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Treatment approaches against rheumatoid arthritis (RA)

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Abstract

Rheumatoid arthritis (RA) disease has inflammatory properties and causes some problems in the joints and decreases the quality of life of the individuals. It is thought that some factors such as environmental, genetic, hormonal, psychological and age have an effect on the development of rheumatoid arthritis, which affects the musculoskeletal system and can cause damage to some organs. Glucocorticoids, disease-modifying anti-rheumatic drugs (DMARDs) and non-steroidal anti-inflammatory drugs (NSAIDs) are preferred within the scope of the treatment.

Introduction

Rheumatoid arthritis (RA), whose etiology is not clearly determined, is an autoimmune disease with inflammatory features and chronic progression. Rheumatoid arthritis is a common joint disease among inflammatory diseases [1,2]. RA joint disease causes disability as well as a decrease in the quality and duration of life [3,4].

Rheumatoid arthritis joint disease is generally more common in the advanced age of the population (40 to 50 years old) and in women (may vary according to geographical conditions) compared to men [2].

In the course of RA disease, which is characterized by damage to the joints; stiffness in the hand, shoulder, elbow, ankle and knee joints, pain in the joints, swelling in the joints, musculoskeletal pain, joint fractures, weakness, fatigue, muscle weakness, weight loss, fever, sleep disorders, joint deformity are among the most common symptoms [1-5].

Although RA affects the musculoskeletal system, it can also cause damage to the nervous and immune system, heart, skin, eyes, lungs, kidneys and vessels [1,5].

Although the exact cause of RA cannot be determined, some factors are thought to be effective in the development of the disease. In general terms, these risk factors include; environmental factors, genetic factors, hormonal factors, psychological factors, age factor [3,5,6]. It is known that genetic factors have a 40%-60% effect on the formation of the disease. Some genetic polymorphisms and epigenetic modifications are considered among genetic factors that may affect the development of the disease. The "Human Leukocyte Antigen (HLA)" locus is very important in the development of RA [7].

Discussion

Although cytokines are thought to have an important role in the pathogenesis of RA, IL-1, IL-6 and TNF-α cytokines are detected at high rates in the synovial fluids of the joints of individuals with rheumatoid arthritis [8-10].

Th17 cells, which have an effect on joint damage, are involved in the pathogenesis of rheumatoid arthritis. Th17 cells; It is responsible for the production of IL-17A, IL-17F, IL-22 and IL-26 cytokines. IL-17 proinflammatory cytokine causes joint damage. TGF-β and IL-6 cytokines (with IL-23) or IL-23 and IL-1β cytokines provide the differentiation of naive T cells into Th17 cells. IL-17A is produced by both Th17 cells and some cells such as neutrophils, eosinophils, and macrophages [7,11-13].

Regulatory T cells, which accumulate in the joints of individuals with rheumatoid arthritis, exist as a subset of naive CD4+ T cells and are involved in autoimmune diseases as suppressive type of cells. These cells are crucial to eliminate the inflammation. Increasing their activity in the joints is one of the immunotherapy approaches against RA [13].

Although there is no definitive treatment method for rheumatoid arthritis, which initially affects the joints and then the internal organs, the symptoms of the disease are handled in an individual way [5]. Early diagnosis of rheumatoid arthritis and initiation of appropriate treatment as a result of this condition allows to increase the quality of life of the individual. In addition, the occurrence and progression of joint damage in 90% of patients is slowed down by early diagnosis of the disease and the application of the necessary treatment method [14].

One of the treatment strategies applied to rheumatoid arthritis patients is drug treatments. In this context, glucocorticoids, non-steroidal anti-rheumatic drugs (NSAIDs) and disease-modifying anti-rheumatic drugs (DMARDs) are used [14,15].

Conclusion

Reducing joint pain and swelling seen in rheumatoid arthritis, preserving the functions of the joints, reducing the sensitivity and damage in the joints, slowing down or stopping the joint destruction, preventing the progression of the disease to an advanced level are possible with the disease-modifying anti-rheumatic drug (DMARD) group [6,14,15].

One of the treatment options applied to reduce pain and inflammation in the treatment of RA disease is the non-steroidal anti-inflammatory drug (NSAID) group. Some drugs included in this drug group (for example, drugs such as ibuprofen, aspirin) have anti-inflammatory and analgesic effects and are therefore preferred as an adjunctive treatment in rheumatoid arthritis [5,15].

Glucocorticoids, which have a strong anti-inflammatory effect, can reduce the progression of damage to cartilage and bone. Diabetes, osteoporosis, gastrointestinal bleeding, hypertension and weight gain are among the side effects of glucocorticoids in sick individuals [6,14].

