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Pattern of Sleep Disturbances in Systemic Lupus Erythematosus Patients - A Case - Control Study from North India

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Abstract

Background: Sleep disturbance is an under-recognised but frequent complaint in patients with systemic lupus Erythematosus (SLE) and has been linked to fatigue, mood disturbance and disease activity. Data on the pattern of sleep disturbance in SLE from the Kashmir valley are lacking.

Objectives: To study the pattern of sleep disturbances in patients with SLE and to correlate sleep disturbance with disease activity and duration of disease.

Methods: This analytical, observational, case–control study was conducted over two years (March 2023–March 2025) in the Departments of Rheumatology and Pulmonary Medicine, SKIMS, Srinagar. Fifty-five adult patients satisfying the 2019 ACR–EULAR classification criteria for SLE and 55 age- and sex-matched healthy controls were enrolled after ethical clearance and informed consent. Sleep quality was assessed in all participants using the Pittsburgh Sleep Quality Index (PSQI); patients scoring >5 underwent level 1/2/3 Polysomnography (PSG) to calculate the Apnoea–Hypopnoea Index (AHI). Disease activity in cases was graded using the SLE Disease Activity Index (SLEDAI). Continuous variables were compared using Student's

independent t-test/Mann–Whitney U-test and categorical variables using the chi-square/Fisher's exact test; $p<0.05$ was considered significant.

Results: The mean age of cases (28.89 ± 7.75 years) and controls (31.29 ± 8.44 years) was comparable (p=0.12), as was residency and literacy status. The mean PSQI score was 3.20 ± 1.85 in cases and 3.53 ± 1.96 in controls (p=0.33); 14.5% of cases and 10.9% of controls had a PSQI >5 (p=0.56), indicating that most participants in both groups had good sleep quality. The mean SLEDAI score in cases was 9.27 ± 4.99 (moderate activity in 63.6%). Among cases with poor sleep quality (PSQI >5), 75% had moderate and 25% had high disease activity, although the overall association between SLEDAI category and PSQI category did not reach statistical significance (p=0.13). None of the eight patients who underwent PSG had an AHI ≥5, i.e. no clinically significant obstructive sleep apnoea was detected. Cases had significantly lower haemoglobin, platelet and lymphocyte counts, and lower serum albumin and total protein, with higher urea and creatinine than controls (all $p<0.05$), while comorbidities were present in 36.4% of cases, hypothyroidism being the commonest.

Conclusion: In this cohort, overall sleep quality did not differ significantly between SLE patients and healthy controls; however, a subset of patients reported poor sleep quality that tended to cluster with moderate-to-high disease activity. Polysomnography did not reveal obstructive sleep apnoea as a driver of poor sleep in these patients, suggesting that disease-related factors such as pain, inflammation and psychological stress, rather than a primary intrinsic sleep disorder, underlie sleep disturbance in SLE.

Keywords: Systemic Lupus Erythematosus, Sleep Quality, Pittsburgh Sleep Quality Index, SLEDAI, Polysomnography, Apnoea –Hypopnoea Index

Introduction

Systemic lupus Erythematosus (SLE) is a complex, relapsing–remitting inflammatory autoimmune disease that affects multiple organ systems and gives rise to a wide array of clinical and serological manifestations.[1] The disease disproportionately affects women of child-bearing age and individuals of African-American or Asian descent, and its course is shaped by an interplay of genetic, hormonal and environmental factors. Among the less appreciated of these factors is sleep disturbance, encompassing difficulty initiating sleep, difficulty maintaining sleep, and early morning awakening[2,3]

Sleep disturbance is common in rheumatic diseases and is thought to contribute to the fatigue that characterizes many of them, including SLE.[4] Poor sleep quality has been linked with depressed mood, chronic corticosteroid use, reduced physical activity and elevated inflammatory cytokines.[5],[8] In SLE specifically, disturbed sleep can compromise quality of life, worsen fatigue, precipitate or aggravate depression, increase work absenteeism, and add to healthcare costs.[9],[13] Experimental partial sleep deprivation—comparable to what patients with chronic illnesses routinely experience - alters immune function in ways that may adversely affect inflammatory disease activity and cardiovascular risk.[14],[16] Despite this, sleep problems remain among the most under-recognised unmet needs of patients with SLE.[17]

