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ORIGINAL ARTICLE

Clinical presentation of children with lupus nephritis from a low- and middle-income country (LMIC): an initial report from the Indian pSLE Nephritis Registry

Sanjukta Poddar[1] · Deblina Dasgupta[1] · Subal Pradhan[3] · Sangeetha Perungo[4] · Mahesh Janarthanan[5] · Kinnari Vala[6] · Priya Pais[7] · Susan Uthup[8] · Jyoti Singhal[9] · Suparna Guha[10] · Sumantra Raut[11] · Shakil Akhtar[1] · Jigna Bathia[2] · Suma Balan[12] · Priyankar Pal[2] · Rajiv Sinha[1] · On behalf, of the Indian pSLE Nephritis consortium

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Abstract

Introduction Limited prospective data exist on pediatric LN (pLN) from low- and middle-income countries (LMIC), where ethnicity, socioeconomic factors, and healthcare access are likely to differ from high-income countries.

Methods The Indian Pediatric Lupus Nephritis registry has been running since 2020 across multiple centers in India. Children (≤ 18 years) diagnosed with lupus (as per 2012 SLICC criteria), presenting with nephritis, and confirmed by kidney biopsy are being prospectively enrolled. Clinical data, laboratory investigations, kidney biopsy results, and treatment responses have been documented prospectively. The current report documents their initial presentation.

Results A total of 154 children (75% female, median age 12 years—IQR 10–14 years) with biopsy-proven LN were enrolled by July 2024. Nearly two-thirds had LN at SLE diagnosis, and the rest developed within a maximum of 5 years of initial presentation. Common manifestations at presentation included edema (75%), hypertension (54%), and proteinuria (98%), of which 68% presented with nephrotic-range proteinuria. Acute kidney injury (AKI) was observed in 43%, with 20% in stage 3. Ninety-four percent of our cohort had low complements (C3, C4, or both), and 96% were ANA-positive. Class IV LN was the most common (45%) histopathological type and had significantly lower estimated glomerular filtration rate in comparison to Class V LN.

Conclusion Kidneys are often involved in the initial presentation of childhood lupus, and the majority have proliferative nephropathy leading to AKI, hypertension, and significant proteinuria. Children enrolled in the registry are under active follow-up to assess the renal responses which will help optimize the management of pLN in LMICs.

Key Points

- *It is a well-known fact that kidney involvement is more common in pediatric lupus and is among one of the most important long-term prognostic factors.*
- *There is scarcity of data on pediatric lupus nephritis (pLN) particularly from low- and middle-income countries (LMIC), and even among them, the majority of the studies are retrospective and limited by a small cohort size.*
- *Through this prospective registry from a LMIC, we demonstrated that 2/3rd of children with lupus have kidney involvement at presentation and almost all (90%) develop LN within 2 years of the diagnosis of lupus.*
- *Acute kidney injury (AKI) is known to increase mortality/morbidity risks independently. Many of the previous studies have under-reported AKI in pLN, probably because the data was collected retrospectively. On the other hand, we found AKI to be very common and to be present in about half of the cases at presentation.*

Keywords Acute kidney injury (AKI) · Anti-nuclear antibody (ANA) · Edema · Estimated glomerular filtration rate (eGFR) · Hematuria · Histopathology · Hypertension · Kidney biopsy · Kidney replacement therapy (KRT) · Lupus nephritis (LN) · Nephrotic syndrome · Proteinuria · PSLE Nephritis Registry · Systemic lupus erythematosus (SLE)

Extended author information available on the last page of the article

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Introduction

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease with usual onset between 15 and 44 years of age and female predominance more prominent in the adolescent age group [1–3]. Lupus nephritis (LN), a major determinant of morbidity and mortality in patients with SLE, is more common among children (50–82%) compared to adult (20–40%) with greater disease severity and earlier accrual of disease damage than in adults [4]. Although the 5-year survival rate of pediatric LN (pLN) has improved markedly, the mortality rate seen in pLN remains around 19 times higher than that in healthy children [4]. To further complicate the management of this challenging disease, there is a lack of robust data on pLN from low- and middle-income countries (LMICs) [3, 5, 6]. A recent systematic review found mostly retrospective data from LMIC including South Asia with the exception of a single-center experience from Eastern India limited by its small sample size of 23 [7].

The Indian pSLE nephritis registry aims to address this lacuna in the medical literature. It is a collaborative effort by pediatric nephrologists and rheumatologists from centers across India aimed at studying the epidemiology, clinicpathologic characteristics, treatment patterns, and kidney outcomes of Indian children with LN, and to assess the factors affecting these outcomes. The current article describes the initial presentation of children with LN at the time of enrollment in the registry.

