The Egyptian Journal of Hospital Medicine (January 2023) Vol. 90, Page 71- 78

Late onset Systemic Lupus Erythematosus: Different Clinical, Serological Presentations and Damage Compared to Adult Lupus in Egypt

Samah A. El Bakry, Mariem Abd-Elrahman,
Marwa Mohamed Elsayed, Fatma Mohammed Badr*
Internal Medicine, Rheumatology and Immunology, Faculty of Medicine, Ain Shams University, Cairo, Egypt
***Corresponding author:** Fatma Mohammed Badr, **Mobile:** (+20)01274307978, **Email:** dr_fatma2020@yahoo.com

ABSTRACT
Background: Comparing cases of adult onset and late onset systemic lupus erythematous (SLE) reveals significant differences in clinical, serological, disease activity, and damage score.
Objective: This study aimed to analyze clinical manifestations, laboratory data, serological markers, and prognosis of late-onset SLE (L-SLE) and for comparing with adult- onset SLE.
Patients and Methods: One hundred fifty individuals with SLE were included in a cross-sectional study conducted at Ain Shams University Hospital. They were divide into: **Group 1** included 100 cases with adult-onset (age of onset ≥ 19 years and below 50 years). **Group 2** included 50 Patients with L-SLE (age of onset ≥ 50 years). All patients were subjected to medical history, physical examination, disease activity measured by the SLE disease activity index (SLEDAI-2K) and a damage score. Laboratory investigations as complete blood count (CBC), blood urea nitrogen (BUN), serum creatinine, anticardiolipin antibodies, lupus anticoagulant, protein creatinine ratio, serum complement (C3, C4), anti-dsDNA antibody, and antinuclear antibodies (ANA).
Results: Mucocutaneous manifestations, frequency of hematuria, proteinuria, urinary cast, consumed C3, positive anti-dsDNA antibodies, anti-cardiolipin antibody and lupus anticoagulant titers had considerably greater rates in-group 1 compared to group 2 (P-value <0.05) while group 2 had significantly more musculoskeletal symptoms (P-value <0.05). The SLEDAI scores of the two groups were equivalent, however the damage index was greater in group 2 (P-value 0.00). Neuropsychiatric, cutaneous, renal, and skin damage were more frequent in group 1, while musculoskeletal, endocrinal, pulmonary, cardiovascular and ocular damage were more frequent in- group 2.
Conclusion: late-onset SLE is different from adult onset SLE with more frequent damage.
Keywords: Adult SLE, L-SLE, SLEADI, Damage score.

INTRODUCTION
Clinical and laboratory symptoms of the autoimmune illness as SLE can vary widely. Despite lacking of understanding of its origin, we know that genetic, hormonal, and environmental variables all play a part in the progression of this disease [(1)].

Systemic signs of SLE include fever and arthritis, but it can also harm the kidneys and the central nervous system. The prognosis and quality of life of a patient can be improved by taking measures to prevent permanent harm to vital organs [(2)]. Both the intensity and clinical manifestations of the disease are influenced by the age at which it first appeared. The onset and progression of L-SLE (50 years) are less obvious and less severe. [(3)] that characterized by a reduced incidence of nephritis and central nervous system involvement compared to the adult-onset type [(4)].

Nonetheless, L- SLE has a worse prognosis because of age and prolonged exposure to vascular risk factors, which results in an increased prevalence of concomitant disorders and increased organ damage [(5)].

Our study aimed to analyze clinical manifestations, laboratory data, serological markers, and prognosis of L-SLE and for comparing with adult-onset SLE.

PATIENTS AND METHODS
I) Patients:
The current cross-sectional study recruited 150 SLE patients diagnosed upon SLE classification criteria established by Systemic Lupus International Collaborating Clinics (SLICC) [(6)] who attended Ain Shams University Hospitals. They were divided into two groups: Group 1 included 100 cases with adult-onset SLE. Adult onset SLE was defined as age of onset ≥ 19 years and below 50 years. Group 2 included 50 patients with L-SLE. L-SLE was defined as age of onset ≥ 50 years. Patients with other autoimmune diseases and those with drug-induced lupus were excluded from the study.

II) Methods:
Complete clinical evaluations were performed for all patients, including a thorough history taking session where age, sex, disease duration, and presenting symptoms were emphasized. A thorough physical examination and a thorough musculoskeletal examination. Laboratory assessment including CBC, urine analysis besides assessing active urinary sediments (white blood cells, red blood cells, as well as casts or proteins), kidney function tests (serum creatinine and BUN), protein creatinine ratio, erythrocyte sedimentation rate (ESR) by Westergren method, ANA and direct immunofluorescence with anti-double-stranded DNA antibodies. Serum complements levels C3 and C4 by Cobas C6000 autoanalyzer, anticardiolipin (IgG & IgM) antibodies by enzyme-linked immunosorbent test (ELISA) and lupus anticoagulant by diluted Russell viper venom time (Roche Diagnostics GmbH, Mannheim, Germany). The

Received: 24/7/2022
Accepted: 25/9/2022

medical records of 51 patients were reviewed for information on the histological evaluation of renal biopsies. A revision to the histopathological classification of lupus nephritis (LN) by the International Society of Nephrology and the Renal Pathology Society was used [(7)].

