Understanding the Development of Rheumatoid Arthritis

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Introduction

Rheumatoid arthritis (RA) is an autoimmune disease that can be caused by genetic factors and environmental triggers that cause modifications to our own antigens such as immunoglobulin G (IgG) antibodies, type II collagen, or vimentin through the process called citrullination.[1] Citrullination is the process of converting the amino acid arginine to citrulline. This procedure confuses the bodies antigens presenting cells to activate CD4 T-helper. Along with the antibodies, T-helper cells enter the circulation to reach the joints. Inside the synovial fluid of the joint T-cells secrete cytokines to recruit macrophages, which also produce more cytokines, then stimulates the synovial cells to proliferate causing inflammation in the joint (Figure 1) .[1]

Rheumatoid arthritis can be diagnosed through blood tests looking for rheumatoid factor and anti-cyclic citrullinated peptide (Anti-CCP) antibody.[2] Additionally, X-rays are used to diagnose rheumatoid arthritis. Treatment for rheumatoid arthritis include nonsteroidal anti-inflammatory drugs which have a chance to increase the need for nutrients and reduce their absorption.[3] Omega-3 fatty acid supplements along with a balanced diet and antioxidants is shown to decrease inflammation.[4] Stem cell injections and surgical procedures are another way to treat arthritis.

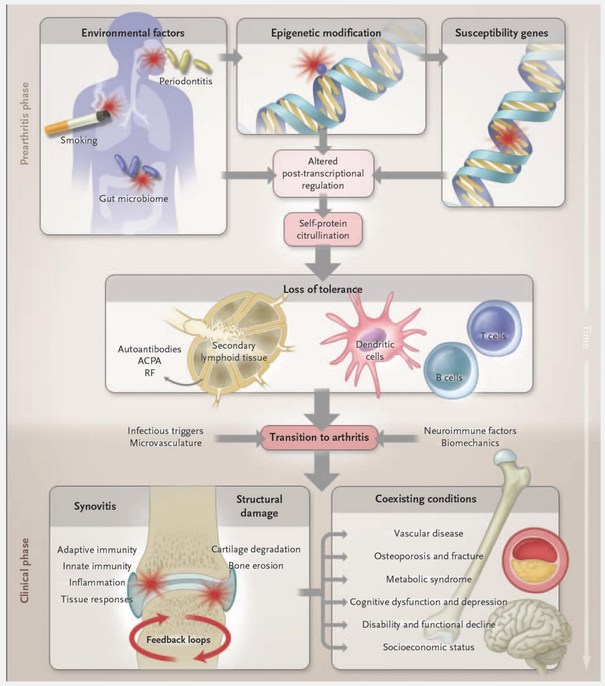

Figure 1. Summarized the process of rheumatoid arthritis.[1]

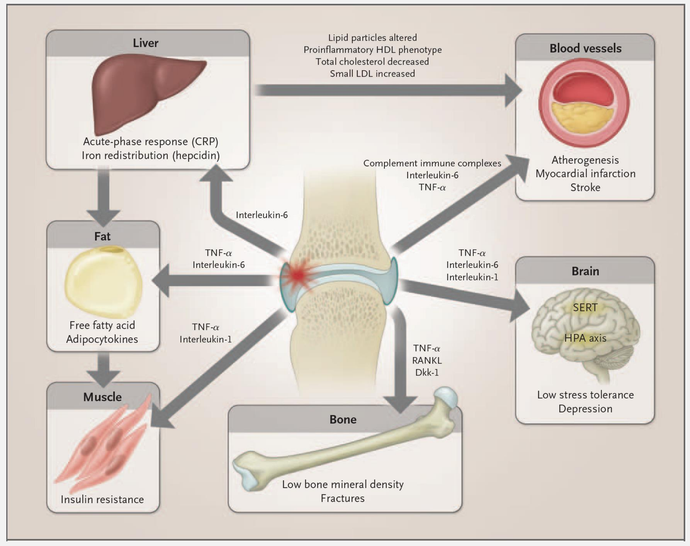

Figure 3. The different anatomy affected by certain inflammatory markers.[1]

