Review

Closing remarks

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Introduction

The past decade has been marked by significant advances in rheumatology, including a better understanding of the pathophysiology of rheumatic diseases and the addition of agents to the therapeutic armamentarium. Studies in cell biology and molecular immunology have clarified the role of cellular and humoral immunity and related processes in the pathogenesis of rheumatoid arthritis (RA) and other inflammatory arthritides. Tumor necrosis factor (TNF) has been identified as a major effector of structural joint disease in patients with RA and the spondyloarthropathies, and the development and introduction of TNF antagonists is a major advance in the management of RA. Notably, the successful use of these agents has not been limited to RA: inroads have been made into other immune-mediated inflammatory diseases, including ankylosing spondylitis and psoriatic arthritis.

The efficacy of the TNF antagonists has been established, and these agents can now be considered the new standard of care for RA and the spondyloarthropathies when disease activity is not controlled by standard non-steroidal anti-inflammatory drugs (NSAIDs) and disease-modifying antirheumatic drugs (DMARDs). Additionally, much has been learned about the safety and adverse event profiles of the TNF antagonists; importantly, we are becoming more adept at managing safety-related issues. With appropriate risk management, the TNF antagonists can be considered to be as safe as the traditional DMARDs and other anti-inflammatory agents.

However, certain questions remain to be answered. Why are some patients not completely responsive to anti-TNF therapy regardless of which TNF antagonist is used? How can we identify patients who are most likely to respond to anti-TNF agents? These questions require further investigation. Additionally, remission in most patients with RA has not yet been achieved; this indicates an unmet need.

Although the TNF antagonists are proving to be effective and safe therapies for RA, there are other agents in development, and some of the preliminary data have been presented at the 65th American College of Rheumatology Conference during October 2002 in New Orleans, Louisiana. When the time comes for such prospective new therapies to be evaluated, they will have to be compared with the TNF biologic response modifiers, the new gold standard for the modification of RA.

Competing interests

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Acknowledgement

The transcript of the World Class Debate for ACR 2002 has been published electronically in *Joint and Bone*. This article, and others published in this supplement, serve as a summary of the proceedings as well as a summary of other supportive, poignant research findings (not included in the World Class Debate ACR 2002).

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DMARDs = disease-modifying antirheumatic drugs; NSAIDs = non-steroidal anti-inflammatory drugs; RA = rheumatoid arthritis; TNF = tumor necrosis factor.