Previous studies have reported a high prevalence of sleep disturbance in SLE, ranging from about 55% to 85%, with variable correlation to disease activity, pain, fatigue and depression. [18],[22] No study has previously examined the pattern of sleep disturbance in SLE

patients from the Kashmir valley. Given the reported high prevalence of sleep disturbance in SLE worldwide and the inconsistent relationship with disease activity reported across studies, this study was undertaken to characterise the pattern of sleep disturbance in a cohort of SLE patients from a tertiary-care centre in North India and to examine its correlation with disease activity and disease duration.

Materials and Methods

Study design and setting

This was an analytical, observational, case–control study conducted jointly by the Departments of Rheumatology and Pulmonary Medicine, Sher-i-Kashmir Institute of Medical Sciences (SKIMS), Soura, Srinagar, a tertiary-care referral centre in North India, over a two-year period (March 2023 to March 2025). Ethical clearance was obtained from the Institutional Ethics Committee, SKIMS (IEC/SKIMS Protocol #013/2023), and written informed consent was obtained from every case and control prior to enrolment.

Study population

Patients were enrolled from the outpatient and inpatient services of the Rheumatology Division. Fifty-five consecutive adult patients (Group A) fulfilling the 2019 ACR–EULAR classification criteria for SLE were recruited, along with 55 age- and sex-matched healthy individuals with no clinical evidence of disease (Group B, controls), in a 1:1 ratio.

Inclusion criteria: (i) age >18 years; (ii) SLE patients satisfying the 2019 ACR–EULAR criteria (entry criterion of ANA titre ≥1:80 on HEp-2 cells, plus additive clinical and immunological criteria yielding a total score ≥10); (iii) healthy volunteers with no clinical evidence of disease for the control group.

Exclusion criteria: cognitive dysfunction, fibromyalgia, a known psychiatric illness, morbid obesity, sinusitis, a pre-existing diagnosis of obstructive sleep apnoea, and patients receiving corticosteroids at a dose ≥0.5 mg/kg/day.

Assessment of sleep quality

All participants were assessed using the Pittsburgh Sleep Quality Index (PSQI), a 19-item self-report instrument evaluating sleep quality and disturbance over the preceding month across seven components—subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. Each component is scored 0–3, and the seven component scores are summed to yield a global score of 0–21, with higher scores denoting poorer sleep quality; a global score >5 has a reported sensitivity of 89.6% and specificity of 86.5% for distinguishing poor from good sleepers. [23] Patients (and controls) with a PSQI score >5 underwent overnight poly somnography (PSG).

Polysomnography was performed at level 1, 2 or 3 depending on feasibility. Level 1 studies are technician - attended, laboratory - based studies recording electro encephalography (EEG), electro - oculography (EOG) and other channels, and are best suited to complex sleep disorders. Level 2–4 studies are unattended, portable, home-based recordings with progressively fewer channels; level 2 requires a minimum of seven channels (EEG, EOG, chin electromyography, oximetry, airflow and respiratory effort) permitting formal sleep staging, level 3 uses a minimum of four channels (airflow/ventilation, heart rate or EEG, and oxygen saturation), and level 4 is used mainly for sleep apnoea screening. The Apnoea–Hypopnoea Index (AHI) derived from PSG was used to grade sleep-disordered breathing:

normal (AHI <5), mild (AHI 5–<15), moderate (AHI 15–<30) and severe (AHI ≥30) sleep apnoea.

Assessment of disease activity

Disease activity in SLE cases was assessed using the SLE Disease Activity Index (SLEDAI-2K), a validated 24-item weighted instrument scoring clinical and laboratory features present over the preceding 30 days (maximum score 105, though in practice most patients score well below this). Activity was categorised as: no activity (SLEDAI=0), mild (1–5), moderate (6–10), high (11–19), and very high (≥20). A rise in SLEDAI of >3 points was defined as a flare, a fall of >3 points as improvement, fluctuation within 3 points as persistently active disease, and a score of 0 as remission.

