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Evaluation of Complete Blood Count in Symptomatic Rheumatoid Arthritis Patients in Sana'a City, Yemen

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Abstract

Background: Rheumatoid arthritis is a chronic inflammatory autoimmune disorder that can affects several organs and systems in human body. It is frequently associated with hematological alterations that reflect disease activity. Data on complete blood count variations among patients with rheumatoid arthritis in Sana' a, Yemen, are limited.
Objectives: This study aimed to evaluate complete blood count variations in symptomatic rheumatoid arthritis patients in Sana'a city, Yemen.
Patients and methods: A comparative cross-sectional study was conducted on 200 participants, including 100 RA patients and 100 healthy controls. The CBC parameters were analyzed using an automated hematology analyzer. ACCP antibodies were measured using a chemiluminescence immunoassay, while RF and CRP were measured using spectrophotometric turbidity and nephelometry methods. The ESR was determined using the Westergren method. Statistical analyses were performed using SPSS version 26.
Results: RA patients showed significantly lower Hb, HCT, MCV, MCH, and MCHC, with significantly higher RDW compared with healthy controls ($p < 0.05$). RBC and total WBC counts were not significantly different between the groups. Neutrophils, monocytes, eosinophils, basophils, platelet count, PCT, NLR, MLR, and PLR were significantly increased, whereas lymphocytes and MPV were significantly decreased in patients with RA ($p < 0.05$). ACCP, RF, CRP, and ESR levels were significantly higher in patients with RA than in controls ($p = 0.001$).
Conclusion: Significant CBC alterations are common in RA patients and may serve as accessible markers of inflammation and disease activity. The most prominent findings were anemia-related changes, thrombocytosis, elevated inflammatory cell ratios, and reduced MPV.

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Keywords:
Rheumatoid arthritis, complete blood count, hematological abnormalities, Sana'a, Yemen

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1. INTRODUCTION

Rheumatoid arthritis (RA) is a chronic systemic autoimmune condition characterized by chronic inflammation of the synovial membrane, resulting in progressive joint damage and functional impairment [1]. Inflammation is a core pathological process in RA and a key driver of the disease activity. This inflammatory response is not static but fluctuates over time, directly affecting disease progression. Therefore, timely and effective control of inflammation is crucial for achieving optimal therapeutic outcomes [2].

In RA, a variety of pro-inflammatory cytokines, such as interleukin-1 (IL-1), IL-15, IL-18, IL-6, and tumor necrosis factor-alpha (TNF-α), drive the inflammatory cascade and are responsible for classic symptoms, including redness, swelling, pain, and warmth in the affected joints. Conversely, cytokines such as IL-10 and IL-4 play anti-inflammatory roles and contribute to immune regulation [3]. The secretion of pro-inflammatory cytokines also leads to the development of inflammatory edema, which further exacerbates the joint stiffness frequently experi-

enced by individuals with RA [4].

In clinical practice, several biomarkers are commonly used to assess disease activity in RA, including complete blood count (CBC) and conventional inflammatory indicators such as rheumatoid factor (RF), anti-cyclic citrullinated peptide (ACCP) antibodies, C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR) [5].

The CBC is a straightforward, cost-effective, and relatively sensitive indicator of inflammation in clinical settings. This is because the interplay among white blood cells (WBCs), red blood cells (RBCs), and platelets, especially the roles of neutrophils, lymphocytes, and platelets as key immune effectors, is essential for modulating inflammatory and immune responses. Consequently, variations in peripheral blood levels can present clinically as anemia, leukocytosis, or thrombocytopenia [6].

The mechanism of anemia in rheumatoid arthritis involves inflammatory cytokines, particularly interleukin-6, which induces hepcidin production, thereby impairing iron metabolism and erythropoiesis [7].

There have been no previous studies on CBC variations in patients with symptomatic RA in Yemen. Conducting this study is crucial as it will provide localized data on the hematological profiles of these patients, which can enhance the understanding of disease activity and guide clinical management. This study aimed to evaluate the hematological abnormalities in patients with RA attending private diagnostic centers providing diagnostic and treatment services for patients with RA in Sana'a, Yemen.

