

STUDY OF SOME IMMUNE PARAMETER AMONG PATIENTS WITH RHEUMATOID ARTHRITIS

Rusul Mahdi Sahib Shukur and Angham Jassim Al-Ramahy*

Pathological Analysis, College of Health and Medical Techniques, Al-Forat Al-Awsat Technical University, Najaf, Iraq.

*e-mail : kin.angh@atu.edu.iq

(Received 3 July 2019, Accepted 5 August 2019)

ABSTRACT : The study was conducted at Al-Sadder Medical City in Al-Najaf province from September 2018 to December 2018. One hundred fifty blood specimens were collected from different age groups and of both sex, these samples included 75 patients and 75 apparently healthy individuals (control). Blood was withdrawn from a vein, for hematology analyzer (total WBCs count, total RBCs count and ESR) while serum were used for immunological tests including IL-33, TNF- α and C-reactive protein by ELISA technique. All patients diagnosed with RA by ACCP test. The study showed that RA more common in females (73.3%) rather than males (26.7%), with incidence among age groups(41-50), (41.3%) and in urban (74.7%) rather than rural (25.3%). Immunological Parameters showed significantly high levels among RA patients, which include HS-CRP (mean = 0.059), TNF- α (mean = 386.713) and IL-33(mean = 733.015). The result of correlation between these parameters appear that TNF- α was stimulated to IL-33, which increased among RA patients compare with control group.

Key words : Rheumatoid arthritis, tumor necrosis factor-alpha, interleukin-33, C-Reactive protein.

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic inflammatory autoimmune disease characterized by symmetrical peripheral polyarthritis that can lead to joint destruction and may be associated with extra articular features such as keratitis, pulmonary granulomas (rheumatoid nodules), pericarditis/pleuritis, small vessel vasculitis and other non-specific extra-articular symptoms (Tracy *et al*, 2017).

Rheumatoid arthritis (RA) is a chronic autoimmune disease affecting 0.3–0.8% of the population (Roux *et al*, 2007). There have been major advances in our understanding of genetic risk from genome-wide association studies. There has also been renewed interest in environmental factors, especially lifestyle factors that are potentially modifiable (Lahiri *et al*, 2011). Smoking is the most consistent association and contributes up to 25% of population attributable risk of RA (Källberg *et al*, 2011). The risk appears to be related, stronger in men and in carriers of the shared epitope (SE) and for anti-citrullinated peptide antibody positive (ACPA+) RA. Some studies support a protective role of breast feeding in women. There are association with alcohol intake (Di Giuseppe *et al*, 2012). Higher education/social class and an increased risk with obesity (Lahiri *et al*, 2014). ACPAs and RFs are produced by synovial tissue B cells

and can be detected in synovial fluid, suggesting that they could play a role in local innate immunity activation and complement fixation (Randen *et al*, 1992). T cells, as one of the most abundant cell population in the RA, are aberrantly activated in RA to drive chronic inflammation and joint destruction (Tran *et al*, 2005). RA T cells interact with other immune and resident cells, including B cells, macrophages, synoviocytes and osteoclasts by secreting a variety of cytokines and chemokines and/or by direct cell-to-cell contact, and ultimately boost their pro-inflammatory action. The role that diverse T-cell populations play in the induction, amplification, and maintenance of inflammatory arthritis has been elucidated in various animal models of RA (Alzabin, Williams, 2011). TNF α is a pro-inflammatory cytokine which plays a pivotal role in the origin and progression of RA. TNF is a trimeric protein cytokine that is produced mainly by monocytes and macrophages. Tumor necrosis factor (TNF) is a pleiotropic cytokine that is transcriptionally activated in response to a variety of stimuli during inflammation, infection and stress TNF is mostly produced by mononuclear phagocytes but is also expressed by neutrophils, lymphocytes, endothelial cells and fibroblasts. The membrane-bound precursor and the cleaved, soluble form of TNF are both biologically active and exert their effects *via* two structurally distinct transmembrane