Statistical analysis

Data were entered in Microsoft Excel and analysed using SPSS version 29.0 (IBM Corp., Armonk, NY, USA) and R software. Continuous variables were expressed as mean ± standard deviation and compared using Student's independent t-test or the Mann–Whitney U-test as appropriate. Categorical variables were expressed as frequencies and percentages and compared using the chi-square test or Fisher's exact test as appropriate. A two-tailed p-value <0.05 was considered statistically significant.

Results

Fifty-five patients with SLE and 55 age- and sex-matched healthy controls were studied.

Table 1: Age distribution of cases and controls

Group	Mean age ± SD (years)	Range (years)	p-value
Cases (SLE)	28.89 ± 7.75	19–47	0.12
Controls	31.29 ± 8.44	20–48	

Inference: Cases and controls were of comparable age, with the majority of participants (about 80%) in the 21–40-year age band, consistent with the known predilection of SLE for young adults of reproductive age.

Table 2: Residence and educational status of cases and controls

Variable	Cases n (%)	Controls n (%)	p-value
Rural residence	22 (40.0)	29 (52.7)	0.18
Urban residence	33 (60.0)	26 (47.3)	
Literate	36 (65.5)	41 (74.5)	0.53
Illiterate	19 (34.5)	14 (25.5)	

Inference: Cases were more often urban dwellers while controls were marginally more rural, but neither residence nor literacy status differed significantly between the two groups, suggesting these demographic factors were well balanced and unlikely to confound the sleep-quality comparison.

Table 3: Co morbidities among SLE cases (n=55)

Co morbidity	Frequency	Percent
Hypertension	8	14.5
Chronic kidney disease	3	5.45
Diabetes mellitus	3	5.45
Hypothyroidism	20	36.3
None	35	63.6

Inference: Almost two-thirds of the SLE cohort had no comorbid illness. Hypothyroidism was the single commonest co morbidity, present in over a third of patients (alone or with hypertension), underscoring the need for routine thyroid screening in SLE.

Table 4: Haematological parameters in cases and controls

Parameter	Cases (mean ± SD)	Controls (mean ± SD)	p-value
Haemoglobin (g/dL)	11.0 ± 0.52	13.2 ± 0.80	0.003
Haematocrit (%)	35 ± 6.3	42 ± 3.2	0.091
MCV (fL)	80 ± 7	90 ± 5	0.081
Neutrophils (×10³/µL)	3.2 ± 0.9	3.5 ± 0.4	0.102
Lymphocytes (×10³/µL)	2.06 ± 0.4	2.56 ± 0.6	0.042
Platelets (×10³/µL)	105 ± 75	145 ± 54	0.040

Inference: SLE cases had significantly lower haemoglobin, lymphocyte and platelet counts than controls, in keeping with the known haematological footprint of SLE (anaemia of chronic disease, lymphopenia and thrombocytopenia related to disease activity and autoantibody-mediated cytopenias).

Table 5: Biochemical profile of cases and controls

Parameter	Cases (mean ± SD)	Controls (mean ± SD)	p-value
Total bilirubin (mg/dL)	1.27 ± 0.78	1.22 ± 0.80	0.52
ALT (U/L)	46 ± 10.2	44 ± 9.6	0.061
ALP (U/L)	93 ± 59.8	96 ± 61.2	0.62
Total protein (g/dL)	6.47 ± 0.59	7.11 ± 0.48	<0.001
Albumin (g/dL)	3.40 ± 0.45	3.82 ± 0.51	<0.001

Parameter	Cases (mean ± SD)	Controls (mean ± SD)	p-value
Urea (mg/dL)	41.02 ± 8.13	39.11 ± 8.23	0.020
Creatinine (mg/dL)	1.11 ± 0.22	0.99 ± 0.14	<0.001
Corrected calcium (mg/dL)	8.59 ± 0.61	8.50 ± 0.61	0.141

Inference: Cases had significantly lower total protein and albumin and significantly higher urea and creatinine than controls, findings consistent with systemic inflammation, protein loss and a degree of renal involvement, a well-recognised complication of SLE.

