

Glomerular Lesions Associated With Systemic Diseases

Lupus nephritis • Henoch–Schönlein purpura

Morphology, immune deposits, and clinicopathologic correlation

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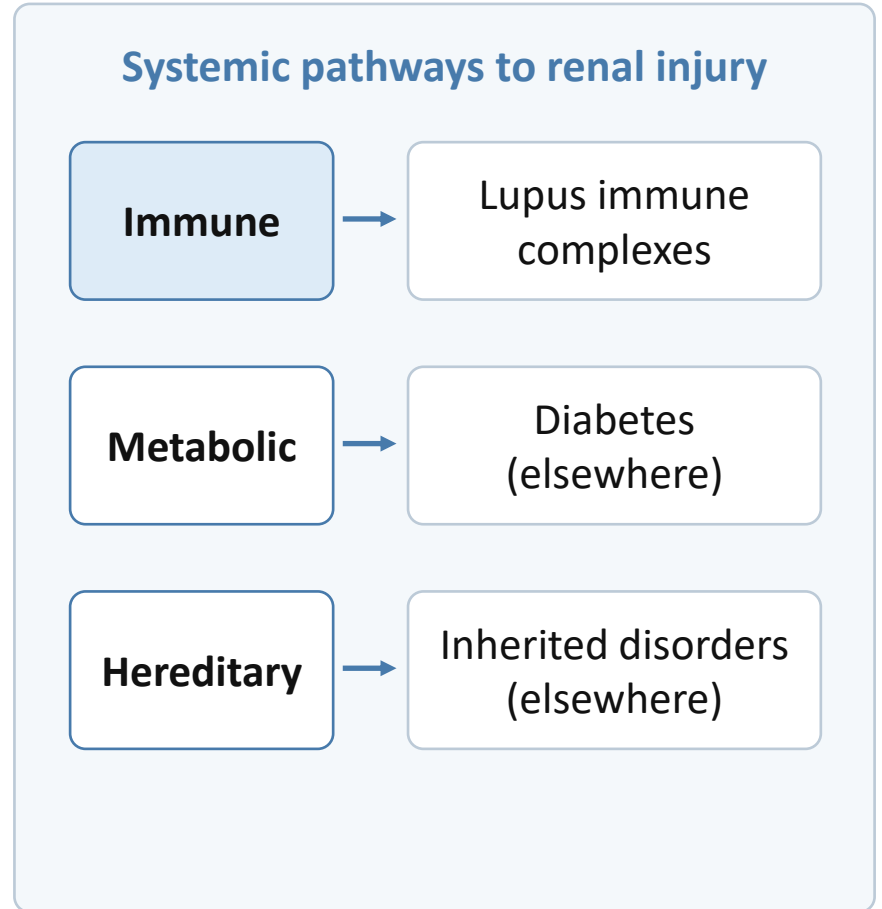
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Learning objectives

- Connect systemic immune diseases with distinct renal morphologic patterns.
- Differentiate the six lupus nephritis classes by anatomic distribution and activity.
- Explain the IgA-associated clinical and renal spectrum of Henoch–Schönlein purpura.
- Interpret clinical syndromes using light microscopy, immunofluorescence, and electron microscopy.

Systemic diseases can injure the glomerulus

- Immunologic, metabolic, and hereditary systemic disorders may produce glomerular injury.
- In systemic lupus erythematosus and diabetes, renal involvement may be a major manifestation.
- This section focuses on lupus nephritis and Henoch–Schönlein purpura; Other systemic lesions are addressed elsewhere.



Schematic classification; Not an exhaustive disease list.

Renal pathology reflects more than one mechanism

- Immune complexes may localize in mesangial or capillary-wall compartments.
- Inflammatory cellular proliferation, capillary necrosis, and crescents express active injury.
- Extrarenal disease does not specify the glomerular lesion without morphologic evaluation.

Immune-complex glomerular injury

Systemic autoimmunity



Glomerular deposits



Inflammatory response



Capillary injury

Causal framework summarized from the described deposits and lesions.

Clinical presentations span several syndromes

- Hematuria may be microscopic or gross and can recur.
- Inflammatory glomerular injury may produce nephritic syndrome or rapidly progressive glomerulonephritis.
- Nephrotic syndrome, renal insufficiency, and hypertension may coexist with other findings.

Patterns of clinical presentation

Hematuria

Nephritic
syndrome

Nephrotic
syndrome

RPGN

Hypertension

Renal failure

*Clinical syndromes are not a direct substitute for
biopsy class.*

Three modalities provide complementary evidence

- Light microscopy identifies cellularity, necrosis, crescents, capillary-wall change, and scarring.
- Immunofluorescence maps immunoglobulin and complement deposits.
- Electron microscopy helps localize immune-complex deposits within glomerular structures.

Complementary tissue modalities

Light microscopy

Cells • matrix •
scarring

Immunofluorescence

Immune-reactant
distribution

Electron microscopy

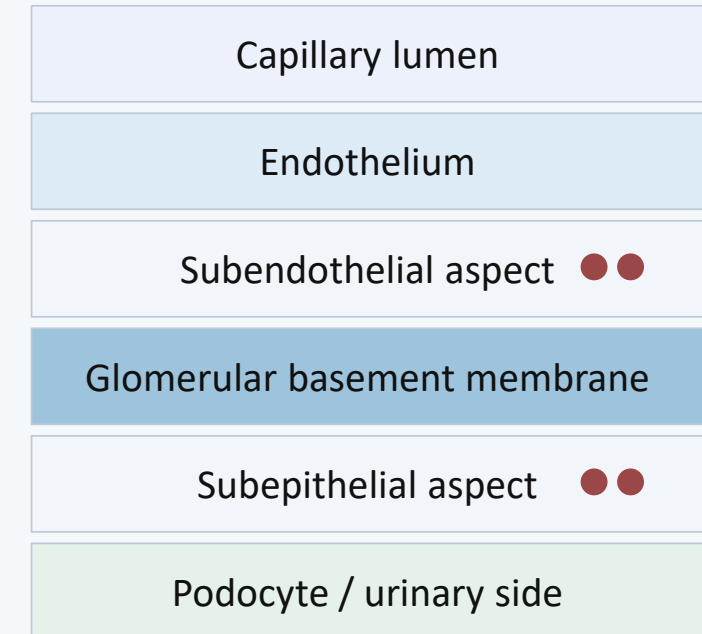
Ultrastructural
deposits

The modalities answer different morphologic questions.

Deposit location is a diagnostic clue

- Mesangial immune complexes are prominent in early lupus patterns and IgA-associated disease.
- Subendothelial deposits contribute to capillary-wall injury and wire-loop appearances in proliferative lupus nephritis.
- Subepithelial deposits are characteristic of membranous lupus nephritis.

Where immune complexes accumulate



Red dots: Schematic immune deposits

Glomerular activity and chronicity differ

- Proliferation, necrosis, and crescent formation signify active tissue injury.
- Global or segmental sclerosis represents scarring after earlier lesions.
- A lesion can resolve or evolve to chronic damage; The current biopsy represents one time point.

Active lesions versus outcomes

Necrosis / proliferation



Healing pathway



Recovery OR segmental/global scar

Repair may restore architecture or leave chronic scars.

