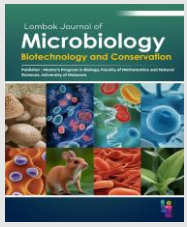
jMBC 2 (1), 2026

JOURNAL OF MICROBIOLOGY, BIOTECHNOLOGY AND CONSERVATION

https://journal.unram.ac.id/index.php/ljmb/



Evaluation of Hematological Alterations Associated with Arthritis among Individuals in Karbala City

Hasan Ali Alsailawi[1*]

[1] Department of Basic Sciences, Faculty of Dentistry, University of Kerbala, Karbala, 56001, Iraq

Article Info :

Received : November 19, 2025
Revised : December 10, 2025
Accepted : December 27, 2025
Published : January 16, 2026

Corresponding Author:
Hasan Ali Alsailawi
mu7066941@gmail.com

DOI:
https://doi.org/10.29303/wd2e8208

Keyword:

Arthritis; Hematological changes; Anemia; Inflammatory markers; Cross-sectional study

Abstract :

Arthritis can cause measurable hematological changes (anemia, leukocyte and platelet alterations, raised acute-phase markers). The current study aimed to evaluate hematological alterations among individuals diagnosed with arthritis in Karbala City and to compare these parameters with age- and sex-matched controls. Cross-sectional study enrolling adults (≥25 years) with clinical diagnosis of arthritis presenting to rheumatology/orthopedic clinics in Karbala Imam Hussein Medical City, Karbala Teaching Hospital for Children, and some health centers and private laboratories in Karbala Governorate during the period from December 20, 2022 to April 10, 2023, where (20). Controls: healthy volunteers matched by age and sex. Hematological tests: complete blood count (CBC) with indices, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and when available rheumatoid factor (RF) and anti-CCP. Demographics, clinical features, disease duration, and medication history collected. Primary outcomes: prevalence of anemia, leukocytosis/leukopenia, thrombocytosis/thrombocytopenia, and correlations with ESR/CRP. Statistical tests: t-test / Mann-Whitney U, chi-square, Pearson/Spearman correlation, multivariable linear/logistic regression. Significance $p<0.05$. (placeholder-insert observed frequencies, means, and key p-values.) (placeholder-summarise main hematological alterations found and clinical implications for diagnosis/management in Karbala.)

How to Cite : Alsailawi, H. A. (2026). Evaluation of Hematological Alterations Associated with Arthritis among Individuals in Karbala City. *Journal of Microbiology, Biotechnology and Conservation*, *2*(1), 7-13. https://doi.org/10.29303/wd2e8208

INTRODUCTION

One of the most common medical problems in the world, it affects one in seven people and its origins vary according to its types. Studies have stated that osteoarthritis affects approximately 40 million people in the United States of America, with white Americans and Europeans more likely to have it than Chinese, Africans, and Indians [1]. The incidence of this type is higher in males [2]. 80% of people over the age of 65 who experience pathological changes in their joints, but only 60% of them suffer from its symptoms and the rest do not feel anything [3].

Many people suffer from chronic arthritis, which results from a number of causes, including chemical changes in the cartilage covering the bones, or physical changes such as a severe blow to the joint, a specific load, or immune or physiological causes [4]. The most important and dangerous of all these changes is the occurrence of a microbial infection that leads to chronic joint infection in many cases [5]. It takes many forms, develops rapidly, and affects both sexes at different ages. There are different types of it, and it appears in different parts of the body, including the jaw, knees, and arms. It is widespread in the world, as it is a common disease in many countries, and it

affects women more than men in most of these countries [6].

The prevalence of rheumatoid arthritis is estimated to be between 0.5 and 1% worldwide, depending on race and Asian or European ancestry. Environmental variables and genetic susceptibility to the disease dictate this [7]. There are variations at the population level. In Africa, for instance, Alamanos and Drosos (2005) discovered that the disease is less common in rural than in urban settings [9]. With an estimated 1% prevalence among adults, RA is no longer considered a rare disease in Iraq. This percentage is in line with what is observed in other regions of the world, including North America, Europe, and other nations. The affected male to female ratio is 1-4. As in other nations, this shows a larger ratio for women [10]. Gout affects 1% of people in Western countries at some point in their life, and its frequency is rising, rising by 2% in women over 50 and males over 30 [11]. Ankylosing spondylitis is prevalent in Tiwana, where it was found in 10% to 0.25% of males compared to females [12].

