Khalij Libya Journal of Dental and Medical Research. 2025;9(1):97-102
https://doi.org/10.47705/kjdmr.25911015
eISSN:2708-888X

Original article

Hematological Disorders of Systemic Lupus Erythematosus: A Cross-Sectional Study

Mohamed Abuagela[1]* , Nadra Elalem[1], Najla Elyounsi[2] , Wasim Elarbi[1], Retaj Almahmoudi[1], Sondos Gaumati[1]

[1]*Department of Anesthesia and Intensive Care, Faculty of Medical Technology, University of Tripoli, Tripoli, Libya.*
[2]*Department of Medical Laboratory Sciences, Faculty of Medical Technology, University of Tripoli, Tripoli, Libya.*
***Corresponding Email**: m.abuagela@uot.edu.ly*

Abstract
Systemic lupus erythematosus (SLE) is an autoimmune disease with unknown etiology. It involves multiple organs and presents as varying clinical and hematological manifestations. Investigations are very important to diagnose SLE, including CBC, ESR. Limited data were reported for the frequency of SLE in Libya. A cross-sectional study aims to assess the prevalence of SLE in Tripoli, Libya. A total of 140 participants, including both genders and aged between 17 and 59 years. Diagnostic Tests and Blood Screening include a complete blood picture and Erythrocyte Sedimentation Rate (ESR). Gender-wise distribution was 93.52% females and 6.48% males. The percentage of SLE was highest in the age group 27 – 36 years (34.26%). The mean ESR was significantly increased in SLE patients compared to the control. While the mean Hgb was decreased compared to the control. The relationship between monocytes and neutrophils in two groups of SLE was also investigated by comparing SLE patients with the control group. The mean Monocytes and neutrophils were significantly increased in SLE patients compared to the control. There are no significant differences in Lymphocytes between SLE patients and control subjects. SLE is a chronic disease that is affected by multiple factors such as age, gender. Alterations in blood tests in SLE patients are a key point in disease diagnosis.
***Keywords**: Systemic Lupus Erythematous, Haematological Disorders, CBC, ESR*

***Received**: 08/03/25*
***Accepted**: 03/05/25*
***Published**: 11/05/25*

Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with a wide spectrum of clinical and hematological manifestations caused by autoantibody production, complement activation, and immune complex deposition. The occurrence of lupus is when the immune system, attacks its tissues. This attack leads to inflammatory disorders and in certain cases permanent tissue damage, which can be widespread – affecting many body organs— joints, skin, kidneys, blood cells, brain, heart, and lungs [1]. SLE is the most common type, accounting for 70% of lupus cases. It affects multiple organs and systems throughout the body. Yet, there are further types, depending on which body parts are affected. Furthermore, to SLE, there are cutaneous lupus (affect skin), drug-induced lupus (DIL), and neonatal lupus [2]. SLE is difficult to diagnose while no single laboratory test can ultimately demonstrate that a person has SLE. An 80-90% of lupus cases may live for more than 10 years after being diagnosed [3]. Late diagnosis and dysregulation of SLE management, the patient may be exposed to many serious health complications as it may lead to renal impairment, pneumonia, arthritis, anemia, infection, and cardiovascular disease leading to death thus care must be taken for early treatment when lupus erythematosus [4]. The etiopathogenesis of lupus is unknown clearly, but it is thought that it results from the complex interaction between genetic, hormonal factors, and environmental factors such as exposure to sun UV light and certain medicines or viral infections, and also has an unpredictable progression that characterized a challenge in the understanding of this disease [5,6]. The prevalence and incidence rates of SLE are different due to various factors. Globally, SLE incidence was estimated to be 5.14 (1.4 to 15.13) per 100,000 person-years and 0.40 million annually, respectively. Yet SLE prevalence was estimated to be 43.7 (15.87 to 108.92) per 100,000 persons and 3.41 million people, respectively [7]. SLE is recurrent in women than men with a female-to-male ratio of 9 to 1. In most cases, lupus develops in women of childbearing age between the ages of 15-44 , suggests hormonal influence in its pathogenesis. People of Asian, Black, African American, Hispanic, and Indigenous descent with higher incidence, prevalence, and mortality in these subpopulations [8,9,10]. Geographically, the incidence of SLE depends on various populations, these differences may, in part, be due to genetics. Recent studies observed higher frequencies of genetic risk variants for lupus in Asians than in Europeans and higher still in Africans. Across the world, it is estimated that genetics explains between 40% - 60% of the risk for lupus, and family history plays an important role in SLE occurrence [11].

