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Evaluation of Parvo Virus B19 infection in systemic lupus erythematosus patients

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ABSTRACT

Background: Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disorder. Its etiology involves genetic and environmental factors, leading to autoantibody production. Infections, particularly viral agents like Parvo Virus B19 **(**B19V), contribute to SLE pathogenesis, triggering flares and autoimmune responses. Our goal was to find out how common B19V infection is in SLE patients and whether there is a correlation between B19V viremia and the severity and activity of the disease. **Methods**: This prospective study carried out on 90 participants were divided into two groups: 60 SLE patients who met Systemic Lupus International Collaborating Clinics (SLICC) classification criteria and 30 healthy controls. Clinical assessments (*medical history, Clinical examination, laboratory* investigations), B19V detection via nested PCR and IgG, IgM anti B19V antibodies by ELISA were conducted. **Results:** There was significant differences between SLE patients and controls regarding B19V PCR positivity (35% vs. 10%, p = 0.011) and B19V IgG positivity (40% vs. 10%, p = 0.009), but no significant differences in B19V IgM positivity. Laboratory parameters did not differ between SLE patients with or without B19V DNA. Additionally, no significant correlations were found between B19V IgG and IgM titer and the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) score. **Conclusion:** This study suggests that B19V may contribute to the exacerbation of SLE symptoms.Although its direct role in the disease's clinical presentation remains unclear due to the overlap of symptoms and lack of significant differences in clinical and laboratory parameters.

Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by systemic inflammation. Although its exact cause is unknown, it results from the interaction of environmental factors and genetic predisposition, leading to autoantibodies production that target self-antigens. The disease predominantly affects women [1].

SLE presents with diverse symptoms, including fever, joint swelling, lymphadenopathy, pleuritic chest pain, oral ulcers, fatigue, alopecia, and malar rash. Periods of remission with minimal symptoms are common [2].

Infections contribute significantly to morbidity and mortality in SLE patients, either by triggering disease onset in genetically predisposed individuals or by inducing flares in known cases.The

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pathophysiology of SLE is linked to a number of microbial entities, including viruses, bacteria, fungi, and parasites, via mechanisms like molecular mimicry and bystander activation [3,4].

Certain pathogens, including viruses and bacteria, can act as superantigens, promoting autoimmune responses by activating autoreactive T and B cells. Production of autoantibodies and cytokines such as interferon-alpha (IFN-α) by Infected B lymphocytes, contributing to autoimmune and inflammatory pathways.B19V has been implicated in the development of SLE [1].

B19V is a small, non-enveloped virus belonging to the *Erythroparvovirus* genus [5].Its single-stranded DNA genome, approximately 5,600 nucleotides in length, encodes three primary proteins: the nonstructural protein NS1 and the structural proteins VP1 and VP2 [6].The NS1 protein, comprising 671 amino acids, is essential for viral DNA replication and has pro-inflammatory effects [7].

VP1 contains a unique region (VP1u), comprising 277 amino acids, distinct from VP2.This region is exposed on the surface of infected cells and is the target of neutralizing antibodies [8,9].

B19V can be transmitted through respiratory droplets, blood transfusion, or vertically from mother to fetus [10]. It can induce transient autoimmune responses that mimic SLE, with manifestations including fever, arthritic pain, myalgia, cytopenia, pleuritis, myopericarditis, acute hepatitis, and malar rash [11]. B19V has also been associated with aplastic crises in patients with hemolytic diseases and persistent pure red cell aplasia in immunocompromised persons [11].

In addition to SLE, this virus has been linked to a number of autoimmune diseases, such as rheumatoid arthritis, polymyositis, and primary biliary cirrhosis [11]. The interaction between B19V and the immune system in SLE patients remains a subject of intense research. The virus is capable of infecting and destroying erythroid progenitor cells in the bone marrow, causing hematologic abnormalities, a feature that is also common in SLE patients. Moreover, B19V can stimulate an immune response that may mimic or trigger autoimmune activity. This makes it difficult to discern whether a patient's symptoms are due to a viral infection or an autoimmune flare-up. The virus may also play a role in triggering the onset of SLE in genetically predisposed individuals or in exacerbating disease activity in those already diagnosed. Therefore, it is important to investigate the relationship between B19V infection and SLE disease progression.[12]

The aim of this work was to assess the prevalence of B19V infection among patients with SLE in comparison with healthy controls and to determine the association between B19V viremia and disease's activity and severity in Menoufia University Hospitals.