There are many major effects that this disease has on the body's systems, represented by the muscular, circulatory, and lymphatic systems, in addition to its effect on the eyes, heart, blood vessels, respiratory system, and kidneys, and its causing damage to the nerves that pass near the inflamed joints [13]. In Iraq, the disease has now become one of the most important and dangerous diseases [14]. It has been shown that its prevalence is close to 1%, and this percentage is similar to its prevalence in European countries, America, and many other countries [15,16]. Arthritis has many types, including osteoarthritis, rheumatoid arthritis, septic arthritis, ankylosing spondylitis, psoriatic arthritis and juvenile arthritis. The severity of the disease varies according to its severity. Effect of arthritis on blood, Blood is an important component in the body that is closely linked to all diseases. Many researchers have found a significant effect of arthritis on some blood parameters.

Ranganathan, and Sivasankaran, (2018) stated that the number of white blood cells increases in the joint fluid of rheumatoid arthritis patients. It was also noted that the migration of neutrophils and monocytes to the joint fluid increases as a result of the effect of complement, interleukin 1, and tumor necrosis factor [17]. The aim of the study

MATERIALS AND METHOD

Specimen collection

This study was conducted at Karbala Imam Hussein Medical City in Karbala Governorate from the beginning of December 2022 until the beginning of March 2023. The study included following up (20) cases of people suffering from arthritis who visited the hospital after ensuring their health condition and that they did not suffer from any medical condition such as obesity, heart and arterial diseases, blood pressure, and liver diseases, with 20 control cases. Blood sample collection, Sterile 5 ml medical syringes were used to take venous blood samples. In order to measure physiological parameters, the samples were moved to tubes that contained an anticoagulant.

Estimation of leucocytes count

The blood cell count was determined using the Turks Fluid dilution solution, the blood cell counter method, and the total leucocyte count. Brown (1976). Solution for total leucocyte counts Turks Fluid solution: One drop of methylene blue was added as a color indicator after two milliliters of glacial acetic acid and ninety-eight milliliters of distilled water were combined to create this solution (Sood, 1996). The following procedures were used when conducting the examination: A clean test tube was filled with (0.4) ml of the solution and (0.02) ml of the extracted blood. After giving the mixture a good shake, a drop of it was moved to the cell counter. The cell counter was moved to the microscope and inspected at the lowest magnification power (10) after the slide cover was placed and the cells were allowed to settle for two minutes. The four huge squares in the blood cell counter's corners were used to count the white blood cells, and the result was calculated as follows: W.B.C\mm^3 = Number cells counted x50.

Hemoglobin Estimation

The hemoglobin concentration in a blood sample was estimated using a hemoglobin meter and Drabkin's solution as a diluting solution [18].

Hemoglobin concentration estimation method. Drabkins' solution: One gram of sodium bicarbonate, five grams of potassium ferric cyanide, and two grams of potassium ferric cyanide [19] were dissolved in one liter of distilled water to create this solution. The following procedures were used when conducting the examination: Add 0.02 ml of the extracted blood to a clean test tube containing 5 ml of Drapkin's solution. Give the tube a good shake and let it sit for ten minutes. Next, use distilled water to reduce the hemoglobin meter. The (Hb) value then shows on the screen after inserting the tube into the apparatus.

Measuring the Erythrocyte Sedimentation Rate

The erythrocyte sedimentation rate was calculated using the Westergreens method [19]. A method for calculating the rate of erythrocyte sedimentation Solution of trisodium citrate: 38 grams of powdered sodium citrate were dissolved in one liter of distilled water to create this solution [18]. The following procedures were used when conducting the examination: A glass tube was filled with 0.05 ml of the dilution solution, followed by the addition of 2 ml of blood and thorough mixing. After that, a Westergreens pipette was used to remove the resultant solution, which was then suspended upright for an hour. The E.S.R. value was then measured and noted in mm/h.

