Original Article

To Estimate the Prevalence and Risk Factors for Nephropathy in Patients with Steady-State Sickle Cell Anemia at A Tertiary Care Centre

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Abstract

Background: Sickle Cell Disease is a common inherited disorder associated with progressive multiorgan complications, including renal involvement. Sickle cell nephropathy (SCN) often begins in childhood and remains subclinical in early stages, making timely detection essential. The present study was undertaken to estimate the prevalence of nephropathy and identify associated risk factors among children with sickle cell anemia in steady state. **Material and Methods:** This hospital-based cross-sectional observational study was conducted over one year in the Department of Paediatrics at Chacha Nehru Bal Chikitsalaya, Indore. A total of 63 children aged 2–14 years with confirmed sickle cell anemia in steady state were included. Clinical data, laboratory investigations (urine analysis, renal function tests), and ultrasonographic findings were recorded. Statistical analysis was performed using SPSS, with $p < 0.05$ considered significant. **Results:** The prevalence of nephropathy was 20.6% (95% CI: 10.6%–30.6%). Among affected children, 6.3% had isolated hematuria, 6.3% had isolated proteinuria, and 7.9% had both. Renal function tests (serum urea, creatinine, eGFR) were normal in all participants, indicating early-stage disease. Increasing age was significantly associated with nephropathy ($p = 0.009$), with combined urinary abnormalities showing strong association ($p = 0.020$). Markers of disease severity, including hospitalisations, blood transfusions, and vaso-occlusive crisis episodes, were significantly higher in affected children ($p < 0.001$). Poor follow-up ($p = 0.008$) and poor drug compliance ($p = 0.031$ to <0.001) were important modifiable risk factors. **Conclusion:** Nephropathy is a common early complication in pediatric sickle cell anemia, strongly associated with age, disease severity, and treatment adherence. Routine urine screening is essential, as renal function tests may remain normal in early stages.

Keywords: Sickle Cell Anemia; Nephropathy; Hematuria; Proteinuria; Pediatric.

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Introduction

Sickle Cell Disease (SCD) is an inherited hemoglobin disorder caused by a point mutation in the β-globin gene, leading to the production of abnormal hemoglobin S (HbS). Under conditions such as hypoxia, acidosis, and dehydration, red blood cells (RBCs) undergo sickling, becoming rigid and prone to hemolysis. These altered cells contribute to recurrent vaso-occlusion, chronic hemolytic anemia, and progressive multiorgan damage. SCD is among the most common monogenic disorders worldwide, with a high prevalence in sub-Saharan Africa, the Middle East, and several regions of India.[1]

The kidneys are particularly vulnerable in SCD due to the unique microenvironment of the renal medulla, which is naturally hypoxic, acidic, and hyperosmolar. These conditions favor RBC sickling, resulting in microvascular obstruction, ischemia, and repeated injury to renal tissues. Over time, this leads to a spectrum of renal abnormalities collectively referred to as sickle cell nephropathy (SCN).[2] SCN includes hyposthenuria, hematuria, proteinuria, renal tubular dysfunction, glomerular hyperfiltration, papillary necrosis, and eventual progression to chronic kidney disease (CKD) and end-stage renal disease (ESRD).[3,4]

Renal involvement in SCD is clinically important due to its early onset and progressive nature. Studies indicate that nephropathy affects approximately 5–18% of patients, with early signs of renal dysfunction detectable even in pediatric populations.[5,6] In children, manifestations such as glomerular hyperfiltration and microalbuminuria often precede overt renal disease. Although advanced CKD is uncommon in early life, findings such as albuminuria (10–27%) and hematuria (4–12%) highlight the presence of early renal damage and the need for timely screening.[7,8]

The pathophysiology of SCN is multifactorial, involving repeated vaso-occlusive episodes, ischemia-reperfusion injury, oxidative stress, and endothelial dysfunction. These processes ultimately result in structural damage, including glomerulosclerosis and tubulointerstitial fibrosis, leading to

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progressive decline in renal function.[2] Various clinical and biochemical factors, such as age, disease severity, frequency of vaso-occlusive crises, hypertension, and transfusion history, have been associated with increased risk of nephropathy.[9]

Despite the recognized burden of renal complications in SCD, data from Indian pediatric populations remain limited. Therefore, assessing the prevalence and identifying risk factors for nephropathy in children with steady-state SCD in a tertiary care centre is essential for early diagnosis, risk stratification, and prevention of long-term renal morbidity.

