



Evaluation of Some Biochemical, Immunological, and Inflammatory Markers in Patients with Rheumatoid Arthritis

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Abstract

Study Background: Rheumatoid arthritis (RA), a chronic autoimmune inflammatory disease that mainly involves the synovial membrane of joints. The disease gradually destroys the cartilage and surrounding bone, causes loss of mobility and motor function, and has systemic extra-articular manifestations. A panel of serological, biochemical and immunological indicators that mirror the inflammation and activity of the disease is critical for an early diagnosis and an appropriate therapeutic follow-up.

Objective The current study was designed to determine the severity of certain inflammatory markers (CRP, ESR, IL-6, TNF- α), well-defined immunological markers for rheumatoid arthritis (RF, Anti-CCP), humoral and complement immune markers (IgG, C3, C4), and liver function and serum protein markers (TP and ALB) in patients with rheumatoid arthritis compared to the group of healthy individuals.

Materials and Methods: The present case-control study was conducted on 50 confirmed patients of rheumatoid arthritis as per the international classification criteria (ACR/EULAR 2010) along with another 50 presumably healthy individuals matched in age and sex as controls. Venous blood samples were obtained, and sera separated to determine the aforementioned markers by enzyme-linked immunosorbent assay (ELISA), immunoturbidimetric assay, and standard biochemical methods. Statistical Analysis The independent samples t-test was used for the statistical analyses, assuming differences were significant at ($p < 0.05$).

Results: The results showed that all inflammatory and immune indicators were significantly elevated in the patient group compared with the healthy individuals; the mean CRP of (18.7 ± 7.4) vs (3.84 ± 1.52) mg/L, the mean ESR of (42.60 ± 15.83) vs (12.40 ± 4.76) mm/hour, the mean IL-6 of (18.35 ± 6.72) vs (4.82 ± 1.63) picogram/mL, and the mean TNF- α of (15.64 ± 5.38) vs (5.17 ± 1.84) picogram/mL. RF was also increased significantly (86.40 ± 38.25) vs (9.72 ± 4.31) IU/mL; $p < 0.01$) and Anti-CCP (112.60 ± 47.84) vs (8.35 ± 3.72) U/mL $p < 0.01$). There was also a marked elevation of the patients' IgG, C3, C4, and total protein as well as a marked decrease of albumin level (3.61 ± 0.42) g/dL when compared to healthy controls (4.31 ± 0.35) g/dL, indicating chronic inflammation and negative acute phase response.

Conclusion: The results suggest that the biochemical, immunological and inflammatory indicators taken together reflected the pathological status of rheumatoid arthritis as an integrate phenomenon. They authenticate that synergism of acute phase indicators, inflammatory cytokines, targeted auto-antibodies, and serum protein indicators can provide a broader scope laboratory evaluation than each singular indicator for augment use in early diagnosis, and while monitoring the therapeutic response.

Keywords: Rheumatoid arthritis; C-reactive protein; Interleukin-6; Tumor necrosis factor-alpha; Rheumatoid factor; Anti-cyclic citrullinated peptide antibodies; Complement C3 and C4; Serum albumin

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1. Introduction

Rheumatoid arthritis (RA) is one of the most prevalent and devastating life-affecting autoimmune rheumatic diseases in the world. It affects 0.5–1% of the global population [1,6], with a marked geographical variation in prevalence. RA is more common in females than males, at a ratio of 2–3:1 [2]. The incidence rates also rise progressively with age, and idea mostly during the sixth and seventh decades of life [2]. This disease is a chronic progressive disease that includes both systemic inflammation and progressively destructive gradual symmetrical small joint, large joint polyarthopathy, which is one of the leading causes of acquired disability worldwide [1].

