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Investigate the levels of soluble vascular cell adhesion molecule 1 (sVCAM-1) and intercellular adhesion molecule 1 (sICAM-1) in the sera of patients with rheumatoid arthritis

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ABSTRACT

Rheumatoid arthritis (RA) is an autoimmune disorder, the synovial joint becomes inflamed and eventually erosive, causing damage to the bones and cartilage. The study aimed to evaluated vascular cell adhesion molecule1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1) serum levels in RA patients. These cellular adhesion molecules (CAMs') serum levels may indicate endothelial cell expression and a possible relationship with other variables. Eighty-eight volunteers, 50 RA patients and 38 healthy controls, ranging in age from 25 to 75, were assessed. The levels of biomarkers were examined in this study: CAM-1, VCAM-1, Rheumatoid Factor (RF), Anti-CCP, C-reactive protein (CRP), ESR, and erythrocyte sedimentation rate (ESR). The findings indicate that there is a significant variation ($p \leq 0.001$) in the levels of RF, Anti-CCP, CRP, and ESR between individuals with RA patients and the control group. Moreover, it was observed that there exist there exist substantial disparities ($p \leq 0.001$) in the concentrations of VCAM-1 between the subjects diagnosed with rheumatoid arthritis (RA) and the control cohort. Furthermore, it was observed that the concentration of ICAM-1 in patients with RA was significantly elevated ($p \leq 0.001$) when juxtaposed with the control cohort. Conversely, the results suggested a correlation that lacked statistical significance between the concentrations of ICAM-1 and VCAM-1 and the serological parameters. In the present investigation, we concluded that elevated levels of VCAM-1 and ICAM-1 in individuals with RA may serve as potential biomarkers for the diagnosis and prognostication of RA, as well as a contributory factor in the pathogenesis of RA as an inflammatory disorder.

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1. INTRODUCTION

Rheumatoid arthritis (RA) is an autoimmune disorder, the synovial joint becomes inflamed and eventually erosive, resulting in bone and cartilage destruction, as well as systemic consequences including heart disease, lung disease, psychological distress, and various skeletal disorders [1,2]. RA can be caused by a combination of genetic and non-genetic factors, such as hormone conditions, environmental factors, and viral infections [3,4]. RA affects anything between 0.5% and 1% of the global population, with a higher incidence in females compared to males [5]. The probability of females experiencing the disease is estimated to be two to three times greater than that of males. It is notably prevalent during the sixth decade of life [6]. Collectively, the pathophysiology of rheumatoid arthritis (RA) is defined by the existence of autoimmune and inflammatory-related mechanisms, which ultimately lead to the deterioration of the bone and cartilage situated within the joints. As a result of this, a considerable amount of time and effort in the field of scientific research has been invested in the study of this disease in order to identify unique biomarkers of the condition. The identification of these biomarkers has been pursued with the intention of achieving both the diagnosis and the prediction of the prognosis. [7].

Specifically, cellular adhesion molecules [CAMs) are glycoproteins located on the cell surface that serve as a molecular correlation between the external and internal regions of the cell, in a process known as cell adhesion [8]. CAMs have been identified in a diverse range of cell types, including endothelium, lymphocytes, macrophages, and several tumor cells lines. [9]. These molecules may function as indicators of endothelial activity and inflammation at the local or systemic level, although the specific pathogenic function of these CAMs compounds in disease states is still unknown [10]. On the other hand, the process of inflammation serves as a protective mechanism that effectively controls infection and promotes the healing of tissue. Specific pro-inflammatory triggers can stimulate the upregulation of CAMs on the outer layer of vascular endothelial cells. These CAMs consist of E-selectin, intracellular adhesion molecule-1 (ICAM-1), and vascular cell adhesion molecule-1 (VCAM-1) [11]. The expression levels of ICAM-1 and VCAM-1 are typically minimal in endothelial cells; however, in the presence of inflammation, whether localized or systemic, there is a notable upregulation of these two cell adhesion molecules (CAMs). This phenomenon is often associated with an elevation in the concentrations of interleukin-1 beta (IL-1b) and tumor necrosis factor (TNF). Consequently, this upregulation facilitates the adhesion and migration of leukocytes to the endothelial surface, which is subsequently followed by their translocation across the endothelial barrier to which they have adhered [12]. Investigations have demonstrated that the existence of soluble cell adhesion molecules within the endothelium, including ICAM-1 and VCAM-1, serves as a reliable indicator of an inflammatory response occurring within this vascular layer [13]. Given that these biomarkers possess the potential to facilitate the early prognostication of endothelial dysfunction and activation, the prompt detection of elevated levels of endothelial biomarkers should significantly enhance the development of innovative therapeutics aimed at mitigating the complications associated with rheumatoid arthritis [14]. Thus, the aim of article is to assess the serum concentrations of VCAM-1 and ICAM-1 in patients diagnosed with rheumatoid arthritis (RA).