Khalij Libya Journal of Dental and Medical Research. 2025;9(1):97-102
https://doi.org/10.47705/kjdmr.25911015
eISSN:2708-888X

Lupus pathogenesis is a complex interaction between the exposome environmental factors (e.g. sex hormones, UV light, infections, and smoking), genetic and epigenetic factors. A complex interaction of genetics, environment, and hormones leads to immune dysregulation and triggers a loss of immune tolerance to self-antigens, resulting in autoantibody production, inflammation, and destruction of end-organs and also aberrant activation of autoimmunity exposure of self-antigens to the immune cells. The ensuing production of autoantibodies and immune complexes, autoreactive T cells and B cells complement activation, and cytokine release result in widespread tissue damage, manifesting as the clinical picture of SLE [3,9,12]. On a cellular level, innate and adaptive immune responses against self-antigen encourage the creation of autoantibodies and the deposition of immune complexes in tissues leading to the activation of complements, accumulation of neutrophils and monocytes, and self-reactive lymphocytes. In SLE, B lymphocyte plays a vital role in adaptive immunity, which is involved in the production of autoantibodies, presentation of autoantigens, and activation of autoreactive T cells. Moreover, T lymphocyte has an important role through co-stimulator-mediated signaling pathways and cytokines secreted by subsets of T cells [13].
Clinically, SLE diagnosis is quite difficult; a diagnosis of lupus depends on medical history, physical exam, blood tests, and imaging studies. Generally, SLE is a relatively rare condition, there are no wide-ranging criteria for diagnosis, and many blood tests or imaging study used to diagnose lupus [14]. Different laboratory researchs reported that SLE diagnosis depended on hematological disorders in 82.7% of patients. The major manifestations are anemia, leucopenia, thrombocytopenia and antiphospholipid syndrome (APS). Primarily, blood tests are used mainly to diagnose features of the disease including complete blood count (CBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP), in addition to autoantibody testing [15, 16]. To evaluate SLE, there are simply available laboratory indicators that evaluate disease activity. Anemia is relatively common and may affect about 50% of cases. Lymphopenia present in 93% of patients during the active phase of the disease. Moreover, neutrophils also a rise due to the complement pathway's inability to clear the lupus neutrophils, thus resulting in their accumulation. In lupus, thrombocytopenia is usually increased [17, 18]. An ESR is a test that measures generalized inflammation in the body, but is mainly used to differentiate inflammatory situations such as SLE from non-inflammatory disease. An increasing ESR may key point of a lupus-associated disorder. Another marker is the CRP an indicator of general inflammation. However, different ESR, do not typically rise with lupus, therefore, it isn't necessarily beneficial for monitoring disease activity [18]. Particularly, there is increasing evidence that early diagnosis and treatment could increase the SLE recovery rate improve patient prognosis, and appear to prevent many complications. The purpose of this study is to evaluate various blood disorders in SLE, including anemia, leukopenia, and other markers like ESR and CRP.

Methods

Ethical approval

The study was conducted in accordance with national ethical guidelines with informed consent. The medical department of Tripoli University Hospital provided ethical approval, and the medical technology faculty provided permission. All met the criteria of classification by the American College of Rheumatology (ACR) for the diagnosis of SLE. Patient data confidentiality was maintained by avoiding personal identification and ensuring the anonymity of the personal data records. Only the patient's data, such as age, sex, and place of residence, were recorded

Study Population and Sample Design

This cross-sectional study aims to assess the prevalence rate of SLE in Tripoli, Libya. A total of 140 participants, 108 were positive SLE patents, while 32 patients were negative (control group). Positive cases including both genders (101 females and 7 males) aged between 17 and 59 years, were included. Patients with a history of liver disease, renal disease (other than SLE), rheumatoid arthritis, ankylosing spondylitis, inflammatory bowel disease, psoriasis, or malignant disease, as well as those with a history of taking anticoagulants, chemotherapy, or a recent blood transfusion, were excluded from the study.

