



# Lupus anticoagulant positivity predicts reduced renal remission in male lupus nephritis

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## ABSTRACT

**Introduction:** Lupus nephritis (LN) represents one of the most severe manifestations of systemic lupus erythematosus (SLE). Although SLE predominantly affects females, male patients often demonstrate more aggressive disease and worse renal outcomes. The impact of lupus anticoagulant (LA) positivity on LN remission among male individuals, remains insufficiently studied.

**Objectives:** To describe characteristics of male patients with LN and evaluate differences in clinical features and remission outcomes between patients with positive lupus anticoagulant (LNpLA) and those without lupus anticoagulant (LNnoLA).

**Patients and Methods:** In a retrospective observational study, adult male patients with SLE and biopsy-proven LN followed at Dubai hospital, a tertiary referral center in the UAE, between 2009 and 2024 were retrospectively analyzed. Clinical features, kidney biopsy findings, treatments, and outcomes were evaluated. Continuous variables were analyzed using independent T-test or Mann-Whitney U test, and categorical variables using  $\chi^2$  test or Fisher's exact test. Statistical significance was defined as  $P < 0.05$ .

**Results:** Thirty-two male patients were included (mean age  $42 \pm 12$  years), with LA positivity identified in 28% ( $n=9$ ). Constitutional symptoms were the most common presentation, while class IV LN was the predominant histologic lesion. Baseline demographic characteristics and renal function were comparable between LNpLA and LNnoLA groups. In addition, antinuclear antibody (ANA) and anti-dsDNA positivity were observed in 81% and 69% of patients respectively. Moreover, LNpLA patients demonstrated higher prevalence of antiphospholipid antibodies and nephrotic-range proteinuria. Treatment regimens were comparable between groups. Renal remission occurred significantly less frequently in LNpLA compared with LNnoLA patients (22% versus 56%,  $P < 0.05$ ).

**Conclusion:** Among male patients with LN, LA positivity was associated with a reduced likelihood of renal remission. These findings support a potential role for antiphospholipid autoimmunity as a prognostic factor in male LN and may have implications for risk stratification and therapeutic decision-making, although larger prospective studies are needed to confirm and further define this association.

## Implication for health policy/practice/research/medical education:

Lupus anticoagulant (LA) positivity may serve as a clinically relevant prognostic marker in male lupus nephritis (LN), supporting routine antiphospholipid antibody assessment to improve risk stratification and individualized therapeutic decision-making. These findings suggest potential implications for treatment optimization and highlight the need for prospective studies evaluating targeted immunomodulatory and antithrombotic strategies to improve renal outcomes in this high-risk population.

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## Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by multisystem inflammation mediated by autoantibody production

and immune complex deposition (1). Lupus nephritis (LN) develops in approximately 40-60% of patients with SLE and remains a major determinant of morbidity and mortality (2,3). Despite advances in immunosuppressive

therapy, progression to chronic kidney disease and end-stage kidney disease continues to occur in a substantial proportion of patients (4).

Although SLE predominantly affects women, male patients potentially exhibit a distinct disease phenotype. Several studies have demonstrated that men with SLE develop higher rates of renal involvement, more severe histological lesions, and poorer long-term outcomes compared with females (5-8).

Antiphospholipid antibodies, including lupus anticoagulant (LA), anti-cardiolipin antibodies and anti- $\beta$ 2-glycoprotein antibodies, are frequently detected in SLE and contribute to thrombotic complications collectively known as antiphospholipid syndrome (9). Increasing evidence suggests that antiphospholipid antibodies may directly contribute to renal injury through microvascular thrombosis, endothelial activation, and chronic vascular damage (10-12). Vascular lesions in LN including those associated with antiphospholipid antibody positivity have been associated with worse renal outcomes, increased proteinuria, and reduced remission rates in LN (12-15).

However, data evaluating the prognostic role of LA specifically among male patients with LN remain scarce, particularly in Middle Eastern populations where epidemiologic characteristics may differ (16-18). Understanding predictors of treatment response is essential for individualized management strategies.

## Objectives

The objective of this study is to evaluate and compare the clinical characteristics and renal outcomes of patients with male lupus nephritis according to lupus anticoagulant status [those with positive lupus anticoagulant (LNpLA) and those without lupus anticoagulant (LNnoLA)], and to assess whether LA positivity is associated with unfavorable renal prognosis in this population.

## Patients and Methods

We conducted a retrospective observational study that included adult male patients diagnosed with SLE with LN who were followed at Dubai hospital, Dubai, United Arab Emirates, between January 2009 and December 2024. Dubai hospital is a large tertiary referral center serving a multiethnic population. Eligible patients were  $\geq 18$  years of age, fulfilled accepted classification criteria for SLE, and had kidney biopsy confirmation of LN. Our study was approved by the institutional review board.

