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RESEARCH ARTICLE

Studying the Level of Amyloid-A and Fibrinogen-Like Protein 1 and Other Biomarkers in the Sera of Rheumatoid Arthritis of Iraqi Patients

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ABSTRACT

Rheumatoid arthritis (RA) is a common chronic autoimmune disease characterized by synovitis causing pain and inflammation and can result in joint destruction, its risk factors involve a family history of disease, obesity, smoking as well as viral infections. In this study, serum amyloid A and fibrinogen-like protein 1 were evaluated in 80 patients and 40 healthy controls (both men and women). Measurement of SAA, FGL1, RF, CRP, and MPO were performed using ELISA kits, the colorimetric method has been employed for estimating lipid profile, and Westergren method has used to measure ESR. The results indicated that levels of SAA, FGL1, MPO, RF, CRP, and ESR were significantly higher mean RA patients compared to the control group, with notable findings include MPO (392.8 ± 62.7 vs. 298.4 ± 44.5 ng/ml), SAA (13.6 ± 5.0 vs. 7.17 ± 1.0 pg/ml), FGL1 (283.7 ± 121.8 vs. 91.1 ± 23.0 ng/ml), RF (78.6 ± 14.7 vs. 27.1 ± 9.1 IU/ml), CRP (7.6 ± 1.7 vs. 2.4 ± 1.1 ng/ml), and ESR (41.2 ± 29.3 vs. 12.0 ± 10 mm/hr). The Receiver operating characteristic curve (ROC) analysis revealed potent diagnostic potencies, yielding an AUC of 0.99 and 0.94 for SAA and FGL1, respectively. Conclusion, the present study revealed that SAA and FGL1 represent as practical diagnostic markers to distinguish RA patients from healthy subjects, providing powerful potency to monitor RA progression and guide the therapeutic intervention.

Keywords: Rheumatoid arthritis (RA), Serum Amyloid A (SAA), Myeloperoxidase (MPO), Fibrinogen like protein 1 (FGL1), C-reactive protein (CRP)

Introduction

Rheumatoid arthritis (RA) is a chronic inflammatory systemic autoimmune pathology that attacks the lining of joints throughout the body, damaging the synovial joints and affecting smaller joints before spreading to larger joints and extra-articular organs like the heart, kidney, lung, digestive tract, eyes, skin, and nervous system.[1] One out of 200 individuals worldwide have rheumatoid arthritis, with women being two to three times more susceptible than men. The peak incidence occurs between the ages of 50 and 59. It is believed that genetics, immune system effects, and sexual hormones contribute to the female preponderance. Men and women experience different disease features, such as extra-articular symptoms, comorbidities, functional outcomes, and disease activity. Treatment responses and treatment choices may also differ.[2] Patients with RA frequently have stiffness, tender, swollen joints, generalized symptoms, and abnormal laboratory tests; in up to 90% of cases, early examination can prevent or reduce the development of the disease symptoms and prevent irreversible disability and joint damage. Therefore, early diagnosis is essential for effective treatment.[3] T cells and B cells are critical in the immune response to RA, secreting pro-inflammatory cytokines and activating B cells to produce RA-specific autoantibodies. Their interaction with T cells is essential to the

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disease's pathogenesis. Therapeutic strategies often target T and B cells, using drugs like methotrexate, corticosteroids, and biologic agents like TNF inhibitors and B-cell depleting therapies.[4] Biomarkers for RA, including Rheumatoid factor (RF), Anti-citrullinated protein antibodies (ACPA), Erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP), have the potential for prognostic, therapeutic, diagnostic, and predictive applications. The American College of Rheumatology and EULAR consider these immunological markers in their classification criteria.[5]

Hepatocytes produce fibrinogen-like protein 1 (FGL1) and belong to the fibrinogen-related protein superfamily. It is crucial in tissue repair, immune response regulation, cell proliferation, and survival. FGL1 is also a critical inhibitory ligand for the T cell inhibitory activity of lymphocyte activation gene 3 (LAG3), an immune checkpoint that helps maintain immune tolerance and prevent excessive immune activation.[6]

