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Diagnostic and Predictive Values of IL-6 in a Group of Iraqi Patients with Rheumatoid Arthritis

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Abstract

Background: Researchers have found that interleukin 6 (IL-6) plays a crucial regulatory function in the onset and progression of a wide range of inflammatory disorders. One of the more prevalent inflammatory illnesses affecting people today is rheumatoid arthritis.

Aim of the study: The purpose of this study was to compare the IL-6 levels of rheumatoid arthritis (RA) patients to those of healthy controls and to examine the relationship between IL-6 and RA-related demographic and clinical factors.

Materials and Method A total of 80 participants: 40 rheumatoid arthritis (RA) sufferers and 40 healthy controls, all of whom ranged in age from 23 to 61. The serum concentrations of (IL-6) were analyzed using an enzyme-linked immunosorbent assay (ELISA).

Results: Increased IL-6 blood levels were associated with rheumatoid arthritis, suggesting that this biomarker may be useful for diagnosing the disease at an early stage. There was no statistically significant correlation between disease severity and the DAS28 score and IL-6 levels in the serum.

Conclusion: The cytokine interleukin 6 (IL-6) has been proposed as a biomarker and possible player in the etiology of rheumatoid arthritis.

Keywords: Rheumatoid arthritis (RA), cytokines, IL-6

Introduction:

Joint and cartilage inflammation is a hallmark of rheumatoid arthritis (RA), an inflammatory disease that can develop into osteoarthritis and a range of symptoms and impairments. The prognosis of RA has been drastically altered by the availability of several therapy alternatives, even when its development and progression are still not fully understood [1]. Patients can maintain control of their condition and lead normal lives with the use of disease-modifying antirheumatic medications (DMARD) like methotrexate (MTX). However, not every patient is helped by these medications, and for those who don't, the quality of life is drastically reduced [2]. In general, environmental and genetic factors contribute to RA development, with the former impacting the latter. It is generally agreed environmental variables [3]. Joint T-cell receptor (TCR) limitation has also been reported, which points to an antigen being attacked by T cells in the joints.

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It may also be useful for forecasting how well standard treatments will work or how severe a patient's illness will be [4]. Smoking, which alters the oral microbiota and may set-off autoimmune cross-reactions, is a major environmental risk for RA [5]. Some infections, such Epstein-Barr and cytomegalovirus (CMV), have a similar effect: When the immune system is overstimulated by an infection, it tends to repress T-regs and promote the growth of Th17 cells [6]. Chronic exposure to citrullinated peptides under situations of chronic inflammation, such as those caused by the aforementioned illnesses, is a crucial stage in the etiology of RA in susceptible persons [7]. One must also recognize the part played by microbiota: The microbiota has long been recognized as an important regulator of inflammation and immunity due to its ability to either stimulate or dampen systemic inflammation and alter the host's cytokine production pattern [8]. The oral microbiome appears to have a key role in RA. Indeed, it promotes cross-reactivity with proteins produced in the joints [9] and the development of aberrant neutrophils, which appear to play a crucial role in RA. As the most common bacterium found in RA patients, *Lactobacillus salivarius* has been shown to exhibit molecular mimicry of antigens implicated in the disease. The oral microbiota of RA patients really changes and becomes more typical of the general population after they begin receiving treatment [10]. IL-6: Because of its pleiotropic and redundant

