# Study on Some Parameters and Anti-Rubella Igg in A Sample of Systemic Lupus Erythematosus Patients Marwah Mohammed Jasim<sup>1</sup>, Dunya Fareed Salloom<sup>1</sup>

Department of Biology, Collage of Science, University of Baghdad, Iraq \*Corresponding author: marwamohammed90n@gmail.com, mobile: +9647707373618

## ABSTRACT

**Background:** Systemic lupus erythematosus (SLE) is an autoimmune syndrome causing both organ and cell damage done by tissue-binding autoantibodies and immune complexes. Skin, joints, kidneys, lungs, and the neurological system can all be affected by this condition. Viral infections are main environmental variables in the pathogenesis of SLE, and they can cause illness initiation and recurrence, as well as modify clinical phenotypes.

**Objective:** The aim of this study was to measure levels of anti-rubella virus IgG in serum of Iraqi patients with SLE disease and to measure the levels of some markers including S. creatinine, blood urea, aspartate transaminase (AST), alanine aminotransferase (ALT), and the reasons for their elevation in serum of patients.

**Materials and Methods:** This study was conducted in Baghdad Teaching Hospital and Typical Rheumatology Unit through the period from October 2021 to January 2022. 103 Iraqi patients with SLE disease and 50 healthy Iraqi individuals were included as control group Blood samples were collected and the anti-rubella IgG for all subjects (for 57 patients and 34 control) was measured by the sandwich enzyme-linked immune-sorbent assay technology. Serum creatinine, blood urea, AST and ALT were measured using the automated method.

**Results:** Anti-Rubella IgG, B. urea, S. creatinine, AST and ALT were significantly higher in patients' group compared to control group. While, there was non- significant difference regarding age between studied groups.

**Conclusion:** Anti rubella virus IgG and some parameter can be used as markers for SLE disease activity.

Keywords: Systemic lupus erythematosus, ELISA, Rubella.

## INTRODUCTION

Systemic lupus erythematosus (SLE) is a systemic chronic autoimmune condition characterized by the onset of autoantibodies, immunological dysfunction, inflammation, and a variety of symptoms <sup>(1)</sup>. Several aspects, especially genetic predisposition, environmental triggers, and hormonal milieu, each play role in disease development and manifestation <sup>(2)</sup>. Arthritis, oral ulcers, fever, blood disorders, serositis, photosensitivity, renal involvement, malar rash, immunological manifestations, discoid rash, neurologic disorders, and hair loss are all typical signs of SLE illness <sup>(3)</sup>. The estimated frequency of lupus in the global population ranged from 6.5 to 178.0 cases per 100,000 individuals. However, the prevalence rate varies greatly by gender, race, and geography, with SLE incidence rates ranging from about 0.3-23.7 per 100,000 person-year <sup>(4)</sup>.

It is important to know about rubella virus. It is a single-stranded RNA togaviridae family virus with a nucleo-capsid made of polypeptide C (C protein) and covered by a lipid-containing envelope. The outer envelope includes many copies of two virus-specific polypeptides (E1, E2) that are essential for viral virulence and immunity <sup>(5)</sup>.

A slight form of measles, usually termed as 'German' (thus, 'German') measles. The infection is marked by a rash, fever, and lymphadenopathy <sup>(6)</sup>. Most rubella infections lead to long-lasting immunity mediated by antibodies it is countered by both humoral and cell-mediated immune responses. IgM and IgG antibodies are identified during infection <sup>(7)</sup>. Autoimmune disorders and viral infections have long been related. These infections frequently occur as a

result of inflammation in the target organ. Bystander activation, molecular mimicry, and viral persistence are three processes that are frequently utilized to understand the link between autoimmune and viral infection <sup>(8)</sup>. The aim of the current study was to estimate the anti-rubella IgG and other parameters such as bl. urea, S. creatinine, ALT and AST in the serum of Iraqi patients with SLE.

## MATERIALS AND METHODS

Blood samples of 103 cases with a recognized diagnosis of SLE based on the information about clinical symptoms and laboratory tests conducted in Baghdad Teaching Hospital and Typical Rheumatology Unit. In addition, a control group included 50 apparently healthy subjects in term of non-SLE whose ages ranged from 12 to 60 years.

Laboratory tests were performed for 57 patients and 34 healthy controls samples to assess disease activity including S. creatinine, ALT, AST and blood urea by using the automated method (Roche, Germany), also measuring of anti-rubella IgG by using sandwich enzyme-linked immunosorbent assay (Monocent, USA). Also, demographic data of the both subject groups were recorded.

## **Ethics approval:**

An approval of this study was obtained from University of Baghdad Academic and Ethical Committee. Informed consents of all patients was taken. This study was carried out in accordance with the World Medical Association Code of Ethics (Declaration of Helsinki) for studies involving humans.