Materials and methods

Indian pSLE Nephritis Registry was initiated after a meeting in Kolkata in June 2018 attended by a group of pediatric nephrologists and rheumatologists having a special interest in pLN from across India. Institute of Child Health, Kolkata, has been serving as the nodal center. Institutes that are currently involved include the following: (a) Amrita Institute of Medical Science (Kochi, Kerala), (b) Government Medical College (Thiruvananthapuram, Kerala), (c) Institute of Child Health (Kolkata, West Bengal), (d) Institute of Kidney Disease and Research Centre (Ahmedabad, Gujarat), (e) King Edward Memorial Hospital (Pune, Maharashtra), (f) North Bengal Medical College and Hospital (Darjeeling, West Bengal), (g) Sardar Vallabhbhai Patel Post Graduate Institute of Pediatrics (Cuttack, Odisha), (h) St John's Medical College (Bengaluru, Karnataka), and (i) Sri Ramachandra Institute of Higher Education and Research (Chennai, Tamil Nadu).

Initially during the June 2018 meeting, the basic protocol was finalized (as mentioned below) and active recruitment of patients for the registry started in August 2020.

Inclusion criteria

Children ≤ 18 years were included if they fulfilled ALL of the following criteria:

(a) Classified as SLE based on 2012 SLICC criteria [8]
(b) Presence of features of nephritis, which include any of the following:
(c) Urine protein to creatinine ratio (PCR) ≥ 0.5
(d) Active urinary sediment defined by >5 red blood cells (RBC)/high power field (hpf) OR> 5 white blood cells (WBC)/hpf in the absence of infection, OR cellular cast of RBCs/WBCs
(e) Kidney biopsy consistent with diagnosis of LN

If a kidney biopsy was not possible because of the clinical conditions at the time of presentation, children were still included provided they fulfilled the other criteria and biopsy was performed once the condition was favorable.

Exclusion criteria

(1) Patients with drug-induced lupus, mixed connective tissue disorders
(2) Parents not willing to participate in the registry

After obtaining informed consent, baseline clinical details were noted and relevant laboratory investigations were sent. Investigations included complete blood count, kidney function tests, urine routine and microscopy, 24-h urine protein and creatinine and/or early morning urine spot sample protein and creatinine ratio, liver function tests, serological parameters like serum complement levels, anti-nuclear factor antibody (ANA) by indirect immunofluorescence, anti-double-stranded DNA antibodies (anti-dsDNA), anti-phospholipid antibodies (APLA), and direct coomb's test (DCT). We calculated estimated glomerular filtration (eGFR) as per the modified Schwartz formula 2009 [9] and diagnosed/classified AKI as per the KDIGO criteria [10]. Kidney biopsy was done following American College of Rheumatology (ACR) guidelines, and the biopsy was classified according to the ISN/RPS classification system (2003) and National Institute of Health (NIH) classification of activity and chronicity indices [11].

Therapeutic interventions were determined at the discretion of the treating physician's in accordance with international guidelines [5, 12, 13]. Treatment details along with the need for kidney replacement therapy (KRT), if any were noted.

After recruitment, clinical and laboratory details were recorded monthly in the induction phase and thereafter

every 3 months during the maintenance phase. Initially, kidney responses were defined by EULAR 2012 criteria and later updated according to KDIGO 2021 Lupus Nephritis guidelines. These response criteria are based on core kidney parameters of proteinuria, serum creatinine, and urine microscopy findings. Responses grouped into:

(a) Complete response: The return of serum creatinine to ±10–15% of the previous baseline, plus a decline in the urinary protein-to-creatinine ratio (uPCR) to <0.5 mg/mg
(b) Partial response: Stabilization of serum creatinine within ± 10–15% of the baseline, along with a reduction in urinary PCR by at least 50% and to below 3 mg/mg
(c) No response: Those who did not fulfill the aforementioned criteria

The current draft focuses on the initial presentation with data analyzed until July 2024, whereas subsequent publications will focus on treatment modalities and outcomes in this cohort of children.

The registry obtained ethical approval at the nodal center, i.e., ICH, Kolkata (IEC/243/2021), as well as from the individual institutional ethics committees of participating centers.

Statistical analysis

For data analysis, we used SPSS version 25. Continuous variables are expressed as mean (± SD) or median with interquartile range (IQR) while categorical data as percentages. Parametric data were analyzed using an independent sample *t*-test and non-parametric data using the Mann-Whitney *U* test. The chi-square test was used to find significant statistical differences between proportions. A p-value<0.05 was considered statistically significant for all tests.

Results

Till the 31st July 2024 over a period of 4 years, 154 children (115 females, 75%) with newly diagnosed biopsy-proven LN have been enrolled prospectively in the registry from 9 different centers across India.

At the time of recruitment, the median age of our cohort was 12 years (IQR 10–14 years) with a median height SDS of 1.15 (IQR −2.27 to 1.6) and weight SDS of 0.825 (IQR −1.55 to 0.3).