In addition to the positive effects of drug treatments on the disease, it can also cause some side effects in sick individuals. Among these side effects; some infections, anemia, kidney and liver damage, loss of appetite, nausea and vomiting, allergies [2].

Different antibody-based therapy approaches are being developed, including immunotherapy, in order to eliminate the side effects of traditional drug treatments for the disease [16-18].

References

1. Kiper, S., Sunal, N. (2009). Romatoid artritli hastalarda uyku kalitesinin değerlendirilmesi. Kocatepe Tıp Dergisi, 10(1), 33-39.
2. Metin, Z. G., & Özdemir, L. (2016). Romatoid Artritte Ağrı ve Yorgunluğun Yönetiminde Aromaterapi ve Refleksolojinin Kullanımı. Hemşirelikte Eğitim ve Araştırma Dergisi, 13(1), 44-49.
3. Altınkesen, E. (2006). Erken ve geç dönem romatoid artrit'li hastalarda semptomlar, fonksiyonel durum ve yaşam kalitesi arasındaki ilişkinin incelenmesi. PHD Thesis, DEÜ Sağlık Bilimleri Enstitüsü
4. Kurt, E., Özdilli, K., & Yorulmaz, H. (2013). Romatoid Artrit'li Hastalarda Beden İmajı ve Benlik Saygısı. Archives of Neuropsychiatry/Noropsikiatri Arsivi, 50(3).
5. Bullock, J., Rizvi, S. A., Saleh, A. M., Ahmed, S. S., Do, D. P., Ansari, R. A., & Ahmed, J. (2018). Rheumatoid arthritis: a brief overview of the treatment. Medical Principles and Practice, 27(6), 501-507.
6. Lin, Y. J., Anzaghe, M., & Schülke, S. (2020). Update on the pathomechanism, diagnosis, and treatment options for rheumatoid arthritis. Cells, 9(4), 880.
7. Terzioğlu, E. (2009). Romatoid Artritin Etyopatogenezi. Ulusal Romatoloji Dergisi, 1(Special Issue), 4-8.
8. Akdoğan, M., & Yöntem, M. (2018). Sitokinler. Online Türk Sağlık Bilimleri Dergisi, 3(1), 36-45.
9. Baykal, Y., Karaayvaz, M., & Kutlu, M. (1998). Interleukins. T Klin J Med Sci, 18, 77-84.
10. Özoran, K., Tulek, N., & Düzgün, N. (1994). Romatoid Artrit (RA) ve Sitokinler: İnterlökin-1 (Il-1), İnterlökin-6 (Il-6), Tümör Nekrozis Faktör Alfa (Tnf-A) Ve İnterferon Gama (Ifn-Y). Ankara Üniversitesi Tıp Fakültesi Mecmuası, 47(3), 495-504.
11. Baskan, E. B. (2013). T hücre immunitesi/T cell immunity. Türkderm: Türk Deri Hastalıkları ve Frengi Arşivi= Turkderm: Turkish Archives of Dermatology and Venereology, 47(1), 18.

12. Düzgün, N. (2014). İmmün sistemin tanıtımı. Ankara Üniversitesi Tıp Fakültesi, İç Hastalıkları Anabilim Dalı, Romatoloji Bilim Dalı, 111.
13. Furst, D. E., Emery, P. (2014). Rheumatoid arthritis pathophysiology: update on emerging cytokine and cytokine-associated cell targets. Rheumatology, 53(9), 1560-1569.
14. Akdağ, H., Kırmusaoğlu, S. (2019) Romatoid Artrit Tedavisinde Uygulanan Güncel Yaklaşımlar. Haliç Üniversitesi Fen Bilimleri Dergisi, 2(2), 197-206.
15. Demirel, A., & Kırnap, M. (2010). Romatoid Artrit Tedavisinde Geleneksel ve Güncel Yaklaşımlar. Sağlık Bilimleri Dergisi, 19(1), 74-84.
16. Reynolds, G., Cooles, F. A., Isaacs, J. D., & Hilkens, C. M. (2014). Emerging immunotherapies for rheumatoid arthritis. Human Vaccines & Immunotherapeutics, 10(4), 822-837.
17. Taylor, P. C. (2003). Antibody therapy for rheumatoid arthritis. Current opinion in pharmacology, 3(3), 323-328.
18. Vitali, C., & Bombardieri, M. S. S. (1993). Immunotherapy in rheumatoid arthritis: a review. *The International Journal of Artificial Organs*, *16*(5_suppl), 196-200.