MARVIX AI

SAMPLE PEDIATRIC NEPHROLOGY SOAP NOTE

STEROID-DEPENDENT NEPHROTIC SYNDROME — AGE 6

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1 PATIENT INFORMATION

NAME	Amara J. Sithole
DOB	02/11/2020 (6 yrs)
AGE / SEX	6 / Female
MRN / PATIENT ID	PED-441209
REFERRAL SOURCE	Dr. Marcus Hale, General Pediatrics (original referral 2024)
PARENT / GUARDIAN PRESENT	Mother (Thandiwe Sithole) present; father by phone
DATE OF SERVICE	July 14, 2026
PROVIDER	Priyanka Raghunathan, MD
CREDENTIALS	MD, Pediatric Nephrology
VISIT TYPE	Pediatric Nephrology Follow-Up — fourth relapse
PRIMARY CARE PROVIDER	Dr. Marcus Hale, Riverside Pediatrics
INFORMANT / COLLATERAL SOURCE	Mother — primary historian; home dipstick log reviewed

CC CHIEF COMPLAINT

Mother reports: “Her eyes are puffy again in the mornings and the dipstick has been showing 3+ for four days.” Fourth relapse of nephrotic syndrome within 12 months, occurring during a prednisolone taper.

S SUBJECTIVE

3a REASON FOR PEDIATRIC NEPHROLOGY EVALUATION

Relapse of idiopathic nephrotic syndrome, detected by the mother on home urine dipstick monitoring. This is the fourth relapse in 12 months and the second occurring during a steroid taper, which meets the definition of steroid-dependent nephrotic syndrome. The visit is to confirm relapse, treat, and — more importantly — to make a decision about steroid-sparing therapy given the accumulating toxicity of repeated corticosteroid courses in a growing child.

3b KIDNEY DISEASE HISTORY

First presentation in April 2024 at age 4 with periorbital edema and 4+ proteinuria. Diagnosed with idiopathic nephrotic syndrome, presumed minimal change disease given age and presentation. Achieved complete remission after 11 days of prednisolone — confirming steroid sensitivity, which carries a favorable long-term renal prognosis. Completed a standard 12-week induction course. Relapses have followed at roughly 3-month intervals: August 2024 (with a viral URI), January 2025, April 2025 (during taper), and now July 2026 (during taper). Kidney function has remained normal throughout, with no hypertension at baseline and no hematuria. No prior AKI. No biopsy performed to date, appropriately, given steroid sensitivity.

3c URINARY SYMPTOMS

Mother reports frothy urine for the past 5 days and reduced urine output over the last 2 days. Amara denies dysuria and there is no gross hematuria. She had been fully toilet trained since age 3 but has had two episodes of nighttime wetting this week, which the mother notes has happened during previous relapses. No flank or abdominal pain. No stone history.

3d UTI / INFECTION HISTORY

No febrile UTIs and no history of recurrent UTI. No vesicoureteral reflux; a renal and bladder ultrasound at diagnosis was normal and no VCUG was indicated. No urinary tract imaging since. No prophylactic antibiotics. No bowel or bladder dysfunction — stooling is regular. Of note, nephrotic children are at elevated risk of spontaneous bacterial peritonitis and invasive pneumococcal infection during relapse, and this risk was specifically reviewed today.

3e BLOOD PRESSURE HISTORY

No history of hypertension at baseline. Home blood pressure is not routinely monitored. Clinic readings have historically tracked at the 60th–75th percentile for age, sex, and height. During the April 2025 relapse her blood pressure rose transiently to the 92nd percentile on high-dose steroids and normalized on taper. No symptoms of hypertension — no headache, visual change, or dizziness. Salt intake at home is moderate; the family has been counseled previously on sodium restriction during relapse.

3f EDEMA / VOLUME SYMPTOMS

Periorbital edema present on waking for 5 days, improving through the day — the classic pattern. New pretibial pitting edema noted by the mother over the past 2 days. Weight is up 1.8 kg over 6 days, from a dry weight of 20.4 kg. Mild abdominal fullness with a slightly protuberant abdomen, though no reported distension or pain. No dyspnea, orthopnea, or respiratory difficulty. Oral intake is preserved. No vomiting or diarrhea, and no dehydration features.