Only 9 children (6%) were aged ≤7 years with the youngest being 5.5 years old. No difference was seen in the severity of kidney involvement in children younger than 7 years at presentation as compared to those who presented beyond 7 years of age.

There was a female preponderance even among the younger age group. Among 25 children who were ≤ 10 years of age, 85% were female.

Nearly two-thirds (61%; $n = 93$) of the children in our cohort had LN at the time of diagnosis of SLE, whereas the rest developed LN subsequently, most within 5 years of the initial presentation.

Kidney manifestation

Table 1 shows the various kidney manifestations at the presentation of which edema (75%, $n = 113$) was the most common. Interestingly, 15 children (10%) did not have any overt manifestation of LN and were only detected by abnormal urinary parameters and/or elevated serum creatinine (3 cases) on routine screening. Seven (47%) of these children had Class IV LN, 5 (33%) had Class III LN, and 3 (20%) had Class II LN. Overall, on urine analysis, proteinuria with or without active urinary sediment and/or hematuria was the most consistent finding ($n = 151$, i.e., 98%). Among the children with proteinuria, 68% ($n = 103$) had nephrotic range proteinuria. The majority of children having AKI were stage 1 ($n = 41$, 62%), while stage 3 AKI was reported in 20%. Kidney replacement therapy (hemodialysis) was necessary at onset in 3 children (2%).

Extra-renal manifestations

Among extra-renal manifestations (Fig. 1), mucocutaneous involvement was the most common (59%, $n = 91$). It included photosensitive rash, oral mucosal ulcer, and

Table 1 Kidney manifestation

Kidney manifestations ($N = 154$)	% (n)
Edema	75 (113)
Oliguria	36 (55)
Hypertension	54 (81)
Gross hematuria	17 (26)
Proteinuria	98 (151)
Nephrotic range proteinuria	68 (103)
Hematuria	67(100)
Pyuria	55 (82)
Cast	14 (21)
Acute kidney injury (AKI)	43 (66)
Estimated glomerular filtration rate (eGFR)	Median 92.9 (IQR 62.1–119.7) ml/min/1.73 m^2