functional activity, interleukin (IL)-6 serves as a paradigmatic cytokine. IL-6 is a member of the IL-6 cytokine family, which also includes IL-11, IL-27, IL-31, oncostatin M (OSM), leukemia inhibitory factor (LIF), ciliary neurotrophic factor (CNTF), cardiotrophin 1 (CT-1), and cardiotrophin-like cytokine factor 1 (CLCF1) [11]. Infection and various forms of inflammation can also trigger the release of IL-6. In fact, IL-6 is quickly generated, mostly by macrophages, in response to infections or inflammation-related damage-associated molecular patterns [12] and has a protective function by eliminating infectious agents and repairing injured tissue via activation of acute phase and immunological responses. Both innate and adaptive immunity rely heavily on IL-6. The synthesis of IL-6 is greatly increased in areas of inflammation and is carried out by a variety of cell types, including monocytes, T-lymphocytes, fibroblasts, and endothelial cells. [1**3**]. Toll-like receptors (TLRs) are involved in the recognition of bacteria, viruses, or fungi during an infection and can stimulate the production of IL-6 and other inflammatory cytokines like interleukin-1 (IL-1) and tumor necrosis factor (TNF-) via the nuclear factor kappa B (NF-kB) signaling pathway. Interestingly, IL-6 production is also stimulated by IL-1 and TNF- [14]. Persistent inflammation results from IL-6 production that is out of whack [1**5**]. IL-6's impact on the immune system is fascinating, as it may be seen in both innate and acquired immunity. Neutrophil migration and mononuclear cell infiltration are both facilitated by IL-6, making it an important component of innate immunity. In addition to its role as a chemoreceptor for monocytes at the site of inflammation [16], IL-6 also exhibits its effect on T-cells and B-cells throughout the process of acquired immunity. Serum gamma-globulin concentrations are raised as a result of its differential influence on plasma-cells. Castelman's illness, which improves with anti-IL-6 therapy, features both of these disorders. The role of IL-6 in T-cells is especially important because of the correlations it has been found to have with a wide range of diseases. In particular, IL-6 operates on CD4+ T-cell differentiation, stimulating the Th17 pathway while suppressing the development of regulatory-T-cells (T-reg). Anti-inflammatory properties of IL-6, which is increased by Th17 differentiation, highlight its significance in the battle against inflammation. Among the cytokines generated by Th17 cells are the pro-inflammatory cytokines TNF, IL-1, IL-17, IL-21, and IL-22 [18]. However, IL-6 blocks T-reg cell development via transforming growth factor (TGF). Under the correct circumstances, IL-6 is an autoinflammatory mediator that not only impairs the body's ability to recognize self from non-self but also promotes fibrosis and the inflammatory response. Evidence for and emphasis on its significance in a variety of autoimmune illnesses [14]. Beyond its inflammatory role, IL-6 is known to play other homeostatic and metabolic roles. More than a decade of anti-IL-6R medication usage in rheumatoid arthritis (RA) [3] has increased our understanding of the wide range of effects produced by this cytokine. Both tocilizumab and etanercept, biological medicines that block IL-6 and TNF, are widely used to treat RA. [19,20].

Materials and Methods

Study population: This is a case-control study on 80 individuals: 40 RA patients and 40 healthy controls almost matched for age and sex. The cases were recruited from the Rheumatology outpatient clinic in Baghdad Teaching Hospital from March - May 2021. The 40 Iraqi RA cases were diagnosed by a rheumatologist according to the American College of Rheumatology (ACR) 1987 criteria or ACR-EULAR (European Alliance of Associations for Rheumatology) 2010 criteria. Of those there was 8 male and 32 female aging from 23 to 60 years. The cases were classified as mild, moderate, and severe by the disease activity score-28 joints (DAS28 score). The 40 healthy age and sex matched controls were recruited from the blood donor bank.

Blood sample collection, preparation and testing: Each participant had five milliliters of blood drawn from them using sterile procedures. Blood samples were centrifuged to separate the serum from the supernatant, then the supernatant was carefully discarded. The separated serum was moved to Eppendorf tubes, then aliquoted and stored at -20°C until the IL-6 Enzyme-linked Immunosorbent Assay (ELISA) test was performed to measure the serum IL-6 concentration as per the manufacturer's instructions (Shanghai / China).

Statistical analysis: The Mean± SD was used for normally distributed numerical variables and the median (interquartile range) was used for non-normally distributed numerical variables in the descriptive statistical analysis. Rates and proportions were calculated for categorical data. The association between socio-demographic characteristics and IL-6, was examined by the Chi-square test the Fisher exact test. The Mann Whitney U test-T and the independent T-test were used to test for differences between sample means. The ROC Curve was utilized to determine the area under the curve (AUC). Using the enter technique, we performed a multiple linear regression analysis to identify the variables influencing or linked with IL-6. SPSS version 24 was used for all statistical analyses. Significant results were accepted at the 0.05 level.