Assessment of disease activity:

Methods for evaluating disease activity in SLE (SLEDAI- 2K)[(8)]. The SLEDAI scores were used to define the following groups of activities: not active when SLEDAI is zero, activity is mild when SLEDAI ranges from one to five, activity is moderate when SLEDAI ranges from six to ten, activity considered high when SLEDAI ranges from eleven to nineteen and activity is considered very high when SLEDAI equals or higher than twenty.

Assessment of SLE damage:

Damage was evaluated using the SLICC/ACR damage index for systemic lupus, developed by the American College of Rheumatology [(9)]. Apart from signs including stroke and myocardial infarction, items have to last six months at least. Damage is defined for 12 organ systems: malignancies (0–2), gonadal (0–1), endocrine (diabetes) (0–1), skin (0–3), musculoskeletal (0–7), gastrointestinal (0–6), peripheral vascular (0–5), cardiovascular (0–6), pulmonary (0–5), renal (0–3), neuropsychiatric (0–6) and ocular (0–2). Damage may either be steady or increase with time (up to a theoretical maximum of 47 points).

Ethical considerations:

We followed the guidelines set in the Declaration of Helsinki by the World Medical Association for this study. All participating patients were given a thorough explanation of the study's goals and methods, and all gave their informed consent before participation. The protocol has been approved by Ain Shams University Ethics Committee.

Statistical analysis

We used SPSS, version 20.0, statistical software to analyse the collected data (SPSS Inc., Chicago, IL, USA). Our quantitative data were displayed as mean ± SD, while our qualitative data were shown in the form of frequencies and percentages. The Chi-square test (X^2) was used to analyse statistical significance between two qualitative groups, whereas the t-test was used to analyse statistical significance between two quantitative groups. To determine whether or not a p-value was statistically significant, we used the following criteria: When the P-value is equal or less than 0.05, it is deemed significant; when it is less than 0.001, it is regarded extremely significant; and when it is greater than 0.05, it is considered insignificant.

RESULTS

Comparing between the two groups as regards demographic data, we found that male to female ratio was 6.6:1 in group (1) while 11.5:1 in group (2) without any statistically significant (P -value > 0.05). Average range of disease duration in-group 1 was 0.5-5 years with a mean of 2.97 ± 1.30 while in-group 2 it was 0.5-5 years with a mean of 2.71 ± 1.48 with no statistical significant (P -value > 0.05). Regarding difference between the two groups concerning clinical manifestations at the onset of the disease, in terms of acute cutaneous lupus, we identified a huge statistically significant difference between the two groups malar rash-photosensitivity and antiphospholipid manifestations (P-value < 0.001) being higher in-group1. Concerning musculoskeletal problems, there was a huge discrepancy between the two groups (p-value <0.001) being higher in-group (2). While, no statistically significant differences were seen between both groups in terms of any other clinical symptoms (P-value > 0.05) (Table 1).

Table (1): Comparative study between group (1) and (2) as regard different demographic and clinical data

Demographic data		Group 1 No. = 100	Group 2 No. = 50	Test value	P-value
Disease Duration (years)	Mean ± SD	2.97±1.30	2.71±1.48	1.120•	0.265
	Range	0.5 – 5	0.5 – 5		
Sex	Male	13 (13.0%)	4 (8.0%)	0.829*	0.362
	Female	87 (87.0%)	46 (92.0%)		
	Female to male Ratio	6.6:1	11.5:1		

Clinical manifestations	Group 1 N=100		Group 2 N=50		Test value*	P-value
	No.	%	No.	%		
Fever	57	57.0%	40	80.0%	7.717	0.005**
Mucocutaneous manifestations	**83**	**83.0%**	**45**	**90.0%**	**1.305**	**0.253**
Non-Scaring Alopecia	56	56.0%	25	50.0%	0.483	0.487
Oral ulcers	38	38.0%	18	36.0%	0.057	0.811
Subacute Cutaneous or Discoid Lupus	11	11.0%	5	10.0%	0.035	0.852
Acute Cutaneous Lupus	60	60.0%	17	34.0%	9.020	0.003**
Malar rash	51	51%	13	26.0%		
Photosensitivity	11	11.0%	5	10.0%		
Musculoskeletal	**61**	**61.0%**	**43**	86%	9.798	0.002**
Serositis	**13**	**13.0%**	**8**	**16.0%**	**0.249**	**0.618**
Renal affection	**27**	**27.0%**	**10**	**20.0%**	**0.879**	**0.348**
Proteinuria	27	27.0%	10	20.0%	0.879	0.348
Hematuria	1	1.0%	0	0.0%	0.503	0.478
Neuro-psychiatric manifestations	**8**	**8.0%**	**1**	**2.0%**	**2.128**	**0.145**
2ry Antiphospholipid	**22**	**22.0%**	**2**	**4.0%**	**8.036**	**0.005****
Recurrent Abortions	14	14.0%	0	0.0%	7.721	0.005
DVT`s	11	11.0%	2	4.0%	2.063	0.151
Raynaud`s phenomena	**13**	**13.0%**	**5**	**10.0%**	**0.284**	**0.594**
Hematological manifestations	**37**	**37.0%**	**24**	**48.0%**	**1.672**	**0.196**
Leucopenia	13	13.0%	3	6.0%	1.714	0.190
Thrombocytopenia (Bleeding tendency)	18	18.0%	12	24.0%	0.750	0.386
Autoimmune Hemolysis	12	12.0%	12	24.0%	3.571	0.059