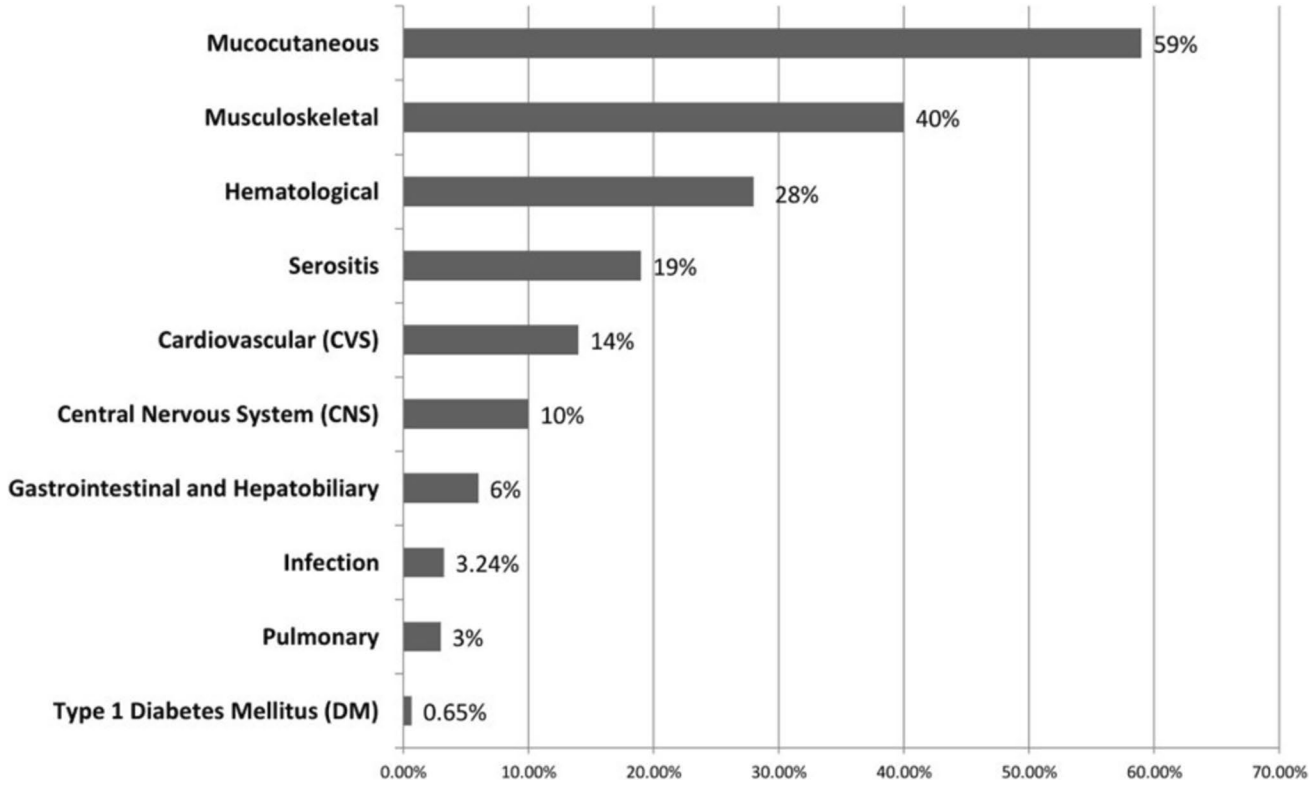

Fig. 1 Extra-renal manifestations

non-scarring alopecia. Hematological manifestation was next of which anemia was the most common ($n = 30$, 19.5%) finding. Though DCT was positive in 53% ($n = 73$), only 9 (6%) children had confirmatory features of autoimmune hemolytic anemia. Neurological symptoms, mostly in the form of convulsion and altered sensorium, were seen in 15 children (10%). Significant infection at the time of diagnosis of LN was seen in only 3% ($n = 5$) of our cohort (2 children had urosepsis, 1 child had sepsis, and 2 had pneumonia). There were two deaths: one child succumbed to severe diffuse alveolar hemorrhage at onset while another child had multi-organ failure and died secondary to macrophage activation syndrome.

Table 2 Serological parameters

Serological markers ($N = 154$)	% (n)
Low complement 3 (C3) and complement 4 (C4)	56 (87)
Low C3 and normal C4	21 (33)
Normal C3 and C4	8 (10)
Normal C3 and low C4	1 (2)
C3 low, C4 missing	14 (22)
Anti-nuclear antibody (ANA) positivity	94 (145)
Anti-double stranded antibody (AntidsDNA) positivity	81 (119)
Anti-phospholipid antibody (APLA) positivity	17(21/117)
Direct Coombs test (DCT) positivity	53 (73)

Immunological parameters

Table 2 shows the serological parameters at presentation with the majority having hypocomplementemia ($n = 144$, 94%). Interestingly, 10 children (6%) had normal complement levels. No difference was found between those with low or normal complements at presentation with the exception of the absence of gross hematuria among those with normal complement levels. ANA was positive in the majority of children (96%, $n = 149$) by either the ELISA technique (28%; $n = 43$) or by indirect immunofluorescence (72%, $n = 111$). However, 5 children were found to be ANA-negative even on repeated testing despite having a kidney biopsy consistent with the diagnosis of LN (3 cases being Class IV+V, 1 Class V, and 1 Class IV). Clinical presentations of children having ANA-negative LN were no different from those having ANA-positive. APLA-positive (17%) children in our cohort did not have any extra-renal manifestations, while two had thrombotic microangiopathy on renal biopsy.

Histopathology features

Figs. 2 and 3 describe the indications for kidney biopsies and the biopsy results respectively. The most common indication for doing a kidney biopsy was proteinuria (either nephrotic or sub-nephrotic). None got biopsy for isolated hematuria.

Class IV (45%, $n = 66$) was the most common histopathological classification on kidney biopsy (Fig. 3). NIH activity and chronicity indices were noted in 134 patients with median values of 6 (IQR 2–9) and 0 (IQR 0–1) respectively.

Fig. 4 shows the clinical presentations across the various classes of LN. Clinical features among children with Class V LN were similar to proliferative LN (class III/IV/III+V/IV+V) except for edema, which was significantly more common in the former group of children, present in 100%. Among laboratory parameters, active urine sediments were significantly less frequent in the Class V LN (p-value = 0.02), whereas nephrotic range proteinuria was present in all cases of Class V LN. Median eGFR value was also significantly better in the Class V LN patients (median eGFR 90.3 ml/min/1.73 m^2) than the proliferative LN cohort (median 84.9 ml/min/1.73 m^2) (p-value= 0.02). Interestingly, there were no cases of AKI or gross hematuria among any of the exclusive Class V LN.

There were only 13 cases of Class II. All of these children had some amount of proteinuria. Among these, 5 children having nephrotic-range proteinuria were found to have lupus podocytopathy on electron microscope. Three of these cases also had AKI at presentation (one child had stage 1 and another two had stage 2 AKI.

