

A Study of Biochemical Parameters Affecting Arthritis Patients and Their Association with Kidney Disease

Shaymaa.H.Fayyadh*

University of AL-Fallujah, Collage of Applied Sciences, Al Ramadi, Iraq

Article Info

Article history:

Received May, 18, 2025

Revised June, 13, 2025

Accepted June, 26, 2026

Keywords:

Arthritis,
Kidney Diseases,
CRP and ESR,
Assessment

ABSTRACT

Arthritis refers to a collection of illnesses and conditions that impact and damage the joints in the body. Common symptoms typically involve pain and stiffness in the joints, along with additional signs like redness, warmth, swelling, and a reduced range of motion in the impacted joints. Some forms of arthritis also impact other organs. The beginning of the illness may be slow or rapid. The purpose of the study is to study the relationship between vital variables and kidney diseases and the relationship between urea and creatinine in patients with arthritis. The study included 60 samples of women and men between the ages of 30 and 55 years old and the samples were divided 30 samples of arthritis patients, 20 samples of women and 10 samples of men. The control group included 30 samples from people without arthritis, 20 samples from women and 10 samples from men. Chronic medical conditions such as cancer, people with chronic inflammation, and pregnant women were excluded. Five milliliters of venous blood samples were collected from patients and control group, blood was clotted at 37°C and centrifuged at 4000 rpm for 10 minutes and blood was separated for direct ESR analysis, serum separation and serum separation for CRP, Uric acid, Urea, creatinine, ALP. The study showed higher levels of erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), creatinine, urea, and uric acid in the blood serum, with a significant difference.

From all of the above, we can conclude that these biomarkers can be used as predictors of arthritis.

Corresponding Author:

* Shaymaa hameed fayyadh

University of AL-Fallujah, Collage of Applied Sciences, Al Ramadi, Iraq

shaymaa.h.fayyadh@uofallujah.edu.iq

1. INTRODUCTION

Recent research shows that individuals with arthritis face a higher likelihood of developing chronic kidney disease (CKD). A meta-analysis indicated that RA patients are 52% more likely to develop CKD than the general population [1]. The occurrence of CKD in arthritis patients varies between 20.8% and 24.5% [2,3], with main risk factors being age, diabetes, cardiovascular conditions, hypertension, and the activity level of arthritis [3]. Moreover, the activity of arthritis and specific medications, especially glucocorticoids and nonsteroidal anti-inflammatory drugs (NSAIDs), could play a role in the onset of CKD [4].

Arthritis is a chronic systemic autoimmune disease associated with potentially debilitating joint inflammation. The bones of a joint are covered with a smooth, spongy, material called cartilage, which cushions the bones and allows the joint to move without pain. The joint is lined by the synovium. This synovium's lining produces a slippery fluid called synovial fluid, that nourishes the joint and helps limit friction within. External to it is a strong fibrous casing

called the joint capsule. Strong bands of tissue called ligaments connect the bones and help keep the joint stable. Muscles and tendons also support the joints and enable us to move. With arthritis, an area in or around a joint becomes inflamed, causing pain, stiffness and sometimes difficult moving. Arthritis can also affect other parts of the body, such as the skin and internal organs [5] The cause of arthritis differs based on the specific kind of arthritis, Osteoarthritis is primarily influenced by factors such as getting older, being female, experiencing joint injuries, and being overweight. Certain genetic factors, such as mutations in genes that code for collagens II, IV, V, and VI, have been identified. [6,7] The most frequent pathological results on kidney biopsies in RA patients were secondary amyloidosis, membranous nephropathy, and occasionally rapidly progressive glomerulonephritis as revealed in clinic-pathological correlations [8].

Gout results from the accumulation of uric acid crystals in the joints, causing inflammation. that occurs due to the development composed of calcium pyrophosphate rhomboid crystals Gouty typically starts in one joint initially, but can eventually spread to multiple joints and become significantly disabling over time. [9] Swelling and impaired function frequently occur in the joints of individuals with gout. Gouty arthritis can become extremely painful and incapacitating if traditional gout treatments are ineffective. When standard medications do not control refractory chronic gout is identified when there are elevated levels of uric acid and symptoms of gout [10] The specific connection between long-term inflammation and CKD is not consistently understood in arthritis patients, yet scientists believe specific inflammatory proteins are involved. This indicates that the greater the severity of your RA (marked by elevated inflammation levels), the increased likelihood of experiencing kidney issues. Unregulated inflammation can harm the linings of the kidneys and result in a condition known as atherosclerosis, in which plaque accumulates within the renal arteries (the arteries of the kidneys) [11].

