Update in Internal Medicine 2026

Saturday, May 2 • 8 a.m. – 4 p.m.

UT Southwestern Medical Center, T. Boone Pickens Medical Education & Conference Center

Navigating Glomerulonephritis for the Internist

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Disclosures

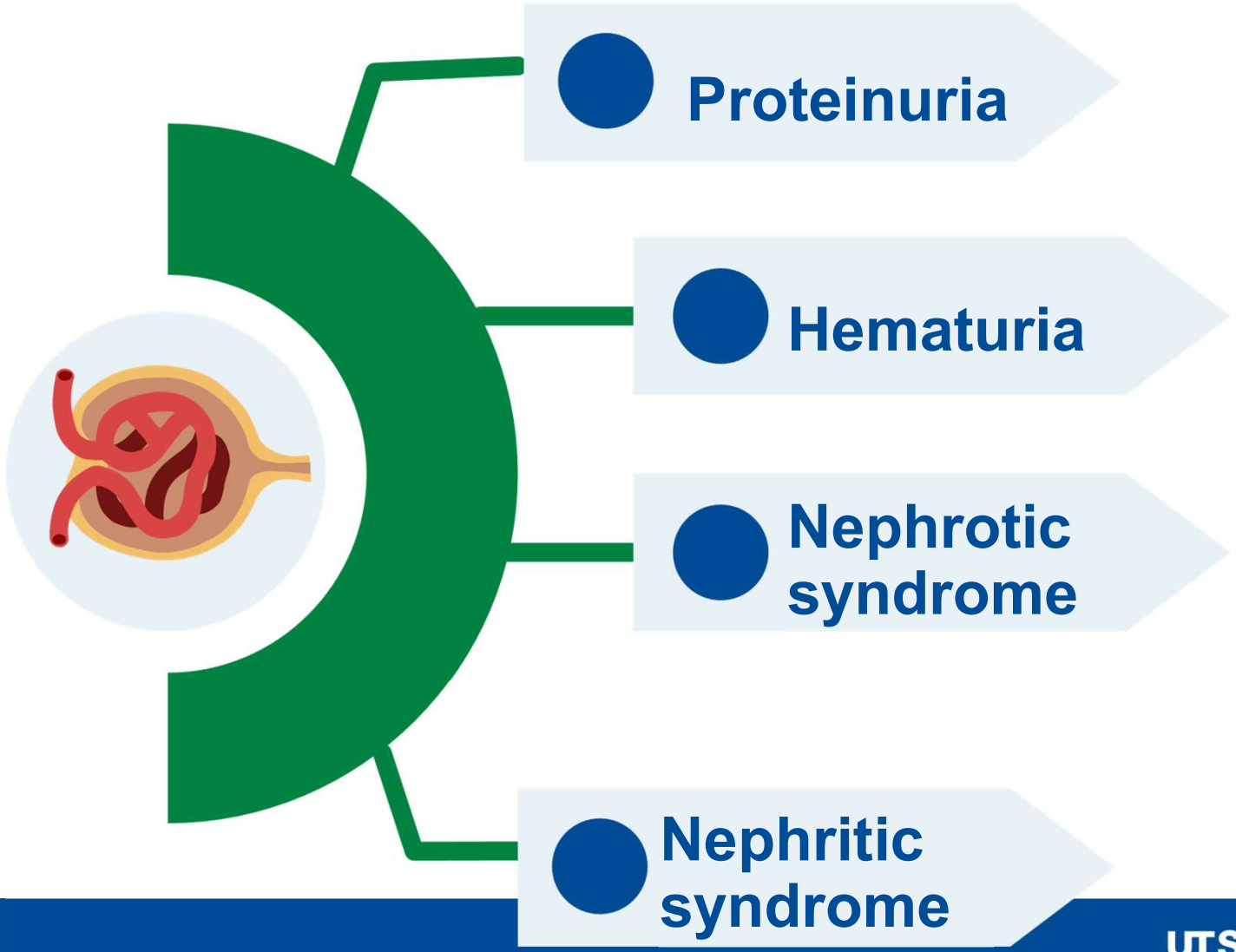
- None

Objectives

- Recognize hematuria and proteinuria as the key clinical entry points to suspected glomerular disease.
- Use urinary patterns and proteinuria severity to assess underlying mechanism, trajectory, and urgency.
- Apply nephritic and nephrotic syndromes as clinical phenotypes, while understanding their overlap and limitations.
- Differentiate mechanisms of acute kidney injury in nephritic versus nephrotic presentations
- Identify targeted history and physical exam clues associated with specific glomerular diseases.
- Initiate appropriate initial evaluation while awaiting definitive diagnosis.
- Recognize red-flag features that require urgent nephrology referral and escalation of care.
- Implement evidence-based supportive care strategies for the management of patients with glomerular disease.

Glomerulonephritis (GN)

- Heterogeneous group of disorders
- Can occur:
  - Restricted to the kidneys
  - Part of a multisystem disease
  - Manifestation of a malignancy
  - Manifestation of monoclonal gammopathy
  - Secondary to external factors
    - Infections
    - Drugs
- Presents as many different patterns or syndromes
- Early presentations are often urine-based, not creatinine based
- Clinical patterns help assess mechanism, severity, and urgency


Proteinuria
Hematuria
Nephrotic syndrome
Nephritic syndrome

Proteinuria

- Proteinuria suggests a predominant permeability defect

Proteinuria Level	Approximate Range	Clinical Implications
Normal to minimal	< 150 mg/day	Functional proteinuria; usually benign unless persistent or associated with hematuria
Low-grade / sub-nephrotic	150 mg – < 3.5 g/day	Does not imply mild disease; trajectory and sediment matter
Nephrotic-range	≥ 3.5 g/day	High risk for AKI, thrombosis, infection; potential urgent evaluation

Diferential Diagnosis	
Functional / hemodynamic proteinuria	Exercise, fever, orthostatic proteinuria (typically transient)
Metabolic / vascular disease	Diabetes, hypertension, obesity-related glomerulopathy
Nephrotic syndromes	Membranous nephropathy, FSGS, minimal change disease may lack hematuria
Tubulointerstitial disease	Low-grade proteinuria from drugs or chronic interstitial nephritis

Sethi et al., The Lancet 2022; KDIGO GN Guideline 2012

Hematuria

- Hematuria alone - think location: structural vs glomerular

Diferential Diagnosis	
Non-glomerular source	Stones, infection, malignancy, trauma
Benign glomerular disorders	Thin basement membrane disease
Early glomerulonephritis	Proteinuria may be minimal initially; creatinine often normal

- Clues suggesting glomerular origin
  - Dysmorphic red blood cells
  - Red blood cell casts
  - Persistent microscopic hematuria
  - Family history of kidney disease

Udomkarnjananun et al., Asian Biomedicine 2025

Hematuria + Proteinuria Combined

Hematuria and proteinuria **together** strongly suggests **glomerular** involvement

Sethi et al., The Lancet 2022

Nephritic Syndrome

Nephritic Syndrome

- Hematuria is the dominant feature
  - Often microscopic
  - May be macroscopic
- Active urine sediment
  - Dysmorphic red blood cells
  - Red blood cell casts

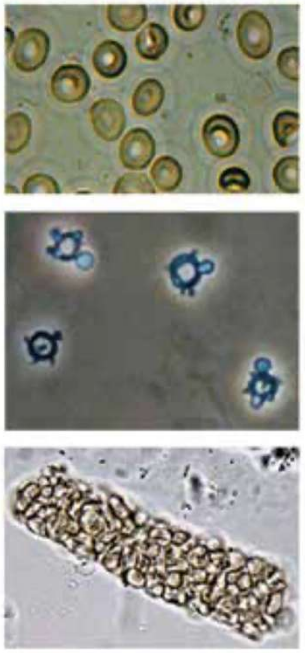


Figure 1. Tedesco et al. 2017.