Myeloperoxidase (MPO) is a protein of monocytes and neutrophil granulocytes in the heme peroxidase-cyclooxygenase family. It plays an indispensable role in the immune system as it converts hydrogen peroxide (H_2O_2) and chloride ions into reactive oxygen species (ROSs), mostly hypochlorous acid (HOCl). This potent oxidative species is essential for the destruction of bacteria, fungi, and other pathogens, increasing the immune response of the body.[7]

Serum Amyloid A (SAA) is an acute-phase protein produced primarily in the liver, that is upregulated in response to pro-inflammatory cytokines, especially during infections and tissues damages, and could noticeably upraise during viral infections.[8]

The overlap between atherosclerotic cardiovascular disease (ASCVD) and RA occurs due to shared inflammatory pathways largely mediated by pro-inflammatory cytokines, namely TNF-α, IL-6, and IL-1. Such cytokines drive chronic inflammation in rheumatoid synovitis, and they also further destabilized atherosclerotic plaque in cardiovascular disease (CVD). This systemic inflammation can be found in both RA and gout, and explains lower ASCVD risk in gout patients, thereby linking RA-mediated systemic inflammations to cardiovascular sequelae. This suggests that by controlling inflammation in RA, ASCVD risk can also be lowered.[9]

The current investigation aims to assess the diagnostic ability of SAA, MPO, and FGL1 as biomarkers for predicting RA progression and estimate the disease severity. In addition, we investigate whether these studied biomarkers effectually monitor RA progression along with predict the associated complications like CVD, to reinforce the strategies used for diagnosis and long-term disease management.

Materials and methods

In the present case-control study, 80 RA patients (they were clinically diagnosed) and 40 healthy individuals as a control group, their age ranged between 16 to 69 years. The patients group included 6 men and 74 women. The blood samples have been collected during the period from September to November at Baghdad Teaching Hospital/Medical City in Iraq. 7ml of venous blood has been taken at 8:30 a.m. from all patients, where 2ml has been placed in sodium citrate tubes for erythrocyte sedimentation rate (ESR) test; 2ml were put EDTA tubes for complete blood count (CBC) analysis, and 3ml were put in gel tubes, the specimens have been allowed for clotting for about 10mins, then centrifuged at 3000rpm for 5mins to obtain the sera. These sera were then divided into Eppendorf tubes and stored at -20°C for up to three months until analysis. The enzyme-linked immunosorbent (ELISA) technique has been applied to evaluate the studied biochemical and immunological biomarkers, which are involving myeloperoxidase (MPO), fibrinogen-like protein 1 (FGL1), serum amyloid A (SAA), rheumatoid factor (RF), as well as C-reactive protein (CRP) by utilizing the commercially kits from Fine Test, China. The colorimetric method using the commercially kits from Linear company/Spain has been utilized to evaluate the lipid profile, while CBC parameters (WBCs, RBCs, hemoglobin, hematocrit, and platelets) were measured employing the Nihon Kohden instrument.

An MPO, FGL1, and SAA antibody-coated 12×8-well plate was used according to the instructions of the manufacturer. Wells were primed to isolate MPO, FGL1, and SAA from the serum. In each well, 100 μl of serum or standard was added, followed by the addition of a biotin-antibody working solution and incubated at 37°C for 60 minutes, and washing was performed to remove unbound components. Then, the HRP-Streptavidin conjugate (SABC) has been appended and allowed to incubate. After further washing to remove unbound reagents, a TMB substrate solution was added. The HRP catalyzed the TMB, producing a blue-colored product that turned yellow after adding a stop solution. The absorbance was measured at 450 nm, with the color intensity corresponding to the concentration of the parameters being analyzed.

Table 1. The statistical distributions of age and BMI study population.

Parameters	Mean ± SD		Probability values
	Healthy controls (n = 40)	RA patients (n = 80)	
Age (Years)	48.2 ± 11.1	49.55 ± 10.1	0.25
BMI (Kg/m^2)	29.6 ± 3.2	29.4 ± 5	0.43

SD: Standard deviation, n: Number, +ev: positive; -ev: negative.

Table 2. Serum levels of MPO, SAA, and FGL1 in RA patients and control.

Parameters	Mean ± SD		P-value
	Healthy controls (n = 40)	RA patients (n = 80)	
MPO (ng/ml)	298.4 ± 44.5	392.8 ± 62.7	0.0001***
SAA (pg/ml)	7.17 ± 1.0	13.6 ± 5	0.0001***
FGL1(ng/ml)	91.1 ± 23.0	283.7 ± 121.8	0.0001***

SD: Standard deviation, N: Number, p-value; +ev: positive; -ev: negative.

