UNDERSTANDING THE IMMUNOLOGY OF INFLAMMATORY ARTHRITIS

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The era of targeted therapies for the management of inflammatory arthritis arose out of two concurrent basic science advances over a generation ago. The first of these was an advance in protein engineering that gave rise to the ability to make monoclonal antibodies with specificity for a single protein target. Secondly, was the cataloguing and documentation of a role for various cells and proinflammatory mediators, in particular cytokines, in the pathobiology of inflammatory arthritis. Subsequent clinical trials unequivocally confirmed the importance of the particular therapeutic target in the pathology of a given disease. But furthermore, by using targeted biologic therapies as probes of pathogenesis, as well as investigating their efficacy in a range of inflammatory rheumatic disorders, it has become clear that while some pro-inflammatory mediators have a role in a wide variety of disease phenotypes, others are more restricted. For example, monoclonal antibodies targeting TNF have efficacy in inflammatory bowel disease (IBD), rheumatoid arthritis (RA), skin psoriasis (PsO), psoriatic arthritis (PsA) and ankylosing spondylitis (AS). IL6 inhibition is effective in rheumatoid arthritis and giant cell arteritis but less so in axial inflammation. IL17A inhibition has been shown to be effective in PsO, PsA and AS but not in RA. Similarly, IL23 is a key cytokine in the pathogenesis of PsO. In addition, understanding the roles of key components of the immune system such as B cell and T cells, how they become activated, and their role in the pathogenesis of inflammatory arthritis has given rise to targeted therapies directed to CD20 and co-stimulation pathways. In this lecture, we will explore the key cells and cytokines involved in inflammatory arthritis and how this knowledge might help inform a management choice.

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