Udomkarnjananun et al., Asian Biomedicine 2025

Nephritic Syndrome

- Proteinuria
  - Usually sub-nephrotic
  - Degree does not reliably predict severity
- Decline in kidney function
  - Acute or subacute rise in creatinine
- Systemic features may be present
  - Hypertension
  - Edema (variable)
  - Constitutional or inflammatory symptoms

- Reduced glomerular filtration from inflammatory injury
- Crescent formation (rapidly progressive GN)
- Tubulointerstitial injury
- Hemodynamic effects and intraglomerular vasoconstriction
- Secondary contributors
  - severe hypertension
  - volume overload
  - superimposed nephrotoxic exposures

Sethi et al., The Lancet 2022

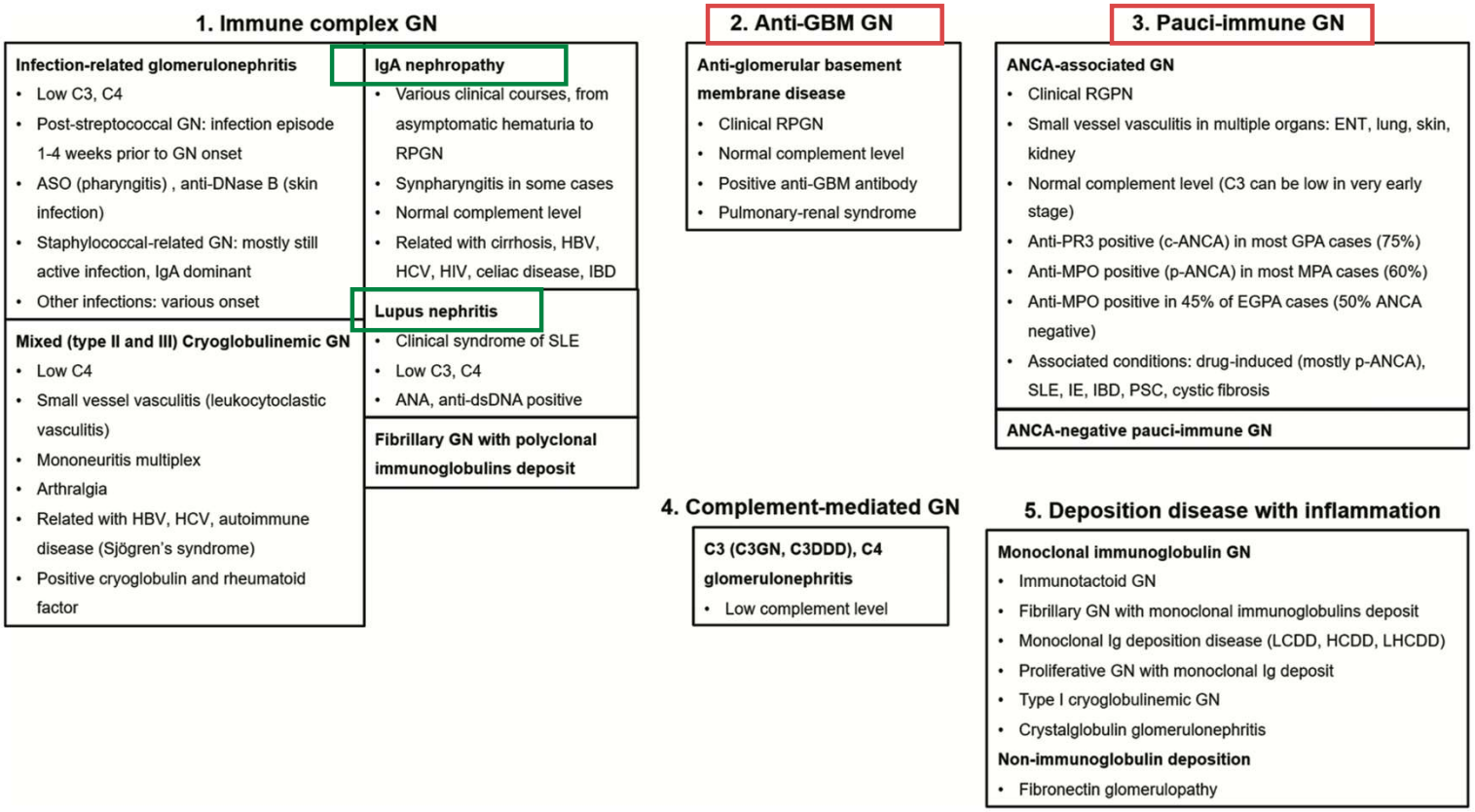
Nephritic Syndrome

- Proteinuria
  - Usually sub-nephrotic
  - Degree does not reliably predict severity
- Decline in kidney function
  - Acute or subacute rise in creatinine
- Systemic features may be present
  - Hypertension
  - Edema (variable)
  - Constitutional or inflammatory symptoms

- Mechanism: increase intravascular hydrostatic pressure leading to activation of renin-angiotensin-aldosterone system
- Manifestation: Dependent edema (dorsum of the feet and pretibial edema)

Udomkarnjananun et al., Asian Biomedicine 2025

Nephritic Syndrome



Udomkarnjananun et al., Asian Biomedicine 2025

Nephrotic Syndrome

Nephrotic Syndrome

Table 2. Diagnostic Criteria for Nephrotic Syndrome

Factor	*Criteria*
Heavy proteinuria	Spot urine showing a protein-to-creatinine ratio of > 3 to 3.5 mg protein/mg creatinine (300 to 350 mg/mmol), or 24-hour urine collection showing > 3 to 3.5 g protein
Hypoalbuminemia	Serum albumin < 2.5 g per dL (25 g per L)*
Edema	Clinical evidence of peripheral edema
Hyperlipidemia (not required for diagnosis)	Severe hyperlipidemia, total cholesterol is often > 350 mg per dL (9.06 mmol per L)

*—Some experts use a cutoff of < 3.0 g per dL (30 g per L).