2. MATERIALS AND METHODS

The study included 60 samples of women and men between the ages of 30 and 55, and was divided into 30 samples of arthritis patients, 20 samples of women, and 10 samples of men. The control group included 30 samples of people without arthritis, 20 samples of women, and 10 samples of men. The control group consisted of 30 samples from people without arthritis, 20 samples from women and 10 samples from men. The patients were enrolled from the rheumatology unit at Ramadi Teaching Hospital. Patients were selected based on the classification principle established by the American College of Rheumatology in 1987. Chronic medical conditions such as cancer, people with chronic inflammation, and pregnant women were excluded. Five milliliters of venous blood samples were collected from patients and control group, blood was clotted at 37°C and centrifuged at 4000 rpm for 10 minutes and blood was separated for direct ESR analysis, serum separation and serum separation for CRP, Uric acid' Urea, creatinine, ALP.

3. RESULTS AND DISCUSSION

3.1. Descriptive statistic for study sample

Table (3-1) this table show Descriptive statistic for study sample as we notice the patient and control groups parameter sorting according to uric A, urea ALP, Creatinine, ESR and CRP test and all patient parameter higher than normal.

Table (3-1): Mean and S.D of biochemical parameters

LABPARAMETES		N.	Minimum	Maximum	Mean	Std.Deviation	
URIC A.	Normal	30	3.00	7.00	4.5700	.91431	
	patient	30	3.00	9.80	5.4172	1.80001	
UREA	Normal	30	18.00	30.00	23.7333	3.68532	
	Patient	30	22.10	104.90	49.6069	18.90287	
ALP	Normal	30	90.00	224.00	178.9033	32.98742	
	Patient	30	67.00	397.00	212.1552	90.10177	
CREATININE	Normal	30	.70	1.10	.8933	.11427	
	patient	30	.50	2.00	1.1931	.40172	
E	Normal	30	5.00	20.00	13.6667	4.23722	
ESR	patient	30	30.00	120.00	51.1379	21.44215	
	Normal	30	1.00	1.00	1.0000	.00000	
CRP	patient	30	12.00	48.00	27.7241	14.73309	

3.2. Uric Acid:

Table (3-2) this table clear there is different in means of study sample according to Uric A. parameter between patient and control group by t value 2.268 and significant level less than 0.05.

Table (3-2) Mean and S.D of Uric acid in patients and Healthy Control

			Independent test			
Uric A.	N	Mean	Std.Deviation	T	P.Value	Significant
Patient	30	5.417	1.8000	2.268	.029	Significant
Normal	30	4.570	.9143			

In a research involving arthritis patients, levels of UA were significantly higher than the control group ($P < 0.01$). [12] Elevated levels of UA in individuals with arthritis were linked to hypertension, according to research by Hence, the recent findings did not align with prior studies. Uric acid is produced from purine breakdown, eliminated through urine by the kidneys, and provides antioxidant benefits. If UA is produced in excess or not eliminated effectively, it can build up and result in crystal formation within the joint, leading to various arthritis-related conditions like gout, kidney stones, RA, and blood disorders like diabetes. [13]. consumption of animal protein rich foods has significantly risen. due to the development of stone, chiefly uric acid stones, because of Several studies [14] have verified that higher consumption of animal proteins, specifically red meat, is closely linked linked to the chance of developing uric acid stones and this clarifies the situation a large percentage of older individuals develop uric acid stones, which are high in acidity study's findings. The cause of the rise in the uric acid percentage. high levels of acid in Anbar province are a result of the type of diet consumed. The population in this area mostly consumes. red meat, which is a significant source of animal proteins. with the idea that excessive consumption of animal products The recurrence of kidney stones is directly related to the levels of proteins [15].

3.3. Urea parameter

Table (3-3) this table clear there is different in means of study sample according to Urea parameter between patient and control group by t value 7.239 and high significant level less than 0.05.

