Lecture 2

Immunology

Fourth Class

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Rheumatoid arthritis, or RA, is an autoimmune and inflammatory disease, which means that your immune system attacks healthy cells in your body by mistake, causing inflammation (painful swelling) in the affected parts of the body.

mainly attacks the joints, usually many joints at once. RA commonly affects joints in the hands, wrists, and knees. In a joint with RA, the lining of the joint becomes inflamed, causing damage to joint tissue. This tissue damage can cause long-lasting or chronic pain, unsteadiness (lack of balance), and deformity (misshapenness).RA can also affect other tissues throughout the body and cause problems in organs such as the lungs, heart, and eyes

symptoms of RA include:

1-Pain or aching in more than one joint

2- Stiffness in more than one joint

3-Tenderness and swelling in more than one joint

4-The same symptoms on both sides of the body (such as in both hands or both knees)

5-Weight loss

6-Fever

7- tiredness

8-Weakness

What causes RA?

RA is the result of an immune response in which the body's immune system attacks its own healthy cells. The specific causes of RA are unknown, but some factors can increase the risk of developing the disease.

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Pathogenesis :

pathogenesis of RA are complex, involving many types of cells, among others macrophages, T and B cells, fibro- blasts, chondrocytes and dendritic cells. Despite well documented role of many genes and epigenetic modifications in the development and evolution of the disease, in most RA patients there is no clear predisposing factor present. Environmental factors involved in RA pathogenesis are cigarette smoke, industrial pollutants like silica crystals, disturbances of intestinal, lung, and oral microbiota and some specific bacterial and viral infectious agents and their components. In the initial disease stage there are qualitative and quantitative disturbances o f peptide citrulination as well as other protein modifications, followed by antigen presenting cell (APC) (macrophages and dendritic cells) and fibroblast . Some microbes foster this processes by APC and and indirect activation. In the second stage APC's elicit specific humral B cell re-sponse resulting in specific antibodies production and T cell auto reactivity. Inherited and acquired defects in T and B cell responses caused by repeated activation of innate immunity as well as loss of tolerance, elicit chronic autoimmune inflammation, primarily of synovial membranes, and development of cellular panus. Pathologic activation of the osteoclasts and release of the immune system effector molecules and the proteolytic enzymes damage the cartilage, bone and tendons composition and structure. Persistent inflammation through its complex mechanisms results in many systemic and extraarticular RA manifestations of almost all organ systems.

Tretment :

Medications

1-**NSAIDs. Nonsteroidal anti-inflammatory drugs (NSAIDs**) can relieve pain and reduce inflammation. Over-the-counter NSAIDs include ibuprofen (Advil, Motrin IB) and naproxen sodium (Aleve). Stronger NSAIDs are available by prescription. Side effects may include stomach irritation, heart problems and kidney damage.

2-**Steroids**. Corticosteroid medications, such as prednisone, reduce inflammation and pain and slow joint damage. Side effects may include thinning of bones, weight gain and diabetes. Doctors often prescribe a corticosteroid to relieve acute symptoms, with the goal of gradually tapering off the medication.

3-**Disease-modifying antirheumatic drugs (DMARDs**). These drugs can slow the progression of rheumatoid arthritis and save the joints and other tissues from permanent damage. Common DMARDs include methotrexate (Trexall, Otrexup, others), leflunomide (Arava), hydroxychloroquine (Plaquenil) and sulfasalazine (Azulfidine).