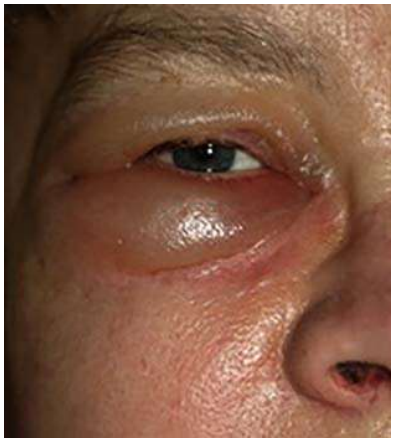
Adapted with permission from Hull RP, Goldsmith DJ. Nephrotic syndrome in adults. BMJ. *2008;336(7654):1185.*

Nephrotic Syndrome: Edema

Underfill Hypothesis

Hypoalbuminemia
Decreased oncotic pressure

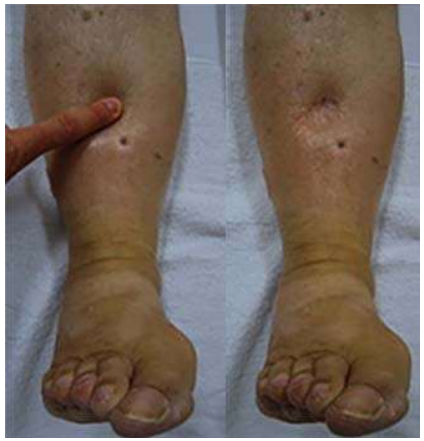
Manifestations: eyelids, genitalia, third spaces (pleural or peritoneal cavities)