•: Independent Sample t-test; *x^2 – Chi-square test

Note: P-value >0.05: Non-significant (NS); P-value <0.05: Significant (S); P-value< 0.01: highly significant (HS).

**Significant test.

In the meantime, we noticed a huge statistical difference between the two groups in terms of ESR when comparing laboratory results (p-value <0.05) with higher values in group (2). On the other hand, there was statistically significant difference between the two groups as regards frequency of albuminuria, anti-DNA antibodies positivity, protein/creatinine ratio and consumed C3 being higher in-group (1) (p-value <0.05). However, there was no discernible difference in any laboratory results between the two groups (p-value >0.05) (Table 2).

Table (2): Comparative study between the two groups as regard different laboratory data

		Group 1 No. = 100	Group 2 No. = 50	Test value	P-value
CBC					
TLC (10^3/ µl)	Mean ± SD	6.00 ± 1.32	6.72 ± 1.23	-1.365•	0.174
HGB (g/dl)	Mean ± SD	10.81 ± 1.68	10.63 ± 1.69	0.610•	0.543
PLT (10^3/ µl)	Mean ± SD	241.49 ± 58.61	224.66 ± 53.32	1.045•	0.298
ESR (mm/h)	Mean ± SD	35 ± 6.34	56.5 ± 12.51	-4.959‡	0.000**
Urine Analysis					
Pus cells (pyuria)	Mean ± SD	3 ± 0.61	4.5 ± 1.1	-1.506‡	0.132
RBCs(hematuria)	Mean ± SD	2 ± 0.41	2 ± 0.42	-0.748‡	0.455
Albumin (Albuminuria)	Negative	40 (40.0%)	29 (58.0%)	4.348*	0.037**
	Positive	60 (60.0%)	21 (42.0%)		
Urinary cast	Negative	91(91.0%)	48(96.0%)	1.226*	0.268
	Positive	9 (9.0%)	2 (4.0%)		
Serum Creatinine(mg/dl)	Mean ± SD	1.10 ± 0.50	1.17 ± 0.48	-0.794•	0.428
ALT(IU/L)	Mean ± SD	31.82 ± 7.12	30.94 ± 6.82	0.283•	0.777
AST(IU/L)	Mean ± SD	29.62 ± 6.63	32.28 ± 6.54	-1.068•	0.287

Investigations at Onset		**Group 1 No. = 100**	**Group 2 No. = 50**	**Test value**	**P-value**
ANA Immunofluorescence (positive)		100 (100.0%)	50 (100.0%)	NA	NA
Anti-DNA(u/ml) (positive)		86 (86.0%)	34 (68.0%)	6.750*	0.009**
ACL Antibodies					
IgG (positive)		37 (37.0%)	12 (24.0%)	2.561*	0.110
IgM (positive)		29 (29.0%)	8 (16.0%)	3.032*	0.082
LAC (positive)		42 (42.0%)	13 (26.0%)	3.675*	0.055**
C3(mg/dl)	Mean ± SD	54.5 ± 12.31	69.5 ± 14.81	-2.016‡	0.044**
C3(consumed)	N%	81 (81.0%)	36 (72.0%)	1.573*	0.210
C4(mg/dl)	Mean ± SD	10 ±2.31	13 ± 4.1	-0.385‡	0.700
C4 (consumed)	N%	58 (58.0%)	30 (60.0%)	0.055*	0.815
Proteinuria					
Protein/Creatinine ratio (mg/gm))	Mean ± SD	0.20 ± 0.041	0.14 ± 0.025	-2.211‡	0.027**
Coombs Test					
Direct	No	85 (85.0%)	40 (80.0%)	0.600*	0.439
	Yes	15 (15.0%)	10 (20.0%)		
Indirect	No	92 (92.0%)	46 (92.0%)	0.000*	1.000
	Yes	8 (8.0%)	4 (8.0%)		

ANA: Antinuclear antibody, **C3**: complement 3, **C4**: complement 4, **LA**: lupus anticoagulant, **ACL**: anticardiolipin**, ESR**: erythrocyte sedimentation rate, TLC: total leucocytic count, HGB: hemoglobin, PLT: platelets, AST: aspartate transaminase, ALT: alanine transaminase **Anti dsDNA**: anti-double-stranded deoxyribonucleic acid antibody

•: Independent Sample t-test; *x^2 – Chi-square test Note: P-value >0.05: Non-significant (NS); P-value <0.05: Significant (S); P-value< 0.01: highly significant (HS). **Significant test.

