



# Spectrum of systemic lupus erythematosus in Oman: from childhood to adulthood

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## Abstract

SLE is a disease that mainly affects women of childbearing age, however, a total of 15–20% of cases present in children. Although adult onset SLE (aSLE) and childhood onset SLE (cSLE) share the same diagnostic criteria, differences have been identified. The aim of this study is to compare the similarities and differences in between cSLE and aSLE in an Arab population from Oman. We evaluated 225 SLE patients, 139 adults and 86 children, who fulfilled the criteria for diagnosis. At disease onset, 99% of SLE cohort fulfilled the SLICC criteria; however the ACR 1997 criteria were fulfilled in 66% aSLE and 80% cSLE. The clinical features of SLE in cSLE showed higher frequency of renal (50 vs 19%;  $p < 0.001$ ), musculo-skeletal (67 vs 53%;  $p = 0.036$ ) and pulmonary involvement (13 vs 2.9%,  $p = 0.005$ ); while aSLE showed higher frequency of hematological (64 vs 49%;  $p = 0.25$ ) and mucocutaneous (24 vs 10%;  $p = 0.13$ ) involvement. The mean disease activity score at disease onset and during disease course was also higher in cSLE (13 vs 8.5;  $p < 0.0005$ ) (16 vs 11.8;  $p < 0.0005$ ), respectively. Differences in autoantibody profile were also noted in cSLE with higher positivity of anti-dsDNA and antiphospholipid antibody (94 vs 84%;  $p = 0.027$ ) (53 vs 37%;  $p = 0.25$ ), respectively. cSLE patients were more likely than aSLE to be treated with immunosuppressant such as cyclophosphamide (51 vs 22%;  $p < 0.001$ ) and MMF (70 vs 54%;  $p = 0.019$ ). Similarities and differences between aSLE and cSLE in a cohort from Oman of Arab ethnicity were identified. It appears that individual races and ethnicities may exhibit differences in disease susceptibility and manifestations.

**Keywords** Childhood SLE · Adult SLE · Systemic lupus erythematosus

## Introduction

Systemic lupus erythematosus (SLE) is a complex autoimmune disease. The clinical heterogeneity of the disease mirrors its complex immune and genetic pathogenesis. Many factors contribute to the diversity of clinical phenotypes of

SLE including race, ancestry, ethnicity, gender and age at disease presentation [1–6]. Although SLE mainly affects women of childbearing age, however, its prevalence is not confined within this population. A total of 15–20% of SLE cases present in children < 16 years of age. Disease onset in children most frequently occurs between the ages 12 and 16 years, while it is uncommon before the age of 10 years and very rare before 5 years. Childhood onset SLE (cSLE) is in fact the same disease that occurs in adult onset SLE (aSLE); however, substantial differences in the initial presentation, disease severity, serology, treatment response and outcome have been reported [5–11].

Several reports demonstrate that cSLE has a more severe disease onset with higher disease activity, and higher major organ involvement, namely renal, neuropsychiatric and hematological [5, 6]. A recent meta-analysis demonstrated that cSLE has a different autoantibody profile than aSLE with increased anti-dsDNA and anticardiolipin antibody, and decreased rheumatoid factor positivity [12]. Comparative

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studies indicate that cSLE is more often treated with higher doses of corticosteroids and immunosuppressive medications than aSLE [6, 13, 14]. In addition, comparative long-term follow-up studies demonstrate that a significantly higher percentage of cSLE are on oral steroids compared to aSLE [15]. Furthermore, statistically significant differences in cumulative organ damage and mortality between cSLE and aSLE were noted [6, 14–16]. Although the age at diagnosis has a strong modulating effect on clinical presentation, disease course, response to treatment and prognosis, the findings are not always consistent across studies due to geographic and ethnic variations.

There is a scarcity of published literature on clinical phenotype of SLE in Oman. However, previous studies have demonstrated that both cSLE and aSLE have clinical characteristics that are unique to the region [17–20]. Given the complexity of clinical features of SLE which is influenced by racial, ethnic, genetic and environmental factors, this study aims to compare the differences and similarities in demographic, clinical presentation, disease severity, serology and treatment between cSLE and aSLE in an Omani population.