Diagnostic Tests and Blood Screening

Diagnostic Tests and Blood Screening include a CBC and ESR. Each patient and control patient had five milliliters of blood extracted under aseptic conditions—two milliliters for the CBC evaluation and three milliliters for the ESR. The hemogram involved counting white blood cells (WBC), platelets, monocytes (N), lymphocytes (L), and 2C hemoglobin (Hb) content (gm/dl). All samples were collected in an EDTA tube, and a simple mixing was made until the patient's blood was well mixed with the anticoagulant. The sample was then placed in the SYSMEX XP-300 device for the CBC test. For the ESR test, the sample was purged using

Khalij Libya Journal of Dental and Medical Research. 2025;9(1):97-102
https://doi.org/10.47705/kjdmr.25911015
eISSN:2708-888X

the EDTA tube and placed in the tube for the ESR test, which contained sodium citrate. The tube was left in a vertical position for 60 minutes. The normal range for ESR is between 0 and 22 mm/hr for men and between 0 and 29 mm/hr for women. For CBC, the normal ranges are between 12-15 g/dl for women and between 15.5 and 18 g/dl for men. The neutrophil ratio should be between 40 and 80%, while the lymphocyte ratio should be between 20 and 40%, and the monocyte ratio between 2 and 7%.

Statistical analysis

In this study, we used the statistical package for the social sciences (SPSS 20) software to analyze the data. Data were presented as mean ± standard deviation (± SD), as well as range or frequencies (number of cases) and percentages when appropriate. A p-value less than 0.05 was considered statistically significant.

Results

The sample size was 140 participants in this cross-sectional case-control study. By using the ESR antibody marker, the result showed that 108 patients (76.64%) were positive (SLE patients), while 32 patients (23.35%) were negative (control group). This study used different values, including Std: deviation, T-Test, and P-Value. Gender-wise distribution was 93.52% females and 6.48% males. The percentage of SLE was highest in the age group 27 – 36 years (34.26%) followed by 37 – 46 (23.15%), and the mean age of the patients was 35.72 ± 10.437 years (Table I). The mean ESR (71.656±29.551) was significantly increased in SLE patients compared to the control (14.968±6.507) ($p < 0.001$*) (T-Test -10.527). While mean Hgb (11.439±1.114) was decreased compared to the control (13.770±1.071) ($p < 0.000$*) (T-Test -5.917). The relationship between monocytes and neutrophils in two groups of SLE was also investigated by comparing SLE patients with the control group. The mean Monocytes (8.430±2.494) and neutrophils (83.825±6.176) were significantly increased in SLE patients compared to the control (4.445±1.536 and 60.685±7.216, respectively) ($p < 0.001$) *) (T-Test -7.863 and -11.589, respectively). The relationship between Lymphocytes in SLE patients was investigated in the current study, there were no significant differences in Lymphocytes between SLE patients and control subjects. In SLE patients, the mean lymphocytes were (29.430±13.363) and in control, 32.805±4.534 (p 0.298) (T-Test 1.071) (Table II).

Table I. Distribution of the subjects in SLE patients (N=108) according to gender and age.

	Age (year)				**Mean ±SD**
	17 – 26	27 -- 36	37 -- 46	47 – 59	
Frequency (N)	25	37	25	21	35.72±10.437
Percentage (%)	23.15	34.26	23.15	19.44	
Gender					
Female	(n=101)	93.52%			
Male	(n=7)	6.48%			

Data were shown as The mean age of the patients (mean±SD) , frequency (N), percentage (%) and gender.

Table II: Hematological parameters of the subjects in both groups (n=137)

Parameters	**(n=108) SLE patients**	**Control (n=32)**	**T -Test**	**P -Value**
ESR (mm in 1st hour	71.656±29.551	14.968±6.507	-10.527	0.001*
Hgb	11.439±1.114	13.770±1.071	5.917	0.000*
Monocytes	8.430±2.494	4.445 ±1.536	-7.863	0.000*
Lymphocytes	29.430±13.363	32.805±4.534	1.071	0.298
Neutrophils	83.825±6.176	60.685±7.216	-11.589	0.000*

Data were shown as mean±SD. Statistical analysis was done by unpaired Student's 't' test. Control= Healthy subjects, SLE= Systemic lupus erythematosus, ESR= Erythrocyte sedimentation rate, Hgb= Hemoglobin level, values are significant at $p < 0.05$.

