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Leucocytes Differential Count and Morphology Abnormalities among Patients with Rheumatoid Arthritis and Systemic Lupus Erythematosus

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ABSTRACT

Background: A diverse collection of illnesses known as immunological connective tissue disorders (ICTDs) are brought on by poorly managed autoimmune reactions and impact connective tissue in different organs. Understanding and diagnosing systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA) depend heavily on the leukocyte morphology and differential count. These illnesses, which include scleroderma, RA, and SLE frequently include immunological dysregulation, which results in distinctive leukocyte abnormalities.

Objective: To investigate abnormalities in leucocyte count, differential, and morphology among patients with RA and SLE.

Patients and Methods: An observational cross-sectional study included a population of 95 patients with RA and SLE who were assessed between March 2023 and June 2024 at the Outpatient Clinics of Rheumatology and Immunology, Menoufia University.

Results: There was a significant difference among the studied groups regarding white cell count, basophils, eosinophils, staff, segmented, monocytes, neutrophils, HCT, MCV, MCH, RDW-CV, and HB ($P<0.05$). Segmented, neutrophils, HCT, MCH, and HB were significantly lower in the high group by (70.2%, 7.6%), (2.8%, 22.2%), (14.1%, 45.8%), (12.3%, 16.9%), (14%, 7.1%) than the other moderate and mild groups; respectively. White cell count, eosinophils, staff, monocytes, and RDW-CV were significantly higher in the high group by (9.82%, 125.2%), (6.31%, 573.3%), (397.3%, 894.7%), (21.9%, 2086%) and (9.51%, 7.75%), than moderate and mild groups; respectively. Basophils were significantly higher in the moderate group by (620%, and 71.4%) than moderate and mild groups; respectively.

Conclusion: The differential count and morphology of leukocytes play a critical role in understanding and diagnosing ICTDs, such as SLE, RA, and scleroderma, often involve immune dysregulation, leading to characteristic leukocyte abnormalities.

Keywords: Systemic lupus, Rheumatoid arthritis, Leucocytes differential count, Leukopenia.

INTRODUCTION

Complement activation, interferon dysregulation, poorly managed autoimmune responses, and related inflammation are the causes of the diverse group of illnesses known as immunological connective tissue disorders (ICTDs), which impact connective tissue in different organs [(1)]. Despite differences in clinical presentation, cross-analysis of genome-wide association studies and shared regulatory mechanisms of autoimmune disorders have shown that these illnesses share important genetic risk factors [(2)].

The development of autoimmune disorders is also significantly influenced by environmental and female-associated variables [(3,4)]. Although a large portion of this knowledge is not yet included into routine clinical management, autoantibody generation and immune dysregulation occur prior to clinical manifestation in almost all systemic autoimmune rheumatic illnesses assessed too far. Enhancing biomarkers for diagnosis, prognosis, treatment selection, and optimal therapy is the subject of ongoing study [(5,6)].

The body's immune cells are called white cells. Often referred to as leukocytes, they come in five different varieties and are helpful in the battle against infections. The body produces around 100 million white cells every day[(7)]. Granulocytes, which comprise neutrophils, basophils, and eosinophils, and non-granular cells, which include lymphocytes and monocytes, are among them. Both the former and monocytes are made in the bone marrow, whilst the lymphocytes are produced in the lymphoid tissue[(8)].

The white blood cell count is a significant subset of the total blood count as the quantity of leukocytes in the blood is frequently a sign of illness. White blood cell counts typically range from 4×10^9/L to 1.1×10^{10}/L[(9)]. Low white cell counts can be brought on by primary or secondary cancers, or by the bone marrow's underproduction of WBC, which can occasionally happen after exposure to toxins like benzene. Antibodies produced by the immune system can also cause the body's white cells to be destroyed in autoimmune diseases like SLE or RA[(10)].

About 50% of SLE patients have leukopenia, making it a frequent condition. Leukopenia often corresponds with clinically active illness and might be caused by lymphopenia or secondary neutropenia[(11)]. The quantity of platelets and White blood cells (WBC) may rise or fall. Additionally, they could have hereditary or acquired morphological defects[(12)]. A differential count is necessary to determine if there is an increase or reduction in the numbers of distinct kinds of white cells. Changes in the shape, function, and/or concentration of one or more kinds of circulating leukocytes are linked to a number of illnesses[(13)].

