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Evaluation of innate immunity in a sample of Iraqi Rheumatoid arthritis patients

Dunya Fareed Salloom* and Ameer Khalid Sabri

Department of Biology, College of Science, University of Baghdad, Baghdad, Iraq.

* Corresponding author: Dunya Fareed Salloom; e-mail: dunyascience@yahoo.com

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ABSTRACT

Rheumatoid arthritis, an autoimmune disease is indicated in the pathogenesis and literature suggests many factors contributed in triggering the disease. In view of this, the present study was designed to evaluate the complement component C_3 and C_4 concentration as a part of innate immunity. The study included 35 patients with RA and 20 individuals as control. The result appeared significant differences ($p<0.05$) between the complement component concentrations in studied groups. This lead to suggest that complement as a part of innate immunity may have a role in the pathogenesis of the disease.

Keywords: rheumatoid arthritis, innate immunity complement.

1. INTRODUCTION

Rheumatoid arthritis (RA) is an autoimmune disease, or it is one of chronic inflammation of the joints. [1]. The pathogenesis of RA is unknown. Autoimmune diseases are illnesses that occur when the body's tissues are mistakenly attacked by their own immune system. The immune system contains a complex organization of cells and antibodies designed normally to "seek and destroy" invaders of the body, particularly infections. Patients with autoimmune diseases have antibodies and immune cells in their blood that target their own body tissues, where they can be associated with inflammation. While inflammation of the tissue around the joints and inflammatory arthritis are characteristic features of rheumatoid arthritis, the disease can also cause inflammation and injury in other organs in the body. Because it can affect multiple other organs of the body, rheumatoid arthritis is referred to as a systemic illness and is sometimes called rheumatoid disease [2].

Rheumatoid arthritis (RA) is a long-lasting autoimmune disorder that primarily affects joints [1]. It typically results in warm, swollen, and painful joints. Pain and stiffness often worsen following rest. Most commonly, the wrist and hands are involved, with the same joints typically involved on both sides of the body. The disease may also affect other parts of the body such as inflammation around the lungs, and inflammation around the heart. Fever and low energy may also be present [3]. Often, symptoms come on gradually over weeks to months [4], while the cause of rheumatoid arthritis is not clear, it is believed to involve a combination of genetic and environmental factors. The underlying mechanism involves the body's immune system attacking the joints. This results in inflammation and thickening of the joint capsule [5]. X-rays and laboratory testing may support a diagnosis or exclude other diseases with similar symptoms. The treatment is important to reduce pain, decrease inflammation, and improve a person's overall functioning [6]. CRP was elevated in rheumatic and other inflammatory diseases; malignancy, tissue injury and necrosis. [7].

The complement is a part of the immune system that enhances the ability of antibodies and phagocytic cells to clear microbes and damaged cells from an organism, promotes inflammation, and attacks membrane. It is part of the innate immune system [8].

2. MATERIALS AND METHODS

2.1 Study group

This study included 35 patients with age range (47±2 years) and control group comprising of 10 (42±1 years) individuals. All studied groups were evaluated for C3 and C4 by single radial immunodiffusion technique, CRP and RF by agglutination test.

2.2 Statistical analysis

The data were expressed as mean ± S.E, probability (*P< 0.05*) considered significant by using IBM computer program SPSS version 24.

3. RESULTS AND DISCUSSION

The present study showed significant differences (*P<0.05*) between the concentration of C3 in patient compared to control concentration, as well, the concentration of C4 showed significant differences (*P<0.05*) between studied group as in table (1). Levels of complement proteins are generally depressed in the synovial fluid of patients with RA, reflecting consumption of complement. On the other hand, elevated levels of several complement cleavage products have been observed in synovial fluid [9].

Table 1: Complement components C_3 and C_4 concentration in studied groups.

Test	Patients group	Control group
C_3 (mg/dl)	120.3 ± 0.03	88 ± 1.2
C_4 (mg/dl)	33.21 ± 1.6	15.66 ± 1.02

The classical pathway is the main complement pathway triggered, presumably via binding of immune complexes containing rheumatoid factor. The alternative pathway is also activated in RA synovium. Increased complement activation via the lectin pathway could also play a role in RA. Changes in glycosylation of IgG in RA cause an increase in binding of mannose-binding lectin resulting in increased complement activation. The complement system might play a role in diseases with an immune component, such as Barraquer-Simons Syndrome, asthma, lupus erythematosus, glomerulonephritis, various forms of arthritis, autoimmune heart disease, multiple sclerosis, inflammatory bowel disease, ischemia-reperfusion injuries, and rejection of transplanted organs. The complement system is also becoming increasingly implicated in diseases of the central nervous system such as Alzheimer's disease and other neurodegenerative conditions such as spinal cord injuries. Additionally, deficiencies in complement proteins produced in the liver can lead to a form of primary (congenital) immunodeficiency, in which the body is more susceptible to disease, particularly autoimmune diseases and severe bacterial infections [10].

The complement system comprises several fluid-phase and membrane-associated proteins. C3 is the point at which all complement pathways converge, and complete deficiency of C3 invariably leads to severe infections, including those caused by meningococci and pneumococci. Deficiencies of the alternative and terminal complement pathways result in an almost exclusive predisposition to invasive meningococcal disease [11-12].

The study also showed seropositivity of RF and CRP in patients group, while negative sera in control as in figure (1) and figure (2). From the present study we suggest the complement may play a role in pathogenesis of RA.

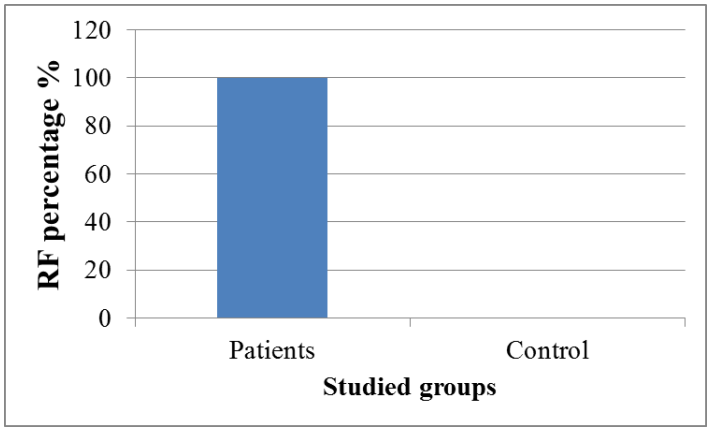

Figure 1: RF in studied groups

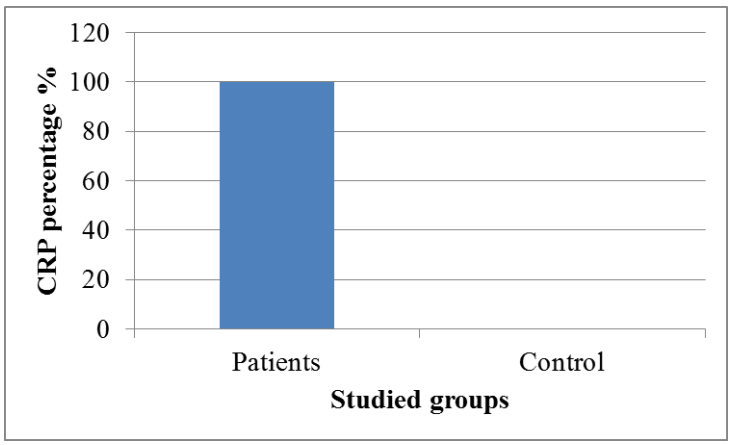

Figure 2: CRP in studied groups.

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