



JUVENILE IDIOPATHIC ARTHRITIS

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Abstract

Juvenile idiopathic (rheumatoid) arthritis (JIA) is a chronic, severe progressive disease of children and adolescents with a predominant lesion of the joints of unknown etiology and complex, autoimmune pathogenesis, which leads to gradual destruction of the joints, is often accompanied by extra-articular manifestations, disrupts the growth and development of the child, and negatively affects the quality of life [2]. Juvenile rheumatoid arthritis remains the most commonly detected disease in childhood Rheumatology. It is accompanied by total damage to connective tissues, which leads to a narrowing of the joint space, the formation of erosive areas on the surface of the joints, atrophic changes in muscle fibers. Basically treat juvenile rheumatoid arthritis in children of different ages in specialized medical institutions or special departments of public hospitals, where specialized doctors work, there is the necessary diagnostic and therapeutic equipment. Childhood juvenile rheumatoid arthritis is a rapidly progressive inflammatory disease in which children and adolescents under 16 years of age affect the joints.

The disease is characterized by destructive changes, severe pain, increasing limitation of motor functions.

Girls get sick 2 times more often than boys. The exact causes of the development of this disease are unknown. The etiology of JIA is multifactorial, with particular emphasis on hereditary and environmental factors such as infections. According to statistics, the incidence of rheumatoid arthritis in relatives of the 1st degree of kinship is higher than in the population. Associations of JIA with Ag histocompatibility (HLA) — A2, B27, B35 and HLA DR-5, DR-8 were revealed. The most common environmental factors are viral or bacterial-viral infection, injuries, insolation or hypothermia, psychological stress and even preventive vaccinations. At the heart of the disease is the activation of the cellular and humoral link Immunity. Alien or altered own Ag is perceived and processed by macrophages or other antigen-presenting cells that present it to T-lymphocytes, leading to the activation and proliferation of T-lymphocytes. Macrophages, activated T-lymphocytes, fibroblasts, synoviocytes produce pro-inflammatory cytokines that cause a cascade of pathological changes with the development of progressive inflammation in the joint cavity and systemic manifestations of the disease. The production of a large number of autoantibodies indicates the involvement of the B-cell link of the immune system. Thus, uncontrolled reactions of the immune system lead to the development of chronic inflammation, non-reversible JIA can be considered a diagnosis Exceptions. It is defined as arthritis of unknown etiology, present for 6 weeks, occurring before the age of 16, with the exclusion of other diseases. There are the following flow options:

- Systemic arthritis is arthritis accompanied by or with prior documented fever for at least 2 weeks in combination with two or more of the following symptoms:
 - or Transient, volatile erythematous eruptions;
 - or Serositis;



or Generalized lymphadenopathy;

or Hepatomegaly;

or Splenomegaly.

- Polyarthritis: negative in the Russian Federation is determined with the defeat of 5 or more joints during the first 6 months of the disease;
- Polyarthritis: positive for the Russian Federation is determined with the defeat of 5 or more joints during the first 6 months of the disease, the presence of a positive RF in two tests for 3 months;
- Oligoarthritis;
- Enthesitic arthritis - is placed in the presence of arthritis and enthesitis;
- Psoriatic arthritis is placed in the presence of arthritis and cutaneous psoriasis [3].

Clinical picture. The basis of JIA is articular syndrome, which is manifested by arthritis. Arthritis is characterized by the following symptoms: swelling, hyperemia, local fever, pain, as well as impaired joint function. All joints with a synovial membrane can be affected. In children, the knee, ankle and wrist joints are more often affected. One of the characteristic signs of JIA is damage to the joints of the cervical spine. Another characteristic feature of JIA is a symptom of morning stiffness, stiffness in the joints in the morning hours lasting from 10-15 minutes to several hours. Systemic form of JIA, in addition to joints, can affect other internal organs and develop : myopericarditis, pleurisy, serous peritonitis, hepatosplenomegaly, lymphadenopathy. Very often, the eyes are affected in JIA and can occur in the form of acute iridocyclitis / uveitis and chronic anterior iridocyclitis, ribbon-like corneal dystrophy with cataract complications.

Oligoarticular variant. The disease begins at the age of 6 months to 6 years. It is characterized by the occurrence of mono- or asymmetric oligoarthritis, mainly knee, wrist, ankle joints. There is an elongation of the affected limb due to irritation of the growth zones. Approximately 50% of patients develop uveitis. In analyzes, 40% reveal an antinuclear factor.