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Even though these alterations are frequently vague, they can offer diagnostic hints for both inherited and acquired diseases. When the concentration of circulating WBC falls below the reference range, it is referred to as leukopenia[14]. Different WBC subpopulations are characterized by decreased concentrations, which are referred to as granulocytopenia, neutropenia, eosinopenia, basopenia, lymphopenia, and monocytopenia[15].

More precisely, the emergence of their possible presentations is crucial for the prompt identification of disease states that require aggressive, targeted, and specific intervention, which is frequently multifaceted. It also helps to raise awareness of SLE and autorheumatoid arthritis and takes a multidisciplinary approach. Having this knowledge enables one to make the crucial, time-sensitive decisions necessary to guarantee the best possible therapeutic result [16]. In this study, we discussed the timeline of differential leucocyte count in RA and SLE.

PATIENTS AND METHODS

Study design: This study is an observational cross-sectional study that was carried out at Menoufia University Hospital. Patients with RA and SLE were recruited from Outpatient Clinics of Rheumatology and Immunology at Menoufia University Hospitals during the study period from March 2023 to December 2024.

Sample size: It was conducted on 105 subjects attending the Rheumatology and Immunology Clinic at Menoufia University Hospital and meeting the eligibility criteria.

All patients under study were selected according to:
Patients' criteria selection:

We included age above 18 and a definitive diagnosis of SLE and RA disease according to the validated diagnostic criteria of each disease as RA by DAS-2 and SLE by SLEDI. However, we excluded critically ill patients, patients with a history of hematological disorders, those who have received blood transfusions within the past three months, those who have smoked or used alcohol, expectant mothers, and those who have recently undergone surgery.

All patients were subjected to the following:
Demographic data: Including age, sex, and occupation.
Clinical history and assessment data: Onset and duration of the disease, number of joint involvements (swelling & tenderness), morning stiffness and exercise tolerance, extra-articular involvement (lung fibrosis, renal involvement, skin lesions,cardiac involvement, or neurological), disease activity clinical scores (SLEDI, DAS-28), drug history and blood pressure, body weight.
Laboratory investigations: Included a complete blood count (CBC) by The Sysmex XN-450/XN-430 a quantitative automated hematology analyzer (Sysmex Company, Germany). Ferritin levels Assayed by electro-chemiluminescence reaction using Cobas 6000 (e 601 module) Roche diagnostics- GmbH, D-68305 Mannheim, Germany. C- reactive protein (CRP) using latex slide test and erythrocyte sedimentation rate (ESR) using Westergren method.
Rheumatoid factor (RF), and anti-cyclic citrullinated peptide antibodies using anti-CCP using Latex agglutination slide test and Anti-nuclear antibody (ANA), Liver enzymes are tested through kinetic method by spectrophotometer wl 540nm. Renal function tests included blood urea and serum creatinine using Cobas 6000 analyzer (c501 module) (Roche Diagnostics- GmbH, D-68305 Mannheim, Germany), and other autoantibodies.

Leucocytes parameters:
Total, relative, and absolute differential leukocyte counts; Absolute differential leukocyte counts (total WBC × WBC type relative percent), TLC (cells/mm^3). Morphology evaluation including overall cell appearance (i.e., reactivelymphocytes), just the nucleus (i.e., hyper- and hypopigmentation, multiple nuclei, nuclear blebbing, Reider forms), or just the cytoplasm (i.e., toxic granulation, vacuolization, a granularity, cytoplasmic blebbing). Immature cells in any leukocyte cell line report and Correlation with platelets and red cell abnormalities.

