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# Real-World Efficacy of Belimumab in childhood-onset systemic lupus erythematosus: A Retrospective Cohort Analysis Using Propensity Score Matching

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**Case Report**

**Keywords:** childhood-onset systemic lupus erythematosus, pediatric lupus nephritis, belimumab, proteinuria

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# Real-World Efficacy of Belimumab in childhood-onset systemic lupus erythematosus: A Retrospective Cohort Analysis Using Propensity Score Matching

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# Abstract

**Objective:** This single-center real-world study aimed to evaluate the efficacy of belimumab in childhood-onset systemic lupus erythematosus (cSLE) and to explore baseline-dependent therapeutic benefits using multivariate logistic regression and propensity score matching (PSM) for mutual validation.

**Methods:** A total of 230 cSLE patients (100 receiving belimumab plus conventional therapy and 130 receiving conventional therapy alone) enrolled from June 2020 to May 2025 were analyzed. Nested logistic regression models were constructed based on SRI-4 response at weeks 24 and 54. After 1:1 PSM, 56 matched pairs were included for comparative analysis of laboratory parameters, disease activity scores, and glucocorticoid dosage.

**Results:** In the overall cohort, multivariate logistic regression showed that belimumab was independently associated with ≥4-point improvement in SLEDAI-2K at week 24 (OR=2.359, 95%CI: 1.114–5.366, P=0.032) and week 54 (OR=4.302, 95%CI: 1.703–13.175, P=0.005). However, these associations became non-significant after adjustment for baseline SLEDAI-2K (week 24: OR=0.608, P=0.418; week 54: OR=1.692, P=0.430). After 1:1 PSM, the belimumab group exhibited significantly higher platelet counts and complement C3 levels, along with lower erythrocyte sedimentation rate (ESR) and anti-dsDNA antibody titers at week 24. At week 54, the belimumab group achieved superior improvements in hemoglobin, platelet count, ESR, anti-dsDNA titers, complement C3, and Physician Global Assessment (PGA) scores (all P<0.05).

**Conclusion:** This single-center real-world study demonstrates that belimumab effectively improves immunological and hematological parameters and reduces disease activity in cSLE, with a favorable safety profile. The benefits are particularly prominent in hematological domains.



**Keywords:** childhood-onset systemic lupus erythematosus; pediatric lupus nephritis; belimumab; proteinuria

# Introduction

Systemic lupus erythematosus (SLE) is a chronic, relapsing inflammatory autoimmune disease characterized by multisystem and multiorgan involvement [1]. Compared with adult-onset SLE, childhood-onset SLE (cSLE) is associated with higher cumulative organ damage, greater disease activity, and increased medication burden, imposing substantial psychosocial and economic burdens on families and society [2]. Persistent disease activity drives progressive organ damage and elevates mortality risk; therefore, timely achievement of disease remission is of paramount importance in long-term management [3].

Conventional therapeutic strategies for cSLE rely primarily on glucocorticoids combined with immunosuppressive agents such as mycophenolate mofetil (MMF) and cyclophosphamide (CTX). Although these regimens can mitigate disease manifestations to some extent, prolonged glucocorticoid use frequently leads to severe adverse effects, including infections, osteoporosis, growth retardation, and metabolic disturbances, which are particularly concerning in pediatric populations due to ongoing physical development. Furthermore, a subset of patients exhibits inadequate response to conventional therapy, with persistent disease activity, refractory proteinuria, and sustained hypocomplementemia. Hence, safer, more precise, and targeted therapeutic options are urgently needed.

Belimumab is a recombinant humanized IgG1κ monoclonal antibody that binds and neutralizes soluble B-lymphocyte stimulator (BLyS), also known as B-cell activating factor (BAFF). Elevated BAFF levels in SLE patients drive aberrant B-cell activation, proliferation, and differentiation, playing a pivotal role in disease pathogenesis [4]. Belimumab is the first targeted biologic approved for cSLE. Previous multicenter prospective studies in Chinese pediatric cohorts have confirmed that belimumab significantly reduces SLEDAI scores and improves clinical remission rates [5]. Nevertheless, real-world evidence for belimumab in cSLE remains limited, and rigorous controlled studies using propensity score matching (PSM) are still lacking.

To address these gaps, this single-center real-world cohort study enrolled children diagnosed with SLE and applied two complementary statistical approaches—multivariate logistic regression and PSM—to adequately balance baseline characteristics between treatment groups. We comprehensively compared the efficacy of belimumab combined with conventional therapy versus conventional therapy alone in terms of disease activity, immunological markers, hematological parameters, and renal involvement, aiming to provide robust evidence for clinical decision-making in cSLE.