Overfill Hypothesis

Renin angiotensive system activation: salt and water retention

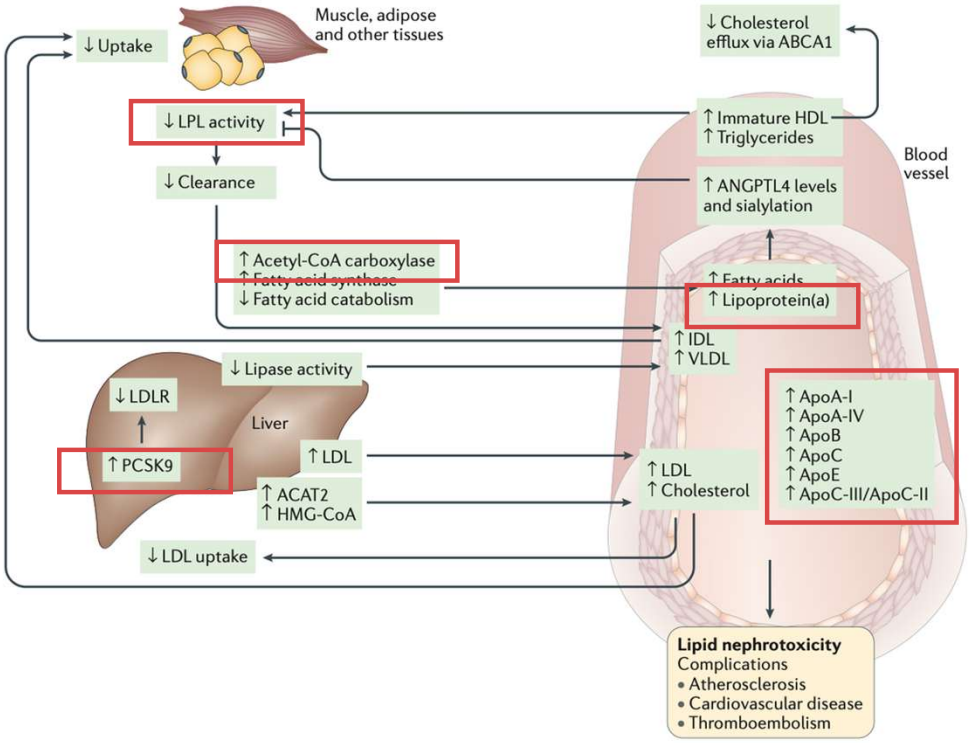
Manifestations: pretibial edema

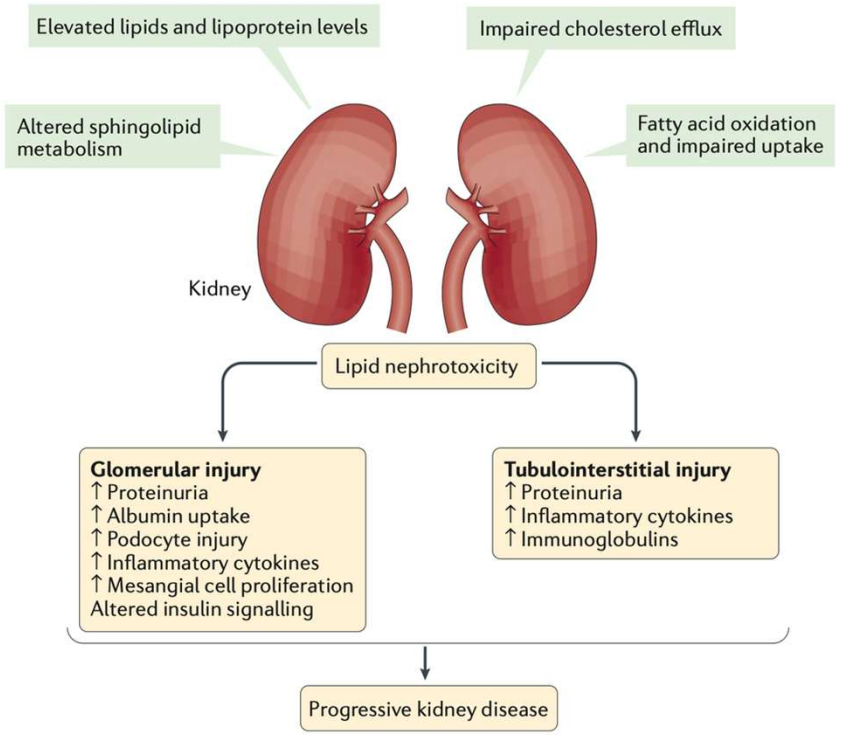


Images from: Welcome Cure

Udomkarnjananun et al., Asian Biomedicine 2025

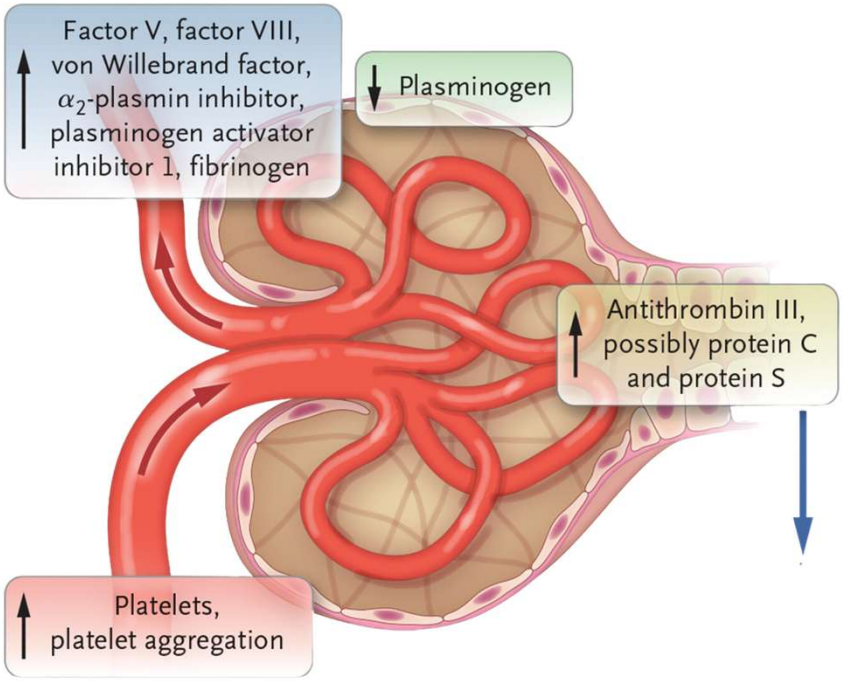
Nephrotic Syndrome: Hyperlipidemia

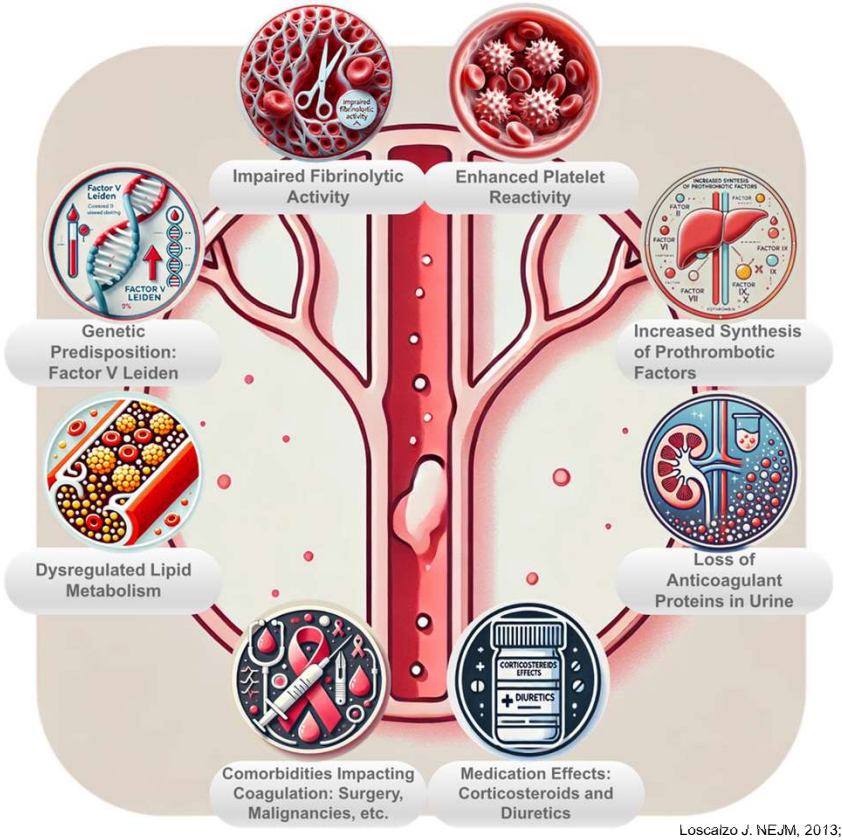




Agrawal et al.,Nature 2018.

Complications: Venous Thromboembolism (VTE)





Loscalzo J. NEJM, 2013;368:956-958.
Ghozloujeh ZG, et al. KI Reports. 2025.

Complications: Acute Kidney Injury

Reduced Effective Circulating Volume

- Severe hypoalbuminemia → decreased plasma oncotic pressure
- Fluid shifts into interstitial space → intravascular volume depletion
- Leads to:
  - Reduced renal perfusion
  - Pre-renal azotemia

Common contributors:

- Aggressive diuresis
- ACEi / ARB initiation in volume-depleted patients
- Gastrointestinal losses

Acute Tubular Injury

- Prolonged renal hypoperfusion
- Increased tubular exposure to filtered proteins
- Ischemia worsened by:
  - NSAIDs
  - Contrast
  - Sepsis

Sethi et al., The Lancet 2022; KDIGO GN 2012. Udomkarnjananun et al., Asian Biomedicine 2025

Complications: Acute Kidney Injury

Renal Vein Thrombosis

- Nephrotic syndrome is a hypercoagulable state
- Loss of anticoagulant proteins (e.g., antithrombin III)
- Most commonly associated with:
  - Membranous nephropathy

Clinical clues:

- Sudden rise in creatinine
- Flank pain
- Hematuria
- Asymmetric kidney size

Interstitial Edema and Renal Congestion

- Marked interstitial edema increases intrarenal pressure
- Leads to reduced filtration and tubular compression
- Especially relevant in:
  - Severe anasarca
  - Heart failure overlap

Superimposed Complications

- Infection (sepsis-associated AKI)
- Drug toxicity

Sethi et al., The Lancet 2022; KDIGO GN 2012. Udomkarnjananun et al., Asian Biomedicine 2025

Nephrotic Syndrome

1. Podocytopathies

Minimal change disease

- Pure nephrotic syndrome (no active sediment)
- Associated infection: TB, syphilis, viral hepatitis, HIV
- Associated neoplasms: hematologic malignancies (solid tumor less common)
- Associated drugs: NSAIDs, lithium, antibiotics, mercury, gold
- Associated with allergy or atopy (e.g., bee stings, pollen, house dust)
- Can present with AKI from hypoalbuminemia and leakage.

Focal segmental glomerulosclerosis

- Nephrotic syndrome ± RBC in urine/AKI
- Primary FSGS: idiopathic
- Genetic FSGS: familial, sporadic, syndromic
- Associated infection: HIV, CMV, viral hepatitis, SARS-COV-2, parvovirus
- Associated drugs: mTORi, CNI, heroin, lithium, interferon, direct-acting antiviral therapy
- Adaptive FSGS: obesity, hypertension, reflux nephropathy, diabetes

2. Immune complex disease

Membranous nephropathy

- Insidious onset (compared with podocytopathies)
- Nephrotic syndrome ± RBC
- Anti-PLA2R positive in primary MN
- Associated neoplasms: lung, renal, gastrointestinal tract, prostate, breast, nasopharynx (hematologic malignancy less common)
- Associated autoimmunity: LN, MCTD, sarcoidosis, IgG4-related disease, Sjögren syndrome, autoimmune thyroiditis
- Associated drugs: penicillamine, gold, NSAIDs
- Associated infection: HBV, HCV, Syphilis, HIV

3. Deposition disease without inflammation

Amyloidosis

- Extrarenal manifestation: shoulder-pad, tongue indentation, LVH, raccoon eyes, cardiac conduction defect, low voltage on limb leads and pseudo-infarct pattern, carpal tunnel syndrome
- Light chain (AL) amyloidosis: most common, with plasma cell clones
- AA amyloidosis: associated with chronic inflammation, autoimmune disease, infection, malignancy, genetics

Collagenofibrotic GN, Lipoprotein GN

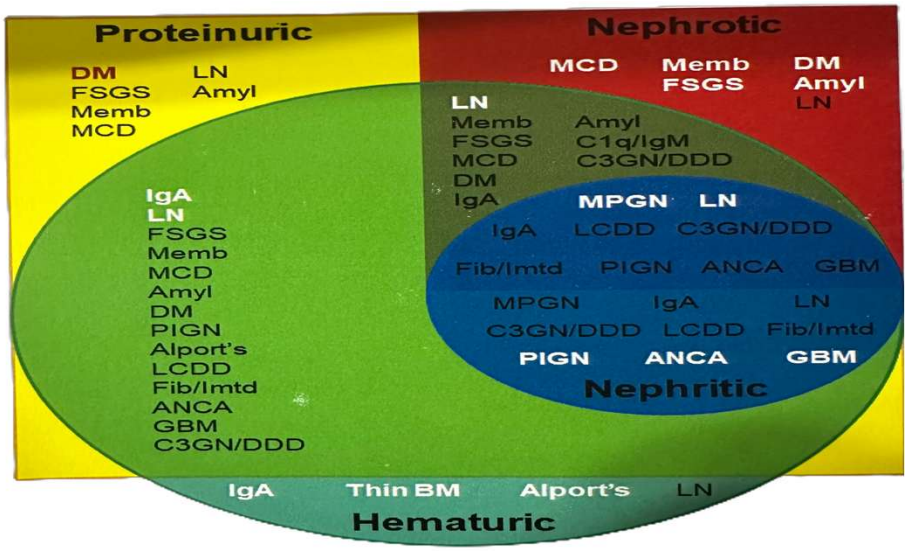
4. Diabetic nephropathy

- Diabetes mellitus
- Features that may raise suspicion for non-diabetic kidney disease
  - Rapid decline of eGFR (>5 mL/min/year)
  - No diabetic retinopathy or type I diabetes less than 5 years
  - Rapid or sudden onset of albuminuria
  - Active urinary sediments: RBC, WBC, or cast
  - Clinical features of another systemic infection or autoimmune disease
- Not all DKD present with nephrotic syndrome or nephrotic-range proteinuria, depends on severity, treatment, and time from DM onset