Table (1): Comparing Table Highlighting the Morphological Changes in White Blood Cells in RA and SLE

Cell Type	RA	SLE
Neutrophils	* Activation and recruitment to synovial tissue. * NET Formation (neutrophil extracellular traps). * Ragocytes in synovial fluid (neutrophils with ingested immune complexes).	* Enhanced NET Formation (plays a key role in SLE). * Lupus neutrophils (prone to apoptosis and NET release). * LE cells (neutrophils/macrophages with phagocytosed nuclei).
Lymphocytes	* T-cell activation (Th17 expansion, CD4+ T-helper cells). * B-cell differentiation into plasma cells producing RF and ACPAs. * Ectopic germinal centers in the synovium.	* T-cell hyperactivation (aberrant signaling, reduced Tregs). * B-cell hyperactivation and plasma cell differentiation (anti-dsDNA, anti-Sm, ANAs). * Ectopic germinal centers in lymphoid tissues.
Monocytes/Macrophages	* M1 Polarization (pro-inflammatory macrophages). * Synovial infiltration and cytokine production (TNF-α, IL-1β, IL-6). * Multinucleated giant cells in chronic inflammation.	* M1 Polarization (pro-inflammatory macrophages). * Enhanced phagocytosis of apoptotic cells and immune complexes. * Foam cell formation (lipid-rich macrophages in vascular inflammation).
Dendritic Cells	• Increased maturation and antigen presentation • Elongated dendrites and cytoplasmic projections	• Increased maturation and antigen presentation • Elongated dendrites and cytoplasmic projections
Eosinophils / Basophils	• Eosinophils: degranulation and release of inflammatory mediators • Basophils: activation and histamine release	• Eosinophils: degranulation and release of inflammatory mediators • Basophils: activation and histamine release
Unique Features	• Rheumatoid Factor (RF) and Anti-Citrullinated Protein Antibodies (ACPAs) • Synovial inflammation and joint destruction	• LE cells (neutrophils/macrophages with phagocytosed nuclei) • Hematoxylin bodies (nuclear remnants in tissues) • Anti-dsDNA, anti-Sm, and ANAs
Cytokine Drivers	• TNF-α, IL-1B, IL-6, and IL-17 drive inflammation	• IFN-α, TNF-α, IL-6, and IL-10 drive inflammation

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Key Differences:

Neutrophils: In RA, neutrophils contribute to synovial inflammation and form ragocytes.
• In SLE, neutrophils are more involved in NET formation and LE cell formation.

Lymphocytes: In RA, T cells (TH17) and B cells (RF, ACPAs) drive joint-specific inflammation.
In SLE, T cells and B cells produce a broader range of autoantibodies (anti-dsDNA, anti-Sm, ANAs) and contribute to systemic inflammation.

Macrophages:

• In RA, macrophages are key players in synovial inflammation and cytokine production.
• In SLE, macrophages are involved in phagocytosis of apoptotic cells and immune complexes, contributing to systemic damage.

Unique Features:

• RA is characterized by synovial inflammation and joint-specific autoantibodies (RF, ACPAs).
• SLE is characterized by systemic autoantibodies (anti-dsDNA, anti-Sm, ANAs) and unique cellular features like LE cells and hematoxylin bodies.

Summary:

• While both RA and SLE involve dysregulated immune responses and morphological changes in WBCs, the specific changes and their clinical implications differ.
• RA is more focused on joint-specific inflammation, whereas SLE involves systemic autoimmunity with broader tissue and organ involvement.

Ethical approval:

The current investigation was conducted in compliance with relevant local regulatory rules and international ethical standards. Following an explanation of the research's goals, methods, risks, and advantages, information permission was acquired from both patients and controls prior to their enrollment in the trial. The Menoufia University Faculty of Medicine's Ethical Committee

examined and approved the study's protocol (No.: 3/2023INTM29). The study adhered to the Helsinki Declaration throughout its execution.

Statistical analysis

The statistical analysis and tabulation of the results were conducted using the SPSS V.25 application for Microsoft Windows 10. The data were described using frequency and percentage for qualitative data and mean ± SD for quantitative data. The total of all observations divided by the total number of observations is the mean. The standard deviation, on the other hand, quantifies how widely apart particular variations are from their mean. X^2-test: It is used to compare one qualitative variable between two or more groups. When comparing two groups in terms of regularly distributed (parametric) quantitative data, the standard student-t test (t) was employed. A nonparametric version of the student's t-test is the Mann-Whitney test (U). It is employed to show whether there is a significant difference between two groups for a quantitative variable that is not regularly distributed. A P-value of less than 0.05 was deemed statistically significant.