## Methods

### Patients

A cross-sectional study was conducted at Sultan Qaboos University Hospital, a tertiary hospital affiliated to an academic institute. We included patients with the diagnosis of SLE who were followed at the pediatric and adult rheumatology department from January 2006 to July 2016. cSLE was defined as onset of SLE in children at  $\leq 16$  years of age, while those diagnosed above the age of 16 were classified as having aSLE. All patients who were included in the study met the 1997 American College of Rheumatology (ACR) revised criteria or the Systemic Lupus International Collaborating Clinics Classification criteria (SLICC) for SLE or both [21, 22]. Patients were excluded from the study if they did not satisfy any of the SLE classification criteria, had other autoimmune rheumatological disorders or had missing data at disease onset. Ethical approval was obtained from the local research and ethics review committee.

### Study variables

Demographic, clinical and immunological data were retrospectively collected from the hospital information system. The following variables were analyzed: demographic characteristics, clinical manifestation of disease at onset and during course of illness, serology, disease severity and treatment. Demographic characteristics included age at disease onset,

gender, disease duration, and geographical regional origin. Clinical data collected included constitutional symptoms such as fever and weight loss, cutaneous manifestations (malar rash, photosensitivity, discoid rash and cutaneous vasculitis), mucosal involvement (oral or nasal ulcers), articular manifestation (arthritis), renal involvement (haematuria, proteinuria and casts), central nervous system manifestations (seizures, psychosis and headache), hematological involvement (leucopenia, lymphopenia, haemolytic anemia and thrombocytopenia), cardiovascular manifestations (pericarditis, myocarditis and hypertension), pulmonary involvement (pneumonitis and pleural effusion), gastrointestinal symptoms (abdominal pain, hepatomegaly, hepatosplenomegaly) and lymphadenopathy. Serological parameters, including antinuclear antibodies (ANA), were determined by immunofluorescence using Hep-2 cells as substrate with a cut of  $> 1:80$ . Autoantibodies including anti-double-stranded DNA (anti-dsDNA), anti-extractable nuclear antigen (anti-ENA) profile including (Ro, La, Smith, and ribonuclear proteins (RNP), as well as antiphospholipid antibodies) were measured qualitatively using enzyme-linked immunosorbent assay (ELISA) technique. Autoantibodies were considered positive if the value was above the cut-offs for the laboratory at least in one determination during the follow-up period, except for anticardiolipin antibodies, which were considered present if there was two positive occasions 12 weeks apart.

Pathological classifications of renal biopsy specimens in patients with lupus nephritis were according to International Society of Nephrology/Renal Pathology Society (ISN/RPS) 2003 [23]. Disease activity at onset and during the course of disease was assessed using SLE disease activity index (SLEDAI) [24]. Treatment with the following medications was recorded: non-steroidal anti-inflammatory drugs (NSAID), hydroxychloroquine, prednisolone, azathioprine, methotrexate, mycophenolate mofetil, cyclophosphamide, cyclosporin, rituximab, intravenous immunoglobulin, calcium and vitamin D.

### Statistical analysis

Descriptive statistics were used to describe the data, and for categorical variables, frequencies and percentages were reported, with differences between groups analyzed using Pearson's chi-square test (or Fisher's exact test for cells  $< 5$ ). For continuous variables, mean and standard deviation (median and interquartile range when not normally distributed) were used to summarise the data. Analyses were performed using a Student's *t* test (or Wilcoxon–Mann–Whitney test as appropriate). An a priori two-tailed level of significance was set at 0.05 and statistical analyses were performed using STATA version 13.1 (STATA Corporation, College Station, Texas, USA).

## Results

A total of 225 patients with the diagnosis of SLE were included in the study comprising 139 (62%) aSLE and 86 cSLE (38%). The demographic data of the patients are summarized in Table 1. cSLE cohort were 79% ( $n=68$ ) girls with a mean age at diagnosis of  $9.5 \pm 4.2$  and with a median disease duration of 8 (5–13) years. The overall female:male (F:M) ratio in cSLE was 4:1; however, differences in ratios were noted across different age groups. The F:M ratio in

children diagnosed < 5 years was 2.5:1, 5–12 years was 3.5:1, and 13–16 years old was 6.7:1. On the other hand, aSLE cohort consisted of 89% ( $n=124$ ) females, had a mean age at diagnosis of  $28 \pm 8.8$  years with median disease duration of 5 (3–8) years. The overall F:M ratio was 8:1; however, differences in ratios were also noted across different groups. The ratio was highest when the diagnosis was made between the ages of 17–25 years 11:1; in the intermediate in age group of 25–50 years the ratio was 6.5:1 and least after the age of 50 years was 3:1.