# Materials and Methods

**Study population**

Pediatric patients (age <18 years) diagnosed with SLE by specialist physicians and with complete treatment records and 54-week follow-up data were enrolled. Multisystem involvement, including renal impairment, was permitted. Exclusion criteria comprised concurrent autoimmune or hereditary nephropathy that could confound disease assessment, severe missing baseline or outcome data, and incomplete 54-week follow-up due to non-disease-related reasons.

**Grouping, treatment Regimens, and ethical Considerations**

Patients were divided into the conventional therapy group and the belimumab combination group. Conventional therapy consisted of individualized glucocorticoids (prednisone or methylprednisolone) and optional immunosuppressants (cyclophosphamide, mycophenolate mofetil, or azathioprine). The belimumab group received additional weight-based intravenous belimumab every 4 weeks, with treatment continued for at least 54 weeks. The study protocol was approved by the institutional ethics committee and complied with the Declaration of Helsinki. All patients or their guardians provided written informed consent for routine treatment, and all data were anonymized during analysis.

**Multivariate logistic regression analysis**

In the overall cohort (n=230), three nested logistic regression models were constructed with SRI-4 response at weeks 24 and 54 as binary outcomes and belimumab treatment as the primary exposure. Model 1 was unadjusted; Model 2 adjusted for age and sex; Model 3 further adjusted for baseline SLEDAI-2K score. Robust standard errors were estimated using the sandwich estimator (HC3), and odds ratios (OR) with 95% confidence intervals (CI) and robust P values were reported.

**Propensity score matching**

To minimize confounding bias inherent to retrospective design, 1:1 nearest-neighbor PSM with a caliper of 0.5 standard deviations of the propensity score was performed. The propensity score was calculated using a logistic regression model with belimumab treatment as the dependent variable and baseline covariates including age, sex, SLEDAI-2K score, complement C3, C4, anti-dsDNA antibody titer, and urinary protein. Ultimately, 56 patients from the belimumab group were successfully matched with 56 controls, yielding 112 patients for subsequent analyses.

**Observation indicators and follow-Up**

Key assessment time points were set at weeks 24 and 54. Primary outcomes included SLEDAI-2K score, immunological markers (anti-dsDNA antibody, complement C3/C4, ESR), hematological parameters (WBC, RBC, hemoglobin, platelet count), and glucocorticoid dosage (expressed as prednisone equivalent).

**Statistical methods**

Normality was assessed using the Shapiro–Wilk test. Normally distributed continuous variables were expressed as mean ± standard deviation (SD) and compared using independent-samples t-test; non-normally distributed variables were presented as median with interquartile range (IQR) and compared using the Mann–Whitney U test. Categorical variables were compared using the chi-square test or Fisher's exact test as appropriate. For subgroup and trend analyses, paired t-test or Wilcoxon signed-rank test was applied. All tests were two-tailed, and $P<0.05$ was considered statistically significant. All statistical analyses and graphing were performed using R software (version 4.4.2).

**Ethics approval and consent to participate**

This retrospective study was approved by the Ethics Committee of Qilu Hospital of Shandong University (Approval No.QLCR20260618 ). The requirement for written informed consent was waived by the Ethics Committee due to the retrospective nature of the study using de-identified clinical data. All procedures complied with the Helsinki Declaration.

**Funding**

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# Results

**Multivariate logistic regression analysis**

In the overall cohort of 230 cSLE patients, nested logistic regression models evaluated the independent effect of belimumab on ≥4-point SLEDAI-2K improvement at weeks 24 and 54. In the unadjusted Model 1, belimumab was significantly associated with response at week 24 (OR=2.359, 95%CI: 1.114–5.366, P=0.032) and week 54 (OR=4.302, 95%CI: 1.703–13.175, P=0.005). After adjustment for age and sex (Model 2), the associations remained significant (week 24: OR=2.541, P=0.023; week 54: OR=4.465, P=0.004). However, further adjustment for baseline SLEDAI-2K (Model 3) rendered the belimumab effect non-significant (week 24: OR=0.608, P=0.419; week 54: OR=1.692, P=0.430), whereas baseline SLEDAI-2K itself strongly predicted improvement (week 24: OR=2.646, P<0.001; week 54: OR=2.111, P<0.001). These findings indicate that baseline disease activity is a major confounder and that belimumab was preferentially prescribed to patients with higher disease activity in real-world practice, underscoring the necessity of PSM adjustment (Table 1, Figure 1).

**Baseline characteristics after propensity score matching**

Using 1:1 nearest-neighbor PSM with a caliper of 0.5, 56 matched pairs (112 patients) were derived from 100 belimumab recipients and 130 controls (Figure 2). Post-matching, most baseline variables were well balanced between groups (all P>0.05). However, the belimumab group still had lower red blood cell count (P=0.041) and serum albumin (P<0.001), higher blood urea nitrogen (P=0.002), and a marginally higher ESR (P=0.057) (Table 2).