3g GROWTH AND DEVELOPMENT

This is an area of active concern. Height has drifted from the 50th percentile at diagnosis to the 34th percentile currently, with height velocity slowing to approximately 4.2 cm/year over the past year — below the expected 5.5–6 cm/year for her age. Weight percentile has risen from the 55th to the 78th, reflecting steroid effect rather than nutritional gain. Developmental milestones are otherwise fully appropriate. She is entering Year 2 and reads well, but has missed 14 school days over the past year for relapses and appointments. Her mother reports Amara has become self-conscious about her facial appearance and has been reluctant to attend birthday parties — a psychosocial dimension that matters at this age.

3h DIET / FLUID HISTORY

No formal fluid restriction currently. Sodium intake has been reduced during relapses per prior counseling, though the mother reports this is difficult to sustain given the marked steroid-induced appetite increase — Amara “asks for food constantly” on high-dose prednisolone. No potassium or phosphorus restriction is required. She is not a picky eater. No formula or specialized feeding history. Protein intake is adequate; no protein restriction indicated.

3i MEDICATION AND NEPHROTOXIN EXPOSURE

Current: prednisolone, presently at 10 mg every other day on the taper when the relapse occurred. Historical: four courses of prednisolone in 12 months. No NSAIDs — the family was counseled at diagnosis and reports strict avoidance, using paracetamol only. No ACE inhibitor or ARB currently. No diuretics at home. No antibiotics recently. No immunosuppressants to date. No contrast exposure. No herbal products or supplements beyond a standard children's multivitamin and the vitamin D and calcium started for steroid bone protection.

3j RELEVANT MEDICAL HISTORY

Born at 34 weeks gestation with a 12-day NICU stay for prematurity and feeding support; no neonatal AKI and no congenital anomalies of the kidney or urinary tract. Prematurity is worth noting as a modest independent risk factor for reduced nephron endowment and long-term renal risk. Mild intermittent asthma, well controlled with as-needed salbutamol. No diabetes, autoimmune disease, cardiac disease, malignancy, or genetic syndrome. Hearing screen normal — relevant to excluding Alport syndrome. No prior surgeries. Immunizations are up to date including pneumococcal conjugate; however, varicella vaccination is incomplete (one dose only), and she had a household varicella exposure through a cousin three weeks ago — a genuine concern in a child about to receive high-dose immunosuppression.

3k FAMILY HISTORY

No family history of nephrotic syndrome, childhood kidney disease, dialysis, or transplant. A maternal grandfather has hypertension and type 2 diabetes. No family history of kidney stones, hematuria, proteinuria, hearing loss, or inherited renal disorders. No consanguinity. This largely reassuring family history supports idiopathic minimal change disease over a genetic podocytopathy, which is relevant because genetic forms are typically steroid-resistant.

3l PRIOR EVALUATION AND TREATMENT

Renal and bladder ultrasound at diagnosis (04/2024): normal-sized kidneys, normal echogenicity, no hydronephrosis or structural abnormality. No VCUG, DMSA, or MAG3 performed — not indicated. No kidney biopsy — appropriately deferred given prompt steroid response. No genetic testing to date, though this would be revisited if steroid resistance emerged. Baseline complements and serologies at diagnosis were normal. Four prednisolone courses with prompt remission each time, typically within 10–14 days. Renal diet counseling provided twice. No urology involvement. No dialysis or transplant planning — not applicable.

3m PERTINENT NEGATIVES

Denies anuria, gross hematuria, severe flank pain, high fever, rapidly worsening edema, severe headache, vision changes, seizure, or altered mental status. No abdominal pain, guarding, or rebound — specifically assessed to exclude spontaneous bacterial peritonitis, a serious complication of nephrotic relapse. No calf pain, unilateral swelling, or respiratory distress to suggest thromboembolism, to which nephrotic children are predisposed. No signs of dehydration despite the reduced urine output. Not currently febrile or systemically unwell.

