmitted infections, it is necessary to treat the patient's sexual partners.

In chlamydia-induced arthritis, the following groups of antibiotics are used: tetracyclines (doxycycline), macrolides (azithromycin, josamycin, roxithromycin, clarithromycin, spiramycin), fluoroquinolones (ofloxacin, ciprofloxacin, levofloxacin, moxifloxacin). Simultaneously with antibacterial agents, flukonazole and other antifungal drugs, multivitamins, and biologics are prescribed.

Despite the presence of a large number of antibacterial agents directed against chlamydia, eradication of the pathogen is not always possible. Limited opportunities for antibacterial therapy of chlamydia are associated with the biological characteristics of this microorganism, in particular, with the existence of a population of elementary forms of chlamydia located in the intercellular spaces of epithelial cells and not subjected to phagocytosis. In addition, chlamydia can be modified into stable forms directly under the influence of antibacterial drugs. Chlamydia strains that are initially resistant to standard antibacterial drugs (tetracyclines, macrolides, etc.) are described. The duration of the course of antibacterial therapy is 1.5-2 months, sometimes more. Taking into account the high risk of developing complications of antibacterial therapy with long-term continuous treatment, a scheme of "pulse therapy" with azithromycin 1.0 gram once a week for 3 weeks has been proposed, which has demonstrated its effectiveness[18].

The most important and complex issue in the treatment of reactive arthritis is determining the timing of antibiotic therapy. Depending on the tasks set, the following schemes of antibacterial therapy are distinguished [3] •

* 10-14 days-treatment is aimed at temporarily suppressing active infection in the focus of inflammation (genitourinary system or intestines); 4-8 weeks of treatment is aimed at achieving short-term remission, however, in the next 6 months, relapses of the disease are observed in 50% of patients [4]

8-12 weeks of treatment are aimed at achieving stable remissions, according to available data, the duration of remission was maintained for more than 2 years [5, 20].

Courses of antibacterial treatment from 8 to 12 weeks are considered as a disease-modifying effect in ReA [8], in contrast to short courses, the results of which are only temporary suppression of inflammation in the infectious focus.

In the enterocolitic variant of Rea, the following antibiotics may be used: levomycetin 500 mg 3 times a day, tetracycline 500 mg 4 times a day, ciprofloxacin 500 mg 2 times a day [13].

The results of meta-analysis of studies on the effectiveness of antibiotics in the treatment of ReA are of interest [5]. We analyzed 12 studies and found that the effect of antibiotics on achieving remission in ReA is quite heterogeneous. The analyzed studies did not establish the effect of antibiotics on joint score, pain, or global health assessment. Antibiotics were associated with gastrointestinal side effects in 97% of cases [22].

Anti-inflammatory therapy is performed using non-steroidal anti-inflammatory drugs (NSAIDs) and glucocorticoids (GC).

Non-steroidal anti-inflammatory drugs (NSAIDs) are used for ReA, but their therapeutic effect is limited to symptomatic non-specific analgesic and anti-inflammatory effects, so the choice of the drug should be determined based on the safety of treatment.

Glucocorticosteroids in ReA with their systemic use have shown their low effectiveness. Glucocorticoids are used as local therapy in the presence of synovial effusion. In case of polyarthritis with pronounced exudative phenomena, high laboratory activity of the disease, pulse therapy of GC 500 mg once a day is recommended once or in case of severe course of the disease - for three consecutive days. In the presence of Antietam GC injected into the area of inflamed entezam. In the presence of nephritis, heart damage, aorta and high laboratory activity, GC is prescribed inside short courses in medium doses (20-40 mg of prednisone). When the eyes and mucous membranes are affected, GC is applied locally.

In the treatment of ReA, sulfasalazine (2 g/day for 6 months) is also used as a "basic drug", the effectiveness of which has been confirmed in double-blind placebo-controlled studies. In the treatment of ReA, there are descriptions of individual cases of the use of drugs used for other chronic arthritis: methotrexate, gold preparations, levonazole, azathioprine [20], however, their therapeutic effect, due to the limited experience of available clinical descriptions of cases, can not be considered established to date.

When the disease is highly active, extracorporeal methods (hemosorption, plasmapheresis, extracorporeal photochemotherapy) are indicated[12].

From physiotherapy procedures, it is recommended to use phonophoresis of drugs (ha and NSAIDs), diadynamic currents, ultrasound with ha, magnetotherapy, laser therapy. Patients with ReA should engage in physical therapy; massage of the muscles in the affected joints improves the functional prognosis of the disease and prevents the development of muscle atrophy [10].

In Rea, several open-label studies have been conducted on the use of TNF-a inhibitors in patients who are refractory to conventional therapy, which were initiated taking into account the high effectiveness of genetically engineered biological drugs in other spondyloarthritis. High therapeutic potential of such therapy without reactivation of trigger infection has been reported. Indications for its implementation are the chronic course of the disease, the inflammatory process in the spine, multiple enthesitis and dactylitis, the ineffectiveness of sulfasalazine, methotrexate and local glucocorticoids [16]. Currently, it is difficult to give an objective assessment of this method of therapy, since a small number of controlled studies have been conducted.

Thus, ReA remains the problem of diagnosing the etiological factor, as well as aspects of early eradication of trigger infection and adequate anti-inflammatory therapy of joint syndrome. These

circumstances dictate the appropriateness of microorganisms (PCR). For detection of chlamydia infection, you must use.

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