Lupus nephritis: A major SLE manifestation

- Clinically significant renal involvement in up to 50% of patients with systemic lupus erythematosus.
- Glomerulopathy and tubulointerstitial nephritis are important forms of renal disease.
- The clinical spectrum extends from asymptomatic urine abnormalities to renal failure.

Patterns of clinical presentation

Hematuria

Nephritic
syndrome

Nephrotic
syndrome

RPGN

Hypertension

Renal failure

Clinical syndromes are not a direct substitute for biopsy class.

Immune-complex deposition underlies most lesions

- Immune complexes accumulate within glomerular compartments.
- Mesangial and glomerular basement membrane deposits are common sites.
- The distribution of deposits helps explain the morphologic diversity across classes.

Immune-complex glomerular injury

Systemic autoimmunity



Glomerular deposits



Inflammatory response



Capillary injury

Causal framework summarized from the described deposits and lesions.

Lupus immunofluorescence involves multiple reactants

- IgG is usually detected within lupus glomerular immune complexes.
- IgA and IgM may accompany IgG.
- C3 and C1q frequently accompany the immunoglobulins and signal complement-associated immune deposition.

Lupus glomerular immune reactants

Often
prominent

IgG

May
accompany

IgA • IgM

Complement
reactants

C3 • C1q

Reactive staining is characteristic; Patterns vary.

One systemic disease, several renal phenotypes

- Renal lupus may manifest with hematuria, nephritic sediment, nephrotic syndrome, or renal insufficiency.
- Even within the same biopsy category, clinical severity can vary.
- Glomerular, tubulointerstitial, and vascular lesions can occur separately or together.

Renal involvement beyond glomeruli

Glomeruli

Immune complexes /
proliferation

Tubules & interstitium

Deposits and
inflammation

Vessels

Deposits ± vasculitis
/ TMA

The same systemic disease may involve multiple compartments.

Lupus nephritis has six morphologic classes

- Classification distinguishes mesangial, proliferative, membranous, and advanced sclerosing patterns.
- Lesions may overlap, and the same patient can shift patterns over time.
- In the source account, class I is least common and class IV the most common pattern.

The six renal morphologic classes

I

Minimal mesangial

II

Mesangial proliferative

III

Focal

IV

Diffuse

V

Membranous

VI

Advanced sclerosing

The biopsy classification is not immutable

- An initial mesangial pattern can coexist with injury in other renal compartments.
- An active proliferative pattern may heal or produce permanent glomerular scars.
- Clinical evolution may require reassessment because biopsy class represents sampled tissue at a particular time.

Active lesions versus outcomes

Necrosis / proliferation



Healing pathway



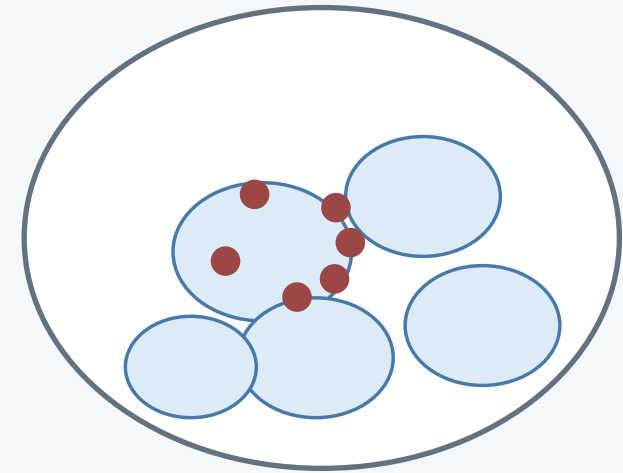
Recovery OR segmental/global scar

Repair may restore architecture or leave chronic scars.

Class I: Minimal mesangial lupus nephritis

- Immune-complex deposits occur within mesangial regions.
- Light microscopy shows no structural glomerular abnormalities.
- The deposits are demonstrable by immunofluorescence and electron microscopy.

Class I: Deposits without LM change



Mesangial immune complexes

Schematic drawing — not a histologic specimen.

Class I illustrates a microscopy mismatch

- Normal-appearing glomeruli on light microscopy do not exclude immune-complex glomerular disease.
- Mesangial deposits may be present without visible mesangial hypercellularity.
- Diagnosis depends on integrating complementary diagnostic modalities.

Complementary tissue modalities

Light microscopy

Cells • matrix •
scarring

Immunofluorescence

Immune-reactant
distribution

Electron microscopy

Ultrastructural
deposits

The modalities answer different morphologic questions.

Class I may have associated non-class lesions

- Lupus podocytopathy may resemble minimal change disease.
- Tubulointerstitial inflammation can be present despite subtle glomerular changes.
- Thrombotic microangiopathy can coexist with otherwise minimal mesangial findings.

Renal involvement beyond glomeruli

Glomeruli

Immune complexes /
proliferation

Tubules & interstitium

Deposits and
inflammation

Vessels

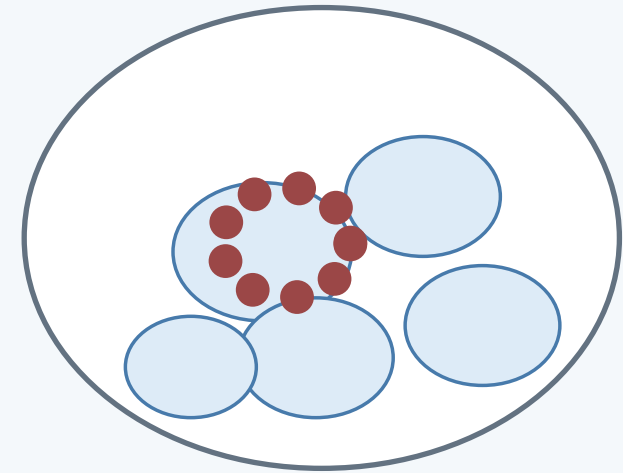
Deposits ± vasculitis
/ TMA

The same systemic disease may involve multiple compartments.

Class II: Mesangial proliferative nephritis

- Mesangial cells proliferate; Mesangial matrix accumulation is often present.
- Immune complexes and complement show granular mesangial deposits.
- Capillary involvement and active endocapillary inflammation are absent in the defining pattern.

Class II: Proliferative mesangium



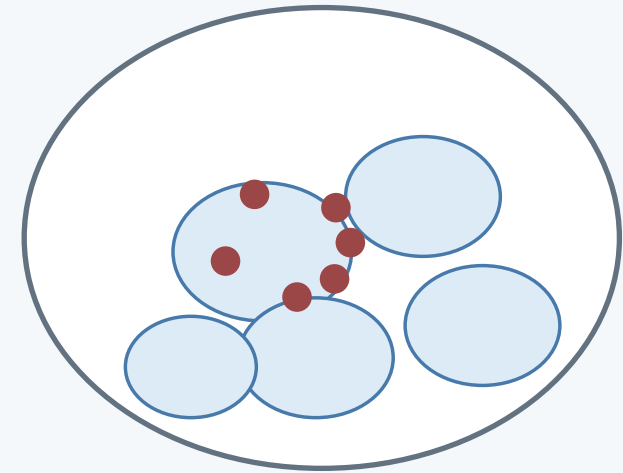
Mesangial hypercellularity

Schematic drawing — not a histologic specimen.

Class II deposits remain mesangial

- The distribution of immunoglobulins and complement is mesangial.
- Increased mesangial cells and matrix distinguish class II from class I on light microscopy.
- This pattern should not be confused with the necrotizing or capillary proliferative lesions of classes III and IV.