Table 6: Global PSQI score in cases and controls

Group	Mean PSQI ± SD	Range	p-value
Cases	3.20 ± 1.85	0–8	0.33
Controls	3.53 ± 1.96	1–9	

Inference: Both groups had, on average, PSQI scores within the 'good sleep quality' range (≤5), and the difference between cases and controls was not statistically significant, indicating that self-reported overall sleep quality was similar between SLE patients and healthy individuals in this cohort.

Table 7: Proportion of good versus poor sleepers (PSQI cut-off of 5)

PSQI category	Cases n (%)	Controls n (%)	p-value
≤5 (good sleep)	47 (85.5)	49 (89.1)	0.56
>5 (poor sleep)	8 (14.5)	6 (10.9)	

Inference: A minority of both cases (14.5%) and controls (10.9%) were classified as poor sleepers, and this difference was not statistically significant, again arguing against a markedly higher burden of poor sleep quality in SLE compared with the general population in this sample.

Table 8: Disease activity (SLEDAI) distribution among cases (n=55)

SLEDAI category	n	Percent
Mild activity (1–5)	9	16.4
Moderate activity (6–10)	35	63.6
High activity (11–19)	7	12.7
Very high activity (≥20)	4	7.3

Mean SLEDAI score: 9.27 ± 4.99 (range 4–25).

Inference: The majority of patients (63.6%) had moderate disease activity at the time of assessment, with only a small minority in remission-equivalent or very-high-activity categories, indicating a cohort that was, on average, in a moderately active but not fulminant disease state.

Table 9: Relationship between disease activity (SLEDAI) and sleep quality (PSQI) in cases

SLEDAI category	PSQI ≤5 n (%)	PSQI >5 n (%)	Total
Mild activity	9 (19.1)	0 (0.0)	9
Moderate activity	30 (63.8)	5 (62.5)	35
High activity	6 (12.8)	1 (12.5)	7
Very high activity	2 (4.3)	2 (25.0)	4
Total	47 (100.0)	8 (100.0)	55

p=0.13 (chi-square test).

Inference: None of the patients with mild disease activity reported poor sleep, whereas both patients with very-high disease activity (2/2, 100%) had poor sleep quality; among poor sleepers, 75% had moderate and 25% had high disease activity. Although the overall association fell short of statistical significance—likely reflecting the small number of poor sleepers (n=8)—the data suggest a activity-dependent gradient in sleep disturbance that merits confirmation in a larger sample.

Table 10: Polysomnography (AHI) findings in the 8 SLE cases with PSQI >5

Patient	AHI
1	1.0
2	1.0
3	2.7
4	4.3
5	3.8
6	4.7
7	3.5
8	3.0

Inference: All eight patients who underwent polysomnography for poor subjective sleep quality had an AHI <5 (range 1.0–4.7), i.e. none met criteria for even mild obstructive sleep apnoea. This indicates that the poor sleep quality reported by these patients was not attributable to sleep-disordered breathing and more likely reflects disease-related factors such as pain, nocturnal symptoms, or psychological distress, as has been suggested in earlier studies.

Among the healthy controls, 6 of 55 (10.9%) had a PSQI score >5; the remaining 49 controls had a mean PSQI of 2.5.

Discussion

This case–control study evaluated the pattern of sleep disturbance in 55 patients with SLE compared with 55 healthy controls from a tertiary-care centre in the Kashmir valley—the first such study from this region.

The mean age of cases (28.89 years) was slightly, but not significantly, lower than that of controls (31.29 years; p=0.12), and around 80% of patients were aged 21–40 years, consistent with the well-documented predilection of SLE for women of reproductive age.[24],[25] Residency and literacy status were also comparable between groups (p=0.18 and p=0.53 respectively), suggesting adequate matching and making it unlikely that these variables confounded the sleep outcomes.