Materials and Methods

After obtaining approval from the Institutional Ethics Committee, this hospital-based cross-sectional observational study was conducted among children aged 2–14 years with Sickle Cell Anemia in steady state attending the Department of Paediatrics at Chacha Nehru Bal Chikitsalaya (CNBC), attached to M.G.M. Medical College, Indore, Madhya Pradesh, over a period of one year. Written informed consent was obtained from parents or legal guardians prior to enrollment, and assent was obtained from children above 12 years of age. Confidentiality of participant data was strictly maintained, and participation was voluntary, with the option to withdraw at any stage without affecting medical care.

The sample size was calculated using Cochran's formula ($N = Z^2 \times P \times Q / d^2$), considering a prevalence (P) of hematuria in sickle cell anemia as 4.1%, with a 95% confidence interval (Z = 1.96) and margin of error (d) of 7%. The minimum calculated sample size was 51, which was increased to 60 to account for a 10–15% non-response rate. Thus, a total of 60 children were included in the study using a consecutive sampling method.

Inclusion and Exclusion Criteria

Children aged 2–14 years with confirmed Sickle Cell Anemia by hemoglobin electrophoresis and in steady state (no acute illness, infection, pain crisis, or hospitalization in the preceding three months) were included. Children were excluded if they had urinary tract infection, exposure to nephrotoxic drugs or radiocontrast agents, any acute illness affecting renal function, other causes of hematuria or proteinuria, were menstruating adolescents, or had recent strenuous physical activity within 24 hours.

Methodology: After obtaining informed consent, eligible children were enrolled consecutively. A detailed history was obtained from parents or caregivers using a predesigned and pretested proforma. Information regarding sociodemographic profile, clinical history (including age at diagnosis, frequency of vaso-occlusive crises, number of hospitalizations, blood transfusions, and drug history including hydroxyurea), family history, dietary history, developmental status, and immunization history was recorded.

A thorough clinical examination was performed, including general physical and systemic examination, along with anthropometric measurements such as height, weight, and body mass index.

Laboratory evaluation included urine examination (dipstick analysis for proteinuria and hematuria and microscopic examination), renal function tests (serum urea and serum creatinine), and ultrasonography of the kidney, ureter, and bladder (USG-KUB) to assess renal size and structural abnormalities. Children were evaluated for nephropathy based on urinary abnormalities and imaging findings.

Outcome Measures: The primary outcome was to determine the prevalence of nephropathy among children with sickle cell anemia in steady state. Secondary outcomes included identification of demographic, clinical, and laboratory risk factors associated with nephropathy.

Statistical Analysis: All data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The Chi-square test or Fisher's exact test was applied for categorical variables, and the Mann–Whitney U test was used for comparison of continuous variables, as appropriate. A p-value of less than 0.05 was considered statistically significant.

Results

The present study included 63 children aged 2–14 years with Sickle Cell Anemia in steady state. The majority of participants (65.1%) belonged to the 5–10 years age group, followed by >10 years (27.0%) and <5 years (7.9%), with a mean age of 8.37 ± 3.15 years (range: 2–14 years). There was a slight male predominance (57.1%), with a male-to-female ratio of 1.3:1. Most participants belonged to the lower socioeconomic class (55.6%), and tribal children constituted 68.3% of the study population [Table 1].

Table 1: Demographic Characteristics of Study Participants (n = 63)

Parameter	Category	n (%)
Age Group (years)	<5	5 (7.9%)
	5–10	41 (65.1%)
	>10	17 (27.0%)
Mean Age (years)		8.37 ± 3.15
Gender	Male	36 (57.1%)
	Female	27 (42.9%)
Socioeconomic Status	Lower	35 (55.6%)
	Middle	27 (42.9%)
	Upper	1 (1.6%)
Caste/Community	Tribal	43 (68.3%)
	Others	20 (31.7%)

The mean age at diagnosis was 4.05 ± 1.80 years, with most children (61.9%) diagnosed before 5 years. The mean

number of hospitalisations was 3.84 ± 3.09, with the majority (55.6%) having 1–5 hospitalisations. Similarly, the mean number of blood transfusions was 4.27 ± 4.40, and 55.6% had received 1–5 transfusions. The mean number of vaso-occlusive crisis (VOC) episodes was 4.90 ± 4.92, with 58.7% experiencing 1–5 episodes. Good follow-up and drug compliance were observed in 66.7% and 60.3% of participants, respectively [Table 2].

