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General Medicine

LEVEL OF ANTI-DSDNA ANTIBODY IN SLE PATIENTS AND ITS RELATIONSHIP WITH DISEASE SEVERITY

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ABSTRACT **Background:** Systemic Lupus Erythematosus (SLE) is a chronic multisystem autoimmune disorder characterized by the production of autoantibodies, particularly anti-double stranded DNA (anti-dsDNA) antibodies. Anti-dsDNA antibodies are considered highly specific for SLE and are often associated with disease activity and organ involvement, especially lupus nephritis. **Objective:** To evaluate the level of anti-dsDNA antibodies in SLE patients and determine its relationship with disease severity. **Methods:** A hospital-based cross-sectional analytical study was conducted among diagnosed SLE patients attending the Rheumatology Department of a tertiary care hospital. Serum anti- dsDNA antibody levels were measured using ELISA. Disease severity was assessed using the SLEDAI score. Statistical analysis included correlation and regression analyses to determine the association between anti-dsDNA levels and disease severity. **Results:** Higher anti-dsDNA antibody levels were significantly associated with increased disease severity scores ($p < 0.05$). Patients with severe disease activity demonstrated markedly elevated anti-dsDNA titers compared to patients with mild or moderate disease activity. A positive correlation was observed between anti-dsDNA antibody levels and SLEDAI scores. **Conclusion:** Anti-dsDNA antibody levels correlate positively with disease severity in SLE patients and may serve as a useful biomarker for monitoring disease activity and predicting severe clinical manifestations.

KEYWORDS : Systemic Lupus Erythematosus, Anti-dsDNA Antibody, Disease Severity, SLEDAI, Autoimmune Disease, Lupus Nephritis

INTRODUCTION

Systemic Lupus Erythematosus (SLE) is a chronic, multisystem autoimmune inflammatory disease characterized by the production of autoantibodies and immune complex deposition, leading to widespread tissue and organ damage. The disease has a relapsing and remittingcourse and can involve almost every organ system of the body, including the skin, joints, kidneys, cardiovascular system, lungs, hematological system, and central nervous system. Because of its heterogeneous clinical presentation, SLE is often referred to as "the disease with a thousand faces." Clinical manifestations may vary from mild constitutional symptoms such as fever, fatigue, and arthralgia to severe complications including lupus nephritis, neuropsychiatric lupus, vasculitis, and hematological abnormalities[1]. SLE predominantly affects women, especially during their reproductive years, with a female-to-male ratio of approximately 9:1. The exact etiology of SLE remains incompletely understood; however, it is believed to result from a complex interaction of genetic predisposition, hormonal influences, environmental triggers, and immunological abnormalities. Factors such as ultraviolet radiation, infections, drugs, and smoking have been implicated in triggering autoimmune responses in genetically susceptible individuals. These triggers lead to loss of immune tolerance and abnormal activation of B lymphocytes, resulting in the production of pathogenic autoantibodies.

One of the most important immunological features of SLE is the formation of autoantibodies against nuclear and cytoplasmic antigens. Among these, anti-double stranded DNA (anti- dsDNA) antibodies are highly specific for SLE and play a significant role in disease pathogenesis. Anti-dsDNA antibodies are included in both the American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR) classification criteria for SLE because of their strong diagnostic value. These antibodies form immune complexes that deposit in tissues, particularly in the kidneys, leading to inflammation and organ damage. Elevated anti-dsDNA antibody levels are especially associated with lupus nephritis, one of the most serious complications of SLE[2].Disease activity in SLE is highly variable and unpredictable, with periods of exacerbation and remission. Accurate assessment of disease activity is essential for appropriate treatment planning, prevention of complications, and monitoring therapeutic response. Various clinical indices and laboratory biomarkers are used to evaluate disease activity, including complement levels, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and autoantibody profiles. Among these biomarkers, anti-dsDNA antibody levels are widely studied because fluctuations in their titers often correlate with disease activity and severity.

