



## Evolving Consensus on Management of Nephrotic Syndrome in Childhood: Focus on KDIGO Guidelines 2025

Nephrotic syndrome, characterized by heavy proteinuria, edema, and hypoalbuminemia, is among the most common chronic kidney diseases of childhood, with an annual incidence of 2.9 (0-6.5) per 100,000 children.<sup>1</sup> In >80% of children, the proteinuria completely remits within 4-6 weeks of corticosteroid therapy, termed steroid-sensitive nephrotic syndrome (SSNS).<sup>2,3</sup> Patients with SSNS may show infrequent or frequent relapses but have satisfactory long-term outcomes.<sup>3</sup> Lack of complete remission following therapy, termed steroid resistance (SRNS), may be observed at the onset of disease (initial steroid-resistance) or following a relapse (late-resistance), and carries a high risk of disease and therapy-associated complications, including kidney failure.<sup>2</sup>

Over the past 3 decades, evidence-based guidelines have refined the therapy of nephrotic syndrome. Guidelines on management of nephrotic syndrome were published by the Indian Society of Pediatric Nephrology (ISPNA) in 2000, and revised in 2008-09 and 2021.<sup>4,5</sup> Clinical practice guidelines (CPG) for therapy of pediatric nephrotic syndrome were proposed by the International Pediatric Nephrology Association (IPNA) in 2020 and 2023,<sup>6,7</sup> and recently by the KDIGO in 2025.<sup>8</sup>

Recent randomized trials in childhood nephrotic syndrome have informed the 2025 KDIGO guideline, which closely aligns with the IPNA guidelines and retains unchanged recommendations for the dose and duration of therapy for first episodes and relapses. Key updates include the introduction of a "confirmation period" for late responders, algorithms for kidney biopsy and genetic testing, and a treatment flowchart for immunosuppression in steroid-sensitive disease. Definitions are harmonized with IPNA recommendations, and routine use of oral steroids during upper respiratory tract infections to prevent relapse is discouraged except in selected patients. Similarities and differences across guidelines have been summarized in Table 1.<sup>4-9</sup> We discuss specific aspects where the CPG differ from the ISPNA guidelines, with the aim to harmonize therapy for children with nephrotic syndrome.

### Evaluation and Management of the First Episode

Evaluation at the onset of nephrotic syndrome focuses on confirming the diagnosis, ruling out secondary causes, and screening for complications. All guidelines advise limited evaluation at the onset, comprising urinalysis and urine protein-creatinine ratio (first morning), blood counts, and levels of creatinine and albumin. Regional variations may exist, depending on the prevalence of systemic infections that affect corticosteroid use.

The current KDIGO, IPNA and ISPNA advice on limiting the duration of prednisone therapy for the initial episode of nephrotic syndrome to 8-12 weeks is based chiefly on meta-analyses from high-quality multicenter trials that showed no differences in outcomes in patients receiving standard (8-12 weeks) or prolonged (16-24 weeks) therapy with prednisone.<sup>10</sup> The KDIGO suggestion to prolong initial therapy in children younger than 4-6 years is based on *post-hoc* subgroup analyses from studies from India and the United Kingdom.<sup>11,12</sup> Similarly, the KDIGO advice for longer duration therapy in young patients with delayed (>7-day) remission is based on low-quality evidence and is rather empiric.<sup>13,14</sup>

### Therapy of Relapses

Therapy for relapses comprises daily prednisone in standard doses until remission, followed by therapy on alternate days (AD) for 4 weeks [Table 1].<sup>4-8</sup> The KDIGO, IPNA, and ISPNA agree that prednisone therapy, for initial episode or relapses, may be dosed either by body surface area or by weight, the former being preferred in young patients.<sup>4,6,8</sup> The use of low-dose or short-duration prednisone therapy is not recommended.

### Frequent Relapses and Steroid Dependence

Early identification of frequent relapses or steroid dependence, which affects one-half of patients with SSNS, is important to prevent morbidities associated with relapses and minimize corticosteroid toxicity through appropriate steroid-sparing strategies. The KDIGO/IPNA definition of frequent relapses, based on cumulative steroid exposure, is sound and likely to be adopted by the ISPNA [Table 1],<sup>4-8</sup> since even short-term use of high-dose corticosteroids carries risks of hypertension, raised intraocular pressure, osteoporosis, obesity, and myopathy.<sup>15</sup>

### Long-term alternate-day (AD) prednisone

Guidelines from the KDIGO, IPNA, and ISPNA recommend 6-12 months of therapy with AD prednisone for frequent relapsers without features of steroid toxicity, although the doses differ [Table 1].<sup>4,6,8</sup> Since children with frequent relapses are expected to have a long disease course and most medications do not have a legacy effect, the initial use of long-term AD prednisone is rational since it may postpone or avoid the need for additional medications in a fraction of children.