Patients and Methods

This prospective study was carried out at Menoufia University Hospitals' Clinical Pathology Department's Microbiology unit in collaboration with Rheumatology and Rehabilitation Department, Faculty of Medicine, Menoufia University; in the duration between December 2022 and June 2024.

Ethical approval for the study protocol was obtained from the ethics committee of the Faculty of Medicine, Menoufia University and an informed consent was obtained from each participant enrolled in the study. (IRB number: 2/2023 CPATH48).

It was conducted on 90 persons and categorized into two groups:

Patients Group: consisted of 60 patients diagnosed with SLE who met Systemic Lupus International Collaborating Clinics (SLICC) classification criteria [13].and ages ranged between 16-65 years.

Control Group: included 30 healthy individuals who were age- and gender-matched with the patients Group.

The Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) was used to evaluate the severity and activity of the disease [14].

Every participant underwent a detailed and systematic evaluation.

Laboratory investigations: were conducted and categorized into routine and specific tests to ensure a comprehensive assessment of the participants' health status.

Blood sampling: eight ml of blood sample were collected from each person under complete aseptic condition by clean venipuncture. The following investigations were done:

Routine investigations including: CBC was measured by Sysmex1 XN-1000, liver function, kidney function & protien/creatinine ratio tests were done using Beckman Au counter (680) Chemistry Autoanalyzer1 using kit supplied by Beckman, ESR was measured by Westeren method, CRP was

measured using Mispa-i2 nephelometer, ANA and anti-dsDNA were measured by ELISA, C3 & C4 were measured by Nephstar analyzer using quantitative turbidimetric assay.

Specific investigations

-Quantification of anti B19V IgM & IgG antibodies serum levels: According to the manufacturer's instructions, the Human (IgG & IgM of B19V) ELISA Kit, given by Sunred, Shanghai, China, with Catalogue Numbers 201-12-211 &4201-12-2113, was used to detect the level of IgG & IgM of B19V.

-Detection of B19V DNA by nested polymerase chain reaction (PCR): First, B19V DNA was extracted by commercially available spin-column1 technique kit for DNA extraction from human whole blood. HigherPurity™ Blood & Cell culture DNA Isolation Kit and procedure was done according to the manufacturer's instructions. The extracted DNA was stored in aliquots at - 80º C till performing PCR. Nested PCR was done according to Zerbini et al.[15]

10µl of DNA template, 2µl of nucleotide primers (F1&R1) (Table 1), 25µl of master mix, and 13µl of nuclease-free water were all included in the 50µl PCR mix used in the first round of amplification.

The thermocycler condition After an initial denaturation step of 5 min at 95°C,the first-round PCR amplification was performed.Then 2µl of first-round product was added to a second 50µl PCR mix.The second round reaction mix contained the same constituents as the first-round mix,but each second primer (F2&R2)was substituted for each first primer, 35 cycles of first- and second-round amplification were carried out: One minute at 95°C, one and half minute at 55°C, and one minute at 72°C.Ten-microliter of second-round PCR products were then analyzed by electrophoresis on a 2% agarose gel.

Statistical Analysis

The collected data were tabulated and analyzed using an IBM-compatible personal computer with the Statistical Package for the Social Sciences **(SPSS)**,version 23 (SPSS Inc., 2018). Data were expressed as Number **(N)**& percentage **(%)**, while quantitative data were expressed as mean **($\bar{x}$)**& standard deviation **(SD)**.Student's t-test **(t)**, The chi-square test **(χ2)** & Fisher's Exact Test **(FE)** were used. The significance of the obtained results was judged at the **5%** level. **P** value of less than 0.05 was considered statistically ssignificant.

Results

The viral markers for B19V, a statistically significant difference was observed between the two groups in B19V PCR results (p = 0.011). Among the SLE patient group,35% tested positive for B19V PCR, compared to 10% in the control group.Similarly,B19V IgG positivity was found in 40% of the SLE patients group, whereas only 10% of the control group tested positive (p = 0.003),also B19V IgG titer was higher in SLE patients group(87.5 pg/ml) than the control group(22.6 pg/ml).However, no significant difference was noted between the two groups concerning B19V IgM positivity & titer **(Table 2&Fig 1)**.

The sociodemographic characteristics of the SLE patients group with and without B19V DNA were compared. The mean age of the group with B19V DNA was 36.1 ± 8.6 years, while the group without B19V DNA had a mean age of 36.8 ± 8.5 years, with no significant difference (t = 0.27, p = 0.788).The age range for both groups was similar,23–59 years for those with B19V DNA and 15–55 years for those without. Regarding sex distribution,14.3% of the patients with B19V DNA were male, compared to 7.7% in the group without B19V DNA, with no significant difference observed between the groups (χ2 = 0.66, p = 0.655).