RESULTS

A flowchart of the study population of 105 patients with RA and SLE who were assessed at the Outpatient Clinics of Rheumatology and Immunology Menoufia University Hospital. 10 patients were excluded from the study (4 patients declined consent, and 6 did not meet the inclusion criteria), and 95 patients participated in the study. Those patients were divided into two main groups; Group one: included 46 patients with systemic lupus divided into mild, moderate, and severe, Group Two included 49 patients with RA that were divided into active and remission (**Figure 1)**.

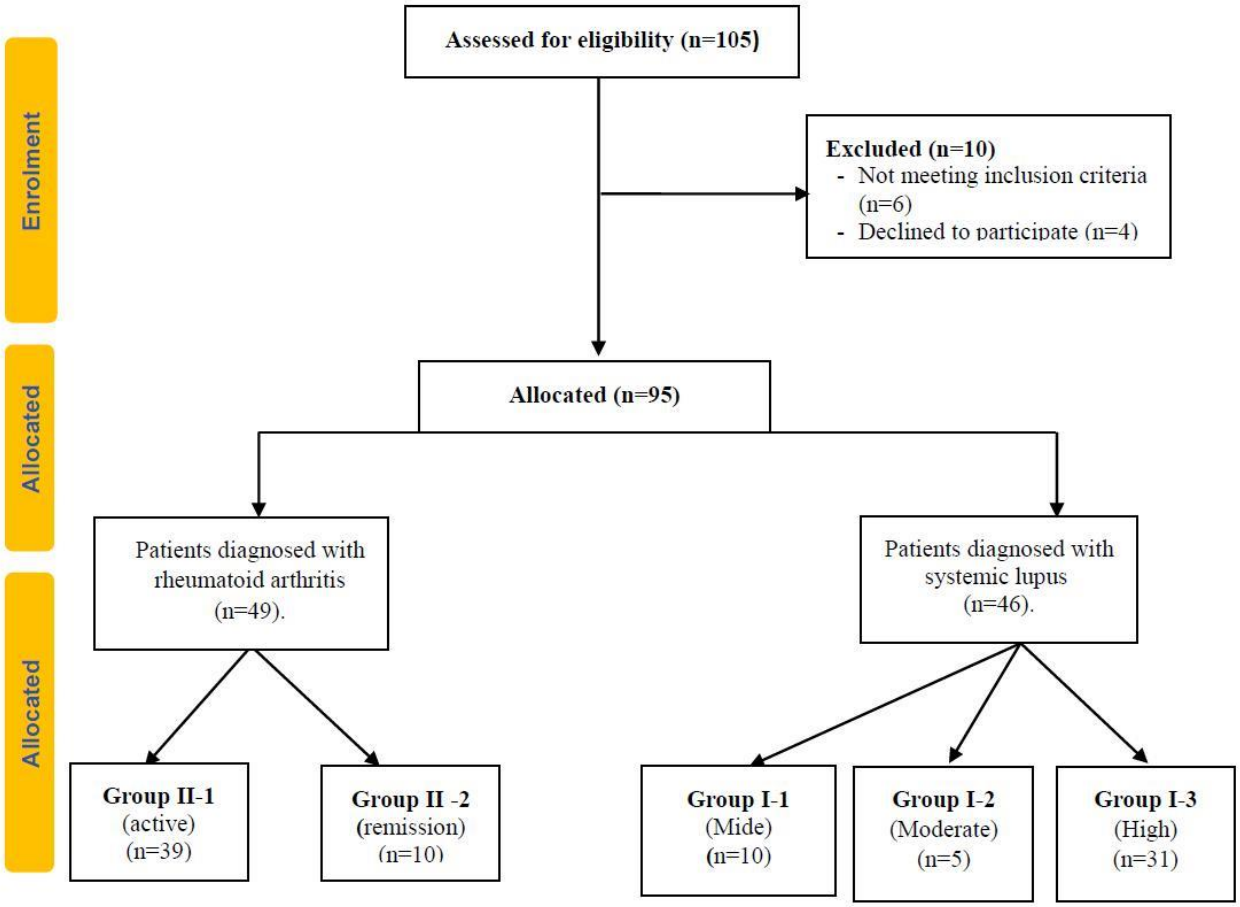

Figure (1): Flowchart of patients with RA and SLE.