Upper respiratory tract viral infections are the trigger for ~50% relapses of nephrotic syndrome. Contrary to data from three studies that found reduced frequency of relapses in patients on AD prednisolone who received

**Table 1: Comparison between the clinical practice guidelines of the Kidney Disease Improving Global Outcomes (KDIGO), International Pediatric Nephrology Association (IPNA), and the Indian Society of Pediatric Nephrology (ISPN)**

Parameter	KDIGO 2025; IPNA 2020 and 2023	ISPN 2021, 2022
Nephrotic syndrome	Nephrotic range proteinuria <sup>a</sup> , and either serum albumin <3 g/dL or edema (when serum albumin is not available)	Nephrotic range proteinuria <sup>a</sup> , serum albumin <3 g/dL, and edema
Kidney biopsy	Age >12 years; steroid resistance; atypical features <sup>b</sup> ; family history or syndromic features if genetic tests not accessible; following prolonged CNI use	Steroid resistance; atypical features <sup>b</sup> ; following (preferably, also before) prolonged CNI use
Genetic testing	Congenital and infantile illness; all initial steroid-resistance; family history of SRNS or FSGS; syndromic features	Congenital NS; initial steroid-resistance with onset in infancy, family history or syndromic features; steroid-resistance with non-response to CNI or progression to KF; prior to transplantation
Steroid sensitive nephrotic syndrome		
Prednisone for the initial episode	4-6 weeks daily and 4-6 weeks AD <sup>c</sup> ; BSA or weight-based dosing <sup>c</sup> ; prolonged therapy (16-24 weeks) in <6-year-olds with delayed remission	6 weeks daily and 6 weeks on AD <sup>c</sup> ; BSA or weight-based dosing <sup>c</sup> ; no indication for prolonging therapy
Prednisone for relapse	Daily until remission, then AD for 4 weeks; BSA or weight-based dosing <sup>c</sup>	Daily until remission, then AD for 4 weeks; BSA or weight-based dosing <sup>c</sup>
Frequent relapses	≥2 relapses in the first 6 months after remission of the initial episode; ≥3 relapses in any 1-year-old	≥2 relapses in the first 6 months after stopping initial therapy; ≥4 relapses in any 1-year-old
Difficult-to-treat disease	Not defined	Both of: (i) frequent relapses, or significant steroid toxicity with infrequent relapses; (ii) failure of ≥2 steroid-sparing agents
Prolonged prednisone therapy	Consider in low doses, ≤0.5 mg/kg AD if frequently relapsing without steroid toxicity	Initial strategy for frequent relapses without steroid toxicity: administer at 0.5-0.7 mg/kg AD for 6-12 months
Prednisone during infections	Do not recommend; consider 0.5 mg/kg/day for 6 days, if on AD prednisone and previous infection-associated relapses	If receiving prednisone AD as above, administer daily for 5-7 days
Steroid-sparing therapy: Indications, choice	Frequent relapses: cyclophosphamide, levamisole; steroid dependence: choose from MMF, CNI, rituximab; cyclophosphamide is less preferred; IPNA advises considering rituximab after failure of ≥1 agent	Failure of AD prednisone: Levamisole or MMF; high steroid threshold, steroid toxicity, or complicated relapses: MMF or cyclophosphamide; difficult-to-treat disease: CNI, then rituximab
Response to therapy	SSNS controlled on therapy: Sustained remission or infrequent relapses during immunosuppression without significant drug-related toxicity	Stable remission: Sustained remission or infrequent relapses during immunosuppressive therapy
Steroid-resistant nephrotic syndrome (SRNS)		
Steroid resistance	Non-response at 4 weeks, or lack of complete remission at 6 weeks; 'late responder' if partial remission at 4 weeks and complete remission in the subsequent 2-week 'confirmation period' <sup>d</sup>	Lack of complete remission at 6 weeks
First line therapy; duration	CNI (cyclosporine A or tacrolimus); ≥1 year (longer if partial remission)	CNI (cyclosporine A or tacrolimus); ≥2 years
Alternative first-line therapy: indication	Mycophenolate mofetil (MMF): if eGFR <30 mL/min per 1.73 m <sup>2</sup> ; cyclophosphamide (IV, oral): if CNI not available	IV (not oral) cyclophosphamide may be used if CNI is not feasible
CNI-responsive SRNS	Partial remission within 6 months and/or complete remission within 12 months of CNI therapy at adequate doses and/or levels	Complete or partial remission within 6 months of CNI therapy at adequate doses and levels
Multi-drug resistant SRNS	Lack of complete remission within 1 year of therapy with two mechanistically distinct steroid-sparing agents at standard doses	No such definition; partial remission to second-line therapy is an acceptable response
Indications for use of MMF	eGFR <30 mL/min/1.73 m <sup>2</sup> ; CNI therapy for >1 year; steroid sensitive relapses; CNI-resistant SRNS	Prolonged CNI use with relapses; add-on in CNI-resistant SRNS
Indications for use of rituximab	CNI-resistant SRNS; allograft recurrence	Prolonged CNI use with relapses; CNI-resistant SRNS; allograft recurrence
Managing CNI-resistant and multidrug-resistant SRNS	Include in clinical trials; switch to MMF or rituximab; consider ofatumumab, lipid apheresis, plasma exchange, immunoabsorption	Rule out monogenic cause; consider rituximab; addition of MMF Avoid immunosuppression if <60 mL/min/1.73 m <sup>2</sup>