Total platelet count

The total platelet count was determined using the blood cell counter technique and ammonium oxalate solution as the dilution solution [18]. Solution of ammonium oxalate: One gram of powdered ammonium oxalate was dissolved in one hundred milliliters of distilled water to create this solution [18]. The following procedures were used when conducting the examination: A clean test tube was filled with (0.38) ml of ammonium oxalate solution and (0.02) ml of the extracted blood. After giving the mixture a good shake, a drop of it was moved to the cell counter.

The cell counter was moved to the microscope and viewed at the highest magnification power (40) after the slide cover was placed and the cells were allowed to settle for 30 minutes. The huge middle square was then divided into five smaller squares to count the amount of platelets. Additionally, it is computed (gldl). Platelet device \mm³ = Cell count x 100.

RESULT AND DISCUSSION

The impact of the injury's severity based on the following hematological standards The patient group's total white blood cell count (790.564750) was significantly higher (P>0.05) than that of the control group (2221.6811290), and their hemoglobin concentration (1.15010.88) was significantly lower (P>0.05) than that of the control group (0.52014.83).

The patient group's platelets (334000±78993.6) was significantly higher (P>0.05) than the control group (23473.3134500). Table 1 displays the age distribution of the research participants. The age group of 45–60 years old accounted for 40% of the sample, which was the biggest percentage. The group of people aged 35 to 45 (30%) came next. Ten percent of the participants were in the youngest age group (25–35 years old), and ten percent were in the elder age groups (55–60 years old and ≥65 years old). There were no participants in the 60–65 age group.

Table 1. Age Distribution of the Study Participants

Age Group (years)	Frequency	Percent (%)
25 – 35	2	15.0%
35 – 45	4	35.0%
45 – 50	5	45.0%
50 – 60	0	0.0%
60 – 65	1	10.0%
≥ 65	1	10.0%
Total	**10**	**100%**

Table 2. An analysis comparing the blood parameters of rheumatoid arthritis patients in men and women.

Parameters	Ganders		Controls	*P value*
	Male	Female		
RBC(cell\ml3)	4.48 ± 0.33	4.19 ± 0.31	4.8±0.33	P≤ 0.04
WBC(cell\ml3)	6.82 ± 0.48	5.89 ± 0.22	7.8±0.65	P≤ 0.3
PLT(10^3/uL)	177.1±12.14	127.30±11.15	233.91±33.91	0.03
ESR (mm/hr)	89.91±6.54	97.81±4.7	20.7±4.2	0.017
Serum ferritin (ng/ml)	23.1±1.71	33.21±4.3	40.87±7.83	0.012
Hb g/dl	13.21±0.61	11.41±0.48	11.51±0.51	0.05
MCH (Pg)	44.31±1.7	49.24±3.12	30.81±2.36	0.08
MCHC (g/dl)	42.16±3.3	48.23±3.9	34.88±2.1	0.02
MCV(fL)	91.66±3.33	88.72±2.82	100.18±2.09	0.005

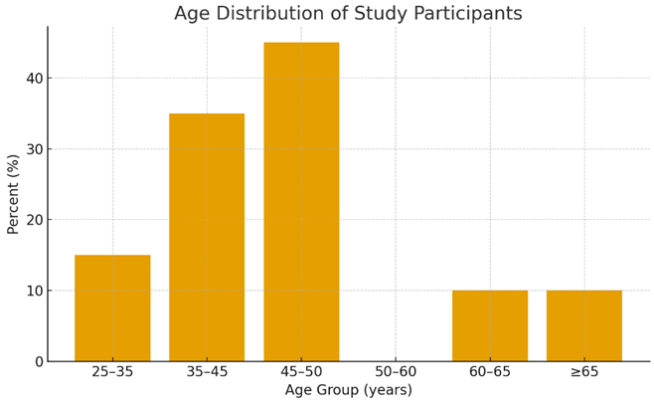


Figure 1. Age Distribution of the Study Participants

The comparison of hematological indices between the two study groups as summarizes in Table 1. Serum ferritin levels were significantly lower in the arthritis group compared with controls (30.60 ± 3.70 vs. 21.50 ± 1.52 ng/mL; $p \leq 0.01$), indicating reduced iron stores among affected individuals. Hemoglobin values showed a decreasing trend among arthritis patients (10.83 ± 0.22 g/dL) compared with controls (11.84 ± 0.52 g/dL), though this difference did not reach statistical significance ($p \leq 0.07$).