In our study, there were significant differences among the studied groups who had systemic lupus regarding age, disease activity, and duration of disease (P<0.05) and who had RA regarding disease activity (p<0.001). Age, disease activity, and duration of disease for patients had systemic lupus were significantly higher in the group with high SLEDAI score (34.00±11.63, 17.81±6.01, 4.77±2.22; respectively) than in the other groups. There wasn't sign. diff. among the studied groups who had systemic lupus regarding gender and who had RA regarding age, gender, and duration of disease (P>0.05) **(Table 2).**

Table (2): Demographic data and clinical history among the patients studied (n=95).

SLE (n=46)	**SLEDAI score**				**Sig. test**	
	Mild (n=10)	**Moderate (n=5)**	**High (n=31)**	**Total N=46**	**F**	***P value***
Age/year Mean ± SD. Range	25.0±5.27 20- 30	27.00±0.00 27 -27	34.00±11.63 17 - 54	28.22±5.60 17-54	3.568	**0.037***
Sex Male Female	0(0%) 10(100%)	0(0%) 5(100%)	5(16.12%) 26(83.87%)	5 (10.86%) 30 (89.1%)	0.65	0.543
Disease activity score Mean ± SD. Range	3.00±1.05 2.00 – 4.00	6.00±0.00 6.00 – 6.00	17.81±6.01 12.00 – 30.00	8.93±2.35 2-30	38.429	**<0.0001***
Duration of disease Mean ± SD. Range	3.00±1.05 2.00 – 4.00	2.00±0.00 2.00 – 2.00	4.77±2.22 2.00 – 8.00	3.25±1.09 2-8	6.588	**0.003***
RA (n=49)	**Remission (N=10)**		**Active (N=39)**	**Total N=49**	**t**	***P value***
Age/year Mean ± SD.	36.60± 5.02		36.74±8.54	36.67±6.78	0.069	0.946
Gender Male Female	1 (10 %) 9 (90%)		14 (35.8%) 25 (64.2%)	15 (30.61%) 34 (69.38%)	1.987	0.652
DAS-28 Mean ± SD.	2.44±0.64		4.67±0.96	3.55±0.8	U=8.768	**<0.0001***
Duration of disease Mean ± SD.	4.15±3.07		6.10±3.15	5.16±3.11	1.783	0.096

SLEDAI: Systemic Lupus Erythematosus Disease Activity Index, **DAS:** Systemic Lupus Erythematosus Disease Activity Index, *: Significant.

There were no significant differences among the studied groups regarding ferritin, red cell count, MCHC, RDW-SD, platelet count, lymphocytes, CRP and ESR 1st hour, albumin in urine, albumin/creatinine ratio in urine and Serum creatinine (P>0.05). While, there was a sig. diff. among the studied groups regarding HCT, MCV, MCH, RDW-CV, white cell count, basophils, eosinophils, staff, segmented, monocytes, neutrophils, HB, ESR 2nd hour, creatinine in the urine, Serum urea, AST, and ALT (P<0.05). HCT, MCH, segmented, neutrophils, HB and serum urea were significantly lower in the high SLDEI score group (31.61±3.48, 26.90±4.29, 61.96±15.03, 53.60±13.31, 10.32±1.36, and 35.71±7.38; respectively) than the other groups. MCV, basophils, AST, and ALT were significantly higher in the moderate group (94.50 ±0, 0.36±0.08, 30.60±0, and 27.00±0; respectively) than other groups. However, ESR 2nd hour and creatinine in urine were significantly higher in mild group (45.90±7.87, 264.60 ±0) than the other groups unlike RDW-CV, white cell count, eosinophils, staff, and monocytes, which were significantly higher in the high group (14.04±0.78), (6.15±1.41), (2.02±0.48), (1.89±0.46), and (6.34±1.51); respectively than the other groups **(Table 3)**.

Table (3): Laboratory investigations among the studied patients of systemic lupus (n=46).