Table (3-3) Mean and S.D of Urea in patients and Healthy Control

			Independent test			
Urea.	N	Mean	Std.Deviation	T	P.Value	Significant
Patient	30	49.607	18.9029	7.239	.000	High
Normal	30	23.733	3.6853			Significant

The outcomes of the experiments conducted in this study are presented in table (3-3) S. urea levels were measured as control (23.733 ± 3.6853) and patient (49.607 ± 18.9029) accordingly. It was evident in this situation that there was a notable distinction between the two groups. The levels of S. urea the levels of were much greater in the group of patients when compared to the controls (12), with a p-value of less than 0.01.

3.4. ALP parameter

Table (3-4) this table clear there is different in means of study sample according to ALP parameter between patient and control group by t value 1.870 and no significant level more than 0.05 level.

Table (3-4) Mean and S.D of ALP in patients and Healthy Control

			Independent test			
ALP	N	Mean	Std.Deviation	T	P.Value	Significant
Patient	30	212.155	90.1018	1.870	.070	No.Significant
Normal	30	178.903	32.9874			

Demonstrates a notable rise, with $P \leq 0.05$ in the ALP enzyme activity in individuals with arthritis related to inflammatory bowel disease. ALP enzyme activity is elevated in individuals suffering from inflammatory bowel disorder and arthritis, aligning with findings of increased ALP effectiveness in rheumatoid arthritis patients [16] (Dubey, et.al,) If RA is confirmed by the results linking ALP alkaline phosphatase level with arthritis, [17] then it can be inferred that its measurement can serve as a biomarker for detecting and forecasting inflammatory conditions in the joints This study also aligns with the results of prior studies on ALP levels in arthritis patients conducted by Makhdoom.et.al discovered that patients with arthritis have higher levels of basal phosphatase than others [18].

3.5. Creatinine parameter:

Table (3-5) this table clear there is different in means of study sample according to creatinine parameter between patient and control group by t value 3.870 and high significant level less than 0.05.

Table (3-5) Mean and S.D of Creatinine in patients and Healthy Control

			Independent test			
Creatinine	N	Mean	Std.Deviation	T	P.Value	Significant
patient	30	1.193	.4017	3.870	.000	High
Normal	30	.893	.1143			Significant

As seen in table (3-5) creatinine level showed a significant difference when compared between group A and Control (0.893 ± 0.1143) patient (1.193 ± 0.4017) mg/dl There was a notable variance in creatinine level between patient group and the control group (0.893 ± 0.1143 patient 1.193 ± 0.4017) mg/dl The findings from the current research show elevated levels of urea and creatinine. In arthritis assistance, the outcomes align with the findings of the prior research. A prior study demonstrated that levels of creatinine were markedly elevated Patients with arthritis had a higher prevalence than the control group ($p < 0.01$). The current findings align with the earlier studies. [12,19].

3.6 ESR parameter

Table (3-6) this table clear there is different in means of study sample according to ESR parameter between patient and control group by t value 9.238 and high significant level less than 0.05.

Table (3-6) Mean and S.D of ESR in patients and Healthy Control

			Independent test			
ESR	N	Mean	Std.Deviation	T	P.Value	Significant
Patient	30	51.14	21.442	9.238	.000	High
Normal	30	13.67	4.237			Significant

As demonstrated in **Table (3-6)** in the analysis made for ESR levels (mm/H), 13.67 ± 4.237 was obtained for Control, while 51.14 ± 21.422 was obtained for patient. The findings of this research demonstrated a rise in the ESR rate for arthritis patients arthritis. This is consistent with what researchers have found in numerous studies, as Wolfe noted and Michaud High erythrocyte sedimentation rate in arthritis patients , even if it is dormant without symptoms, but this diagnosis is insufficient It must be attributed with other criteria He observed through his study Shrivastava.et.al the high ESR with other inflammatory parameters such as CRP and TNF in patients with inflammation These results are also consistent with the study of Kay, et al, who observed an increase in arthritis ESR rate when studying the level of ESR and CRP together for arthritis patients. Rheumatoid arthritis [20,21] The findings of this research demonstrated a rise in the This increase may be due to the increased production of acute condition proteins as a result of the inflammation in the body, it leads to an increase in the sedimentation rate of red blood cells as a result of the increased viscosity of the blood [22].

3.7. C-Reactive protein parameter:

Table (1-7) this table clear there is different in means of study sample to CPR parameter between patient and control group by t value 8.901 and high significant level less than 0.05.