2. SUBJECTS AND METHODS

This comparative cross-sectional study was conducted at private diagnostic centers in Sana'a, Yemen, which provide diagnostic and therapeutic services for patients with RA. The four specific centers involved were the Science and Technology Teaching Hospital Laboratory Department, New Scan Medical Laboratories, One Lab Medical Laboratory, and I Lab Medical Laboratory. The study enrolled 200 participants, including both males and females aged 15–70 years. The sample consisted of 100 individuals with symptomatic RA and 100 apparently healthy controls. The timeline for sample collection was from September 2024 to September 2025.

The case group included patients with a prior confirmed diagnosis of Rheumatoid Arthritis (RA) and who met the specified age range, with no sex restrictions. The control group comprised healthy individuals of similar age with no history of acute or chronic illnesses. The exclusion criteria were as follows: RA patients undergoing methotrexate treatment, individuals with hematological disorders or any condition known to influence CBC parameters, and participants receiving antibiotics or chemotherapy.

A standardized questionnaire was used to gather sociodemographic information, such as age, sex, marital status, and place of residence. From each participant, 5 mL of venous blood was collected aseptically and allocated into labeled tubes:K_3EDTA tubes for CBC, trisodium citrate Westergren tubes for erythrocyte sedimentation rate (ESR) testing, and plain gel separator tubes for serum isolation to measure RF, CRP and ACCP antibodies. Serum samples were stored at -20℃ for repeat analyses or further testing.

For diagnosis, these parameters, RF and CRP, were measured using the spectrophotometric turbidity and nephelometry method on a BECKMAN AU 480 chemistry autoanalyzer (USA), while ACCP antibodies were assessed using a chemiluminescence immunoassay (CLIA) on the ARCHITECT i1000 system (Abbott, USA). CBC parameters were analyzed using a MINDRAY five-part automated hematology analyzer (Model BC-5800, China), and ESR was determined using the one-hour Westergren method.

Data analysis was performed using SPSS software (version 26; LEAD Technologies Inc., USA). This study received ethical clearance from the Committee of Postgraduate Studies and Scientific Research at the Faculty of Medicine and Health Sciences, Sana'a University.

3. RESULTS

The study included 200 participants, comprising 100 patients diagnosed with rheumatoid arthritis (RA) and 100 apparently healthy controls. The mean age of the RA patients was 44.9 ± 14.1 years, which was similar to that of the control group (45.4 ± 14.5 years), with no significant difference observed (p = 0.831). In terms of sex distribution, females predominated in both the RA (88.0%) and control groups (87.0%), while males represented 12.1% and 13.1% of participants, respectively. There was no statistically significant difference in sex distribution between the two groups (p = 0.831).

Regarding place of residence, a significantly larger proportion of patients with RA lived in urban areas (73.0 %) than those in the control group (43.0 %), while rural residence was more common among healthy controls (57.0 %) than among patients with RA (27.0%). This difference was statistically significant (p = 0.001). In terms of marital status, most participants in both groups were married (67.0% of patients with RA and 73.0% of controls). The distribution of single, divorced, and widowed participants was similar between the groups, with no statistically significant difference observed (p = 0.438) (Table 1).

Patients with RA had significantly lower levels of hemoglobin (Hb), hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC) than healthy controls (P = 0.05). Conversely, red cell distribution width (RDW) indices, including RDW-CV and RDW-SD, were significantly elevated in patients

Table 1. Demographic features of the study participants

Parameters	RA(n=100)	Healthy controls(n=100)	*p*. value
Age (Mean± SD)	44.9±14.0	45.4±14.5	0.831
Sex n(%) Male Female	 12(12.0%) 88(88.0%)	 13(13.0%) 87(87.0%)	0.831
Residence n(%) Rural Urban	 27(27.0%) 73(73.0%)	 57(57.0%) 43(43.0%)	0.001*
Marital status n(%) Single Married Divorce Widow	 17(17.0%) 67(67.0%) 8(8.0%) 8(8.0%)	 15(15.0%) 73(73.0%) 3(3.0%) 9(9.0%)	0.438

Comparisons were conducted using the independent sample t-test and Chi-square test. Data are presented as mean ± standard deviation (SD) or frequency and percentage. A p-value < 0.05 was considered statistically significant, whereas a p-value >0.05 was considered not significant.

with RA compared to those in the control group (P = 0.001). No significant difference was observed in red blood cell (RBC) counts between the two groups (P = 0.802). Total white blood cell (WBC) counts did not differ significantly between patients with RA and controls (P = 0.261). However, the percentage of neutrophils was significantly higher in patients with RA (P = 0.049), whereas lymphocyte percentages were significantly lower than those in healthy individuals (P = 0.001). Monocyte percentages were also elevated in patients with RA (P = 0.007), and eosinophil and basophil percentages were significantly higher than those in controls (P = 0.001).

