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Diagnostics and physiotherapy in rheumatoid arthritis

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Abstract

Rheumatoid arthritis (RA) is one of the most common autoimmune diseases of connective tissue. The first symptoms of the disease usually appear between the ages of 30 and 50 and include a chronic inflammatory process that causes progressive destruction of the osteoarticular system and organ changes. The etiology of RA is not sufficiently understood so far, and both genetic and environmental factors play a role in the pathogenesis of RA. The diagnosis is based on the criteria of the American Collegium of Reumathology (ACR) and the European League Against Rheumatism (EULAR) from 2010. Among the methods of physical therapy used in the treatment of RA, cryotherapy (cryostimulation) plays a special role. According to the guidelines of the Ottawa Panel, functional kinesiotherapy should play a special role in RA patients, which aims to restore movement patterns closest to physiology. When using kinesiotherapy in patients with RA, the course of compensatory processes should be monitored. The lack of supervision by a physician and a physiotherapist over the above-mentioned process leads to overload and then deformation of the musculoskeletal system.

Key words: rheumatoid arthritis, physiotherapy

INTRODUCTION

Rheumatoid arthritis (RA) is one of the most common autoimmune diseases of connective tissue. It is characterized by a chronic inflammatory process that causes progressive destruction of the osteoarticular system and organ changes. [1] The first symptoms of the disease usually appear between the ages of 30 and 50. Currently, about 1% of the human population is affected by this disease, and the incidence in women is twice as high as in men [2]. The disease is characterized by symmetrical arthritis, degradation of the articular cartilage and epiphyses, and the presence of extra-articular lesions and systemic complications, including vasculitis, reactive amyloidosis, and pulmonary fibrosis. RA is progressive, significantly worsens the quality of life of patients, contributes to disability and premature death associated with an increased risk of cardiovascular complications [3, 4]. Early diagnosis of rheumatoid arthritis and the introduction of appropriate treatment are of great importance for improving the prognosis and quality of life of patients [5]

DESCRIPTION OF THE STATE OF KNOWLEDGE

Etiology and pathophysiology

The etiology of RA is not sufficiently understood so far, and both genetic and environmental factors (including smoking) play a role in the pathogenesis of this disease [2]. Genetic factors are considered to contribute 40-60% to the etiopathogenesis of RA [6–9].

There are several potential genetic factors associated with an increased incidence of RA, including the occurrence of the so-called common epitope in HLA class II molecules, gene polymorphisms of some matrix metalloproteinases (MMPs), including matrix metalloproteinase 1 (MMP-1) and matrix metalloproteinase 3 (MMP-3), as well as polymorphism of proinflammatory cytokine genes, especially tumor necrosis factor α (TNF- α), inteleukin 1 β (IL-1 β) and interleukin 4 (IL-4) [6, 9, 10].

In the etiopathogenesis of RA, the genetic basis is also formed by polymorphic genes outside the HLA system, and most of them regulate the course of the acquired response. The most important of them are polymorphic variants of genes such as: PTPN22, which is responsible for the encoding of tyrosine phosphatase, Lyp - an enzyme regulating lymphocyte activation, genes encoding molecules that provide co-stimulation signals (CD40, CD28) or quench the activity of T lymphocytes (CTLA4), or the STAT 4 protein, which is involved in the initiation and stabilization of the synthesis of pro-inflammatory cytokines such as IL-12, IL-15, IL-23 [10, 11].

Apart from genetic factors, environmental factors play a special role in the etiopathogenesis of RA, including smoking, prolonged stress, improper diet and intense exercise [6, 12–14]

Smoking is currently the best known environmental factor that increases not only the development of RA, but also the severity of the disease and a weaker response to pharmacotherapy [11].

Diagnostics of RA

The diagnosis of RA is based on the criteria of the American Collegium of Reumathology (ACR) and the European League Against Rheumatism (EULAR) from 2010. These criteria allow the detection of both early and chronic erosive forms. These criteria take into account 4 categories: location of inflammation, markers of inflammation (CRP, ESR), serological tests (RF IgM, anti-CCP), duration of the disease.

There is no single laboratory test that can make a definitive diagnosis of RA. The sensitive and specific markers of inflammation are constantly searched for, which would enable the detection of the disease at its initial stage and the implementation of an effective therapy that would inhibit the inflammation process [15, 16].

