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ORIGINAL ARTICLE

# Late-onset systemic lupus erythematosus: characteristics and outcome in comparison to juvenile- and adult-onset patients—a multicenter retrospective cohort

Basma M Medhat[1] · Mervat Essam Behiry[2] · Nesreen Sobhy[1] · Yomna Farag[3] · Huda Marzouk[3] · Noha Mostafa[3] · Iman Khalifa[4] · Marwa Elkhalifa[5] · Basma M. Eissa[1] · Eman Hassan ElSayed Hassan[5]

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## Abstract

**Introduction** The aim of this study was to investigate the characteristics and outcome of systemic lupus erythematosus (SLE) among elderly-onset patients.

**Methods** This study included 575 SLE patients managed at Cairo, Alexandria, and Helwan universities from August 2014 to 2018: of whom 49 (8.5%), 420 (73%), and 106 (18.4%) were elderly- (> 50 years), adult- (17–50 years), and juvenile- (≤ 16 years) onset patients, respectively. Cumulative characteristics were recorded. Disease activity at the last visit was investigated through the Systemic Lupus Erythematosus Disease Activity Index-2K (SLEDAI-2K), whereby lupus low disease activity (LLDA) was defined as a SLEDAI-2K score ≤ 4. The disease outcome was assessed through investigating disease damage (Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index (SDI)) and the prevalence of mortality. Quantitative and categorical data were compared using Kruskal–Wallis and Mann–Whitney tests, and chi-square ($\chi^2$) test, respectively.

**Results** Late-onset SLE (LSLE) patients demonstrated the lowest prevalence of constitutional and mucocutaneous manifestations ($p < 0.001$), serositis ($p = 0.006$), nephritis ($p < 0.001$), neuropsychiatric involvement ($p < 0.001$), and hypocomplementinemia ($p < 0.001$), but showed the highest prevalence of comorbidities and multimorbidity (comorbidities ≥ 2) ($p < 0.001$), and positive anti-ds DNA antibodies ($p < 0.001$). Elderly-onset patients demonstrated the lowest SLEDAI-2K and SDI scores, achieved LLDA the most ($p < 0.001$), and developed any damage (SDI ≥ 1) the least ($p < 0.001$). The prevalence of mortality was comparable across the three age groups ($p = 0.6$).

**Conclusions** Late-onset SLE patients (8.5%) showed the lowest prevalence of major organ involvement and the highest prevalence of comorbidities, and demonstrated more favorable disease activity and damage indices.

**Key Points**

- *The disease characteristics and outcome among LSLE patients are characterized by being controversial, with studies from the Middle East being limited. Our cohort constituted of 8.5% elderly-onset SLE patients—who were characterized by the lowest prevalence of major organ involvement and the lowest activity and damage indices—making the disease pattern more favorable in this age group, despite being characterized by the highest prevalence of comorbidities.*

**Keywords** Characteristics · Disease outcome · Elderly onset · Juvenile · Late onset · Systemic lupus erythematosus

✉ Basma M Medhat
basmamedhat@kasralainy.edu.eg

[1] Rheumatology and Rehabilitation Department, Faculty of Medicine, Cairo University, Kasr Al Ainy St., Cairo 11562, Egypt

[2] Internal Medicine Department, Faculty of Medicine, Cairo University, Cairo, Egypt

[3] Pediatric Rheumatology Department, Faculty of Medicine, Cairo University, Cairo, Egypt

[4] Pediatric Department, Faculty of Medicine, Helwan University, Helwan, Egypt

[5] Rheumatology Unit, Internal Medicine Department, Faculty of Medicine, Alexandria University, Alexandria, Egypt

## Introduction

Systemic lupus erythematosus (SLE) is an obscure autoimmune disease distinguished by the wide variation in its clinical and immunological characteristics, with several factors contributing to the disease's heterogeneity including the influence of female sex hormones, hence making it mostly a disease of child-bearing women [1].