Table 3. The statistical distributions of RF, CRP, and ESR in RA patients and control.

Parameters	Mean ± SD		P-value
	Healthy controls (n=40)	RA patients (n=80)	
RF (IU/ml)	27.1 ± 9.1	78.6 ± 14.7	0.0001***
CRP (ng/ml)	2.4 ± 1.1	7.6 ± 1.7	0.0001***
ESR (mm/hr)	12.0 ± 10	41.2 ± 29.3	0.0001***

SD: Standard deviation, N: Number, p-value; +ev: positive; -ev: negative.

Inclusion criteria

Patients with rheumatoid arthritis were diagnosed with clinical and an X-ray.

Exclusion criteria

RA patients diagnosed via X-rays, congenital or acquired dysplasia, pregnancy, diabetes, anemia, elevated liver enzymes, genetic disease, cancer, and any other inflammatory disease like OA or hepatitis C, all of the above were excluded from the study.

Statistically analysis

Statistical analysis has been achieved employing Statistical Program for Social Sciences (SPSS).[10] The individual correlation has been employed to underscore the association between SAA and FGL1 with other studied markers of RA patients. A significant level of 0.05 or less had considered significant from a statistical perspective. ROC curve mythology examines the optical cutoff for MPO, SAA, and FGL1.

Results and discussion

As shown in Table 1, the average age difference between the RA patient group and the control group is not statistically significant ($p > 0.05$). Similarly, there are no statistically significant changes in BMI, with the RA patient group having an average of 29.4 ± 5 compared to 29.6 ± 3.2 in the control group.

Table 2 presents the three key parameters. The serum levels in the RA patient group were significantly elevated compared to the control group, with a p-value of less than 0.05. Specifically, MPO levels were 392.8 ± 62.7 ng/dl in patients versus 298.4 ± 44.5 ng/dl in controls; SAA levels were 13.6 ± 5 pg/ml in patients compared to 7.17 ± 1.0 pg/ml in controls; and FGL1 levels were 283.7 ± 121.8 ng/dl in patients versus 91.1 ± 23.0 ng/dl in controls.

Table 3 presents significant differences between the studied groups (RA patients and controls) for the three parameters, with a p-value of less than 0.05. The results are as follows: RF levels were 78.6 ± 14.7 IU/ml in patients compared to 27.1 ± 9.1 IU/ml in controls; CRP levels were 7.6 ± 1.7 ng/dl in patients versus 2.4 ± 1.1 ng/dl in controls; and ESR levels were 41.2 ± 29.3 mm/hr in patients compared to 12.0 ± 10 mm/hr in controls.

Table 4 demonstrates significant differences in total white blood cell count (WBC) between RA patients 7.6 ± 2.2 and the control group 8.44 ± 1.9. In addition, the results of statistical analysis revealed a significant differences in the levels of red blood cells (RBCs), hemoglobin (HB), and hematocrit (HCT), where RA cases exhibited 4.4 ± 0.5 (RBCs), 11.9 ± 1.5 (HB), and 37.8 ± 4.3 (HCT) compared with healthy individuals values of 4.8 ± 0.6 (RBCs), 13.0

Table 4. The statistical distributions of CBC values in RA patients and controls.

Parameters	Mean ± SD		P-value
	Healthy control (n = 40)	RA cases (n = 80)	
WBC (10^3/uL)	8.44 ± 1.9	7.6 ± 2.2	0.035*
RBC (10^6/uL)	4.8 ± 0.6	4.4 ± 0.5	0.0001 ***
HB (g/dl)	13.0 ± 1.7	11.9 ± 1.5	0.0003***
HCT (%)	41.2 ± 5.4	37.8 ± 4.3	0.0004***
PLT (10^3/uL)	254 ± 97	278.1 ± 97	0.16

Table 5. The statistical distributions of the lipid profile in RA patients and control groups.