The pathogenesis of rheumatoid arthritis (RA) involves multiple genetic, environmental and immunologic factors; class II tissue specific antigens are major contributors to genetic risk of disease, particularly HLA-DR4 and HLA-DR1, while smoking remains one of the most prominent environmental risk factors precipitating anti-citrullinated protein antibodies [3,5]. The first step of the pathogenic pathway is the loss of self-tolerance against these modified proteins, causing the appearance of specific antibodies—including Rheumatoid Factor (RF) and Anti-Cyclic Citrullinated Peptide (Anti-CCP)—that may antedate the clinical manifestations of the disease by years [6,7]. This aberrant immune response is then followed by the infiltration of the synovial membrane with T and B lymphocytes and macrophages, and the local overproduction of a complicated, and typically circular, network of pro-inflammatory cytokines (now and again, referred to as cytokine storm), led by Tumor Necrosis Factor-alpha (TNF- α) and consequently the notably, and notable, important, Interleukin-6 (IL-6) -that is uniformly implicated as a key mediator in the development of synovial membrane procreation and therefore the imaging of a separatist tissue, namely the destructive pannus that breakfast, erodes cartilage and subchondral bone [3,4]. The diagnosis of rheumatoid arthritis and monitoring of its therapeutic activity depends largely on biochemical and immunological serological markers. Non-specific indicators of systemic inflammation severity, such as C-Reactive Protein (CRP) and Erythrocyte Sedimentation Rate (ESR), are utilized as acute phase indicators, whereas inflammatory cytokines such as IL-6 and TNF- α provide information that is more directly related to the underlying pathogenic mechanism [7,8,9]. Regarding the specific autoantibodies, particularly RF and Anti-CCP, both exhibit high diagnostic and predictive utility, with the sensitivity and specificity of Anti-CCP being superior to the classical rheumatoid factor in many recent studies [7,13,15]. Of note, they also predict the detrimental behavior of the disease before detection by radiological markers. The activation of the immune system and the formation of immune complexes are reflected in cellular immune indicators such as IgG and complement components (C3 and C4) [16,17], whereas serum protein indicators, especially the reduction in albumin as one of the negative acute phase proteins, and the increase in total protein reflect the chronic inflammation related to the disease and its adverse effects on liver function and general metabolism [19,22].

In view of the complicated pathology of rheumatoid arthritis it may be inappropriate to rely on a single laboratory indicator for accurate characterization of the pathological condition or for differentiating it from other inflammatory autoimmune conditions with similar clinical manifestations [7,24]. Thus, the aim of the current work was to evaluate a comprehensive set of biochemical, immunological and inflammatory markers (acute phase reactants, inflammatory cytokines, specific autoantibodies, humoral and complement immunity markers, serum protein and liver function indicators) between a cohort of patients with rheumatoid sarthritis and matched healthy controls, in order to provide a comprehensive laboratory picture contributing to the early diagnosis and further understanding the immune and inflammatory changes induced by this condition.

Objectives of the Current Study The current study aims to: 1. Estimating serum levels of inflammatory markers in patients with rheumatoid arthritis in comparison with that of healthy controls (CRP, ESR, IL-6, TNF- α). 2. Particular immunological markers of the disease RF and Anti-CCP and their diagnostic value. Assessment of the particular immunological indicators of the disease (RF and Anti-CCP) and their diagnostic function. 3. Determination of humoral and complement immunity indicators (IgG, C3, C4) in the sera of studied groups. 4. Liver Function Indicators and Serum Proteins (Total Protein and Albumin) Related to Disease Study 5. Purpose To model and examine the role of these aggregated prognostic indicators in conveying the status of RA-associative chronic inflammation.

2. Materials and Methods

2.1. Study Design and Location

The present study is of case-control study design as it was designed to assess a panel of biochemical, immunological and inflammatory markers in the serum of rheumatoid arthritis subjects as compared to a control group of apparently healthy individuals.

2.2. Study sample

The study sample consisted of two groups, group(1):50 patient (clinically and laboratory diagnosed with rheumatoid arthritis) and group (2):50 healthy individuals which are clinicians and laboratory diagnosed healthy individuals as a control and the two groups are matched in age and gender in order to eliminate the effect of confounding variables in a more accurate way.