Table 2: Clinical Profile of Study Participants (n = 63)

Parameter	Category	n (%)
Age at Diagnosis (years)	<5	39 (61.9%)
	5–10	24 (38.1%)
	>10	0 (0.0%)
	Mean ± SD	4.05 ± 1.80
Hospitalisations	0	8 (12.7%)
	1–5	35 (55.6%)
	6–10	19 (30.2%)
	>10	1 (1.6%)
	Mean ± SD	3.84 ± 3.09
Blood Transfusions	0	11 (17.5%)
	1–5	35 (55.6%)
	6–10	10 (15.9%)
	>10	7 (11.1%)
	Mean ± SD	4.27 ± 4.40
VOC Episodes	0	7 (11.1%)
	1–5	37 (58.7%)
	6–10	10 (15.9%)
	>10	9 (14.3%)
	Mean ± SD	4.90 ± 4.92
Follow-Up	Good	42 (66.7%)
	Poor	21 (33.3%)
Drug Compliance	Good	38 (60.3%)
	Poor	25 (39.7%)

Renal assessment showed that microscopic hematuria and proteinuria were present in 14.3% of participants each, while 85.7% had no urinary abnormalities. Serum urea, serum creatinine, and eGFR were normal in all participants (100%), indicating preserved renal function. Ultrasonographic evaluation revealed normal kidney size in most cases, with only 4.8% of right kidneys and 6.3% of left kidneys being small [Table 3].

Table 3: Renal and Laboratory Parameters (n = 63)

Parameter	Category	n (%)
Urine Hematuria	Absent	54 (85.7%)
	Present (1+)	9 (14.3%)
Urine Proteinuria	Absent	54 (85.7%)
	Present	9 (14.3%)
Serum Urea	Normal	63 (100.0%)
	Elevated	0 (0.0%)
Serum Creatinine	Normal	63 (100.0%)
	Elevated	0 (0.0%)
eGFR	Normal	63 (100.0%)
	Abnormal	0 (0.0%)
Right Kidney Size	Normal	60 (95.2%)
	Small	3 (4.8%)
Left Kidney Size	Normal	59 (93.7%)
	Small	4 (6.3%)

The mean haemoglobin level was 7.46 ± 1.12 g/dL. A majority (47.6%) had haemoglobin levels between 7–9 g/dL, while 39.7% had severe anemia (<7 g/dL), consistent with chronic hemolytic anemia [Table 4].

Table 4: Haematological Profile (n = 63)

Haemoglobin Level (g/dL)	n (%)
<7.0	25 (39.7%)
7.0–9.0	30 (47.6%)
>9.0	8 (12.7%)
Total	63 (100.0%)
Mean ± SD	7.46 ± 1.12

Nephropathy was present in 13 (20.6%) children (95% CI: 10.6%–30.6%). Among these, 6.3% had isolated hematuria,

6.3% had isolated proteinuria, and 7.9% had both. The majority (79.4%) had no evidence of nephropathy. Renal function tests remained normal in all participants, indicating early or subclinical renal involvement. [Table 5].

Table 5: Prevalence of Nephropathy (n = 63)

Nephropathy Status	n (%)
Isolated Hematuria	4 (6.3%)
Isolated Proteinuria	4 (6.3%)
Both (Hematuria + Proteinuria)	5 (7.9%)
Absent	50 (79.4%)
Total	63 (100.0%)

Analysis of risk factors demonstrated that age was significantly associated with combined nephropathy (p = 0.020). Poor follow-up was significantly associated with hematuria (p = 0.008), while poor drug compliance showed strong associations with proteinuria (p = 0.031), hematuria (p < 0.001), and combined abnormalities (p = 0.017). No significant associations were observed with gender, socioeconomic status, or caste [Table 6].

Table 6: Association of Demographic and Clinical Variables with Nephropathy and Renal Parameters (n = 63)

