



# Detection of Hematological and Biochemical Manifestations Associated with Women infected by Systemic Lupus Erythematosus

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Received: May 14, 2023 / Accepted: June 7, 2023/ Published: September 23, 2024

**Abstract.** Systemic lupus erythematosus (SLE) is an autoimmune disease in which the immune system attacks its own tissues. This study was aimed to investigate the hematological and chemical manifestations of SLE Iraqi women sample by focused on tests which related with SLE disease, as hematological tests that included: complete blood count (CBC) and erythrocyte sedimentation rate (ESR), also, chemical tests to evaluate renal function (blood urea and serum creatinine), and the ratio of protein calculated by urine strip only. The results (mean  $\pm$  SE) of patients vs. control showed that ESR level was 40.08 vs. 10.96 mm/hour, Blood urea was 33.41  $\pm$ 1.40 vs. 27.11  $\pm$ 0.82 mg/dl with highly significant difference at  $P \leq 0.01$ , Creatinine was 0.758  $\pm$ 0.04 vs. 0.651  $\pm$ 0.02 mg/dl, with significant difference at  $P \leq 0.05$  and protein urea percentages was 50 (83.33%) vs. 3(5.00%) with highly significant difference at  $P \leq 0.01$ . while hemoglobin was 11.37  $\pm$ 0.20 vs. 12.90  $\pm$ 0.08 g/dl, and platelets was 230.75  $\pm$ 13.86 vs. 285.83  $\pm$ 10.06  $\times 10^3 / \mu\text{l}$  with highly significant difference at  $P \leq 0.01$ , white blood cells were 7.28  $\pm$ 0.46 vs. 6.29  $\pm$ 0.13 cell  $\times 10^3 / \mu\text{l}$ , with significant difference at  $P \leq 0.05$ . It concluded that it is evident that hematological testing and SLE disease are known to be related. And may interact with a variety of biomarker results and be used to forecast disease severity, which could be beneficial for SLE patients by expanding their options for diagnosis and treatment.

**Keywords:** Hemoglobin, Proteinuria, WBC, SLE, EULAR/ACR

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## Introduction

Systemic lupus erythematosus the most typical form of lupus is characterized by the immune system attacking its own tissues, which results in extensive inflammation and tissue destruction in the organs that are afflicted. It can have an impact on the blood vessels, brain, lungs, kidneys, skin, joints, and skin; moreover, lupus cannot be cured, although it can be managed by medical procedures and lifestyle changes (1). Systemic lupus erythematosus is a systemic multi-organ autoimmune disease characterized by

autoantibody response to nuclear and/or cytoplasmic antigens (2). According to Accapezzato and colleagues (1), it is characterized by the development of many autoantibodies, with a wide range of sickness histories and symptoms, Systemic lupus erythematosus patients frequently develop hematologic abnormalities both at the time of diagnosis and during the course of the illness. Anemia, leukopenia, and thrombocytopenia are the main hematologic symptoms of SLE (3). These anomalies might be a symptom of

SLE, connected to another concurrent illness, or brought on by an SLE medication. Patients with SLE have also shown changes in hemostasis, which are probably caused by autoantibodies (4). On the other hand, some autoantibodies may impede the action of clotting factors, increasing the risk of major bleeding (5). Patients with lupus can develop renal problems. Nephritis due to deposition of immune complex in the renal glomeruli region (6). In view of the importance of the disease and its pathological sequelae, this study was designed to investigate the hematological and biochemical manifestations of SLE Iraqi women sample.

#### **Materials and methods**

This study was based on approval of ethics committee of Institute of Genetic Engineering and Biotechnology for Postgraduate Studies, University of Baghdad. Samples were collected from 60 females suffered from SLE with age ranged between 19 and 57 years from medical lab, rheumatology units in Baghdad teaching hospital at medicine city, also study employed 60 healthy volunteer females as control group, the physicians ascertained their health status which did not have any autoimmune diseases. Permission was taken from all subjects after viewing about the aim the study. The diagnosis of the SLE disease was done by the rheumatologists at the unit clinic and followed EULAR/ACR diagnostic criteria for SLE (6). Three ml of whole blood was drawn by venipuncture using an aseptic approach and a sterile disposable syringe, from each subject, and divided into three sterile tubes: EDTA, gel and tri-sodium citrate tubes. The EDTA tube was used directly after shaking, to determine the hemoglobin and WBCs count by fully automated analyzer, while the sera was separated

from in gel tube within an hour of collection by centrifuging at 5000 rpm it for 5 minutes to measure renal function test (urea and creatinine levels) by fully automated analyzer Selectra ProXS and the ratio of protein calculated by urine strip only, along with, tri-sodium citrate tubes to measure ESR test by Westergren assay technique, and routine blood testing to evaluate the complete blood count (CBC).

#### **Statistical analysis**

Results analysis was done by using the program of Statistical Analysis System-SAS (7) to estimate the effect of difference factors in work parameters. T-test was used to significantly compare between means, as well as Chi-square test was used to significantly compare between percentages (0.05 and 0.01 probability).