**Table 1** Demographics, clinical and laboratory characteristics of aSLE and cSLE patients at disease onset

Characteristics, $n(\%)$ unless stated otherwise	aSLE ( $n=139$ )	cSLE ( $n=86$ )	$p$ value
Age, mean $\pm$ SD, years	$34 \pm 9$	$18 \pm 7.1$	<0.001
Age at diagnosis, mean $\pm$ SD, years	$28 \pm 9$	$9.5 \pm 4.2$	<0.001
Disease duration, median (IQR), years	5 (5)	8 (7)	<0.001
Male gender	15 (11%)	18 (21%)	0.037
Region			
Muscat	36 (26%)	16 (19%)	0.002
Al Batinah	42 (30%)	20 (23%)	
Sharqiya	22 (16%)	33 (38%)	
Ad Dakhliyah	16 (12%)	11 (13%)	
Others	23 (16%)	6 (7%)	
ACR fulfillment	92 (66%)	69 (80%)	0.023
SLICC fulfillment	138 (99%)	85 (99%)	0.731
Clinical features at presentation			
Malar	33 (24%)	9 (10%)	0.013
Discoid	7 (5.0%)	0	0.046
Oral ulcer	18 (13%)	6 (7.0%)	0.158
Photosensitivity	17 (12%)	0	<0.001
Alopecia	22 (16%)	22 (26%)	0.073
Arthritis	74 (53%)	58 (67%)	0.036
Serositis	21 (15%)	17 (20%)	0.365
Renal	27 (19%)	43 (50%)	<0.001
Hematological	89 (64%)	42 (49%)	0.025
Hemolytic anemia	22 (16%)	23 (27%)	0.047
Lymphopenia	62 (45%)	19 (22%)	<0.001
Neurologic	12 (8.6%)	15 (17.4%)	0.048
Pulmonary	4 (2.9%)	11 (13%)	0.005
Constitutional	84 (60%)	10 (12%)	<0.001
Complement status, $n(\%)$			
C3	90 (65%)	77 (90%)	<0.001
C4	90 (66%)	69 (80%)	0.019
ANA	136 (98%)	84 (98%)	0.934
Anti-dsDNA	117 (84%)	80 (94%)	0.027
Anti-smith	51 (37%)	18 (21%)	0.015
Antiphospholipid antibody	53 (37.4%)	46 (53.5%)	0.024
SLEDAI at disease onset, mean $\pm$ SD	$8.5 \pm 4.7$	$13.3 \pm 6.4$	<0.001

aSLE systemic lupus erythematosus, cSLE child systemic lupus erythematosus, SD standard deviation, IQR interquartile range, ACR American College of Rheumatology, SLICC systemic lupus international collaborating clinics classification criteria (SLICC), SLEDAI systemic lupus erythematosus disease activity index

**Table 2** The clinical features of cSLE and aSLE throughout disease course

Clinical features	aSLE (n=139)	cSLE (n=86)	p value
Malar	54 (39%)	12 (14%)	<0.001
Discoid	12 (8.6%)	1 (1.2%)	0.019
Oral ulcer	28 (20%)	7 (8.1%)	0.016
Photosensitivity	31 (22%)	2 (2.3%)	<0.001
Alopecia	30 (22%)	24 (28%)	0.280
Arthritis	84(60%)	64 (74%)	0.032
Serositis	31 (22%)	26 (30%)	0.184
Renal	46 (33%)	55 (64%)	<0.001
Lymphopenia	91 (65%)	41 (48%)	0.008
Hemolytic anemia	31 (22%)	32 (37%)	0.016
Neurologic	33 (24%)	26 (30%)	0.282
Pulmonary	10 (7.2%)	20 (23%)	0.001
Constitutional	95 (68%)	53 (62%)	0.302
SLEDAI over disease course, mean $\pm$ SD	11.8 $\pm$ 6.4	16.3 $\pm$ 7.0	<0.001

aSLE systemic lupus erythematosus, cSLE child systemic lupus erythematosus, SLEDAI systemic lupus erythematosus disease activity index, SD standard deviation

Interestingly, there was a difference in geographical distribution of disease among cSLE and aSLE ( $p=0.002$ ). Geographical clustering of cSLE was identified in Sharqiya region (38%), while aSLE had a more equal distribution of disease among residents from Al Batinah (30%) and Muscat (26%) regions.