2.3. Exclusion criteria

Patients with concomitant autoimmune inflammatory diseases (such as systemic lupus erythematosus or psoriasis arthritis), chronic liver or kidney disease, pregnant, recently used immunosuppressive drugs or antibiotics before blood collection and any condition that may interfere with the results of these indicators were also excluded from the study.

2.4. Sample collection

After 8–10 hours of fasting, blood samples were taken from peripheral venous blood for all the participants and were divided into sterile tubes without anticoagulant to obtain serum after centrifugation at (3000) RPM for (10) minutes. The samples were kept at (-20°C) until the analyses were performed.

2.5. Laboratory measurements

C-reactive protein (CRP) was measured using the immunoturbidimetric assay, and the erythrocyte sedimentation rate (ESR) was measured using the Westergren method. At the same time, concentrations of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), rheumatoid factor (RF) and anti-cyclic citrullinated peptide (Anti-CCP) antibodies were measured using the enzyme-linked immunosorbent assay (ELISA, supplier's protocol). Serum immunoglobulin G (IgG) and complement components C3 and C4 were measured using the immunoturbidimetric method, total protein was measured using the Biuret method and albumin using the Bromocresol Green method.

2.6. Statistical Analysis

The results were presented as mean $\pm$ standard deviation (Mean $\pm$ SD) and independent samples t-test was used to compare the two groups of patients and healthy individuals for each of the studied indicators. Statistical significance was accepted at a probability level of ($p < 0.05$). Data was analysed through the statistical software (SPSS) and was also plotted through illustrative graphs by using the Python Programming language (Matplotlib library).

3. Results

The current study revealed a significant variation ($p < 0.05$) between LART patient group and healthy reference group regarding every one of the assessed biochemical, immunological and inflammatory parameters illustrated in the tables and graphs to follow.

Table 1: Inflammatory markers in the study groups

Inflammatory Biomarker	RA Patients (n = 50), Mean $\pm$ SD	Healthy Controls (n = 50), Mean $\pm$ SD	P-value
C-reactive protein (CRP) mg/L	18.72 $\pm$ 7.46	3.84 $\pm$ 1.52	<0.001
Erythrocyte sedimentation rate (ESR) mm/h	42.60 $\pm$ 15.83	12.40 $\pm$ 4.76	<0.001
(IL-6) pg/mL	18.35 $\pm$ 6.72	4.82 $\pm$ 1.63	<0.001
Tumor necrosis factor-alpha (TNF- α) pg/mL	15.64 $\pm$ 5.38	5.17 $\pm$ 1.84	<0.001

The clinical data presented in table (1) and figure (1) showed that the averages of CRP, ESR, IL-6, and TNF- α were significantly higher in the patient group when compared to the healthy group. Patients had an average CRP approximately five-times that of healthy individuals (18.72 vs. 3.84 mg/L), and an average ESR three times that of healthy individuals (42.60 vs. 12.40 mm/hour). Correspondingly, serum levels of IL-6 and TNF- α significantly elevated in the patients, indicating the status of active systemic inflammation induced by the disease.