2. METHOD

Blood samples collection

The study included a sample of 50 subjects, comprising 45 females and 5 males, who engaged in the study between November 2023 and March 2024. The participants were in pursuit of medical care at the Rheumatology Unit of Baghdad Teaching Hospital and Baquba General Hospital, and control group consisted of 38 individuals, of which 28 were female and 10 were male, with ages ranging from 25 to 75 years. All of the cases had a confirmed diagnosis of RA, and every patient met the criteria for RA categorization that were established by the American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR) in 2010 [15].

A total of 5 millilitres of blood was obtained using a disposable syringe. Three milliliteres of blood were taken and placed in a gel clot activator tube at room temperature until the coagulant started forming. Following that, the samples were centrifuged at a speed of 3000 rpm per minute for 15 minutes to collect serum for the tests of rheumatoid factor (RF) kit (DIRUI / China), antibodies to cyclic citrullinated peptides (Anti-CCP) kit (Snibe / China), and C-reactive protein (CRP) kit (Dorset / UK), as well as 2 ml EDTA tubes for the erythrocyte sedimentation rates (ESR) test, kit (MONOSED/ USA).

Measurement of cell adhesion molecules levels

Using enzyme-linked immunosorbent assay (ELISA) sandwich kits (Elabscience, USA), measurements of cell adhesion molecules (ICAM-1 and VCAM-1) in the blood samples were carried out in line with the methods provided by the manufacturer protocols. VCAM-1 Cat. No. E-EL-H5587 and ICAM-1 Cat. No. E-OSEL-H0018.

Patients Consent

Prior to data collection, signed consent from each of the participants was obtained after explaining the purpose of the study and ensuring the privacy of the data.

Statistical Analysis

Through using of the SPSS Statistical software (Version 27, SPSS, IBM), statistical analysis was carried out. Mean ± standard deviation was used to express the data. We employed Pearson's correlation coefficient and Student's t-test for our statistical study. Significant differences were considered statistically significant when the p-value was less than 0.05.

3. RESULTS AND DISCUSSION

Recurrent synovitis is a hallmark of RA, A commonly occurring persistent inflammatory tissue lesion affecting the synovial tissue of the joints that can lead to articular dysfunction and damage to intra-articular cartilage and bone mass [16]. Table 1 displays the outcomes of the laboratory parameters that were measured during this investigation for both the patients and the control group separately, of all the people diagnosed with RA, approximately 36 (72%) are positive for RF, 14 (28%) are negative for RF, and 38 (100%) healthy control individuals are 100% negative for RF, with statistically significant differences between the RA patients and the control group ($p \leq 0.001$). About 32 (64%) of the patients were positive for anti-CCP, while 18 (36%) of the patients were negative for anti-CCP. Additionally, 38 (100%) healthy controls were negative for anti-CCP, with a statistically significant difference between the RA patients and the control group ($p \leq 0.001$).

Also, the mean level of CRP and ESR were highly significantly (p≤0.001) increased when compared patient group (18.96±11.13; 33.42±15.08) with the control group (9.16±3.03; 17.68±8.54) respectively. Recently, X-ray analysis, laboratory data, and clinical symptoms are used to diagnose RA. The serological markers are an essential component in the process of diagnosing and determining the prognosis of patients who suffer from RA. (RF) has been applied for the diagnosis of RA for several years, and it is included in the ACR diagnostic criteria. In addition, high positive findings were obtained for the ESR and CRP tests. Although these tests do not provide a definitive diagnosis of RA, they play an essential role in the diagnostic process of RA. The Anti-CCP test is said to possess outstanding performance characteristics and it's as a component of the criteria established by the ACR [17]. In the current study, patients who demonstrated positive RF and Anti-CCP test results were more prevalent than those with negative test results. Recently, new diagnostic criteria for RA have been established, augmenting the existing criteria of Anti-CCP and RF. Additionally, we noted a high correlation between anti-CCP antibodies and a variety of joint pains, including those affecting multiple joints, the hands, and both sides of the body, as well as an increase in CRP and RF levels. [18]. Consequently, these markers can serve as precise serologic indicators for RA. The results of our study are consistent with a recent study, which documented that none of these biomarkers (ESR, CRP, RF, anti- CCP) could accurately monitor RA activity. The anti- CCP antibody test, which was developed as a novel serum marker for RA, was able to aid in the early diagnosis of RA patients and change decisions regarding their treatment. When combined with the RF test, the anti- CCP test provides a more accurate early diagnosis of RA and has been found to have important diagnostic value for this disease [19]. Furthermore, the present study demonstrated that the levels of C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were significantly increased in patients compared to the control group. The findings of our study aligned with prior research that validated elevated levels of ESR and CRP in patients as compared to the control group [17, 20]. Given the function of CRP in the inflammatory response and the host's defence mechanism against infections, there are several reasons why CRP production in the liver is stimulated by various proinflammatory cytokines derived either from monocytes or macrophages. The inflammatory response leads to, an increase in the production of IL-1 and TNF-α, which in turn leads to the release of the messenger cytokine, IL-6, which in turn stimulates the liver to secrete CRP [21,22]. In contrast, ESR is usually elevated in RA. However, ESR testing does not provide a definitive diagnosis of RA because other conditions can cause inflammation. It may also be repeated after initial evaluation and diagnosis to assess and monitor systemic inflammation [23]. However, ESR and CRP are considered suitable biochemical markers for long-term monitoring of RA disease activity [24].