Variables	SLEDAI score				Sig. test	
	Mild (n=10)	Moderate (n=5)	High (n=31)	Total (n=46)	F	*P value*
Ferritin (ng/mL) Mean ± SD.	50.00±12.18	60.20±14.87	69.39 ± 16.39	59.86±0.27	0.964	0.389
Red cell count (mcL) Mean ± SD.	3.80 ±0 .70	3.90 ± 0.01	4.15 ± 0.61	3.95±0.44	1.392	1.389
HCT (Mean ± SD.)	58.35 ± 14.41	36.80 ± 0.01	31.61 ± 3.48	42.25±10.4	14.187	**<0.0001***
MCV (µm^3) Mean ± SD.	52.25 ± 12.73	94.50 ±0.01	81.75 ± 7.01	76.17±11.3	23.636	**<0.0001***
MCH (Mean ± SD.)	32.40±0.63	30.70 ± 0.01	26.90±4.29	30.0±1.64	9.856	**<0.0001***
MCHC (Mean ± SD.)	33.00±001	32.60±0.001	32.47±3.03	32.69±1.10	0.164	0.85
RDW-CV (Mean ± SD.)	13.03±1.09	12.82±0.04	14.04±0.78	13.3±0.64	8.867	**0.001***
RDW-SD (Mean ± SD.)	44.82±2.65	44.88±1.61	44.28±1.82	44.66±2.03	0.399	0.673
Platelet count (mcL) Mean ± SD.	277.00±28.46	225.0±0.001	271.71±67.28	257.9±11.58	1.489	0.237
White cell count (mcL) Mean ± SD**.**	2.73±0.67	5.60±0.001	6.15±1.41	4.83±1.10	5.757	**0.006***
Basophils (Mean ± SD.)	0.05±0.011	0.36±0.08	0.21±0.04	0.36±0.08	3.474	**0.040***
Eosinophils Mean ± SD.	0.30±0.03	1.90±0.46	2.02±0.48	1.41±0.34	17.616	**<0.0001***
Staff (Mean ± SD.)	0.19±0.03	0.38±0.08	1.89±0.46	0.82±0.20	9.975	**<0.0001***
Segmented (Mean ± SD.	67.10±0.32	208.00±50.18	61.96±15.03	97.35±24.10	5	**0.011***
Lymphocytes Mean ± SD.	29.10±0.32	39.60±0.001	38.29±9.49	35.66±4.44	2.951	0.063
Monocytes Mean ± SD.	0.29±0.06	5.20±0.001	6.34±1.51	3.94±0.98	20.557	**<.0001***
Neutrophils Mean ± SD.	68.90±0.32	55.20±0.001	53.60±13.31	59.23±4.72	6.714	**.003***
HB (g/dL) Mean ± SD.	11.11±1.15	12.00±0.001	10.32±1.36	11.14±0.84	4.662	**.015***
CRP (mg/L) Mean ± SD.	6.96±1.46	7.54±1.64	6.85±1.47	7.12±1.52	0.472	0.627
ESR 2nd hour Mean ± SD.	45.90±7.87	32.80±6.26	41.32±8.89	40.01±7.67	3.988	**.026***
ESR 1st hour Mean ± SD.	14.80±3.61	14.60±3.41	16.00±3.98	15.13±3.71	0.254	0.777
Creatinine in urine (mg/dl) Mean ± SD.	264.60 ±0.001	36.46±9.10	54.01±13.40	118.36±29.41	46.841	**<.0001***
Albumin in urine Mean ± SD.	6196.70 ±107.73	6167.20 ±99.51	6142.52±94.01	6168.81±90.42	0.012	0.988
Albumin/creatinine ratio in urine Mean ± SD.	4082.70±484.30	3594.40±540.62	3879.06±79.69	3852.23±65.2	0.792	0.459
Serum urea (mg/dL) Mean ± SD.	43.90±4.77	44.20±7.16	35.71±7.38	41.27±6.44	7.311	**.002***
Serum creatinine (mg/dl) Mean ± SD.	0.99±0.23	1.12±0.27	0.97±0.23	1.03±0.24	0.359	0.701
AST (U/L) Mean ± SD.	18.40±4.30	30.60±0.001	20.94±5.19	23.31±4.83	6.316	**.004***
ALT (U/L) Mean ± SD.	14.90±3.71	27.00±0.001	17.19±4.12	19.7±3.56	9.565	**<.0001***

*: Significant

In the current study, among mild systemic lupus patients, WBCS were significantly increased at 6th month of follow-up compared to admission (16%), while among moderate and severe patients, WBCs were significantly lower or decreased at 6th month of follow-up compared to admission (20%, 30%; respectively) (**Table 4**).