There was no statistically significant difference between SLE patients with B19V DNA and those without B19V DNA regarding clinical manifestations such as fever, rash, arthritis, serositis, CNS manifestations, alopecia, vasculitis, mucosal ulcers, myositis, disease remission/activity, and disease duration. These clinical features were similarly distributed across both groups. **Table (3)**

We could not find any statistically significant difference in the laboratory data between the SLE patients with B19V DNA and those without B19V DNA. The parameters measured included RBC count, Hb levels, PLT count,WBC count, MCV, MCH), liver function markers (ALT and AST),renal function markers (UREA and creatinine),protein/creatinine ratio, ESR, CRP, and complement levels (C3 and C4).All of these markers showed no significant differences between the two groups, suggesting that the presence of B19V DNA did not have a discernible impact on these laboratory parameters in the study population.

The laboratory manifestations were compared between SLE patients with and without B19V DNA. Anemia, Thrombocytopen, ESR at the first hour,high CRP levels, macroproteinuria and microproteinuria, low C3 and C4 levels. No statistically significant differences were found in these laboratory manifestations between the two groups **Table (4)**.

Between SLE patients with B19V DNA and those without, there was no statistically significant difference in the levels or positivity of viral antibodies (IgM and IgG) **Table (5)**.

The correlation between the SLEDAI score and various factors(age,IgM titer & IgG titer) was also evaluated. There was a weak negative correlation between age and the SLEDAI score ($r = -0.24$), which was not statistically significant ($p = 0.065$). Additionally, no significant correlation was found between the SLEDAI score and IgM titer ($r = 0.05$, $p = 0.696$) or IgG titer ($r = -0.13$, $p = 0.322$) **Table (6)**.

Table 1. Sequences of the primers used in nested polymerase chain reaction for detection of B19V.

Name	**Sequences (5' > 3')**	**Product size**
F1	CTTTAGGTATAGCCAACTGG	1112 bp
R1	ACACTGAGTTTACTAGTGGC	
F2	CAAAAGCATGTGGAGTGAGG	104 bp
R2	CCTTATAATGGTGCTCTGGG	

Table 2. Viral Markers in the studied groups.

Viral Markers of B19V		**SLE Patients group (N = 60)**		**Control group (N = 30)**		**Test of sig**	**P value**
		NO.	**%**	**NO.**	**%**		
B19V PCR	Positive	21	35	3	10	**χ^2 6.39**	**0.011***
	Negative	39	65	27	90		
B19V IgM	Positive	4	6.7	1	3.3	**FE 0.42**	**0.661**
	Negative	56	93.3	29	96.7		
B19V IgG	Positive	24	40	3	10	**χ^2 8.57**	**0.003***
	Negative	36	60	27	90		
B19V IgM (pg/ml)	Mean ±SD Range Median	12.1 ± 46.9 0.10–237 0.50		7.6 ± 38.8 0.10–213 0.50		**U 0.09**	**0.925**
B19V IgG (pg/ml)	Mean ±SD Range Median	87.5 ±123.5 0.30–391 1.45		22.6 ± 72.3 0.10 –346 1		**U 2.63**	**0.009***

χ^2 =Chi-square test. *= statistically significant U= Mann-Whitney test SD= Standard deviation,FE=Fisher's exact test.

Table 3. Comparison between B19 V positive and negative SLE patients regarding clinical manifestations.

Clinical manifestations		SLE cases group				FE test (Test of sig)	P- value
		With B19V DNA (N = 21)		Without B19V DNA (N = 39)			
		NO.	%	NO.	%		
Fever		16	76.2	29	74.4	**$\chi 2$ =0.02**	**0.876**
Rash		9	42.9	10	25.6	**$\chi 2$ =1.87**	**0.172**
Arthritis		6	28.6	4	10.3	**FE=3.29**	**0.143**
Serositis		3	14.3	2	5.1	**FE=1.49**	**0.332**
CNS		0	0	5	12.8	**FE=2.94**	**0.152**
Alopecia		2	9.5	8	20.5	**FE=1.19**	**0.470**
Vasculitis		7	33.3	10	25.6	**$\chi 2$ =0.39**	**0.528**
Mucosal ulcer		4	19	11	28.2	**$\chi 2$ =0.61**	**0.435**
Myositis		3	14.3	4	10.3	**$\chi 2$ =0.22**	**0.687**
Remission/ACTIVITY **No activity** **Mild activity** **Moderate activity** **High activity** **Very high activity**		1 5 12 3 0	4.8 23.8 57.1 14.3 0	1 14 13 10 1	2.6 35.9 33.3 25.6 2.6	**$\chi 2$** **4.04**	**0.401**
SLEDAI SCORE	**Mean ±SD**	8 ± 4.7		8 ± 5.3		**U** **0.16**	**0.877**
	Range	0 −18		0 −23			
Disease duration	**Mean ±SD**	5.2 ± 4.5		6.2 ± 5		**U** **0.97**	**0.333**
	Range	1 −16		1 −21			