Results

Table 1 shows the description of cases and controls in terms of age, gender, smoking and BMI. It shows that patients and controls were well matched for the first three variables (P = 0.576, 1.000, and 0.552, respectively). A statistically significant difference was found between the two groups in terms of body mass index (P=0.008).

Table 1: Description of the Cases and Controls by Gender, Smoking, Age and BMI

Variables	Categories	Patients		Control		P- Value
		N	%	N	%	
Gender	Male	9	22.5	7	17.5	0.576[a]
	Female	31	77.5	33	82.5	
Smoking	Non-Smoker	37	50.0	37	50.0	1.000[b]
	Ex-smoker	3	50.0	3	50.0	
		Mean	SD	Mean	SD	
Age (years)		47.0	10.32	45.6	10.62	0.552[c]
BMI (Kg/m^2)		26.5	5.57	29.8	5.15	0.008[c]

Table 2 shows that the mean CCN3 level for the patients was 4.0±19.64 while for the controls was 0.1±0.26. The mean IL-6 for the patients was 121.8±60.93 while for the controls was 71.3±50.47. Regarding WBC, the mean among patients was 4.7±14.20, HB 8.5±15.70, platelets 150±450, ESR mean score was 25 ± 9.01, Urea 15±42.01, Creatinine 0.8±41.01, SGPT 6.2±40.02, SGOT 7.8±53.01.

Table 2: Laboratory findings for the cases and controls

Variables	**Patients**		**Controls**	
	Mean	**±SD**	**Mean**	**±SD**
CCN3ng/dl	3.98	19.64	0.13	0.26
Interleukin 6ng/dl	121.82	60.39	71.25	50.47
White blood cells (10^3/**μ**l)	4.70	14.20	-	-
Hemoglobin (mg/dl)	8.5	15.70	-	-
Platelets (10^3/**μ**l)	150	450	-	-
Erythrocyte sedimentation rate (mm/hr.)	25	9.01	-	
Urea (mg/dl)	15	42.01	-	-
Creatinine (mg/dl)	0.8	41.01	-	-
Serum glutamic pyruvic transaminase (**μ**/l)	6.2	40.02	-	-
Serum glutamic oxaloacetic transaminase (μ/l)	7.8	53.01	-	-

Comparing the IL-6 levels in cases and controls has shown that the median and the IQR were 102.54 / 28.19 ng/dl and 80.55 / 62.11 respectively which was statistically significant (P< 0.001).

Figure 1 depicts the correlation between patients and controls with regards to IL-6. They were linked in a way that was statistically significant (p< 0.001).

Table 3: Serum levels IL-6 concentrations in patients and controls

Variables	Patients		Control		P-value
	Median ng/dl	IQR	Median ng/dl	IQR	
IL-6	102.54	28.19	80.55	62.11	<0.001

IL-6= Interleukin 6, IQR= Inter quartile range

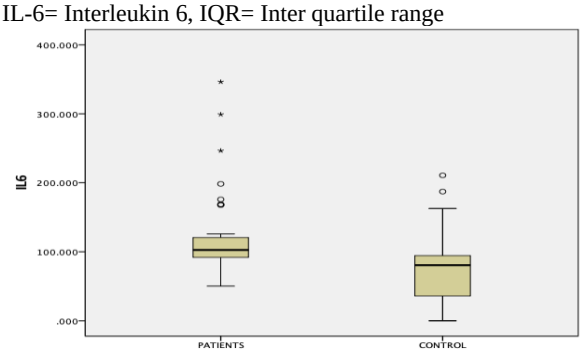

Figure 1: Error bars of IL-6 in patients and controls

Disease activity was measured using the DAS28 score and there were 3 cases with a remission, one case classified as mild, 30 as moderate and 6 as severe. Table 3 shows the mean±SD of IL-6 for each of these four classifications. And its correlation with IL-6 level is shown in Table 5. At P = 0.928 and P = 0.429, respectively. There was no statistically significant difference between IL-6 mean levels of the four DAS28 score categories. Mean IL-6 levels were highest in participants with moderate illness severity.