Table (4): White cells count among patients with systemic lupus (n=46).

WBCS	Mild (n=10)	Moderate (n=5)	Severe (n=31)	Total (n=46)
At admission				
Mean±SD	2.73±0.67	5.6±0.1	6.15±1.41	4.83±1.55
3rd month				
Mean±SD	2.75±1.93	5.13±1.63	4.16±1.70	19.84±2.09
different %	(0.73%) ↑	(8.30%) ↓	(32.30%) ↓	
6th month				
Mean±SD	3.97±0.11	4.84±1.22	4.03±1.89	4.28±1.74
different %	(16%) ↑	(20%) ↓	(30%) ↓	
H	9.51	4.22	17.9	
P value	**0.026***	**0.039***	**0.005***	

-DISCUSSION

Circulating WBCs frequently experience relative alterations under systemic inflammation, which are usually shown as neutrophilia and lymphopenia. This might account for the increased neutrophil to lymphocyte ratio (NLR) in SLE patients, particularly when activity is high. Moreover, platelet counts typically drop when lupus activity increases; however, the lymphocyte count decreases more than the platelet count, which may account for the elevated platelet-to-lymphocyte ratio (PLR) associated with SLE activity [16].

Our study showed that there was a significant difference among the patients studied regarding disease activity score and duration of disease. The high group had substantially greater disease activity scores (17.81±6.01) and duration of disease (4.77±2.22) compared to the other groups. In a study by **Wong *et al.*** [17] individuals with RSLE and SLE had a mean diagnosis duration of 12•4 ± 6•3 years (range 1•7-26•6) and 9•0 ± 6•8 years (range 0•3-25•6 years), respectively. RSLE and SLE patients had SLEDAI scores of 7•9 ± 5•9 (range 0-20) and 2•8 ± 5•6 (range 0-32), respectively.

We found no significant difference in ferritin, red cell count, MCHC, RDW-SD, platelet count, and lymphocytes among the individuals we investigated. Compared to the other groups, the high group had considerably lower levels of HCT, MCH, segmented neutrophils, and HB. Compared to the other groups, the high group had considerably greater RDW-CV, white cell count, eosinophils, staff, and monocytes. The Moderate group had much more MCV and basophils than the other groups.

Similarly, **Abdulrahman *et al.*** [18] reported that patients' NLR and PLR ratios were considerably greater than controls' ($p = 0.007$ for both). Compared to individuals without lupus nephritis, those with the disease had considerably higher NLR and PLR ratios ($p < 0.001$). Patients with lupus nephritis who were relapsing and those who were naïve had similar NLR and PLR ratios (5.2 ± 1.1 vs 5 ± 1.2 and 246.4 ± 67.6 vs 259.4 ± 57.3, respectively) ($p = 0.81$). Patients with active lupus nephritis had their laboratory tests and parameters compared to those without the condition. However, **Soliman *et al.*** [19] found that among SLE patients with extremely active SLE illness, both the NLR and PLR values were considerably higher.

Additionally, **AbdElkader *et al.*** [20] discovered a statistically significant positive connection ($p<0.001$) between NLR and PLR. In terms of SLICC DI, comprising renal and musculoskeletal symptoms, as well as the overall score, there was a statistically significant difference between L/M activity SLE and H/VH SLE activity ($p<0.001$).

According to our study, there were no appreciable differences between the patients in terms of CRP, ESR at the first hour, lupus anticoagulant, anti-cardiolipin 1gM, complement (C3), and complement (C4). Compared to the other groups, the Mild Group's ESR was noticeably higher at the second hour.

According to a different research **Qin *et al.*** [16], NLR had a favorable correlation with SLEDAI ($r=0.471$, $p<0.01$), ESR ($r=0.610$, $p<0.01$), and CRP ($r=0.509$, $p<0.01$). According to **Qin *et al.*** [16], there was a favorable correlation between PLR and SLEDAI ($r=0.44$, $p<0.01$). NLR, PLR, and MPV did not show statistically significant correlations with urine protein, C3, C4, or anti-dsDNA antibodies. Also, PLR showed a good correlation with SLEDAI score, ESR, and CRP levels, according to **Soliman *et al.*** [19]. A substantial negative association was also observed between PLR and C4, whereas a nonsignificant negative correlation was observed with C3. They discovered a correlation between PLR and anti-DNA.