Additionally, patients with RA showed significantly higher platelet count (P = 0.001) and plateletcrit (PCT) levels (P = 0.011), whereas mean platelet volume (MPV) was significantly lower than that in the control group (P = 0.004). No significant differences were observed between the groups in terms of platelet distribution width (PDW) or platelet large cell ratio (P-LCR) (P > 0.05). Furthermore, the neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), and platelet-to-lymphocyte ratio (PLR) were significantly higher in patients with RA than in healthy controls (P = 0.001). (Table 2).

Patients with RA exhibited significantly elevated median levels of ACCP, RF, CRP, and ESR compared to healthy controls (P = 0.001) (Table 3).

4. DISCUSSION

Inflammatory processes in rheumatic diseases result in alterations in peripheral blood cell counts, morphology, and cell size, making blood cell indices valuable indicators of inflammation and markers of disease activity [8]. In the present study, the mean age of the RA patients was slightly lower than that of the healthy controls, with no statistically significant difference observed (p = 0.831). This finding is consistent with the systematic review and meta-analysis by Rudan et al. [9], who analyzed 60 cohorts from low- and middle-income countries and found no significant effect of mean age on the reported RA prevalence across different populations (P = 0.0599, P > 0.05). The authors concluded that differences in age structure between the study samples were not the main determinant of the observed heterogeneity in RA prevalence. In contrast to this observation, Kaçar et al. [10] reported that the mean age of patients with RA was significantly greater than that of non-RA participants, with a p-value of < 0.001. This discrepancy suggests that the absence of an age difference in the present study is likely due to the age-matched design of the control group rather than a true lack of association between age and RA.

In the present study, there was a strong female predominance in the RA group, while the control group also had a high proportion of women, with no statistically significant difference between the two groups (p = 0.831). This finding is consistent with that of Kaçar et al. [10], who found that among 12 patients with RA in their Antalya-based cross-sectional study, 11 were female and only one was male. Furthermore, a systematic review by Rudan et al. [9] demonstrated that the prevalence of female RA in low- and middle-income countries was 0.75% (95% CI: 0.60–0.90%), while that of males was 0.16% (95% CI: 0.11–0.20%), with a highly significant difference (P < 0.0001). The strong female predominance in RA may be attributed to hormonal factors, pregnancy-related immune modulation, X-chromosome inactivation patterns, and gene dose effects [11, 12].

Table 2. Comparison of CBC Parameters between RA Patients and Healthy Individuals

Parameters	RA (n=100)		Healthy controls (n=100)		*p. value*
	Mean± SD	Min–Max	Mean± SD	Min–Max	
Hb (g/dl)	13.0±1.4	9.15-17.0	13.8±1.2	12.0-17.9	0.001*
RBCs count ($\times 10^6 \mu$L)	4.9±0.6	3.46-6.5	4.9±0.5	3.85-6.19	0.802
HCT (%)	39.8±4.3	29.7-51.1	40.9±3.8	34.9-55.0	0.038*
MCV (fL)	80.1±6.8	61.5-96.7	83.4±5.1	72.0-97.4	0.001*
MCH (pg)	26.0±2.7	18.3-32.4	28.1±2.4	22.0-35.3	0.001*
MCHC (g/dl)	32.4±1.2	28.9-34.8	33.4±1.4	30.0-37.0	0.001*
RDW-CV (%)	14.5±1.9	11.3-23.0	13.4±1.2	11.0–18.7	0.001*
RDW-SD (fL)	37.4±4.5	28.7-48.6	34.8±3.5	29.0-47.1	0.001*
WBCs count ($\times 10^3 \mu$L)	7.2±2.5	3.35-17.2	6.8±1.8	3.4-10.8	0.261
Neutrophils %	54.7±12.6	22.6-87.4	51.6±9.4	25.9-70.6	0.049*
Lymphocyte %	33.8±10.2	10.2-61.0	39.1±8.7	15.9-61.2	0.001*
Monocytes %	7.5±2.9	0.94-17.8	6.4±2.6	1.1-14.2	0.007*
Eosinophils %	3.9±3.1	0.0-14.2	2.6±1.7	0.0-11.1	0.001*
Basophils %	0.2±0.2	0.0-0.90	0.005±0.02	0.0-0.10	0.001*
Platelets-count ($\times 10^3 \mu$L)	385.9±131.9	157.0-977.0	313.6±79.4	175.0-536.0	0.001*
MPV (fL)	8.5±0.7	6.2-10.5	8.7±0.6	7.6-10.0	0.004*
PCT (%)	0.31±0.10	0.15-0.64	0.28±0.09	0.11-0.80	0. 010*
PDW (%)	10.5±2.4	6.90-18.8	10.3±2.1	7.8-17.0	0.682
P-LCR (%)	21.7±6.3	9.8-40.6	22.5±5.8	12.0-36.6	0.374
NLR	1.9±1.3	0.40-8.17	1.5±0.6	0.45-4.44	0.001*
MLR	0.24±0.1	0.04-1.0	0.17±0.1	0.04-0.47	0.001*
PLR	188.4±95.6	81.73-623.94	130.1±52.7	43.09-374.73	0.001*