Parameter	Mean ± SD		P-value
	Healthy controls (n = 40)	RA Patient (n = 80)	
Cholesterol (mg/dl)	165.6 ± 31.9	205.5 ± 25.6	0.0001***
Triglyceride (mg/dl)	118.4 ± 9.4	220 ± 6.0	0.0001***
HDL (mg/dl)	45.2 ± 8.8	33.0 ± 5.6	0.0001***
LDL (mg/dl)	96.4 ± 32.8	129.0 ± 28.1	0.0001***
VLDL (mg/dl)	23.7 ± 2.0	43.9 ± 1.2	0.0001***

SD: Standard deviation, n: Number, p-value; +ev: positive; -ev: negative, HDL: high-density lipoprotein, LDL: low-density lipoprotein, VLDL: very low-density lipoprotein.

Table 6. Correlation analysis for MPO with other parameters in the study population.

Parameter	Healthy controls		patients	
	R	p	R	P
SAA	–0.01	0.96	–0.07	0.52
FGL1	–0.31*	0.05	0.13	0.22
ESR	–0.11	0.52	–0.03	0.78
RF	–0.27	0.09	0.05	0.60
CRP	–0.2	0.22	–0.12	0.27
CHOL	–0.16	0.32	0.04	0.72
TG	0.09	0.56	0.28*	0.01
HDL	–0.42*	0.01	0.32*	0.00
LDL	–0.05	0.75	0.06	0.6
VLDL	0.13	0.42	0.27*	0.02
WBC	–0.09	0.6	0.09	0.42
RBC	–0.05	0.77	–0.09	0.41
HB	–0.04	0.82	0.07	0.52
HCT	–0.06	0.71	0.02	0.83
PLT	0.00	0.99	–0.07	0.51

n: number, R: person correlation, p-value.
The strength MPO of correlations between variables is estimated by linear regression analysis.

± 1.7 (HB), and 41.2 ± 5.4 (HCT). Conversely, the counts of platelet (PLT) demonstrated non-significant differences between RA patient (278.1 ± 97) and control group (254 ± 97).

Table 5 exhibits the results of lipid profile (LP), emphasized a significant difference between cases and control groups. Cholesterol levels were significantly higher in the patient group 205.5 ± 25.6 compared to the control group 165.6 ± 31.9. Triglyceride (TG) levels also considerably increased in the patient group, 220 ± 6.0, versus the control group, 118.4 ± 9.4. Additionally, high-density lipoprotein (HDL) levels significantly decreased in the patient group 33.0 ± 5.6 compared to the control group 45.2 ± 8.8. Low-density lipoprotein (LDL) and very low-density lipoprotein (VLDL) levels significantly increased in the patient group 129.0 ± 28.1 and 43.9 ± 1.2, respectively, compared to the control group 96.4 ± 32.8 and 23.7 ± 2.0, with a p-value of less than 0.0001.

Table 6 presents the personal correlation of MPO with other parameters in patients as compared with healthy individuals. In RA patient, MPO levels exhibit a significant positive correlation with triglycerides (TG), high-density lipoprotein (HDL), and very low-density lipoprotein (VLDL). Conversely, in the control group, there is a negative correlation between MPO FGL1 and HDL.

Table 7. Correlation analysis for SAA with other parameters study population.

	Healthy controls		patients	
Parameter	R	p	R	P
MPO	–0.01	0.96	–0.07	0.53
Fib-like-1	0.07	0.67	–0.14	0.22
ESR	0.09	0.58	0.03	0.78
RF	–0.15	0.37	0.32*	0.00
CRP	–0.02	0.92	0.00	1.0
CHOL	–0.04	0.78	0.13	0.26
TG	–0.01	0.97	–0.11	0.34
HDL	–0.22	0.17	–0.08	0.5
LDL	0.02	0.91	0.07	0.56
VLDL	–0.05	0.77	–0.11	0.34
WBC	0.24	0.13	0.08	0.46
RBC	–0.13	0.42	–0.07	0.55
HB	–0.16	0.32	0.15	0.18
HCT	–0.14	0.38	0.07	0.56
PLT	0.10	0.53	–0.26*	0.02

The linear regression analysis was utilized to rate the mightiness of SAA with other parameters