Variable	Parameter	Abnormal n (%)	Normal n (%)	p-value
Age Category	Proteinuria	9 (14.3%)	54 (85.7%)	0.096
	Hematuria	9 (14.3%)	54 (85.7%)	0.096
	Both (Hematuria + Proteinuria)	5 (7.9%)	58 (92.1%)	0.020
	Kidney Size (Right)	3 (4.8%)	60 (95.2%)	0.276
	Kidney Size (Left)	4 (6.3%)	59 (93.7%)	0.515
Gender	Proteinuria	9 (14.3%)	54 (85.7%)	0.640
	Hematuria	9 (14.3%)	54 (85.7%)	0.640
	Both	5 (7.9%)	58 (92.1%)	0.737
	Kidney Size (Right)	3 (4.8%)	60 (95.2%)	0.147
	Kidney Size (Left)	4 (6.3%)	59 (93.7%)	0.412
Socioeconomic Status	Proteinuria	9 (14.3%)	54 (85.7%)	0.342
	Hematuria	9 (14.3%)	54 (85.7%)	0.342
	Both	5 (7.9%)	58 (92.1%)	0.514
	Kidney Size (Right)	3 (4.8%)	60 (95.2%)	0.911
	Kidney Size (Left)	4 (6.3%)	59 (93.7%)	0.713
Caste	Proteinuria	9 (14.3%)	54 (85.7%)	0.294
	Hematuria	9 (14.3%)	54 (85.7%)	0.068
	Both	5 (7.9%)	58 (92.1%)	0.276
	Kidney Size (Right)	3 (4.8%)	60 (95.2%)	0.565
	Kidney Size (Left)	4 (6.3%)	59 (93.7%)	0.393
Follow-Up	Proteinuria	9 (14.3%)	54 (85.7%)	0.056
	Hematuria	9 (14.3%)	54 (85.7%)	0.008
	Both	5 (7.9%)	58 (92.1%)	0.070
	Kidney Size (Right)	3 (4.8%)	60 (95.2%)	0.530
	Kidney Size (Left)	4 (6.3%)	59 (93.7%)	0.855
Drug Compliance	Proteinuria	9 (14.3%)	54 (85.7%)	0.031
	Hematuria	9 (14.3%)	54 (85.7%)	<0.001
	Both	5 (7.9%)	58 (92.1%)	0.017
	Kidney Size (Right)	3 (4.8%)	60 (95.2%)	0.113
	Kidney Size (Left)	4 (6.3%)	59 (93.7%)	1.000

Comparison of continuous variables showed that children with nephropathy had significantly higher mean age (10.42 ± 2.77 vs. 7.84 ± 3.05 years; p = 0.009) and significantly greater disease burden, reflected by higher hospitalisations, blood transfusions, and VOC episodes (p < 0.001). No significant differences were observed for haemoglobin levels or kidney size [Table 7].

Table 7: Comparison of Continuous Variables Between Nephropathy Groups (n = 63)

Parameter	Nephropathy Present (n=13) Mean ± SD	Median (Range)	Nephropathy Absent (n=50) Mean ± SD	Median (Range)	U value	p-value
Age (years)	10.42 ± 2.77	10.00 (5.50–14.00)	7.84 ± 3.05	7.50 (2.00–14.00)	477.5	0.009
Age at Diagnosis (years)	3.50 ± 1.96	3.00 (1.00–8.00)	4.20 ± 1.75	4.00 (0.58–8.00)	239.0	0.141
Hospitalisations	7.00 ± 3.65	7.00 (0–14)	3.02 ± 2.34	2.50 (0–9)	534.5	<0.001
Blood Transfusions	9.15 ± 5.37	8.00 (1–20)	3.00 ± 3.07	2.00 (0–14)	559.5	<0.001
VOC Episodes	10.15 ± 6.05	8.00 (3–24)	3.54 ± 3.53	3.00 (0–14)	567.5	<0.001
Haemoglobin (g/dL)	7.19 ± 1.02	6.80 (5.90–8.80)	7.53 ± 1.14	7.50 (5.50–10.00)	278.5	0.434
Right Kidney Size (cm)	6.60 ± 0.64	6.40 (6.00–8.00)	6.60 ± 0.62	6.50 (5.30–8.50)	309.0	0.792
Left Kidney Size (cm)	6.72 ± 0.52	6.60 (6.20–8.20)	6.64 ± 0.57	6.50 (5.70–8.20)	363.5	0.517

Discussion

The present hospital-based cross-sectional study evaluated the prevalence and risk factors of nephropathy in children with Sickle Cell Anemia in steady state. A total of 63 children aged 2–14 years were included, providing region-specific insight from Central India, a known high-burden area.

The overall prevalence of nephropathy in this study was 20.6% (95% CI: 10.6%–30.6%), defined by the presence of hematuria and/or proteinuria. This finding is consistent with existing literature, though variability exists depending on definitions and diagnostic methods. Wigfall et al,[10] (2000) reported persistent proteinuria in 6.2% of children, increasing to approximately 12% in adolescents, which is lower than our estimate as their study assessed only proteinuria. In contrast, Eke et al (2012),[11] reported microalbuminuria in 18.5% of children, closely aligning with our findings. Similarly, Nnaji et al. (2020),[8] demonstrated early renal involvement in children with evidence of hyperfiltration and mild urinary abnormalities.

Higher prevalence rates have been documented in some studies. Ranque et al (2014),[12] observed that more than one-quarter of children had albuminuria, increasing with age. Kamble et al. (2020),[13] from Central India reported renal involvement in nearly 50% of children, possibly due to inclusion of patients in crisis and use of microalbuminuria as a diagnostic marker. Machila et al. (2020),[14] found renal abnormalities in 36% of children, while Isaza-López et al. (2020),[15] reported nephropathy in up to 70% using broader criteria. Thus, the prevalence observed in our study lies within the global range (6–70%) and reflects early-stage renal involvement in steady-state patients.