Class I: Deposits without LM change



Mesangial immune complexes

Schematic drawing — not a histologic specimen.

Classes I and II: Decisive distinction

Feature	Class I	Class II
Light microscopy	No glomerular structural change	Mesangial hypercellularity ± matrix
IF / EM	Mesangial immune deposits	Mesangial immune deposits
Endocapillary activity	Not defining	Not part of defining pattern
Interpretive point	Deposits may be occult on LM	Proliferation visible on LM

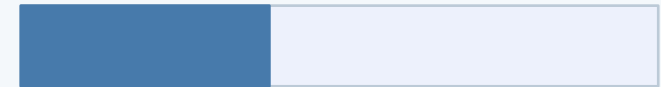
Class III: Focal lupus nephritis

- Fewer than 50% of sampled glomeruli are involved in the source definition.
- Affected glomeruli can show segmental or global lesions.
- Focal describes how many glomeruli are affected, not how much of any single tuft is injured.

Extent of glomerular involvement

Focal: Class III

Fewer than half of
glomeruli involved



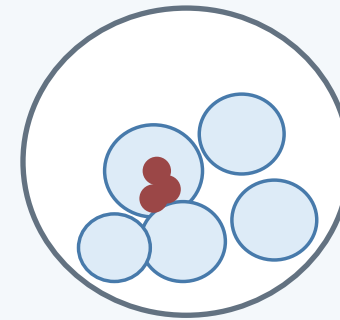
< 50%

Bar illustrates the definition, not a case dataset.

Segmental and global describe lesion extent

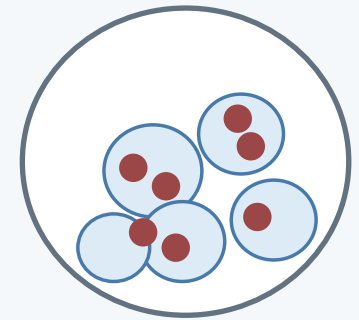
- A segmental lesion occupies part of an individual glomerular tuft.
- A global lesion involves the entire individual glomerulus.
- Either can occur within the focal pattern of class III lupus nephritis.

How far a lesion extends within one glomerulus



Segmental

Portion of tuft



Global

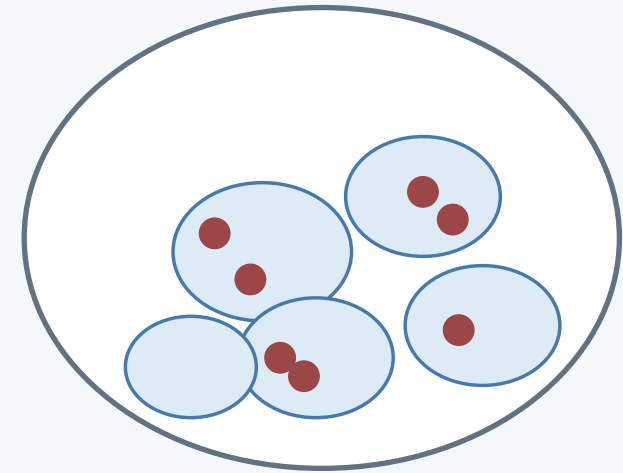
Entire tuft

Extensiveness within a glomerulus differs from focal/diffuse.

Class III: Active inflammatory morphology

- Endothelial and mesangial cells may swell or proliferate.
- Leukocytes accumulate in affected glomerular capillary loops.
- Hyaline thrombi and necrotizing lesions may be present.

Capillary hypercellularity



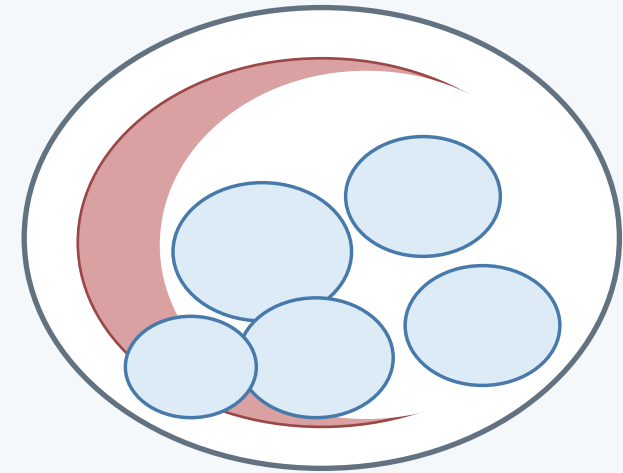
Endocapillary hypercellularity

Schematic drawing — not a histologic specimen.

Class III: Necrosis and crescents

- Focal capillary necrosis may accompany extracapillary proliferation.
- Cellular crescents form in Bowman space near injured capillary loops.
- These lesions indicate active glomerular inflammation rather than simple mesangial deposit accumulation.

Extracapillary crescent formation



Cellular crescent at tuft periphery

Schematic drawing — not a histologic specimen.

Class III: Nephritic urinary sediment

- Hematuria and variable proteinuria are common presentations.
- Red cell casts correlate with active disease and a nephritic urinary sediment.
- Clinical severity ranges from relatively mild urinary abnormalities to acute renal insufficiency.

Patterns of clinical presentation

Hematuria

Nephritic
syndrome

Nephrotic
syndrome

RPGN

Hypertension

Renal failure

Clinical syndromes are not a direct substitute for biopsy class.

Class III can heal or become chronic

- Inflammatory lesions may regress with restoration of glomerular architecture.
- Other lesions heal by segmental or global glomerular scarring.
- Some cases progress to diffuse glomerulonephritis, underscoring the changing disease pattern.

Active lesions versus outcomes

Necrosis / proliferation



Healing pathway



Recovery OR segmental/global scar

Repair may restore architecture or leave chronic scars.

Class IV: Diffuse lupus nephritis

- The source describes involvement of more than 50% of glomeruli.
- It is the most common and among the most severe morphologic forms.
- Inflammatory lesions resemble class III but are more extensive across the biopsy.

Class IV: Diffuse involvement

Diffuse: Class IV

More than half of glomeruli affected (source wording)



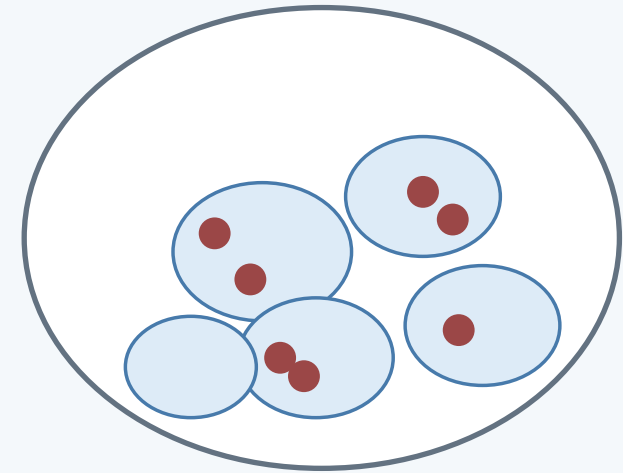
> 50%

Illustrative bar; No patient percentages are implied.

Class IV: Marked cellular proliferation

- Endothelial and mesangial proliferation increase glomerular cellularity.
- Epithelial proliferation can create prominent extracapillary crescents.
- Capillary lumina may be distorted or compromised by the inflammatory response.