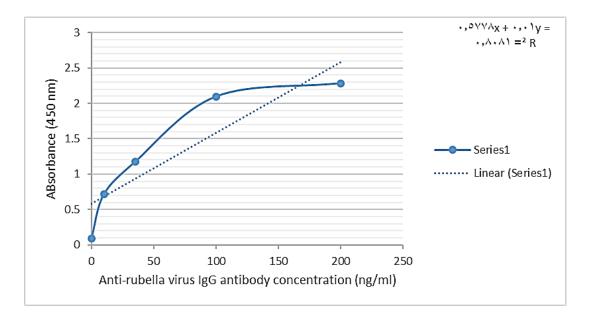


Figure 1: Standard curve of anti- rubella IgG antibody concentration (ng/ml).

## Statistical Analysis

The IBM SPSS version 26.0 (IBM Corp. Released 2019) was used to calculate the linearity, homogeneity, and normal distribution tests were done for study parameters (anti-rubella IgG serum level, age, blood urea, S. creatinine, ALT and AST). In addition, the calculation of the mean, standard error, and the probability were calculated to determine the statistically significant difference.  $P \leq 0.05$  was considered significant.

## RESULTS

Table (1) showed no significant differences in age between SLE patients and the control group.

Groups	Age mean ± SE (Years)	Probability
Patients group	$35.74 \pm 1.59$	0.06
Control group	$41.09\pm2.38$	

There was a significant difference in the levels of bl urea, S. creatinine, ALT and AST between the healthy control group and SLE patients as shown in table (2) and table (3).

**Table (2)**: Comparison between SLE patients and control regarding bl. urea and S. creatinine

Renal test	SLE group	Control group	<i>p</i> - value
B. urea	$46.46 \pm$	23.34 ±	2.52 x
(mg/dl)	1.30	2.0	10-16
S. creatinine	1.65 ±	$0.65 \pm$	1.11 x
(mg/dl)	0.28	0.19	10-39

**Table (3):** Comparison between SLE patients and control regarding ALT and AST

~					
	Liver	SLE	Control	<i>p</i> - value	
	function test	group	group		
	ALT (u/l)	$50.47 \pm$	$14.56 \pm$	5.59 x 10 <sup>-22</sup>	
		1.57	2.42		
	AST (u/l)	53.09 ±	16.13 ±	9.79 x 10 <sup>-35</sup>	
		1.26	1.10		

As shown in table (4), there was a significant difference in anti- rubella IgG serum level among studied groups.

**Table (4):** Comparison between healthy control and

 SLE patients regarding anti- rubella IgG level in

 serum

Groups	Anti-rubella IgG antibody level mean ± SE (mg/ml)	Probability
Patients group	$141.08\pm4.17$	2.35 x 10 <sup>-24</sup>
Control group	$48.22 \pm 4.90$	

## DISCUSSION

In this study the statistical analysis showed that there was no significant difference (p > 0.05) between SLE group and control group regarding mean age. The current study is consistent with prior research findings indicating that SLE can be present at any age <sup>(9)</sup>. it is most frequently diagnosed between ages 15 and 45 <sup>(10)</sup>.

The finding of this study suggested that antirubella IgG antibody may be contributory in aetiopathogenesis of SLE. This result is in agreement with other study. Because of the high level of virus seropositivity that has been discovered. Several research have focused on the function of viral infection in the presence of autoimmune illnesses such as SLE. Furthermore, it is proposed that viruses have the ability to trigger autoimmune problems <sup>(11)</sup>. So, it was reported that anti-rubella IgG viral antibodies may be implicated in the etio-pathogensis of nephritis and arthritis in SLE cases <sup>(12)</sup>. Viral infections are significant environmental variables in the pathogenesis of SLE, and they can cause clinical initiation and recurrence, as well as modify clinical traits. A diverse set of viral-mediated immunomodulatory pathways host immune system, can dramatically affect the host immune system and interact to influence B-cell differentiation in SLE pathogenesis <sup>(13)</sup>.

In addition, abnormal levels of bl. urea, S. creatinine, ALT and AST have been noticed in SLE patients compared to the control group. Perhaps the reason for inflammatory conditions is microbial infection. This is in agreement with what was found by some studies. Assessment of some immunological markers in Iraqi patients with Systemic Lupus Erythematosus and Lupus Nephritis (LN) showed increased levels of blood urea (BU) and creatinine (Cr), in groups of patients (SLE with LN, SLE without LN) in comparison with controls <sup>(14)</sup>. A recent study proved that one of the factors associated with loss kidney function was doubled their serum creatinine (15). SLErelated hepatitis (LH) is a separate entity that affects 3-8% of SLE patients and is characterized by subclinical and slight elevations of liver enzymes. Although the specific mechanism of LH-induced liver injury is unknown, a great body of research demonstrates that LH is due to SLE activity in the majority of patients and SLE therapy reduces liver enzymes <sup>(16)</sup>.

