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(Askandar Tjokroprawiro)

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Abstract

RA (rheumatoid arthritis) is a chronic progressive autoimmune disease with a high socio-economic burden. This disease is more common in women compared to men (3:1). Joint involvement occurs early in the natural history of the condition. At the cellular level, the immune system continues to recognize and respond to the autoantigen that leads to persistent activation CD4+ T cells and B cells. Activated B produces rheumatoid factor that lead to the formation of immune complexes in the synovial space, activating complement to stimulate migration of neutrophils into the synovial space. Chemokine production enhances the migration of mononuclear cells into the joint space, adding further to the exudative component of the disease. Proinflammatory cytokines also stimulate the production of collagenases, OPGL (osteoprotegerin ligand), and IL-6. IL-6, IL-18, and other growth factors stimulate fibroblast proliferation, resulting in invasive pannus formation, leading to osteoclast activation and bone destruction. In the final stage, the inflammatory reaction extinguishes and is replaced by fibrosis, which causes tendon adhesions and fixed deformities. One of the end results of this pathological inflammation is the disruption of the balance between flexor and extensor tendons, which produces the characteristic hand deformities.

Keyword: hand, deformities, rheumatoid, arthritis, ,

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