Impact to the System

In rheumatoid arthritis, the inflammatory cytokines travel beyond the joints. Interleukin-1 or interleukin-6 in the brain and acts as pyrogens to induce fever.[7] In skeletal muscles the cytokines promote protein break down.[7] In the skin or visceral organs, the cytokines produce rheumatoid nodules which can lead to tissue death.[7] Blood vessels also become inflamed and can produce atheromatous plaques that can be the cause blockage in the blood vessels.[7] Heart attacks or strokes are the risks of atherosclerosis. The liver produces hepcid a protein that decreases serum iron levels by inhibiting the absorption by the duodenum and small intestine and trapping them in macrophages and liver cells.[7] In the lung interstitium fibroblasts become activated and produce scar tissue that decreases gas exchange. In the pleural cavities of the lung they become inflame and lead to pleural effusion.[7] Pleural effusion is the presence of fluid in the pleural cavity of the lungs that causes complications with lung expansion. Felty syndrome is a serious condition that is caused by rheumatoid arthritis, splenomegaly and granulocytopenia which can lead to life-threatening infections.[7] Splenomegaly is an abnormal enlargement of the spleen. Granulocytopenia is an acute condition that involves the lowering of white blood cells in the body. Felty syndrome causes people with the disease to become more susceptible to infections and develop leg ulcers (Figure 3).[7]

Diagnostic Imaging (MRI)

Diagnostic imaging for rheumatoid arthritis includes radiograph imaging to look at the bone destruction and tissue swelling. Early detection of RA can be found by the use of magnetic resonance imaging (MRI).[5] MRI machines use a strong magnetic field to measure the way hydrogen atoms receive and deliver electromagnetic energy then produce the image. With the use of MRI, radiologist can have a better in-depth imaging diagnosis of tissue and joint space. Another way to diagnose RA early is the use of a prediction model for persistent arthritis. This model uses diagnostic criteria to differentiate, at the first visit, between self-limiting, persistent nonerosive and persistent erosive arthritis (Figure 2).[5]

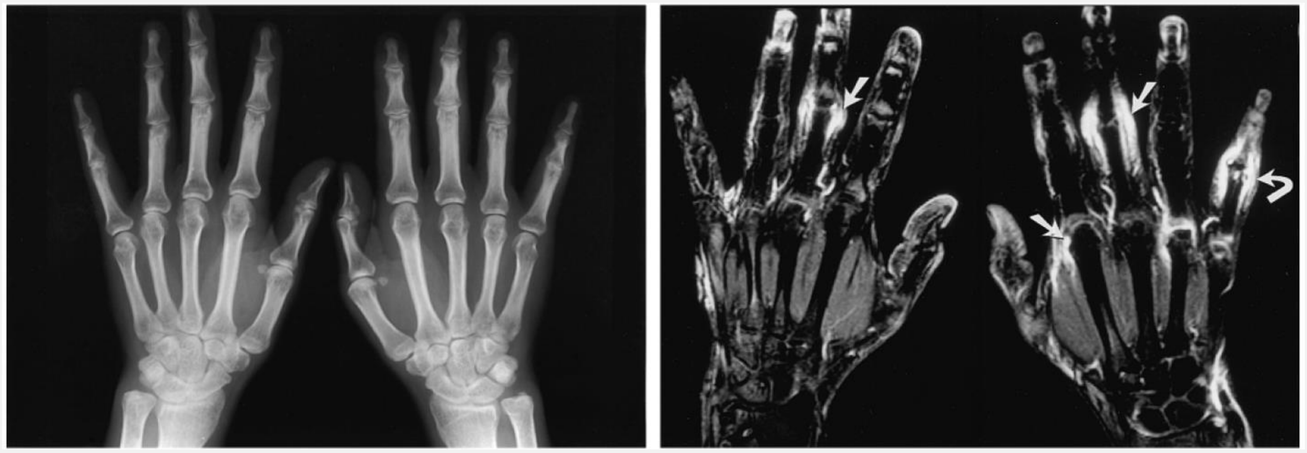

Figure 2. Demonstrates an X-ray on the left and a MRI on the right of a 42-year-old woman who tested true-positive for RA.[6]

References

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