Comparisons were made using the independent sample t-test, with data presented as mean ± SD. A p-value < 0.05 was considered statistically significant, while $p > 0.05$ was considered not significant. Abbreviations used include: Hb (hemoglobin), RBCs (red blood cells), HCT (hematocrit), MCV (mean corpuscular volume), MCH (mean corpuscular hemoglobin), MCHC (mean corpuscular hemoglobin concentration), RDW-CV (red cell distribution width–coefficient of variation), RDW-SD (red cell distribution width–standard deviation), WBCs (white blood cells), MPV (mean platelet volume), PCT (plateletcrit), PDW (platelet distribution width), P-LCR (platelet–large cell ratio), NLR (neutrophil to lymphocyte ratio), MLR (monocyte to lymphocyte ratio), and PLR (platelet to lymphocyte ratio).

Table 3. Comparison of ACCP, RF, CRP and ESR between RA Patients and Healthy Individuals

Parameters	RA Median (Min - Max)	Healthy controls Median (Min - Max)	*p*. value
ACCP	200(6.5-680)	0.5(0.5-5)	0.001*
RF	77.1(3-1389)	4.5(0.5-13.1)	0.001*
CRP	8.1(0.4-153)	2.5(0.2-20)	0.001*
ESR	32.5(5-158)	11(3-37)	0.001*

Comparisons were conducted using the Mann-Whitney test, with data presented as median and range. Statistical significance was set at $p < 0.05$. The abbreviations used include Min (minimum), Max (maximum), ACCP (anti-cyclic citrullinated peptide), RF (rheumatoid factor), CRP (C-reactive protein), and ESR (erythrocyte sedimentation rate).

In the present study, a significantly larger proportion of patients with RA lived in urban areas than healthy controls, whereas rural residence was more common among healthy controls than patients with RA. This difference was statistically significant (P = 0.001). This finding is consistent with Deseatnicova et al. [12], who suggested in their review that

Urbanization may be associated with increased exposure to risk factors, such as pollution and unhealthy dietary habits. In contrast to these observations, a systematic review and meta-analysis by Rudan et al. [9] examined the difference in RA prevalence between urban and rural populations and found no significant difference (P = 0.353). The authors concluded that urban dwelling does not contribute significantly to disease development in low- and middle-income countries. This disagreement suggests that the urban predominance observed in the present study may be explained by ascertainment bias, as urban residents generally have better access to rheumatology services and diagnostic facilities than a true biological increase in RA risk among urban dwellers.

In the present study, no significant difference in marital status distribution was observed between patients with RA and healthy controls (p = 0.438), with most participants in both groups being married. This finding aligns with Ranjbaran and Kazemi [13], who reported no significant correlation between marital status and RA (p = 0.39). From a pathophysiological perspective, marital status is not a causal or protective determinant of RA but rather a prognostic factor influencing disease outcomes and not disease onset [13].