Table (3-7) Mean and S.D of CRP in patients and Healthy Control

		Independent test				
CRP	N	Mean	Std.Deviation	T	P.Value	Significant
Patient	30	27.7241	14.73309	8.901	.000	High
Normal	30	3.2000	1.78885			Significant t

Table (3-7) shows significant difference in the level of CRP (mg/L) between Control (1.0000 ± 0.0000) compared to patient (27.72 ± 14.73) accordingly significant correlation has been seen between serum CRP levels and tissue inflammation samples in patients with arthritis [23] CRP, a protein, created by the liver, acts as an initial sign of infection. [24].

3.8 Pearson Correlation

Table (3-8) this table show correlation level between case status and lab parameters as notice there are weak correlation at Uric A. ALP and Creatinine by Value 0.290, 0.243 and 0.461 respectively while there are moderate correlation in Urea, ESR and CRP by value 0.698, 0.779 and 0.796 respectively.

Table (3-8) Pearson Correlation with biochemical parameters affecting arthritis patients and Healthy Control

.	Uric A,	Urea	ALP	Creatinine	ESR	CRP
N	60	60	60	60	60	60
P	.290	.698	.243	.461	.779	.796
Sig	.013	.000	.032	.000	.000	.000

3.9. Multi Regression:

Table (3-9) this table to determine association between case status patient or healthy and independent lab parameter that explain 73% from dependent variable and significant by f value 23.16 and highly significant level 0.000 less than 0.01 and there are two lab parameter (Urea and ESR) with significant level less than 0.05.

.				Multi regression				
Dependent v.	Independent V.	R	R ²	F	F Sig	B	t	T
Case	Uric A.	.853	.728	23.157	0.001	.029	.882	0.38
	Urea					.007	2.297	2
	ALP					6.04	0.93	.026
	Creatinine					.050	.315	.926
	ESR					.008	2.210	.754
	CRP					.008	1.454	.032
								.152

Multiple regression indicates that there is for infected patients and the healthy group with the vital independent variables urea, creatinine, ESR, CRP, ALP, and uric acid studied to find through this statistical analysis a correlation with the symbol R and the correlation value is(0.853) and R² is 0.728 and the f value is (23.157) and the probability value F sig is 0.001 from here we conclude that there is a high correlation between the variables and patients and it is a significant correlation The F Sig indicates that there is a significant multiple regression for the two variables urea and ESR.

4. CONCLUSION

We conclude that there is a significant increase in urea and creatinine levels in patients compared to the control group, and this issue should be studied in detail in current studies, A significant increase in the erythrocyte sedimentation rate in patients with arthritis, with a significant difference compared to the control group, Significant increase in CRP level in arthritis patients with significant difference from the control group, No significant difference in the ALP of arthritis patients and healthy subjects in the control group.