Udomkarnjananun et al., Asian Biomedicine 2025

Nephritic and nephrotic syndromes as clinical phenotypes

- “Nephritic” and “nephrotic” syndromes are pattern-based clinical phenotypes
- They reflect the dominant manifestation of glomerular injury:
  - Inflammation-predominant
  - Permeability-predominant
- These phenotypes are starting points, not diagnoses
- Overlap is common and expected



Udomkarnjananun et al., Asian Biomedicine 2025.
Field Guide to the Kidney.

Evaluation: History and Physical Examination

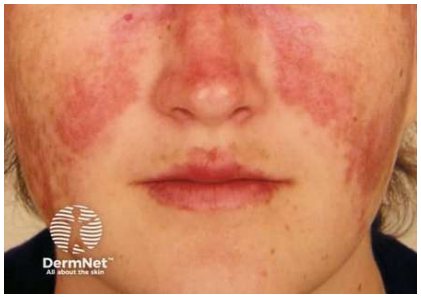
Comprehensive History, Exposure, and Physical Exam Clues for Glomerular Diseases

History / Exposure	Key Exam / Urine Clue	Mechanistic Insight	Associated GN / Pattern
Chronic inflammatory disease (RA, seronegative arthritis, IBD)	Nephrotic syndrome ± CKD	Chronic SAA overproduction	AA amyloidosis
Paraproteinemia / plasma cell dyscrasia	Nephrotic syndrome, CKD	Monoclonal protein deposition	AL amyloidosis, MGRS
Chronic infection	Proteinuria, CKD	Persistent inflammatory stimulus	AA amyloidosis
HIV infection	Rapid nephrotic syndrome, AKI	HIV-mediated podocyte collapse	**Collapsing FSGS (HIVAN)**
Parvovirus B19, EBV, CMV, HTLV-1	AKI, nephrotic syndrome	Viral-triggered podocytopathy	**Collapsing FSGS**
African ancestry	Proteinuria, rapid progression	APOL1 risk alleles	FSGS (esp. collapsing)
Obesity, HTN, sickle cell disease	Proteinuria, CKD	Hyperfiltration injury	Secondary FSGS
Reduced renal mass	Progressive proteinuria	Adaptive hyperfiltration	Secondary FSGS
NSAID exposure	Nephrotic syndrome, bland urine ± AKI	T-cell–mediated podocyte injury	**Minimal change disease ± AIN**
Hodgkin lymphoma	Nephrotic syndrome	Cytokine-mediated podocyte injury	Minimal change disease
Immunizations	New nephrotic syndrome	Immune activation	Minimal change disease

Sethi et al., The Lancet 2022; KDIGO GN 2012. Udomkarnjananun et al., Asian Biomedicine 2025

Comprehensive History, Exposure, and Physical Exam Clues for Glomerular Diseases

History / Exposure	Key Exam / Urine Clue	Mechanistic Insight	Associated GN / Pattern
Stem-cell transplant (months–years)	Nephrotic syndrome	GVHD / alloimmune injury	**Membranous nephropathy**
Solid tumors	Nephrotic syndrome	Paraneoplastic immune complexes	Membranous nephropathy
SLE	Hematuria ± nephrotic syndrome	Immune complex GN	Lupus nephritis
Sjögren's, MCTD, RA	Proteinuria, CKD	Autoimmune immune complexes	Membranous, MPGN
Syphilis, leprosy	Proteinuria	Infection-related immune GN	Membranous GN
Parasites, sarcoidosis, IgG4 disease	Proteinuria	Granulomatous / immune	Membranous GN

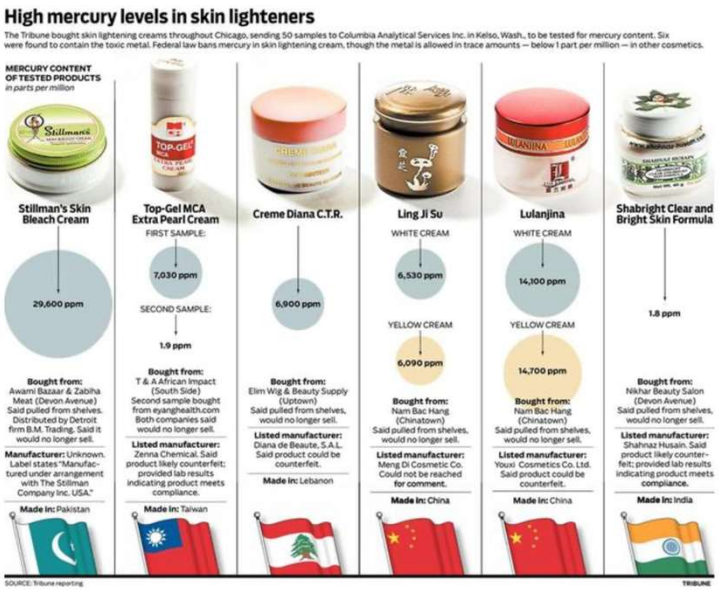


Sethi et al., The Lancet 2022; KDIGO GN 2012. Udomkarnjananun et al., Asian Biomedicine 2025

Comprehensive History, Exposure, and Physical Exam Clues for Glomerular Diseases

History / Exposure	Key Exam / Urine Clue	Mechanistic Insight	Associated GN / Pattern
Gold, penicillamine, **mercury,** tiopronin, captopril	Nephrotic syndrome	Drug-induced podocytopathy	**Membranous GN**

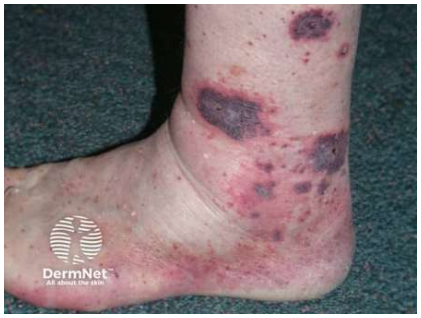
Beauty Products: skin whitening cosmetics, anti-aging creams



Sethi et al., The Lancet 2022; KDIGO GN 2012. Udomkarnjananun et al., Asian Biomedicine 2025

Comprehensive History, Exposure, and Physical Exam Clues for Glomerular Diseases

History / Exposure	Key Exam / Urine Clue	Mechanistic Insight	Associated GN / Pattern
HCV	Low C3, purpura, neuropathy	Cryoglobulin-mediated injury	MPGN
Chronic sinusitis, epistaxis	Purpura, AKI	Small-vessel necrotizing vasculitis	ANCA GN
PTU, hydralazine, penicillamine	AKI, hematuria	Drug-induced autoimmunity	**ANCA-associated GN**
Anti-GBM disease	AKI ± hemoptysis	Linear GBM antibodies	Anti-GBM GN