In the present study, Hb levels were significantly lower in patients with RA than in healthy controls ($p = 0.001$). This finding is consistent with that of Farouk et al.[8], who reported reduced Hb levels among patients with RA, reflecting the high prevalence of anemia in chronic inflammatory diseases. The reduction in Hb levels may be attributed to anemia of chronic disease, iron sequestration, impaired erythropoietin response, and the suppressive effects of pro-inflammatory cytokines, such as IL-6 and TNF-α on erythropoiesis [14, 15]. A similar observation was reported by Tecer et al. [16], confirming that low Hb levels are a common hematological manifestation of RA. The RBCs count showed no statistically significant difference between patients with RA and healthy individuals in the current study ($p = 0.802$). This finding is consistent with those of previous studies [17, 18]. Farouk et al. [8] and Al-Yasiri [19] observed a significantly lower RBC count in patients with RA. The absence of a significant difference in RBC count in this study may be explained by the exclusion of patients with hematological disorders or those receiving methotrexate and chemotherapy, which may preserve RBC production despite reductions in hemoglobin and RBC indices. In addition, the relatively small sample size may have reduced the ability to detect a significant difference in RBC counts between patients with RA and healthy controls.

In the present study, HCT levels were significantly lower in patients with RA than in controls ($p = 0.038$). This finding supports the results of Farouk et al. [8] and further reflects the reduced red cell mass and impaired erythropoiesis in RA. HCT decrease is commonly associated with chronic inflammation and anemia of chronic disease, both of which are frequent complications of RA[20].

In the current study, patients with RA exhibited significantly lower RBC indices, including MCV, MCH, and MCHC, than healthy controls ($p = 0.001$). These findings are consistent with those reported by Farouk et al.[8] and indicate a tendency toward microcytic or normocytic hypochromic anemia in patients with RAA. The observed reductions in MCV, MCH, and MCHC reflect impaired erythropoiesis and reduced Hb synthesis, which are commonly associated with chronic inflammatory diseases. This pattern is likely mediated by inflammation-induced disturbances in iron metabolism, particularly increased hepcidin levels, leading to restricted iron availability and inhibition of erythrocyte maturation [14, 21].

In the present study, RDW was significantly higher in patients with RA than in healthy controls ($p = 0.001$). This finding is in strong agreement with that of Farouk et

al.[8], who reported a significant elevation in RDW% in patients with RA. Similar observations were documented by Tecer et al. [16], Al-Rawi et al. [22] and Lin et al. [23]. Elevated RDW reflects increased anisocytosis and may result from ineffective erythropoiesis, disturbances in iron metabolism, and chronic systemic inflammation. Furthermore, Lee and Kim [24] demonstrated that RDW values were significantly higher in patients not only compared with RA than in healthy controls and patients with osteoarthritis, suggesting a strong association between RDW elevation and inflammatory disease activity in RA.

In this study, the total WBCs count was slightly higher in patients with RA than in healthy controls, but the difference was not statistically significant ($p = 0.261$). This finding is consistent with those of previous studies [16, 18, 25]. Al-Yasiri [19] and Raza et al. [26] reported a significant increase in total WBCs in RA patients, attributing this to the chronic inflammatory nature of the disease. In RA, elevated WBC counts are attributed to increased immune-mediated activity, possible infection, and recruitment of leukocytes to inflamed synovial tissues due to systemic inflammation [26]. The lack of a significant difference in the total WBC count may be related to the moderate inflammatory status of many patients with RA recruited from outpatient diagnostic centers and the relatively limited sample size.

Neutrophil percentages were significantly higher in patients with RA than in controls ($p = 0.049$). This finding is in agreement with a study conducted by Al-Yasiri [19], who reported significant neutrophilia in patients with RA, which was associated with elevated IL-6 levels. IL-6 is a multifunctional cytokine that stimulates bone marrow production and release of neutrophils and plays a key role in regulating acute-phase inflammatory responses [27, 28].

Lymphocyte percentages were significantly decreased in RA patients ($p = 0.001$). This finding aligns with that of Al-Yasiri [19], who reported low lymphocyte counts in RA due to chronic synovial inflammation. Lymphocytes migrate to inflamed joints, and local cytokine signaling attracts them to the synovium, reducing their numbers in the peripheral blood [19]. Additionally, impaired signaling pathways, including CD69, sphingosine-1-phosphate, and CXCR4, may limit lymphocyte egress from tissues, contributing to lymphopenia [29].

