Rheumatoid Arthirits Definition, Causes, Pathophysiology, clinical features, diagnosis and treatment

Arthritis is a descriptive term applied to more than 100 rheumatic diseases, ranging from localized, self-limiting conditions to those that are systemic autoimmune processes. Arthritis can affect persons of all age groups and is the second leading cause of disability.

Although the condition cannot be cured completely, much can be done to control its progress.

In systemic rheumatic diseases—those affecting body systems in addition to the musculoskeletal system, the inflammation is primary, resulting from an immune response.

In rheumatic conditions limited to a single or few diarthrodial joints, the inflammation is secondary, resulting from a degenerative process and the resulting joint irregularities that occur as the bone attempts to remodel itself

Rheumatoid arthritis is defined as a chronic systemic inflammatory disease characterized by bilateral involvement of synovial or diarthrodial joints

The initial joint changes involve the synovial cells that line the joint. In the process, the inflammatory cells accumulate and angiogenesis and pannus formation follow proceeding to cover the articular cartilage and isolate it from its nutritional synovial fluid take place.

Causes and pathogenesis of rheumatic arthritis

The exact cause of rheumatoid arthritis is idiopathic meaning it isn't exactly known. Although there is evidence of a genetic predisposition and immunologically mediated joint inflammation.

The development of rheumatoid arthritis is initiated in a genetically predisposed person by the activation of a T-cell–mediated response to an immunologic trigger, such as a microbial agent.

It has been shown that certain major histocompatibility complex (MHC) genes are expressed in a nonrandom manner in persons with RA.

An important genetic locus that predisposes to RA is present on the human leukocyte antigen (HLA) loci on the MHC class II molecules, with a specific set of HLA DR alleles (DR4, DR1, DR10,

DR14) being consistently increased in persons with RA.

These HLA DR alleles form a shared epitope in the hypervariable segment of the HLA-DRB1 gene, which forms a rheumatoid pocket on the HLA molecule.

Pathogenesis of rheumatoid arthritis

The pathogenesis of RA is an aberrant immune response that leads to synovial inflammation and destruction of the normal joint architecture. This process is usually initiated by the activation of helper T cells, the release of cytokines, and antibody formation. The majority of people suffering from rheumatoid arthritis have detectable, self-produced antibodies (IGF) known as the rheumatoid factor (RF). This rheumatoid factor reacts with a fragment of immunoglobulin G (IgG) to form immune complexes.

This Immune complexes of immunoglobulin G and immunoglobulin F (Ig RF + IgG) together with complement proteins are found in the synovium, synovial fluid, and extra-articular lesions.

When lab tests are done in patients suffering from rheumatoid factor, these individuals with RA may be seronegative without any Ig RF present in their serum.

Conversely, the presence of a high RF titer is associated with severe disease and systemic complications.

At the basic cellular level, neutrophils, macrophages, and lymphocytes are attracted to the area. These neutrophils and macrophages phagocytize the immune complexes and, in the process, they release lysosomal enzymes capable of causing destructive changes in the joint.

The inflammatory response that follows attracts additional inflammatory cells, setting into motion a chain of events that perpetuates the condition. As the inflammatory process progresses, the synovial cells and subsynovial tissues undergo reactive hyperplasia. Vasodilation and increased blood flow cause warmth and redness.
The joint swelling that occurs is the result of the increased capillary permeability that accompanies the inflammatory process.

Characteristics of rheumatoid arthritis

The characteristic of rheumatoid arthritis is the development of an extensive network of new blood vessels in the synovial membrane that contributes to the advancement of rheumatoid synovitis.

This destructive vascular granulation tissue, which is called pannus, extends from the synovium to involve the "**bare area**," which is a region of unprotected bone at the junction between cartilage and subchondral bone.

Pannus is a feature of RA that differentiates it from other forms of inflammatory arthritis.

The inflammatory cells found in the pannus have a destructive effect on the adjacent cartilage and bone. Eventually, pannus develops between the joint margins, leading to reduced joint motion and the possibility of eventual ankylosis.

With the progression of the disease, joint inflammation and the resulting structural changes lead to joint instability, muscle atrophy from disuse, stretching of the ligaments, and involvement of the tendons and muscles.

The effect of the pathologic changes on joint structure and function is related to the degree of disease activity, which can change at any time. These destructive changes in the joints are irreversible.

Signs and symptoms of rheumatoid arthritis

Classical features of rheumatoid arthritis are associated with extra-articular as well as articular manifestations.

Its onset is insidious in nature and is marked with systemic features such as excessive fatigue, loss of appetite, weight loss, and generalized body malaise and stiffness.

Rheumatoid arthritis characterized by exacerbations and remissions may involve only a few joints for brief durations, or it may become relentlessly progressive and debilitating.

Joint Manifestations.

RA has a characteristic symmetrical and polyarticular Joint involvement consisting of any diarthrodial joint.

Patients may complain of joint pain and stiffness that lasts for 30 minutes and frequently for several hours.

There is a limited joint motion that occurs early in the disease mainly as a result of joint pain; later, this limited joint motion results from fibrosis.

The commonly affected joints initially are the fingers, hands, wrists, knees, and feet. Later, other diarthrodial joints may become involved.

Spinal involvement usually is limited to the cervical region. In the hands, there usually is bilateral and symmetric involvement of the proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints in the early stages of RA; the distal interphalangeal (DIP) joints rarely are affected.

The fingers often take on a spindle-shaped appearance because of inflammation of the PIP joints.

Progressive joint destruction may lead to subluxation or a dislocation of the joint resulting in misalignment of the bone ends and instability of the joint and limitation of movement.

Swelling and thickening of the synovium can result in stretching of the joint capsule and ligaments. When this occurs, muscle and tendon imbalances develop, and mechanical forces applied to the joints through daily activities produce joint deformities.

In the metacarpophalangeal joints, the extensor tendons can slip to the ulnar side of the metacarpal head, causing ulnar deviation of the fingers.

Subluxation of the MCP joints may develop when this deformity is present.

Hyperextension of the PIP joint and partial flexion of the DIP joint is called a swan neck deformity.