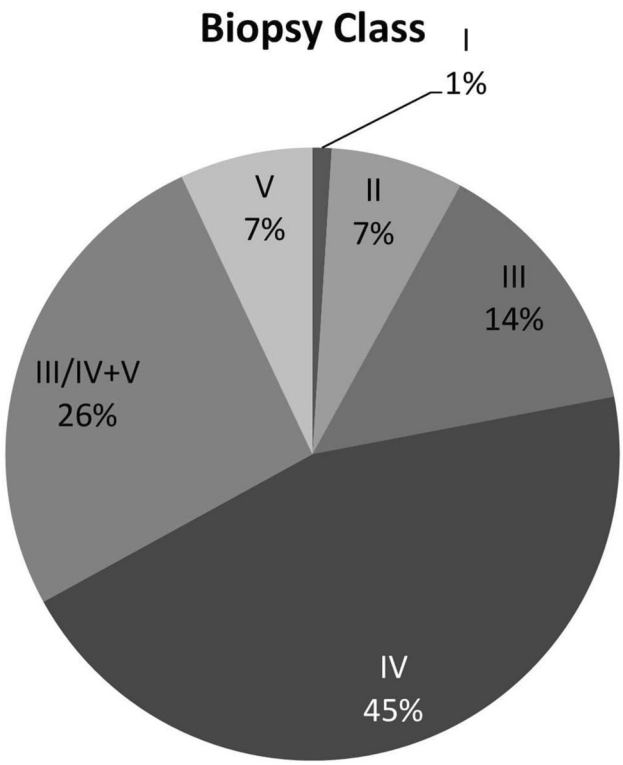

Fig. 3 Renal histopathology classification

Discussion

To the best of our knowledge, this registry is the very first initiative for a longitudinal database of presentation, management, and outcomes of pLN from an LMIC. While some

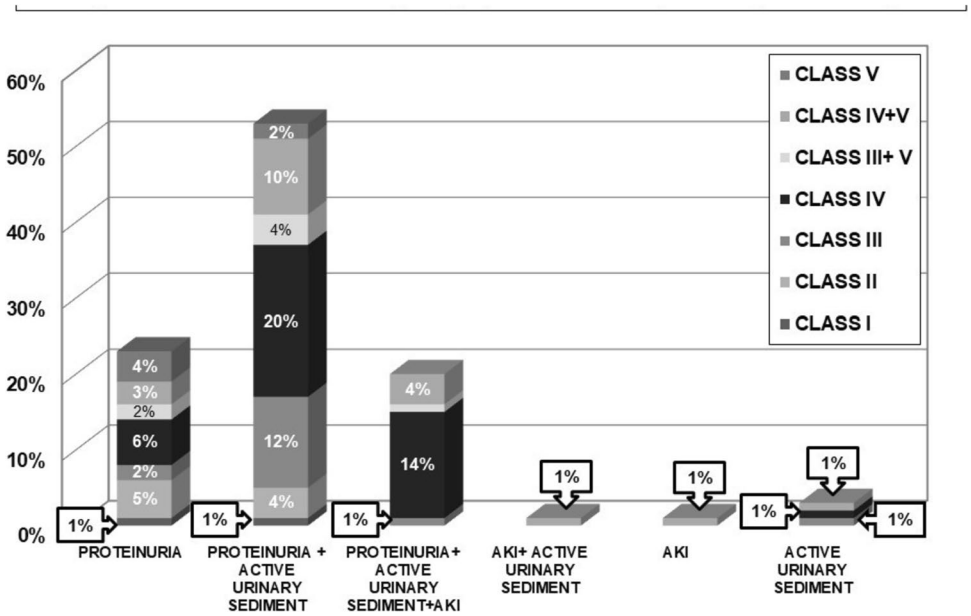

Fig. 2 Indications for kidney biopsy and the histopathological findings

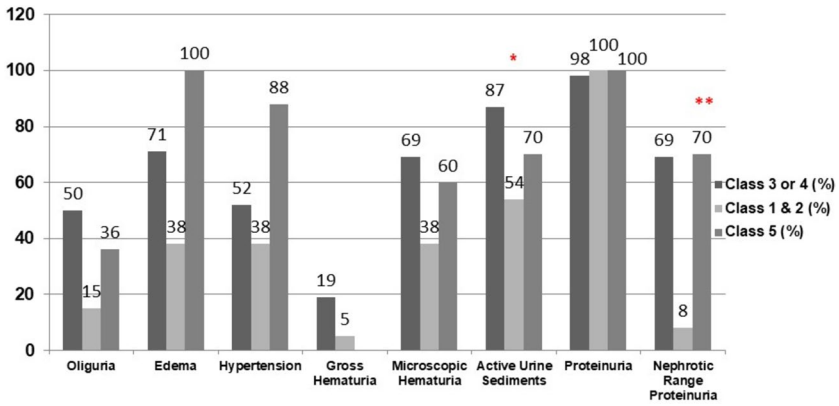

Fig. 4 Clinical presentation across various histopathologic classifications

of our findings were similar to the previous retrospective cohort [4, 5, 8–11, 14–16], this study did help to get a clearer view of many severe manifestations like AKI and nephrotic range proteinuria, which were often under-reported in retrospective studies. It was important to note that the majority of pLN (nearly two-thirds) registered were diagnosed at the onset of SLE. This underscores the importance of checking kidney parameters at the initial diagnosis of lupus and thereafter monitoring kidney parameters regularly. Similarly, the retrospective study by Samanta et al. [17] from Eastern India showed that 82% developed nephritis at the onset of SLE whereas the Childhood Arthritis and Rheumatology Research Alliance (CARRA) Registry showed that 73% developed nephritis within the same calendar year of SLE onset and 93% developed LN within 2 years [18]. The German Registry had similar findings with 75% having features of nephritis at diagnosis of SLE [19].

