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Biologic Agents in Rheumatic Diseases: Lessons Learned and Perspective

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SUMMARY

Autoimmune rheumatic diseases are chronic, systemic inflammatory disorders of complex etiology and pathophysiology. Uncontrolled inflammation results in organ damage and functional impairment.

During the past 15 years we have enhanced the understanding of molecular pathogenesis of autoimmune and autoinflammatory rheumatic diseases. This has enabled the development of innovative biological agents that target specific parts of the immune system. Biologic therapies such as monoclonal antibodies and fusion proteins have revolutionized the management of rheumatic disease. By targeting key cytokines (TNFα, IL-1, IL-6) and immune cells (T-cells, B-cells), biologics have provided more specific therapeutic interventions with less immunosuppression. These treatments have changed the course and face of inflammatory arthritides and outcomes for patients and society. Clinical use, however, has revealed that their theoretical simplicity hides a more complex reality. Residual disease activity, immunogenicity, specific infections, are issues which have been emerged in clinical application of novel therapies.

Concerning pathophysiology of rheumatoid arthritis, new knowledge has emerged of how environmental factors interact with susceptibility genes and the immune system in the pathogenesis of the disease. Research undertaken on the longitudinal disease process and molecular pathology of joint inflammation has led to new therapeutic strategies that promote early use of disease-modifying drugs with tight disease control and distinct and quantifiable treatment goals. Today, such approaches can halt most cases of joint destruction but not all instances of joint inflammation and comorbidity. Understanding the cause and pathogenesis of different rheumatoid arthritis and other autoimmune and autoinflammatory diseases, will lead not only to individualized treatments during early phases of the illness but also, possibly, to disease prevention