NGN NCLEX 2026 · RENAL / GENITOURINARY SYSTEM

Nephrotic *Syndrome*

When the glomerular filter loses its grip — massive protein loss, sweeping edema, and a cascade of cardiovascular and immunologic risk.

SYSTEM · RENAL | HIGH-YIELD TOPIC | PRIORITY: FLUID VOLUME + INFECTION | TEST PLAN: PHYSIOLOGICAL ADAPTATION

SOURCE DISCLOSURE

Nephrotic syndrome was not present in the uploaded reference notes. Content was synthesized from standard NCLEX references — Saunders Comprehensive Review for the NCLEX-RN (Silvestri, 9th ed.), ATI Adult Medical Surgical Nursing (RN 11.0), Lippincott Illustrated Reviews: Pharmacology — together with the KDIGO 2021 Clinical Practice Guideline for the Management of Glomerular Diseases.

1 Definition & Overview

The classic clinical tetrad — what defines the syndrome and why it matters.

Nephrotic syndrome is a **glomerular disorder** — not a single disease — in which damage to the glomerular filtration barrier allows large amounts of **plasma protein** (primarily albumin) to leak into the urine. The body cannot replace what it loses, and the resulting drop in plasma oncotic pressure drives fluid out of the vasculature into tissues.

01
Proteinuria
`> 3.5 g / 24 hr`
Massive urinary protein loss; urine appears foamy / frothy.

02
Hypoalbuminemia
`Albumin < 3.0 g/dL`
Serum protein depleted faster than the liver can synthesize.

03
Edema
`Periorbital → Anasarca`
Generalized; worst in morning around eyes, evening in feet.

04
Hyperlipidemia
`↑ Cholesterol & ↑ LDL`
Compensatory hepatic lipid synthesis; lipiduria possible.

WHY IT MATTERS ON THE NCLEX

Nephrotic syndrome is a **fluid volume + infection + clotting triple-threat.** The exam tests your ability to prioritize edema management, recognize a child or adult at risk for infection from immunoglobulin loss, and anticipate thromboembolism from urinary loss of antithrombin III. Differentiating it from nephritic syndrome (hematuria + HTN dominant) is a recurring trap.

2 Pathophysiology

How a damaged glomerular filter drives every sign and symptom.

1 Glomerular injury
Immune-mediated, structural, or systemic disease damages podocytes & basement membrane.

↓

2 Loss of filter selectivity
The negatively charged barrier no longer repels plasma proteins; albumin slips through.

↓

3 Massive proteinuria → hypoalbuminemia
Urinary albumin loss outpaces hepatic production; serum albumin drops.

↓

4 ↓ Plasma oncotic pressure
Without albumin to hold water inside vessels, fluid shifts to interstitial space.

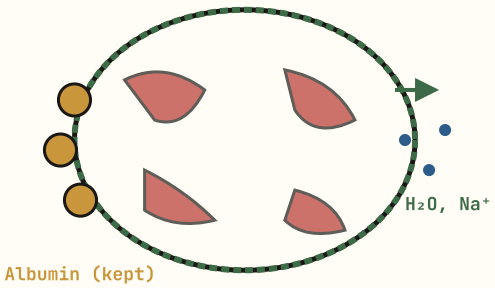
↓

5 Generalized edema + activation of RAAS
Intravascular volume drops → kidneys retain Na^+ and H_2O → edema worsens.

↓

6 Compensatory hyperlipidemia + hypercoagulability + ↓ immunity
Liver upregulates lipid synthesis. Urinary loss of antithrombin III → clot risk. Loss of IgG → infection risk.

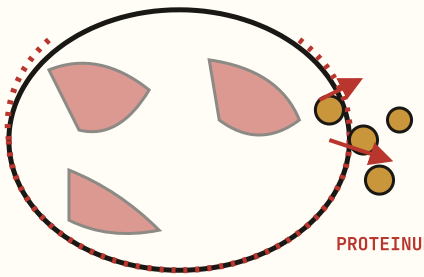
Healthy Glomerulus



Filter intact · Proteins retained

Negative charge + intact podocytes block large proteins.

Nephrotic Glomerulus



Damaged filter · Proteins LEAK out

Loss of charge + podocyte injury → proteins escape into urine.

Primary & Secondary Causes

PRIMARY (KIDNEY-ONLY)	SECONDARY (SYSTEMIC)
Minimal Change Disease — most common in **children**	Diabetes mellitus (diabetic nephropathy)
Focal Segmental Glomerulosclerosis (FSGS)	Systemic Lupus Erythematosus (lupus nephritis)
Membranous Nephropathy — most common in **adults**	Amyloidosis, multiple myeloma
Membranoproliferative Glomerulonephritis	Infections: HIV, Hepatitis B/C, malaria

- Drugs: NSAIDs, gold, penicillamine

3 Signs & Symptoms

Early presentation → progressive findings → red flags requiring immediate intervention.

