Anifrolumab in systemic lupus erythematosus: real-world experience from a single academic tertiary care centre

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Abstract

Objective

To evaluate the effectiveness and safety of anifrolumab, a human monoclonal antibody to type I interferon receptor subunit 1, on systemic lupus erythematosus (SLE) at a German academic tertiary care centre in a real-life setting.

Methods

We evaluated disease activity, clinical course and adverse events in a single-centre prospective observational cohort study of 26 SLE patients at baseline, 3, 6, 9, 12, 18 and 24 months of anifrolumab treatment. The decision to initiate therapy was made according to current guidelines (EULAR 2024).

Results

There was a significant reduction in the disease activity indices SLE-Disease Activity Index 2000 (SLEDAI-2k) (6.0 ±3.9 vs. 2.8 ±2.6, p≤0.001) and European Consensus Lupus Activity Measurement Index (ECLAM) (1.92 ±1.16 vs. 0.94 ±0.99, p=0.001) after just three months of treatment. After 12 months, definition of remission in SLE (DORIS) was achieved in 53% of patients and lupus low disease activity state (LLDAS) in 89% of patients. Mucocutaneous manifestations responded quickly and there were significant improvements in fatigue and arthritis/arthralgia. A favourable response was also seen in patients who had received previous therapies or after long duration of the disease. This was accompanied by a reduction in the glucocorticoid dose. Overall, the drug was safe and well tolerated.

Conclusion

In our real-world experience, anifrolumab achieved sustained remission after just 3 months of treatment and a significant reduction in disease activity in most patients. These data suggest that SLE patients with active disease benefit from anifrolumab therapy regardless of prior therapies or disease duration.

Key words

systemic lupus erythematosus, anifrolumab, type I interferon blockade, real-world experience

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Introduction

In recent years, it has become increasingly evident that type I interferons play a key role in the pathogenesis of SLE and that the interferon-signaling pathway is a central therapeutic target (1, 2). Moreover, the so-called IFN-signature was correlated with disease activity and there is evidence suggesting that type I interferons are already involved in disease initiation (2-4). Anifrolumab, a human monoclonal antibody to type I interferon receptor subunit 1, has been approved as a new SLE-specific therapy. Following approval of anifrolumab in 2021 and 2022 by the FDA and EMA, it is now also recommended by the new EULAR recommendations as an add-on therapy for SLE patients, who do not respond adequately to hydroxychloroquine alone or in combination with glucocorticoids. The recommendation is especially strong for patients presenting with extra-renal SLE with non-major organ involvement, but extensive disease of the skin or joints (5).

Efficacy data for the treatment of active lupus nephritis are still lacking, although data from the TULIP-LN Phase II study are promising and efficacy is currently being tested in the IRIS Phase III study (6). The data from the TULIP-2 study show efficacy for anifrolumab (7). *Post-hoc* analyses of the TULIP trials demonstrated that anifrolumab treatment leads to earlier, more frequent, and more sustained achievement of low disease activity and allows for a greater reduction in glucocorticoid use compared to placebo (8). However, apart from small case series, there is still little real-life data on the effects of anifrolumab in everyday clinical practice (9-11). This gap is to be addressed by the ASTER study, which aims to collect real-world data on anifrolumab and is expected to conclude in 2029 (12).

In the meantime, we aim to provide real-world data on the effectiveness and safety of anifrolumab in SLE patients in a real-world clinical setting.