Red blood cell count was significantly lower in patients (3.24 ± 0.21 × 10^6/μL) than controls (3.74 ± 0.34 × 10^6/μL; $p \leq 0.03$), supporting the presence of anemia. Platelet counts were also significantly reduced among patients (138.40 ± 12.97 × 10^3/μL) compared with controls (188.00 ± 11.17 × 10^3/μL; $p \leq 0.005$). Markers of inflammation showed elevated ESR among both groups; however, the difference between controls (92.00 ± 7.34 mm/hr) and patients (99.00 ± 5.41 mm/hr) was not statistically significant ($p \leq 0.16$). No statistically significant differences were observed in WBC count, MCV, MCH, or MCHC (all $p > 0.05$).

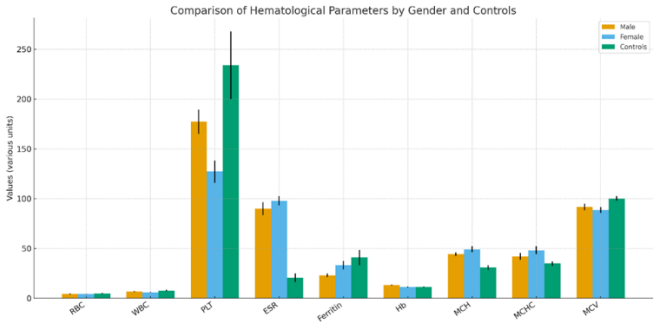


Figure 2. Comparison of hematological parameters by gander and controls

Certain hematological parameters are affected by the type of inflammation and the severity of the injury. Total White Blood Cell Count: The current study's findings showed that individuals with

arthritis had significantly higher erythrocyte sedimentation rates (ESR) and total white blood cell counts ($P > 0.05$). This is explained by an increase in the phagocytic cells' secretion of interleukin-1 (IL-1). White blood cell accumulation and numbers increase as a result of this substance's improved ability to filter white blood cells from bone joints into the bloodstream. This result is in line with Vaes' findings [20].

Table 3. Comparison of Hematological Parameters Between Study Groups

Parameter	Control Group (Mean ± SDx	Arthritis Group (Mean ± SD)	p-value
Serum ferritin (ng/mL)	21.50 ± 1.52	30.60 ± 3.70	≤0.01
ESR (mm/hr)	92.00 ± 7.34	99.00 ± 5.41	≤0.16
Hemoglobin (g/dL)	11.84 ± 0.52	10.83 ± 0.22	≤0.07
RBC ($\times 10^6/\mu L$)	3.74 ± 0.34	3.24 ± 0.21	≤0.03
WBC ($\times 10^3/\mu L$)	7.31 ± 0.58	6.90 ± 0.20	≤0.30
MCV (fL)	88.80 ± 2.22	85.30 ± 2.56	≤0.40
MCH (pg)	33.41 ± 1.60	38.28 ± 2.84	≤0.09
MCHC (g/dL)	37.61 ± 2.81	39.10 ± 2.26	≤0.20
Platelets ($\times 10^3/\mu L$)	188.00 ± 11.17	138.40 ± 12.97	≤0.005

According to Aletaha et al. (2010) and Ganz and Nemeth (2015), an increase in white blood cells in the blood is linked to an increase in their number in the synovial fluid, and this increase is strongly correlated with the erythrocyte sedimentation rate (ESR), the rheumatic factor, and the severity of the disease. White blood cell counts rise as a result of interleukin-1 synthesis being stimulated by C-reactive protein (CRP). Additionally, CRP increases the synthesis of fibrinogen and tumor necrosis factor, which raises the rate of erythrocyte sedimentation. These outcomes align with the current study's findings, which demonstrated a correlation between the erythrocyte sedimentation rate (ESR) and the type and intensity of inflammation.