Similar to other studies [4, 5, 8, 11, 14, 15], edema and hypertension (54%) seem to be the most common clinical presentations. When compared with the German Registry (82%), hypertension seems to be slightly less common among our cohort. Among our children, 32% had nephrotic range proteinuria, while in the German cohort, 59% had nephrotic range proteinuria and 33% had nephrotic syndrome at presentation [19]. Hematuria along with proteinuria was present in 67% ($n = 103$) of our cohort, of which 50% had microscopic hematuria. This is unlike the cohort from the Western world, where microscopic hematuria seems to be more common (93%). Similarly, Das et al. [14] from Northeast India reported a lower incidence of gross hematuria, but Samanta et al. [17] reported an overall incidence of proteinuria with hematuria similar to ours (30%). Compared to prospective studies, there is always a risk of under-reporting of clinical findings such as gross hematuria in retrospective studies because of recall bias. Notable in our cohort was the finding of 10% of children having no symptoms of kidney involvement yet having proteinuria and/or active urine sediments and/or AKI. SLE was diagnosed from extra-renal manifestations, while nephritis was detected on renal screening and later confirmed by kidney biopsy. This emphasizes the importance of actively screening for kidney involvement in pSLE.

Another notable point in our prospective cohort was the documentation and staging of AKI at presentation, which was often not included in most of the previous Indian cohort [14, 17]. In our LN cohort, 43% of cases had some degree of AKI, and the median eGFR was 92.9 ml/min/1.73 m^2 (IQR 62.1–119.7 ml/min/1.73 m^2). Our AKI incidence was a bit lower than the German Registry, which reported AKI in 61% at presentation with a median eGFR of 75 ml/min/1.73 m^2 (IQR 49–107 ml/min/1.73 m^2), with 3% lying below 15 ml/min/1.73 m^2 [19].

Regarding extra-renal manifestations, mucocutaneous involvement, though the most common, was found in 59% while most previous cohorts showed higher (70–80%) mucocutaneous involvement at presentation [19, 20]. The fact that over a third of our cohort did not have any mucocutaneous involvement at presentation needs to be highlighted. Mucocutaneous involvement including rashes/ulcers is often the initial signs/symptoms that lead clinicians to suspect lupus, but our findings suggest that among LN, reliance on this finding may result in late diagnosis. The study by Samanta et al. [17] from India reported some form of neurological involvement in 28%, whereas in our cohort, only 10% had neurological manifestations. The German Registry also reported 18% CNS involvement at presentation [19]. It needs to be noted that as per the UK-jSLE registry (including 422 patients of ≤ 16 years of age from different ethnic backgrounds like White Caucasian, Black African as well as Asian), the Asian population seems to have more mucocutaneous involvement, whereas the Caucasians mostly had musculoskeletal involvement at presentation. Hematological manifestation was significantly higher in the African population in comparison to Asians and Caucasians [20].

According to current American College of Rheumatology guidelines, ANA positivity is a mandatory criterion to classify the disease as SLE. However, in our cohort, despite the classical presentation of SLE, 5 children were

persistently ANA-negative. A probable reason can be lab variability which is an inherent challenge in any multicenter registry [24]. Anti-dsDNA was positive in 81%. Previous Indian studies like Das et al. reported ANA positivity in only 62% and anti-dsDNA in 64% of cases [14]. In the UK-jSLE cohort, all were ANA-positive and 70% were anti-dsDNA-positive [20].

Similar to previous reports, Class IV LN was the most common histopathological category [4, 7, 14, 16–19, 21–23] with significantly lower eGFR in comparison to Class V LN.

Conclusion

Our initial analysis of the longitudinal data collected through the pan-India multicenter registry highlights the need for routine kidney screening for children with SLE right from their time of diagnosis. Apart from the usual renal manifestations of edema, proteinuria, and hypertension, a substantial proportion also had AKI wherein an early diagnosis and appropriate treatment could prevent progression to chronic kidney disease. Notably, 10% of our cohort had sub-clinical nephritis, again highlighting the need for routine evaluation of renal parameters in pediatric lupus. We intend to follow up these children for at least 2 years, with the goal of informing improved management strategies for children with LN in our low- and middle-income setting.