Capillary hypercellularity



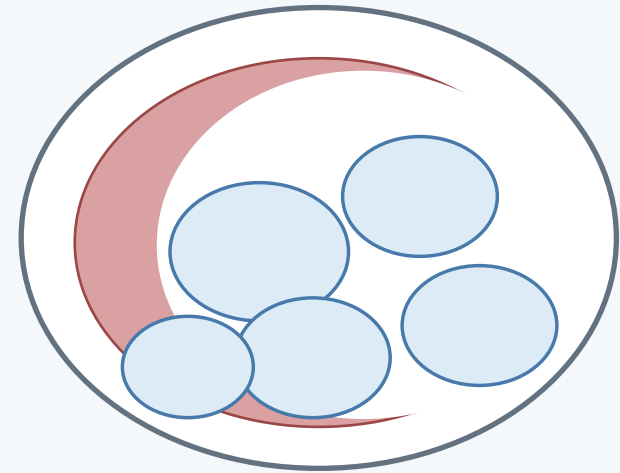
Endocapillary hypercellularity

Schematic drawing — not a histologic specimen.

Class IV: Crescents may fill Bowman space

- Injured glomeruli may develop extensive epithelial-cell proliferation.
- Cellular crescents can occupy the urinary space around the tuft.
- Necrosis and active proliferation often occur with other immune-complex-mediated lesions.

Extracapillary crescent formation

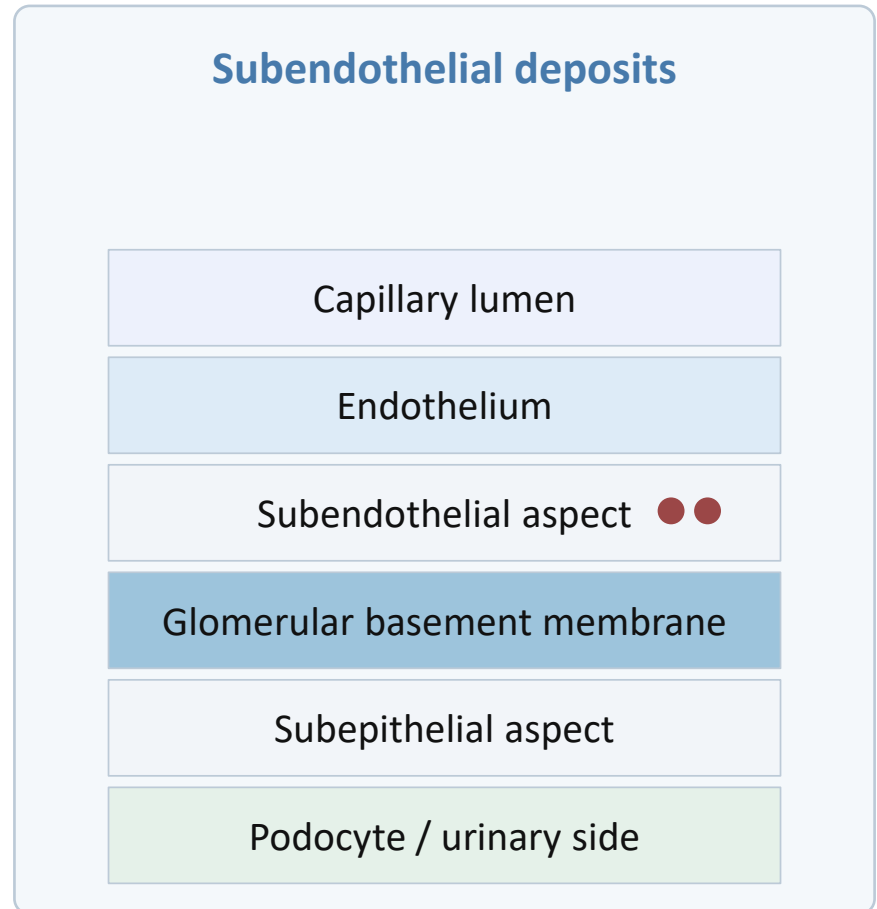


Cellular crescent at tuft periphery

Schematic drawing — not a histologic specimen.

Wire loops indicate subendothelial deposits

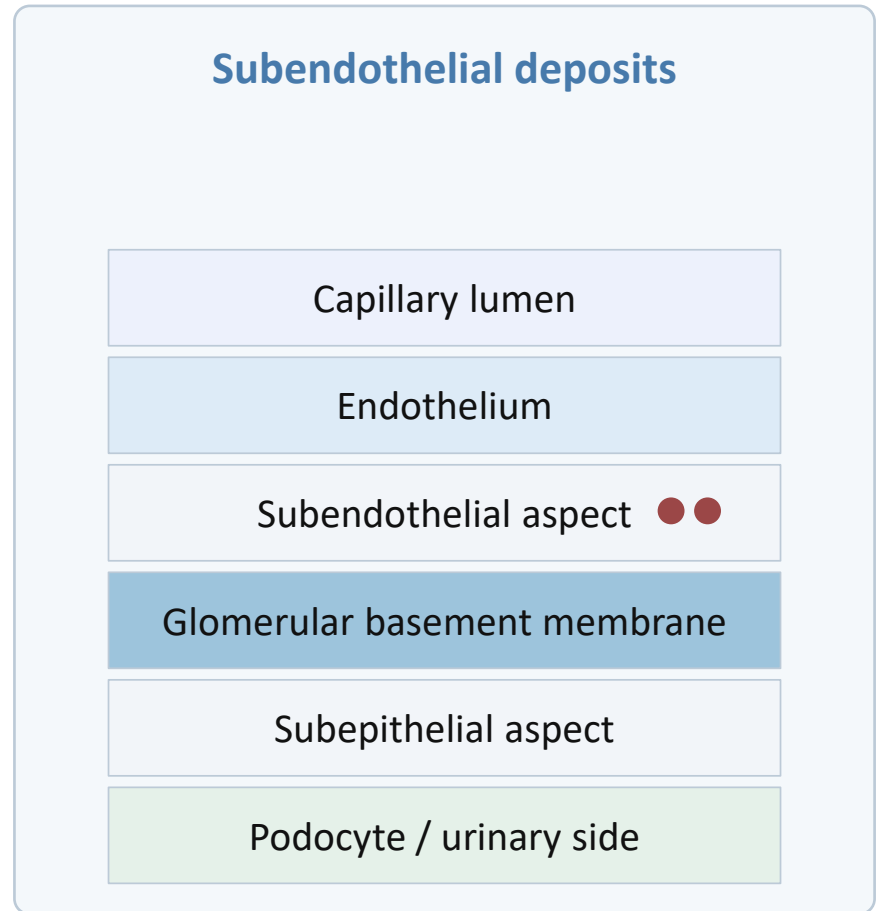
- Extensive subendothelial immune complexes thicken capillary walls.
- Circumferential wall thickening creates the light-microscopic “wire loop” appearance.
- The morphologic observation is supported by electron-dense deposits on ultrastructural examination.



Red dots: Schematic immune deposits

The subendothelial position is mechanistic

- Subendothelial deposits lie between endothelium and glomerular basement membrane.
- Immune complexes at this interface accompany capillary-wall inflammatory changes.
- Their abundant accumulation distinguishes the classic wire-loop lesion from a purely mesangial process.