Nevertheless, the distinctive disease features among other age groups warrant attention, with patients developing the disease at or above the age of 50 being characterized by the presence of potentially independent, yet superimposing, factors that could influence the disease pattern and outcome such as the high prevalence of comorbidities [2–5], especially with the increase of aging populations over the past few decades [6].

Interestingly, other key players in the pathogenesis of the disease such as race and ethnicity could affect disease characteristics and outcome [4]; yet, data about LSLE in the Middle East is rather scarce [7], with studies investigating LSLE in Egypt, to the best of our knowledge, lacking.

In this study, our aim was to retrospectively investigate the disease characteristics and outcome of LSLE patients in comparison with those with adult and juvenile onset, through a cohort managed at Cairo, Alexandria, and Helwan universities.

## Patients and methods

### Data collection

Seven hundred consecutive medical records of patients fulfilling the 2012 Systemic Lupus Collaborating Clinics (SLICC) classification criteria for SLE [8] and managed at the internal medicine, rheumatology and rehabilitation, and pediatric rheumatology departments of Cairo, Alexandria, and Helwan universities from August 2014 to 2018 were retrospectively viewed, of whom 125 patients were excluded due to the absence of more than 50% of the data, having a disease duration of less than 6 months, incomplete lupus, mere discoid lupus, and/or drug-induced lupus.

The following data were collected from the patients' medical records:

1. Demographic data: (i) age at the last recorded visit or mortality; (ii) age at onset which was determined as the age at the onset of the initial manifestation(s) to be juvenile (onset ≤ 16 years), adult (16–50 years), or late (> 50 years) onset. Adult patients at the time of data collection with juvenile onset were not included in the study due to a potentially longer disease duration; (iii) disease duration was calculated from the onset of symptom(s) until the last recorded visit or mortality
2. Clinical characteristics: cumulative clinical manifestations were recorded. Manifestations were defined according to the SLICC classification criteria [8], in addition to recording the prevalence of comorbidities and multimorbidity which was defined as the presence of two or more comorbidities (comorbidities ≥ 2) [9]
3. Patients were diagnosed with secondary antiphospholipid syndrome (APS) according to the modified Sapporo criteria [10]
4. Serological markers recorded at any time throughout the course of the disease and were marked as positive and negative. Investigated serologic investigations included antinuclear antibody (ANA), anti-double stranded deoxyribonucleic acid (anti ds-DNA), anti-Ro/SSA; anti-La/SSB, and anti-Smith (anti-Sm) antibodies. Presence of either a positive anticardiolipin IgG, anticardiolipin IgM, or lupus anticoagulant was considered a positive antiphospholipid antibody (aPL). Complement 3 and complement 4 were recorded as normal or low
5. Disease activity at the last visit was assessed through the Systemic Lupus Erythematosus Disease Activity Index-2K (SLEDAI-2K) [11]. Lupus low disease activity (LLDA) was defined as a SLEDAI-2K ≤ 4 in the absence of activity in major organ systems (renal, CNS, cardiopulmonary, vasculitis, fever) [12]
6. Disease outcome was investigated through assessing disease damage and the prevalence of mortality. Damage was studied utilizing the Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index (SDI) [13], which was recorded both as continuous (total damage score) and dichotomous variables in order to assess the presence or absence of any damage (SDI ≥ 1)
7. Treatment received by the patients at the last visit was recorded, in which a low dose of glucocorticoids (GC) was defined as oral prednisolone ≤ 7.5 mg/day (mg/d), moderate dose as > 7.5–30 mg/d, and high dose as > 30–100 mg/d [14]

The study was approved by the local ethics committee, according to the provisions of the World Medical Association Declaration of Helsinki.

### Statistical analysis

Data were coded and entered using the statistical package SPSS (Statistical Package for the Social Sciences) version 25. Data was summarized using mean, standard deviation, median, minimum, and maximum in quantitative data and using frequency (count) and relative frequency (percentage) for categorical data. Comparisons between quantitative

variables were done using the non-parametric Kruskal–Wallis and Mann–Whitney test corrected by Bonferroni correction was used as a post hoc test. For comparing categorical data, chi-square ($\chi^2$) test was performed. Exact test was used instead when the expected frequency is less than 5. $p$ values less than 0.05 were considered statistically significant.