contd..

**Table 1: Continued**

Managing monogenic SRNS	ACEi/ARB; immunosuppression not usually advised	ACEi/ARB; immunosuppression not usually advised
Renal transplantation	Counsel families regarding outcomes; evaluation of recipient and donor; managing recurrent FSGS	

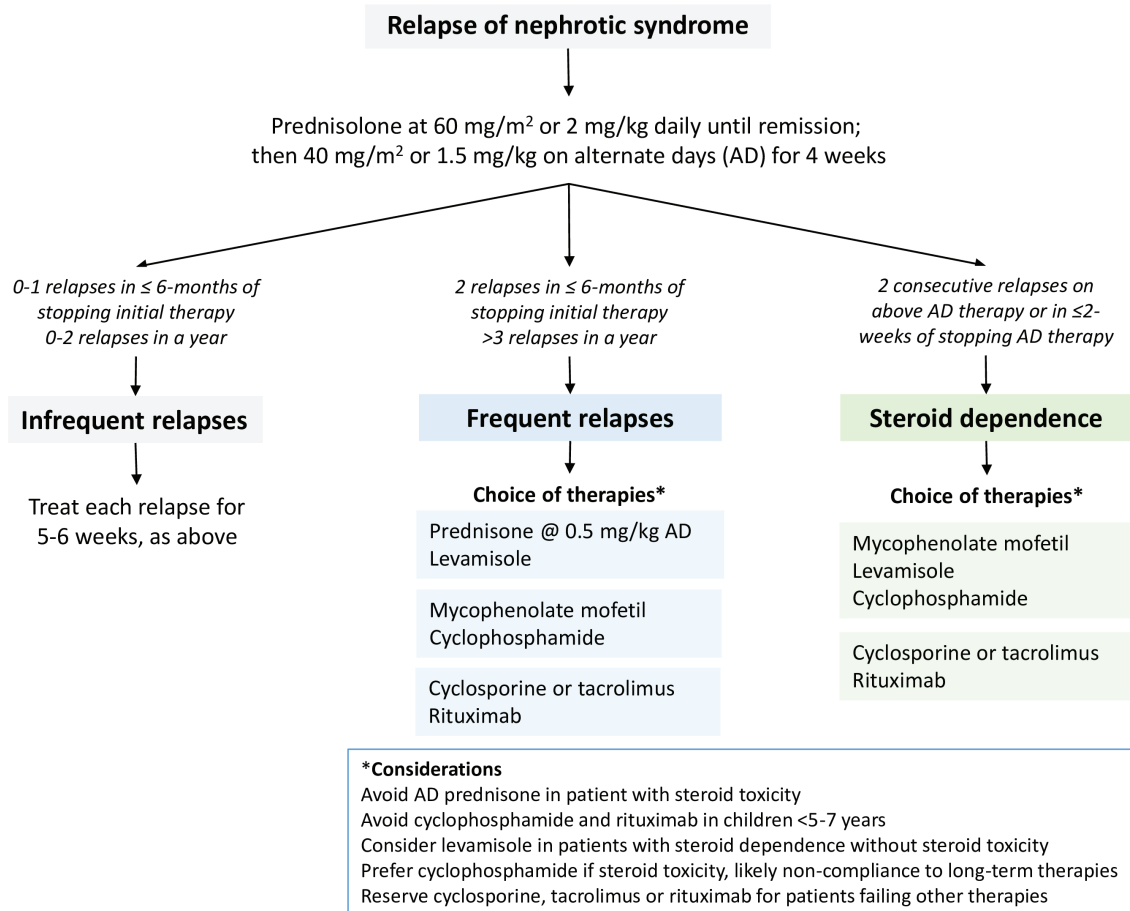
ACEi: Angiotensin converting enzyme inhibitor, AD: Alternate day, ARB: Aldosterone receptor blocker, CNI: Calcineurin inhibitor, eGFR: Estimated glomerular filtration rate, FSGS: Focal segmental glomerulosclerosis, KF: Kidney failure, MMF: Mycophenolate mofetil, SRNS: Steroid resistant nephrotic syndrome, UPCR: Urine protein to creatinine