Red dots: Schematic immune deposits

Class IV: Immunofluorescence and EM correlate

- Immune-complex deposition is readily demonstrable by immunofluorescence.
- Electron microscopy identifies electron-dense deposits within involved capillary walls.
- Correlating stain distribution with the light-microscopic lesion strengthens interpretation.

Complementary tissue modalities

Light microscopy

Cells • matrix •
scarring

Immunofluorescence

Immune-reactant
distribution

**Electron
microscopy**

Ultrastructural
deposits

The modalities answer different morphologic questions.

Class IV commonly produces symptomatic renal disease

- Patients generally have hematuria together with proteinuria.
- Hypertension may accompany extensive glomerular inflammation.
- Renal insufficiency may range from mild impairment to severe dysfunction.

Patterns of clinical presentation

Hematuria

Nephritic
syndrome

Nephrotic
syndrome

RPGN

Hypertension

Renal failure

Clinical syndromes are not a direct substitute for biopsy class.

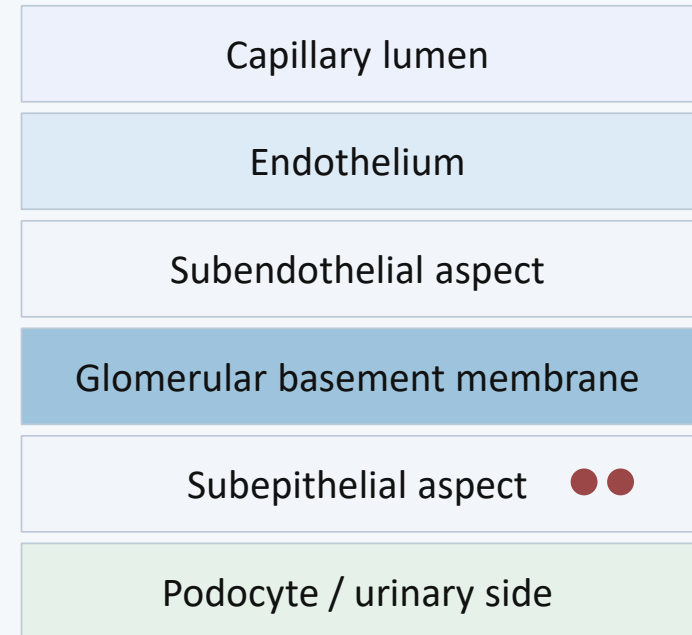
Classes III and IV differ chiefly in extent

Comparison	Class III — focal	Class IV — diffuse
Glomerular distribution	Fewer than 50% involved	More than 50% involved (source)
Inflammatory pattern	Segmental or global active lesions	Similar lesions, more extensive
Necrosis / crescents	May be present	May be prominent
Clinical spectrum	Mild urine findings → AKI	Common hematuria / proteinuria; Renal insufficiency
Long-term lesion	May scar or progress	May progress to glomerulosclerosis

Class V: Membranous lupus nephritis

- Diffuse capillary-wall thickening is the principal light-microscopic feature.
- The thickening reflects subepithelial immune-complex deposits.
- Basement membrane–like material accumulates around the deposits.

Subepithelial deposits



Red dots: Schematic immune deposits

Subepithelial deposits alter the filtration wall

- The deposits lie external to the glomerular basement membrane beneath podocytes.
- Capillary walls become diffusely thickened as additional basement membrane material is produced.
- The pattern parallels primary membranous nephropathy morphologically.

Capillary-wall compartments

Capillary lumen

Endothelium

Subendothelial aspect

Glomerular basement membrane

Subepithelial aspect

Podocyte / urinary side

Class V commonly presents with nephrosis

- Severe proteinuria or nephrotic syndrome is a typical clinical presentation.
- The lesion emphasizes filtration-barrier injury rather than obligatory florid endocapillary proliferation.
- Mesangial immune-complex deposits are also frequently seen.

Site of deposits shapes injury

III–IV

Subendothelial

Proliferative injury

V

Subepithelial

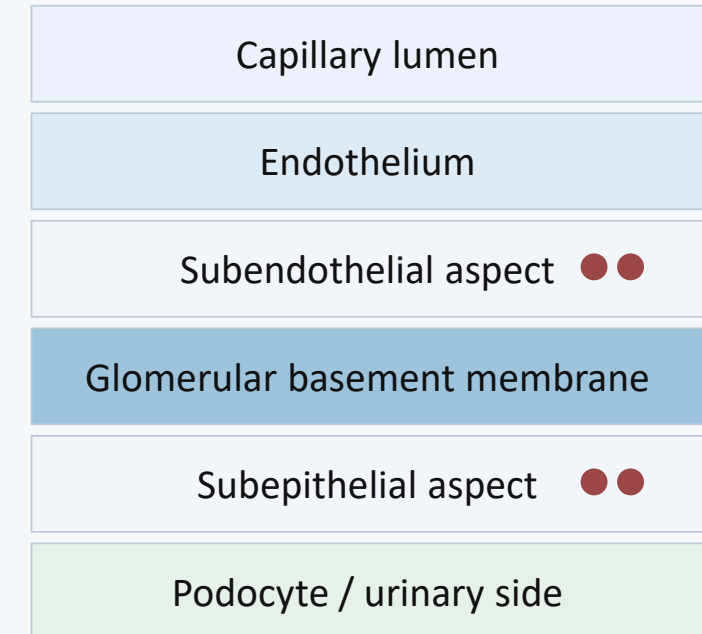
Proteinuria / nephrosis

Simplified clinicopathologic mapping.

Class V may coexist with proliferative lesions

- Membranous lupus nephritis can occur together with focal lupus nephritis.
- It can also coexist with diffuse proliferative lupus nephritis.
- Interpret the full biopsy; One deposit compartment need not exclude another lesion pattern.

Where immune complexes accumulate

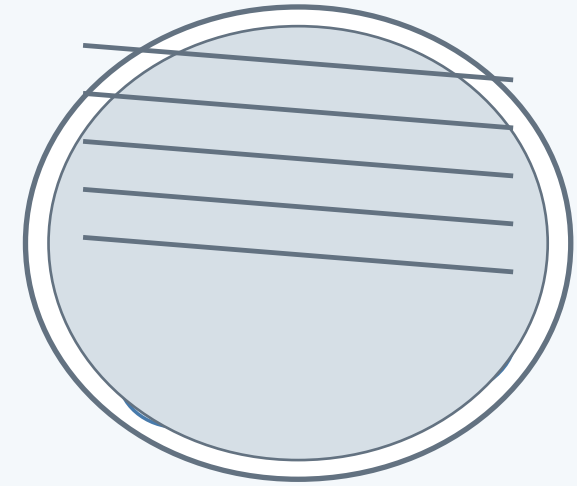


Red dots: Schematic immune deposits

Class VI: Advanced sclerosing lupus nephritis

- The defining finding is global sclerosis in 90% or more of glomeruli.
- This is a predominantly chronic, scarred morphologic pattern.
- It should be distinguished from the active inflammatory findings of proliferative classes.

Class VI is defined by scar burden



≥90% global glomerular sclerosis

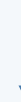
Schematic: One glomerulus, not a quantified biopsy.

Activity and sclerosis answer different questions

- Necrosis and crescents represent active injury detectable on microscopy.
- Global sclerosis reflects replacement of functioning glomerular architecture by scar.
- Severe chronic sclerosis may persist after earlier immune-mediated inflammation has subsided.