Discussion

SLE is a multifactorial disease, which is autoimmune in origin and characterized by autoantibody production. Generally, the etiology of the disease is unknown, hematological abnormalities are common in patients with SLE [19,20]. In the Laboratory, different hematological tests are routinely used to assess SLE disease activity in clinical practice, and some are included in the disease activity measures such as CBC), ESR additionally to immune and genetic tests [21]. Results of the study show that the most susceptible age for SLE is 27 - 36 followed by 37 - 46. According to the gender clearly, SLE in females is more common than males (females,93.52% and males, 6.48%). Accordingly, various studies estimated that gender differences

related to incidence among patients with SLE were especially evident in the reproductive age groups (20-40 year). Most studies report an impressive female-to-male ratio (9:1). In contrast to males, the occurrence of SLE increases intensely in post-pubertal females and still high during the reproductive ages. It is hypothesized that due to hormonal changes in the reproductive period [22,23,24].

Typically, SLE is hard to diagnose because its symptoms can be vague. Many researchers have assessed independently different hematological markers that may reflect disease activity. In addition, unlike other diseases, doctors cannot diagnose it with a single lab test. ESR is a marker used as an indicator of inflammation. Inflammation could indicate lupus activity. ESR and the association with inflammation is complex, however, ESR has a critical role in lupus activity. This test could be used to monitor inflammation, which could indicate changes in disease activity or response to treatment [25].

Red cell distribution width (RDW) positively correlates with SLEDAI score and 24 h urinary proteins, so it reflects disease activity as well as renal affection, but the lymphocytic and leukocyte count had no significant correlation with these activity indices. Therefore, RDW could be used as a surrogate marker of the inflammation rather than the neutrophil and lymphocyte ratio (NLR). It is a simple and easy testing included in CBC, thus RDW can be used as a possible indicator to assess disease activity. It can reflect the ongoing inflammatory state in this autoimmune disease and needs to be studied in various aspects of this complex autoimmune disorder in relation to different organ affection in a large group of patients [25].

The results of the laboratory data showed that the SLE patients have statistically significantly lower mean hemoglobin levels and a clear increase in ESR. Study in Bangladesh 2018 observed significantly higher ESR levels in SLE patients compared to the control group ($p < 0.001$). On the other hand, RBC, WBC, and Platelet Counts were significantly lower in SLE patients compared to the control group ($p < 0.001$). This study concludes that CBC and ESR were altered in SLE patients and it was related to the duration of disease [26]. An Indonesian study in 2023 showed that anemia is a risk factor for worsening in SLE patients and plays a role in indicating lupus activity [27]. In active lupus, most of the research reported that anemia is both a short-term and long-term prognostic factor for the assessment of the disease. Also, a high ESR without clear reasons for it as infection, indicates that lupus is active [26,28,29,30].

Hematologic manifestations in lupus are relatively frequent and range from mild to severe. During active SLE, all blood cell lines can be affected. These disorders may be caused by the lupus disease itself or by medications for the disease [31]. interestingly, abnormal peripheral neutrophils are an indicator in SLE patients, and neutropenia is associated with underlying SLE activity and might increase susceptibility to infection [32]. Typically, a disease flare is accompanied by an increasing ESR, and a dropping complement and lymphocyte [2]. During SLE activity, studies stated that the Neutrophil to lymphocyte ratio (NLR) correlates with disease activity. NLR is raised in patients with SLE compared to healthy persons and is associated with key immunopathological events. Neutrophils and lymphocytes play a significant role of SLE pathogenesis and activity [33]. Laboratory findings may change according to Lupus types; therefore, an Egyptian study in 2020 shows that lymphopenia is a common finding in SLE patients and was significantly related with lupus nephritis, complement consumption, higher steroid doses, and cyclophosphamide administration. Lymphopenia might be a promising marker for renal involvement in SLE [34]. Another study in 2024 concluded that the studied hematological parameters were distinguishable including the active and inactive SLE patients. Cases with active SLE had higher values of NLR, monocyte to lymphocyte ratio (MLR), platelet to lymphocyte ratio (PLR), RDW and lower value of monocyte to lymphocyte ratio (MLR), compared to patients with inactive SLE. were useful for determining active disease particularly in patients with active lupus nephritis [35].