Table 1: Serological parameters results for RF, anti-CCP, CRP, ESR in the study groups.

Parameter	Result	Patient N=50	Control N=38	P-Value
RF	Positive	36 (72%)	0	≤0.001
	Negative	14 (28%)	38 (100%)	
Anti-CCP	Positive	32 (64%)	0	≤0.001
	Negative	18 (36%)	38 (100%)	
CRP mg/L (mean ± SD)		18.96±11.13	9.16±3.03	≤0.001
ESR, mm/hr. (mean ± SD)		33.42±15.08	17.68±8.54	≤0.001

On the other hand, the study's most noteworthy result was that RA patients had elevated serum levels of VCAM-1 and ICAM-1 compared to the control group. The mean serum VCAM-1 level of the patients' group was determined to be 127.18±26.15 ng/ml, which was shown to be significantly higher (p≤0.001) than the mean serum level of the control group, which was 97.45±11.96 ng/ml. The estimated level of mean serum ICAM-1 for patients with RA was found to be significantly different from that of the control group, as indicated by the difference in significance (p ≤ 0.001). According to Table 2, the patients with RA had an estimated mean serum ICAM-1 level of 51.86±4.91ng/ml, whereas the control group had a level of 37.88±9.35 ng/ml. The results of our study are consistent with the results of a recent study that showed an increased level of VCAM-1 in the serum of patients with RA compared to the control group [25]. The results of our study were similar to those of Sodergren et al. (2019) who showed in their study that serum VCAM-1 levels were shown to be considerably greater in patients diagnosed with RA, when compared to the control group [26]. The elevation in VCAM-1 levels in autoimmune disorders is attributed to the existence of autoantibodies, heightened synthesis of proinflammatory cytokines, and oxidative stress. Vascular cell adhesion molecules-1 exert an impact on the inflammatory process, potentially associated with the onset and advancement of RA [27].

Another recent study reported that this increase in VCAM-1 levels is due to the activation and upregulation of CAMs, including integrins and VCAM-1, enhance the adherence to the extracellular matrix (ECM) in the inflamed synovium in RA, therefore promoting joint injury and destruction [28]. This provides evidence that the adhesion of T cells to synovial cells or endothelium through VCAM-1 and its ligand VLA-4, a major integrin for T cells, can contribute to the activation of effector cells (both T cells and synovial cells) and the release of more cytokines and destructive enzymes from them. Which causes inflammation and formation of synovial RA [29, 30]. On the other hand, with respect to ICAM-1. The findings of our research align with those of a previous study which confirmed a significant increase in ICAM-1 levels in the bloodstream of individuals diagnosed with rheumatoid arthritis when compared to the control group. Moreover, it was demonstrated that individuals with a positive Anti-CCP status for the condition exhibited heightened concentrations of adhesion molecules (31). A recent investigation has revealed that endothelial chemokines and cytokines elicit the stimulation of ICAM-1 and VCAM-1, which subsequently play a significant role in various pathways associated with inflammatory development, particularly the localized infiltration of inflammatory cells. The interaction between leukocyte integrins and ICAM-1 as well as VCAM-1 facilitates the rolling motion of leukocytes during their migration across the endothelium, thereby instigating a cascade of inflammatory reactions within the synovial membrane infiltrating RA [32]. Nonetheless, recent investigations have indicated that the expression levels of CAMs on the endothelial cell membrane constitute a pivotal determinant in assessing the relative impact of VCAM-1 and ICAM-1 on leukocyte recruitment under specific pathological circumstances. ICAM-1 assumes an indispensable role in processes associated with inflammation and T-cell-mediated immunity. Conversely, VCAM-1 represents an additional molecule that facilitates adhesion to inflammatory cells and possesses potential therapeutic implications in the realms of immunological disorders and malignancies. Under physiological conditions, both ICAM-1 and VCAM-1 exhibit markedly low levels within the organism; however, their concentrations may experience substantial elevation during states of dysfunction. The augmented expression of ICAM-1 and VCAM-1 results in enhanced leukocyte adhesion, a phenomenon attributed to the inherent inflammatory response and damage to endothelial cells. Consequently, these molecules may function as a dependable biomarker for the inflammatory processes occurring within the endothelium [33,34]. This gives implications and support to the results of our study in terms of considering these adhesion molecules as inflammatory markers affecting the development of RA.