Table 8. Correlation analysis for FGL1 with other parameters study population

	Healthy controls (n = 40)		patients (n = 80)	
Parameters	R	p	R	P
MPO	–0.31*	0.05*	0.14	0.23
SAA	0.07	0.67	–0.14	0.22
ESR	–0.07	0.68	–0.18	0.11
RF	0.08	0.63	–0.1	0.36
CRP	–0.06	0.7	–0.03	0.83
CHOL	0.12	0.47	–0.07	0.54
TG	–0.12	0.48	–0.02	0.88
HDL	0.06	0.73	–0.02	0.84
LDL	0.11	0.51	–0.01	0.95
VLDL	–0.15	0.36	–0.02	0.88
WBC	0.27	0.09	–0.27*	0.02
RBC	–0.07	0.65	–0.01	0.94
HB	0.31*	0.05	0.02	0.88
HCT	0.3	0.06	0.05	0.63
PLT	0.15	0.34	–0.06	0.6

The linear regression analysis was utilized to rate the mightiness of FGL1 correlations with other parameters.

Table 7 illustrates the personal correlation of SAA with other parameters in the patient group compared to the control group. In the patient group, SAA levels demonstrate a significant positive correlation with RF, exhibiting a correlation coefficient of 0.32 and a p-value of 0.00. Additionally, PLT shows a significant negative correlation with SAA, with a correlation coefficient of -0.26 and a p-value of 0.02. In contrast, no significant correlations were observed in the control group.

Table 8 presents the personal correlation of FGL1 with other parameters in the patient group compared to the control group. In the patient group, FGL1 levels exhibit a significant negative correlation with WBC, showing a correlation coefficient of -0.27 and a p-value of 0.02. Conversely, a significant negative correlation with MPO is noted in the control group, with a correlation coefficient of -0.31 and a p-value of 0.05. Additionally, a significant positive correlation is observed with HB in the control group, featuring a correlation coefficient of 0.31 and a p-value of 0.05.

The receiver operating characteristic curve (ROC) is a statistical tool that reveals the relationships between sensitivity and specificity, using to compare biomarker levels between patients and healthy individuals to point out the optimal diagnostic biomarkers. As displayed in Table 9 and Fig. 1, ROC analysis for MPO, SAA, and FGL1 biomarkers revealed specific cut-off values, showed sensitivities values of 92%, 97.5%, and 93.7%, along with specificities values of 84%, 100%, and 80%, respectively. The findings of current study showed that the studied biomarkers are potent predictors to diagnosis the RA cases.

Table 9. ROC analysis for MPO, SAA, FGL1.

Parameters	AUC value	Sensitivity	Specificity	Cutoff value
MPO	92%	92%	84%	>323.0
SAA	99.8%	97.5%	100%	>8.83
Fib like 1	94%	93.7%	80%	>94.54

AUC: The area under the curve.

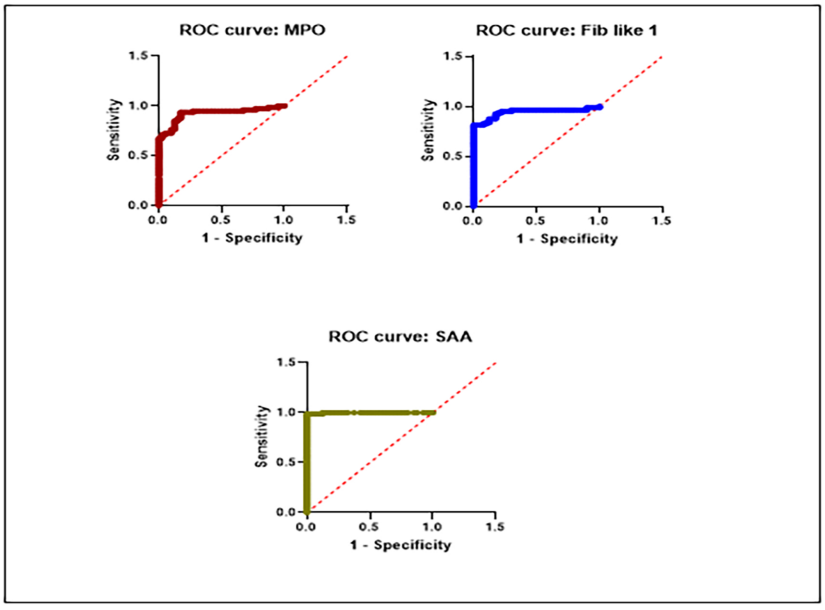

Fig. 1. The curves of ROC analysis for SAA, FGL1, and MPO.

