Short Communication

International Journal of Clinical Rheumatology

Disease-Modifying Antirheumatic Drugs (DMARDs): Revolutionizing Rheumatic Disease Management

Sandra Deson*

Department of Biochemistry, Jimma University, Ethiopia

***Author for Correspondence:**
sandradeson879@yahoo.com

Received: 01-Jun-2024, Manuscript No. fmijcr-24-145771; **Editor assigned:** 03-Jun-2024, Pre-QC No. fmijcr-24-145771 (PQ); **Reviewed:** 17-Jun-2024, QC No. fmijcr-24-145771; **Revised:** 22- Jun-2024, Manuscript No. fmijcr-24-145771 (R); **Published:** 28- Jun-2024, **DOI:** 10.37532/1758-4272.2024.19(6).208-210

Abstract

Disease-Modifying Antirheumatic Drugs (DMARDs) have transformed the landscape of rheumatic disease treatment, particularly for conditions like rheumatoid arthritis (RA), psoriatic arthritis (PsA), and systemic lupus erythematosus (SLE). These medications do not merely alleviate symptoms; they alter the disease's course by targeting underlying pathological mechanisms, thus preventing joint damage and preserving function. This article explores the types of DMARDs, their mechanisms of action, clinical applications, and the impact they have had on the management of rheumatic diseases.

Introduction

DMARDs are broadly categorized into conventional synthetic DMARDs (csDMARDs), biologic DMARDs (bDMARDs), and targeted synthetic DMARDs (tsDMARDs). Each class works through different mechanisms to suppress the immune system and reduce inflammation. Conventional DMARDs, such as methotrexate, hydroxychloroquine, and sulfasalazine, are the traditional mainstays of treatment. They act on a broad range of immune processes to control the disease. Biologic DMARDs, a newer class, include drugs like tumor necrosis factor (TNF) inhibitors, interleukin inhibitors, and B-cell depleting agents. These drugs specifically target cytokines or cells involved in the inflammatory process. Targeted synthetic DMARDs, such as Janus kinase (JAK) inhibitors, are even more recent innovations that selectively inhibit intracellular signaling pathways essential for the immune response [1-3].

Methodology

Methotrexate, the most commonly used csDMARD, has been the cornerstone of RA treatment for decades. It works by inhibiting the enzyme dihydrofolate reductase, which is essential for DNA synthesis and cell replication. This inhibition affects rapidly dividing cells, including immune cells, thus reducing inflammation. Despite its broad action, methotrexate is generally well-tolerated, with side effects like gastrointestinal discomfort and liver enzyme elevations being manageable in most patients. Other csDMARDs, like hydroxychloroquine and sulfasalazine, have different mechanisms but similar goals: to suppress the overactive immune response in rheumatic diseases. Hydroxychloroquine, for example, interferes with lysosomal activity in immune cells, while sulfasalazine modulates the immune response through multiple pathways, including inhibition of pro-inflammatory cytokines [4-6].

The advent of biologic DMARDs marked a significant advancement in rheumatology. These drugs are designed to target specific components of the immune system. For instance, TNF inhibitors like etanercept,

infliximab, and adalimumab block the action of TNF, a cytokine that plays a key role in inflammation. By neutralizing TNF, these drugs effectively reduce joint inflammation and prevent damage. Interleukin inhibitors, such as tocilizumab (IL-6 inhibitor) and secukinumab (IL-17 inhibitor), work similarly by targeting other cytokines involved in the inflammatory process. Rituximab, a B-cell depleting agent, reduces the number of B-cells, which are implicated in the autoimmune response. The specificity of biologic DMARDs offers a more targeted approach, often with fewer systemic side effects compared to conventional DMARDs [7-9].