Age was identified as a significant risk factor. Children with nephropathy were older (10.42 ± 2.77 vs. 7.84 ± 3.05 years; $p = 0.009$), and combined hematuria with proteinuria showed a significant association with age ($p = 0.020$). Although individual associations for hematuria and proteinuria did not reach statistical significance ($p = 0.096$), the trend toward higher prevalence in older children was evident. These findings are supported by Wigfall et al. (2000),[16] Eke et al. (2012),[11] and Ranque et al. (2014),[12] all of whom demonstrated increasing renal involvement with age. Maurício et al. (2021),[17] Elzorkany et al. (2024),[18] and Thakkar et al. (2021),[19] further confirmed age as a key determinant of nephropathy progression.

Markers of disease severity showed strong associations with nephropathy. Children with nephropathy had significantly higher numbers of hospitalisations, blood transfusions, and vaso-occlusive crisis episodes (all $p < 0.001$). These findings are consistent with Eke et al. (2012),[11] who reported hospitalization frequency as an independent predictor of renal involvement. Xu et al. (2016),[20] identified markers of hemolysis and disease severity as predictors of nephropathy progression. Similar associations have been reported by Bukar et al. (2019),[21] Machila et al. (2020),[14] and Nnaji et al. (2020),[8] reinforcing the role of recurrent vaso-occlusion and chronic hemolysis in renal injury.

Importantly, modifiable factors such as follow-up and drug compliance were significantly associated with nephropathy. Poor follow-up was associated with higher prevalence of hematuria ($p = 0.008$), while poor drug compliance showed strong associations with proteinuria ($p = 0.031$), hematuria ($p < 0.001$), and combined abnormalities ($p = 0.017$). These findings highlight the importance of adherence to treatment and regular monitoring. Although direct comparisons are limited, studies such as Xu et al (2016),[20] and Wigfall et al (2000),[16] indirectly support this association by linking disease control to renal outcomes.

Despite urinary abnormalities, all participants had normal serum urea, creatinine, and eGFR, indicating preserved renal function. This observation is consistent with Nnaji et al. (2020),[8] Machila et al. (2020),[14] and Ephraim et al. (2015),[22] who reported normal or increased eGFR in pediatric populations. In contrast, adult studies such as Bukar et al. (2019),[21] and Elzorkany et al. (2024),[18] have demonstrated declining renal function, highlighting the progressive nature of sickle cell nephropathy.

Ultrasonographic evaluation revealed predominantly normal kidney size, with only a small proportion showing reduced size and no significant association with nephropathy. These findings are comparable to Kamble et al. (2020),[13] suggesting that structural changes occur later in disease progression.

Hemoglobin levels did not show a significant association with nephropathy ($p = 0.434$), although previous studies such as Eke et al. (2012),[11] Ranque et al. (2014),[12] and Maurício et al. (2021),[17] have reported lower hemoglobin as a risk factor. This discrepancy may be due to uniformly low hemoglobin levels in our cohort and the relatively small sample size.

In summary, this study demonstrates that nephropathy is a common early complication in pediatric sickle cell anemia, significantly associated with increasing age, disease severity, and modifiable factors such as follow-up and drug compliance. Early detection through routine urine screening is essential, as renal function tests may remain normal in early stages.

Limitations: The present study has certain limitations. Being a cross-sectional study, causal relationships could not be established. The relatively small sample size ($n = 63$) may have limited the statistical power to detect some associations. Nephropathy was assessed using dipstick methods rather than quantitative measures such as urine albumin-to-creatinine ratio, which may underestimate early renal involvement. Additionally, the single-centre design may limit generalizability of the findings. Finally, other markers of tubular dysfunction and long-term follow-up data were not evaluated.

Conclusion

Nephropathy was observed in 20.6% of children with Sickle Cell Anemia in steady state, indicating early renal involvement despite normal serum urea, creatinine, and eGFR. Increasing age and markers of disease severity were significantly associated with nephropathy. Importantly, poor drug compliance and inadequate follow-up emerged as key modifiable risk factors. These findings highlight that renal damage begins early and may remain subclinical. Routine urine screening is essential for early detection, as conventional renal function tests may be normal. Improving treatment adherence and regular follow-up could play a crucial role in preventing progression to chronic kidney disease.

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Conflicts of interest
There are no conflicts of interest.

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