U= Mann-Whitney test
SD= Standard deviation
FE= Fisher's Exact Test
$\chi 2$ =Chi-square test

Table 4. Laboratory manifestations according to B19 V DNA in SLE patients group.

Llaboratory manifestations	SLE patients group				FE test (Test of sig)	P value
	With B19V DNA (N = 21)		Without B19V DNA (N = 39)			
	NO	%	NO	%		
Anemia	9	64.3	18	62.1	**0.020**	**0.888**
Thrombocytopenia	3	14.3	6	15.4	**1.75**	**0.415**
High ESR (1st hour)	15	71.4	20	51.3	**2.27**	**0.131**
High CRP	9	42.9	19	48.7	**0.188**	**0.664**
Macro proteinuria	4	19	3	7.7	**2.41**	**0.299**
Micro proteinuria	1	4.8	5	12.8	**2.41**	**0.299**
Low C3 level	21	100	39	100	**0**	**1**
Low C4 level	10	47.6	16	41	**0.242**	**0.623**

Table 5. Comparison between B19V IgM and IgG antibody in the SLE patients group.

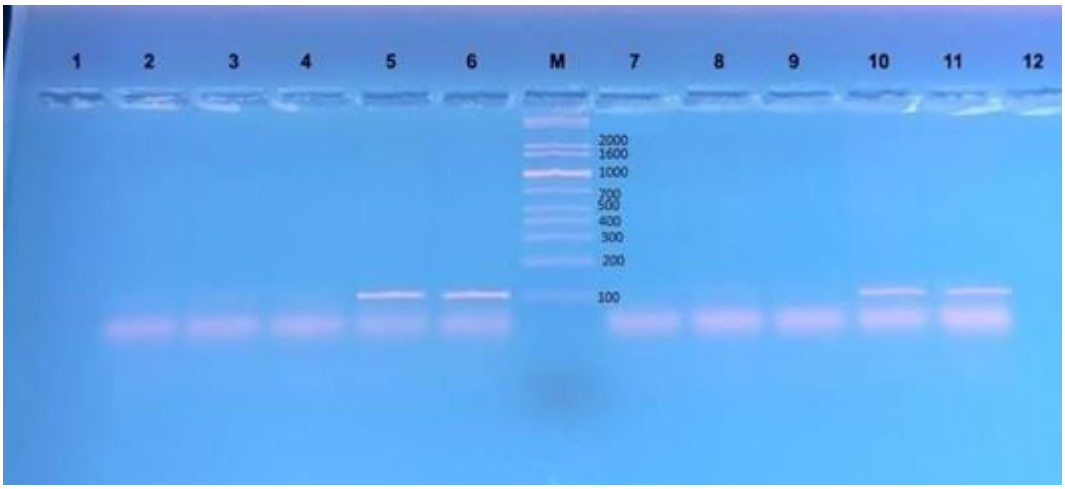
B19V		SLE patients group				Test of sig	P value
		With B19V DNA (N =21)		Without B19V DNA (N =39)			
B19V IgM (pg/ml)	Median	0.4		0.5		**U 0.41**	**0.680**
	IQR	0.2 −0.75		0.2 −0.70			
	Mean ±SD	19 ± 59.1		8.3 ± 39.2			
	Range	0.10 −217		0.10 −237			
B19V IgM	Positive	2	9.5	2	5.1	**FE 0.42**	**0.606**
	Negative	19	90.5	37	94.9		
B19V IgG (pg/ml)	Median	1.2		1.5		**U 0.32**	**0.750**
	IQR	1 −121		0.9 −196			
	Mean ±SD	75.7 ± 114.9		93.8 ± 128.9			
	Range	0.30 −321		0.40 −391			
B19V IgG	Positive	8	38.1	16	41	**χ2 0.049**	**0.825**
	Negative	13	61.9	23	59		

U= Mann-Whitney test SD= Standard deviation IQR=Interquartile Range χ2 =Chi-square test, FE=Fisher's exact test.