Comparing frequency of renal biopsy classes between two groups, we found that frequency of all renal biopsy classes being higher in-group 1 compared to group 2 with no statistical significance (p value <0.05). The common classes in adult group were class II and class III, while the most common classes among L-SLE were class III then class IV as presented in table (3).

Table (3): Comparative study between the two groups as regard frequency of renal biopsy classes

Renal Biopsy classes	Group 1 No. = 100	Group 2 No. = 50	Test value	P-value
Not done	**63 (63.0%)**	**36 (72.0%)**	**6.659***	**0.247**
Class I	**1 (1.0%)**	**0 (0.0%)**		
Class II	**11 (11.0%)**	**1 (2.0%)**		
Class III	**11 (11.0%)**	**7 (14.0%)**		
Class IV	**10 (10.0%)**	**6 (12.0%)**		
Class V	**4 (4.0%)**	**0 (0.0%)**		

P-value >0.05: Non-significant (NS); P-value <0.05: Significant (S); P-value< 0.01: highly significant (HS).
**Significant test.

On the SLEDAI, there was no statistically significant difference between the two groups in terms of the overall score. (P-value >0.05), but in moderate disease activity score was statistically significant higher in-group (1) (P-value <0.05) (Table 4).

Table (4): Comparative study between the two groups as regard SLE activity according to SLEDAI score

		Group 1 No. = 100	Group 2 No. = 50	Test value	P-value
Total score of SLEDAI	Median (IQR)	6 (4 – 10)	6 (4 – 8)	-0.898‡	0.369
	Range	0 – 31	0 – 20		
Classification of activity according to SLEDAI score	No Activity (0)	2 (2.0%)	4 (8.0%)	3.125	0.077
	Mild Activity (1-5)	36 (36.0%)	12 (24.0%)	2.206	0.137
	Moderate Activity (6-10)	39 (39.0%)	29 (58.0%)	4.856	0.028**
	High activity (11 - 19)	18 (18.0%)	4 (8.0%)	2.663	0.103
	Very high activity (>20)	5 (5.0%)	1 (2.0%)	0.781	0.377

SLEDAI: Systemic Lupus Disease Activity Index Note: P-value >0.05: Non-significant (NS); P-value <0.05: Significant (S); P-value< 0.01: highly significant (HS). **Significant test.

Regarding damage score according to SLICCA/ACR damage index, L- SLE patients had significantly higher SLICCA damage score median (IQR): 2.5 (2 – 4); range: 0-6] compared to adult patients [median (IQR): 1 (1–2); range 0-7], (P-value 0.00). Meanwhile, comparing between the two groups regarding frequency of damage parameters, ocular damage, musculoskeletal damage, endocrine damage, lung damage, and heart damage all differed significantly between the two groups (P-value <0.05) being more in-group (2). On the other hand, frequency of renal damage being significantly more in-group (1) (P-value < 0.05) (Figure 1 & 2).