Several studies have reported that increased anti-dsDNA antibody levels are associated with active disease, renal involvement, vasculitis, and hematological manifestations. Rising antibody titers may precede disease flares, making anti-dsDNA a potentially useful marker for predicting disease exacerbation. However, some studies have demonstrated inconsistent correlations between anti-dsDNA levels and overall disease severity, suggesting that the relationship may vary according to ethnicity, disease duration, laboratory methods, and clinical characteristics of the study population. Consequently, the clinical significance of anti- dsDNA antibody levels as a reliable marker of disease severity remains an area of ongoing research. In developing countries, including India, limited data are available regarding the relationship between anti-dsDNA antibody levels and disease severity among SLE patients. Understanding this association in the local population may help clinicians improve disease monitoring, identify patients at higher risk of severe complications, and optimize treatmentstrategies. Furthermore, early detection of increased disease activity through reliable biomarkers may reduce morbidity, prevent irreversible organ damage, and improve quality of life in patients with SLE[3]. Therefore, the present study is designed to evaluate the levels of anti-dsDNA antibodies in patients diagnosed with SLE and to determine their relationship with disease severity. The findings of this study may contribute to better understanding of the role of anti-dsDNA antibodies in disease monitoring and may support their use as an important biomarker in the clinical management of SLE patients[4,5].

Aim

To assess the level of anti-dsDNA antibodies in SLE patients and determine its relationship with disease severity.

Objectives

1. To measure serum anti-dsDNA antibody levels in SLE patients.
2. To assess disease severity using the SLEDAI score.
3. To evaluate the correlation between anti-dsDNA antibody levels and disease severity.

Review of Literature

Several studies have highlighted the important role of anti-double stranded DNA (anti- dsDNA) antibodies in the diagnosis, assessment, and monitoring of Systemic Lupus Erythematosus (SLE). Anti-dsDNA antibodies are considered highly specific serological markers for SLE and are included in the disease classification criteria established by the American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR). These antibodies are not only valuable for diagnosis but also play a major role in disease pathogenesis through the formation of immune complexes that deposit in tissues and cause inflammation[6].

Earlier studies demonstrated that anti-dsDNA antibodies are closely associated with disease activity, particularly lupus nephritis. Research conducted by George C. Tsokos reported that elevated anti-dsDNA antibody levels were significantly correlated with active clinical manifestations of SLE, especially renal involvement. The study emphasized that patients with high antibody titers frequently exhibited increased disease activity and were more likely to develop severe organ complications. Similar findings were reported in several other studies where increased anti-dsDNA titers showed a positive association with disease activity indices such as the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI)[7].

A number of investigators have evaluated the relationship between anti-dsDNA antibody levels and lupus nephritis. It has been observed that rising anti-dsDNA titers are often accompanied by declining complement levels and worsening renal function. Some studies suggested that anti-dsDNA antibodies may serve as predictive markers for renal flare-ups, thereby assisting clinicians in identifying patients at risk for severe kidney involvement. Furthermore, patients with persistently elevated anti-dsDNA levels were found to have higher rates of hospitalization and increased disease morbidity[8].

Research by various immunological and rheumatological groups has also explored the role of anti-dsDNA antibodies in monitoring treatment response. Reduction in antibody titersfollowing immunosuppressive therapy has been associated with clinical improvement in many patients. Therefore, serial measurement of anti-dsDNA antibodies is considered useful in routine clinical follow-up of SLE patients[9].

Despite these findings, the relationship between anti-dsDNA antibody levels and overall disease severity remains controversial. Some studies have reported inconsistent or weak correlations between antibody titers and disease activity. Certain patients with clinically active disease may demonstrate low or negative anti-dsDNA levels, while others with high antibody titers may remain clinically stable. These discrepancies indicate that anti-dsDNA antibodies alone may not be sufficient to predict disease severity accurately[10].

Several factors have been proposed to explain the variability in study findings. Ethnic and genetic differences are believed to influence autoantibody production and disease expression in SLE patients. Variations in laboratory techniques used for measuring anti-dsDNA antibodies, including ELISA, Farr assay, and Crithidia luciliae immunofluorescence assay, may also contribute to differences in sensitivity and specificity among studies. Additionally, disease duration, treatment status, and environmental factors may affect antibody levels and clinical manifestations[11].

Studies conducted in Asian populations, including Indian patients, have shown a relatively higher prevalence of severe disease manifestations such as lupus nephritis. However, limited research is available regarding the relationship between anti-dsDNA antibody levels and disease severity in the Indian population. Further investigation is therefore necessary to determine whether anti-dsDNA antibodies can reliably reflect disease activity and severity in different clinical settings[12].

Overall, the literature suggests that anti-dsDNA antibodies remain one of the most important immunological biomarkers in SLE. Although their exact relationship with disease severity may vary among populations, they continue to play a significant role in diagnosis, disease monitoring, prediction of disease flares, and assessment of therapeutic response in patients with SLE[12].

MATERIALS AND METHODS

Study Design

Hospital-based cross-sectional analytical study.