**Distribution of organ system involvement**

After matching, the belimumab group exhibited significantly higher proportions of renal involvement (80.36% vs. 42.86%, P<0.001), anemia (58.93% vs. 19.64%, P<0.001), thrombocytopenia (30.36% vs. 10.71%, P=0.019), and neurological involvement (30.36% vs. 10.71%, P=0.019). No significant intergroup differences were observed for rash, leukopenia, fever, or arthritis (all P>0.05) (Table 3).

**Intergroup comparisons at week 24**

At week 24, the belimumab group showed significantly higher platelet counts and complement C3 levels, along with lower ESR, anti-dsDNA antibody titers, and SDI scores (all P<0.05). No significant differences were found in other hematological/immunological parameters, SLEDAI-2K, or glucocorticoid dosage.

Notably, the PGA score was higher in the belimumab group (P=0.046), which is consistent with its more severe baseline disease and renal impairment (Table 4).

**Intergroup comparisons at week 54**

At week 54, the belimumab group demonstrated superior recovery in red blood cell count, hemoglobin, and platelet count, with persistently lower ESR and anti-dsDNA titers, higher complement C3, and a significantly lower PGA score (all P<0.05). Complement C4, SLEDAI-2K, SDI, and steroid dosage were comparable between groups (Table 5).

**Longitudinal trend analysis**

For hematological parameters, platelet counts remained significantly higher in the belimumab group from week 24 onward, with an expanded advantage at week 54. Hemoglobin showed a borderline improvement at week 24 (P=0.070) and became statistically superior at week 54; red blood cell counts were significantly elevated only at week 54. For inflammatory and immunological markers, complement C3 increased significantly from week 24 and persisted through week 54; anti-dsDNA titers and ESR were markedly reduced at both time points. No intergroup difference in complement C4 was observed throughout follow-up. Urinary protein positivity declined progressively in both groups: from 78.6% at baseline to 26.8% at week 24 and 8.9% at week 54 in the control group, versus from 82.1% to 25.0% and then to 16.1% in the belimumab group. Although the rates were comparable at week 54, the belimumab group exhibited clinically meaningful relative improvement given its more severe baseline renal involvement (Figure 3).

**Safety**

Belimumab was well tolerated throughout the follow-up period. No severe infections, severe hematological adverse events, anaphylaxis, or infusion-related reactions were recorded. All patients completed the scheduled treatment and follow-up without treatment discontinuation due to adverse events.

# Discussion

Belimumab, as the first biologic approved for cSLE, has seen increasing clinical application, yet real-world data remain scarce. This study retrospectively analyzed 56 children with cSLE receiving belimumab at a single center to evaluate its efficacy and safety.

Our results demonstrate that multiple serological and hematological parameters—including hemoglobin, platelet count, complement C3, and anti-dsDNA antibodies—were significantly improved in the belimumab group as early as week 24, indicating that belimumab rapidly ameliorates hematological involvement while suppressing aberrant immune responses. These findings are consistent with the adult BLISS-LN trial [6]. The therapeutic advantage of belimumab was further consolidated at week 54.

SLEDAI-2K and PGA are core instruments for assessing SLE disease activity, and the treat-to-target (T2T) principle has been widely endorsed in SLE management [7]. Previous studies have confirmed that belimumab reduces SLEDAI-2K scores and relapse risk. Wahadat et al. reported that all 51 children with cSLE achieved the low lupus disease activity state (LLDAS) after a mean treatment duration of 186 days [8]. A multicenter real-world study with Chinese participation showed that 40.5% and 9% of SLE patients achieved LLDAS and complete remission (CR) at 28 weeks with belimumab, versus 8.8% and 3.5% in the conventional treatment group, respectively [9]. In our study, PGA scores were significantly better in the belimumab group at week 54, suggesting a progressive cumulative benefit of belimumab on overall disease control. Notably, the intergroup difference in SLEDAI-2K disappeared at week 54 (2.00 vs. 2.00, P=0.799). This apparently paradoxical result reflects a crucial clinical implication: the belimumab group had a substantially higher baseline SLEDAI-2K (11.32 vs. 8.93, P=0.001), and the equalization of scores at week 54 demonstrates that belimumab enabled a "catch-up" improvement, achieving comparable disease control despite a heavier baseline burden. Thus, belimumab is effective not only in mild cSLE but also in patients with high baseline disease activity, making it a valuable supplementary option in real-world practice.