In this study, monocyte percentages were significantly elevated in patients with RA ($p = 0.007$), consistent with McGarry et al. [30], who explained that monocytes contribute to RA pathogenesis by differentiating into macrophages within the synovial tissue, producing pro-inflammatory cytokines, and promoting joint destruction, with the observed monocytosis reflecting ongoing immune activation and chronic inflammation.

Eosinophil percentages were significantly higher in patients with RA ($p = 0.001$), consistent with previous studies [19, 26]. Eosinophilia in patients with RA may result from autoimmune activity, drug reactions, or allergic responses. It has also been linked to the presence of autoantibodies, such as RF, and is considered a marker of disease activity [19].

Basophil percentages were significantly increased in RA patients compared to controls ($p = 0.001$). In RA, circulating basophil levels vary with disease type and stage, being decreased in adult RA with a dominant Th1 response, increased in juvenile RA with a dominant Th2 response, and showing an activated phenotype associated with disease activity [31].

In the present study, platelet counts were significantly higher in patients with RA than in healthy controls ($p = 0.001$). This finding is consistent with that of Al-Yasiri [19], who also reported a significant increase in platelet counts among patients with RA ($p = 0.021$), as well as with the results of Nghiem et al. [14]. In RA, thrombocytosis is attributed to several mechanisms, including acute and chronic blood loss associated with anemia of chronic disease, leading to increased erythropoietin and thrombopoietin levels, compensatory platelet overproduction due to increased platelet consumption, and immunological responses secondary to microthrombosis within chronically inflamed synovial tissue [32]. Furthermore, it may also occur as a consequence of inflammation-mediated cytokine activity, particularly IL-6, which indirectly enhances thrombopoietin production and stimulates reactive megakaryocytopoiesis and thrombopoiesis, resulting in increased platelet production and hyperactivity [33].

The MPV was significantly lower in patients with RA than in healthy controls in the current study ($p = 0.004$). This finding is in agreement with González-Sierra et al. [34] and Al-Yasiri [19], who reported a significantly reduced MPV in patients with RA compared with controls. In RA, decreased MPV during periods of high-grade inflammation is attributed to increased platelet consumption at sites of synovial inflammation, with MPV increasing following suppression of inflammation by disease-modifying and anti–TNF-α therapies [35].

The PCT values were significantly higher in patients with RA than in controls in the present study ($p = 0.011$). This observation is consistent with the findings of Al-Yasiri [19], who also reported significantly elevated PCT levels in RA patients ($p = 0.013$).

In the current study, PDW showed no statistically significant difference between patients with RA and healthy controls ($p = 0.736$). This finding contrasts with that of Al-Yasiri [19], who reported significantly higher PDW values in patients with RA ($p = 0.007$).

This study had several limitations that should be considered when interpreting the findings. First, the cross-sectional design limited our ability to establish causal relationships between CBC alterations and RA progression. Second, the association between CBC parameters and disease severity indices, such as the Disease Activ-

ity Score (DAS28), was not evaluated. Third, information regarding disease duration and detailed treatment history was limited, which may have influenced the hematological parameters. In addition, the study was conducted in selected diagnostic centers in Sana'a, which may limit the generalizability of the findings to all patients with RA in Yemen.

5. CONCLUSION

This study demonstrated that symptomatic patients with RA in Sana'a, Yemen, exhibited significant alterations in CBC parameters compared with healthy individuals, reflecting the systemic inflammatory nature of the disease. RBC indices showed reduced hemoglobin, HCT, MCV, MCH, and MCHC, with elevated RDW, indicating anemia secondary to chronic inflammation. WBC differentials revealed increased neutrophil, monocyte, eosinophil, and basophil counts with reduced lymphocyte counts, suggesting persistent immune activation. Platelet abnormalities were characterized by significant thrombocytosis, increased PCT, and decreased MPV, which were consistent with inflammation-driven platelet production and consumption.

These findings highlight the clinical value of CBC parameters as simple and accessible markers for monitoring inflammation and disease activity in RA. Future research should involve longitudinal studies to correlate CBC variations with specific disease activity scores (DAS) and treatment responses over time.

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