Early / Mild

Onset

- **Periorbital edema** — worst on waking (the hallmark first sign)
- **Foamy / frothy urine** — classic teaching cue for proteinuria
- Unexplained weight gain (fluid)
- Fatigue, malaise, mild anorexia
- Pallor (anemia of CKD developing)
- Mild ankle edema by end of day

Late / Severe

Progression

- **Anasarca** — generalized body-wide edema
- **Ascites & scrotal/labial edema**
- Pleural effusion → dyspnea, decreased breath sounds
- Hypertension (variable; depends on cause)
- Decreased urine output, dark or tea-colored urine
- Hyperlipidemia, lipiduria (oval fat bodies)
- Skin breakdown over edematous areas
- Malnutrition signs (muscle wasting masked by edema)

⚠ Red Flag Findings — Notify Provider Immediately

THROMBOEMBOLISM

Sudden chest pain, dyspnea, calf swelling, neuro changes. Loss of antithrombin III drives clot risk — **DVT/PE or renal vein thrombosis**.

INFECTION / SEPSIS

Fever, hypotension, lethargy, abdominal tenderness. Loss of IgG → **spontaneous bacterial peritonitis** is classic in children with ascites.

ACUTE KIDNEY INJURY

Oliguria < 0.5 mL/kg/hr, rising BUN/creatinine, hyperkalemia, metabolic acidosis — hypovolemia or progression to renal failure.

4 Priority Nursing Interventions

NCLEX prioritization through the ABC + Safety lens.

A — Airway / Breathing
Assess for pleural effusion & ascites compromising ventilation. Position semi-Fowler's. Pulse ox q4h.

B — Circulation / Volume
Strict I&O. Daily weights at same time, same scale. Monitor BP — risk for both hypovolemia and HTN.

C — Infection & Skin
Hand hygiene, visitor screening, skin integrity checks. Loss of IgG creates immunocompromise.

ASSESS	**Edema** — periorbital, sacral, scrotal, ankles, abdominal girth daily. Grade pitting edema (1+ to 4+). Compare findings shift-to-shift.
MONITOR	**Daily weight** at the same time, same scale, same clothing — **1 kg = 1 L of fluid**. The single most reliable indicator of fluid status.
MONITOR	**Strict I&O** every shift. Urine dipstick for protein every void in acute phase. Watch for oliguria (< 30 mL/hr in adults; < 1 mL/kg/hr in children).
MONITOR	**Labs** — serum albumin, total protein, BUN/creatinine, electrolytes (esp. K^+ with diuretics), lipid panel, 24-hr urine protein, urinalysis.
ADMINISTER	**Corticosteroids** (prednisone) as ordered — the cornerstone of therapy. Give **with food**, do **NOT abruptly stop**, monitor blood glucose.
ADMINISTER	**Loop diuretics** (furosemide) and/or **salt-poor IV albumin** as ordered. Albumin may be given before furosemide to pull fluid back into vessels.
PREVENT	**Skin breakdown** — reposition q2h, pressure-relief mattress, support edematous extremities with pillows, meticulous skin care, no IM injections in edematous tissue.
PREVENT	**Infection** — strict hand hygiene, limit visitors with illness, monitor temp q4h, no live vaccines while on high-dose steroids, prompt culture if febrile.
PREVENT	**Thromboembolism** — encourage ambulation/ROM, SCDs, anticoagulation per order. Assess for Homan's sign, calf tenderness, sudden dyspnea.
ADMINISTER	**Diet:** moderate (not low) protein 0.8–1.0 g/kg/day, sodium restriction (1–2 g/day), fluid restriction if hyponatremic, low saturated fat.
EDUCATE	Teach **steroid safety**: take with food, never stop abruptly, recognize Cushingoid changes, infection precautions, weight diary, and what symptoms to report.

5 Pharmacology

The four drug classes that drive nephrotic syndrome management.