Abbreviations ANA: Anti-nuclear antibody; AKI: Acute kidney injury; cSLE: Childhood onset systemic lupus erythematosus; CR: Complete response; DAH: Diffuse alveolar hemorrhage; DCT: Direct Coomb's test; eGFR: Estimated glomerular filtration rate; ESKD: End stage kidney disease; IQR: Interquartile range; NR: No response; pSLE: Pediatric systemic lupus erythematosus; PR: Partial response; LIF: Light immunofluorescence; LMIC: Low- and middle-income countries; LN: Lupus nephritis; SCr: Serum creatinine; SLE: Systemic lupus erythematosus; UPCR: Urine protein creatinine ratio

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Author contribution The conception or design of the work: Prof. Rajiv Sinha, Prof. Priyankar Pal. Acquisition, analysis, or interpretation of data for the work: acquisition done by all authors. Analysis and interpretation were done by Dr. Sanjukta Poddar, Dr. Deblina Dasgupta, and Dr. Rajiv Sinha. Drafting the work or revising it critically for important intellectual content: all authors. Final approval of the version to be published: all authors. Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved: all authors.

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Data availability All data is available through Institute of Child Health Research academic council email: ichrac23@gmail.com.

Declarations

Ethics approval The registry obtained ethical approval at the nodal center, i.e., ICH, Kolkata (IEC/243/2021), as well as through the individual ethics committees of all the participating centers.

Consent for publication Taken from all patients and authors.

Disclosures None.

References

1. Chatterjee R, Aggarwal A (2023) Challenges in the diagnosis and management of SLE in India. Clin Immunol Commun 4:65–9
2. Rovin BH, Ayoub IM, Chan TM, Liu ZH, Mejía-Vilet JM, Floege J (2024) Kdigo 2024 Clinical Practice Guideline for the management of Lupus Nephritis. Kidney Int 105(1):S1-69
3. Hiraki LT, Feldman CH, Liu J, Alarcón GS, Fischer MA, Winkelmayer WC et al (2012) Prevalence incidence, and demographics of systemic lupus erythematosus and lupus nephritis from 2000 to 2004 among children in the US medicaid beneficiary population. Arthritis Rheum. 64(8):2669–76
4. Pinheiro SVB, Dias RF, Fabiano RCG, Araujo S de A, Silva ACS e. (2019) Pediatric lupus nephritis. Braz J Nephrol. 41(2):252–65
5. Khandelwal P, Govindarajan S, Bagga A (2023) Management and outcomes in children with lupus nephritis in the developing countries. Pediatric Nephrology. 38(4):987–1000
6. Alarcon GS, McGwin Jr, G, Bastian HM, Roseman J, Lisse J, Fessler BJ, et al. 2001 Systemic lupus erythematosus in three ethnic groups. VIII. Predictors of early mortality in the LUMINA cohort. Arthritis Rheum. 45(2):191–202
7. Nandi M, Mondal R (2012) Renal involvement in childhood lupus: a study from Kolkata India. Saudi J Kidney Dis Transpl 23(4):871
8. Petri M, Orbai AM, Alarcón GS, Gordon C, Merrill JT, Fortin PR, Bruce IN, Isenberg D, Wallace DJ, Nived O, Sturfelt G, Ramsey-Goldman R, Bae SC, Hanly JG, Sánchez-Guerrero J, Clarke A, Aranow C, Manzi S, Urowitz M, Gladman D, Kalunian K, Costner M, Werth VP, Zoma A, Bernatsky S, Ruiz-Irastorza G, Khamashta MA, Jacobsen S, Buyon JP, Maddison P, Dooley MA, van Vollenhoven RF, Ginzler E, Stoll T, Peschken C, Jorizzo JL, Callen JP, Lim SS, Fessler BJ, Inanc M, Kamen DL, Rahman A, Steinsson K, Franks AG Jr, Sigler L, Hameed S, Fang H, Pham N, Brey R, Weisman MH, McGwin G Jr, Magder LS (2012) Derivation and validation of the Systemic Lupus International Collaborating Clinics classification criteria for systemic lupus erythematosus. Arthritis Rheum. 64(8):2677–86. https://doi.org/10.1002/art.34473
9. Muhari-Stark E, Burckart GJ (2018) Glomerular filtration rate estimation formulas for pediatric and neonatal use. J Pediatr Pharmacol Ther 23(6):424–31
10. Khwaja A (2012) KDIGO clinical practice guidelines for acute kidney injury. Nephron Clin Pract 120(4):c179-84. https://doi.org/10.1159/000339789
11. Weening JJ, D'Agati VD, Schwartz MM, Seshan SV, Alpers CE, Appel GB et al (2004) The classification of glomerulonephritis in systemic lupus erythematosus revisited. J Am Soc Nephrol 15(2):241–50
12. Fanouriakis A, Kostopoulou M, Cheema K, Anders HJ, Aringer M, Bajema I et al (2020) 2019 Update of the Joint European League Against Rheumatism and European Renal Association-European Dialysis and Transplant Association (EULAR/ERA–EDTA) recommendations for the management of lupus nephritis. Ann Rheum Dis 79(6):713–23