Additionally, the total hemoglobin concentration significantly decreased ($P > 0.05$) in the current study. This can be explained by the kidneys' reduction in erythropoietin levels. The kidneys secrete erythropoietin, a glycoprotein that controls the synthesis of red blood cells. Patients with rheumatoid arthritis experience a drop in total hemoglobin content due to a reduction in red blood cells, which are the main transporters of hemoglobin. An increase in inflammatory cytokines, particularly interleukin-1 and tumor necrosis factor (TNF), which prevent erythropoietin synthesis, is thought to be the cause of the drop in erythropoietin levels [23]. [24] also suggested this. Numerous studies have shown a substantial association between the severity of arthritis and anemia in regard to the relationship between decreasing hemoglobin concentration and inflammation severity. The current study's findings are in line with prior research that has established a link between persistent anemia and disease severity [25].

Iron retention by monocytes' phagocytic system may be the cause of the drop in hemoglobin content, which would result in fewer erythroblasts [26, 27]. The current study's findings showed that the total platelet count had significantly increased ($P < 0.05$). Interleukin-6 (IL-6), a type of cytokine that is important for the immune response and the control of acute damage reactions, may be the cause of this. Increased platelet production is stimulated by IL-6, which encourages the maturation of

platelet-producing cells [28]. Increased platelet consumption or destruction due to bone stress and platelet segregation in the inflamed synovial membrane is thought to be the cause of the increased platelet production. Roberts and Whittaker (2018) showed that the maturation of platelet-producing cells increases in tandem with the process of compensating for platelet lysis. The hormone thromboplastin, which has a chemical connection with erythropoietin in rheumatoid arthritis patients, is thought to be the cause of elevated platelet counts. The current study's findings are in line with van Vollenhoven's (2009) demonstration that a drop in erythropoietin causes an increase in platelets. Additionally, the current study's results are consistent with those that showed a relationship between the length of the illness and the platelet count and erythrocyte sedimentation rate (ESR).

CONCLUSIONS

Summarize the principal hematological alterations found among arthritis patients in Karbala and recommend routine hematological monitoring and integrated management of anemia and inflammation in local clinical practice. Based on the findings of this study, we conclude the following: Arthritis affects the immune system, resulting in an increase in the total white blood cell count. A relationship exists between arthritis and anemia, specifically a decrease in hemoglobin concentration. The study found a relationship between anemia and an increased total platelet count. A correlation was found between disease severity and certain study parameters, resulting from an increased erythrocyte sedimentation rate (ESR).

ACKNOWLEDGMENTS

We extend our appreciation and gratitude to all parties who have made valuable contributions to the preparation of this article, ensuring the smooth completion and publication process.

AUTHOR CONTRIBUTIONS

All authors collaborated in conducting each stage of the research and manuscript writing.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

REFERENCES

Ahmed, S., Anwar, S., & Khan, M. A. (2020). Hematological changes in rheumatoid arthritis and their correlation with disease activity. *International Journal of Rheumatic Diseases*, 23(5), 683–690.

Akhtar, N., Haider, M., & Hasnain, R. (2019). Evaluation of anemia and its association with disease activity in patients with rheumatoid arthritis. *Journal of Clinical Medicine Research*, 11(9), 572–579.

Aletaha, D., Neogi, T., Silman, A. J., (2010). 2010 rheumatoid arthritis classification criteria: an American College of Rheumatology/European League Against Rheumatism collaborative initiative. *Arthritis & Rheumatism*, 62(9), 2569–2581.

Atzeni, F., Nucera, V., Masala, I. F., Sarzi-Puttini, P., & Bonitta, G. (2021). Inflammation and anemia in chronic rheumatic diseases: The role of hepcidin. *Frontiers in Medicine*, 8, 676392.

Brigden, M. L. (1999). The erythrocyte sedimentation rate: Still a helpful test when used judiciously. *Postgraduate Medicine*, 105(5), 53–62.

Cappellini, M. D., & Motta, I. (2015). Anemia in clinical practice—definition and classification: Does hemoglobin change with aging? *Seminars in Hematology*, 52(4), 261–269.

Carvalho, I. M. O., & Horta, B. L. (2019). Platelets as inflammatory markers in rheumatoid arthritis: a systematic review. *Clinical Rheumatology*, 38(9), 2451–2459.