Discussion

The present study demonstrates a significant relationship between MPO levels and progression of RA. Increment MPO levels indicate magnified the inflammation and immune responses, leading to tissues damage and the possible onset of CVD in patients with RA.[11] A previous study of Foer's et al.[12] emphasized MPO pathogenic roles, which is a reactive protein present in neutrophil azurophilic granules in patients with RA. The upraised MPO levels in RA synovium leading to oxidative stress, tissue damages, and development the atherosclerosis as well as other cardiovascular complications, highlighting the demand to track the MPO as a biomarker for RA.

The levels of SAA showed a significant increase in RA patients as compared with healthy individuals. These increased SAA levels in RA patients are linked to increased liver's production in the course of acute inflammatory response, revealing the crucial role of SAA in the inflammatory process and probable disease pathogenesis, thereby it a critical biomarker.[13]

With regard to FGL1 levels, these levels being significantly higher in RA patients than in healthy individuals. The current finding aligns with those in the study of Laurindo et al., which explained that the interaction between the immune regulator LAG-3 and FGL1 enhance the cytokine releasing and guides the inflammations. This underscores FGL1 roles in immunological regulation and its engagement in the inflammatory proceeding in RA.[14] The study of Yousif et al.[15] demonstrates that LAG-3 is expressed on aged T-cells within atherosclerotic plaques, and it is independently and positively correlated with CVD.

In the context of ESR, its levels showed significant increment in RA patients than in controls, revealed that the inflammations are frequently linked with upraised disease activity and damage to joint; this finding agree with the result of Alturaiki et al.[16] Another study proposed that high ESR levels signify the inflammations, participating to develop the atherosclerosis and other CVD conditions, thus ESR serve as a potential predictor for CVD.[17]

As for CRP levels, the present study showed that its levels were significantly increased in RA patients than in controls, thereby it indicates active RA, since it intensifies the inflammations in influenced joints.[18]

The present study also revealed a significant increase in RF levels in RA patients as compared with healthy individuals, this finding in line with the

finding of Conforti et al.,[19] who as well demonstrated high RF concentrations in RA patients. However, the study underscored the lack of specificity of RF in identifying RA and suggested that it should be combined with other parameters for a more precise diagnosis.

Regarding to lipid profile results, the present study showed a strong association in RA patients. This finding aligns with the study of Rodriguez et al.,[20] which demonstrated increased TG levels and reduced HDL levels, thus supported the current study's outcomes. In the study conducted by Van den Oever et al.,[21] the cholesterol was significantly increased in RA patients than in control group, which in line with the observations of current study. In another study indicated that dyslipidemia, particularly high cholesterol levels, is a usual feature in RA patients, likely contributed to increase CVD risk in RA patients.[22]

The levels of WBCs reveal significant decreased in RA cases as compared with controls. A similar finding is reported by Mykola et al.,[23] which supported the finding of present study. The RBCs and Hb levels exhibited significant decreased in RA cases as compared with controls. These findings agree with Farouk et al.,[24] who highlighted the significance of CBC parameters in realization RA impacts. The levels of HCT exhibited significant decreased in RA cases as compared with controls; this is in line with findings of Xue et al.,[25] who also revealed a diminished HCT levels in RA patients. The PLT levels show non-significant difference between patients and controls. The study of Sağ S et al.[26] also reported that PLT levels showed non-significant difference between RA patients and controls, matching our findings.

The current study demonstrated non-significant alteration in the mean age when compared RA patients with healthy individuals. These findings align with those of Alanzy et al.,[27] who also reported absent of significant difference in age between two groups. This proposed that RA could develop at any age and is affected more by genetic, environmental, and immunological determents than by age alone.