ROS PEDIATRIC NEPHROLOGY REVIEW OF SYSTEMS

CONSTITUTIONAL / GROWTH

Positive for increased appetite and weight gain; height velocity slowing; no fever, poor feeding, or decreased activity

VOLUME

Positive for periorbital and pretibial edema, 1.8 kg gain, mild abdominal fullness; no dyspnea

URINARY

Positive for frothy urine, reduced output, and two nights of secondary enuresis; no dysuria, hematuria, or urgency

GASTROINTESTINAL

Denies flank or abdominal pain, nausea, vomiting, or

NEUROLOGIC / BP

Denies headache, vision changes, dizziness, or

diarrhea — no features of peritonitis

seizures

IMMUNE / OTHER

Recent household varicella exposure; denies rash, joint pain, or hearing concerns

MEDICATION / ADHERENCE

Good adherence reported; strict NSAID avoidance; sodium restriction difficult during steroid courses

O OBJECTIVE

5a VITALS

TEMPERATURE

36.8 °C

HEIGHT

112.4 cm (34th percentile)

BLOOD PRESSURE

108/68 mmHg

WEIGHT

22.2 kg (78th percentile; dry weight 20.4 kg)

BLOOD PRESSURE PERCENTILE, IF APPLICABLE

88th percentile for age, sex, and height — elevated but below stage 1 hypertension

BMI

17.6 kg/m² (85th percentile)

HEART RATE

96 bpm, regular

GROWTH PERCENTILES

Height 34th (was 50th at diagnosis); weight 78th (was 55th); height velocity 4.2 cm/yr

RESPIRATORY RATE

20 / min

ORTHOSTATIC VITALS, IF OBTAINED

Not obtained — no orthostatic symptoms

OXYGEN SATURATION

99% on room air

5b EXAMINATION

GENERAL APPEARANCE

Alert, interactive, cooperative 6-year-old in no distress; visibly cushingoid with rounded facies and mild central adiposity; well hydrated; appropriately engaged with her mother and with the examiner

GROWTH / DEVELOPMENT

Developmentally appropriate for age — speech, comprehension, and social interaction all normal. Growth trajectory is the concern: height percentile has fallen from the 50th to the 34th with reduced velocity, while weight percentile has risen. Prepubertal, Tanner stage 1. No dysmorphic features.

CARDIOVASCULAR

Regular rate and rhythm; no murmur; peripheral pulses 2+ and symmetric; capillary refill under 2 seconds; 1+ pitting pretibial edema bilaterally; no JVD appreciable

PULMONARY

Clear to auscultation bilaterally; no crackles or wheeze; no increased work of breathing; no pleural effusion signs

ABDOMEN

Soft with mild fullness and a shifting dullness suggesting a small volume of ascites; non-tender throughout with no guarding or rebound — no peritonitis features; no hepatosplenomegaly, mass, or bruit; no flank tenderness; bladder not distended

GENITOURINARY

Normal external female genitalia; mild labial edema noted, consistent with nephrotic fluid distribution; no catheter

EXTREMITIES

1+ pitting pretibial and pedal edema bilaterally; pulses intact; perfusion normal; no joint swelling or tenderness; no wounds; no dialysis access

SKIN

Marked periorbital edema, most prominent on waking per mother; faint striae over the lower abdomen from cumulative steroid exposure; no rash, purpura, or vasculitic lesions; no varicella lesions identified on careful full-skin inspection

NEUROLOGIC

Alert and fully oriented to age-appropriate level; interacts and plays normally; no weakness, tremor, or asterixis; no focal deficit; no seizure activity

DA RENAL / DIALYSIS ACCESS EXAMINATION, IF APPLICABLE

Not applicable — the patient has normal kidney function with no dialysis access and no indication for renal replacement therapy. Her steroid-sensitive disease course carries a favorable long-term renal prognosis, and progression to kidney failure is not anticipated.

L LABORATORY AND DIAGNOSTIC RESULTS

Kidney Function:

Creatinine 0.38 mg/dL — normal for age and body size. eGFR by the bedside Schwartz equation approximately 108 mL/min/1.73m², consistent with her prior baseline. BUN 14 mg/dL. Kidney function has remained normal through all four relapses, which is expected and reassuring in steroid-sensitive disease. Cystatin C not obtained.

Electrolytes / Acid-Base:

Sodium 134 mmol/L (mild dilutional hyponatremia, characteristic of nephrotic relapse), potassium 4.2, chloride 100, bicarbonate 23, calcium 8.1 mg/dL (low, largely reflecting hypoalbuminemia — ionized calcium normal), phosphorus 4.6, magnesium 2.0. Anion gap 11.

Urine Studies:

Urinalysis: protein 4+, no blood, no leukocyte esterase or nitrites, no casts on microscopy. Urine protein/creatinine ratio 6.8 g/g — firmly in the nephrotic range (above 2.0 g/g). Urine albumin/creatinine ratio markedly elevated. Urine calcium/creatinine ratio 0.12 (normal for age). Urine culture negative. Home dipstick log reviewed — the mother has documented 3–4+ protein for 5 consecutive days, confirming relapse by standard criteria.