Demographic characteristics, clinical manifestations at presentation, thromboembolic complications, treatment exposure, and follow-up outcomes were collected from electronic medical records. Body mass index (BMI) was calculated using weight in kg divided by height in meters squared ( $\text{kg}/\text{m}^2$ ). Laboratory data including hemoglobin level, leukocyte count, platelet count, serum creatinine, estimated glomerular filtration rate (eGFR), using updated CKD-EPI (chronic kidney disease epidemiology

collaboration) equation (19) serum albumin, urine analysis, urine protein-creatinine ratio, antinuclear antibody (ANA), anti-double-stranded DNA antibodies (anti-dsDNA), complement levels (C3 and C4), LA, anti-cardiolipin antibodies and anti- $\beta$ 2-glycoprotein antibody levels were collected.

All renal biopsy specimens were reviewed using light microscopy, immunofluorescence, and electron microscopy. Histopathologic classification was assigned according to the revised International Society of Nephrology/Renal Pathology Society (ISN/RPS) 2018 LN classification system (20).

Patients were categorized into two groups according to LA status; lupus nephritis with positive lupus anticoagulant (LNpLA) and lupus nephritis without lupus anticoagulant (LNnoLA). Renal remission was defined based on improvement in renal function and reduction in proteinuria consistent with accepted clinical response criteria for LN (21).

## Statistical analysis

Continuous variables were expressed as mean  $\pm$  standard deviation for normally distributed data and median with interquartile range for non-normally distributed variables. Categorical variables were summarized as frequencies and percentages. Comparisons between groups were performed using independent T-test or Mann-Whitney U test for continuous variables and Pearson's  $\chi^2$  test or Fisher's exact test for categorical variables. A *P* value  $< 0.05$  was considered statistically significant. Statistical analyses were conducted using SPSS version 29.

## Results

A total of 32 adult male patients with biopsy-proven LN were included in the study. The mean age at study inclusion was  $42 \pm 12$  years and the mean age at diagnosis was  $28 \pm 12$  years. Approximately half of the cohort was younger than 40 years of age. Most patients were of Emiratis (66%), whereas the rest where a mix of multiple origins and ethnicities.

At presentation, constitutional symptoms were the most frequently reported clinical manifestation, occurring in 43% of patients, followed by hematological involvement in 38% and polyarthralgia or polyarthritis in 34% and dermatologic manifestations in 31%. Peripheral edema was observed in 22%, while neurological manifestations occurred in 19%. Less frequent presentations included cardiac symptoms observed in 6% of patients (Table 1).

Regarding immunological profile, ANA positivity was present in 81% of patients, while anti-double-stranded DNA antibodies were detected in 69%. LA positivity (LNpLA) was identified in 28% of the cohort, whereas anti-cardiolipin antibodies and anti- $\beta$ 2-glycoprotein antibodies positivity were each observed in 19% of patients. Complement consumption was common, with low C3 and C4 levels observed in 53 % and 44% of patients,

**Table 1.** Baseline characteristics in full cohort and in patients with lupus nephritis (with and without a positive lupus anticoagulant)