Table 6: Spearman correlation of SLEDAI SCORE with age,IgM titer &IgG titer in the SLE patients group.

	SLEDAI SCORE	
	r	**p-value**
Age	**-0.24**	**0.065**
IgM titer	**0.05**	**0.696**
IgG titer	**-0.13**	**0.322**

Figure 1. Agrose gel electrophoresis of amplification of B19V at 104 bp. Lane 7: negative control, lane 6: positive control, lanes 5,10 &11 show amplification product of B19V at 104 bp.



Discussion

The relationship between B19V infection and SLE is an issue of interest.The present study was carried out on 60 SLE patients.The mean age of SLE patients was 36.6 ± 8.5 years, with 90% females and 10% males (p = 1.00).

This is consistent with findings by Shafik et al.[16], who found a median age of 29 years for SLE patients, with 20% males and 80% females, and Abd El-Hamid et al.[17],who found 96.7% of SLE patients were females with a mean age of 24.2 years.El-mak et al.[18] also reported that majority of study patients were female (89%) rather than male. This result may be attributed to the predominance of SLE among females and its higher incidence in middle age suggesting that the female sex hormones

like estrogen have roles in autoimmunity including SLE.[19]

Our results showed that 35% of SLE patients tested positive for B19V PCR, significantly higher than the 10% of controls (p = 0.011). B19V IgM positivity was 6.7% in SLE patients versus 3.3% in controls (p = 0.661), with similar IgM levels (p = 0.925). B19V IgG positivity was significantly higher in SLE patients (40% vs. 10%; p = 0.003), along with elevated mean IgG levels (p = 0.009).

The findings align with Shafik et al.[16],who noted PCR positivity in active SLE cases, with or without IgM, indicating variability in acute immune responses.Also our results were in agreement with Valencia et al study [20]. in Mexico, reported the presence of IgM, IgG, and viral load in Mayan women with established SLE,as well as the high incidence of B19V in Yucatan suggest that B19V infection could be an environmental factor to reactivate or trigger SLE.

In our study, B19V IgG positivity was more prevalent in SLE patients, with higher mean IgG levels than controls. Shafik et al.[16] also observed IgG positivity in SLE, often with other markers, suggesting its role in SLE's immunological profile. In contrast, IgG positivity was lower and often isolated in controls.Also Hod et al study[21] reported that only two of 51 patients were positive for B19V DNA,but elevated titre of IgG Abs to B19V were found in 25 (49%) SLE patients, and slightly elevated IgM Abs titre to B19V were detected in 8 (15.7%) .

Our research show a higher prevalence of B19V markers in SLE patients, with significant PCR positivity suggesting an association with SLE. However, no significant differences in IgM positivity or levels were noted, indicating that it is very difficult to detect IgM positivity or levels.This could be attributed to its early infection of B19V, immunosuppressive therapies, chronic inflammation affecting antibody kinetics, or variability in viral reactivation patterns. Methodological factors, including assay sensitivity and timing of sample collection, might also influence IgM detection [22].

In contrast, Bengtsson et al.[23] showed no higher prevalence of B19V infection in SLE cases compared to the general population, also Abd El-Hamid et al.[17]found no significant difference in B19V DNA positive between SLE cases and healthy controls.

Many studies support the idea of B19V infection inducing flares of SLE and that treatment for B19V infection in SLE cases might be useful to decrease flare-ups of such a serious illness,If discontinuation of the immunosuppressive therapy is not option, the administration of immunoglobulins could be helpful to control infection.[24]

While Cope et al.[25] proposed that B19V infection might trigger autoimmune responses leading to SLE. Some researchers, such as Pavlovic et al.[26], suggested that B19V could cause symptoms resembling lupus, like vasculitis and arthralgia.These contradictory results highlight the need for further investigation into the possible involvement of B19V in the etiology and development of SLE.

In this study the laboratory data of SLE patients with (n=21) and without (n=39) viremia.Has no statistically significant differences in hemoglobin (Hb) levels,red blood cell (RBC) count, white blood cell (wBC), or platelet count (PLT).Other parameters, including MCV, MCH, ALT, AST, urea, creatinine, protein/creatinine ratio,ESR,CRP,C3, and C4 levels, showed no significant statistically differences, indicating similar laboratory profiles regardless of B19V DNA presence.