Methods

Patients

This single-centre prospective observational cohort study includes patients with a diagnosis of SLE treated with anifrolumab. Between January 2022 and December 2024, all patients who were prescribed anifrolumab in our university lupus clinic were included in this observational study, with all patients providing informed consent. Anifrolumab was initiated in patients with a moderate to severe active flare (defined as a ≥4-point increase in the SLEDAI-2K score), a high symptom burden (PGA ≥2), or other clinical considerations of unmet therapeutic needs. The maximum observation period was 2 years, with a mean follow-up time of 17.5 months. All patients had to meet the classification criteria of SLE according to 2019 EULAR/ACR classification. The patients included received 300 mg of anifrolumab intravenously in our outpatient clinic every 4 weeks. A medical consultation took place at least every three months during which a routine blood and urine sample was taken. In addition, the SLE-disease activity index 2000 (SLEDAI-2k), the Systemic Lupus International Collaborating Clinics (SLICC)/ACR Damage Index, the European Consensus Lupus Activity Measurement Index (ECLAM) and the Physician Global Assessment (PGA), as well as patient reported clinical symptoms were recorded. Low disease activity was assessed according to the lupus low disease activity state (LLDAS) criteria, and remission was assessed according to the definition of remission in SLE (DORIS) criteria. The categorisation of SLE manifestations (Articular, Fatigue, Neuropsychiatric, Serositis, Renal, Haematological, Mucocutaneous, Cardiovascular) was based on the fulfillment of corresponding criteria in the SLEDAI-2K and ECLAM disease activity indices. A manifestation was classified as 'fulfilled' if at least one associated item from either index was present. For detailed mappings between clinical categories and specific SLEDAI-2K/ECLAM criteria, see Supplementary Table S1. Adverse events were recorded at each visit. All participating physicians were trained in the application of SLEDAI-2k, PGA and ECLAM to ensure consistency in assessments. Symptoms were only recorded when considered to be explained by SLE.

Patient and public involvement

Patients were not directly involved in the design, conduct, reporting, or dissemination plans of our research. However, the study addresses frequent patient inquiries regarding therapy effectiveness and the selection of optimal therapeutics in SLE, as observed during clinical consultations.

Ethics

This study was approved by the Independent Ethics Committee of the state of Rhineland-Palatinate, 2019-14659. Participants gave informed consent to participate in the study before taking part.

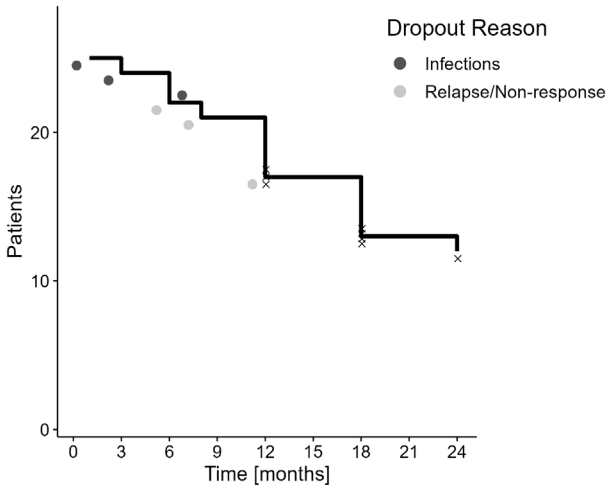

Fig. 1. Kaplan-Meier plot showing the number of treated patients over time.
Dropout reasons are visually differentiated.
The black line represents the number of patients over time. Coloured markers indicate dropout reasons: light grey (relapse/non-response), dark grey (infections), black 'x' markers denote censored patients.

Statistical analysis

Missing data were not imputed. Due to the observational nature of the study, no formal sample size calculation was performed. All patients meeting the inclusion criteria and receiving anifrolumab were included in the analysis. In addition to the primary analyses, we explored the impact of certain influencing factors (*e.g.* prior therapies, disease activity level) by stratifying patients into subgroups. Due to the limited sample size and the exploratory nature of this study, we did not perform formal interaction testing or sensitivity analyses.

R 4.4.1 (R Core Team (2022). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. URL https://www.R-project.org/) and RStudio version 2024.04.2 (Posit team (2024). RStudio: Integrated Development Environment for R. Posit Software, PBC, Boston, MA. URL http://www.posit.co/.) were used for data analysis and graphic design. The packages 'ggplot2' (v. 3.5.1, Wickham 2016), 'gtsummary' (v. 2.0.0, Sjoberg *et al.* 2021) and 'fmsb' (v. 0.7.6, Nakazawa 2024) were used for statistical analysis and data visualisation. Quantitative data are expressed as mean ± standard deviation (SD)/standard error (SEM), as indicated. The t-test was used to compare two groups with continuous variables. For comparison of more than two groups of continuous data Kruskal Wallis test and for comparison more than two groups of categorical data Pearson's Chi-squared test and Fisher's exact test were applied. Our complete data analysis is exploratory. Hence, no adjustments for multiple testing were performed. For all tests, we used a 0.05 level of significance to define statistically relevant deviations from the respective null hypothesis.