Table 2: The mean levels of VCAM-1 and ICAM-1 in the blood among the research groups.

Cell Adhesion Molecules	Patients	Control	P value
Serum VCAM-1 (ng/ml)	127.18±26.15	97.45±11.96	≤0.001
Serum ICAM-1 (ng/ml)	51.86±4.91	37.88±9.35	≤ 0.001

Regarding correlations, the analysis of Pearson's correlation was utilized to investigate the relationships that existed between the levels of serum VCAM-1 and ICAM-1 and the serological parameters.no circulating VCAM-1 and ICAM-1 levels correlated with clinically apparent serological parameters in RA patients. Negative correlations were observed between serum VCAM-1 and serological parameters such as anti-CCP, CRP, ESR, and ICAM-1. The statistical significance of these correlations was not observed ($p \geq 0.05$). On the other hand, it was shown that between serum ICAM-1 and both RF and CRP, there was a negative correlation that was not statistically significant ($p \geq 0.05$) as shown in table 3. Differences from previously reported findings [25,35] in patients with RA may suggest that distinct pathogenic pathways play a role in various diseases. The possible elucidation is that VCAM-1 and ICAM-1 represent molecules that are induced through the activation of cytokines, particularly TNF-α and IL-1β, which are two prevalent cytokines present in the inflamed synovium of RA [35]. Furthermore, various inflammatory diseases exhibit unique patterns of alterations in circulating adhesion molecules and demonstrate a certain degree of specificity, even among related symptoms such as the different types of vasculitis. Studying fluctuations of adhesion molecules along disease outcome may improve assessment of subclinical disease activity, the effects of treatment on specific pathophysiological mechanisms, as well as the involvement of different cell types in the pathogenesis of the inflammatory response across various disorders [36]. It is possible that the subsequent factors may have influenced the findings drawn from our research. To adequately assess the results of this study, it is imperative to consider elements such as the potential influence of age and, most probably, gender as well. Additionally, there exists the possibility that the minor variations in the levels of the adhesion molecules that were examined could serve as an indication of the effects that pain and stress exert on the immune system. Moreover, one may postulate regarding whether the immunological response to the adhesion molecules that were investigated differs between males and females.

Table 3: Correlation between VCAM-1 and ICAM-1 and other serological parameters in patients with RA.

Parameters	VCAM-1	ICAM-1
RF	r= 0. 051 P= 0. 727 NS	r=-0.003 P=0.985 NS
Anti-CCP	r=-0.087 P=0.549 NS	r=0.038 P=0.795 NS
CRP	r=-0.226 P=0.114 NS	r=-0.019 P=0.893 NS
ESR	r=-0.159 P=0.271 NS	r=0.084 P=0.564 NS
ICAM-1	r= -0.148 P=0.306 NS	r=1

r= correlation coefficient, NS= not significant

4. CONCLUSION

The current study's findings show that the levels of RF, Anti-CCP, CRP, and ESR in the blood of RA patients were found to be considerably greater than those found in the control group. In contrast, the levels of ICAM-1 and VCAM-1 that were found in the blood of RA patients were shown to be considerably greater when compared to the levels found in the serum of controls. Additionally, we found that there was no significant correlation between VCAM-1 and ICAM-1 and the serological parameters. In summary, we arrived at the conclusion that elevated concentrations of VCAM-1 and ICAM-1 in individuals afflicted with RA may serve as a potential biomarker for the diagnosis and prognostication of RA, in addition to being a contributory factor in the pathogenesis of RA as an inflammatory condition.

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Ethical approval

The local ethics committee of the University of Diyala and Council College of Education for Pure Sciences authorized the study.

Declaration of conflicting interest

Authors claim no conflict of interest.

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