Study Area

Department of Rheumatology/Medicine of a tertiary care hospital.

Study Duration

6–12 months.

Study Population

Patients diagnosed with SLE according to the: American College of Rheumatology criteria or European League Against Rheumatism classification criteria.

Inclusion Criteria

- Confirmed SLE patients aged ≥13 years.
- Patients willing to provide informed consent.

Exclusion Criteria

- Patients with overlap autoimmune syndromes.
- Pregnant women.
- Patients with active infections or malignancy.

Clinical Assessment

Detailed history and physical examination were performed.

Laboratory Investigations

- Complete blood count
- ESR/CRP
- ANA profile
- Serum complement levels (C3, C4)
- Anti-dsDNA antibody assay by ELISA
- Urinalysis
- Renal function tests

Disease Severity Assessment

Disease activity was assessed using:

SLEDAI

Weight	SCORE	Descriptor	Definition
8	❐	Seizure	Recent onset, exclude metabolic, infectious or drug causes.
8	❐	Psychosis	Altered ability to function in normal activity due to severe disturbance in the perception of reality. Include hallucinations, incoherence, marked loose associations, impoverished thought content, marked illogical thinking, bizarre, disorganized, or catatonic behavior. Exclude uremia and drug causes
8	❐	Organic brain syndrome	Altered mental function with impaired orientation, memory, or other intellectual function, with rapid onset and fluctuating clinical features, inability to sustain attention to environment, plus at least 2 of the following: perceptual disturbance, incoherent speech, insomnia or daytime drowsiness, or increased or decreased psychomotor activity. Exclude metabolic, infectious, or drug causes.
8	❐	Visual disturbance	Retinal changes of SLE. Include cytoid bodies, retinal hemorrhages, serous exudate or hemorrhages in the choroid, or optic neuritis. Exclude hypertension, infection, or drug causes.
8	❐	Cranial nerve disorder	New onset of sensory or motor neuropathy involving cranial nerves.
8	❐	Lupus headache	Severe, persistent headache; may be migrainous, but must be nonresponsive to narcotic analgesia.
8	❐	CVA	New onset of cerebrovascular accident(s). Exclude arteriosclerosis.
8	❐	Vasculitis	Ulceration, gangreene, tender finger nodules, periungual infarction, splinter hemorrhages, or biopsy or angiogram proof of vasculitis.
4	❐	Arthritis	≥ 2 joints with pain and signs of inflammation (i.e., tenderness, swelling or effusion).
4	❐	Myositis	Proximal muscle aching/weakness, associated with elevated creatine phosphokinase/aldolase or electromyogram changes or a biopsy showing myositis.
4	❐	Urinary casts	Heme-granular or red blood cell casts.
4	❐	Hematuria	>5 red blood cells/high power field. Exclude stone, infection or other cause.
4	❐	Proteinuria	>0.5 gram/24 hours
4	❐	Pyuria	>5 white blood cells/high power field. Exclude infection.
2	❐	Rash	Inflammatory type rash.
2	❐	Alopecia	Abnormal, patchy or diffuse loss of hair.
2	❐	Mucosal ulcers	Oral or nasal ulcerations.
2	❐	Pleurisy	Pleuritic chest pain with pleural rub or effusion, or pleural thickening.
2	❐	Pericarditis	Pericardial pain with at least 1 of the following: rub, effusion, or electrocardiogram or echocardiogram confirmation.
2	❐	Low complement	Decrease in CH50, C3, or C4 below the lower limit of normal for testing laboratory
2	❐	Increased DNA binding	Increased DNA binding by Farr assay above normal range for testing laboratory.
1	❐	Fever	>38° C. Exclude infectious cause.
1	❐	Thrombocytopenia	<100,000 platelets / x10^9/L, exclude drug causes.
1	❐	Leukopenia	< 3,000 white blood cells / x10^9/L, exclude drug causes.
TOTAL SCORE			

SLEDAI Interpretation

- 0–4 = Mild activity
- 5–9 = Moderate activity
- 10–14 = High activity
- ≥15 = Very high activityResults and analysis

Table 1: Distribution of Age Among Study Participants

Age Group (Years)	Number of Patients	Percentage (%)
14–18	4	5.7
19–28	25	35.7
29–38	18	25.7
39–48	17	24.2
49–58	4	5.7
59–68	2	2.8
>69	0	0
Total	70	100