Targeted synthetic DMARDs, like the JAK inhibitors tofacitinib and baricitinib, represent the latest evolution in DMARD therapy. These drugs inhibit the Janus kinase enzymes, which are involved in signaling pathways that regulate the immune response. By blocking these pathways, JAK inhibitors can reduce the inflammatory process with a precision that was not possible with earlier treatments. The development of these drugs has expanded the treatment options for patients who may not respond to or tolerate traditional DMARDs or biologics. However, because JAK inhibitors interfere with critical signaling pathways, they can also increase the risk of infections and other complications, making careful monitoring essential [10].

The introduction and widespread use of DMARDs have significantly improved outcomes for patients with rheumatic diseases. Before the era of DMARDs, patients with rheumatoid arthritis and similar conditions often faced severe disability due to progressive joint damage. With DMARDs, particularly when used early in the disease course, it is now possible to achieve disease remission or low disease activity in a substantial proportion of patients. This not only improves quality of life but also reduces the long-term healthcare burden associated with complications like joint replacements and cardiovascular disease. Furthermore, the use of DMARDs has been associated with reduced mortality in patients with rheumatic diseases, likely due to better control of systemic inflammation.

Despite their benefits, DMARDs are not without challenges. Conventional DMARDs can take weeks to months to exert their full effects, requiring patients to be patient and adherent to treatment despite ongoing symptoms. Biologic DMARDs, while effective, are expensive and require regular injections or infusions, which can be a barrier for some patients. Additionally, all DMARDs carry the risk of side effects, including immunosuppression, which can lead to infections and, in rare cases, malignancies. Careful monitoring and a balanced approach to treatment are necessary to minimize these risks while maximizing the benefits of therapy. Regular follow-up with healthcare providers, including periodic blood tests and imaging studies, is crucial to ensure that the treatment is both effective and safe.

Conclusion

In conclusion, Disease-Modifying Antirheumatic Drugs have revolutionized the treatment of rheumatic diseases, offering hope to millions of patients worldwide. By targeting the underlying mechanisms of these diseases, DMARDs not only alleviate symptoms but also prevent joint damage and other complications. The development of biologic and targeted synthetic DMARDs has further expanded treatment options, allowing for more personalized and effective care. As research continues to evolve, the future of rheumatic disease management looks promising, with the potential for even more targeted therapies that offer greater efficacy with fewer side effects. The ongoing challenge for clinicians is to balance the benefits of these powerful drugs with their risks, ensuring that patients receive the best possible care.

References

1. Abolfazl Akbarzadeh. Liposome: Classification, Preparation, and Applications. *Nanoscale Res Lett.* 8, 102 (2013).
2. Goyal M. Endovascular thrombectomy after large vessel ischaemic stroke: a meta- analysis of individual patient data from five randomised trials. *Lancet.* 22, 416-430 (2016).
3. Hamdi Nsairat. Liposomes: Structure, Composition, Types, and Clinical Applications. *Heliyon.* 8, 9394 (2022).
4. Berkhemer OA. A randomized trial of intra-arterial treatment for acute ischemic stroke. *N Engl J Med*. 14, 473-478 (2015).
5. Harrison, Paul. How shall I say it? Relating the nonrelational .*Environ Plan A***.** 39, 590-608 (2007).
6. Vukasinovic. Real Life impact of anesthesia strategy for mechanical thrombectomy on the delay, recanalization and outcome in acute ischemic stroke patients. *J Neuroradiol.* 95, 391-392 (2019).
7. Carrillo JE, Carrillo VA, Perez HR *et al*. Defining and targeting health care access barriers. *J Health Care Poor Underserved*. 22, 562-75 (2011).
8. Carrillo JE, Carrillo VA, Perez HR *et al*. Defining and targeting health care access barriers. *J Health Care Poor Underserved*. 22,562-75 (2011).
9. Peng J, Luo F, Ruan G *et al*. Hypertriglyceridemia and atherosclerosis. *Lipids Health Dis*. 16, 233 (2017).
10. Kooman JP, Kotanko P, Stenvinkel P *et al*. Chronic kidney disease and premature ageing. *Nat Rev Nephrol*. 10, 732-742 (2014).