Conclusion and future respective

Hematological disorders are common findings in patients with SLE. This study showed that females are more frequent than males by 9:1 and reproductive age is the most common age. Hematological parameters CBC, ESR can be partially helpful as preliminary markers. In this study, simple parameters were used to diagnose SLE. However, more tests, such as Antinuclear antibody (ANA) test, Tissue biopsies, and urine test, should be used.

Conflict of Interest

There are no financial, personal, or professional conflicts of interest to declare.

References

1. Ali A, Sayyed Z, Ameer MA, Arif AW, Kiran F, Iftikhar A, Iftikhar W, Ahmad MQ, Malik MB, Kumar V, Dass A, Sami SA, Sapna F, Waqas N. Systemic Lupus Erythematosus: An Overview of the Disease Pathology and Its Management. Cureus [Internet]. Cureus Inc.; 2018 Sep 11;10(9). Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6235637/

2. Lupus: Causes, symptoms, and research [Internet]. 2020 [cited 2025 Mar 5]. Available from: https://www.medicalnewstoday.com/articles/323653
3. Rose T, Rose T, Dörner T, Dörner T. Drivers of the immunopathogenesis in systemic lupus erythematosus. Best Practice & Research: Clinical Rheumatology [Internet]. Best Pract Res Clin Rheumatol; 2017 Jun 1;31(3):321–333. Available from: https://pubmed.ncbi.nlm.nih.gov/29224674/
4. Crow MK. Collaboration, genetic associations, and lupus erythematosus. The New England Journal of Medicine [Internet]. Massachusetts Medical Society; 2008 Feb 28;358(9):956–961. Available from: https://www.nejm.org/doi/full/10.1056/NEJMe0800096
5. Parks CG, de Souza Espindola Santos A, Barbhaiya M, Costenbader KH. Understanding the role of environmental factors in the development of systemic lupus erythematosus. Best Practice & Research: Clinical Rheumatology [Internet]. Best Pract Res Clin Rheumatol; 2017 Jun 1;31(3):306–320. Available from: https://pubmed.ncbi.nlm.nih.gov/29224673/
6. Manson JJ, Rahman A. Systemic lupus erythematosus. Orphanet J Rare Dis. 2006 Mar 27;1:6. doi: 10.1186/1750-1172-1-6. PMID: 16722594; PMCID: PMC1459118.
7. Tian J, Zhang D, Yao X, Huang Y, Lu Q. Global epidemiology of systemic lupus erythematosus: a comprehensive systematic analysis and modelling study. Ann Rheum Dis. 2023 Mar;82(3):351-356. doi: 10.1136/ard-2022-223035. Epub 2022 Oct 14. PMID: 36241363; PMCID: PMC9933169.
8. Barber MRW, Falasinnu T, Ramsey-Goldman R, Clarke AE. The global epidemiology of SLE: narrowing the knowledge gaps. Rheumatology (Oxford). 2023 Mar 29;62(Suppl 1):i4-i9. doi: 10.1093/rheumatology/keac610. PMID: 36987602; PMCID: PMC10050933.
9. Ameer MA, Chaudhry H, Mushtaq J, Khan OS, Babar M, Hashim T, Zeb S, Tariq MA, Patlolla SR, Ali J, Hashim SN, Hashim S. An Overview of Systemic Lupus Erythematosus (SLE) Pathogenesis, Classification, and Management. Cureus. 2022 Oct 15;14(10):e30330. doi: 10.7759/cureus.30330. PMID: 36407159; PMCID: PMC9662848.
10. Justiz Vaillant AA, Goyal A, Varacallo MA. Systemic Lupus Erythematosus. [Updated 2023 Aug 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK535405/
11. Lupus UK | [Internet]. Diagnosis | Lupus UK; 2024 Oct 1 [cited 2025 Mar 11]. Available from: https://lupusuk.org.uk/diagnosis/.