<sup>a</sup>24-h urine protein >1 g/m<sup>2</sup> surface area; spot UPCR >2 mg/mg; ≥3+ by dipstick; relapse is diagnosed on first morning urine on 3 consecutive days

<sup>b</sup>Gross or persistent microscopic hematuria; hypocomplementemia; acute kidney injury not explained by hypovolemia; features of vasculitis or lupus; persistent hypertension

<sup>c</sup>Therapy based on body surface area (BSA): 60 mg/m<sup>2</sup> daily and 40 mg/m<sup>2</sup> on alternate days; weight-based dosing: 2 mg/kg/day and 1.5 mg/kg on alternate days; maximum daily dose: 60 mg (KDIGO, IPNA, ISPN); maximum dose on alternate days: 50 mg (IPNA), 40 mg (KDIGO, ISPN)

<sup>d</sup>KDIGO, IPNA: Therapy with ACEi/ARB and IV methylprednisolone pulses may be administered during the ‘confirmation period.’



**Figure 1:** Summarizes the choice of steroid-sparing medications, determined by the pattern of relapses, patient age, presence of steroid toxicity, and physician and patient preference.

daily steroid therapy for 5-7 days during upper respiratory infections,<sup>16-18</sup> a large placebo-controlled RCT (PREDNOS-2) did not show similar benefits.<sup>19</sup> The inconsistent results might reflect differences in eligibility criteria, disease severity, ethnicity, and concurrent therapy with other medications. Based on the findings of PREDNOS-2, the KDIGO advises that administration of daily prednisone during infections be limited to patients with previous infection-associated relapses [Table 1].<sup>8</sup> This advice is at

variance from that of ISPN, which routinely recommends 5-7 days of prednisone therapy during upper respiratory infections.<sup>4</sup>

**Steroid-sparing medications**

All guidelines recommend the use of steroid-sparing agents in patients with frequent relapses or steroid dependence [Table 1].<sup>4,6,8</sup> While the guidelines differ in therapeutic choices, it is important to note that few

**Table 2: Therapies for frequently relapsing, steroid-dependent, or steroid-resistant nephrotic syndrome**

Drug	Dose and duration	Adverse effects	Monitoring
Prednisolone	0.5-0.7 mg/kg on alternate days for 6-12 months <sup>a</sup> ; given daily during infections	Weight gain, Cushingoid features; reduced height velocity; hypertension; raised intraocular pressure; cataract; impaired glycemic tolerance	At each visit: Anthropometry, blood pressure q6-12 months: Height velocity; ocular examination; HbA1c, fasting glucose and lipids
Levamisole	2-2.5 mg/kg on alternate days for 1-3 yr	Neutropenia, vasculitic rash; raised transaminases, seizures	At each visit: history, examination q2-3 months: complete blood counts q6 months: hepatic transaminases, ANCA
Cyclophosphamide	2-2.5 mg/kg/day orally for 8-12 weeks <sup>b</sup>	Leukopenia, alopecia, hyperpigmented nails; gonadal toxicity and malignancies	At each visit: history, examination q2 weeks: total and differential leukocyte counts
Mycophenolate mofetil	900-1200 mg/m <sup>2</sup> /day for 2-3 years	Abdominal pain, diarrhea, nausea; viral warts; leukopenia; elevated transaminases	At each visit: history, examination q2-3 months: complete blood counts q3-6 months: hepatic transaminases
Cyclosporine	4-5.5 mg/kg/day (target trough levels) for 2-3 years	Nephrotoxicity; hyperkalemia; gum hyperplasia, hirsutism; hypertension; dyslipidemia; hepatotoxicity	At each visit: history, examination q2-3 months: blood urea, creatinine, electrolytes q3-6 months: uric acid, fasting lipids, magnesium, glucose
Tacrolimus	0.1-0.2 mg/kg/day (target trough levels) for 2-3 years	Nephrotoxicity, hyperkalemia; tremors, seizures, headache; diarrhea; glucose intolerance; hypomagnesemia; hepatotoxicity	q3-6 months: uric acid, fasting lipids, magnesium, glucose
Rituximab	375 mg/m <sup>2</sup> as IV infusion, 2 doses 1 week apart (target B cell depletion)	Infusion reactions; neutropenia; <i>P. jirovecii</i> pneumonia; acute lung injury; low IgG; reactivation of hepatitis B or JC virus	Before infusion: HBsAg, anti-HCV, anti-HIV; IgG After infusion: CD19 (>48-h after 2 <sup>nd</sup> infusion) q1-3 months: complete blood counts; IgG

