

MODERN STRATEGIES IN THE TREATMENT OF SYSTEMIC LUPUS ERYTHEMATOSUS

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Introduction. Systemic lupus erythematosus (SLE) is an autoimmune disease affecting multiple organs, leading to significant morbidity and mortality. Despite treatment advancements, SLE activity, comorbidities, and drug toxicity still cause irreversible damage and increased mortality. SLE treatment now includes biologics, with belimumab being the first approved. New therapies targeting interferons, cytokines, intracellular signals, plasma cells, T lymphocytes, and co-stimulatory molecules are under evaluation.

Aim of the study. To investigate modern strategies in SLE treatment. **Material and methods.** A systematic review of the published in the past 5 years literature was conducted which focused on modern strategies in the treatment of SLE.

Results. Approved biologics for SLE include belimumab and anifrolumab. Belimumab reduces mortality in SLE patients (0.4/100 person-years) compared to the general population (1.63/100 person-years), despite a slow response rate. Non-corticosteroid immunosuppressants like cyclophos-

phamide, mycophenolate mofetil, and azathioprine are crucial for reducing SLE activity and are used for both initiating and maintaining therapy. Resistance to glucocorticoids and these immunosuppressants is common. Calcineurin inhibitors like cyclosporin A indirectly affect B cells by suppressing T cells and are safe for pregnant women. Biologics offer an alternative for patients not responding to conventional drugs, with sequential therapy enhancing B-cell depletion effectiveness. **Conclusion.** Devising a standardized treatment approach for all SLE patients poses a challenge due to the intricate pathogenesis and diverse clinical manifestations of the disease. Immunological profiling and precision medicine, based on transcriptome analysis, can help identify immune phenotypes and gene signatures in SLE patients, leading to better understanding and treatment outcomes. **Keywords:** Systemic Lupus Erythematosus, Immunosuppressants, Biological therapy.

THE ROLE OF JANUS KINASE INHIBITOR IN THE TREATMENT OF RHEUMATOID ARTHRITIS

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Introduction. Rheumatoid arthritis is a chronic inflammatory disease that is autoimmune in nature. A JAKi may be utilized to treat RA. It is a member of the DMARDs family. The purpose of this study is to investigate the efficacy of JAKi in the treatment of RA. **Material and methods.** A narrative literature review study was carried out. A bibliographic search was conducted using databases such as PubMed, WebMed, Google Scholar, National Institutes of Health, and Med Central. Articles published between 2018 and 2024 were selected using various combinations of keywords such as rheumatoid arthritis, Janus kinase inhibitor, tofacitinib, baricitinib, and Upadacitinib. **Results.** This study validates that it is capable of being taken orally due to its patient friendly nature. Patients who have not responded adequately to biologic DMARDs may use it. Drugs like tofacitinib, baricitinib, and Upadacitinib that have FDA approval are widely utilized. A number of people receiving

JAKi therapy have side effects that include elevated liver enzymes, cardiovascular risks, hematological abnormalities, and infections like herpes zoster. For optimal effects in order to lessen the severity of adverse effects, it is utilized as monotherapy. In the future, the challenge of using JAKi appropriately will arise in clinical settings. Future use of these medications is anticipated to rise, which means that more clinical data regarding the long-term outcomes and adverse effects will need to be gathered for the safe administration of JAK inhibitors. **Conclusions.** JAKi are tiny compounds that specifically target intracellular signaling molecules that function by preventing the activity of the Janus kinase enzyme, hence reducing inflammation. In addition to being widely used as an alternative to biologics, JAKi has become standard therapy for RA patients who are not responding to csDMARDs. **Keywords:** Rheumatoid arthritis, Janus kinase inhibitor, tofacitinib, baricitinib, Upadacitinib.