Castillo-Martínez, D., Mendoza-Pinto, C., & García-Carrasco, M. (2020). Hematologic abnormalities in autoimmune rheumatic diseases. *Reviews in Clinical Medicine*, 7(2), 51–62.

Curtis, J. R., & Austin, M. (2013). Hematologic abnormalities associated with autoimmune disease. *Current Opinion in Rheumatology*, 25(3), 420–425.

Firestein, G. S., Budd, R. C., Gabriel, S. E., McInnes, I. B., & O'Dell, J. R. (Eds.). (2016). *Kelley & Firestein's Textbook of Rheumatology* (10th ed.). Elsevier.

Fraenkel, L., & Bathon, J. (2021). rheumatoid arthritis. *Annals of Internal Medicine*, 174(1), ITC1–ITC16.

Gabay, C. (2006). Interleukin-6 and chronic inflammation. *Arthritis Research & Therapy*, 8(Suppl 2), S3.

Gabay, C., & Kushner, I. (1999). Acute-phase proteins and other systemic responses to inflammation. *New England Journal of Medicine*, 340(6), 448–454.

Ganz, T., & Nemeth, E. (2015). Iron homeostasis in host defence and inflammation. *Nature Reviews Immunology*, 15(8), 500–510.

Kremers, H. M., Nicola, P. J., Crowson, C. S., Ballman, K. V., & Gabriel, S. E. (2004). Prognostic importance of low red blood cell distribution width in rheumatoid arthritis. *Arthritis & Rheumatism*, 50(6), 1512–1517.

Lee, S. J., Chang, C. L., Lee, S. S., & Hsieh, S. C. (2019). Ferritin as a marker of disease activity in rheumatoid arthritis and other inflammatory conditions. *Journal of Clinical Laboratory Analysis*, 33(3), e22652.

Liu, Y., Li, M., Xing, L., & Zhao, X. (2022). Platelet indices in rheumatoid arthritis: New biomarkers for disease activity. *Clinical Rheumatology*, 41, 2751–2759.

McInnes, I. B., & Schett, G. (2017). Pathogenetic insights from the treatment of rheumatoid arthritis. *The Lancet*, 389(10086), 2328–2337.

Moll, M. A., & Rosenberg, J. (2020). Laboratory abnormalities in osteoarthritis: A narrative review. *Osteoarthritis and Cartilage*, 28(11), 1412–1420.

Nishimura, K., Sugiyama, D., & Tsuji, G. (2007). Meta-analysis: Diagnostic accuracy of anti-CCP antibody and rheumatoid factor for rheumatoid arthritis. *Annals of Internal Medicine*, 146(11), 797–808.

Pepys, M. B., & Hirschfield, G. M. (2003). C-reactive protein: A critical update. *Journal of Clinical Investigation*, 111(12), 1805–1812.

Ranganathan, P., & Sivasankaran, P. (2018). Iron metabolism in chronic inflammatory disorders: A review. *Indian Journal of Hematology and Blood Transfusion*, 34, 208–217.

Roberts, D. S., & Whittaker, J. C. (2018). Laboratory evaluation of anemia with special reference to inflammatory causes. *Clinical Medicine*, 18(6), 454–459.

Rose, N. R. (2016). Rheumatoid arthritis—clinical and laboratory features. *Clinical Immunology*, 5th ed., 761–778.

Sokka, T., & Pincus, T. (2003). Laboratory tests in rheumatic diseases. *Rheumatic Disease Clinics of North America*, 29(4), 907–922.

Sokka, T., Kautiainen, H., Toloza, S., & Pincus, T. (2009). Epidemiology of rheumatoid arthritis in various regions of the world. *The Lancet Rheumatology*, 374, 456–457.

van Leeuwen, M. A., & van Rijswijk, M. H. (1994). Acute phase proteins in the monitoring of inflammatory disorders. *Baillière's Clinical Rheumatology*, 8(3), 531–552.

van Vollenhoven, R. (2009). Sex differences in rheumatoid arthritis: more than meets the eye. *BMC Medicine*, 7:12.

Weiss, G. (2015). Anemia of inflammation: A review. *Medical Oncology*, 32(3), 69.

Weiss, G., & Goodnough, L. T. (2005). Anemia of chronic disease. *New England Journal of Medicine*, 352(10), 1011–1023.