Results

Patient cohort

In our lupus cohort, 26 patients were treated with anifrolumab between 2022 and 2024. We were able to observe 21 of these patients for 1 year (Fig. 1). The

Table I. Baseline characteristics at the start of treatment with anifrolumab.

Characteristics			n=26 [1]
Female sex	24 / 26 (92%)	Leukocytes, cells/nl	6.06 ± 2.20
Age, years	44 ± 15	Hb, g/dl	13.46 ± 1.65
Time from initial diagnosis to start of therapy, months	154 ± 136	Thrombocytes, cells/nl	236 ± 88
SLEDAI-2k	6.0 ± 3.9	CRP, mg/l	1.99 ± 1.85
ECLAM	1.92 ± 1.16	Complement c3, g/l	1.02 ± 0.33
SLICC	1.08 ± 1.60	Complement c3, % of patients below cut-off	5 / 26 (19%)
PGA score	1.65 ± 0.63	Complement c4, g/l	0.18 ± 0.06
Glucocorticoids	17 / 26 (65%)	Complement c4, % of patients below cut-off	15 / 26 (58%)
Glucocorticoid dose, mg/d	8 ± 12	ds-DNA-Ab, IU/ml	305 ± 335
Antimalarial agent	16 / 26 (62%)	ds-DNA-Ab, % of patients above cut-off	10 / 26 (38%)
Belimumab	0 / 26 (0%)	ANA	
MMF	6 / 26 (23%)	>1:320	9 / 26 (35%)
MTX	1 / 26 (3.8%)	>1:80 bis ≤1:320	12 / 26 (46%)
Cyclosporine	2 / 26 (7.7%)	≤1:80	5 / 26 (19%)
Number of agents [§]	2.65 ± 0.89		

[1] n (%); mean ± SD. [§] Number of different concomitant immunosuppressive agents for SLE treatment.
SLEDAI-2k: SLE-Disease Activity Index 2000; ECLAM: European Consensus Lupus Activity Measurement Index; SLICC: Systemic Lupus International Collaborating Clinics; PGA: Physician Global Assessment; MMF: mycophenolate mofetil; MTX: methotrexate; Hb: haemoglobin (standard: 12-15 g/dl); ANA: antinuclear antibodies (standard: <1:80); ds-DNA-Ab: anti-double stranded DNA antibody (standard: <200 IU/ml); CRP: C-reactive protein (standard: <5 mg/l), leucocytes standard 3.5-10/nl, thrombocytes standard: 150-360/nl, C3c standard: 0.8-1.9 g/l, C4 standard: 0.2-0.6 g/l.