Table 3: Mean± SD IL-6 levels in the four disease categories according to the DAS28 score

Variables	**Remission (N=3)**		**Low (N=1)**		**Moderate (N=30)**		**Severe (N=6)**		**P value**
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
IL-6	84.7	14.78	120.0	---	130.3	68.99	99.4	8.89	0.429

Table 4 shows the multiple linear regression which was calculated to predict IL-6 related factors. There was no significant association between age, gender, BMI, duration of the disease, disease activity, DAS28 score and IL-6 with P value (0.895, 0.850, 0.389 ,0.717, 0.500, 0.710) respectively.

Based on Regression coefficient β, BMI had the strongest relationship with IL-6 (β =.248) followed by CRP. The R square value was 0.158 that means the model success to explain 15.8% of the factors related to IL-6.

Table 4: Demographic, clinical and lab characteristics of the cases and their impact on IL-6through multiple linear regression analysis

Variables	Regression coefficient B Beta	*P value*	95% Confidence Interval
Age (Years)	.038	.895	3.644
Gender	.050	.850	92.674
BMI (Kg/m^2)	.248	.389	8.975
Disease duration (Years)	.087	.717	5.819
Disease activity	-.278	.500	74.677
DAS28	.141	.710	62.211
White Blood Cells (10^3/μl)	-.150	.667	17.551
Haemoglobin (mg/dl)	-.048	.877	24.958
Platelet (10^3/μl)	.042	.894	.736
C-Reactive Protein (mg/dl)	.156	.521	33.940
Erythrocyte Sedimentation Rate (mm/hr.)	.137	.697	3.068
Urea (mg/dl)	.034	.887	4.848
Creatinine (mg/dl)	-.146	.570	3.089
SGPT(μ/l)	.097	.782	5.791
SGOT(μ/l)	-.048	.861	3.912
CCN3	.090	.762	2.113

Figure 2 shows the Receiver Operating Characteristics (ROC) curve. The area under the curve (AUC) was 0.796 with a statistically significant result (P<0.001) 95% CI (0.696 - 0.895).

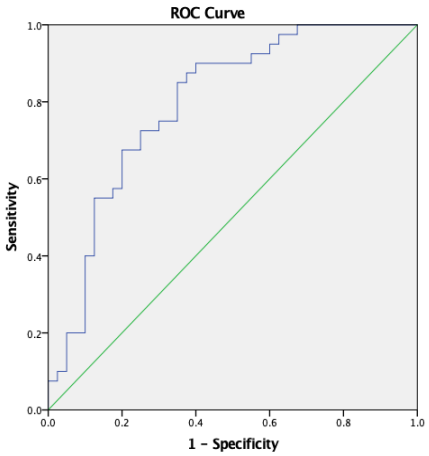

Figure 2: Roc analysis to test IL-6 ability to differentiate between RA patients and controls

Table 5 shows the validity parameters (Sensitivity, Specificity, positive predictive value, negative predictive value, and accuracy) of the cut-off value of IL-6 to differentiate between RA patients and controls

Table 5: Validity parameters of the IL-6 cut-off value

Variable	AUC	Optimum cut-off value	Sensitivity	Specificity	Accuracy	PPV	NPV
IL-6	0.796	72.9	92.5 %	42.5 %	67.5 %	61.7%	85.0%

The area under the curve (AUC) is 0.796 with the optimum cut-off value for IL-6 being 72.9 to differentiate between RA patients and controls. The sensitivity, specificity, and accuracy of IL-6 in identifying RA patients from controls was only 67.5% and 61.7%. The positive predictive value (PPV) was 61.7% which means among those who had severe RA, the probability of disease was 61.7%. The negative predictive value was 85%. which means that among controls, the probability of being disease-free was 85%.