## Results

This retrospective cohort included 575 patients, of whom 49 (8.5%) were LSLE patients, and 420 (73%) and 106 (18.4%) were ASLE and JSLE patients, respectively. Demographic features of the three age groups are shown in Table 1.

Several clinical and immunologic differences were detected across the three groups (Table 2). Among the studied constitutional manifestations, fever was least common among LSLE patients ($p < 0.001$), yet weight loss ($p = 0.003$) and lymphadenopathy ($p < 0.001$) were most common; furthermore, they demonstrated the lowest prevalence of several mucocutaneous manifestations, including malar rash ($p < 0.001$), photosensitivity ($p = 0.02$), and cutaneous vasculitis ($p = 0.023$), and were characterized by the absence of discoid rash ($p = 0.03$). Serositis ($p < 0.001$) and leukopenia ($p = 0.006$) were least prevalent in this age group.

Major organ involvement in the form of nephritis (7/49 (14.3%)) and neuropsychiatric (NP) involvement (2/49 (4.1%)) were statistically least prevalent among LSLE patients ($p < 0.001$). The highest prevalence of nephritis belonged to JSLE patients (67%), whereas that of neuropsychiatric (NP) involvement belonged to ASLE patients (25.2%).

Among the investigated serologic investigations, hypocomplementemia ($p < 0.001$) was least commonly detected among LSLE patients, whereas anti-ds DNA was most commonly detected ($p < 0.001$). Antiphospholipid antibodies were significantly higher among ASLE as opposed to LSLE and JSLE patients ($p < 0.001$).

### Comorbidities

Comorbidities were present in almost 75% of elderly-onset patients, thus showing the highest prevalence ($p < 0.001$). Moreover, the occurrence of multimorbidity (comorbidities $\geq 2$) was highest among LSLE patients being present in about 35% ($p < 0.001$). The most prevalent comorbidities among LSLE patients were hypertension (17/49 (34.7%)), followed by osteoporosis (16/49 (32.7%)) ($p < 0.001$). Malignancy occurred in 2/49 (4.1%) of LSLE patients as opposed to its absence among JSLE and ASLE patients ($p = 0.007$). The prevalence and nature of comorbidities across the three groups are shown in Table 3.

### Disease activity and outcome

Late-onset lupus patients showed the lowest SLEDAI-2K score (median 3; interquartile range (IQR) 1–5) with the highest score belonging to JSLE patients (median 10; IQR 6–21) ($p < 0.001$). Moreover, the number of LSLE patients achieving LLDA (SLEDAI-2K $\leq 4$) was highest compared with other age groups ($p < 0.001$). On the other hand, the SDI score, one of the studied disease outcome parameters, was the lowest (median 0; IQR 0–1) among elderly-onset patients ($p < 0.001$), who showed the lowest prevalence of any damage (SDI $\geq 1$) ($p < 0.001$), with the highest SDI score (median 1; IQR 0–3) and highest prevalence of any damage (78.1%) belonging to ASLE patients. The prevalence of mortality ($p = 0.6$) was comparable across the three age groups and there was no difference in the causes of mortality between the three groups ($p = 0.1$) (Table 4).

### Medications

Lower glucocorticoid (GC) dosage and immunosuppression intake were noticed among LSLE patients ($p < 0.001$). Medications received by the patients are shown in Table 5.

