Prthritis Pathogenesis | rheumatoid arthritis pathogenesis centre of excellence

RHEUMATOID ARTHRITIS (RA) - CLINICAL OVERVIEW

Dr Florian MP. Meier, Brian E. Morton I University of Glasgow

PATHOGENESIS

ENVIRONMENT







GENES



INFECTIONS



And many other unknown factors...

TOLERANCE

- The immune system, capable of recognizing and fighting foreign organisms, is no longer tolerant of 'self' structures
- Instead, it attacks healthy tissues, which leads to tissue inflammation and destruction
 In rheumatoid arthritis,
- inflammation manifests as a painful disease of the joints

CHRONIC **AUTOIMMUNE** DISEASE

- · Chronic means the opposite of acute: it is a long-term disease
- Autoimmune means that the immune system fights against substances naturally present in the body
- Autoimmunity associated with rheumatoid arthritis does not exclusively affect the joints.

Who gets RA?

RA is the most common form of autoimmune arthritis, affecting more than 400,000 people in the UK. Of these, about 75% are women. In fact. 1-3% of women may get rheumatoid arthritis in their lifetime. The disease most often begins between one's thirties to fifties, However, RA can start

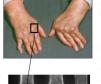
Normal joint



Early RA



Late RA



- Kidneys Eyes Lymph nodes
- Spleen

Heart Bone marrow

Lungs

Peripheral nerves

Organs also affected Cardiovascular system

- Muscles

Therapy for RA has *improved greatly* in the past 30 years. Current treatments give most patients good or excellent relief of symptoms and let them keep functioning at, or near, normal levels. With the right medications, many patients can achieve "remission" — that is, have no signs of active disease.

There is no cure for RA. Current treatments aim to lessen symptoms. Doctors do this by starting proper medical therapy as soon as possible, before joints have lasting damage. No single treatment works for all patients. Many people with RA must change their treatment at least once during their lifetime.

Drugs used to treat RA patients, referred to as disease-modifying anti-rheumatic drugs (DMARDs), have been designed to target specific immune cells involved in the pathogenesis of RA. Many of these drugs gain entry to their target cells and inhibit important pro-inflammatory molecules, while others act outside the cell to 'mop up' harmful proteins produced by these immune cells.

Macrophages





B Cells









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THERAPY