CKD / Mineral Bone Disease Labs:

PTH 42 pg/mL (normal), 25-OH vitamin D 22 ng/mL (insufficient, relevant given cumulative steroid exposure and bone health risk), corrected calcium normal, phosphorus 4.6, alkaline phosphatase 210 U/L (within the normal range for a growing child).

Anemia Labs:

Hemoglobin 12.8 g/dL, hematocrit 38% — normal, with a component of hemoconcentration expected in nephrosis. Iron studies not indicated. No anemia of CKD, consistent with preserved kidney function.

Serologic / Immune Workup:

Complements C3 and C4 both normal — an important discriminator, as low complement would point toward post-infectious or membranoproliferative glomerulonephritis rather than minimal change disease. ANA negative. ASO and anti-DNase B not repeated (normal at diagnosis). Hepatitis B and C serologies negative. HIV testing not clinically indicated. IgA normal. **Varicella IgG negative** — confirming she is non-immune, which is directly relevant to the recent household exposure and to intensifying immunosuppression.

Stone / Metabolic Workup:

Not indicated — no stone history and a normal urine calcium/creatinine ratio. Serum uric acid 4.1 mg/dL.

Imaging:

No new imaging required this visit. Renal and bladder ultrasound at diagnosis (04/2024) was normal with symmetric kidneys, normal echogenicity, no hydronephrosis, and no structural abnormality. Bedside assessment today suggests a small volume of ascites clinically; formal imaging deferred as it would not change management.

Pathology / Genetics:

No kidney biopsy performed — appropriately deferred throughout, as biopsy is not indicated in steroid-sensitive childhood nephrotic syndrome. No genetic testing to date. Both would be reconsidered only if steroid resistance emerged or if the response pattern changed.

Prior Records Reviewed:

Full pediatric nephrology record from 04/2024 forward including all four relapse episodes and treatment responses; general pediatrics notes from Dr. Hale; complete growth charts with height and weight plotted from birth, which made the velocity decline evident; serial lab trends across relapses; the diagnostic renal ultrasound report; the neonatal discharge summary documenting 34-week prematurity; the immunization record confirming incomplete varicella coverage; and the mother's home dipstick diary.

A ASSESSMENT

Steroid-dependent nephrotic syndrome, presumed minimal change disease, in a 6-year-old girl — fourth relapse in 12 months, second during taper. Relapse is confirmed by 5 consecutive days of 3–4+ proteinuria, a urine protein/creatinine ratio of 6.8 g/g, hypoalbuminemia, and clinical edema. Because two of these relapses occurred while on a taper, she now formally meets criteria for steroid dependence rather than simple frequent relapsing. This is a meaningful reclassification: it changes the treatment question from "how do we treat this relapse" to "how do we stop relying on steroids."

Kidney function is entirely preserved with a normal creatinine and eGFR, normal complements, no hematuria, and no hypertension at baseline — all consistent with minimal change disease and a favorable long-term renal prognosis. Biopsy remains unindicated. Progression to CKD is not the concern here; *treatment toxicity is*.

Cumulative corticosteroid toxicity is now the dominant clinical problem, and it is the reason to change course. Four courses in 12 months have produced converging evidence of harm: height percentile falling from the 50th to the 34th with velocity dropping to 4.2 cm/year, weight percentile rising from the 55th to the 78th, cushingoid facies, abdominal striae, blood pressure creeping to the 88th percentile, vitamin D insufficiency with bone-health implications, and — not to be dismissed — a real psychosocial cost, with school absence and emerging social withdrawal related to her appearance. In a growing child, linear growth suppression is not a cosmetic issue; it is a hard endpoint. Continuing to treat each relapse with steroids alone would predictably worsen all of these.

Non-immunity to varicella with a recent household exposure — a time-critical safety issue. Varicella IgG is

negative, immunization is incomplete at one dose, and there was a household exposure three weeks ago. Disseminated varicella in a child on high-dose immunosuppression is potentially life-threatening. The exposure window has largely passed without symptoms, which is reassuring, but this materially shapes both the immediate plan and the choice and timing of any steroid-sparing agent. Live vaccination cannot be given during active immunosuppression, creating a genuine sequencing problem that needs deliberate planning rather than deferral.