12. Moulton VR, Suarez-Fueyo A, Meidan E, Li H, Mizui M, Tsokos GC. Pathogenesis of Human Systemic Lupus Erythematosus: A Cellular Perspective. Trends Mol Med. 2017 Jul;23(7):615-635. doi: 10.1016/j.molmed.2017.05.006. Epub 2017 Jun 13. PMID: 28623084; PMCID: PMC5650102.
13. Pan L, Lu MP, Wang JH, Xu M, Yang SR. Immunological pathogenesis and treatment of systemic lupus erythematosus. World J Pediatr. 2020 Feb;16(1):19-30. doi: 10.1007/s12519-019-00229-3. Epub 2019 Feb 22. PMID: 30796732; PMCID: PMC7040062.
14. How Is Lupus Diagnosed? [Internet]. [cited 2025 Mar 9]. Available from: https://www.verywellhealth.com/how-lupus-is-diagnosed-2249846
15. Bashal F. Hematological disorders in patients with systemic lupus erythematosus. Open Rheumatol J. 2013 Oct 18;7:87-95. doi: 10.2174/1874312901307010087. PMID: 24198852; PMCID: PMC3816272.
16. Team E. What Is The Link Between Blood Disorders and Lupus? [Internet]. [cited 2025 Mar 9]. Available from: https://lupus.net/blood-abnormalities
17. Abdalhadi S, Khalayli N, Al-Ghotani B, Kudsi M. Systemic lupus erythematosus disease activity and neutrophil to lymphocyte ratio and platelet to lymphocyte ratio: a Cross-Sectional case control study. Ann Med Amp Surg [Internet]. 2023 Apr 6 [cited 2025 Mar 10];Publish Ahead of Print. Available from: https://doi.org/10.1097/ms9.0000000000000477
18. Lupus Canada [Internet]. Everything Lupus:Resource Hub | Lupus Canada; [cited 2025 Mar 10]. Available from: https://www.lupuscanada.org/resources/fact-sheets/blood-disorders-in-lupus/.
19. Santacruz JC, Mantilla MJ, Rueda I, Pulido S, Rodriguez-Salas G, Londono J. A Practical Perspective of the Hematologic Manifestations of Systemic Lupus Erythematosus. Cureus [Internet]. 2022 Mar 7 [cited 2025 Mar 10]. Available from: https://doi.org/10.7759/cureus.22938
20. Sibilia J. Treatment of systemic lupus erythematosus in 2006. Jt Bone Spine [Internet]. 2006 Dec [cited 2025 Mar 10];73(6):591-8. Available from: https://doi.org/10.1016/j.jbspin.2006.09.003
21. Connelly K, Kandane-Rathnayake R, Hoi A, Louthrenoo W, Hamijoyo L, Cho J, Lateef A, Fen Luo S, Wu YJ, Li Z, Navarra S, Zamora L, Sockalingam S, Hao Y, Zhang Z, Katsumata Y, Harigai M, Oon S, Chan M, Chen YH, Bae SC, O'Neill S, Goldblatt F, Kikuchi J, Takeuchi T, Ling Ng KP, Tugnet N, Basnayake BM, Ohkubo N, Tanaka Y, Sing Lau C, Nikpour M, Golder V, Morand EF. Associations of improvement in laboratory tests with clinical outcomes in patients with active systemic lupus erythematosus: a multinational longitudinal cohort study. Lancet Rheumatol [Internet]. 2022 Nov [cited 2025 Mar 10]. Available from: https://doi.org/10.1016/s2665-9913(22)00307-1
22. Santamaría-Alza Y, Motta JZ, Fajardo-Rivero JE, Pineda CL. Systemic lupus erythematosus, gender differences in Colombian patients. Clin Rheumatol [Internet]. 2018 Jun 2 [cited 2025 Mar 10];37(9):2423-8. Available from: https://doi.org/10.1007/s10067-018-4161-8
23. Margery-Muir AA, Bundell C, Nelson D, Groth DM, Wetherall JD. Gender balance in patients with systemic lupus erythematosus. Autoimmun Rev [Internet]. 2017 Mar [cited 2025 Mar 10];16(3):258-68. Available from: https://doi.org/10.1016/j.autrev.2017.01.007