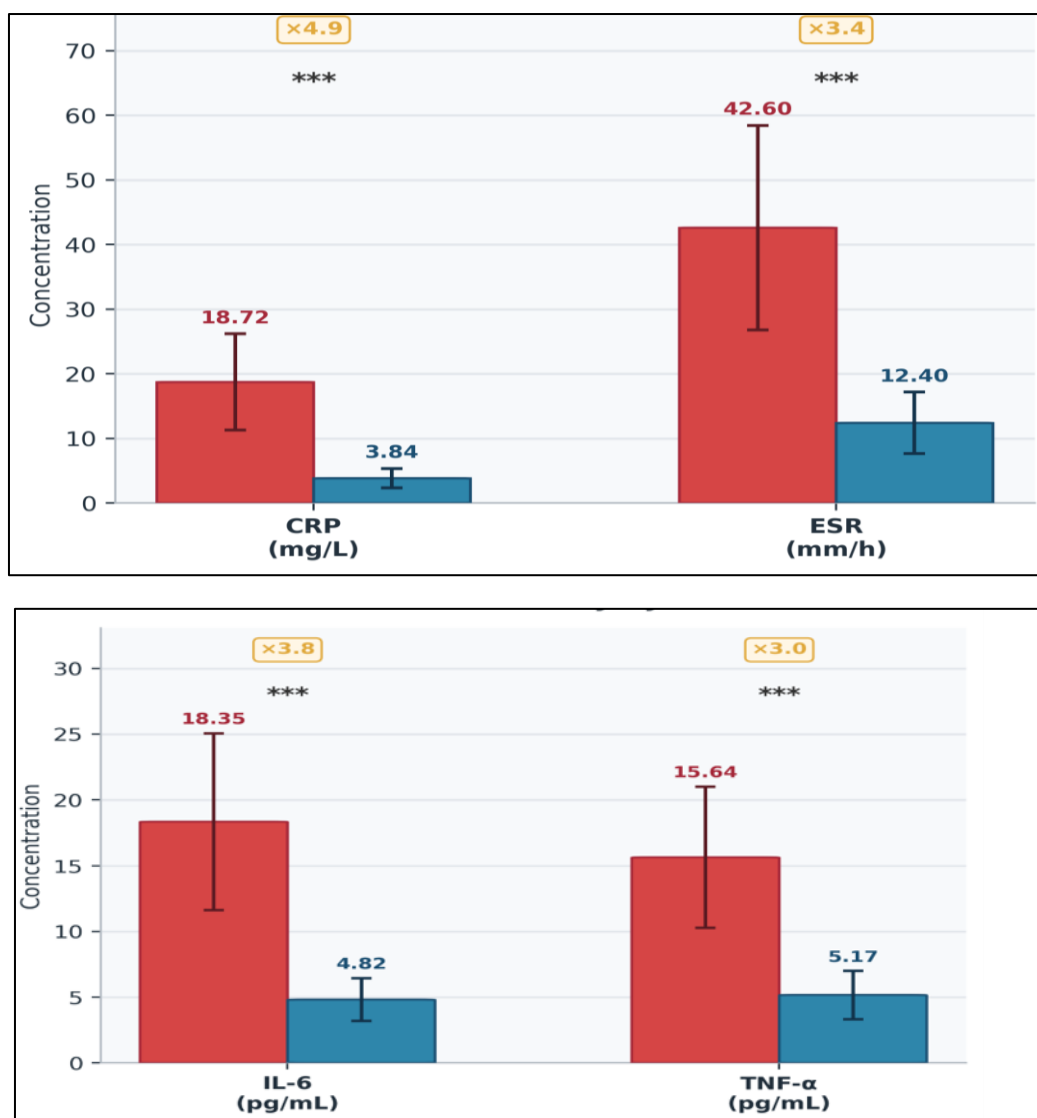


Figure 1: Comparison of inflammatory markers (CRP, ESR, IL-6, TNF-α) between rheumatoid arthritis patients and healthy individuals

Table 2: Specific Immunological Indicators for Rheumatoid Arthritis

Immunological Marker	RA Group (n = 50), Mean ± SD	Healthy Group (n = 50), Mean ± SD	P-value
Rheumatoid factor (RF) IU/mL	86.40 ± 38.25	9.72 ± 4.31	<0.001
Anti-cyclic citrullinated peptide antibodies (Anti-CCP) U/mL	112.60 ± 47.84	8.35 ± 3.72	<0.001

The data have been summarized in Table (2) and simple impact can be seen in a distinct porcupine figure (figure 2), the levels of, RF in patients increased sharply (86.40 ± 38.25 IU/ml) in comparison to healthy individuals (9.72 ± 4.31 IU/ml) an increase by almost nine-fold. Similarly, Anti-CCP was (112.60 ± 47.84 IU/ml) in patients vs. (8.35 ± 3.72 IU/ml) in healthy; almost thirteen-fold higher and the high specific marker for pathology.