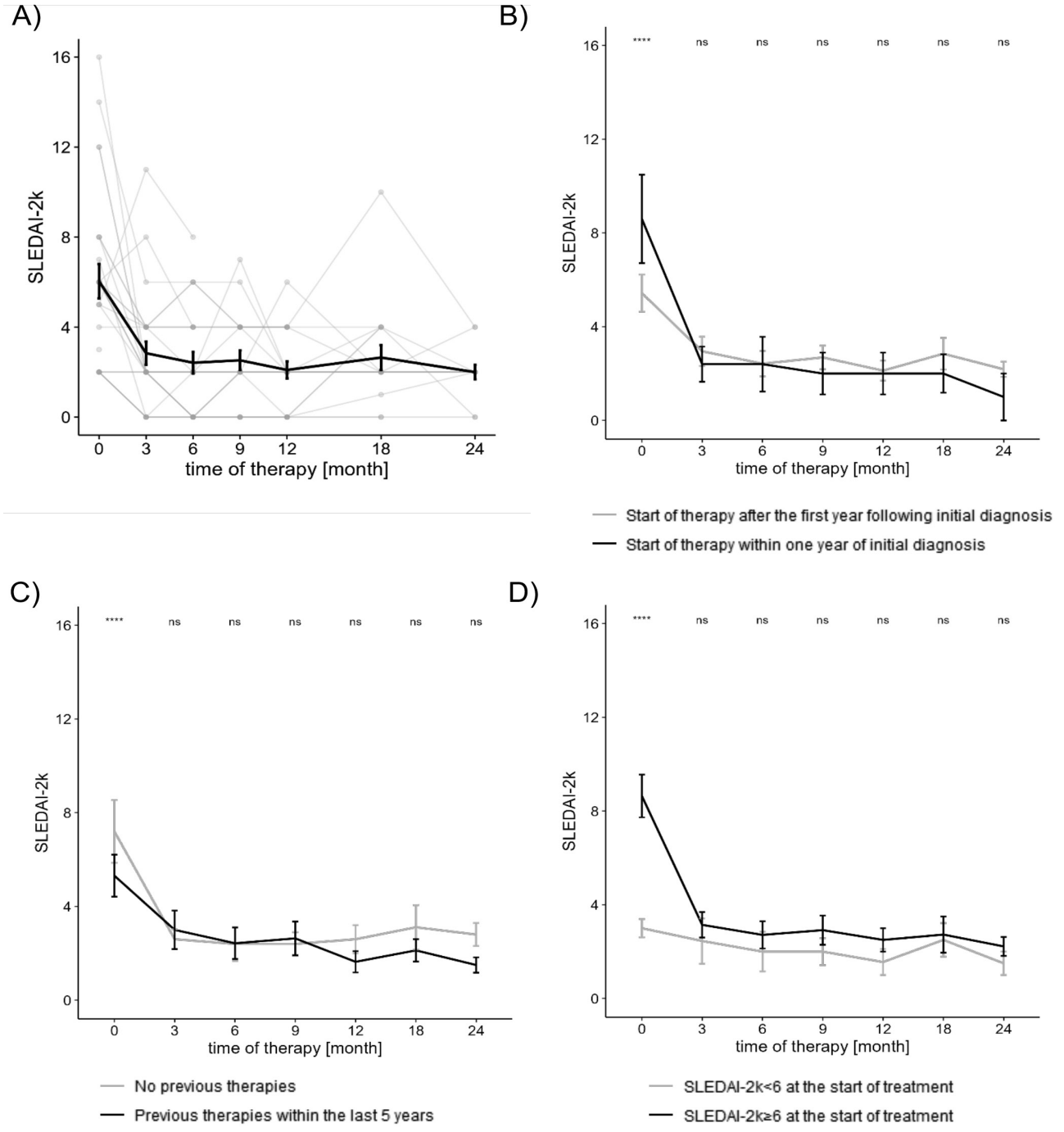

Fig. 2. **A**: SLEDAI-2k over the treatment period. Individual courses (grey lines) as well as mean values (black line) with standard error are shown.
B: Course of SLEDAI-2k dependent on the time of therapy initiation with anifrolumab within the first year after initial diagnosis (n=5, 0 month) compared to a later start of therapy (n=21, 0 month). Mean values with standard error are shown. Statistics with t-test.
C: Progression of SLEDAI-2k depending on prior therapies with strong immunosuppressants (belimumab, MMF, MTX, azathioprine, cyclopsorine, tacrolimus, rituximab) (n=16, 0 month) versus the use of anifrolumab as the first immunosuppressant in addition to glucocorticoids and hydroxycholoroquine (n=10, 0 month). Mean values with standard error are shown. Statistics with t-test.
D: Progression of SLEDAI-2k depending on SLE activity at the time of treatment initiation with anifrolumab with SLEDAI-2k ≥ 6 (n=14, 0 month) compared with SLEDAI-2k < 6 (n=12, 0 month). Mean values with standard error are shown. Statistics with t-test.
SLEDAI-2k: SLE-Disease Activity Index 2000; MMF: mycophenolate mofetil; MTX: methotrexate; ns: not significant. ****$p<0.0001$.

baseline characteristics are shown in Table I. Sixty-five percent of patients were on glucocorticoids at the start of treatment. The average dose was 8 mg prednisolone equivalent per day. If there was no contraindication or intolerance, the patients were treated with hydroxychloroquine. The majority of patients received hydroxychloroquine (16/26, 62%). On average, 2.65 agents were used in treatment. Of the other immunosuppressants used in addition to glucocorticoids and anifrolumab, mycophenolate mofetil (MMF) was the most common at 6/26 (23%). The mean

SLEDAI-2k at inclusion was 6. 14/26 patients had a SLEDAI-2k score ≥6 (6-16) at the time of inclusion, 12/26 patients had a SLEDAI-2k score <6 (2-5). At the start of treatment, 16/26 patients suffered from arthralgia or arthritis, 20/26 patients had mucocutaneous manifestations and 12/26 patients suffered from fatigue. Less frequent were cardiovascular, haematologic, neuropsychiatric manifestations, renal involvement or serositis (Fig. 3A).