Relapse-associated risks requiring active vigilance: spontaneous bacterial peritonitis (abdomen benign today, but the family must know the warning signs), thromboembolism from urinary antithrombin loss (no current features), and hypovolemia if diuresis is pursued too aggressively against a low intravascular volume. **Prematurity at 34 weeks** is a minor additional consideration for lifelong renal reserve and supports long-term follow-up even after remission is durable.

P PLAN

Kidney function monitoring:

Repeat urine protein/creatinine ratio, serum albumin, and basic metabolic panel in 1 week, then every 2 weeks until stable remission. Continue daily home dipstick monitoring with the diary — the mother's log directly enabled early detection of this relapse and she was commended for it. Growth to be plotted at every visit, with height velocity as a tracked outcome, not an incidental measurement.

CKD or AKI management plan:

No CKD or AKI present — kidney function is normal and expected to remain so. Continue strict nephrotoxin avoidance, particularly NSAIDs. Long-term nephrology follow-up will continue even after durable remission, given prematurity and cumulative disease burden.

Blood pressure management:

Blood pressure at the 88th percentile is elevated for age but below the stage 1 hypertension threshold, and is likely steroid- and volume-related. Begin home blood pressure monitoring with a correctly sized pediatric cuff — cuff supplied and technique demonstrated to the mother today. Recheck at each visit. Reinforce sodium restriction during relapse. No antihypertensive indicated at present; if readings persist above the 95th percentile after remission and steroid reduction, ambulatory blood pressure monitoring would be the next step.

Proteinuria or hematuria management:

Nephrotic-range proteinuria is the relapse itself and will be treated with induction therapy below. No ACE inhibitor or ARB indicated in steroid-sensitive disease with normal function — these are reserved for steroid-resistant or persistently proteinuric cases. No serologic re-workup needed given normal complements. Biopsy and genetic testing remain unindicated unless steroid resistance emerges.

UTI or urinary tract management:

No UTI — urine culture negative and no urinary symptoms beyond relapse-related enuresis, which will resolve with remission. No antibiotic prophylaxis, no urology referral, and no further urinary tract imaging indicated.

Nephrotic syndrome or glomerular disease management:

Induce remission: prednisolone 60 mg/m²/day (40 mg daily for her body surface area) as a single morning dose until urine protein is negative or trace for 3 consecutive days, then transition to alternate-day dosing with a slow taper — deliberately slower than her previous tapers, which is where she has twice relapsed. **Steroid-sparing therapy — the key decision of this visit:** she now meets clear criteria, and the accumulating growth and metabolic toxicity makes continuing steroid monotherapy indefensible. Discussed levamisole, mycophenolate mofetil, calcineurin inhibitors, and rituximab with the mother in detail, covering efficacy, monitoring burden, and side-effect profiles. Recommending **mycophenolate mofetil** as first-line steroid-sparing therapy given its favorable balance of efficacy and tolerability in children, absence of nephrotoxicity (unlike calcineurin inhibitors), and oral administration. To be initiated once remission is achieved, with baseline CBC and LFTs and monitoring every 2 weeks initially. **Edema management:** conservative — sodium restriction and monitoring rather than diuretics, given only mild edema and the hypovolemia risk in nephrosis; furosemide reserved for symptomatic or severe edema and, if used, with albumin cover. **Infection precautions:** family educated in explicit detail on spontaneous bacterial peritonitis — fever, abdominal pain, or vomiting warrants same-day assessment, not watchful waiting. **Thrombosis precautions:** encourage mobilization and adequate hydration; avoid prolonged immobility; report calf pain or unilateral swelling.

Volume and electrolyte management:

No fluid restriction — she is drinking appropriately and restriction risks hypovolemia. Sodium restriction to approximately 2 g/day during relapse, with practical guidance provided given the steroid-driven appetite. Mild hyponatremia at 134 is dilutional and requires no specific treatment; it will correct with remission. No potassium or phosphorus restriction. Repeat electrolytes with the 1-week labs.

Growth and nutrition plan:

Pediatric renal dietitian referral placed — focused on managing steroid-induced appetite, maintaining sodium restriction palatably for a 6-year-old, and ensuring adequate protein and calcium without excess calories. Growth velocity to be formally tracked and plotted at every visit. If height velocity does not recover within 6–12 months of steroid reduction, pediatric endocrinology referral for growth assessment. Continue calcium and vitamin D; increase cholecalciferol given the level of 22 ng/mL.