**Table 1** Demographic characteristics of the three age groups

| | Juvenile onset ($N = 106$) | Adult onset ($N = 420$) | Late onset ($N = 49$) | $P$ value |
|---|---|---|---|---|
| Age (years) | Mean (SD) (range) | | | |
| Age at last visit or mortality | 12.7 (2.6) (6–16) | 30.8 (8.6) (17–56) | 58.4 (6.3) (51–79) | |
| Age at onset | 9.9 (2.5) (4–15) | 25.3 (7.9) (17–50) | 55 (5.5) (51–77) | |
| Gender | $N$ (%) | | | |
| Male | 20 (18.9) | 49 (11.7) | 3 (6.1) | 0.04 |
| Female | 86 (81.1) | 371 (88.3) | 46 (93.9) | |
| | Median (IQR) | | | |
| Diagnostic lag (months) | 2 (1–3) | 2 (1–6) | 3 (2–5) | 0.01 |
| Disease duration (months) | 24 (12–48) | 60 (24–96) | 24 (11–60) | < 0.001 |

*N*, number; *SD*, standard deviation; *IQR*, interquartile range

**Table 2** Cumulative clinical and immunologic characteristics across the three age groups

| | Juvenile onset ($N = 106$) | Adult onset ($N = 420$) | Late onset ($N = 49$) | $p$ value* |
|---|---|---|---|---|
| Clinical characteristics | | | | |
| Constitutional | 83 (78.3) | 231 (55) | 19 (38.3) | < 0.001* |
| Fever | 76 (71.7) | 227 (54) | 16 (32.7) | < 0.001* |
| Weight loss | 20 (18.9) | 40 (9.5) | 11 (22.4) | 0.003* |
| Lymphadenopathy | 14 (13.2) | 8 (1.9) | 8 (16.3) | < 0.001* |
| Mucocutaneous | 81 (76.4) | 374 (89) | 36 (73.5) | < 0.001* |
| Malar rash | 63 (59.4) | 250 (59.5) | 16 (32.7) | 0.001* |
| Photosensitivity | 52 (49.1) | 183 (43.6) | 13 (26.5) | 0.02* |
| Alopecia | 52(49.1) | 214 (51) | 22 (44.9) | 0.7 |
| Oral ulcers | 28 (26.4) | 138 (32.9) | 14 (28.6) | 0.4 |
| Discoid rash | 1 (0.9) | 23 (5.5) | 0 | 0.03* |
| Cutaneous vasculitis | 26 (24.5) | 109 (26) | 4 (8.2) | 0.02* |
| Arthritis | 18 (17) | 280 (66.7) | 28 (57.1) | < 0.001* |
| Serositis | 39 (36.8) | 204 (48.6) | 14 (28.6) | 0.006* |
| Pleurisy and/or pleural effusion | 22 (20.8) | 172 (41) | 13 (26.5) | < 0.001 |
| Pericarditis and/or pericardial effusion | 24 (22.6) | 88 (21) | 5 (10.2) | 0.1 |
| Neuropsychiatric | 13 (12.3) | 106 (25.2) | 2 (4.1) | < 0.001* |
| Seizures | 9 (8.5) | 39 (9.3) | 1 (2) | 0.24 |
| Psychosis | 9 (8.5) | 41 (9.8) | 0 | 0.04* |
| Peripheral or cranial neuropathy | 1 (0.9) | 5 (1.2) | 1 (2) | 0.65 |
| Nephritis | 71 (67) | 222 (52.9) | 7 (14.3) | < 0.001* |
| Hematologic | 77 (72.6) | 300 (71.4) | 28 (57.1) | 0.1 |
| Hemolytic anemia | 29 (27.4) | 102 (24.3) | 8 (16.3) | 0.32 |
| Thrombocytopenia | 30 (28.3) | 111 (26.4) | 11 (22.4) | 0.7 |
| Leukopenia and/or lymphopenia | 62 (58.5) | 275 (65.5) | 21 (42.9) | 0.006* |
| Secondary APS | 9 (8.5) | 54 (12.9) | 7 (14.3) | 0.42 |
| Other manifestations | | | | |
| Avascular necrosis | 1 (0.9) | 62 (14.8) | 0 | < 0.001* |
| Sicca manifestations | 2 (1.9) | 42 (10) | 5 (10.2) | 0.01* |
| Immunologic characteristics | | | | |
| ANA | 102/104 (96.2) | 396/408 (97.1) | 49 (100) | 0.5 |
| Anti-ds DNA | 72/101 (71.3) | 222/374 (59.4) | 41/48 (85.4) | < 0.001* |
| Hypocomplementemia | 89/105 (84.8) | 289/415 (69.6) | 19/42 (45.2) | < 0.001* |
| aPL | 11/101 (10.8) | 165/332 (49.7) | 12/38 (31.6) | < 0.001* |
| Anti-Ro/SSA | 6/78 (7.7) | 14/123 (11.4) | 3/18 (16.7) | 0.4 |
| Anti-La/SSB | 5/52 (9.6) | 15/97 (15.5) | 2/14 (14.3) | 0.6 |
| Anti-Sm | 16/79 (20.3) | 21/108 (19.4) | 9/47 (19.1) | 0.9 |