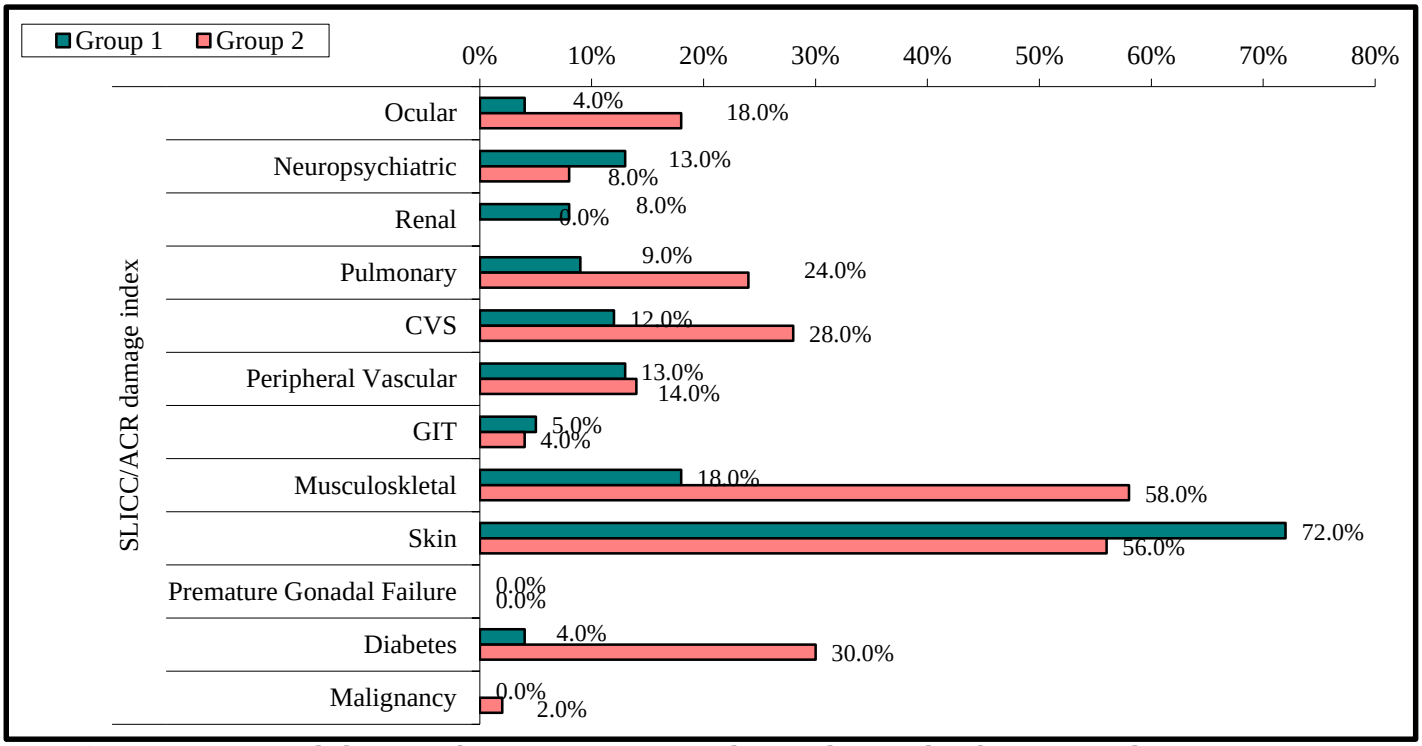

Fig. (1): Comparative study between the two groups as regard organ damage distribution according to SLICC /ACR damage index score

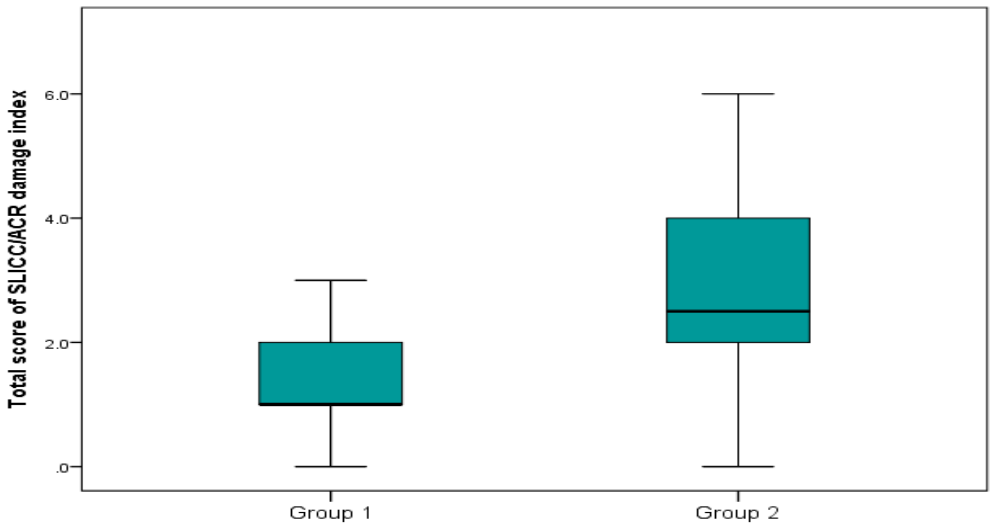

Fig. (2): Comparative study between the two groups as regard total SLICC /ACR damage index score

DISCUSSION

SLE is chronic multisystem autoimmune illness that primarily affects women, has a wide range of clinical presentations and a complicated etiology. Genes, epigenomes, immune regulators, cultures, hormones, and the environment all play roles in the pathogenesis of SLE [(11)].

L-SLE prevalence varies in the literature, likely due to ethnic characteristics and the use of different cutoff values in different studies. Although, onset can occur at any age, SLE more commonly affects women of childbearing age. The prognosis for patients with L-SLE is worse, despite the reduced frequency of significant organ involvement and the milder clinical course, which may be due to age-related situations such increased comorbidities and longer exposure to cardiovascular risk factors [(12)].

Our cross-sectional study aimed to evaluate demographic, clinical, and serological data from L-SLE and adult-onset SLE to determine whether or not these two forms of lupus are different.

L- SLE has a significantly higher sex ratio than adults do (11.5:1), whereas adults have a much lower sex ratio of 6.6:1, nonetheless, there was no discernible difference between the two groups statistically (p-value= 0.362). This is in agreement with **Cildag *et al.*** [(3)] and **Alonso *et al.*** [(12)] who discovered no gender differences between adults and people with L- SLE at a statistically significant level. While, **Medhat *et al.*** [(13)] who discovered a decreased male to female ratio in adults with SLE (11.7: 88.3 to 6.1:93.9) respectively. However, Brazilian study conducted by **Sassi *et al.*** [(4)] found that there were significantly fewer females than males in the late-onset group. That could be due to a variety of cultural and geographical variables.