Multiple domestic and international studies have validated belimumab’s efficacy in cSLE. A multicenter study encompassing both adults and children reported that 65% of cSLE patients achieved ≥50% improvement in baseline symptoms without deterioration of other organ functions after 6 months, with the mean prednisone dose

decreasing from 17 mg/d to 11 mg/d within 3 months, and 35% discontinuing corticosteroids during follow-up [10]. A Chinese multicenter prospective study of 193 active cSLE patients (92.2% female) from 15 centers showed that proportions of patients achieving LLDAS and CR were 12.4% (1.0%), 25.6% (4.5%), and 70.3% (29.7%) at 3, 6, and 12 months, respectively. Mean SLEDAI-2000 scores declined from 11.0 at baseline to 3.7, 2.9, and 1.7 at 3, 6, and 12 months, and the mean prednisone dose was reduced from 31.0 (18.2) mg/d at baseline to 19.4 (10.8), 12.6 (7.2), and 6.7 (5.3) mg/d at the corresponding time points, accompanied by significant improvements in clinical and immunological parameters [5]. A single-center controlled study in China further verified that the belimumab group had a mean prednisone dose of 14.12±5.86 mg/d at week 24, significantly lower than the conventional group, with no significant difference in adverse events [11]. Collectively, these data support belimumab as an effective and safe therapeutic option for cSLE.

Regarding glucocorticoid tapering, current guidelines recommend maintaining the lowest effective dose (≤5 mg/d of prednisone equivalent) during remission and maintenance phases [12]. Chinese multicenter studies have shown that belimumab enables more pronounced steroid reduction within 28 weeks compared with conventional therapy [9]. In a cohort of 26 children receiving belimumab, 42.3% discontinued glucocorticoids and 65.4% achieved a final dose ≤5 mg/d at last follow-up [13]. In our study, the belimumab group achieved a lower mean glucocorticoid dose at week 24 and a higher proportion of patients tapered to ≤5 mg/d by week 54. Therefore, belimumab offers a promising strategy for minimizing steroid exposure in cSLE.

In terms of safety, no hypersensitivity or infusion reactions were observed in our belimumab-treated patients, indicating favorable tolerability in the pediatric population. This is consistent with previous domestic and international reports [14,15]. Combined with its robust efficacy, belimumab effectively controls disease activity without increasing serious adverse event risk, further supporting its clinical use in children with SLE.

Several limitations warrant consideration. The retrospective real-world design may introduce selection bias. The relatively small sample size and single-center setting limit generalizability. Additionally, renal biopsy pathological classification data were unavailable, and long-term renal hard endpoints (e.g., sustained eGFR decline, end-stage renal disease) were not assessed. Future multicenter, prospective, large-scale studies are needed to validate these findings and to develop predictive models for treatment response in cSLE.

In conclusion, this retrospective study of 56 cSLE patients treated with belimumab demonstrates that combination therapy with belimumab and conventional treatment significantly improves serological and hematological laboratory parameters, reduces systemic disease activity, and facilitates glucocorticoid tapering. Longitudinal

follow-up further clarified the distinct temporal patterns of dynamic changes in various clinical and laboratory indicators during treatment.