| Patient characteristics                               | Full cohort (n =32) | LN with positive LA (n =9) | LN with negative LA (n = 23) |
|-------------------------------------------------------|---------------------|----------------------------|------------------------------|
| Age (years) mean $\pm$ SD                             | 42 $\pm$ 12         | 40 $\pm$ 11                | 43 $\pm$ 13                  |
| Nationality                                           |                     |                            |                              |
| UAE, n (%)                                            | 21 (66)             | 6 (67)                     | 15 (65)                      |
| Other, n (%)                                          | 11 (34)             | 3 (33)                     | 8 (35)                       |
| BMI (kg/m <sup>2</sup> ) mean $\pm$ SD                | 28 $\pm$ 8          | 29 $\pm$ 11                | 28 $\pm$ 7                   |
| Associated comorbidities                              |                     |                            |                              |
| Diabetes mellitus, n (%)                              | 19 (59)             | 1 (11)                     | 18 (78)                      |
| Hypertension, n (%)                                   | 18 (56)             | 6 (67)                     | 12 (52)                      |
| Smoking, n (%)                                        | 8 (25)              | 3 (33)                     | 5 (22)                       |
| SLE organ involvement                                 |                     |                            |                              |
| CNS manifestations present, n (%)                     | 6 (19)              | 2 (22)                     | 4 (17)                       |
| Cardiac manifestations, n (%)                         | 2 (6)               | 1 (11)                     | 1 (4)                        |
| Skin manifestations, n (%)                            | 10 (31)             | 5 (56)                     | 5 (22)                       |
| Arthralgia/arthritis, n (%)                           | 11 (34)             | 3 (33)                     | 8 (35)                       |
| Hematologic manifestations, n (%)                     | 12 (38)             | 5 (56)                     | 7 (30)                       |
| Constitutional symptoms, n (%)                        | 14 (43)             | 6 (67)                     | 8 (35)                       |
| Hemoglobin (g/dL), median (IQR)                       | 13 (2)              | 13 (2)                     | 13 (2)                       |
| WBC ( $\times 10^3/\mu\text{L}$ ), median (IQR)       | 6.5 (2.4)           | 6.2 (2.5)                  | 6.6 (2.3)                    |
| Platelets ( $\times 10^3/\mu\text{L}$ ), median (IQR) | 206 (55)            | 194 (61)                   | 212 (53)                     |
| Complement C3 low, n (%)                              | 17 (53)             | 5 (56)                     | 12 (52)                      |
| Complement C4 low, n (%)                              | 14 (44)             | 5 (56)                     | 9 (39)                       |
| Anti-nuclear antibody positivity, n (%)               | 26 (81)             | 8 (89)                     | 18 (78)                      |
| Anti-dsDNA positivity, n (%)                          | 22 (69)             | 8 (89)                     | 14 (61)                      |

LN: Lupus nephritis; LA: Lupus anticoagulant; SD: Standard deviation; SLE: Systemic lupus erythematosus; IQR: Interquartile range; WBC: White cell count; UAE: United Arab Emirates; BMI: Body mass index; CNS: Central nervous system; dsDNA: Double stranded DNA.

respectively.

Hematological parameters demonstrated a mean hemoglobin level of 13 g/dL, mean leukocyte count of  $6.5 \times 10^9/\text{L}$ , and mean platelet count of  $206 \times 10^9/\text{L}$ . Likewise, anemia, leukocytopenia, leukocytosis and thrombocytopenia were observed in 12%, 16%, 6%, and 19% of patients, respectively. Renal function at presentation showed a median serum creatinine of 1.19 mg/dL, across with a median estimated glomerular filtration rate of 77.9 mL/min/1.73m<sup>2</sup>. Mean proteinuria was  $4.4 \pm 2.7$  g/day. Histopathological analysis demonstrated that class IV LN was the most frequent biopsy finding across the cohort (34%), followed by class V disease (19%).

When comparing patients with lupus anticoagulant positivity (LNpLA) to those without (LNnoLA), no significant differences were observed in baseline age or age at diagnosis. Clinical manifestations were broadly comparable between the two groups, although thromboembolic events showed a distinguishing pattern, with cerebrovascular accidents occurring exclusively in the LNpLA group. Constitutional symptoms were the most frequent presentation in both groups. Skin and hematologic manifestations were numerically more frequent in LNpLA patients, whereas polyarthritis and

edema were slightly more common among LNnoLA patients. Immunologically, LNpLA patients exhibited higher frequencies of antiphospholipid-associated serologies, including anti- $\beta 2$ -glycoprotein antibodies and anti-cardiolipin antibodies, along with higher proportions of ANA and anti-dsDNA positivity, although most of the differences were not statistically significant. Hematological indices were largely comparable between the two groups; however, leukopenia was more frequently observed in LNpLA patients, whereas thrombocytopenia and anemia were more common among LNnoLA patients.

Renal parameters, including serum creatinine, eGFR, and overall proteinuria, were similar between groups. However, nephrotic-range proteinuria was more frequently observed in the LNpLA group. Histologically, class IV LN remained the dominant lesion in both groups, while class V disease was relatively more frequent among LNnoLA patients (Table 2).

Regarding chronic kidney disease stage distribution, patients with LNpLA demonstrated a broader distribution across all stages, including a relatively higher proportion of advanced disease stages. In terms of therapy, most common induction utilized was double therapy with prednisone and either mycophenolate or cyclophosphamide (66%