Rheumatoid factor (RF) in the IgM class is one of the most important serological markers of arthritis. This factor is present in approximately 75% of RA patients. Its absence indicates a seronegative form of the disease. Unfortunately, RF IgM has a low specificity for RA (87%). High titers of this autoantibody are observed in other systemic connective tissue diseases, such as Sjgren's syndrome, systemic scleroderma, systemic lupus erythematosus, and polymy / intramuscular inflammation or sarcoidosis [16–19].

Anti-CPP antibodies present in RA patients can be detected in the early stages of the inflammatory process involving the synovium of the joints [20, 21]. In the diagnosis of RA, a-CCP antibodies have a diagnostic sensitivity similar to RF IgM, but with greater specificity. The diagnostic sensitivity of commonly used tests for the determination of a-CCP antibodies is about 61% in the early stage of RA and 75% in the advanced stage of the disease [16].

Anti-keratin antibodies (AKA) are present in 36-59% of RA patients. They are determined by indirect immunofluorescence method, using slices of the rat esophagus. The sensitivity and specificity of AKA tests are at the level of 40-60% and 88-99%.

Antiphilagrin antibodies (AFA) are present in 41% of RA patients. They are determined using immunoblotting or ELISA methods. Recombinant filaggrin is used for these tests, which significantly increases the sensitivity of these tests up to 52%. AFA antibodies have a high specificity for RA - over 90%.

Anti-Sa antibodies react with the SA antigen, which is a citrullinated transition form of vimentin white filaments. Antibodies are determined by the ELISA method. The level of antibodies is related to the clinical condition of the patient - it increases with the severity of the changes. They are characterized by a sensitivity in the range of 31-84% and specificity of 83-93% for RA [16, 22, 23]

Antibodies to carbomylated proteins (anti-CarP) are relatively recently discovered antibodies. They are found in about 45% of RA patients. Interestingly, 30% of patients who do not have ACPA antibodies have anti-CarP antibodies. The

sensitivity and specificity of these antibodies are 2-29% and 95-100%, respectively [16, 24].

In the diagnostic procedure, auxiliary tests are also performed, such as CRP, ESR, fibrinogen concentration, proteinogram, AST and ALT activity, serum uric acid, creatinine and electrolyte levels. CRP and ESR protein levels are often elevated in active RA and are> 7mg / L and> 30mm, respectively, after 1h. In addition, an increased concentration of fibrinogen is observed, and in blood counts normocytic and hypochrominic anemia, slight leukocytosis with a normal percentage increase. Increased levels of alpha-1 and alpha-2 globulins are observed in the proteinogram. The above parameters are also used to assess the severity of the disease.

Physical therapy in the treatment of RA

Physical therapy is an auxiliary method that complements pharmacotherapy and treatment with movement. The most effective physiotherapy treatments used in RA patients are: thermotherapy (mainly cryotherapy), laser biostimulation, ultrasounds, and TENS electrostimulation [25, 26].

Among the methods of physical therapy used in the treatment of RA, cryotherapy (cryostimulation) plays a special role [25]. In the studies by Krawczyk-Wasielewska [26], in patients with RA, cryotherapy was recognized as the most effective method of analgesic physical therapy. It has also been proven that cryostimulation is a safe method that does not have a negative impact on peripheral vessels and heart function (it does not cause adverse changes in the electrocardiogram). The analgesic effect of cryotherapy in RA patients is, inter alia, associated with the destimulating effect on pain receptors and the regulation of the production, release and degradation of histamine [27].

According to the guidelines of the Ottawa Panel, functional kinesiotherapy should play a special role in RA patients, which aims to restore movement patterns closest to physiology. An important function is also played by the improvement of the gait stereotype and exercises increasing the patient's cardiovascular capacity, eg cycling, swimming in a pool or walking [25]. According to the recommendations proposed by the American College of Sports Medicine (ACSM), in RA patients aged 50–64 years, attention should be paid to exercises to strengthen the muscular corset, balance and improve the flexibility of the musculoskeletal system [28]. A review of the literature on physiotherapy in RA conducted by Dutch authors ended with the preparation of recommendations according to which resistance and aerobic exercises are recommended for patients with RA, while passive mobilization and manipulations should not be abandoned, but also not promoted [29].