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Comprehensive History, Exposure, and Physical Exam Clues for Glomerular Diseases

History / Exposure	Key Exam / Urine Clue	Mechanistic Insight	Associated GN / Pattern
High dose Vitamin C, Starfruit, Turmeric, Chaga mushroom	AKI, proteinuria	Oxalate deposition	**Oxalate nephropathy**
Herbal supplements: Traditional Chinese herbal mixtures, Chaga mushroom	Nephrotic syndrome	Similar pathway to NSAID-associated MCD	Minimal change disease

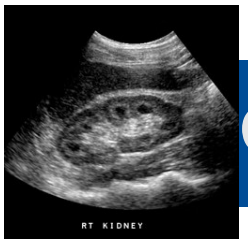
Sethi et al., The Lancet 2022; KDIGO GN 2012. Udomkarnjananun et al., Asian Biomedicine 2025

Evaluation: Testing and Further Steps

Evaluation: Tests

Essential first tests

- Urinalysis with microscopy
  - Hematuria
  - Dysmorphic RBCs
  - RBC casts
- Quantify proteinuria
  - UPCR and ACR (spot sample)
- Basic metabolic panel
  - Serum creatinine



Consider ordering renal US

Serologic Evaluation

Panel: **Initial laboratory evaluation in patients suspected as having glomerulonephritis**

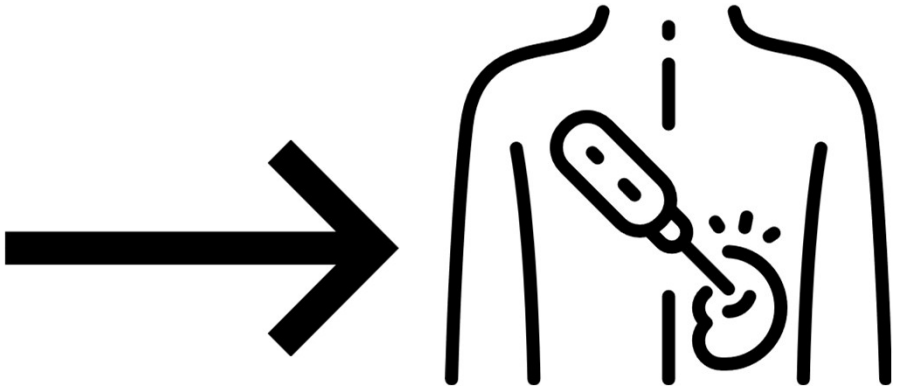
- Complete blood count
- Urinalysis with a careful search for red blood cell casts
- Proteinuria quantification (on a 24 h urine sample)
- Complete metabolic panel
- C3 and C4 complement concentrations
- Anti-double stranded DNA antibody
- Anti-neutrophil cytoplasmic antibodies and anti-glomerular basement membrane serology
- Hepatitis B, hepatitis C, and HIV serology
- Monoclonal protein studies and plasma free light chains (in patients aged >50 years)
- C-reactive protein
- Cryoglobulins and rheumatoid factor (in patients presenting with palpable purpura, arthralgia, or arthritis; peripheral neuropathy; and hypocomplementemia [low C4 concentration] or both)
- Anti-streptolysin O titre, anti-deoxyribonuclease B, and blood cultures (when infection-related glomerulonephritis is suspected)

Sethi et al., The Lancet 2022; KDIGO GN 2012. Udomkarnjananun et al., Asian Biomedicine 2025

Identify Red Flags That Change Management Now

Findings requiring urgent nephrology referral

- Rapid rise in creatinine (days–weeks)
- Active urine sediment
- Nephrotic syndrome with AKI
- Pulmonary symptoms + kidney findings
- Severe hypertension or malignant HTN
- Pregnancy with GN features

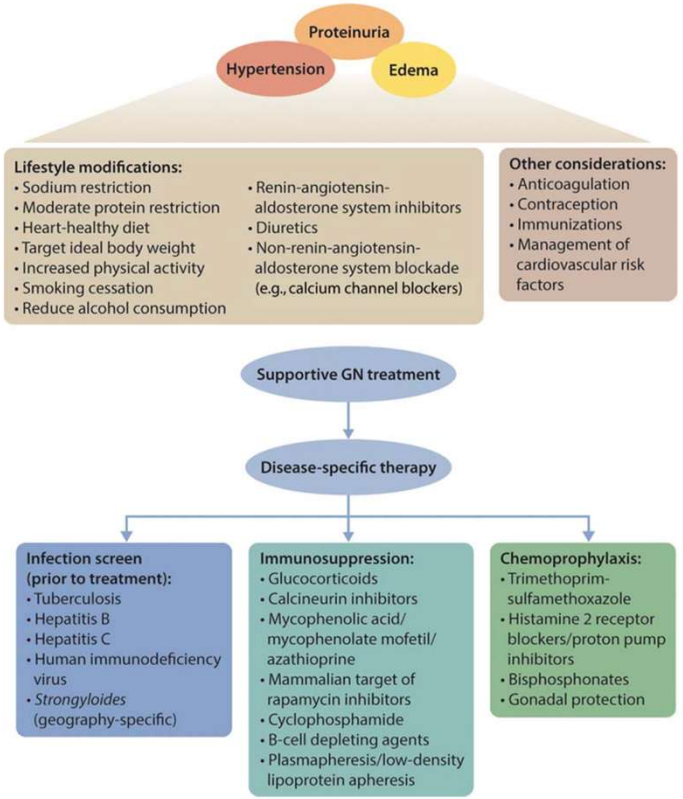


Kidney Biopsy

Sethi et al., The Lancet 2022; KDIGO GN 2012. Udomkarnjananun et al., Asian Biomedicine 2025

Management

Supportive Management of Glomerular Disease



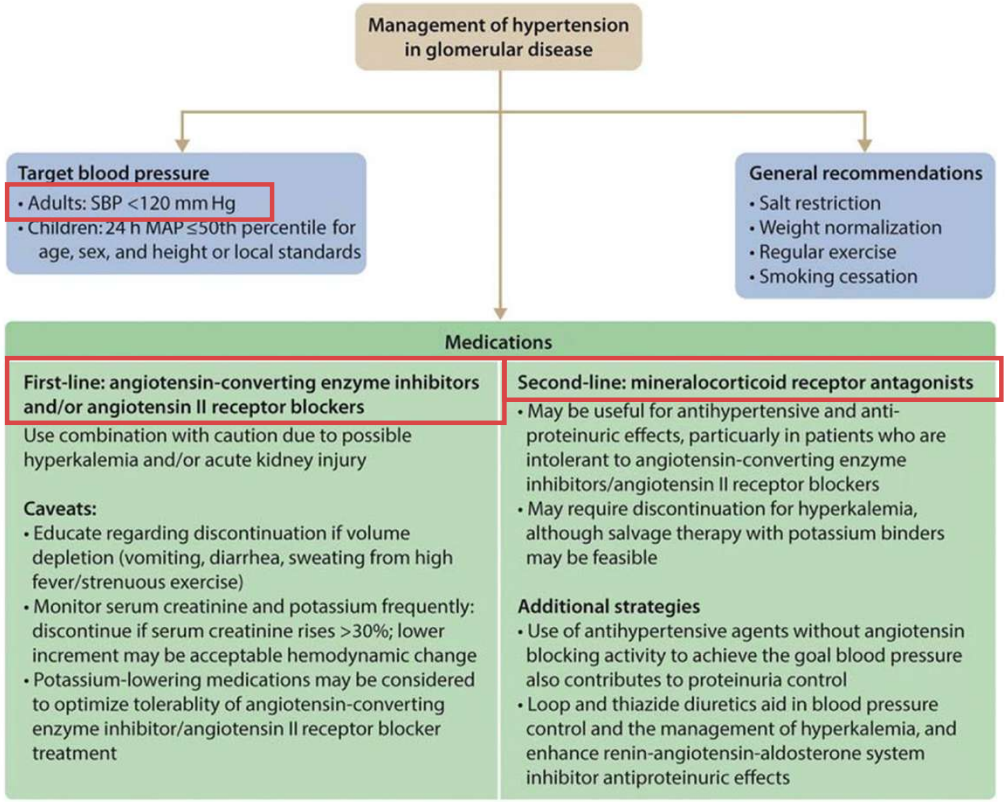
KDIGO GN 2012.