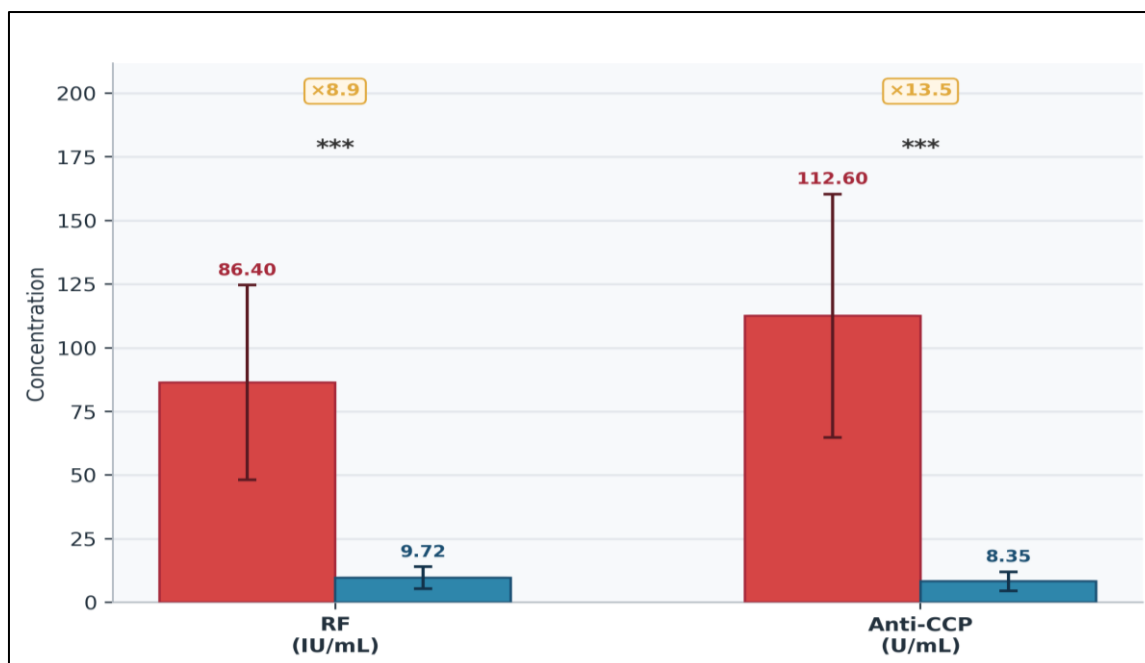


Figure 2: Comparison of rheumatoid factor (RF) and anti-cyclic citrullinated peptide (Anti-CCP) antibody levels between the two groups

Table 3: Indicators of humoral and complement immunity

Immune Parameter	RA Group Mean $\pm$ SD	Healthy Controls Mean $\pm$ SD	P-value
Immunoglobulin G (IgG), g/L	16.84 $\pm$ 3.72	11.92 $\pm$ 2.46	<0.001
Complement component C3, g/L	1.48 $\pm$ 0.31	1.21 $\pm$ 0.22	<0.001
Complement component C4, g/L	0.34 $\pm$ 0.09	0.27 $\pm$ 0.07	<0.001

As is shown in Table (3) and Figure (3), there were significant ($P < 0.05$) higher immunoglobulin IgG levels in the serum of patients (16.84 ± 3.72 g/L) compared with healthy control subjects (11.92 ± 2.46 g/L), reflecting the hyper activity of the humoral immune response of the disease. Complement components C3 and C4 showed a large increase in patients versus healthy individuals, which is expected for these positive acute-phase proteins elevated in response to systemic inflammation in rheumatoid arthritis. This is in contrast to findings in other autoimmune diseases characterized by immune complex deposition, where these components are decreased, such as in systemic lupus erythematosus.