#### **Results and discussion**

The results of present study appeared that about 33(55%) of patients suffered from anemia and this has considered as the highest prevalence among hematological manifestations, followed by thrombocytopenia which was found in 9 (15%) of patients and leukopenia was 6 (10%) in patients. Also the results showed that mean of patients *vs.* control for ESR level was (40.08 *vs.* 10.96 mm/hour), and blood urea was (33.41 ± 1.40 *vs.* 27.11 ± 0.82 mg/dl) with highly significant difference ( $P \leq 0.01$ ), while creatinine was (0.758 ± 0.04 *vs.* 0.651 ± 0.02 mg/dl) with significant difference ( $P \leq 0.05$ ) and positive proteinuria percentages was 50 (83.33%) *vs.* 3 (5.00%) with highly significant difference ( $P \leq 0.01$ ), while only about 10(16.67%) of patients was negative in comparison of 57(95.00%) of control as illustrated in Tables (1,2). The hemoglobin level was (11.37 ± 0.20 *vs.* 12.90 ± 0.08 g/dl), but platelets was (230.75 ± 13.86 *vs.* 285.83 ± 10.06 \*10<sup>3</sup> /

$\mu\text{l}$ ) with highly significant difference ( $P \leq 0.01$ ), while the count of white blood cells were ( $7.28 \pm 0.46$  vs.  $6.29$

$\pm 0.13 \text{ cell} \times 10^3 / \mu\text{l}$ ) with significant difference ( $P \leq 0.05$ ) as shown in Table (3).

**Table 1: Comparison between patients and control groups in ESR, Urea and Creatinine**

Group	Mean $\pm$ SE		
	ESR (mm/hr)	Urea (mg/dl)	Creatinine (mg/dl)
Patients	$40.08 \pm 2.85$	$33.41 \pm 1.40$	$0.758 \pm 0.04$
Control	$10.96 \pm 0.49$	$27.11 \pm 0.82$	$0.651 \pm 0.02$
T-test	5.746 **	3.227 **	0.0957 *
P-value	0.0001	0.0002	0.0293

\* ( $P \leq 0.05$ ), \*\* ( $P \leq 0.01$ )

**Table 2: Proteinuria percentages in patients and control groups**

Group	No	Positive (+ve) No. (%)	Negative (-ve) No. (%)	P-value
Patients	60	50 (83.33%)	10 (16.67%)	0.0001 **
Control	60	3 (5.00%)	57 (95.00%)	0.0001 **

\*\* ( $P \leq 0.01$ )

**Table 3: Comparison between patients and control groups in WBC, Hb and PLT**

Group	Mean $\pm$ SE		
	WBC ( $10^3 / \mu\text{l}$ )	Hb (g/dl)	Platelets ( $10^3 / \mu\text{l}$ )
Patients	$7.28 \pm 0.46$	$11.37 \pm 0.20$	$230.75 \pm 13.86$
Control	$6.29 \pm 0.13$	$12.90 \pm 0.08$	$285.83 \pm 10.06$
T-test	0.9597 *	0.4328 **	33.925 **
P-value	0.0451	0.0001	0.0017

\* ( $P \leq 0.05$ ), \*\* ( $P \leq 0.01$ )

Patients with SLE frequently experience hematological problems, which can be a symptom or an indication of the illness. Leucopenia, anemia, and thrombocytopenia are frequently the most significant clinical symptoms (8,9). The presented study appeared about 33(55%) of patients suffered from anemia and this has considered as the highest prevalence among hematological manifestations, followed by thrombocytopenia found in 9(15%) and leukopenia in 6(10%) patients. The 10% of patients who had leukopenia was roughly consistent with numerous earlier investigations. More recent sources indicate higher percentages of leucopenia such as, in 2013 evaluation research (10) found that 57.3% of SLE patients had leukopenia (11) in comparison to patients in

Pakistan (12). Anemia was identified in 55% of SLE patients in the current study. Regarding thrombocytopenia, it was discovered in 15% of patients in this study, which was higher than the 6% observed in an earlier Pakistani study (12). The pathophysiological mechanisms behind the hematological symptoms of SLE disease and their causes are still incompletely understood (13). Leukopenia is a consequence of an autoimmune process in the bone marrow, while anemia could be explained by many mechanisms, such as the anti-erythrocyte antibodies (warm-type IgG, and antiphospholipid (aPL) antibodies) which are associated with Coombs-positive anemia (8). Additionally, it has been discovered that hepcidin overexpression and CD55 and CD59 expression are expressed at lower

levels in SLE patients, due to CD55 and CD59 were membrane proteins that protect against complement-induced cell lysis, so any decrease in their expression results in autoimmune hemolysis, respectively (14). In roughly 50% of cases of SLE, the hepcidin prevents iron from integrating with RBC for development, causing anemia (12).

For thrombocytopenia, several mechanisms were suggested like when the bone marrow impaired production of the platelets, reservation of PLTs in the spleen, or accelerated the destruction of PLTs in the peripheral circulation (4).

The ESR is a non-specific and indirect test for assessing the body's overall inflammatory response, which comprises three main stages (15). ESR is thought to be a beneficial inflammatory parameter due to many reasons included chronic inflammatory response and increased immunoglobulin synthesis cause ESR to rise in SLE patients (16), which is a symptom of CKD and is indirectly brought on by proteinuria due to its capacity to trigger glomerular inflammation (17).

A poor glomerular filtration rate (GFR) is caused by multi vascular damage, which is associated with persistently elevated proteinuria (18,19). Proteinuria causes structural or functional changes in kidney cells. Further protein diseases result from its precipitation in the proximal and distal collecting ducts or from subsequent fibrosis, which decline the kidneys function gradually (19). Proteinuria is regarded as a risk factor for the advancement of CKD because of the pro-fibrotic and pro-inflammatory effects it has on the kidney (21,22).

### Conclusion

In conclusion, it is evident that hematological testing and SLE disease are known to be related. And may interact with a variety of biomarker

results and use to forecast disease severity, which could be beneficial for SLE patients by expanding their options for diagnosis and treatment.

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