24. Schwartzman-Morris J, Putterman C. Gender Differences in the Pathogenesis and Outcome of Lupus and of Lupus Nephritis. Clin Dev Immunol [Internet]. 2012 [cited 2025 Mar 10];2012:1-9. Available from: https://doi.org/10.1155/2012/604892
25. Mohamed OS, Azmy GJ, Elfadl EM. Clinical significance of red blood cell distribution width in systemic lupus erythematosus patients. Egypt Rheumatol Rehabil [Internet]. 2020 Sep 18 [cited 2025 Mar 10];47(1). Available from: https://doi.org/10.1186/s43166-020-00037-y
26. Abira M, Akhter QS. Relationship between CBC and ESR and duration of disease in patients with systemic lupus erythematosus (SLE). J Bangladesh Soc Physiol [Internet]. 2018 Dec 26 [cited 2025 Mar 10];13(2):41-6. Available from: https://doi.org/10.3329/jbsp.v13i2.39476
27. Aji G, Sudaryo MK, Djuwita R, Sukamto S. Risk Factors of Worsening of Systemic Lupus Erythematosus in Patients at Two Tertiary Hospitals in Jakarta. Kesmas [Internet]. 2023 Aug 31 [cited 2025 Mar 10];18(3):197. Available from: https://doi.org/10.21109/kesmas.v18i3.6964
28. Samohvalov E, Samohvalov S. The Pattern of Anemia in Lupus. IntechOpen J. 2018:10.5772/intechopen.71293.
29. Aringer M. Inflammatory markers in systemic lupus erythematosus. J Autoimmun [Internet]. 2020 Jun [cited 2025 Mar 11];110:102374. Available from: https://doi.org/10.1016/j.jaut.2019.102374
30. Siegel, MD, MS CH. Hospital for Special Surgery [Internet]. Lupus Blood Test Results: What to Know | HSS Rheumatology; 2022 Feb 7 [cited 2025 Mar 11]. Available from: https://www.hss.edu/conditions_understanding-laboratory-tests-and-results-for-systemic-lupus-erythematosus.asp#:~:text=Erythrocyte%20sedimentation%20rate,-Erythrocyte%20sedimentation%20rate&text=If%20inflammation%20is%20present,%20these,suggests%20that%20lupus%20is%20active.
31. Palm M, Erkan D. Hospital for Special Surgery [Internet]. Lupus and Blood Cell Disorders | HSS Rheumatology; 2023 Feb 15 [cited 2025 Mar 11]. Available from: https://www.hss.edu/conditions_top-ten-points-about-lupus-and-blood-cell-disorders.asp
32. Littlejohn E, Calabrese C. Cleveland Clinic [Internet]. Leukopenia: Lupus Flare, Medication Effect or Infection?; 2019 Apr 16 [cited 2025 Mar 11]. Available from: https://consultqd.clevelandclinic.org/leukopenia-lupus-flare-medication-effect-or-infection
33. Han BK, Wysham KD, Cain KC, Tyden H, Bengtsson AA, Lood C. Neutrophil and lymphocyte counts are associated with different immunopathological mechanisms in systemic lupus erythematosus. Lupus Sci Amp Med [Internet]. 2020 May [cited 2025 Mar 11];7(1):e000382. Available from: https://doi.org/10.1136/lupus-2020-000382
34. Sobhy N, Niazy MH, Kamal A. Lymphopenia in systemic lupus erythematosus patients: Is it more than a laboratory finding? Egypt Rheumatol [Internet]. 2020 Jan [cited 2025 Mar 11];42(1):23-6. Available from: https://doi.org/10.1016/j.ejr.2019.04.003
35. Ahmeda HH, Faragb HS, Sobhy N. Correlation of simple hematological parameters with disease activity and damage indices among Egyptian patients with systemic lupus erythematosus. Asociaci ´on Colombiana de Reumatolog´ıa [Internet]. 2024;7. Available from: Asociaci ´on Colombiana de Reumatolog´ıa