The clinical features at time of diagnosis of cSLE and aSLE are described in Table 1. At disease onset 99% of SLE cohort fulfilled the SLICC criteria; however, a significant difference was noted in fulfilling the ACR criteria (66% aSLE vs 80% cSLE) at disease onset. Similarly, at disease onset, cSLE had significantly higher frequency of major organ involvement including renal (50 vs 19%;  $p<0.001$ ), neuropsychiatric (17 vs 8.6%;  $p=0.048$ ), pulmonary (13 vs 4%;  $p=0.005$ ), hemolytic anemia (27 vs 16%;  $p=0.045$ ) and arthritis (67 vs 53%;  $p=0.036$ ). While aSLE had significantly higher involvement of cutaneous manifestations (malar, 24 vs 10%;  $p=0.013$ ; discoid rash, 5 vs 0%;  $p=0.045$ ; photosensitivity, 12 vs 0%;  $p<0.001$ ), lymphopenia (45 vs 22%;  $p<0.001$ ) as well as constitutional symptoms (60 vs 12%;  $p<0.010$ ).

The clinical features of SLE throughout the disease course showed a similar trend to the clinical features at disease onset in the majority of clinical presentation between cSLE and aSLE with renal, pulmonary, hemolytic and arthritis being predominant in cSLE while cutaneous manifestation and hematological manifestation being predominant in aSLE. However, it was interesting to note that neuropsychiatric manifestations during disease course in aSLE matched cSLE while constitutional manifestation in

cSLE matched aSLE showing no difference during disease course (Table 2).

As shown in Table 1, disease activity at disease onset, measured by SLEDAI, was higher in cSLE (13 vs 8.5;  $p<0.001$ ). Similarly, as shown in Table 2, the highest mean SLEDAI score measured during disease course was also highest among cSLE (16 vs 11.8;  $p<0.001$ ).

At disease onset and during disease course, there was higher tendency of renal involvement in cSLE compared to aSLE. However, despite the difference in frequency of renal involvement, the clinical presentation of renal manifestation was similar. Hematuria, proteinuria and active sedimentation rate occurred in similar frequency (data not shown). Similarly, there was comparable distribution of lupus nephritis histological classes among cSLE compared to aSLE with diffuse lupus nephritis (class IV) occurring in highest frequency followed by focal lupus nephritis (class III) as shown in Table 3. Interestingly, there was higher occurrence of minimal mesangial lupus nephritis (class I) in cSLE as (8.6 vs 0%), while there was higher occurrence of membranous lupus nephritis (class V) in aSLE (15.6 vs 6.4%).

The relative incidence of autoantibodies detected in SLE is summarized in Table 4. ANA and Anti-dsDNA antibodies were the most common autoantibodies detected with a higher frequency of anti-dsDNA positivity in cSLE (94 vs 84%). Additionally, antiphospholipid antibodies (lupus anticoagulant, anticardiolipin antibodies, antiglycoprotein antibodies) were more often detected in children (54 vs 37%;  $p=0.026$ ) while anti-smith antibodies occurred more commonly among adults (37 vs 21%;  $p=0.025$ ). The incidence of presence of other antibodies was the same both in children and in the adult SLE population.

The prescribed treatment of SLE at disease onset and during course of illness is described in Table 5. Glucocorticoid was the most frequently prescribed treatment without

**Table 3** Comparing characteristics of lupus nephritis between aSLE and cSLE

Characteristics	Adults n=139 (62%)	Children n=86 (38%)
Lupus nephritis	46 (33%)	55 (64%)
Patients with renal biopsy	32 (70%)	46 (84%)
WHO classification		
Class I	0	3 (6.5%)
Class II	2 (6.3%)	4 (8.7%)
Class III	7 (22%)	7 (15%)
Class IV	16 (50%)	29 (63%)
Class V	5 (16%)	3 (6.5%)
Class IV	2 (6.3%)	0

aSLE systemic lupus erythematosus, cSLE child systemic lupus erythematosus, WHO World Health Organization

**Table 4** Comparing autoantibody profile in aSLE and cSLE

Characteristics	aSLE ( <i>n</i> = 139) (%)	cSLE ( <i>n</i> = 86) (%)	<i>p</i> value
C3	90 (65)	77 (90)	<0.001
C4	90 (66)	69 (80)	0.019
ANA	136 (98)	84 (98)	1.000
Anti-dsDNA	117 (84)	81 (94)	0.045
Anti-Smith	51 (37)	18 (21)	0.025
Antiphospholipid antibody	51 (37)	46 (54)	0.026

aSLE systemic lupus erythematosus, cSLE child systemic lupus erythematosus, ANA antinuclear antibody