Efficacy

The average observation period was 17.5 month. During this period, we observed a significant reduction in the disease activity indices SLEDAI-2k and ECLAM (Fig. 2A, Table II). There was a significant improvement in SLEDAI-2k (6.0±3.9 *vs*. 2.3±1.9, $p \leq 0.001$) and ECLAM (1.92±1.16 *vs*. 0.81±0.81, $p \leq 0.001$) after just 3 months. This was accompanied by a lower Physician Global Assessment (PGA) Score (1.65±0.63 *vs*. 0.81±0.81 after 3 months, $p \leq 0.001$). These effects persisted or continued to improve over the entire observation period.

After a treatment period of 1 year, almost 90% of patients under treatment achieved a lupus low disease activity state (LLDAS) (0/26 (0%) *vs*.19/21 (90%), $p \leq 0.001$) and almost half (0/26 (0%) *vs*. 10/21 (48%), $p \leq 0.001$) achieved a complete remission according to the definition of remission in SLE (DORIS) criteria.

The glucocorticoid dose was reduced from 7.85±11.80 mg to 3.52/1.71/2.31 mg after 6/12/24 months during the course of therapy ($p=0.022$).

We observed that patients benefit from anifrolumab regardless of whether therapy was started soon after initial diagnosis or at a later point in time (Fig. 2B). Regardless of previous therapies with belimumab, MMF, MTX, azathioprine, cyclosporine, tacrolimus or rituximab, there was a comparable reduction in disease activity as measured by SLEDAI-2k (Fig. 2C). There were signs that patients with lower disease activity as measured by SLEDAI-2k <6 could also benefit from anifrolumab, although its use in patients with high disease activity naturally showed greater reductions in disease activity (Fig. 2D). Patients' symptoms improved noticeably under therapy with anifrolumab. In particular, there was a rapid response to mucocutaneous manifestations, while fatigue and articular manifestations improved more slowly (Fig. 3).

Safety

Therapy was discontinued in six cases within the first year; 3 of these patients due to infections (one of which was herpes zoster) and in 3 patients this was due to non-life-threatening infections, one of which was herpes zoster. Two patients had a relapse/non-response after 6 months, one patient after 12 months of treatment (Fig. 1). No patient was hospitalised or died during the observation period.