Active lesions versus outcomes

Necrosis / proliferation



Healing pathway



Recovery OR segmental/global scar

Repair may restore architecture or leave chronic scars.

Classes I–VI: Morphology in one comparison

Class	Principal morphology	Key deposit/location clue
I	No LM structural change	Mesangial IF/EM deposits
II	Mesangial cell/matrix increase	Mesangial deposits
III	Focal active ± necrotizing lesions	Variable immune complexes
IV	Diffuse proliferative ± wire loops	Frequent subendothelial deposits
V	Diffuse capillary-wall thickening	Subepithelial deposits
VI	≥90% global sclerosis	Advanced chronic scar

Renal lupus is not restricted to the glomerulus

- Tubular and interstitial changes are frequent in lupus nephritis.
- In occasional patients, tubulointerstitial injury dominates the renal abnormalities.
- Immune deposits may involve tubular or peritubular capillary basement membranes.

Renal involvement beyond glomeruli

Glomeruli

Immune complexes / proliferation

Tubules & interstitium

Deposits and inflammation

Vessels

Deposits ± vasculitis / TMA

The same systemic disease may involve multiple compartments.

Interstitial inflammation may be organized

- Lupus kidneys sometimes contain well-formed interstitial B-cell follicles.
- Plasma cells may accompany the follicles.
- The source notes that some interstitial plasma cells may produce autoantibodies locally.

Renal involvement beyond glomeruli

Glomeruli

Immune complexes /
proliferation

Tubules & interstitium

Deposits and
inflammation

Vessels

Deposits ± vasculitis
/ TMA

The same systemic disease may involve multiple compartments.

Vascular immune deposits may accompany lupus

- Immune-complex deposits may occur in the intima and media of renal arteries.
- Vascular pathology can coexist with glomerular and tubulointerstitial disease.
- Active vasculitis is less frequent than mere vascular immune deposition.

Renal involvement beyond glomeruli

Glomeruli

Immune complexes / proliferation

Tubules & interstitium

Deposits and inflammation

Vessels

Deposits ± vasculitis / TMA

The same systemic disease may involve multiple compartments.

Vasculitis and thrombotic microangiopathy differ

- Occasionally, vascular inflammation includes necrosis of the vessel wall.
- Thrombotic microangiopathy can coexist with lupus-associated renal disease.
- Distinguish these vascular findings from proliferative glomerular lesions in tissue interpretation.

Renal involvement beyond glomeruli

Glomeruli

Immune complexes / proliferation

Tubules & interstitium

Deposits and inflammation

Vessels

Deposits ± vasculitis / TMA

The same systemic disease may involve multiple compartments.

Evaluate the entire renal biopsy

- Count the distribution of involved glomeruli and describe segmental or global lesions.
- Identify necrosis, crescents, capillary-wall thickening, and chronic scarring.
- Examine the tubulointerstitium and vessels in addition to glomerular deposits.

Pathology interpretation sequence

Distribution

Focal versus diffuse

Compartment

Mesangial vs capillary wall

Activity

Necrosis / crescents

Chronicity

Glomerular sclerosis

Renal syndrome does not determine lupus class

- Hematuria and renal insufficiency may be seen in several active classes.
- Marked proteinuria commonly accompanies class V, but membranous and proliferative lesions may coexist.
- Tissue evidence reconciles the clinical presentation with the underlying injury.

Patterns of clinical presentation

Hematuria

Nephritic
syndrome

Nephrotic
syndrome

RPGN

Hypertension

Renal failure

Clinical syndromes are not a direct substitute for biopsy class.

Applied case: Focal proliferative lupus

- A patient with SLE develops hematuria, red cell casts, and proteinuria.
- Biopsy shows necrosis and crescents in fewer than half of glomeruli.
- Question: Which lupus nephritis class best fits the distribution and injury pattern?

Extent of glomerular involvement

Focal: Class III

Fewer than half of
glomeruli involved



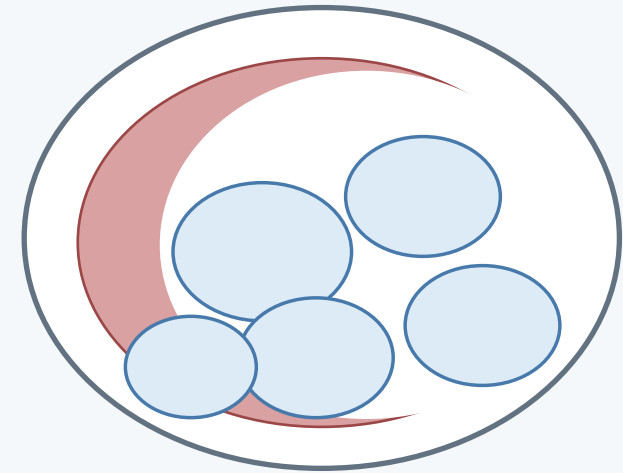
< 50%

Bar illustrates the definition, not a case dataset.

Case interpretation: Class III

- Fewer than half of glomeruli affected is the defining focal distribution.
- Necrotizing capillary injury with crescents supports active proliferative nephritis.
- Red cell casts are compatible with a nephritic sediment during active disease.

Extracapillary crescent formation

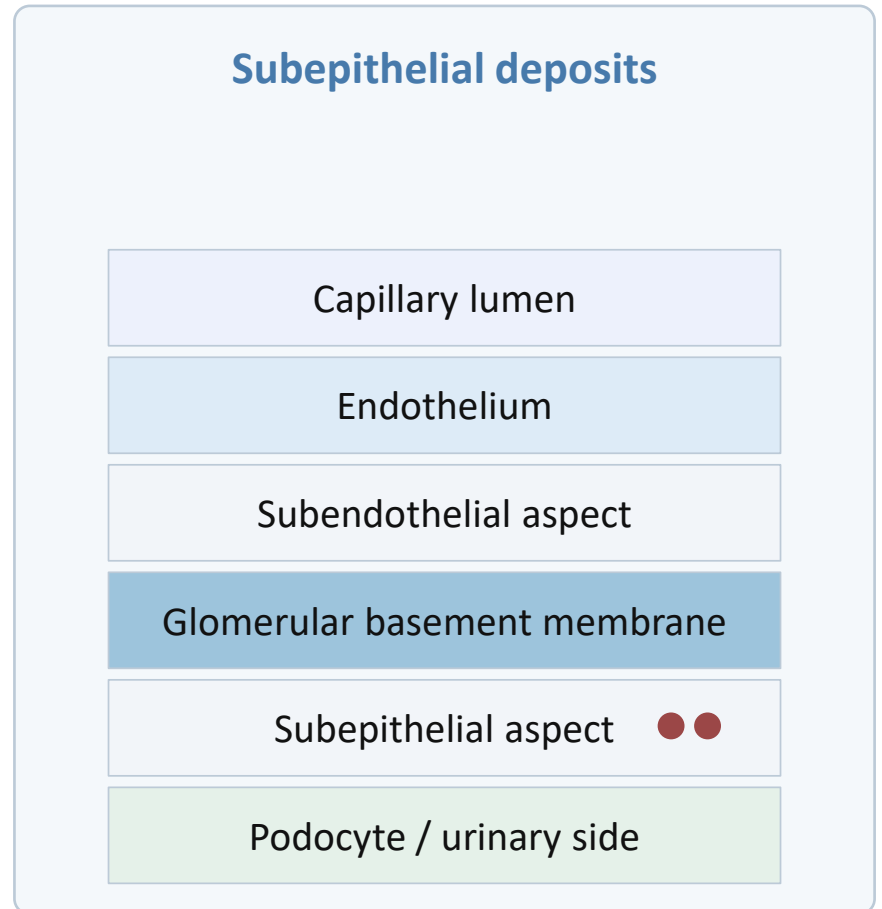


Cellular crescent at tuft periphery

Schematic drawing — not a histologic specimen.