L-SLE patients had a greater mortality rate because of the more gradual disease development, longer diagnostic delay, and subsequent lack of treatment. Thus, special care should be taken with this subset of SLE patients to prevent unnecessary delays or misdiagnoses. [(14)].

Our investigation found a statistically significant difference between the two groups in terms of the incidence of acute cutaneous lupus (malar rash-photosensitivity) (P-value < 0.05) being higher in adult onset SLE. This is in agreement with most of literatures like **Medhat *et al.*** [(13)]**, Cildag *et al.*** [(3)]**, and Ferucci *et al.*** [(15)].

Meanwhile, musculoskeletal symptoms (Arthritis/Arthralgia) was significant higher in L-SLE compared to adult SLE. This is in agreement with **Feng *et al.*** [(16)] but in contrast to **Choi *et al.*** [(17)] who found that musculoskeletal manifestation are more in adult lupus than in L-SLE.

Antiphospholipid manifestations were more frequent in- adult onset SLE compared to L-SLE (p-value <0.05). This is in accordance with **Medhat *et al.*** [(13)] and **Choi *et al.*** [(17)]. While, against **Cildag *et al.*** [(3)] and **Sousa *et al.*** [(18)] who both found no significant difference between both groups as regards antiphospholipid syndrome.

Among different laboratory data, we found that positive anti DNA antibodies were higher in adult SLE than in L-SLE (86% vs 68%) (P value=0.009). This is against **Medhat *et al.*** [(13)] who found that the presence of positive anti-DNA antibodies were significantly greater in the L-SLE than in adult group. While, our study is matching with results obtained by **Choi *et al.*** [(17)] and **Sassi *et al.*** [(4)].

In this study, we found that frequency of proteinuria and hematuria were statistically significant higher in adult SLE than in L-SLE, but there was no statistically significant difference between both groups as presence of urinary cast but still higher in adult than in L-SLE. These results are in accordance with results obtained by **Choi *et al.*** [(17)] who discovered a statistically significant difference between the two groups (with adult cases being more common than L-SLE cases in terms of hematuria, proteinuria, and urinary cast) (p value =0.02). However, **Xu *et al.*** [(19)] found that between adult and L-SLE regarding renal affection, there was no

statistical significant difference as regards hematuria, proteinuria and urinary cast.

In our study, we found that protein creatinine ratio was higher in adult SLE than in L-SLE. These results were in agreement with **Song *et al.*** [20] who found that there was statistically significant increase in 24-hour urinary proteinuria in adult in comparison with L-SLE patients. Also, **Sassi *et al.*** [4] and **Kutky and Aloudat** [21] found that frequency of proteinuria was significantly higher in adult SLE patients than in L-SLE. Our result pointed to more aggressive disease nature in younger age.

Studying different histopathological types of lupus nephritis according to WHO classification, we found that the most common class of lupus nephritis in adult SLE was class II & III (11%), while the most common class of lupus nephritis in L-SLE was class III (14%). Our result is close to **Xu *et al.*** [19] and **Song *et al.*** [20] who found that in terms of renal function, L-SLE patients fared better than their counterparts. Although there was no statistically significant difference between the L-SLE and adult-onset groups, it appeared that the L-SLE group had a smaller proportion of class IV patients and a higher proportion of class V patients.

The risk of developing LN, adult-onset SLE was significantly higher among those who regularly consumed C3, according to our findings. Our findings are comparable to those of several other studies [22, 17].

There is strong association between positive anti DNA antibodies active nephritis [23]. That can explain the higher frequency of nephritis, proteinuria and positive anti DNA in adults patients in comparison with L-SLE.

We also found that positive anti cardiolipin antibodies (IgG and IgM) and lupus anticoagulant antibodies were higher in adult SLE patients with no statistical significant difference. These results are in agreement with different studies who found that anti cardiolipin antibodies and lupus anticoagulant antibodies positivity were higher in adult SLE than in L-SLE (p value< 0.001) [12 , 13, 17], while another one found that the incidence of antiphospholipid antibodies were higher in elderly patients (p<0.01) [24].

We discovered no statistically significant difference in overall SLEDAI scores between the two groups (P= 0.369) where the SLEDAI score ranged from 0-31 with the median (IQR) 6 (4 – 10) in adult group, while it ranged from 0-20 with the median (IQR) 6 (4 – 8) in L-SLE group. This agrees with two studies who found that there was no difference between these two age groups in the incidence of severe flares [25, 5]. On other hand, different studies found that Elderly-onset patients significantly demonstrated the lowest median SLEDAI-2K score (p < 0.001) [13, 4].

Our findings may be explained by the fact that patients of advanced age with low disease activity rarely visited our clinic and that the majority of our elderly population came from inpatients who had been hospitalized with severe illness and high disease activity. The prognosis in L-SLE worsens with age and prolonged exposure to vascular risk factors, which increases the likelihood of developing several comorbid conditions and further damages multiple organs. [3].