Polyarticular variant. There are two options: seropositive in the Russian Federation and seronegative in the Russian Federation. Seropositive in the Russian Federation: symmetrical lesion of the proximal interphalangeal, metacarpal-phalangeal, wrist joints is characteristic. The peculiarity is that in this disease a rheumatoid factor is detected. Seronegative in the Russian Federation: symmetrical lesions of large and small joints are characteristic, but without rheumatoid factor in the analyzes.

Enthesitant option. In this case, the joints of the foot are affected, pain in the heels appears, as well as pain on palpation in the areas of attachment of ligaments and tendons. It is possible to involve the sacroiliac joints in the pathological process. Uveitis develops in 10% of cases.



System option. There are two options: the allergoseptic variant and the Still variant. The allergoseptic variant is characterized by a sudden onset with a high long-term fever up to 39-40 ° C. The rash usually appears with fever or after 2-3 weeks. It is spotty-papular and localized over the affected joints, usually polymorphic. The disease is accompanied by hepatosplenomegaly, lymphadenopathy, pericarditis or myocarditis. The knee, hip, ankle joints are affected. Key A feature of the Still variant is the involvement in the process, in addition to the knee hip, ankle joints, cervical spine, as well as temporomandibular joints [2].

Differential diagnosis. The diagnosis of JIA is a diagnosis of exclusion and is made last. First, pathologies such as sepsis, infections (yersiniosis, toxoplasmosis, etc.), oncohematology, solid tumors, diffuse connective tissue diseases (SLE, systemic vasculitis), inflammatory bowel disease (UC, CD) are excluded.

Diagnostics. There is no specific diagnosis of JIA. There are diagnostic criteria proposed by the American Rheumatic Association:

- Onset of the disease before 16 years of age;
- Damage to one or more joints, characterized by swelling / effusion or having at least 2 of the following symptoms: limitation of function, pain on palpation, increase in local temperature.
- The duration of articular changes is at least 6 weeks.
- Exclusion of other rheumatic diseases.

The analyzes note an increase in ESR, CRP, Ig M, Ig G. Only in 6% of cases, RF and antibodies to cyclic citrulline peptide are detected - markers of rheumatoid arthritis. An important role is played by instrumental diagnostic methods - MRI, CT, X-ray examination, ultrasound [2].

Treatment. The following drugs are used in the treatment of JIA:

1. Nonsteroidal anti-inflammatory drugs (diclofenac);
2. Glucocorticosteroids (prednisolone)
3. Basic antirheumatic drugs (methotrexate, sulfasalazine)
4. Genetically engineered biological drugs (etanercept, tocilizumab in the systemic variant) [2].

Prognosis and prevention. JIA belongs to the group of lifelong diagnoses. Timely therapy and regular visits to a rheumatologist will help to achieve sustainable remission without reducing the quality of life and severe restrictions in joint mobility. The risk of pathology going into the acute stage is significant. An optimistic prognosis is formed with an early onset of the disease. Late manifestations of JRA are characterized by a continuously renewed course. The patient suffers from limited mobility of the limbs and receives a disability. Preventive measures involve parental supervision of young children to prevent excessive insolation or hypothermia. Babies should limit contact with carriers of viral and bacterial infections.