DRUG / CLASS	MECHANISM	KEY SIDE EFFECTS	NURSING CONSIDERATIONS
Prednisone CORTICOSTEROID · FIRST-LINE	Suppresses immune-mediated glomerular inflammation & reduces proteinuria.	Hyperglycemia, immunosuppression, weight gain, mood changes, osteoporosis, peptic ulcer, Cushingoid features, ↑ infection risk.	Give **with food**. **NEVER stop abruptly** — taper. Monitor glucose, weight, BP, signs of infection. No live vaccines.
Furosemide LOOP DIURETIC	Inhibits $Na^+/K^+/2Cl^-$ reabsorption in loop of Henle → diuresis & edema reduction.	**Hypokalemia**, hyponatremia, ototoxicity (esp. IV push), dehydration, hypotension, hyperuricemia.	Monitor K^+, daily weight, BP. Encourage K^+-rich foods. Push IV **slowly** (over 1–2 min) to prevent ototoxicity.
Lisinopril / Losartan ACE-I / ARB	Reduces glomerular pressure & proteinuria; renoprotective; lowers BP.	**Hyperkalemia**, dry cough (ACE-I), angioedema, hypotension, ↑ creatinine (transient acceptable rise).	Monitor K^+ & creatinine. Hold for SBP < 90 unless ordered otherwise. Teach to report swelling of face/tongue.
Albumin 25% COLLOID · PLASMA EXPANDER	Pulls fluid from interstitium into vasculature — restores oncotic pressure.	Fluid overload → **pulmonary edema**, HTN, allergic reaction, headache.	Monitor lung sounds, BP, JVD **during and after** infusion. Often given **before furosemide** to mobilize edema.
Atorvastatin STATIN · HMG-CoA INHIBITOR	Lowers cholesterol synthesis to address persistent hyperlipidemia.	Myopathy, **rhabdomyolysis**, ↑ LFTs, GI upset.	Teach to report muscle pain/weakness/dark urine. Monitor LFTs and CK. Take in the evening.
Enoxaparin / Warfarin ANTICOAGULANT (HIGH-RISK PT)	Prevents thromboembolism in patients with serum albumin < 2.0–2.5 g/dL or prior clot.	Bleeding, HIT (heparin), bruising, hematuria.	Monitor PT/INR (warfarin) or anti-Xa. Assess for bleeding. Bleeding precautions; soft toothbrush, no IM injections.
Cyclophosphamide / Tacrolimus IMMUNOSUPPRESSANT · STEROID-RESISTANT	Used when steroids fail or for relapsing disease; suppresses immune activity.	Bone marrow suppression, **hemorrhagic cystitis** (cyclophosphamide), nephrotoxicity (tacrolimus), infection.	Push fluids with cyclophosphamide (prevent cystitis). Monitor CBC, drug levels. Strict infection precautions.

6 NCLEX Test Traps

The wrong-answer logic the test loves — and the right thinking beside it.

TRAP	RIGHT
Confusing nephr**OTIC** with nephr**ITIC** — selecting "hematuria + HTN" as the priority finding.	Nephr**OTIC** = **O**edema + Pr**O**teinuria + Hyp**O**albuminemia. Nephr**ITIC** = Inflammation, hematuria, HTN.
Restricting protein severely to "protect the kidneys."	**Moderate** protein (0.8–1.0 g/kg/day). Severe restriction worsens malnutrition. They're losing protein — don't starve them of it.
Stopping prednisone when the child "looks better."	Steroids must be **tapered**. Abrupt withdrawal → adrenal crisis & relapse. Teach families this in every discharge plan.
Identifying weight gain as a non-priority concern.	Daily weight is the **#1 indicator of fluid status**. A 2–3 lb overnight gain = fluid shift, not nutrition.
Picking "monitor for hyperkalemia" as priority while patient is on furosemide alone.	Loop diuretics cause **HYPO**kalemia. Hyperkalemia is the concern with ACE-I/ARB/potassium-sparing diuretics — know the combo.
Giving an IM injection in an edematous thigh because it's the largest site available.	**Never inject into edematous tissue** — absorption is unpredictable and breakdown risk is high. Use a non-edematous site.
Administering a live vaccine (MMR, varicella) to a child on high-dose prednisone.	**No live vaccines** on high-dose steroids or immunosuppressants. Inactivated vaccines (flu shot) are OK.

7 Patient & Family Education

Discharge teaching that prevents readmission and relapse.

D Diet & Fluids

- → Limit sodium to 1–2 g/day; **no added salt**, read labels, avoid processed foods, deli meat, canned soups
- → Moderate protein at meals — lean meat, eggs, fish, dairy
- → Choose low-saturated-fat foods (heart-healthy)
- → Fluid restriction *only if ordered* — usually with hyponatremia
- → No grapefruit juice with tacrolimus or statins

A Activity & Monitoring

- → Daily weight **same time, same scale, same clothing** — report > 2 lb gain in 24 hr
- → Test urine for protein at home (dipstick) — track in a daily log
- → Balance rest with activity; avoid prolonged immobility (clot risk)
- → Elevate edematous extremities when sitting
- → Keep all follow-up labs & nephrology appointments