Dietary Modifications

Practice Point 1.14.1.	Restrict dietary sodium to reduce edema, control blood pressure, and control proteinuria	• Dietary sodium <2.0 g/d (<90 mmol/d)
Practice Point 1.14.2.	Restrict dietary protein based on degree of proteinuria	• Nephrotic-range proteinuria: 0.8–1 g/kg/d protein intake* • Add 1 g per g of protein losses (up to 5 g/d) • The safety of protein restriction in GN has not been established in children • Plant-based diets may be preferred
Practice Point 1.14.3.	Restrict dietary protein based on kidney function	• Estimated glomerular filtration rate <60 ml/min/1.73 m^2 with nephrotic-range proteinuria • Limit or target intake to 0.8 g/kg/d • Avoid <0.6 g/kg/d due to safety concerns and risk of malnutrition • Emphasis on vegetable (plant) sources of protein is appropriate
Practice Point 1.14.4.	Restrict caloric intake to achieve normal body mass index and limit central adiposity in order to reduce chronic kidney disease progression, development of kidney failure, cardiovascular events, and mortality	• Target caloric intake 35 kcal/kg/d • Estimated glomerular filtration rate <60 ml/min/1.73 m^2: 30–35 kcal/kg/d
Practice Point 1.14.5.	Restrict dietary fats in patients with elevated serum cholesterol to prevent cardiovascular complications	• Heart-healthy diet • Dietary fat <30% of total calories • Mono- or polyunsaturated fat 7%–10% of total calories

KDIGO GN 2012.

Optimize Blood Pressure Control



SGLT2i is an option as well

Especially in patients with:

- Diabetes
- Heart Failure
- Lupus nephritis
- IgA Nephropathy

KDIGO GN 2012.

Edema Management in Nephrotic Syndrome

Practice Point 1.4.1.	Use loop diuretics as first-line therapy for treatment of edema in the nephrotic syndrome	• Twice daily dosing preferred over once daily dosing; daily dosing may be acceptable for reduced GFR • Increase dose of loop diuretic to cause clinically significant diuresis or until maximally effective dose has been reached • Switch to longer acting loop diuretic such as bumetanide or torsemide/torasemide if concerned about treatment failure with furosemide, or if concerned about oral drug bioavailability
Practice Point 1.4.2.	Restrict dietary sodium intake	• Restrict dietary sodium to <2.0 g/d (<90 mmol/d)
Practice Point 1.4.3.	Use loop diuretics with other mechanistically different diuretics as synergistic treatment of resistant edema in the nephrotic syndrome	• All thiazide-like diuretics in high doses are equally effective. None is preferred. • Thiazide diuretics, administered with an oral or i.v. loop diuretic, will impair distal sodium reabsorption and improve diuretic response • Amiloride may provide improvement in edema/hypertension, and counter hypokalemia from loop or thiazide diuretics • Acetazolamide may be helpful for the metabolic alkalosis of diuresis • Spironolactone may provide improvement in edema/hypertension, and counter hypokalemia from loop or thiazide diuretics
Practice Point 1.4.4.	Monitor for adverse effects of diuretics	• Hyponatremia with thiazide diuretics • Hypokalemia with thiazide and loop diuretics • Impaired GFR • Volume depletion, especially in pediatric/elderly patients • Hyperkalemia with spironolactone and eplerenone especially if combined with RAS blockade

If associated AKI:

- Hold RASi, SGLT2i, MRA
- Allow systolic blood pressure of 130-150mmHg

KDIGO GN 2012.

Hyperlipidemia Management

Practice Point 1.6.1.	**Treatment of hyperlipidemia may be considered in patients with the nephrotic syndrome, particularly for patients with other cardiovascular risk factors, including hypertension and diabetes**	High quality data are lacking to guide treatment in these patients
Practice Point 1.6.2.	**Use lifestyle modifications in all patients with persistent hyperlipidemia and glomerular disease:** • **Heart-healthy diet** • **Increased physical activity** • **Weight reduction** • **Smoking cessation**	• Not well studied as primary means of reducing lipids in nephrotic syndrome • Can be used as primary therapy in low-risk individuals with mild to moderate hyperlipidemia • Additive to pharmacologic treatment of hyperlipidemia • Considered first-line treatment of hyperlipidemia in children • Consider a plant-based diet • Avoid red meat
Practice Point 1.6.3.	**Consider starting a statin drug as first-line therapy for persistent hyperlipidemia in patients with glomerular disease:** • **Assess ASCVD risk based on LDL-C, Apo B, triglyceride and Lp (a) levels, age group, and ASCVD 'risk enhancers'** • **Align statin dosage intensity to ASCVD risk** • **Statins can be initiated in children aged > 8 years with concerning family history, extremely elevated LDL-C or Lp(a), in the context of informed shared decision-making and counselling with patient and family**	• Reduced eGFR (<60 ml/min/1.73 m^2 not on dialysis) and albuminuria (ACR >30 mg/g) are independently associated with an elevated risk of ASCVD • ASCVD risk enhancers include chronic inflammatory conditions such as systemic lupus erythematosus, rheumatoid arthritis, history of preeclampsia, early menopause, South Asian ancestry, chronic kidney disease and human immunodeficiency virus/AIDS (accuracy of ASCVD risk estimators have not been well validated for adults with chronic inflammatory disorders or human immunodeficiency virus) • Adherence to changes in lifestyle and effects of LDL-C lowering medication should be assessed by measurement of fasting lipids and appropriate safety indicators 4–12 weeks after statin initiation/dose adjustment or inflammatory disease-modifying therapy/antiretroviral therapy, and every 3–12 months thereafter based on need to assess adherence or safety
Practice Point 1.6.4.	**Consider initiation of non-statin therapy in those individuals who cannot tolerate a statin, or who are at high ASCVD risk and fail to achieve LDL-C or triglyceride goals despite maximally tolerated statin dose:** • **Bile acid sequestrants** • **Fibrates** • **Nicotinic acid** • **Ezetimibe** • **PCSK9 inhibitor** • **Lipid apheresis**	• Bile acid sequestrants have a high rate of gastrointestinal side effects limiting their use • Bile acid sequestrants and fibrates have been shown in small studies to reduce serum cholesterol in nephrotic syndrome • Fibrates will increase serum creatinine level due to direct action on the kidney • Ezetimibe has limited vascular and clinical benefits, but is used in statin-intolerant patients as salvage therapy • Nicotinic acid and ezetimibe have not been studied in patients with nephrotic syndrome • PCSK9 inhibitors may be beneficial in nephrotic syndrome; trials ongoing