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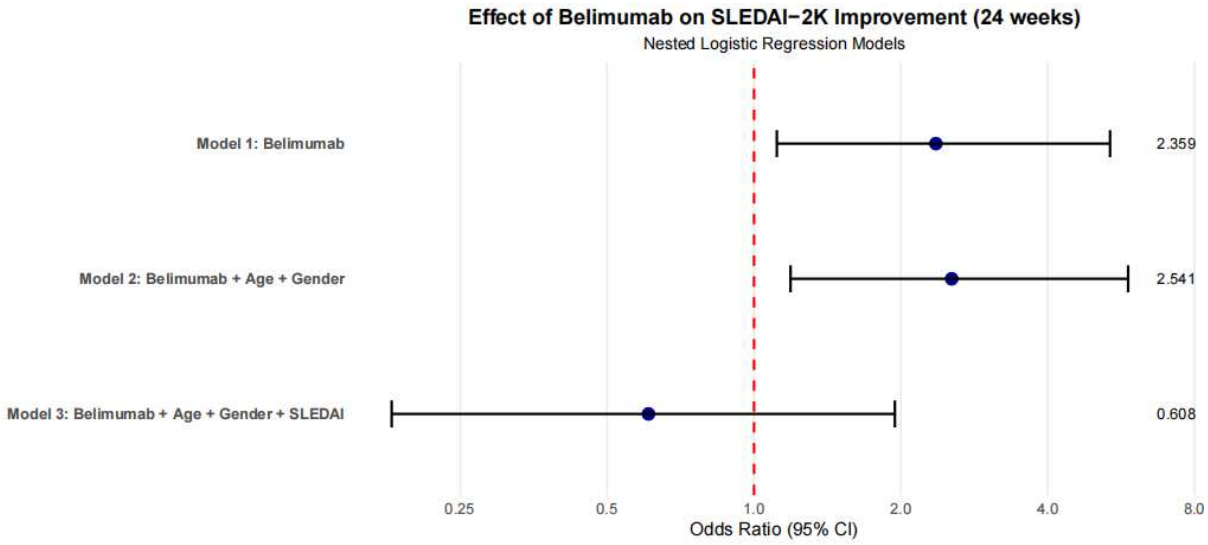
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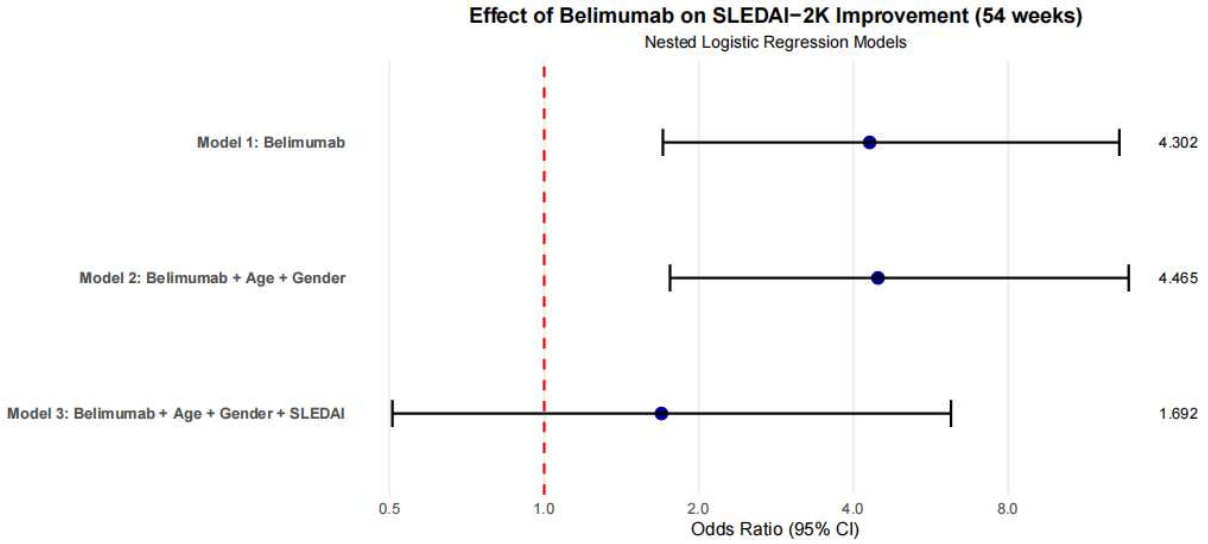
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413 Tables/Figures




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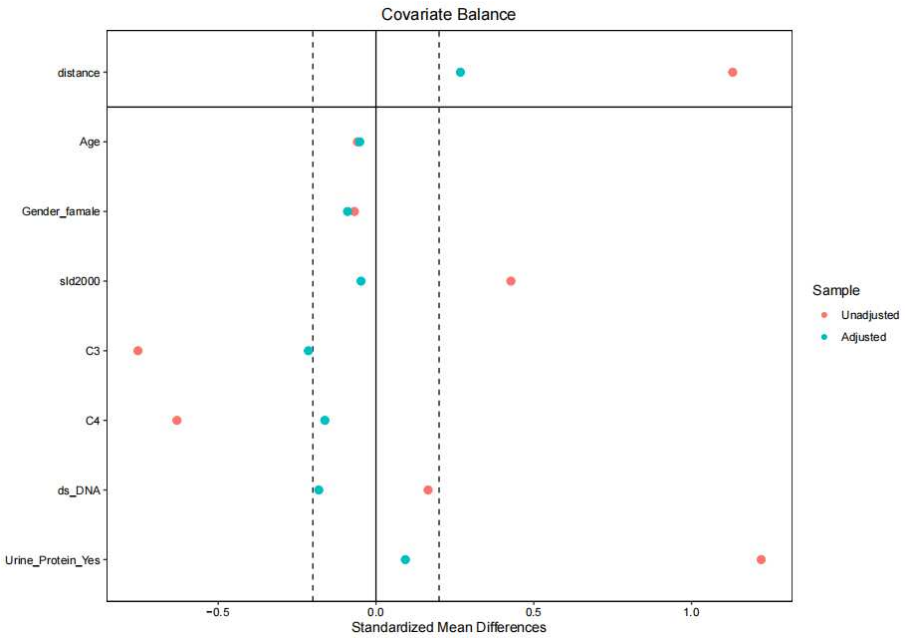
416 Figure 1 Belimumab forest plot