When using kinesiotherapy in patients with RA, the course of compensatory processes should be monitored. Lack of supervision by a physician and a physiotherapist over the above process leads to overload and then deformation of the musculoskeletal system [30]. In compensation, orthoses (orthoses) and auxiliary equipment are of great importance. The use of hand orthoses in the acute phase of arthritis reduces friction of the articular surfaces, prevents excessive stress on the joints, thus inhibiting the progression of hand deformation. The results of many

studies show that in patients with arthritis, the orthosis reduces pain and improves joint function during daily activities [31, 32]

Conclusion

Rheumatoid arthritis (RA) is one of the most common autoimmune diseases of connective tissue. The first symptoms of the disease usually appear between the ages of 30 and 50 and include a chronic inflammatory process that causes progressive destruction of the osteoarticular system and organ changes. The etiology of RA is not sufficiently understood so far, and both genetic and environmental factors play a role in the pathogenesis of RA. The diagnosis is based on the criteria of the American Collegium of Reumathology (ACR) and the European League Against Rheumatism (EULAR) from 2010. Among the methods of physical therapy used in the treatment of RA, cryotherapy (cryostimulation) plays a special role. According to the guidelines of the Ottawa Panel, functional kinesiotherapy should play a special role in RA patients, which aims to restore movement patterns closest to physiology. When using kinesiotherapy in patients with RA, the course of compensatory processes should be monitored. The lack of supervision by a physician and a physiotherapist over the above-mentioned process leads to overload and then deformation of the musculoskeletal system.

REFERENCES

1. Scott DL, Wolfe F, Huizinga TWJ. Rheumatoid arthritis. In: The Lancet. 2010. p. 1094–108. doi:10.1016/S0140-6736(10)60826-4.

2. Rindfleisch AJ, Muller D. Diagnosis and Management of Rheumatoid Arthritis - American Family Physician. 2005. www.aafp.org/afp. Accessed 19 Apr 2021.

3. Lee DM, Weinblatt ME. Rheumatoid arthritis. Lancet. 2001;358:903–11. doi:10.1016/S0140-6736(01)06075-5.

4. Olewicz-Gawlik A, Hrycaj P. Original paper
br>Health-related quality of life among patients with rheumatoid arthritis – original results and short literature review. Termedia; 2007. www.statsoft.com. Accessed 19 Apr 2021.

5. Matuszewska A, Madej M, Wiland P. Immunological markers of rheumatoid arthritis. Postepy Hig Med Dosw. 2016;70:251–7.

6. Jakub T, Agnieszka PG, Ewa MP, Maria RB, Wojciechowska B, Jan KŁ. Wpływ czynników genetycznych na rozwój i ciężkość przebiegu reumatoidalnego zapalenia stawów Część i. Polski Merkuriusz Lekarski. 2009;27:157–60. https://fbc.pionier.net.pl/details/nnz53Wv. Accessed 19 Apr 2021.

7. Alamanos Y, Drosos AA. Epidemiology of adult rheumatoid arthritis. Autoimmunity Reviews. 2005;4:130-6. doi:10.1016/j.autrev.2004.09.002.

8. Orozco G, Barton A. Update on the genetic risk factors for rheumatoid arthritis. Expert Review of Clinical Immunology. 2010;6:61–75. doi:10.1586/eci.09.72.

9. Orozco G, Rueda B, Martin J. Genetic basis of rheumatoid arthritis. Biomed Pharmacother. 2006;60:656–62. doi:10.1016/j.biopha.2006.09.003.

10. Ostanek M, Ciechanowicz A. Artykuł przeglądowy/Review paper. Termedia; 2009.

11. Kontny E. Pathogenesis of rheumatoid arthritis. Part I: acquired immunity, genetic and environmental factors. Termedia; 2011.

12. I. Z-G. Wspó³czesne podej cie do leczenia reumatoidalnego zapalenia stawów. Alerg Astma Immun. 1999;4:83–90.

13. Silman AJ, Pearson JE. Epidemiology and genetics of rheumatoid arthritis. Arthritis research. 2002;4 Suppl 3:S265–72. doi:10.1186/ar578.

14. Bang SY, Lee KH, Cho SK, Lee HS, Lee KW, Bae SC. Smoking increases rheumatoid arthritis susceptibility in individuals carrying the HLA-DRB1 shared epitope, regardless of rheumatoid factor or anti-cyclic citrullinated peptide antibody status. Arthritis Rheum. 2010;62:369–77. doi:10.1002/art.27272.

15. Aho K, Heliövaara M. Risk factors for rheumatoid arthritis. Annals of Medicine. 2004;36:242–51. doi:10.1080/07853890410026025.

16. Polińska B, Matowicka-Karna J, Kemona H. Markery wczesnego stadium reumatoidalnego zapalenia stawów Markers of the early stage of rheumatoid arthritis. 2016.