Applied case: Thickened glomerular loops

- A patient with SLE has substantial proteinuria.
- Biopsy shows diffuse capillary-wall thickening and subepithelial immune deposits.
- Question: Which lupus class explains these structural findings?



Red dots: Schematic immune deposits

Case interpretation: Class V

- Diffuse capillary-wall thickening with subepithelial immune complexes characterizes class V.
- Severe proteinuria and nephrotic syndrome are typical clinical consequences.
- Assess separately for concurrent focal or diffuse proliferative lupus nephritis.

Site of deposits shapes injury

III–IV

Subendothelial

Proliferative injury

V

Subepithelial

Proteinuria / nephrosis

Simplified clinicopathologic mapping.

Henoch–Schönlein purpura: A systemic syndrome

- The source describes a childhood-predominant vasculitic syndrome with skin, intestinal, joint, and renal manifestations.
- Purpuric skin lesions are accompanied by abdominal symptoms and arthralgias.
- Glomerular injury is associated with IgA-containing deposits.

Systemic clinical pattern

Skin

Purpuric lesions

Gut

Pain • bleeding

Joints

Arthralgia

Kidney

Hematuria ±
syndrome

The four-organ pattern supports the systemic diagnosis.

Age distribution influences clinical severity

- The syndrome is particularly common in children aged 3–8 years.
- Adults may also develop the disease.
- Renal manifestations tend to be more severe in adults than in children.

Age modifies the renal presentation

Children (often 3–8 years)

Adults (less frequent)



Renal disease usually more severe in adults

Clinical onset may follow respiratory infection

- The source notes that onset often follows an upper respiratory infection.
- Approximately one-third of patients have a background of atopy.
- These associations provide context but are not sufficient to establish renal histologic severity.

Systemic clinical pattern

Skin

Purpuric lesions

Gut

Pain • bleeding

Joints

Arthralgia

Kidney

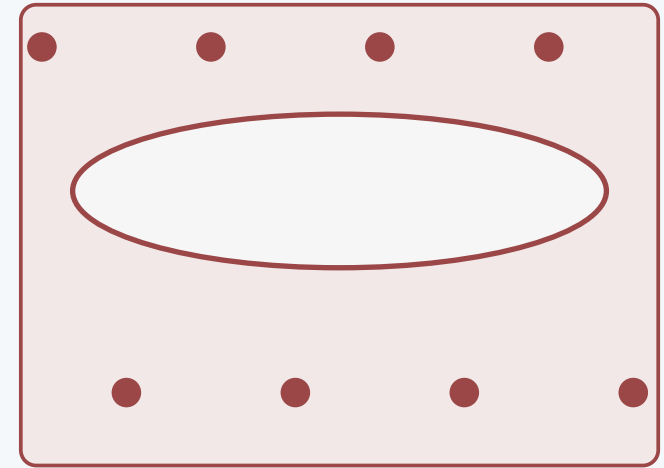
Hematuria ±
syndrome

The four-organ pattern supports the systemic diagnosis.

Purpura has a characteristic distribution

- Purpuric lesions often involve extensor surfaces of the arms and legs.
- Buttocks are another characteristic site.
- Skin biopsy may reveal small-vessel necrotizing vasculitis and subepidermal hemorrhage.

Small-vessel injury in the skin



IgA ± IgG/C3 in dermal
vessels

Simplified vascular diagram; Not a skin section.

Gastrointestinal symptoms are integral

- Abdominal pain is common and may be accompanied by vomiting.
- Intestinal bleeding can occur as part of systemic vascular involvement.
- The presence of gastrointestinal involvement helps distinguish a systemic syndrome from isolated renal disease.

Systemic clinical pattern

Skin

Purpuric lesions

Gut

Pain • bleeding

Joints

Arthralgia

Kidney

Hematuria ±
syndrome

The four-organ pattern supports the systemic diagnosis.

Arthralgia reinforces the multisystem pattern

- Joint symptoms occur alongside skin and abdominal manifestations.
- The combination of purpura, abdominal symptoms, arthralgia, and renal findings is diagnostically informative.
- Systemic involvement should be considered when interpreting IgA-associated glomerular disease.

Systemic clinical pattern

Skin

Purpuric lesions

Gut

Pain • bleeding

Joints

Arthralgia

Kidney

Hematuria ±
syndrome

The four-organ pattern supports the systemic diagnosis.

Renal disease affects about one-third

- Renal manifestations in approximately one-third of patients.
- Gross or microscopic hematuria is a common renal sign.
- Nephritic or nephrotic syndrome may occur, including mixed clinical presentations.

Patterns of clinical presentation

Hematuria

Nephritic
syndrome

Nephrotic
syndrome

RPGN

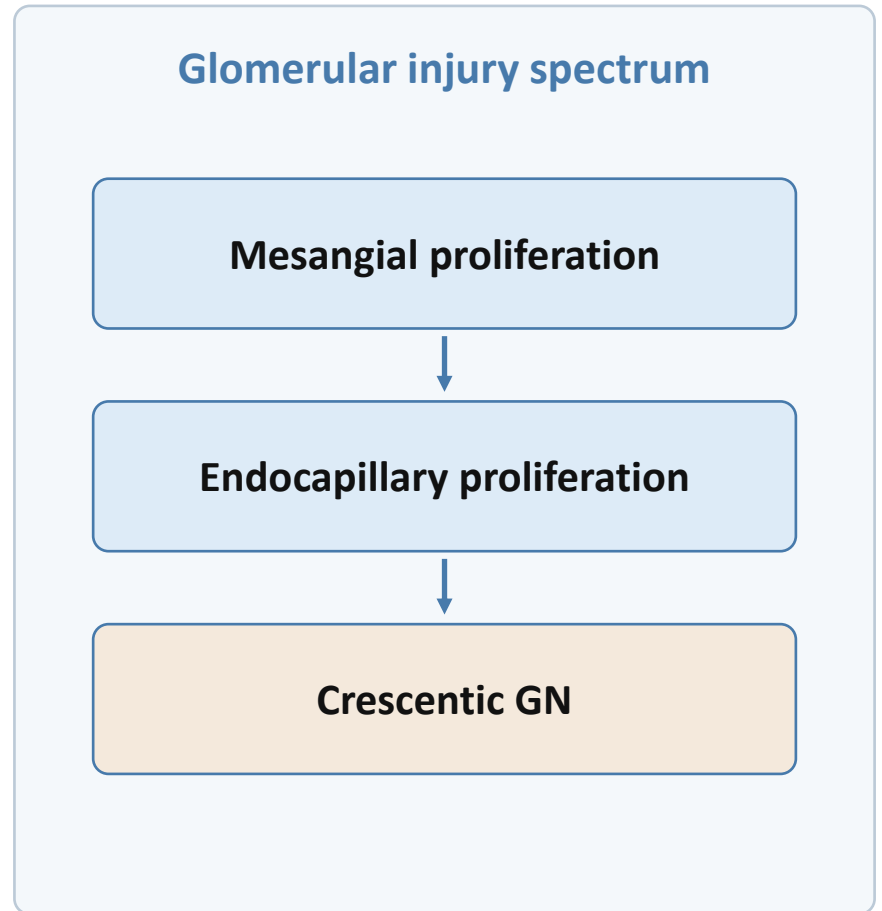
Hypertension

Renal failure

Clinical syndromes are not a direct substitute for biopsy class.