Discussion

Published research supports the finding of a statistically significant difference in IL-6 levels between patients and controls in the current investigation, [21, 22]. The ratio of IL-17-producing Th17 cells to regulatory T cells is heavily influenced by IL-6 (T reg). Both Th17 cells and T reg cells serve pivotal roles in the immune system, with the former playing a role in the development of autoimmune disorders and the latter serving to rein in overactive effector T-cell responses. It is generally agreed that Th17 cells are primarily responsible for RA pathogenesis. Possible therapeutic value in autoimmune and inflammatory illnesses may come from IL-6 pivotal function in shifting the balance between T reg and Th17 cells, Nishimoto et al. We found no significant association between IL-6 and disease severity which disagrees with previous studies and may be attributed to the small sample size in the current study [23, 24].. The highest mean value of IL-6 was found among patients with moderate disease severity. There was no significant association between IL-6 and gender, duration of the disease, disease activity, or DAS28 score. AS for laboratory investigations, there was no significant association between WBC, HB, PLT, CRP, ESR, Urea, Creatinine, SGPT, SGOT, and IL-6 which disagrees with previous studies [25, 26] and may be due to the small sample size in the current study.

Conclusion

With the serum levels of IL-6 being significantly higher in RA patients than in healthy controls, an optimum cut-off value for IL-6 of 72.90 was used to differentiate between RA patients and controls. However, the sensitivity and specificity of IL-6 in identifying RA patients were moderate. IL-6 showed no correlation with demographic, clinical or laboratory parameters.

Authors' declaration

Conflicts of Interest: None.-

We hereby confirm that all the Figures and Tables in the manuscript are ours. Besides, the Figures and images, which are not ours, have been given permission for re-publication attached with the manuscript.-Authors sign on ethical consideration's approval-

Ethical Clearance: The project was approved by the local ethical committee in College of Medicine,

University of Baghdad according to the code number (125.3.2.2021).

Authors` contribution:

Abeer M. Mohammed contribute in Concept and design of study,

Sarmad M. Zayni : Drafting the article or revising it critically for important intellectual content;

Muhammad M. AL-Anni& Faiq I. Corial: contribute in acquisition of data or analysis and interpretation of data

Adnan Al- Rubaee :Final approval of the version to be published

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القيم التشخيصية والتنبؤية لIL-6
في عينات المرضى العراقيين المصابين بالتهاب المفاصل الروماتويدي

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الخلاصة:
الخلفية: التهاب المفاصل الرَثَيَاني أو الداء الرثياني أو الالتهاب المفصلي الروماتويدي هو مرض مزمن، من الأمراض الانضدادية التي تؤدي بالجهاز المناعي لمهاجمة المفاصل، مسببة التهابات وتدميرًا لها. ومن الممكن أيضًا أن يدمر جهاز المناعة أعضاء أخرى في الجسم مثل الرئتين والجلد. وفي بعض الحالات، يسبب المرض الإعاقة، مؤدية إلى فقدان القدرة على الحركة والإنتاجية. ويتم تشخيص المرض بواسطة تحاليل دم مخبرية مثل تحليل العامل الرثياني والأشعة المقطعية.
الهدف من الدراسة:لتقييم مستوى IL6 في مصل مرضى التهاب المفاصل الرثوي مقارنة بالاصحاء,لتقييم فائدتهما التشخيصية والتنبؤية في المرضى وللتحقيق في ارتباط مستوياتهما بالخصائص الاجتماعية والديموغرافية والسريرية لمرضى التهاب المفاصل الرثوي.
المرضى والطريقة: ثمانون شخصا, 40 شخصا مصاب بمرض التهاب المفاصل الرثوي تتراوح اعمارهم (23-60)و40 شخص سليم تتراوح اعمارهم (23-61). تم استخدام مقايسة الممتز المناعي المرتبط بالانزيم(ELISA) لتحليل مستويات IL6 في الدم.
النتائج:كانت مستويات المصل من IL6 مرتفة في مرضى التهاب المفاصل الرثوي مقارنة بالاصحاء .وكانت هناك قدرة تشخيصية جيدة للتنبؤ بمرض التهاب المفاصل الرثوي.تلك المستويات لم تكن مرتبطة مستوى نشاط المرض ومعدل الترسيب .
الاستنتاجات:IL6 يلعب دورا مهما في التسبب بمرض التهاب المفاصل الرثوي وقد اثبت انه عامل بيولوجي لتشخيص مرض التهاب المفاصل الرثوي.
مفتاح الكلمات: التهاب المفاصل الرثوي, IL6.