17. Burska AN, Hunt L, Boissinot M, Strollo R, Ryan BJ, Vital E, et al. Autoantibodies to posttranslational modifications in rheumatoid arthritis. Mediators of Inflammation. 2014;2014. doi:10.1155/2014/492873.

18. Taylor P, Gartemann J, Hsieh J, Creeden J. A systematic review of serum biomarkers anticyclic citrullinated peptide and rheumatoid factor as tests for rheumatoid arthritis. Autoimmune Diseases. 2011;1. doi:10.4061/2011/815038.

19. Šenolt L, Grassi W, Szodoray P. Laboratory biomarkers or imaging in the diagnostics of rheumatoid arthritis? BMC Medicine. 2014;12. doi:10.1186/1741-7015-12-49.

20. Li X, Tian F, Wang F. Rheumatoid arthritis-associated microrna-155 targets socs1 and upregulates TNF- α and IL-1 β in PBMCs. Int J Mol Sci. 2013;14:23910–21. doi:10.3390/ijms141223910.

21. Trenkmann M, Brock M, Gay RE, Michel BA, Gay S, Huber LC. Tumor necrosis factor α -induced microRNA-18a activates rheumatoid arthritis synovial fibroblasts through a feedback loop in NF- κ B signaling. Arthritis Rheum. 2013;65:916–27. doi:10.1002/art.37834.

22. Alicja Grim, Katarzyna Komosińska-Vassev, Paweł Olczyk. Rodzaje autoprzeciwciał w chorobach reumatycznych i metody ich oznaczeń. J Lab Diagnostics Diagn Lab. 2015. https://documen.site/download/rodzaje-autoprzeciwcia-w-chorobach-reumatycznych-i-metody-ich_pdf. Accessed 19 Apr 2021.

23. Farid SS, Azizi G, Mirshafiey A. Anti-citrullinated protein antibodies and their clinical utility in rheumatoid arthritis. International Journal of Rheumatic Diseases. 2013;16:379–86. doi:10.1111/1756-185X.12129.

24. Shi J, Van De Stadt LA, Levarht EWN, Huizinga TWJ, Hamann D, Van Schaardenburg D, et al. Anti-carbamylated protein (anti-CarP) antibodies precede the onset of rheumatoid arthritis. Ann Rheum Dis. 2014;73:780–3. doi:10.1136/annrheumdis-2013-204154.

25. Kuncewicz E, Samborski P SA i wsp. Polskie podejście fizjoterapeutyczne usprawniania w reumatoidalnym zapaleniu stawów a zalecenia Panelu Ottawskiego. Chir Narządów Ruchu Ortop Pol. 2009. https://documen.site/download/wpyw-fizjoterapii-na-poziom-odczuwanego-bolu-u-chorych-na_pdf. Accessed 19 Apr 2021.

26. Agnieszka Krawczyk-Wasielewska EKMSWS. Assess of physical therapy effectiveness in pain treatment in rheumatoid arthritis. Nowa Med. 2007.

27. Wojtecka-Lukasik E, Ksiezopolska-Orlowska K, Gaszewska E, Krasowicz-Towalska O, Rzodkiewicz P, Maslinska D, et al. Cryotherapy decreases histamine levels in the blood of patients with rheumatoid arthritis. Inflamm Res. 2010;59 SUPPL. 2. doi:10.1007/s00011-009-0144-1.

28. Kerschan-Schindl K, MacHold K. Rehabilitation von Patienten mit rheumatoider Arthritis. Phys Medizin Rehabil Kurortmedizin. 2011;21:297–310. doi:10.4061/2011/681640.

29. Hurkmans EJ, Li L, Verhoef J, Vliet Vlieland TPM. Physical Therapists' Management of Rheumatoid Arthritis: Results of a Dutch Survey. Musculoskeletal Care. 2012;10:142–8.

30. Kazimiera Milanowska WD. Rehabilitacja medyczna. 2003. https://medbook.com.pl/ksiazka/pokaz/id/1657/tytul/rehabilitacja-medyczna-milanowskadega-wydawnictwo-lekarskie-pzwl. Accessed 19 Apr 2021.

31. Beasley J. Osteoarthritis and rheumatoid arthritis: Conservative therapeutic management. J Hand Ther. 2012;25:163–72. doi:10.1016/j.jht.2011.11.001.

32. Zuk B, Księzopolska-Orłowska K. Ochrona stawów w reumatoidalnym zapaleniu stawów. Zaopatrzenie ortopedyczne. Reumatologia. 2009;47:241–8.