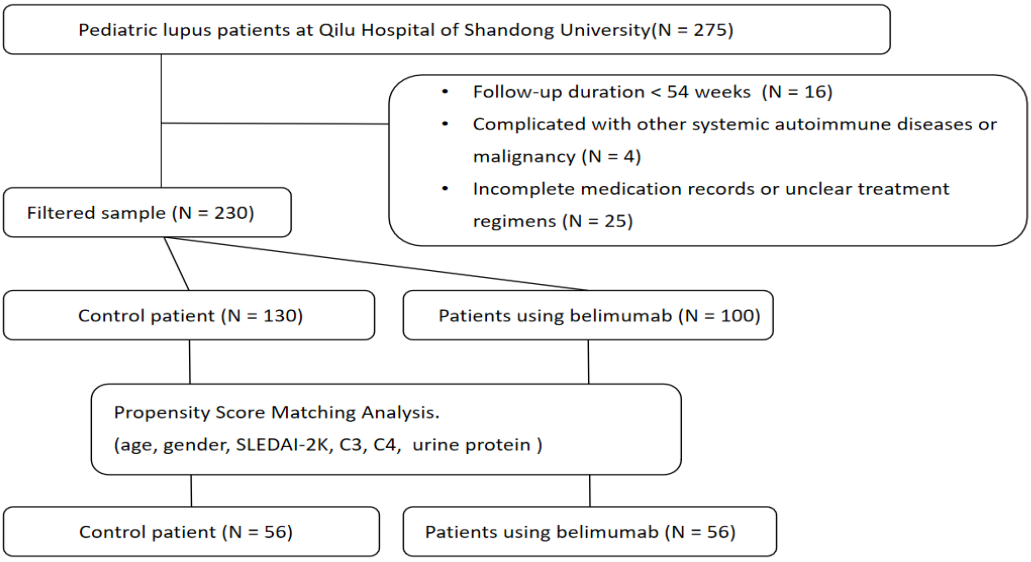
| time point | model | OR（95%CI） | Robust standard error | P value | significance |
|---|---|---|---|---|---|
| 24 weeks | Model 1: Belimumab | 2.359 (1.114-5.366) | 0.401 | 0.032 | * |
| 24 weeks | Model 2: Belimumab + Age + Gender | 2.541 (1.188-5.844) | 0.411 | 0.023 | * |

| | | | | | |
|---|---|---|---|---|---|
| 24 weeks | Model 3: Belimumab + Age + Gender + SLEDAI | 0.608 (0.181-1.944) | 0.616 | 0.418 | ns |
| 54 weeks | Model 1: Belimumab | 4.302 (1.703-13.175) | 0.516 | 0.005 | ** |
| 54 weeks | Model 2: Belimumab + Age + Gender | 4.465 (1.758-13.739) | 0.523 | 0.004 | ** |
| 54 weeks | Model 3: Belimumab + Age + Gender + SLEDAI | 1.692 (0.506-6.194) | 0.666 | 0.430 | ns |

Note: Model 1 only includes belimumab; Model 2 adjusts for age and gender; Model 3 further adjusts for baseline SLEDAI-2K.

Table 1 Nested Logistic Regression Results for the Effect of Belimumab on the Improvement of SLEDAI-2K






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424 Figure 2 Sample screening and PSM matching

425

| Variable | Control group（n=56） | Belimumab group（n=56） | P-value |
|---|---|---|---|
| woman [n (%)] | 47/56 (83.9%) | 45/56 (80.4%) | 0.622 |
| age（year） | 13.00 [10.00, 14.00] | 13.00 [10.00, 14.00] | 0.914 |
| hormone dosage（mg/d） | 60.00 [50.00, 60.00] | 60.00 [48.75, 60.00] | 0.861 |
| SLEDAI-2K | 12.00 [8.00, 14.00] | 11.00 [9.00, 14.00] | 0.515 |
| SDI | 0.00 [0.00, 1.00] | 0.00 [0.00, 1.00] | 0.741 |
| PGA | 2.00 [1.50, 2.00] | 2.00 [1.50, 2.00] | 0.551 |
| leucocyte count（$\times10^9$/L） | 4.25 [2.63, 6.27] | 4.70 [3.66, 7.15] | 0.463 |
| red-cell count（$\times10^{12}$/L） | 3.74 (0.74) | 3.43 (0.82) | 0.041 |
| Hemoglobin（g/L） | 103.39 (19.51) | 98.59 (20.70) | 0.209 |
| platelet count（$\times10^9$/L） | 176.50 [100.25, 236.00] | 168.00 [73.25, 265.25] | 0.827 |
| C reactive protein（mg/L） | 2.00 [0.51, 4.01] | 1.92 [0.39, 3.13] | 0.374 |
| E.S.R.（mm/h） | 36.50 [22.75, 60.00] | 49.00 [30.50, 77.50] | 0.057 |
| ALT（U/L） | 33.50 [16.00, 49.25] | 19.00 [11.00, 38.50] | 0.069 |
| AST（U/L） | 35.50 [19.75, 59.25] | 29.00 [18.00, 48.25] | 0.621 |
| LDH（U/L） | 314.00 [246.00, 412.67] | 297.50 [245.00, 402.00] | 0.699 |