**Table 5** Treatment used in SLE cohorts

Drug name	Adults ( <i>n</i> = 139; 62%)	Children ( <i>n</i> = 86; 38%)	<i>p</i> value
NSAIDs	50 (36%)	43 (50%)	0.038
HCQ	128 (92%)	86 (100%)	0.008
Prednisolone	133 (96%)	86 (100%)	0.085
Methyl prednisolone	74 (53%)	51 (59%)	0.374
Cal vit D	115 (83%)	84 (98%)	<0.001
MMF	75 (54%)	60 (70%)	0.019
Cyclophosphamide	30 (22%)	44 (51%)	<0.001
Rituximab	29 (21%)	20 (23%)	0.673
Azathioprine	96 (69%)	59 (69%)	0.942
Methotrexate	21 (15%)	12 (14%)	0.812
IVIG	8 (5.8%)	7 (8.1%)	0.486

SLE systemic lupus erythematosus, NSAIDs non-steroidal anti-inflammatory drugs, HCQ hydroxychloroquine, MMF mycophenolate mofetil, IVIG intravenous immunoglobulin

a significant difference between the two groups. Cyclophosphamide and mycophenolate mofetil were more often administered to children with SLE. While exposure to other immunosuppressant drugs, including azathioprine, methotrexate, and biological therapies were similar between the two groups.

## Discussion

This study compares childhood and adult onset SLE patients from similar geographical background in Oman of Arab ethnicity. Comparing SLE phenotypes across different age groups revealed similarities but also important differences in demographic features, clinical manifestation, disease activity, serology as well as treatment in our cohort.

We found the female predominance of disease is less pronounced in both age spectrums, < 5 years and > 50 years with a female to male ratio of 3:1. The highest female predominance was between the ages of 18–25 years with a ratio of 11:1. This distinction between the two groups supports the role of hormonal factors, including sex hormones in

modifying the disease either in facilitating or suppressing symptoms [4, 25]. The age of onset of cSLE in our cohort was  $9.5 \pm 4.2$  years, which is consistent with age of onset of cSLE in other Arab ethnicity [22], yet younger than Caucasian ethnicity (11.5–13.1 years) [26, 27]. The difference could be partly due to the lack of consensus regarding the cut-off age for defining cSLE, which varies from 13 to 18 years in the literature. In most Arab health care facilities, the transitional age between children to adults is 13 years, however, in our studies, we considered 16 years as the upper limit to define cSLE.

Geographical distribution of aSLE in Omani regions follows the population density of Oman as most of the aSLE being from Al Batinah (*n* = 42, 30%) and Muscat (*n* = 36, 26%) region, which together constitute 50% of population of Oman. However, there was geographical clustering of cSLE with 38% of cSLE originating from Al Sharqiyah region, which constitutes 15% of the population of Oman. This could be explained by higher frequency of familial SLE in the subgroup of cSLE patients from Al Sharqiya region [18].

The results of our study confirms the results of earlier studies that demonstrate that SLICC-2012 classification criteria to be more sensitive than ACR-1997 criteria for the diagnosis of SLE [22]. At disease onset, 99% of our SLE cohort fulfilled the SLICC criteria; however, a significant difference was noted in fulfilling the ACR criteria (66% aSLE vs 80% cSLE). Petri et al., demonstrated that SLICC had higher sensitivity (97 vs 83%;  $p < 0.001$ ) but a lower specificity (84 vs 96%;  $p < 0.001$ ) than ACR criteria [22]. In another large study multicenter study from Spain and Portugal comparing the two classification criteria in 2044 SLE patients, it demonstrated that the differences in sensitivity is highest when disease duration is < 5 years (76 vs 90%;  $p < 0.001$ ), hence the SLICC criteria may allow patients with SLE to be diagnosed earlier in the disease course [28]. Similarly, the results of UK JSLE cohort study, which included 225 cSLE patients demonstrated that SLICC-2012 was more sensitive than ACR-1997 at diagnosis (93 vs 84%;  $p < 0.001$ ) and after follow-up (100 vs 92%;  $p < 0.001$ ) [29].

Comparing the clinical manifestation of SLE in our study, cSLE experienced a higher frequency of renal, neurological, hemolytic anemia, alopecia, arthritis and pulmonary

involvement than their adult counterparts, while aSLE experienced higher frequency of photosensitivity and lymphopenia. A recent systematic review and meta-analysis comparing 1560 cSLE with 8701 aSLE demonstrated similarities as well as differences to the results of our study [30]. Our results were similar to the meta-analysis with higher frequency of renal and neurological manifestation in cSLE and with a higher frequency of cutaneous manifestation such as photosensitivity in aSLE. However, aSLE had a higher likelihood of developing arthritis and pulmonary manifestation in the meta-analysis, which is different from our results. Furthermore, in contrast to meta-analysis, our cSLE cohort did not display a higher tendency of constitutional manifestation; they were equal to aSLE with no significant difference.