Discussion

This study reports on the efficacy and safety of anifrolumab in patients with SLE in a real-world setting. Patients with moderate to high disease activity (SLEDAI-2k ≥6, 14/26, 53.8%) as well as patients with lower disease activity as measured by SLEDAI-2k (SLEDAI-2k <6, 12/26, 46.2%) were included. Overall, anifrolumab was effective and well tolerated. Consistent with previous trials, our findings demonstrate a rapid and sustained reduction in disease activity, although most prior evidence has been derived from controlled trial settings rather than real-world cohorts. After 52 weeks of treatment with anifrolumab, the disease activity indices SLEDAI-2k and ECLAM were significantly reduced. Furthermore, measured by LLDAS, almost 90% of patients achieved a low level of activity during treatment. The observed decline in the proportion of patients achieving LLDAS (from 90% to 69%) and DORIS remission (from 48% to 38%) between months 12 and 24 stands in contrast to stable SLEDAI-2K (2.00±1.15) and PGA (0.62±0.65) scores at the final follow-up. This apparent discrepancy likely reflects the stringent definitions of LLDAS and DORIS remission rather than a genuine loss of disease control. Furthermore, the decreasing number of cases under observation over time may have introduced a bias, potentially influencing the interpretation of these outcomes. In patients who no longer met LLDAS or DORIS criteria after 12 months, the most common contributing factors were transient increases in low-dose glucocorticoids during planned tapering attempts and isolated serological abnormalities, such as asymptomatic hypocomplementaemia (*e.g*. isolated C4 decline), which resulted in elevated SLEDAI-2K scores without corresponding clinical disease activity. The LLDAS rate observed in our study after one year is significantly higher than in a *post-hoc* analysis of the TULIP trials. Here, 30% of patients with anifrolumab showed LLDAS after 52 weeks (13). We interpret this finding as a result of the noticeably higher overall disease activity in the TULIP collectives and our per protocol approach. Our higher LLDAS/DORIS rates are consistent with another study which, to our knowledge, is the only larger study in a real-world setting (9). However, in this study only patients from a compassionate use program for the use of anifrolumab in active adult SLE were considered, in whom all available treatment choices had failed, were not tolerated, or were contraindicated (9). In line with this study, we also found that the glucocorticoid dose could be significantly reduced during the course of therapy. After 24 months, all patients were either steroid-free or receiving less than 5 mg/day prednisone equivalent. These real-world findings align with recent claims-based analyses demonstrating reduced disease flares and oral corticosteroid use with anifrolumab over a 6-month period, while our data extend these observations to a 2-year follow-up (14). Thus, anifrolumab appears to offer significant clinical benefits even outside the controlled conditions of randomised trials, further strengthening its role in SLE treatment.

Our data also indicate that anifrolumab is suitable for patients who have already received various prior therapies (Fig. 2C). Even long-standing SLE (Fig. 2B) can benefit from a new therapy with anifrolumab despite many previous therapies. Anifrolumab could also improve disease activity in patients with lower disease activity (Fig.

Table II. Disease activity indices, therapy and laboratory parameters at therapy start and after 3, 6, 9, 12, 18 and 24 months of treatment with anifrolumab.