| | | | |
|---|---|---|---|
| urea nitrogen（mmol/L） | 4.58 [3.59, 6.34] | 6.54 [4.24, 10.22] | 0.002 |
| creatinine（μmol/L） | 49.50 [40.00, 63.25] | 53.50 [43.00, 86.25] | 0.128 |
| blood albumin（g/L） | 37.21 (4.96) | 31.28 (7.04) | <0.001 |
| Anti-dsDNA antibodies（IU/mL） | 490.19 [244.24, 800.00] | 323.20 [149.30, 752.44] | 0.229 |
| Complement C3（g/L） | 0.43 [0.26, 0.60] | 0.33 [0.23, 0.53] | 0.099 |
| Complement C4（g/L） | 0.07 [0.05, 0.11] | 0.07 [0.06, 0.07] | 0.593 |
| Positive urine protein [n (%)] | 44/56 (78.6%) | 46/56 (82.1%) | 0.634 |

Note: Categorical variables are expressed as n (%); normally distributed continuous variables are expressed as mean±SD; non-normally distributed continuous variables are expressed as median [IQR]

Table 2 Comparison of baseline characteristics between the two groups after PSM

| Types of System Affected | control group（n=56） | Belimumab group（n=56） | P-value |
|---|---|---|---|
| erythra [n (%)] | 18/56（32.14%） | 16/56（28.57%） | 0.837 |
| aleucocytosis [n (%)] | 10/56（17.86%） | 10/56（17.86%） | 1.000 |
| anemia [n (%)] | 11/56（19.64%） | 33/56（58.93%） | <0.001 |
| thrombocytopenia [n (%)] | 6/56（10.71%） | 17/56（30.36%） | 0.019 |
| renal involvement [n (%)] | 24/56（42.86%） | 45/56（80.36%） | <0.001 |
| fever [n (%)] | 22/56（39.29%） | 18/56（32.14%） | 0.554 |
| neurological involvement [n (%)] | 6/56（10.71%） | 17/56（30.36%） | 0.019 |
| joint involvement [n (%)] | 11/56（19.64%） | 9/56（16.07%） | 0.805 |

Table 3 Comparison of affected systems between the two groups after PSM

| target | control group（n=56） | Belimumab group（n=56） | P-value |
|---|---|---|---|
| leucocyte count（$\times10^9$/L） | 7.81 [4.58, 11.26] | 6.94 [4.62, 9.57] | 0.283 |
| red-cell count（$\times10^{12}$/L） | 4.24 (0.70) | 4.40 (0.66) | 0.209 |
| Hemoglobin（g/L） | 121.78 (17.09) | 127.77 (17.49) | 0.070 |
| platelet count（$\times10^9$/L） | 247.95 (94.12) | 298.09 (63.20) | 0.001 |
| E.S.R.（mm/h） | 17.00 [9.75, 34.00] | 10.06 [4.53, 21.63] | 0.002 |
| Anti-dsDNA antibodies（IU/mL） | 37.00 [8.63, 186.57] | 17.19 [7.87, 39.12] | 0.002 |
| Complement C3（g/L） | 0.86 (0.23) | 1.05 (0.26) | <0.001 |
| Complement C4（g/L） | 0.18 [0.13, 0.27] | 0.20 [0.16, 0.28] | 0.176 |

| | | | |
|---|---|---|---|
| SLEDAI-2K 评分 | 4.00 [2.00, 5.25] | 2.00 [2.00, 4.00] | 0.266 |
| SDI | 0.00 [0.00, 1.00] | 0.00 [0.00, 0.00] | 0.021 |
| PGA | 0.50 [0.50, 1.00] | 1.00 [0.50, 1.00] | 0.046 |
| hormone dosage（mg/d） | 20.00 [7.50, 30.00] | 15.00 [10.00, 26.25] | 0.967 |

Note: Normal-distributed continuous variables are expressed as mean ± SD; non-normal distributed continuous variables are expressed as median [IQR].

Table 4 Comparison of efficacy indicators between the two groups after 24 weeks of treatment