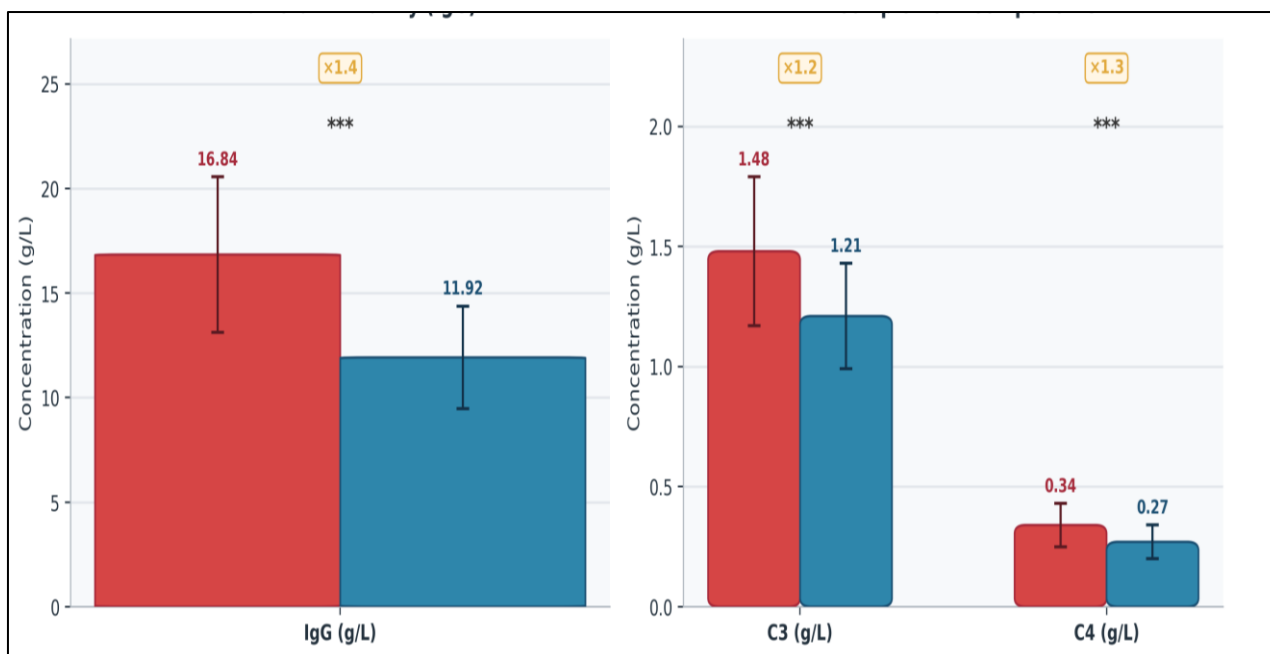


Figure 3: Comparison of IgG, C3, and C4 levels between the study groups (C3 and C4 values multiplied by 10 for illustrative purposes).

Table 4: Indicators of Liver Function and Serum Proteins

Biochemical Parameter	RA Group Mean $\pm$ SD	Healthy Controls Mean $\pm$ SD	P-value
Total serum proteing/dL	7.82 $\pm$ 0.64	7.21 $\pm$ 0.48	0.003
Serum albuming/dL	3.61 $\pm$ 0.42	4.31 $\pm$ 0.35	<0.001