Characteristic	Therapy start n=26 [1]	3 mo n=25 [1]	6 mo n=24 [1]	9 mo n=21 [1]	12 mo n=21 [1]	18 mo n=17 [1]	24 mo n=13 [1]	*p*-value [2]
SLEDAI-2k	6.04 ± 3.88	2.84 ± 2.58	2.42 ± 2.36	2.52 ± 1.99	2.10 ± 1.73	2.65 ± 2.26	2.00 ± 1.15	<0.001
PGA score	1.65 ± 0.63	0.84 ± 0.47	0.83 ± 0.76	0.67 ± 0.48	0.57 ± 0.60	0.65 ± 0.61	0.62 ± 0.65	<0.001
ECLAM	1.92 ± 1.16	0.94 ± 0.99	0.75 ± 1.08	0.57 ± 0.71	0.50 ± 0.82	0.56 ± 0.70	0.50 ± 0.76	<0.001
LLDAS	0 / 26 (0%)	17 / 25 (68%)	18 / 24 (75%)	16 / 21 (76%)	19 / 21 (90%)	12 / 17 (71%)	9 / 13 (69%)	
DORIS	0 / 26 (0%)	5 / 25 (20%)	8 / 24 (33%)	7 / 21 (33%)	10 / 21 (48%)	5 / 17 (29%)	5 / 13 (38%)	
Glucocorticoids	17 / 26 (65%)	18 / 25 (72%)	17 / 24 (71%)	15 / 21 (71%)	15 / 21 (71%)	7 / 17 (41%)	6 / 13 (46%)	0.28
Glucocorticoid dose, mg/d	7.85 ± 11.80	4.48 ± 3.51	3.79 ± 2.73	3.52 ± 2.62	3.38 ± 2.67	1.71 ± 2.28	2.31 ± 2.59	0.022
Antimalarial agent	16 / 26 (62%)	15 / 25 (60%)	15 / 24 (63%)	13 / 21 (62%)	13 / 21 (62%)	10 / 17 (59%)	8 / 13 (62%)	>0.99
Belimumab	0 / 26 (0%)	0 / 25 (0%)	1 / 24 (4.2%)	1 / 21 (4.8%)	1 / 21 (4.8%)	0 / 17 (0%)	0 / 13 (0%)	0.73
MMF	6 / 26 (23%)	5 / 25 (20%)	5 / 24 (21%)	3 / 21 (14%)	3 / 21 (14%)	3 / 17 (18%)	2 / 13 (15%)	0.99
MTX	1 / 26 (3.8%)	1 / 25 (4.0%)	1 / 24 (4.2%)	0 / 21 (0%)	0 / 21 (0%)	0 / 17 (0%)	0 / 13 (0%)	>0.99
Cyclosporine	2 / 26 (7.7%)	1 / 25 (4.0%)	1 / 24 (4.2%)	1 / 21 (4.8%)	1 / 21 (4.8%)	1 / 17 (5.9%)	1 / 13 (7.7%)	>0.99
Tacrolimus	0 / 26 (0%)	0 / 25 (0%)	0 / 24 (0%)	1 / 21 (4.8%)	1 / 21 (4.8%)	1 / 17 (5.9%)	1 / 13 (7.7%)	0.33
Azathioprine	2 / 26 (7.7%)	1 / 25 (4.0%)	2 / 24 (8.3%)	2 / 21 (9.5%)	3 / 21 (14%)	3 / 17 (18%)	2 / 13 (15%)	0.76
Number of agents [§]	2.65 ± 0.89	2.64 ± 0.81	2.75 ± 0.74	2.71 ± 0.78	2.71 ± 0.85	2.47 ± 0.62	2.54 ± 0.78	0.94
ds-DNA-Ab, % of patients above cut-off	10 / 26 (38%)	9 / 25 (36%)	8 / 24 (33%)	7 / 21 (33%)	5 / 21 (24%)	8 / 17 (47%)	5 / 13 (38%)	0.87
Complement c3, % of patients below cut-off	5 / 26 (19%)	3 / 25 (12%)	2 / 24 (8.3%)	2 / 21 (9.5%)	4 / 21 (19%)	1 / 17 (5.9%)	1 / 13 (7.7%)	0.82
Complement c4, % of patients below cut-off	15 / 26 (58%)	15 / 25 (60%)	13 / 24 (54%)	12 / 21 (57%)	13 / 21 (62%)	13 / 17 (76%)	10 / 13 (77%)	0.72
Leukocytes, cells/nl	6.06 ± 2.20	6.37 ± 1.65	6.71 ± 2.07	6.59 ± 1.79	6.51 ± 2.62	6.65 ± 1.81	7.08 ± 2.61	0.86
Hb, g/dl	13.46 ± 1.65	13.72 ± 2.00	13.63 ± 1.47	13.56 ± 1.52	13.37 ± 1.57	13.74 ± 1.43	13.71 ± 1.55	0.99
Thrombocytes, cells/nl	236 ± 88	264 ± 91	264 ± 91	274 ± 84	285 ± 85	281 ± 96	307 ± 108	0.33

[1] n (%); mean ± SD. [2] Kruskal-Wallis rank sum test; Pearson's Chi-squared test; Fisher's exact test. [§] Number of different concomitant immunosuppressive agents for SLE treatment.
mo: months; SLEDAI-2k: SLE-Disease Activity Index 2000; ECLAM: European Consensus Lupus Activity Measurement Index; PGA: Physician Global Assessment; LLDAS: lupus low disease activity state; DORIS: definition of remission in SLE; MMF: mycophenolate mofetil; MTX: methotrexate; ds-DNA-Ab: anti-double stranded DNA antibody; Hb: haemoglobin.