Mean Age: 37.14 ± 17.36 years

Table 2: Distribution of Study Participants by Gender

Gender	Number of Patients	Percentage (%)
Female	67	95.7
Male	3	4.3
Total	70	100

Table 3: Distribution of Study Participants by Duration of Disease

Duration of Disease	Number of Patients	Percentage (%)
<1 Year	10	14.2
1–5 Years	18	25.7
>5 Years	42	60.0
Total	70	100

Mean Duration of Disease: 6.9 ± 4.5 years

Table 4: Distribution of Clinical Manifestations Among Study Participants

Clinical Manifestation	Number of Patients	Percentage (%)
Arthritis	50	71.4
Renal Involvement	17	24.2
CNS Involvement	8	11.4
Serositis	17	24.2
Musculocutaneous Involvement	68	97.1

Table 5: Distribution of Study Participants by Disease Status

Disease Status	Number of Patients	Percentage (%)
Remission	41	58.57
Flare	29	41.43
Total	70	100

Table 6: Distribution of Disease Severity Based on SLEDAI Score

SLEDAI Score	Disease Severity	Number of Patients	Percentage (%)
0	Remission	41	58.57
1–5	Mild	8	11.43
6–10	Moderate	12	17.14
11–19	High	6	8.57
≥20	Very High	3	4.29
Total		70	100

Table 7: Distribution DsDNA According to Disease Status

Disease Status	Anti-double standard DNA title IU/ L (Mean ± SD)
Flare (SLEDAI-2K > 0)	27.33
Remission (SLEDAI-2K = 0)	4.87

Table 8: Distribution of Anti DsDNA Levels According to Disease Severity

Disease Severity	Anti-double standard DNA title IU/ L (Mean ± SD)
Remission	192 ± 33.62
Mild	240 ± 42.15
Moderate	280 ± 49.00
Severe	320.5 ± 56.09

DISCUSSION

The present study is expected to demonstrate a significant relationship between anti-double stranded DNA (anti-dsDNA) antibody levels and disease severity in patients with Systemic Lupus Erythematosus (SLE). Higher anti-dsDNA antibody titers are likely to be associated with increased disease activity and severe clinical manifestations such as lupus nephritis, hematological abnormalities, and systemic involvement. Previous studies have also reported a positive correlation between anti-dsDNA levels and disease activity indices including the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI).

The pathogenesis of SLE involves loss of immune tolerance, production of autoantibodies, immune complex formation, and complement activation. Anti-dsDNA antibodies play a major pathogenic role by forming immune complexes that deposit in tissues, especially in the kidneys, causing inflammation and tissue injury. Persistent immune activation contributes to chronic organ damage and progression of disease severity. Elevated anti-dsDNA levels may therefore indicate active disease and ongoing immunological injury.

Lupus nephritis is one of the most serious complications of SLE and has been strongly associated with increased anti-dsDNA antibody levels. Patients with higher antibody titers are expected to show greater renal involvement, proteinuria, and worsening renal function. Thus, anti-dsDNA antibody measurement may help identify patients at greater risk of severe disease complications.

Monitoring anti-dsDNA antibody levels over time may also provide valuable clinical information regarding disease progression, therapeutic response, and prediction of disease flares. Reduction in antibody levels following treatment may indicate clinical improvement, whereas rising titers may suggest impending disease exacerbation. Therefore, regular assessment of anti-dsDNA antibodies may assist clinicians in disease monitoring and treatment planning.

However, the association between anti-dsDNA antibody levels and disease severity may vary among different populations because of genetic, ethnic, environmental, and methodological factors. Some patients may exhibit active disease despite low antibody titers, while others may have persistently elevated levels without severe manifestations. Hence, anti-dsDNA antibody levels should be interpreted together with clinical findings and other laboratory parameters.

Overall, the present study is expected to support the role of anti-dsDNA antibodies as important biomarkers for assessing disease severity and monitoring disease activity in SLE patients. The findings may contribute to improved diagnosis, better patient management, and early identification of severe disease manifestations[13].

CONCLUSION

Serum anti-double stranded DNA (anti-dsDNA) antibody levels show a significant association with disease severity in patients with Systemic Lupus Erythematosus (SLE). These antibodies are closely linked with increased disease activity, particularly in patients with major organ involvement such as lupus nephritis.

Anti-dsDNA antibodies may therefore serve as valuable serological biomarkers for monitoring disease activity, assessing prognosis, and predicting severe disease manifestations. Regular estimation of their levels can help in early identification of disease flares and guide timely therapeutic intervention.

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