Our study showed that the active group had substantially more disease activity than the remission group. Regarding the length of the illness, there was no discernible difference between the groups under study. **Abd-Elazeem and Mohamed** [21] demonstrated that there was a significant difference in the duration of the illness between the active and remission groups (6.9 ± 3.4 years, 4.8 ± 3.04 years; $p = .02$).

Our study showed that the active group had more segments and neutrophils than the remission group,

whereas the remission group had more lymphocytes than the active group. Red cell count, HCT, MCH, MCV, MCHC, RDW-CV, RDW-SD, platelet count, ferritin, white cell count, basophils, eosinophils, staff, monocytes, and HB did not significantly differ among the groups under study.

In the same line, **Abd-Elazeem and Mohamed** [21] demonstrated that when patients were compared to controls in terms of CBC parameters, there was a significant difference in platelet count between groups A and B (p =.04) and group A and control (p =.048). Although neutrophils, mast cells, and B and T lymphocytes play essential roles in the pathophysiology of RA, it has been discovered that the NLR is an extremely relevant indicator of systemic inflammation that is easily calculated [22].

Abd-Elazeem and Mohamed [21] demonstrated that the NLR and PLR were 2.8±2.1 and 1.7±0.9 in all patients, which were similar to the control group's 2.1±0.59 and 1.27±0.46 (p=0.15 and p=0.09, respectively). There was no significantly different between men and females in terms of NLR (2.2±0.6 and 3.5±3.02) and PLR (1.7±1.1, 1.8±1.2) (p=0.42 and p=0.91; respectively). Between active patients and control active patients, there was a significant difference in both NLR and PLR; RF-positive (n=19) cases had a considerably greater NLR than RF-negative (n=6) cases (p=0.03), but PLR was similar (p=0.22). RF positive revealed no significant difference in NLR or PLR among remission patients (p=0.77 and p=0.9, respectively). The NLR and PLR of active anti-CCP positive (n=19) and negative (n=6) patients did not vary significantly (p=0.7 and p=0.47, respectively), nor did those in remission (p=0.91 and p=0.74; respectively). NLR is a helpful metric for evaluating the efficacy of anti-TNF-a medications and for displaying inflammation alongside CRP.

According to our study, there was no discernible variation in the patients' ESRs at the first hour (p=.402). The active group had greater CRP and ESR during the second hour than the remission group, which was a significant difference (p=0.001). In individuals with RA, the inflammatory response status is indicated by the CRP level and ESR [22].

Abd-Elazeem *et al.*'s study [21] revealed that while only the CRP was greater than those in remission, the ESR and CRP were considerably higher in active patients when compared to control. **Mercan *et al.*** [23] enrolled 117 controls and 136 RA patients in their research. In RA, the NLR was greater than the control and showed a strong correlation with both ESR and CRP.

AST, ALT, serum creatinine, albumin in urine, and the albumin/creatinine ratio in urine did not significantly change between the groups under investigation when comparing those in remission and those in active status based on liver and renal function tests. The remission group's serum urea and urine creatinine levels were significantly greater than those of the active group.

Similarly, **Tang *et al.*** [24] demonstrated that 90 (71.4%) of the 126 RA patients in the research had a diagnosis of renal impairment. MAU and PU were the primary indicators of its presence, and they considerably outperformed ($p<0.05$) comparable indices in AHPs and the group of RA patients without renal injury. Our investigation compared autoantibodies between individuals in remission and those who were active. There was no significant difference between all complements (C3), complement (C4), lupus anticoagulant, anti-cardiolipin 1gM, and anti-cardiolipin 1gG. This study's findings are similar to those of a study conducted by **Ulvestad *et al.*** [25]**.**

CONCLUSION

The differential count and morphology of leukocytes play a critical role in understanding and diagnosing RA and SLE. These diseases, such as SLE, RA, and scleroderma, often involve immune dysregulation, leading to characteristic leukocyte abnormalities.

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