Co morbidities were relatively infrequent in this cohort, with 63.6% of patients having none; hypothyroidism was the commonest association, found in over a third of patients either alone or with hypertension. This mirrors the reported epidemiological association between SLE and primary hypothyroidism.[26]

Cases showed significantly lower haemoglobin, lymphocyte and platelet counts than controls, findings that are a well-recognised haematological signature of SLE and often track with disease activity.[27] Lower serum albumin and total protein together with higher urea and creatinine in cases point to systemic inflammation and a degree of renal involvement, a common and clinically important complication of lupus.[28]

With respect to the primary outcome, global PSQI scores were similar between cases (3.20 ± 1.85) and controls (3.53 ± 1.96), and the proportion of 'poor sleepers' (PSQI >5) did not differ significantly (14.5% vs 10.9%). This finding contrasts with several previous reports of a substantially higher prevalence of poor sleep quality in SLE—ranging from 55% to 85% in various series[18],[21],[22] and 62% in the study by Chandrasekhara et al.[18]—and may reflect differences in disease activity level, corticosteroid dosing (patients on ≥0.5 mg/kg/day steroids were excluded here), co morbidity burden, or cultural and psychosocial factors influencing self-reported sleep quality in this population. Nonetheless, among the subset of cases with poor sleep quality, three-quarters had moderate and one-quarter had high disease activity, and no patient with mild disease activity reported poor sleep—an activity-dependent gradient consistent with the broader literature linking sleep disturbance to disease activity, pain and fatigue in SLE.[18],[21],[29]

Notably, none of the eight patients with poor subjective sleep quality who underwent polysomnography had a clinically significant AHI (all <5). This suggests that obstructive sleep apnoea was not a major contributor to poor sleep in this cohort, echoing the findings of Gudbjörnsson and Hetta, who reported that SLE patients obtain a fairly normal amount of sleep overall but are frequently disturbed by pain and vegetative symptoms such as breathlessness, sweating and palpitations, implicating possible autonomic nervous system involvement rather than a primary respiratory sleep disorder.[7] Similarly, Palagini et al. concluded that psychosocial and psychological factors—particularly depression—are among the most frequently implicated mechanisms linking sleep disorders with SLE, alongside pain and disease activity.[21] Valencia-Flores et al., using formal polysomnography, did report an excess of intrinsic sleep disorders (respiratory and movement-related) in SLE patients together with reduced sleep efficiency and delta sleep correlating with disease

activity;[30] our small PSG subgroup did not replicate the respiratory component of this finding, though our sample of eight patients limits the ability to detect such an association.

Taken together, these findings suggest that although SLE patients as a whole may not universally experience clinically poor sleep quality, a subgroup with more active disease is at higher risk of disturbed sleep, and that this disturbance is unlikely to be driven by obstructive sleep apnoea. Attention to pain control, mood, disease activity and other lupus-specific symptoms—rather than screening for sleep apnoea alone—may therefore be more productive in managing sleep complaints in SLE.

Limitations

This study has several limitations. The sample size, particularly the number of patients undergoing Polysomnography (n=8), was small, limiting statistical power to detect associations between disease activity and sleep quality or sleep-disordered breathing. The cross-sectional design precludes assessment of temporal or causal relationships between disease activity and sleep quality. Depression, anxiety and fibromyalgia-related symptoms, which are known confounders of sleep quality in SLE, were not formally quantified (although patients with a known psychiatric illness or fibromyalgia were excluded). Finally, single-centre recruitment may limit generalisability of the findings.

Conclusion

In this case–control study from the Kashmir valley, overall sleep quality—measured by the PSQI—did not differ significantly between patients with SLE and healthy controls. However, poor sleep quality, when present, tended to occur in patients with moderate-to-high disease activity, and Polysomnography excluded obstructive sleep apnoea as a cause of poor sleep in the affected patients. These findings suggest that sleep disturbance in SLE is more likely driven by disease activity, pain and psychosocial factors than by an intrinsic respiratory sleep disorder, and support incorporating routine sleep-quality screening (e.g. with the PSQI) into the clinical assessment of patients with active SLE, reserving Polysomnography for those with specific features suggestive of sleep-disordered breathing.

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