Our data showed that the total SLICC/ACR damage index score was significantly higher in L-SLE than in adult SLE, with a P-value of 0.00. This is in accordance with **Tomic-Lucic *et al.*** [26] and **das Chagas Medeiros *et al.*** [11] who found that SLICC damage index showed a greater damage in L-SLE. While, against study of **Medhat *et al.*** [13] who found that L-SLE patients showed the lowest median SLICC damage index score and the lowest prevalence of occurrence of any damage (p < 0.001). Meanwhile, **Cartella *et al.*** [25] and **Feng *et al.*** [16] demonstrated no difference in SLICC damage index scores between various age groups.

When comparing the two groups according to the extent of damage to certain organs, we discovered a statistically significant difference (P 0.05) favouring L-SLE in terms of ocular damage. This is in agreement with **Ugarte and Alarcón** [27].

Musculoskeletal, endocrinal damage, pulmonary and cardiovascular damage were more frequent in L-SLE group compared to adult one (P-value <0.05). This is in agreement with different studies [3, 26, 28]. But, against **Choi *et al.*** [17] who found that musculoskeletal damage are more in adult lupus than in L-SLE.

Meanwhile, renal system damage was more in adult group than in L-SLE group (P-value < 0.05). This is in agreement with **Sassi *et al.*** [4] while against **Chen *et al.*** [29] who found that renal damage was more among L-SLE group than adult group. Logistic regression research demonstrated that renal involvement in L-SLE is a prediction for damage; chronic renal failure is a univariate predictor of death, but renal problem is not [30]. These variations may result from the interaction of several factors, such as the severity of the disease and the length of time it has been present [31], GC dose [32], and higher disease activity [33]. Damage caused by the disease itself and that caused by the natural ageing process cannot be differentiated using the SLICC-DI. Furthermore, our study did not include other outcome measures like mortality.

CONCLUSION

Compassionate treatment that strikes a balance between controlling disease activity and medicine side effects can assist improve long-term results in L-SLE by reducing accumulated damage, which is one way in which the disease differs from adult onset SLE.

Conflict of interest: The authors declared no conflict of interest.

Sources of funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author contribution: Authors contributed equally in the study.

