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The Role of Innate Immune Response And IL-6 IN Osteoarthritis Patients in Basrah Province

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Abstract

Background: Osteoarthritis (OA) has been observed as degeneration disease of joint cartilage and the underlying bone affecting millions of people worldwide. OA has traditionally been classified as a non-inflammatory arthritis but with evidences accumulation indicates the role of immune response and inflammation in OA onset and progression.

Aim of study: To shed the light on role of innate immune response, CRP and IL-6 level in the development of OA in Basra province.

Patients and methods: 55 osteoarthritis patients (11 males and 44 females) and 70 healthy individuals as control (24 males and 46 females) were participate in this study. Total and differential WBCs count in whole blood in addition to the levels of all ESR, CRP and IL-6 were measured in serum for all participants.

Results: The results were recorded a significant increasing in total WBCs count, neutrophils and monocyte in addition to the level of all inflammatory markers (ESR, CRP and IL-6) in osteoarthritis patients as compared with healthy control. While lymphocytes and platelets count record an increase in OA patients but insignificant . A significant positive correlation was found between IL-6 level in serum and each of CRP, ESR, WBCs and the platelet. In addition to a significant correlation was found between CRP and both of WBCs and ESR.

Conclusion: The results indicates the active participation of inflammatory response in development of osteoarthritis in addition to the importance role for IL-6 as considered multifunctional cytokines effect different immunological parameters in the body and has adverse effect in development of OA.

Keywords: Osteoarthritis, inflammatory response, CRP, ESR, IL-6

Introduction

Osteoarthritis (OA) is a chronic and dynamic disorder characterized by a progressive degeneration of articular cartilage , bone remodeling leading to joint space narrowing (JSN) associated with pain, and loss of function ([1] ,[2],[3]) .Any joint can be affected by OA but more frequently the weight-bearing joints particularly knees and hips. ([3])

Numerous risk factors have been related to that disease including genetic predisposition, age, sex, obesity, reproductive status (e.g., postmenopausal) in females, individuals variation in physical activity and a history of previous joint trauma. ([1],[3],[4],[5])

Initially, OA involves a low-grade inflammatory disease (innate immune response) before a mild degree of adaptive immunity. ([6] ,[7],[8]) During tissue damage, damage-associated molecular pattern (DAMP) which are group of endogenous molecules, provide signals to the innate immune cells (like macrophages and mast cells) mediating a protective response and enhancing both wound healing and tissue repair. Mast cells regulate vascular permeability, facilitating leukocyte infiltration ([8])while macrophages display a plasticity function depending on the environment where they are present. Upon prolonged stimulation by DAMP molecules and chronic activation , macrophages can lead to more producing of proinflammatory cytokines that make OA joints worst. ([9], [10])

Several proinflammatory cytokines have been found to play a role in OA progression, one of them is IL-6. IL-6, is an important pro-inflammatory cytokine produced by many non-immune tissues and by many immune and nonimmune cells like T cells, B cells, monocytes, macrophages as well as fibroblasts and osteoblasts ; in vivo IL-6 is considered as a central mediator for regulating immune and inflammatory responses. ([7],[11],[12])

Healthy chondrocytes produce IL-6 in low amount without stimulation but upon exposure to certain cytokines during inflammation like TNF-α and IL-1β, chondrocytes will increase its production([3],[7]). The exact mechanism action of IL-6 in OA is not yet understood , some *in-vitro* studies showed that IL-6 will inhibit type II collagen production by rabbit articular chondrocytes ([13]) and the combination of IL-6 with IL-1β and TNF will regulate matrix metalloproteinases (MMP-1) production in human and bovine chondrocytes as