**Table 2.** Characteristics of renal disease and antiphospholipid features in full cohort and in patients with lupus nephritis (with and without a positive lupus anticoagulant)

| Patient characteristics                                           | Full cohort (n =32) | LN with positive LA (n =9) | LN with negative LA (n = 23) |
|-------------------------------------------------------------------|---------------------|----------------------------|------------------------------|
| Renal parameters                                                  |                     |                            |                              |
| Age at diagnosis of LN, mean $\pm$ SD (year)                      | 28 $\pm$ 12         | 28 $\pm$ 12                | 28 $\pm$ 12                  |
| Creatinine (mg/dL), median (IQR)                                  | 1.19 (2)            | 1.29 (8)                   | 1 (1)                        |
| eGFR (mL/min/1.73m <sup>2</sup> ), median                         | 77.8                | 78.3                       | 76.5                         |
| Proteinuria (g/day), median (IQR)                                 | 4.4 (2.3)           | 3.2 (1.9)                  | 3.6 (3)                      |
| Proteinuria $\geq$ 3 g/day, n (%)                                 | 16 (50)             | 5 (56)                     | 11 (48)                      |
| Hematuria, n (%)                                                  | 18 (56)             | 6 (67)                     | 12 (52)                      |
| LN Class based on kidney biopsy                                   |                     |                            |                              |
| Class I                                                           | 1 (3)               | 1 (11)                     | 0 (0)                        |
| Class II                                                          | 1 (3)               | 0 (0)                      | 1 (4)                        |
| Class III                                                         | 2 (6)               | 0 (0)                      | 2 (9)                        |
| Class IV                                                          | 11 (34)             | 3 (33)                     | 8 (35)                       |
| Class V                                                           | 6 (19)              | 1 (11)                     | 5 (22)                       |
| Combined classes/other                                            | 2 (6)               | 0                          | 2 (9)                        |
| LA positivity, n (%)                                              | 9 (28)              | 9 (100)                    | 0 (0)                        |
| Anti- $\beta$ 2-glycoprotein antibody positivity *, n (%)         | 6 (19)              | 4 (44)                     | 2 (9)                        |
| Anticardiolipin antibody positivity*, n (%)                       | 6 (19)              | 5 (55)                     | 1 (4)                        |
| Antiphospholipid syndrome present, n (%)                          | 2 (6)               | 2 (22)                     | 0                            |
| DVT/PE, n (%)                                                     | 1 (3)               | 0 (0)                      | 1 (4)                        |
| Stroke/TIA*, n (%)                                                | 2 (6)               | 2 (22)                     | 0 (0)                        |
| Most common induction utilized                                    |                     |                            |                              |
| Double therapy with Prednisone and MMF or cyclophosphamide, n (%) | 21 (65)             | 6 (66)                     | 15 (65)                      |
| Most common maintenance utilized                                  |                     |                            |                              |
| Prednisone and MMF, n (%)                                         | 19 (59)             | 4 (44)                     | 15 (65)                      |
| Achieved remission*, n (%)                                        | 15 (47)             | 2 (22)                     | 13 (57)                      |

LN: Lupus nephritis; LA: Lupus anticoagulant; SD: Standard deviation; IQR: Interquartile range; DVT/PE: Deep vein thrombosis/pulmonary embolism; eGFR: Estimated glomerular filtration rate; TIA: Transient ischemic attack; MMF: Mycophenolate.

\* Indicates statistically significant difference ( $P < 0.05$ ) between LN with positive LA and LN with negative LA.

in LNpLA and 65% in LNnoLA). The most common maintenance regimen utilized was prednisone and mycophenolate combination (44% in LNpLA and 65% in LNnoLA). In our study, a total of 75% of patients were on hydroxychloroquine.

Importantly, renal outcomes differed significantly between the two groups. Renal remission was achieved in 56% of LNnoLA patients compared with only 22% of LNpLA patients, indicating a substantially lower likelihood of achieving remission in patients with LA positivity ( $P < 0.05$ ).

## Discussion

In this single-center study of male patients with biopsy-proven LN, we observed that LA positivity was associated with a markedly lower likelihood of achieving renal remission. While baseline demographic characteristics, renal function, and histologic class distribution were broadly comparable between groups, LNpLA patients demonstrated features suggestive of a more aggressive disease phenotype, including higher prevalence of antiphospholipid antibodies and increased nephrotic-

range proteinuria. These findings support the growing concept that antiphospholipid autoimmunity contributes not only to vascular thrombosis but also to adverse renal outcomes in LN.

Male sex itself has been recognized as an unfavorable prognostic factor in SLE and LN, and previous studies have shown that men develop LN more frequently and can present with higher disease activity, with irreversible organ damage earlier than female patients (5-8). Our cohort from UAE reflects these observations, with a predominance of proliferative disease and substantial baseline proteinuria. The relatively young age at diagnosis despite later study age further suggests prolonged disease burden among affected males. The reasons underlying sex differences remain incompletely understood but may involve hormonal influences, delayed diagnosis and genetic or immunologic variations affecting immune regulation.