Anemia and CKD-mineral bone disease management:

No anemia — hemoglobin normal, no intervention. For bone health under cumulative steroid exposure: increase vitamin D supplementation to 1,000 IU daily and continue dietary calcium optimization; recheck vitamin D in 3 months. DEXA not routinely indicated at her age but would be considered if steroid exposure continues to accumulate despite steroid-sparing therapy.

Medication review:

Full reconciliation with the mother. Reinforced absolute NSAID avoidance and confirmed paracetamol as the appropriate antipyretic and analgesic. Continue as-needed salbutamol for asthma. All medications dosed by weight and reviewed. Written medication schedule provided, with the prednisolone taper laid out day by day on a printed calendar — the previous tapers were confusing to follow and this may have contributed to inconsistency.

Dialysis or transplant planning:

Not applicable and explicitly discussed for reassurance — the mother had read about kidney failure online and was anxious. Explained clearly that steroid-sensitive minimal change disease very rarely progresses to kidney failure, that Amara's kidney function is entirely normal, and that most children outgrow the condition by adolescence.

Referral plan:

Pediatric renal dietitian (placed today). **Infectious disease consultation — priority** for varicella non-immunity management: guidance on post-exposure prophylaxis eligibility, and, critically, on sequencing varicella vaccination before mycophenolate initiation if a safe window exists, since live vaccines are contraindicated during immunosuppression. Pediatric endocrinology deferred pending growth response. School liaison letter provided to support absence and to arrange bathroom access without restriction. Social work referral offered for caregiver support and discussed with the mother, who accepted.

Patient and caregiver education:

Extended discussion with the mother, with Amara included at an age-appropriate level. Explained the shift from "frequently relapsing" to "steroid-dependent" and, in plain terms, why adding a second medication is intended to *reduce* total medication harm rather than add to it — the mother's initial reaction was reluctance to "add another drug," and this reframing was important. Reviewed that the cushingoid appearance and appetite are steroid effects that will improve as the dose falls. Warning signs reviewed and provided in writing: fever, abdominal pain, vomiting, breathing difficulty, leg swelling or pain, reduced urine output, or severe headache — all warranting same-day contact. Home dipstick technique reconfirmed and the diary praised. Addressed Amara directly about her worries regarding her appearance and about missing school.

F FOLLOW-UP

Telephone check in 3 days to confirm the dipstick is trending toward negative and that no red-flag symptoms have developed. Clinic review in 1 week with repeat urine protein/creatinine ratio, albumin, and electrolytes, and to confirm remission. Once remission is established, a dedicated visit in 2–3 weeks to initiate mycophenolate mofetil with baseline CBC and LFTs — timed after infectious disease input on varicella vaccination sequencing. Fortnightly monitoring during mycophenolate initiation, then monthly. Growth and blood pressure plotted at every visit. Dietitian within 2 weeks. Infectious disease within 1 week given the vaccination-timing question. Immediate return for fever, abdominal pain, vomiting, breathlessness, or reduced urine output.

TD TIME DOCUMENTATION, IF APPLICABLE

TOTAL TIME SPENT

60 minutes

CAREGIVER COUNSELING TIME

30 minutes (steroid-sparing decision, toxicity, varicella risk, warning signs)

COUNSELING / COORDINATION OF CARE TIME

40 minutes

RECORDS REVIEW TIME, IF APPLICABLE

15 minutes (growth charts, relapse history, immunization record)

TB BILLING CONSIDERATIONS

E/M LEVEL

99215 — Established patient, high complexity

PROCEDURE CODE(S), IF APPLICABLE

None this visit

BASIS FOR BILLING

Medical Decision Making (high) — also supported by total time of 60 minutes

PRIMARY PEDIATRIC RENAL DIAGNOSIS CODE + DESCRIPTION

N04.0 — Nephrotic syndrome with minor glomerular abnormality (steroid-dependent, relapsed)

SECONDARY DIAGNOSIS CODE(S)

E27.0 (steroid-induced adrenal/metabolic effect); R62.52 (short stature / growth failure); E55.9 (vitamin D deficiency); Z23 (immunization needed); Z20.820 (contact with and exposure to varicella); P07.36 (preterm newborn, 34 weeks — history)

SO SIGNATURE

PROVIDER NAME	CREDENTIALS	SPECIALTY	DATE	TIME
Priyanka Raghunathan, MD	MD, FAAP	Pediatric Nephrology	07/14/2026	10:20