<sup>a</sup>Taper to 0.3-0.5 mg/kg if remission is sustained and continued beyond 1 year

studies have prospectively compared various interventions for comparative efficacy and safety. Hence, the choice of therapy is dictated by relative efficacy, toxicity, costs, and physician preference. Figure 1 summarizes the choice of steroid-sparing medications, determined by the pattern of relapses, patient age, presence of steroid toxicity, and physician and patient preference. Table 2 summarizes the dosage and practical considerations in their prescription.<sup>4-8</sup>

### Steroid-resistant nephrotic syndrome

The ISPN defines steroid-resistance as a lack of complete remission following 6 weeks' therapy with prednisone that aligns with the duration of daily corticosteroid therapy for the initial episode.<sup>5</sup> The IPNA and KDIGO define steroid-resistance differently, using the terms 'confirmation period' and 'late responder', which are not evidence-based, confusing, and do not significantly affect management.<sup>7,8</sup> Patients with steroid-resistance require quantification of proteinuria, eGFR, and a kidney biopsy. While KDIGO (and IPNA) advise genetic testing for all patients with infantile onset and steroid-resistant disease,<sup>7,8</sup> the cost of testing and limited access to interpretation of results preclude its application across India. We advise that all infants with congenital nephrotic syndrome should undergo genetic studies. Beyond infancy, the likelihood of detecting monogenic disease in initial steroid-resistance is ~20%, and ISPN guidelines limit genetic testing to disorders with a high likelihood of detecting a monogenic disease [Table 1].<sup>5</sup>

The management of patients with steroid-resistance is challenging because of variable response to immunosuppression, high incidence of therapy- and disease-related adverse effects, and risk of progression to kidney failure.<sup>2,3</sup> Across guidelines, treatment with calcineurin inhibitors (CNI), either cyclosporine or tacrolimus, titrated to their trough levels is recommended, since they induce complete or partial remission of proteinuria in 60-70% patients by 6 months of therapy.<sup>5,7,8</sup> Management choices for patients who are refractory to CNI are limited, empirical, and lack evidence-base. Guidelines suggest the use of rituximab or MMF as add-on therapies in such a context.<sup>5,7,8</sup> Although KDIGO guidelines advise the use of MMF if the eGFR is <30 mL/min/1.73 m<sup>2</sup>, the ISPN has not advised the use of immunosuppression in CKD stage 4-5.<sup>5,7,8</sup> While recognizing that patients with monogenic steroid-resistance may occasionally show partial remission following CNI therapy, expert guidelines do not recommend their use.<sup>5,7,8</sup>

All these guidelines advise regarding management of complications and supportive care, summarized in Supplementary Table S1.<sup>5,7,8</sup> Use of an inhibitor of the renin-angiotensin-aldosterone system is recommended to reduce proteinuria and manage hypertension. Patients with steroid-resistance with non-response to CNI and/or monogenic disease are at risk of progression to kidney failure. More than half the cases with non-genetic disease

that undergo kidney transplantation show recurrent disease that requires specialized management.<sup>5,7</sup>


Review of KDIGO guidelines in the context of IPNA and ISPN guidelines shows reasonable consensus on the management of steroid-sensitive and steroid-resistant nephrotic syndrome in children. Minor differences reflect practice patterns and physician preferences rather than ethnic differences in therapeutic response.

In the Indian context, key considerations include the use of optional vaccines not included in the regular national immunization schedule to reduce infection-related morbidity, and individualized therapeutic decision-making based on age, cumulative steroid exposure, cost, and resource availability. Access to generic second-line immunosuppressive agents through schemes such as the Pradhan Mantri Bharatiya Janaushadhi Kendras facilitates sustained therapy. Caregiver education should focus on infection prevention, home urine protein monitoring to detect early relapses, and lifestyle measures to mitigate steroid-related metabolic and psychosocial complications. Planned transition to adult services is important for adolescents with persistent or relapsing disease. The KDIGO advice is broadly in agreement with ISPN guidance on childhood nephrotic syndrome, with recommendations that are largely applicable across the subcontinent.

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