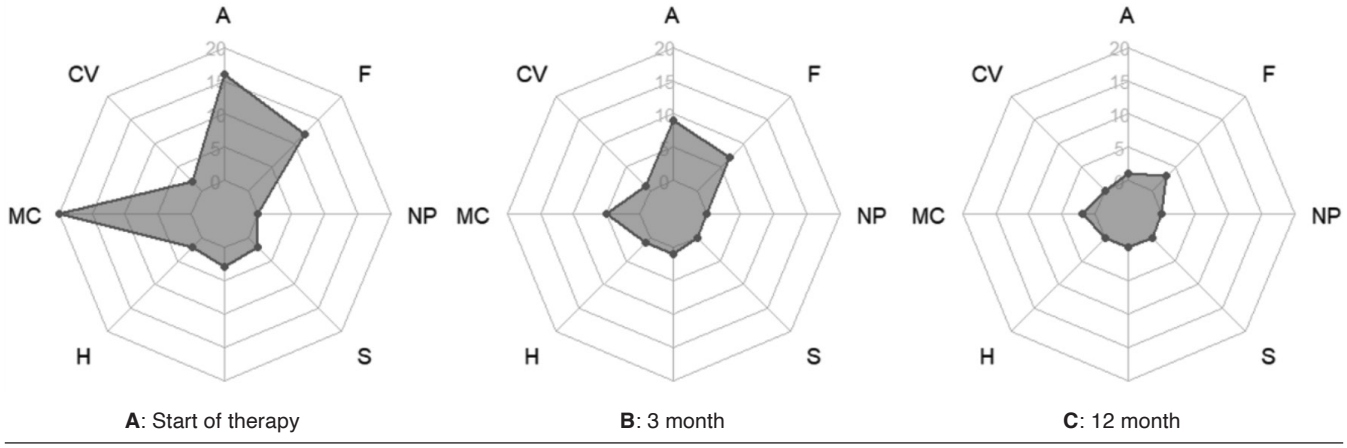

Fig. 3. Manifestations at the start of therapy, after 3 months and 12 months were recorded according to SLEDAI-2k, ECLAM, laboratory parameters and complaints expressed by patients during the doctor's consultation. Symptoms were only recorded when considered to be explained by SLE.
A: articular; **F**: fatigue; **NP**: neuropsychiatric; **S**: serositis; **R**: renal; **H**: haematological; **MC**: mucocutaneous; **CV**: cardiovascular.

2D), supporting its use in a broad spectrum of SLE patients.

Notably we observed a rapid response to the treatment for distinct manifestations, particularly in mucocutaneous involvement. In addition, arthralgia/arthritis and fatigue symptoms also improved. This corresponds to a pooled *post-hoc* analysis of the TULIP studies, which shows that fatigue improves over the duration of treatment (15). Whether this is directly due to anifrolumab or a secondary effect as a result of the general reduction in disease activity remains to be seen. The rapid response to mucocutaneous manifestations in particular is in line with another *post-hoc* analysis of the TULIP

studies (16). Similarly, a large case series evaluating patients with multi-refractory skin disease over a median follow-up of 8.5 months also demonstrated a marked improvement within the first three months of anifrolumab therapy (17). Our data indicate that this effect is sustained over time.

The findings of this study demonstrate that anifrolumab is generally well tolerated. Discontinuation of treatment due to adverse events was required in a small number of patients within the initial six-month period, with these cases being associated with infections, including herpes zoster. These results are in line with those of previous reports and underscore the significance of monitoring for infections (7, 18). Given the rise in infections at the start of therapy, it is vital to consider the impact of increased dosages of other immunosuppressive agents. In the second year of therapy, however, there were no treatment discontinuations due to increasing activity or complications in the small collective, which underlines the potential role of the drug as a long-term therapeutic agent.

While our study provides real-world insights, certain limitations must be acknowledged. Study findings indicate limitations due to small group size, especially for interpreting long-term effects, given the small number of patients especially in the second year. Our single-centre design may limit generalisability. Additionally, as an observational study, there is an inherent risk of selection bias and confounding factors. Fatigue was evaluated based on clinician documentation rather than validated patient-reported outcome measures. Due to the lack of a control group, no conclusions can be drawn about the course of the disease without anifrolumab treatment.

In conclusion, our real-world experience confirms that anifrolumab is an effective and well-tolerated treatment option, demonstrating sustained efficacy and safety over 12–24 months. Our data suggest that SLE patients with active disease benefit from anifrolumab therapy regardless of prior therapies or disease duration. Patients with lower disease activity also appear to benefit. The observed glucocorticoid-sparing effect and broad efficacy across various patient subgroups confirm the findings from the clinical study setting and further support its integration into routine clinical practice. Due to the promising observations in our cohort, an evaluation of the long-term treatment with anifrolumab is planned. Further studies, such as the assessment of the patients' interferon signature, will show whether patients can be identified who particularly benefit from anifrolumab.

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