REFERENCES

- [1] Raksasuk, S., & Ungprasert, P. (2020). Patients with rheumatoid arthritis have an increased risk of incident chronic kidney disease: A systematic review and meta-analysis of cohort studies. *International Urology and Nephrology*, 52(1), 147–154. <https://doi.org/10.1007/s11255-019-02346-4>
- [2] Mori, S., Yoshitama, T., Hirakata, N., et al. (2017). Prevalence of and factors associated with renal dysfunction in rheumatoid arthritis patients: A cross-sectional study in community hospitals. *Clinical Rheumatology*, 36(12), 2673–2682. <https://doi.org/10.1007/s10067-017-3804-5>
- [3] Tokoroyama, T., Ando, M., Setoguchi, K., et al. (2017). Prevalence, incidence and prognosis of chronic kidney disease classified according to current guidelines: A large retrospective cohort study of rheumatoid arthritis patients. *Nephrology Dialysis Transplantation*, 32(12), 2035–2042. <https://doi.org/10.1093/ndt/gfw315>
- [4] Kochi, M., Kohagura, K., Shiohira, Y., et al. (2016). Inflammation as a risk of developing chronic kidney disease in rheumatoid arthritis. *PLoS ONE*, 11(8), e0160225. <https://doi.org/10.1371/journal.pone.0160225>
- [5] David, Z. (2017). *Rheumatology and rheumatic diseases: Effects of arthritis* (Vol. 49, pp. 159–500). American College of Rheumatology.
- [6] Okubo, M., & Okada, Y. (2013). [Destruction of the articular cartilage in osteoarthritis]. *Clinical Calcium*, 23(12), 1705–1713. [In Japanese].
- [7] Abraham, S., & Patel, S. (2025). Monoarticular arthritis. In *StatPearls* [Internet]. StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK459313/>
- [8] Liu, Y., Wen, H. Y., Wang, L. H., & Wang, C. (2017). Focal segmental glomerulosclerosis lagged behind the onset of rheumatoid arthritis by 7 years: A case report and literature review. *Medicine (Baltimore)*, 96(1), e5789. <https://doi.org/10.1097/MD.0000000000005789>
- [9] Becker, M. A. (2005). *Arthritis and allied conditions: A textbook of rheumatology* (15th ed., pp. 2303–2339). Lippincott Williams & Wilkins.
- [10] Ali, S., & Lally, E. V. (2009). Treatment failure gout. *Medicine and Health, Rhode Island*, 92(11), 369–371. <https://doi.org/10.1.1.608.3812>
- [11] Kochi, M., Kohagura, K., Shiohira, Y., et al. (2016). Inflammation as a risk of developing chronic kidney disease in rheumatoid arthritis. *PLoS ONE*, 11(8), e0160225. <https://doi.org/10.1371/journal.pone.0160225>
- [12] Cockel, R., Kendall, M. J., Becker, J. F., & Hawkins, C. F. (1971). Serum biochemical values in rheumatoid disease. *Annals of the Rheumatic Diseases*, 30(2), 166–170. <https://doi.org/10.1136/ard.30.2.166>
- [13] Maiuolo, J., Oppedisano, F., Gratteri, S., et al. (2016). Regulation of uric acid metabolism and excretion. *International Journal of Cardiology*, 213(7), 8–14. <https://doi.org/10.1016/j.ijcard.2016.04.027>
- [14] Ramello, A., Vital, C., & Marangella, M. (2000). Epidemiology of nephrolithiasis. *Journal of Nephrology*, 13(Suppl. 3), 45–50.
- [15] Abdel-Halim, R. E. (2005). Urolithiasis in adults: Clinical and biochemical aspects. *Saudi Medical Journal*, 26(5), 705–713.
- [16] Dubey, A., et al. (2017). Study of serum alkaline phosphatase level in rheumatoid arthritis. *International Journal of Medical Research Professionals*, 3(3), 318–321.
- [17] Olago-Rakuomi, A., et al. (2017). Prevalence of abnormal liver function tests in rheumatoid arthritis. *African Journal of Rheumatology*, 5(2), 70–75.
- [18] Chawla, A., et al. (2020). Elevated alkaline phosphatase: The initial laboratory abnormality in an atypical presentation of Takayasu arteritis. *International Journal of Clinical Rheumatology*, 15(2), 33–36.

- [19] Isaacs, J. D., et al. (2014). Changes in serum creatinine in patients with active rheumatoid arthritis treated with tofacitinib: Results from clinical trials. *Arthritis Research & Therapy*, 16, 1–12. <https://doi.org/10.1186/ar4497>
- [20] Sunar, I., & Ataman, Ş. (2020). Serum C-reactive protein/albumin ratio in rheumatoid arthritis and its relationship with disease activity, physical function, and quality of life. *Archives of Rheumatology*, 35(2), 247–254. <https://doi.org/10.46497/ArchRheumatol.2020.7439>
- [21] Akl, N. E., et al. (2019). Role of interleukin-35 in rheumatoid arthritis pathogenesis and its relation to disease activity and joint damage. *Egyptian Rheumatology and Rehabilitation*, 46, 177–182. https://doi.org/10.4103/err.err_33_19
- [22] Kwaan, H. C. (2010). Role of plasma proteins in whole blood viscosity: A brief clinical review. *Clinical Hemorheology and Microcirculation*, 44(3), 167–176. <https://doi.org/10.3233/CH-2010-1241>
- [23] Orr, C. K., et al. (2018). The utility and limitations of CRP, ESR and DAS28-CRP in appraising disease activity in rheumatoid arthritis. *Frontiers in Medicine*, 5, 185. <https://doi.org/10.3389/fmed.2018.00185>
- [24] Marnell, L., Mold, C., & Du Clos, T. W. (2005). C-reactive protein: Ligands, receptors and role in inflammation. *Clinical Immunology*, 117(2), 104–111. <https://doi.org/10.1016/j.clim.2005.08.004>