Hepatic lesions caused by SLE pathogenetic processes are assumed uncommon, although multiple studies have revealed that SLE patients have a 25-50% probability of acquiring abnormal liver tests over their lifetime. Common causes of liver enzyme abnormalities in SLE patients include hepatotoxic medications, concurrent viral hepatitis, non-alcoholic fatty liver disease, hepatic arteritis, and nodular regenerative hyperplasia. In 19.4% of the patients, the reason of liver enzyme abnormalities could not be confirmed. Several studies have identified SLE individuals with abnormal liver enzymes but no diagnostic results <sup>(17)</sup>. In these studies, the cause of liver enzyme abnormalities was SLE itself, with biochemi\cal abnormalities correlating with disease activity.

## CONCLUSION

The conclusion of this study has suggested that the virus and some parameters can be used as markers for SLE disease activity.

## REFERENCES

- 1. Sallam R, El-Sherbeeny A, El-Sayed H *et al.* (2021):Serum level of CXCL 12 in patients with systemic lupus erythematosus: Is it worthy for predilection of lupus nephritis? Egyptian Rheumatologist, 43 (1): 71–75.
- 2. Fava A, Petri M (2019): Systemic lupus erythematosus:

Diagnosis and clinical management. Journal of Autoimmunity, 96: 1–13.

- **3.** Tayel S, Muharram N, Fotoh D *et al.* (2021): Prognostic Impact of Genetic Variants of MECP2 and TIRAP on Clinical Outcomes of Systemic Lupus Erythematosus with and without Nephritis. Biomolecules, 11(9): 1378.
- 4. Nasonov E, Soloviev S, Davidson J *et al.* (2014): The prevalence and incidence of Systemic Lupus Erythematosus (SLE) in selected cities from three Commonwealth of Independent States countries (the Russian Federation, Ukraine and Kazakhstan). Lupus, 23 (2): 213–219.
- 5. Mawson A, Croft A (2019): Rubella virus infection, the congenital rubella syndrome, and the link to autism. International Journal of Environmental Research and Public Health, 16(19): 16604601.
- Mali A, Giri P (2018): A mini review on rubella virus. Acta Scientific Medical Sciences. Prevention., 2 (9): 10– 14.
- Altman A, Szyper-Kravitz M, Agmon-Levin N et al. (2012): Prevalence of rubella serum antibody in autoimmune diseases. Revista Brasileira de Reumatologia, 52 (3): 307–318.
- 8. Christen U, Hintermann E, Holdener M et al.(2010): Christen, U. et al. (2010) 'Viral triggers for autoimmunity: Is the "glass of molecular mimicry" half full or half empty?', Journal of Autoimmunity, 34 (1): 38–44.
- **9.** Gorial F (2021): Assessment immunological test with creactive protein in a newly diagnosed systemic lupus erythematosus patients. Turkish Journal of Physiotherapy and Rehabilitation, 32 (3): 2651-4451.
- **10.** Connelly K, Morand E (2021): Systemic lupus erythematosus: a clinical update. Internal Medicine Journal, 51 (8): 1219–1228.
- **11. Muhsin J** (2017): Study the possible Seropositivity connection of EBV, Rubella Virus & CMV infection with four groups of autoimmune diseases in sample of Iraqi patients. Journal of Al-Nisour University College, 2 (2): 108–117.
- **12.** Hafedh A, Krikor M, Hussein A (2019): Autoantibody Profile in Systemic Lupus Erythematosus Patients. Journal of Physics, Conf. Series, 1294 (6): 062006.
- **13. Iwata S, Tanaka Y (2022):**Association of Viral Infection With the Development and Pathogenesis of Systemic Lupus Erythematosus. Frontiers in Medicine, 9 : 1–10.
- 14. Ibrahim N, Allawi A , Ghudhaib K *et al.* (2020): Estimation of some immunological markers of Iraqi patients in systemic lupus erythematosus with lupus nephritis. Prof.(Dr) RK Sharma, 20 (4): 4668.
- **15.** Zavala-Miranda M, Perez-Arias A, Márquez-Macedo S *et al.* (2022): Characteristics and outcomes of a Hispanic lupus nephritis cohort from Mexico. Rheumatology, 61 (9): 3507-38.
- **16.** Afzal W, Haghi M, Hasni S *et al.* (2020): Lupus hepatitis, more than just elevated liver enzymes. Scandinavian Journal of Rheumatology, 49 (6):427–433.
- **17.** Efe C, Purnak T, Ozaslan E *et al.* (2011): Autoimmune liver disease in patients with systemic lupus erythematosus: A retrospective analysis of 147 cases. Scandinavian Journal of Gastroenterology, 46 (6): 732–737.