Unless indicated, the prevalence of the studied disease characteristics is calculated from the cohort of each age group

*APS*, antiphospholipid syndrome; *ANA*, antinuclear antibody; *Anti-ds DNA*, anti-double-stranded deoxyribonucleic acid antibody; *aPL*, antiphospholipid antibodies; *Anti-Sm*, anti-Smith antibodies

*Significant $p$ value < 0.05

## Discussion

The characteristics and outcome of SLE among patients with a late onset are characterized by being controversial, with studies from the Middle East being limited [7].

Driven by the hormonal milieu, the occurrence of SLE among the elderly is rather uncommon [5], with a prevalence ranging from 3.5 to 20% across various reports [2, 5, 7, 15–23]. LSLE patients constituted 8.5% of our cohort, which is higher than the prevalence reported in studies from Turkey (3.6%) [7] and Saudi Arabia (2.9%) [24], yet lower than that

**Table 3** The nature and prevalence of comorbidities across the three age groups

| | Juvenile onset ($N = 106$) | Adult onset ($N = 420$) | Late onset ($N = 49$) | $p$ value* |
|---|---|---|---|---|
| *N* (%) | | | | |
| Comorbidities | 41 (38.7) | 238 (56.7) | 37 (75.5) | < 0.001* |
| Multimorbidity (comorbidities ≥ 2) | 7 (6.6) | 102 (24.3) | 17 (34.7) | < 0.001* |
| Nature of comorbidities | | | | |
| Osteoporosis | 6 (5.7) | 75 (17.9) | 16 (32.7) | < 0.001* |
| Hypothyroidism | 2 (1.9) | 15 (3.6) | 9 (18.4) | < 0.001* |
| Diabetes mellitus | 4 (3.8) | 72 (17.1) | 9 (18.4) | 0.002* |
| Hypertension | 31 (29.2) | 157 (37.4) | 17 (34.7) | 0.29 |
| Ischemic heart disease | 0 | 16 (3.8) | 1 (2) | 0.08 |
| CVA | 0 | 19 (4.5%) | 1 (2) | 0.04* |
| HCV infection | 5 (4.7) | 22 (5.2) | 7 (14.3) | 0.05 |
| Malignancy | 0 | 0 | 2 (4.1) | 0.007* |
| CKD | 5 (4.7) | 37 (8.8) | 4 (8.2) | 0.41 |
| ESRD | 1 (0.9) | 8 (1.9) | 0 | 0.86 |

*CVA*, cerebrovascular accident; *HCV*, hepatitis C virus; *CKD*, chronic kidney disease; *ESRD*, end-stage renal disease

*Significant $p$ value < 0.05

reported among Tunisians (19%) [17]. This variation in the prevalence of LSLE across various cohorts could be attributed to the different cutoffs in the age chosen to determine elderly onset, which ranged from 50 to 65 years [2, 7, 15–17, 20–22, 24]. Elderly-onset SLE was determined in our study at an age of more than 50 years, in concordance with previous studies in the Middle East [17, 24] and worldwide [18–21].