M Medication Safety

- → **NEVER stop prednisone abruptly** — always taper as prescribed
- → Take steroids **with food** in the morning to mimic natural cortisol
- → Wear a medical alert ID stating "long-term steroid use"
- → Carry an updated med list; avoid NSAIDs (worsen kidney injury)
- → Get the seasonal flu shot and pneumococcal vaccine (inactivated only)

! When to Call the Provider

- → Sudden weight gain > 2 lb in 24 hr or worsening edema
- → Fever > 100.4°F, chills, sore throat — any sign of infection
- → Decreased urine output, dark cola-colored urine, or new blood in urine
- → Calf pain, sudden chest pain, shortness of breath (DVT/PE)
- → Persistent abdominal pain (peritonitis — especially in children with ascites)

8 Memory Boosters

Mnemonics & pattern-recognition cues that stick under exam pressure.

The Four Hallmarks

P A L E

- **P** — **Proteinuria** (massive, > 3.5 g/day)
- **A** — **Albumin** low (hypoalbuminemia)
- **L** — **Lipids** high (hyperlipidemia)
- **E** — **Edema** (periorbital → anasarca)

OTIC vs ITIC

O = O O O

- **O** — nephr**OTIC** has triple-O findings:
  - **O** edema · pr**O** teinuria · hyp**O** albuminemia
- **I** — nephr**ITIC** = **I** nflammation, hematur**I** a, HTN

If the question stem says "tea-colored urine + HTN" → nephr**ITIC**, not nephrotic

// QUICK ASSOCIATIONS & PATTERN CUES

- **Foamy urine** → proteinuria (the classic teaching cue)
- **Periorbital edema in a child** → minimal change disease
- **Adult + heavy proteinuria** → membranous nephropathy
- **Diabetic + proteinuria** → diabetic nephropathy
- **Sudden flank pain + hematuria** → renal vein thrombosis
- **Ascites + fever in a child** → spontaneous bacterial peritonitis
- **Dipstick 3+ or 4+ protein** → nephrotic-range
- **Albumin then furosemide** → pulls fluid in, then off

9 NCJMM Clinical Judgment Scenario

Six-step Next Generation Clinical Judgment Measurement Model walkthrough.

CASE SCENARIO

A 6-year-old child is admitted with a 4-day history of progressive periorbital and lower-extremity swelling. The mother reports the child's urine has "looked bubbly" in the toilet for the past week and the child has gained 5 lb in 5 days despite a poor appetite.

Vital signs: T 98.9°F · HR 118 · RR 24 · BP 102/68 · SpO_2 96% on room air. **Weight today:** 24 kg (baseline 21.7 kg).

Labs: Serum albumin 1.8 g/dL · Total cholesterol 412 mg/dL · 24-hr urine protein 4.2 g · BUN 22 mg/dL · Cr 0.6 mg/dL · Na^+ 132 · K^+ 4.1. **Urinalysis:** 4+ protein, no blood, no casts.

STEP 1

Recognize Cues

Identify which client findings are clinically significant.

Cues:

Periorbital + LE edema · 5 lb / 5 day weight gain · foamy urine · albumin 1.8 g/dL · 24-hr protein 4.2 g · cholesterol 412 · 4+ urine protein · mild hyponatremia.

STEP 2

Analyze Cues

Cluster cues and link them to a clinical condition.

Analysis:

The tetrad — massive proteinuria, hypoalbuminemia, edema, hyperlipidemia — in a child fits nephrotic syndrome (most likely minimal change disease). No hematuria/HTN rules out nephritic.

STEP 3

Prioritize Hypotheses

Rank what's most pressing right now.

Top priorities:

(1) Fluid volume excess (edema) with paradoxical intravascular depletion ·
(2) Infection risk from IgG loss + future steroids ·
(3) Thromboembolism risk from antithrombin III loss.

STEP 4

Generate Solutions

Plan evidence-based interventions.

Plan:

Daily weight + strict I&O · low-Na diet · prednisone with food · monitor BP/glucose · skin protection · infection precautions · IV salt-poor albumin then furosemide if severe edema · parent education on steroid taper.

STEP 5

Take Action

Implement the highest-priority interventions first.

Actions:

Obtain weight in gown only · strict I&O begun · 1-g sodium diet ordered · prednisone 2 mg/kg/day initiated · daily urine dipstick · skin assessment q shift · teach mom weight log and S/Sx of infection.

STEP 6

Evaluate Outcomes

Measure whether the plan is working.

Evaluation:

Expected within 1–2 weeks of steroids: ↓ urine protein (negative or trace), ↑ serum albumin > 2.5 g/dL, weight returning to baseline, edema resolving, no signs of infection or thrombosis. If no response in 4–8 weeks → steroid-resistant; reassess.