The prevalence of LA positivity in our cohort (28%) is consistent with previously reported antiphospholipid antibody frequencies in LN (22). Increasing experimental and clinical evidence indicates that antiphospholipid

antibodies may directly induce renal injury through endothelial activation, complement-mediated inflammation, and chronic microvascular thrombosis, collectively described as antiphospholipid syndrome nephropathy (2-10,23). These vascular lesions may coexist with immune complex-mediated glomerulonephritis and potentially limit response to conventional immunosuppressive therapy (2-10,23).

Importantly, remission rates were substantially lower among LNpLA patients compared with LNnoLA patients. Similar associations between antiphospholipid antibodies and poor renal outcomes have been reported in prior LN cohorts (5-13,24). The persistence of proteinuria despite comparable baseline renal function suggests that vascular or thrombotic mechanisms may contribute to ongoing renal injury independent of inflammatory activity. This observation has important clinical implications, as traditional treatment strategies primarily target immune complex inflammation rather than microvascular injury (5-13,24).

The occurrence of stroke exclusively in the LNpLA group further highlights the systemic implications of LA positivity and reinforces its role as a marker of thrombotic risk (25). The findings of this study emphasize the importance of routine antiphospholipid antibody screening in patients with LN, particularly among male patients who already represent a higher-risk subgroup. Early identification of LNpLA patients may allow closer monitoring, optimization of cardiovascular risk factors, and consideration of adjunctive antithrombotic strategies in selected cases (26,27).

An important interpretation emerging from our study is that LA positivity may represent a distinct pathogenic phenotype of LN characterized by overlapping immune-mediated and vasculopathic injury. Traditional classifications of LN focus primarily on glomerular immune complex deposition; however, vascular lesions increasingly appear to influence long-term renal prognosis (11,15). The higher frequency of nephrotic-range proteinuria observed in LNpLA patients in our cohort may reflect combined podocyte injury and microvascular injury. Recent international recommendations emphasize a precision-medicine approach to SLE management, recognizing that immunologic heterogeneity influences therapeutic response (28,29). Our findings support incorporating antiphospholipid antibody status into future risk stratification models for LN.

From a therapeutic perspective, the optimal management of LN patients with LA remains uncertain. Standard induction regimens using mycophenolate mofetil or cyclophosphamide have demonstrated efficacy in inflammatory LN (30,31), yet outcomes remain suboptimal in patients with concomitant antiphospholipid antibodies. The lower remission rate observed among LNpLA patients in this study suggests that conventional immunosuppression alone may be insufficient for this

subgroup. Whether adjunctive anticoagulation, intensified immunosuppression, or vascular-protective strategies improve renal outcomes warrants prospective evaluation. Additionally, regional data on LN in Middle Eastern populations remain limited (16-18); therefore, our study contributes valuable real-world evidence from the UAE and highlights the need for multicenter regional registries focusing on sex-specific and immunologic predictors of outcome.

## Conclusion

In summary, this study demonstrates that LA positivity identifies a subgroup of male patients with LN who experience significantly lower rates of renal remission despite comparable baseline clinical and histopathologic characteristics. Our findings suggest that antiphospholipid autoimmunity may contribute to a distinct disease phenotype characterized by combined inflammatory and vasculopathic renal injury, potentially limiting response to standard immunosuppressive therapy. Given the recognized association between antiphospholipid antibodies, thrombotic risk, and adverse renal outcomes, routine assessment of LA status should be considered an important component of risk stratification in male LN patients. These results highlight the need for heightened clinical vigilance, individualized therapeutic strategies, and prospective studies evaluating whether adjunctive antithrombotic or targeted treatment approaches can improve renal outcomes in this high-risk population. Larger multicenter studies from the Middle East and other underrepresented regions are warranted to validate these findings and refine prognostic models incorporating sex and immunologic profiles in LN.

## Limitations of the study

Despite its strengths, including detailed biopsy confirmation and long observation period, this study has limitations. The retrospective design and modest sample size limit causal inference. Treatment decisions were clinician-dependent rather than protocolized, introducing potential confounding related to therapeutic selection and disease severity that could not be fully adjusted for in the analysis. Nonetheless, the consistent association between LA positivity and reduced remission supports the biological plausibility of our findings and justifies further investigation in larger prospective cohorts.

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## Authors' contribution

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### Conflicts of interest

The authors declare no conflicts of interest associated with the publication of this paper.

### Ethical issues

The research conducted in this study adhered to the principles outlined in the Declaration of Helsinki and was approved by the Ethics Committee of Dubai Health (reference number MBRU-2024-527/DSREC-12/2024\_11). Prior to any intervention, all participants provided written informed consent. The authors have fully complied with ethical issues, such as plagiarism, data fabrication, and double publication.

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