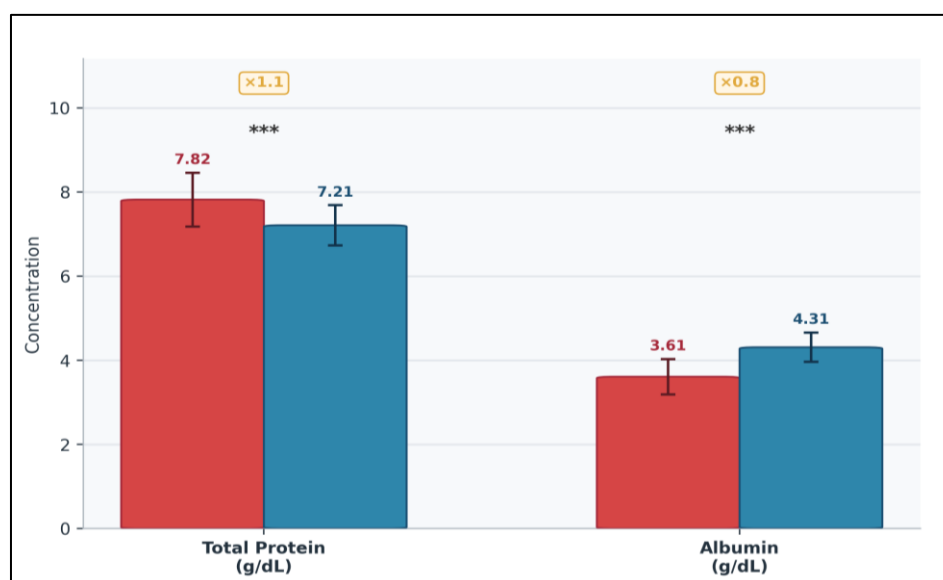


Figure 4. Serum levels of Total Protein and Albumin in patients compared to healthy controls. Bars represent mean values ($\pm$ SD), showing significantly higher Total Protein and lower Albumin in the patient group ($P < 0.001$).**

Table (4) and Figure (4) displayed a highly significant increase in the mean TP level in patients (7.82 ± 0.64 g/dL) versus healthy persons (7.21 ± 0.48 g/dL). This increase in Ig is mainly due to hypergammaglobulinemia related to chronic inflammatory reaction. Conversely, the level of serum albumin significantly decreased in patients (3.61 ± 0.42 g/dL) than healthy individuals (4.31 ± 0.35 g/dL), which can be explained by the known fact that albumin behaves as one of the

negative acute phase proteins, of which concentrations decrease in response to a shift in the liver to a predominantly positive acute phase protein production during chronic inflammation.

4. Discussion

The significance of the increase in markers of inflammation (CRP, ESR, IL-6 and TNF- α) in rheumatoid arthritis patients, as shown in the present study, was in a good agreement with the study of [8], which demonstrated that IL-6 and CRP levels positively correlated with the disease activity score DAS28. Notably, this reinforces their dual function; these markers assess the systemic inflammation severity but also the systemic clinical disease activity as well. The postulates were also in agreement with the study of [9]. Note however that this analysis followed up cohorts with [9], which found that the high that IL-6 levels were only slightly reduced in remission cohorts can't be rapidly translated that IL-6 observed in remission; not only did this increase drive up transmission, but also drive up transmission, but could not be diagnostic marker; IL-6 for disease activity [11], but more importantly remains a marker of disease severity and progression. This is also in agreement with the report by study [10] on the positive correlation between CRP and other inflammatory markers including SAA in rheumatoid arthritis patients. From a pathophysiological perspective, this rise derives from the consideration that IL-6 is one of the key promoters of hepatic acute phase protein production [3], including CRP, and furthermore its ability to induce Th17 helper T cell differentiation that supports the persistence of synovial inflammation.

For TNF- α , the results of the present investigation are consistent with another study [11] which also proved to be a reliable marker for evaluating disease activity in the early stages of rheumatoid arthritis as its increased levels correlate with increase in number of painful and swollen joint and deterioration in the functional disability index. TNF- α : Involves in the pathogenesis of the disease by being the major trigger for activating pannus cells, inducing secretion of matrix metalloproteinases (mostly MMPs) causing cartilage destruction and that is why many modern biological therapies (TNF- α inhibitors) depend on controlling the disease [3,4].