As for BMI comparison, it showed non-significant differences between RA patients and healthy control, revealing that RA influences the individuals regardless of their BMI values, highlighting the roles of immunological responses and genetic determents. This aligns with the findings of van den Ibrahim et al.,[28] who also demonstrated non-significant difference in BMI. On the other hand, another study has suggested that high BMI, particularly in women, could boost the likelihood of progressing RA, exacerbated symptoms, and lead to damage the joint and inflammations.[29]

Conclusion

The biomarkers SAA, MPO, and FGL1 were significantly positively associated with RA disease. The findings show that SAA and RF had a direct correlation while PLT had an inverse relationship. The third row shows the correlation of MPO was positive with TG, HDL, and VLDL while FGL1 was negatively correlated with WBC. These discoveries indicate that these biomarkers are involved in the pathogenesis of RA, which can facilitate the enhancement of RA management. They were also identified as major risk factors and possibly better predictors of CVD in RA compared to other cohorts; future studies are recommended to investigate the longitudinal role of SAA, MPO, and FGL1 as predictors of RA disease progression and cardiovascular risk to better inform the development of tailored therapy regimens as well as to improve monitoring and management.

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Authors' declaration

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are ours. Furthermore, any Figures and images, that are not ours, have been included with the necessary permission for republication, which is attached to the manuscript.
- No animal studies are present in the manuscript.
- Authors signed on ethical consideration's approval.
- Ethical Clearance: The project was approved by the local ethical committee at University of Baghdad.

Authors' contribution statement

A. W. A. collect the samples, did the experiment, analysis the results, and elucidated them. B. F. H. had achieved the design and over sighted of the work.

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دراسة مستوى الاميلويد- أ والبروتين الشبيه بالفيبرينوجين - 1 والعلامات الحيوية الأخرى في مصل التهاب المفاصل الرثوي لدى المرضى العراقيين

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الملخص

التهاب المفاصل الرثوي هو اضطراب مناعي ذاتي مزمن يؤثر على المفاصل، مسببًا الألم والتورم وتلف المفاصل. يصيب نحو 1 من كل 200 شخص، مع انتشار أعلى بين النساء. تشمل عوامل الخطر التاريخ العائلي، السمنة، التدخين، والعدوى الفيروسية. شملت الدراسة 120 مشاركًا: 80 مصابًا بالمرض و40 من الأصحاء، تتراوح أعمارهم بين 16 و69 عامًا من الجنسين. تم قياس مستويات مصل أميلويدA ، أميلوبيروكسيداز، بروتين شبيه الفايبرينوجين 1، معدل ترسيب كريات الدم الحمراء، البروتين التفاعلي سي، العامل الرثوي، وتعداد الدم الكامل، بالإضافة إلى تحليل الدهون الكلية. أظهرت النتائج ارتفاعًا ملحوظًا في المتوسط الحسابي للمؤشرات الحيوية(SAA ، FGL1، MPO، RF، ESR) لدى المرضى مقارنة بالأصحاء. كما موضح:

(MPO ٣٩٢,٨ ± ٦٢,٧ 44.5 ± 298.4 مقابل (، نانو غرام/مل) SAA ١٣,٦ ± ٥,٠ 1.0 ± 7.17 مقابل(، بيكو غرام/مل) FGL1 ٢٨٣,٧ ± ١٢١,٨ ±23.0 91.1 مقابل(، نانو غرام/مل) RF ٧٨,٦ ± ١٤,٧ 9.1 ± 27.1 مقابل(، وحدة دولية/مل CRP(7.6 ± 1.7 ±1.1 2.4 ± مقابل (نانو غرام/مل) ESR, ٤١,٢ ± ٢٩,٣ ١٢,٠ ± مقابل (.ملمتر/ساعة

أظهر تحليل منحنى خصائص تشغيل المستقبل قدرات تشخيصية قوية، حيث بلغت المنطقة تحت المنحنى (AUC) 0.99 لأميلويد A (SAA) و0.94 لبروتين .FGL1خلصت الدراسة إلى أن SAA وFGL1 يمثلان مؤشرات حيوية فعالة للتمييز بين مرضى التهاب المفاصل الرثوي والأصحاء، مما يوفر إمكانية لمراقبة تقدم المرض وتوجيه التدخلات العلاجية. وقد تسهم هذه المؤشرات الحيوية في التنبؤ بمضاعفات مرتبطة بالمرض مثل أمراض القلب والأوعية الدموية، مما يعزز من فوائدها السريرية.

الكلمات المفتاحية: التهاب المفاصل الرثوي، مصل الاميلويد اي، الفايبرينوجين الشبيه بالبروتين 1، الاميلوبيروكسيديس، بروتين سي التفاعلي.