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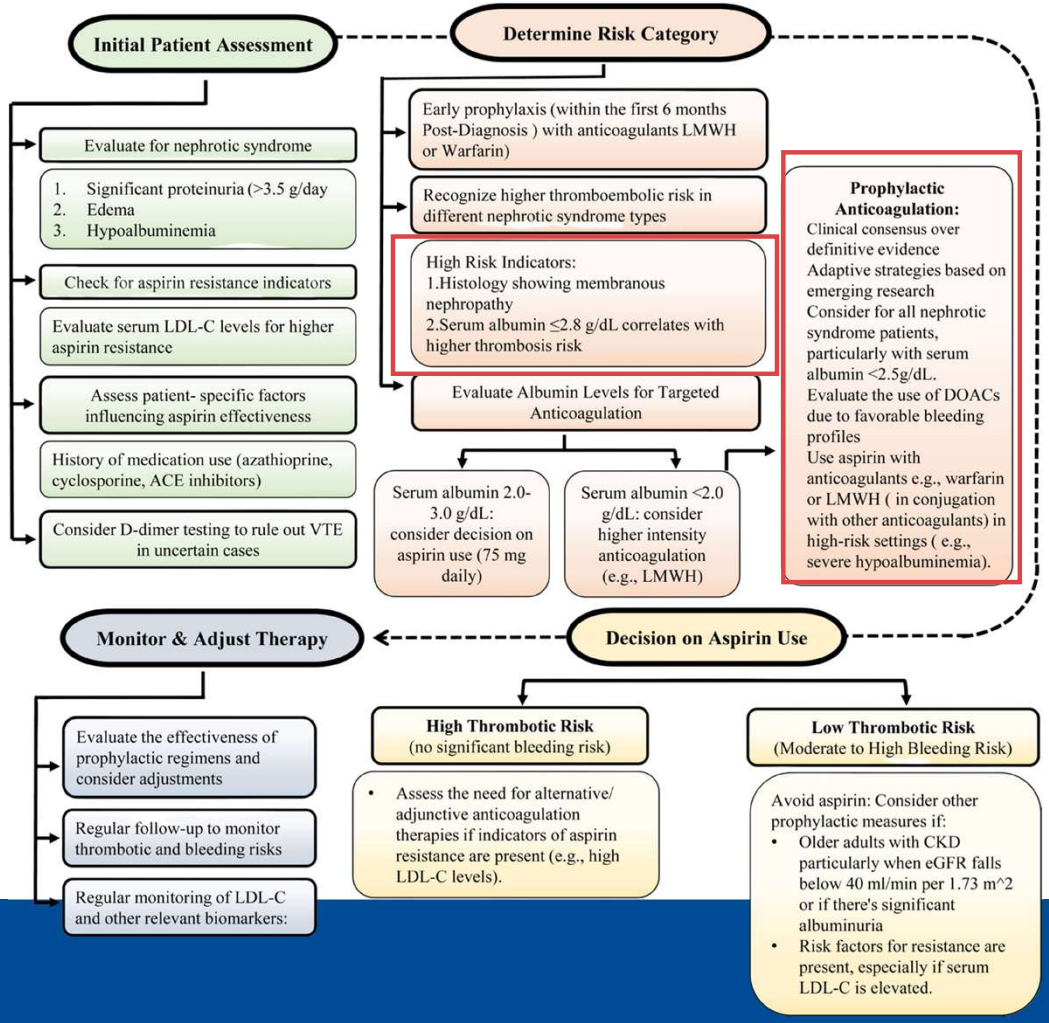
Anticoagulation in Nephrotic Syndrome



Ghozloujeh ZG, et al. KI Reports. 2025.

Screening/prophylaxis for all patients with glomerular disease on immunosuppression

Assessment	Measures
Peptic ulcer disease	H_2 blockers Proton pump inhibitors
Bone health and protection	Individual fracture risk assessment/bone mineral density Calcium and vitamin D supplementation Bisphosphonates Growth hormone (pediatric population)
Infection risk	Assess medical history of herpes zoster infection Screening for hepatitis B virus, hepatitis C virus, human immunodeficiency virus Hepatitis B virus vaccination Zoster vaccination Screening for tuberculosis Screening for strongyloides Pneumocystis prophylaxis Influenza and pneumococcal vaccination* Meningococcal vaccination (if C5 antagonists are used) Monitor gammaglobulin levels and white blood cells levels (rituximab, cyclophosphamide)
Ultraviolet light protection	Limit ultraviolet exposure Broad-spectrum sunscreen
Cancer screening	Evaluate individual risk factors for malignancy Age-specific malignancy screening Annual dermatology exam Bladder cancer (cyclophosphamide cumulative dose >36 g)

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Reproductive health for patients with glomerular disease

Effective contraception	Individual evaluation (preference, thrombosis risk, age)

Contraceptive method	Unintended pregnancy rate within 1st year of use (%)†		Contraindications in glomerular disease	Other considerations
	Perfect use	Typical use		
Estrogen-containing methods (pill, patch, ring)	0.3	9	• Lupus • Venous thromboembolism • Vascular disease	• Breast cancer risk • Cervical cancer risk with immunosuppression • Venous thromboembolism risk in nephrotic syndrome
Progesterone-only pill	0.3	9	• None	• Longest re-dosing interval with desogestrel (may improve typical use) • Possible breast cancer risk, especially >40 yr
Progesterone intrauterine device (Mirena)	0.2	0.2	• None	• Possible breast cancer risk, especially >40 yr • Effective with immuno-suppression, no evidence of increased infection
Progesterone implant (Nexplanon)	0.05	0.05	• None	• Possible breast cancer risk, especially >40 yr
Copper intrauterine device	0.6	0.8	• None	• No associated hormonal risk
Male condom	2	18	• Ineffective for long-term use	• Protects against human immunodeficiency virus and sexually transmitted infection
Female condom	5	21		
None	85	85		

If patient desires pregnancy:

- Encourage patient to discuss with nephrologist:
  - Discuss timing
  - Explain potential complications
  - Change to non-teratogenic medications

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Take Home Points

- **Glomerulonephritis is a syndrome, not a diagnosis**: early care is guided by clinical patterns, not pathology.
- **Urine findings often precede changes in serum creatinine**; creatinine is a late marker of injury.
- **Hematuria and proteinuria together strongly suggest glomerular disease.**
- **Severity is defined by trajectory and context**, not protein quantity alone.
- **Nephritic and nephrotic syndromes are useful clinical heuristics**, but overlap is common.
- **Mechanisms of AKI differ**:
  - *Nephritic*: inflammatory and crescentic injury
  - *Nephrotic*: hemodynamic, tubular, or thrombotic processes
- **Medication, supplement, infection, malignancy, and transplant histories matter**: many cases are secondary and potentially reversible.
- **Supportive management is not benign**:
  - Optimize blood pressure carefully
  - Manage edema thoughtfully, especially in AKI
  - Address hyperlipidemia and thrombosis risk
  - Avoid nephrotoxins and inappropriate RAAS blockade during instability
- **Active urine sediment, rapid creatinine rise, pulmonary involvement, or systemic features are emergencies.**
- **Early nephrology referral and timely biopsy improve outcomes**: delay is a major predictor of irreversible kidney loss.

Thank you!