REFERENCES

1. **Wang F, Zhang W, Wang S *et al.* (2018):** Protective effects of antimalarial in Chinese patients with systemic lupus erythematous. Ann Rhum Dis., 78: 80. doi: 10.1136/annrheumdis-2018-213819
2. **Shaharir S, Said M, Rajalingham S *et al.* (2020):** Distinct clinical features of late onset systemic lupus erythematosus among Malaysian multi-ethnic cohort. Annals of the Rheumatic Diseases, 79: 368-369.
3. **Cildag S, Kara Y, Cakir E *et al.* (2019):** Comparison of clinical and laboratory findings in patients with systemic lupus erythematosus with regard to age at onset. The Eurasian Journal of Medicine, 51 (1): 17-22.
4. **Sassi R, Hendler J, Piccoli G *et al.* (2017):** Age of onset influences on clinical and laboratory profile of patients with systemic lupus erythematosus. Clinical Rheumatology, 36 (1): 89-95.
5. **Sohn I, Joo Y, Won S *et al.* (2018):** Late-onset systemic lupus erythematosus: Is it "mild lupus"? Lupus, 27 (2): 235-242.
6. **Petri M, Orbai A, Alarcón G *et al.* (2012):** Derivation and validation of the Systemic Lupus International Collaborating Clinics classification criteria for systemic lupus erythematosus. Arthritis & Rheumatism, 64 (8): 2677-2686.
7. **Weening J, D'agati V, Schwartz M *et al.* (2004):** ON Behalf of the International Society of Nephrology and Renal Pathology Society Working Group on the Classification of Lupus Nephritis. The classification of glomerulonephritis in systemic lupus erythematosus revisited. Kidney Int., 65: 521–530.
8. **Gladman D, Iban˜ ez D, Urowitz M (2002):** Systemic lupus erythematosus disease activity index 2000. J Rheumatol., 29: 288–291.
9. **Gladman D, Ginzler E, Goldsmith C *et al.* (1996):** The development and initial validation of the Systemic Lupus International Collaborating Clinics/American College of Rheumatology damage index for systemic lupus erythematosus. Arthritis Rheum., 39: 363–9.
10. **Zucchi D, Elefante E, Schilirò D *et al.* (2022):** One year in review 2022: systemic lupus erythematosus. Clinical and Experimental Rheumatology, 40 (1): 4-14.
11. **Das Chagas Medeiros M, Bezerra M, Braga F *et al.* (2016):** Clinical and immunological aspects and outcome of a Brazilian cohort of 414 patients with systemic lupus erythematosus (SLE): comparison between childhood-onset, adult-onset, and late-onset SLE. Lupus, 25 (4): 355-363.
12. **Alonso M, Martinez-Vazquez F, de Teran T *et al.* (2012):** Late-onset systemic lupus erythematosus in Northwestern Spain: differences with early-onset systemic lupus erythematosus and literature review. Lupus, 21 (10): 1135-1148.
13. **Medhat B, Behiry M, Sobhy N *et al.* (2020):** Late-onset systemic lupus erythematosus: characteristics and outcome in comparison to juvenile and adult-onset patients-a multicenter retrospective cohort. Clin Rheumatol., 39 (2): 435-442.
14. **Kernder A, Richter J, Fischer Betz R *et al.* (2021):** Delayed diagnosis adversely affects outcome in systemic lupus erythematosus: Cross sectional analysis of the LuLa cohort. Lupus, 30: 431-8.
15. **Ferucci E, Johnston J, Gaddy J *et al.* (2014):** Prevalence and incidence of systemic lupus erythematosus in a population-based registry of American Indian and Alaska Native people, 2007-2009. Arthritis Rheumatol., 66: 2494–2502.
16. **Feng X, Zou Y, Pan W *et al.* (2014):** Associations of clinical features and prognosis with age at disease onset in patients with systemic lupus erythematosus. Lupus, 23 (3): 327-334.
17. **Choi J, Park D, Kang J *et al.* (2015):** Comparison of clinical and serological differences among juvenile-, adult-, and late-onset systemic lupus erythematosus in Korean patients. Lupus, 24 (12): 1342–1349.
18. **Sousa S, Gonçalves M, Inês L *et al.* (2016):** Clinical features and long-term outcomes of systemic lupus erythematosus: comparative data of childhood, adult and late-onset disease in a national register. Rheumatol Int., 36 (7): 955–960.
19. **Xu Y, Tan Y, Yu F *et al.* (2011):** Late onset lupus nephritis in Chinese patients: classified by the 2003 International Society of Nephrology and Renal Pathology Society system. Lupus, 20 (8): 801-808.
20. **Song K, Liu X, Liu J *et al.* (2020):** Analysis of clinical and laboratory characteristics and pathology of lupus nephritis-based on 710 renal biopsies in China. Clinical Rheumatology, 39 (11): 3353-3363.
21. **Kutky M, Aloudat S (2018):** Late-onset systemic lupus erythematosus with lupus nephritis in a 74-year-old male: a brief case and review. Canadian Journal of Kidney Health and Disease, 5: 2054358118793397. doi: 10.1177/2054358118793397
22. **Lalani S, Pope J, De Leon F *et al.* (2010):** Clinical features and prognosis of late-onset systemic lupus erythematosus: results from the 1000 faces of lupus study. J Rheumatol., 37 (1): 38–44.
23. **Rastin M, Mahmoudi M, Sahebari M *et al.* (2017):** Clinical & immunological characteristics in systemic lupus erythematosus patients. Indian J Med Res., 146: 224–229
24. **Chanprapaph K, Tubtieng I, Pratumchat N *et al.* (2021):** Cutaneous, systemic features and laboratory characteristics of late-versus adult-onset systemic lupus erythematosus in 1006 Thai patients. Lupus, 30 (5): 785-794.
25. **Cartella S, Cavazzana I, Ceribelli A *et al.* (2013):** Evaluation of mortality, disease activity, treatment, clinical and immunological features of adult and late onset systemic lupus erythematosus. Autoimmunity, 46 (6): 363– 368.
26. **Tomic-Lucic A, Petrovic R, Radak-Perovic M *et al.* (2013):** Late-onset systemic lupus erythematosus: clinical features, course, and prognosis. Clin Rheumatol., 32 (7): 1053–1058.
27. **Ugarte-Gil M, Alarcón G (2013):** Is there an effective treatment for late-onset systemic lupus erythematosus? Aging Health, 9 (4): 437-450.
28. **Bultink I (2012):** Osteoporosis and fractures in systemic lupus erythematosus. Arthritis Care & Research, 64 (1): 2-8.
29. **Chen T, Wong C, Lee C *et al.* (2009):** Systemic lupus erythematosus in the elderly. International Journal of Gerontology, 3 (2): 108-113.
30. **Lin H, Wei J, Tan C *et al.* (2012):** Survival analysis of late-onset systemic lupus erythematosus: a cohort study in China. Clin Rheumatol., 31: 1683–1689.
31. **Prasad R, Ibanez D, Gladman D *et al.* (2006):** Anti-dsDNA and anti-Sm antibodies do not predict damage in systemic lupus erythematosus. Lupus, 15 (5): 285–291.
32. **Al Sawah S, Zhang X, Zhu B *et al.* (2015):** Effect of corticosteroid use by dose on the risk of developing organ damage over time in systemic lupus erythematosus—the Hopkins lupus cohort. Lupus Sci Med., 2(1): e000066. doi: 10.1136/lupus-2014-000066
33. **Nossent J, Kiss E, Rozman B *et al.* (2010):** Disease activity and damage accrual during the early disease course in a multinational inception cohort of patients with systemic lupus erythematosus. Lupus, 19 (8): 949–956.