Among the studied demographic features, LSLE patients were characterized by a pronounced female predominance, which is similar to several reports [3, 18, 22, 25] yet contradictory to several others [18, 20, 23] that showed significant decline in this universal disease feature. LSLE is characterized by insidious onset [5], hence explaining the significantly longer diagnostic lag among our elderly patients ($p = 0.01$).

Of the clinical characteristics investigated, elderly-onset patients in our cohort demonstrated the lowest prevalence of mucocutaneous and constitutional involvement ($p < 0.001$), serositis ($p = 0.006$), and leukopenia ($p = 0.006$), but showing the highest prevalence of sicca manifestations ($p = 0.01$). There was disparity in the prevalence of several manifestations among LSLE across various studies, with some cohorts showing a lower prevalence of constitutional involvement [2, 4, 18, 23], several mucocutaneous [4, 16, 18–21, 25–28] and hematologic [4, 16, 18, 23, 25, 27] manifestations, and serositis [9, 24], and a higher prevalence of sicca manifestations [20, 23], thus resembling our patients, whereas other reports detected a higher prevalence of hematologic [15] involvement and serositis [4] among elderly-onset patients.

This incongruity among various cohorts could be attributed to several factors including racial differences [29]. Interestingly, apart from two studies from the Far East [30, 31], LSLE of heterogeneous ethnicities [2–4, 15, 18–22, 25] showed the lowest prevalence of nephritis ($p < 0.001$) as opposed to that of other age groups, thus resembling our patients.

**Table 4** Disease activity and outcome across the three age groups

| | JSLE ($N = 106$) | ASLE ($N = 420$) | LSLE ($N = 49$) | $p$ value* |
|---|---|---|---|---|
| Disease activity | | | | |
| SLEDAI-2K (median (IQR)) | 10 (6–21) | 4 (2–8) | 3 (1–5) | < 0.001* |
| SLEDAI-2K ≤ 4 (*N* (%)) | 8 (7.5) | 180 (42.9) | 24 (49) | < 0.001* |
| Disease outcome | | | | |
| Disease damage | | | | |
| SDI (median (IQR)) | 1 (0–1) | 1 (0–3) | 0 (0–1) | < 0.001* |
| SDI ≥ 1 (*N* (%)) | 74 (69.8) | 328 (78.1) | 22 (44.9) | < 0.001* |
| Mortality (*N* (%)) | 2 (1.9) | 16 (3.8) | 2 (4.1) | 0.68 |

*N*, number; *IQR*, interquartile ratio; *SLEDAI*, Systemic Lupus Erythematosus Disease Activity Index; *SDI*, Systemic Lupus International Collaborating Clinics Damage Index

*Significant $p$ value < 0.05

**Table 5** Nature of medications received by the cohort at the last visit

| | Juvenile onset ($N = 106$) | Adult onset ($N = 420$) | Late onset ($N = 49$) | $p$ value* |
|---|---|---|---|---|
| Glucocorticoid dosage ($N$ (%)) | | | | |
| None | 0 | 23 (5.5) | 12 (24.5) | < 0.001* |
| Low | 35 (33) | 92 (21.9) | 23 (46.9) | |
| Moderate | 16 (15.1) | 181 (43.1) | 4 (8.2) | |
| High | 55 (51.9) | 124 (29.5) | 10 (20.4) | |
| HCQ | 102 (96.2) | 362 (86.2) | 39 (79.6) | 0.004* |
| Immunosuppressives | 103 (97.2) | 336 (80) | 28 (57.1) | < 0.001* |
| Azathioprine | 34 (32.1) | 195 (46.4) | 21 (42.9) | 0.02* |
| Cyclophosphamide | 10 (9.4) | 95 (22.6) | 3 (6.1) | < 0.001* |
| MMF | 59 (55.7) | 46 (11) | 0 | < 0.001* |
| Cyclosporine | 0 | 0 | 4 (8.2%) | < 0.001* |
| Rituximab | 2 (1.9%) | 1 (0.2%) | 0 | 0.17 |

*HCQ*, hydroxychloroquine; *MMF*, mycophenolate mofetil

*Significant $p$ value < 0.001

Moreover, NP involvement was least common among our elderly-onset patients ($p < 0.001$), which, although contradictory to two previous reports from Latin America [3, 19] that showed a higher prevalence of NP manifestations, is similar to several others [2, 4, 7, 25].