Over disease course, the difference in frequency of clinical manifestation did not change between aSLE and cSLE compared to disease onset except for neurological manifestation. It is interesting to note that during the disease course in aSLE, there was increased frequency of neurological manifestations to match cSLE at disease onset. Our result are similar to previous studies that demonstrate that the neuropsychiatric involvement with SLE is at least as common in children as it is in adults, with the former experiencing symptoms more commonly at diagnosis SLE (70 vs 28%) [31, 32].

Generally, renal involvement in SLE, detected by abnormal urinalysis and/or renal function, occurs more frequently in cSLE than in aSLE [11]. Although the frequency of renal involvement in our SLE cohort was higher in children than adults, however the overall frequency was lower than reported at disease onset occurring in 49% of cSLE and 20% of aSLE. Previous studies support a similar distribution of lupus nephritis (LN) histological classes in cSLE compared to aSLE, with diffuse proliferative LN (Class IV) occurring in 40–60% of the patients, focal proliferative LN (Class III) in 10–20%, and membranous LN (Class V) in 3–18% [5, 33, 34]. Similarly, in our cohort of patients with lupus nephritis, Class IV was the most frequent glomerulonephritis (50% aSLE vs 61.7% cSLE) followed by Class III (21.9% aSLE vs 14.9% cSLE) as shown in Table 3. When investigating the clinical characteristics of LN in patients before and after puberty, Torr et al. [35] concluded that the post-pubertal form of SLE is similar to adult one, while pre-pubertal form of SLE had different histological LN profile. Although, the majority of our cohort had disease onset in the pre-pubertal period before the age of 12 years ( $n = 65$ ; 76%), they had similar distribution of LN as aSLE.

Similar to recent meta-analysis, cSLE patients in our cohort had increased anti-dsDNA antibodies and anticardiolipin antibody compared to aSLE [12]. Infectious processes may result in transient and nonpathogenic anticardiolipin antibody positivity, which occurs more commonly in children. In general, antiphospholipid antibody-related thrombosis is rare in

children, and prothrombotic or vasculopathic factors are more frequent in adults. A recent review demonstrated an association of positive RF with Sjogren's syndrome and inflammatory arthritis [36]. Interestingly, our cohort showed that cSLE were more commonly affected with arthritis, although there was no difference in occurrence of RF between the two groups [12]. Additionally, our cohort showed increased anti-Smith antibody in aSLE. The frequency of anti-Smith antibody in our aSLE cohort (37%) was comparable to other Asian countries (30%), higher than Caucasians (26%) and Latin Americans (11%) but much lower than African Americans (49%) [36–39]. However, our results were similar to recent meta-analysis and reviews as there was no predilection of other autoantibodies to either age groups (ANA, anti-Smith, anti-RNP, anti-U1RNP, anti-Ro and anti-La, antiphospholipid, lupus anticoagulant, complements, dsDNA, and Coomb's test).

This study is not without limitations. As this was a retrospective study, missing data from chart review may affect the overall results. While cSLE share many similarities with aSLE, awareness of its differences is crucial, namely higher disease activity and a more aggressive clinical course. The result of our study on SLE patients of Arab ethnicity from Oman confirms the results of other studies that emphasize the importance of ethnicity on disease burden [40]. The current treatment strategies in cSLE are hampered by lack of robust evidence-based studies. To establish optimum care and improve long-term outcomes, we need streamline treatments and improved transition programs from pediatric to adult care.

## Conclusion

In conclusion, the results of our study showed similarities and differences between aSLE and cSLE. Our results were similar to other studies reporting cSLE as having a more severe disease onset with higher major organ involvement and having a more severe disease course requiring more aggressive treatment than aSLE. The overall treatment approach in SLE is similar in children to adults. However, as cSLE is a more aggressive disease, the requirements of high doses of corticosteroids and immunosuppressive treatment may be greater than adults to control disease. In our cohort, cSLE received more mycophenolate mofetil and cyclophosphamide therapy than aSLE, two drugs commonly used to treat severe lupus manifestations including lupus nephritis and central nervous system involvement.

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