In this regard, with respect to the specific immune indicators and compared to data from 1998, the present work revealed that both RF and Anti-CCP increased significantly, with a clear superiority of the increase percentage in favor of Anti-CCP. This corresponds with the findings of many recent studies which have shown that Anti-CCP has a much greater diagnostic specificity than traditional RF. This has been reaffirmed in comparative studies [12] which showed that, although RF is diagnostic, the diagnostic accuracy of Anti-CCP is unambiguously top-performing, and, moreover, Anti-CCP has greater discriminatory ability than ESR and CRP for early diagnosis of rheumatoid arthritis [13], especially in phases where acute phase indicators have not yet elevated remarkably. A study [15] further confirmed that the specificity of Anti-CCP (94.2%) is higher than that of Anti-MCV (84.4%) and of RF (84.8%) as well. This is because Anti-CCP targets citrullinated proteins produced in the inflamed synovial tissue, in contrast to RF, which can be positive in a number of other diseases so has lower diagnostic specificity but adequate sensitivity [14].

With respect to humoral immunity and complement, the elevation in IgG, C3, and C4 detected by this study could be attributed to the fact that these proteins are positive acute phase proteins. This support the previous works which studied the role of complement in differentiating type of autoimmune disorders [16,18]. High levels of C3 and C4 were noticed in a study investigating rheumatoid arthritis patients while these levels were lower in patients with systemic lupus erythematosus due to complement consumption because of the deposition of immune complexes in the tissues [17]. This adds more value to the measurement of these indicators in the differential diagnosis between the two conditions in terms of clinical symptoms. Elevated IgG also reflects chronic B lymphocyte activation and differentiation into IgG-secreting plasma cells, a hallmark of the humoral-autoimmune nature of disease.

In the present study, the reduction of serum albumin in the patient group coincided with results of several recent studies reporting a marked correlation between albumin and the disease activity index DAS28-CRP [19]. Excessive decrease in albumin, it has been demonstrated that as activity increases of disease, this distinction is more evident [19,23]. The study [22] based on NHANES data also brought further confirmation, suggesting a potential causal role of low albumin in disease progression. Such decrease is due to the diversion of the hepatic synthetic machinery to the synthesis of positive acute phase proteins at the expense of albumin during chronic inflammatory state in addition to an anticoagulative action from IL-6 per se that inhibits the gene activity of hepatic cells transcription that regulates the albumin. Therefore, these recent studies proposed to use the composite ratios such as CRP to albumin ratio (CAR) [21] and fibrinogen to albumin ratio (FAR) [20] as more sensitive composite indicators of inflammation severity than single indicator, which can provide directions for future studies and possible investigations based on the findings of the present study.

Importantly, this concerted pattern of increases (in acute phase reactants, inflammatory cytokines, autoantibodies, and complement) and decrements (albumin) reflects the many interrelated derangements in the pathophysiological network seen in rheumatoid arthritis [4,24]. This pattern emphasizes the importance of the contribution of both humoral and cellular immunity in the maintenance of synovial inflammation and joint destruction. Endeavoring towards a multi-biomarker diagnostic solution rather than a standalone marker [7,24] continue to support the modern scientific strategy of enhancing the likelihood of the early diagnosis and the gauging of response to disease-modifying interventions.

5. Conclusion

The present study found that there is a significant increase in inflammatory markers (CRP, ESR, IL-6, TNF- α), particular immune markers (RF, Anti-CCP), humoral, and complement immune markers (IgG, C3, C4), and total protein among rheumatoid arthritis patients as compare to healthy control with significant decrease in serum albumin level. In conclusion, these findings together corroborate the inflammatory and immune complex features of this disease and this supports the incremental diagnostic benefit of using a panel of laboratory markers rather than a single marker, thereby improving the efficacy of the diagnosis and monitoring of treatment response in these patients.

Statements on compliance with ethical standards

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Disclosure of conflict of interest

The authors declare no conflicts of interest regarding this manuscript.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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