These findings are consistent with Abd El-Hamid et al.[17],who reported no significant differences in proteinuria, hypocomplementemia, ESR, CRP, or other serological markers between B19V-positive and negative SLE patients.While Hsu et al.[24] showed that individuals with B19V viremia had a considerably higher incidence of hypocomplementemia compared to those lacking B19 DNA.Also El-Saadany et al.[27]demonstrated a higher prevalence of hypocomplementemia in patients with viremia than in those without B19V.WBC counts were slightly higher in the B19V-negative group, but this difference was not statistically significant, suggesting no major impact on the immune response. Additionally, there were no significant variations in liver and kidney function markers (ALT, AST, urea, creatinine), suggesting that B19V infection does not correlate with hepatic or renal impairment in SLE patients. The lack of differences in protein/creatinine ratios, ESR, and CRP levels further supports the conclusion that B19V DNA presence does not affect systemic inflammation or kidney involvement in SLE [28].

The findings emphasize the importance of a comprehensive approach to SLE management, incorporating both clinical assessments and laboratory monitoring to guide treatment decisions and improve patient outcomes. The variability in disease activity further underscores the need for individualized care tailored to the specific needs of each patient [29].

In this study, B19V IgM and IgG levels showed no significant differences between SLE cases with and without B19V DNA (p=0.680 and p = 0.750, respectively).Median IgM and IgG levels, as well as positivity rates. IgM positivity was 9.5% in B19V-positive patients and 5.1% in negative patients (p = 0.606), while IgG positivity was 38.1% in B19V-positive patients and 41% in negative patients (p = 0.825).

In line with our results Hsu et al. [24] B19V DNA was detected in 17 of 72 (24%) SLE patients by PCR and was confirmed by Southern blotting and the prevalence of IgG and IgM anti-B19 antibodies in serum of SLE patients with B19 DNA was much lower than in patients without B19 DNA ($p<0.05$).

On the other hand, Shafik et al.[16] found 25% of SLE cases had B19V DNA, with a higher rate of positive IgM and IgG antibodies in the B19V-positive group compared to those without detectable B19V DNA. Pacheco et al.[1] also found that women with established SLE had significant prevalences of B19V infection markers, such as IgM, IgG, and viral load,suggesting that B19V infection could be reactivation factor or an environmental trigger for SLE.

Our study, shows no significant correlation between IgG and IgM levels, with the negative correlation suggesting a very weak inverse relationship that is not statistically significant. Therefore, the presence of one antibody does not predict the presence of the other in SLE patients.

Furthermore, there was a weak negative association between the SLEDAI score and age (r= -0.24, p= 0.065), which was not statistically significant, according to the correlation analysis. The SLEDAI score did not significantly correlate with either the IgM titer (r= 0.05, p = 0.696) or the IgG titer (r= -0.13, p = 0.322), indicating that neither age nor antibody levels were significantly associated with disease activity.

Hod et al.[21] found no change in SLEDAI scores between SLE cases with and without high IgG or IgM levels, which is in line with these findining.

In contrast, Shafik et al.[16] found a significant positive association between SLEDAI,viral load and IgM titer, but not with IgG titer. They also found that higher IgG levels were associated with increased disease activity, while IgM levels showed no significant correlation with SLEDAI.

Our study has some limitations, like a small sample size that restricts the generalizability of the results and the inability to establish causal relationships due to its cross-sectional design.

Conclusion:

The idea that B19V infection may be an environmental component that triggers, induces, or reactivates SLE patients is supported by our findings. Lack of anti-B19 antibodies in SLE patients may result from the immunocompromised nature of the illness or from the use of immunosuppressive medications.

Recommendation:

SLE patients should be investigated for B19V routinely as lowering the infection's rate helps preventing flare-ups of this serious illness. In individuals who are genetically susceptibl, B19V may cause idiopathic SLE or SLE-like symptom. Further research is needed to fully understand B19V's role in SLE pathogenesis and increasing sample size.

Conflict of interest

No coflict of interest.

Financial disclosure

Self-funded contributors.

Data availability

All data generated or analyzed during this study are included in this puplished article.

Authors' contribution

All authors made significant contributions to the work presented, whether in the areas of ideation, study design, implementation, data collection, analysis, and interpretation, or all of these. They also contributed to the article's writing, revising, or critical evaluation, gave final approval for the version to be published, chose the journal to which the article was submitted, and agreed to be responsible for all aspects of the work.

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