13. Hahn BH, McMahon MA, Wilkinson A, Wallace WD, Daikh DI, FitzGerald JD et al (2012) American College of Rheumatology guidelines for screening, treatment, and management of lupus nephritis. Arthritis Care Res (Hoboken). 64(6):797–808
14. Das J, Kalita P, Dey B, Raphael V, Mishra J, Khonglah Y et al (2023) Clinicopathological immunological and laboratory parameters of childhood lupus nephritis: a study from Northeast India. J Lab Physicians 15(03):361–4
15. Biswas D, Dasgupta D, Pal P, Sinha R (2023) Presentation and outcome of pediatric lupus nephritis from a large single centre contemporary cohort in Eastern India. Lupus 32(12):1440–6
16. Al-Mayouf S, AlAmeer A, Alfattani A, Alsonbul A (2017) Outcome of childhood lupus nephritis in Saudi children. Saudi J Kidney Dis Transpl 28(5):1015
17. Samanta M, Nandi M, Mondal R, Hazra A, Sarkar S, Sabui T et al (2017) Childhood lupus nephritis: 12 years of experience from a developing country's perspective. Eur J Rheumatol 4(3):178–83
18. Vazzana KM, Daga A, Goilav B, Ogbu EA, Okamura DM, Park C et al (2021) Principles of pediatric lupus nephritis in a prospective contemporary multi-center cohort. Lupus 30(10):1660–70
19. Suhlrie A, Hennies I, Gellermann J, Büscher A, Hoyer P, Waldegger S et al (2020) Twelve-month outcome in juvenile proliferative lupus nephritis: results of the German registry study. Pediatric Nephrology 35(7):1235–46
20. Massias JS, Smith EM, Al-Abadi E, Armon K, Bailey K, Ciurtin C et al (2021) Clinical and laboratory phenotypes in juvenile-onset systemic lupus erythematosus across ethnicities in the UK. Lupus 30(4):597–607
21. Wei Q, Wang W, Dong Y, Zhong L, Song H (2021) Five years follow-up of juvenile lupus nephritis: a single-center retrospective cohort study. Ann Palliat Med 10(7):7351–9
22. Ahmadzadeh A, Derakhshan A, Ahmadzadeh A (2008) A clinicopathological study of lupus nephritis in children. Saudi J Kidney Dis Transpl 19(5):756–60
23. Wenderfer SE, Eldin KW (2019) Lupus nephritis. Pediatr Clin North Am. 66(1):87–99
24. Khalifah MJ, Almansouri O, Aga SS, Aljefri AA, Almalki A, Alhmdan N, Al-Mazain W, Alsalmi K, Alamri A (2022) Comparison of indirect immunofluorescence and enzyme immunoassay for the detection of antinuclear antibodies. Cureus. 14(11):e31049. https://doi.org/10.7759/cureus.31049.PMID:36475172;PMCID:PMC9719102

Authors and Affiliations

Sanjukta Poddar[1] · Deblina Dasgupta[1] · Subal Pradhan[3] · Sangeetha Perungo[4] · Mahesh Janarthanan[5] · Kinnari Vala[6] · Priya Pais[7] · Susan Uthup[8] · Jyoti Singhal[9] · Suparna Guha[10] · Sumantra Raut[11] · Shakil Akhtar[1] · Jigna Bathia[2] · Suma Balan[12] · Priyankar Pal[2] · Rajiv Sinha[1] · On behalf, of the Indian pSLE Nephritis consortium

✉ Rajiv Sinha
rajivsinha_in@yahoo.com

1 Division of Pediatric Nephrology, Institute of Child Health, Kolkata, India

2 Pediatric Rheumatology Unit, Institute of Child Health, Kolkata, India

3 Division of Pediatric Nephrology, Sardar Vallabhbhai Patel Post Graduate Institute of Pediatrics, Cuttack, India

4 Pediatric Nephrology, Sri Ramachandra Institute of Higher Education and Research, Chennai, India

5 Department of Clinical Immunology & Rheumatology, Sri Ramachandra Institute of Higher Education and Research, Chennai, India

6 Department of Pediatric Nephrology, Smt. GR Doshi and Smt. KM Mehta Institute of Kidney Diseases and Research Center, Gujarat University of Transplantation Sciences, Ahmedabad, India

7 Department of Pediatric Nephrology, St John's Medical College, St John's National Academy of Health Sciences, Bengaluru, India

8 Department of Pediatric Nephrology, Government Medical College, Thiruvananthapuram, India

9 Pediatric Nephrology Service, Renal Unit, King Edward Memorial Hospital, Pune, India

10 Department of Pediatrics, Vivekananda Institute of Medical Sciences, Kolkata, India

11 Department of Nephrology, North Bengal Medical College and Hospital, Siliguri, India

12 Department of Rheumatology and Clinical Immunology, Amrita Institute of Medical Sciences, Cochin, India