Among the serologic features investigated, hypocomplementemia was least commonly detected among LSLE patients ($p < 0.001$) which is similar to previous studies [2, 16, 18, 25, 30, 32]. On the other hand, contradictory to several reports [18, 25, 27], positive anti-ds DNA antibodies were highest among our LSLE patients ($p < 0.001$), which could be explained by the more stringent diagnosis of SLE among elderly.

The main challenge of managing LSLE patients stems from the high prevalence of comorbidities, a finding reported by various studies [2–5] and demonstrated among our elderly-onset patients ($p < 0.001$). Several authors detected a higher prevalence of osteoporosis [3, 23], hypothyroidism [2, 3], diabetes mellitus [2, 3, 30], and hypertension [2, 3, 18, 23, 30] among their LSLE patients, thus resembling our cohort. Moreover, multimorbidity (comorbidities ≥ 2) was most commonly detected among our LSLE patients which is similar to previous reports [4, 31].

The high prevalence of comorbidities among elderly-onset SLE patients could have contributed to the paradigm shift [2–4, 16] toward the previously suggested more benign nature of the disease [5, 16, 25] in this age group. Nevertheless, our LSLE patients demonstrated more favorable activity and damage indices. Elderly-onset patients in our cohort demonstrated the lowest median SLEDAI-2K score ($p < 0.001$), achieved LLDA (SLEDA-2K ≤ 4) the most, and were less inclined to receive GC and immunosuppressive drugs at the last visit ($p < 0.001$). These findings are similar to several former reports that detected lower activity scores among their LSLE patients [2, 3, 18, 25, 27] and lower GC [19, 21] and immunosuppression [19, 21, 31] administration. Furthermore, our LSLE patients showed the lowest median SDI score and the lowest prevalence of occurrence of any damage (SDI ≥ 1) ($p < 0.001$). These findings are contradictory to several reports that detected higher SDI scores [2–4, 15] and a higher occurrence of any damage (SDI ≥ 1) among their elderly-onset patients [2, 3, 19], while other studies demonstrated no difference in SDI scores between various age groups [22, 30, 31]. Indeed, several intermingling factors could contribute to disease damage including the disease duration [33], GC dosage [34], and higher disease activity [35]. It is of note that the highest median SDI score in our cohort, and the highest prevalence of any damage belonged to ASLE patients, who demonstrated the longest disease duration ($p < 0.001$) and a substantially high medication intake.

The prevalence of mortality was comparable across the three age groups ($p = 0.6$), which is contradictory to that of previous reports [3, 21, 22, 31] but similar to another [19].

The main limitation of our study lies in its retrospective nature, thus leading to the absence of some data. On the other hand, our study has many strengths as it is, to the best of our knowledge, the first study from Egypt investigating the disease characteristics and outcome among elderly-onset SLE patients, and included patients from several centers.

To conclude, LSLE patients in our cohort were characterized by the lowest prevalence of major organ involvement and demonstrated the lowest disease activity and damage scores, the highest prevalence of LLDA, and the least damage developed (SDI ≥ 1), but they showed the highest prevalence of comorbidities and multimorbidity (comorbidities ≥ 2), hence showing several similarities and disparities to their peers across the globe which could be attributed to several factors including the retrospective nature of the studies, the rather small number of elderly-onset patients, and the different inclusion criteria. Moreover, race and ethnicity are

multidimensional and surpass genetic ancestry per se [29] to include environmental, social, and cultural aspects [36], thus further adding to the complexity of this intricate disease.

**Compliance with ethical standards** The study was approved by the local Ethics Committee, according to the provisions of the World Medical Association Declaration of Helsinki.

**Disclosures** None.

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