| target | control group（n=56） | Belimumab group（n=56） | P-value |
|---|---|---|---|
| leucocyte count（×$10^9$/L） | 5.30 [2.81, 7.42] | 5.88 [4.05, 7.89] | 0.063 |
| red-cell count（×$10^{12}$/L） | 4.08 [3.71, 4.51] | 4.51 [4.17, 4.87] | <0.001 |
| Hemoglobin（g/L） | 116.69 (18.31) | 126.73 (15.10) | 0.002 |
| platelet count（×$10^9$/L） | 241.00 [186.00, 283.33] | 315.50 [245.00, 377.50] | <0.001 |
| E.S.R.（mm/h） | 18.00 [7.50, 26.75] | 7.17 [3.04, 20.08] | 0.017 |
| Anti-dsDNA antibodies（IU/mL） | 115.50 [17.42, 184.20] | 10.67 [6.51, 68.62] | <0.001 |
| Complement C3（g/L） | 0.78 (0.29) | 0.98 (0.35) | 0.001 |
| Complement C4（g/L） | 0.19 [0.12, 0.24] | 0.20 [0.10, 0.30] | 0.490 |
| SLEDAI-2K 评分 | 2.00 [0.00, 4.00] | 2.00 [0.00, 2.00] | 0.172 |
| SDI | 0.00 [0.00, 0.00] | 0.00 [0.00, 0.00] | 1.000 |
| PGA | 0.50 [0.00, 1.00] | 0.00 [0.00, 0.50] | 0.018 |
| hormone dosage（mg/d） | 5.00 [5.00, 10.62] | 5.00 [4.38, 8.12] | 0.051 |

Note: Normal-distributed continuous variables are expressed as mean ± SD; non-normal distributed continuous variables are expressed as median [IQR].

Table 5 Comparison of efficacy indicators between the two groups after 54 weeks of treatment

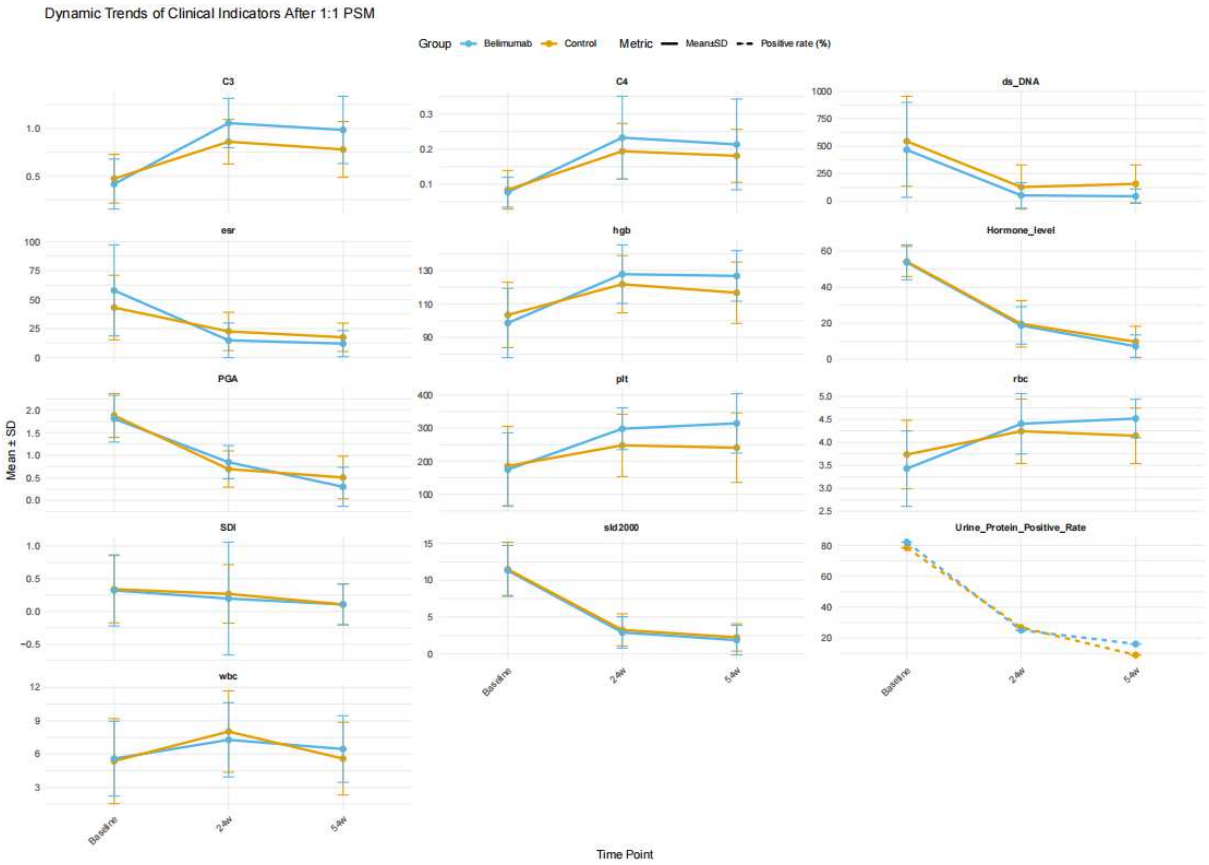



Figure 3: Dynamic Trends of Indicators

Figures (Figures can be included at the end of this document but will be requested as separate files upon article acceptance)

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