References

1. Balabanova R. M. Rheumatoid arthritis with systemic manifestations (clinic, treatment, prognosis): diss. Doc. med sciences / R. M. Balabanova; Inst. Rheumatol. RAMS. M.; 1990.
2. Ibragimova D. T. Clinical features of juvenile rheumatoid arthritis // Young scientist. — 2019. — №22.
3. Clinical recommendations for medical practitioners. Moscow, 2002.
4. N.N. Kuzmina, A.V. Shaikov "Juvenile rheumatoid arthritis: terminological and classification aspects", J. Scientific and practical rheumatology, No. 1, 2000.
5. Rational pharmacotherapy of rheumatic diseases. Edited by V.A. Nasonova, E.L. Nasonov, Moscow, 2003.
6. Dolgopolova A.V., Bisyarina V.P., Alekseev L.S. et al. Questions of Rheumatism 1979, 4,3-5.
7. Dolgopolova A.V., Yakovleva A.A., Isaeva L.A. Classification of JRA. Pediatrics. 1981, 7,3-5.
8. Melikhova N.I. Juvenile rheumatoid arthritis. M. "Medicine", 1991, 208.
9. Kovalenko VM, Omelchenko LI, Yaremenko OB et al. (2016). Continuity and efficacy of juvenile idiopathic arthritis therapy: directions of personalized therapy. Ukrainian Rheumatology Journal. 2: 9–16. <http://lib.inmeds.com.ua:8080/jspui/handle/lib/1961>.
10. Guide to pediatric arthrology. Edited by M.Y. Studenikin and A.A. Yakovleva. Flax. "Medicine", 1984.
11. Shaikov A.V. Criteria for exclusion of JRA Children's Rheumatology, 1996, 1, 57-61.
12. Shaikov A.V., Kuzmina N.N. Problem of JRA at the modern stage. Clinical Rheumatology, 1994, 2, 12-14.
13. Boykinov I.N. Juvenile chronic arthritis. Sofia. "Medicine and physical education." 1983, 229.
14. Brewer E.J., Giannini E.M., Person D.A. (eds). Juvenile Rheumatoid Arthritis. W.B.Saunders, Philadelphia, 1982.
15. Cassidy J.T, Levinson J.E., Bass J.G et al. A study of classification criteria for diagnosis of Juvenile Rheumatoid Arthritis. Arthritis Rheum, 1986, 29, 274.
16. Cassidy J.T, Petty R.E. Juvenile Rheumatoid Arthritis In: Textbook of pediatric rheumatology. New York, Edinburg, Melbourne, Tokyo, 1990, 113-220.
17. Cornil M.V. Memoire on the pathological coincidences of rheumatic fever. CR Mem.Soc.Biol. (Paris), 1864. 3.3, series 4.
18. Coss J.A, Boots R.M. Juvenile rheumatoid arthritis. A study of fifty-six cases with a note on skeletal changes. Pediatr, 1946, 29,143.
19. Diamantberger M.S. Rheumatic nodosa (deforming polyarthritis) in children. These for the doctoral in medicine. Lecrosnier and Bade Livraires Publishers. Paris, 1890.
20. Fink C.W. The Task Force for Classification Criteria: Proposal for the development of classification criteria for idiopathic arthritides of childhood. J. Rheumatol, 1995, 22,1566-9.
21. Kolle G. Juvenile rheumatoid arthritis and related collagen diseases. Klinische Aspekte Mschr Kinderheilk. 1976, 124,779.



22. Kvien T.K., Hayeraal H.M. Kass E. Diagnostic criteria of rheumatoid arthritis in children. *Scand.J. Rheumatol*, 1982, 11,187.
23. Petty R.E. Classification of childhood arthritis: a work of progress. *Baillieres Clin. Rheumatol*, 1998, 12(2), 181-190.
24. Still J.F. On a form of chronic joint disease in children. *Med.Chir.Frans*, 1897, 80:47. (Reprinted in *Arch.Dis.Cild*, 1941, 16,156).
25. Mirrakhimova M.Kh., Kurbanova D.R., Nishanbaeva N.Yu. . Identification of clinical and laboratory changes of the gastrointestinal tract in atopic dermatitis in children and improvement of the principles of diagnosis and treatment/ISSN 2181-712X. EISSN 2181-2187 «Тиббиётда янги кун» 6 (38/1) 2021,pp.720-726. 32374956; PMID: PMC7281714.
26. Mirrakhimova M.H., Nishanbaeva N.Y., Clinical Manifestations Of Connective Tissue Dysplasia In Children With Glomerulonephritis //Journal of Pharmaceutical Negative Results/Volume 13/Special Issue 9 | 2022,rr.4203-4205.
27. Nishanbayeva N.Y., Mirrakimova M.X. Improving the ability to detect, diagnose, and treat clinical laboratory changes in the gastrointestinal tract in atopic dermatitis in children /"New Day in Medicine" 6(38/1)2021 ISSN 2181-712X. EISSN 2181-2187 pp.720-726.
28. Wood P.H.N. Special meeting on: Nomenclature and classification of arthritis in children. In: *The care of Rheumatic Children* (Ed. E.Munthe). EULAR Publishers, Basel, 1978, 47.
29. Oshlyanska OA, Omelchenko LI, Okhotnikova OM. (2017). Juvenile Arthritis Management Errors. *Clinical immunology. Allergology. Infectology*. 99(2): 67–78.
30. *Clinical medicine. Handbook of a practical doctor*. M., 1997, T.1
31. *Clinical immunology of childhood*, D.V. Stefani, Y.E. Velti shchev, 1996
32. Pariyskaya T.V.. *Pediatrician's Handbook*. EKSMO, Moscow, 2004.
33. *Guide to Pediatric Arthrology*. Ed. acad. Academy of Medical Sciences of the USSR M.Y. Studenikin and prof. A.A.Yakovleva. – L., 1987. –With. 162- 170.
34. *Handbook of a general practitioner*. Edited by Acad. RAMS. N.R.Paleeva. EKSMO 2002
35. *Pediatrician's handbook*. St. Petersburg, Moscow, 2004
36. "Handbook of the family doctor" (Pediatrics), - Minsk, 2000