HSP glomerular morphology is heterogeneous

- Mild focal mesangial proliferation may be the only finding.
- Some biopsies show diffuse mesangial or endocapillary proliferation.
- The severe end includes crescentic glomerulonephritis.

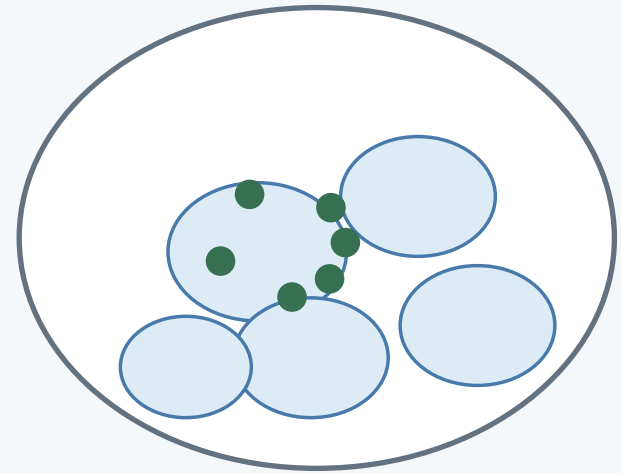


Range of observed morphologies; Not inevitable progression.

Mesangial IgA is the defining IF clue

- IgA deposits are found in the mesangial regions on immunofluorescence.
- IgG and C3 may accompany IgA.
- Deposits can sometimes extend from mesangial areas to capillary loops.

IgA-associated mesangial pattern



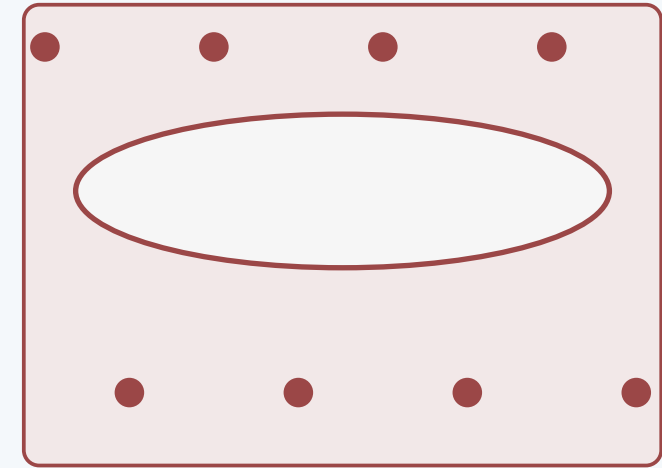
Mesangial IgA deposits

Schematic drawing — not a histologic specimen.

The skin and glomerulus share immune deposits

- Dermal small vessels show necrotizing vasculitis with subepidermal hemorrhage.
- IgA, sometimes with IgG and C3, is demonstrable in involved vessels.
- These deposits support an immunologically mediated multisystem vascular process.

Small-vessel injury in the skin



IgA $\pm$ IgG/C3 in dermal vessels

Simplified vascular diagram; Not a skin section.

IgA nephropathy and HSP are closely related

- Both conditions show a mesangial distribution of IgA deposits.
- The source considers them manifestations of a closely related disease process.
- The systemic skin, gastrointestinal, and joint findings distinguish the purpura syndrome clinically.

Immunofluorescence patterns

Lupus nephritis

IgG; Often also IgA, IgM, C3, C1q

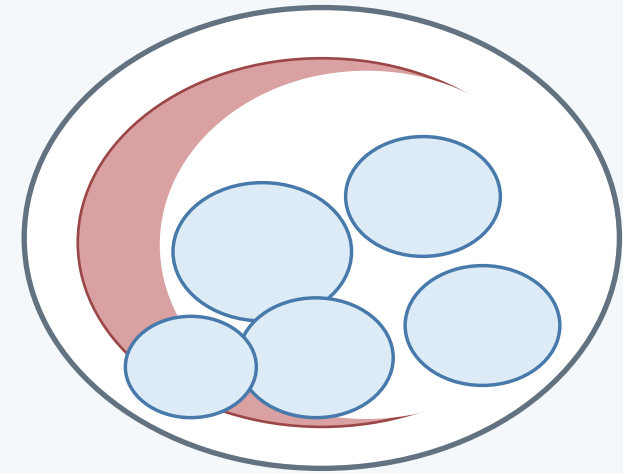
Henoch–Schönlein purpura

Mesangial IgA ± IgG/C3

Crescents can signal a severe renal variant

- A small subset develops rapidly progressive glomerulonephritis with numerous crescents.
- Severe crescentic disease is described particularly in adults.
- This lesion is more concerning than a mild isolated mesangial proliferation.

Extracapillary crescent formation



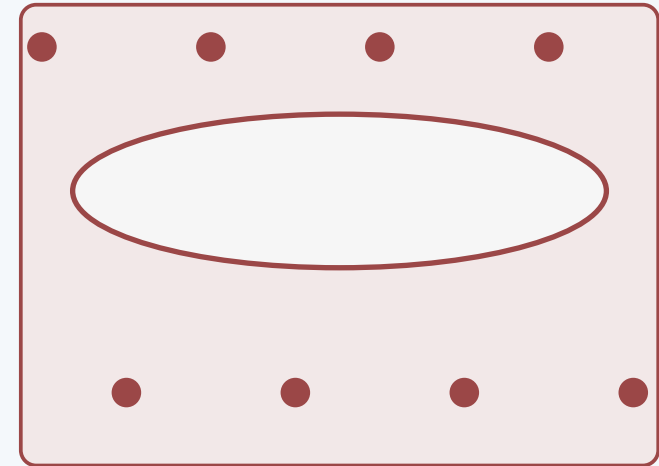
Cellular crescent at tuft periphery

Schematic drawing — not a histologic specimen.

Extra-renal vasculitis is usually more conspicuous

- Small-vessel vasculitis characterizes cutaneous lesions.
- The gastrointestinal tract may also have vasculitic injury.
- Vasculitis is relatively uncommon within the kidney itself.

Small-vessel injury in the skin



IgA ± IgG/C3 in dermal
vessels

Simplified vascular diagram; Not a skin section.

The course and prognosis vary

- Hematuria may recur over many years following onset.
- Most children have an excellent overall prognosis.
- Diffuse glomerular lesions, crescents, or nephrotic syndrome are associated with less favorable renal outcomes.

Morphology associated with poorer course

Diffuse renal involvement

Crescents

Nephrotic syndrome

Risk associations as described; No numeric score is implied.

Lupus nephritis versus Henoch–Schönlein purpura

Feature	Lupus nephritis	Henoch–Schönlein purpura
Systemic setting	SLE	Purpura + gut + joint manifestations
Immune pattern	IgG often with IgA/IgM, C3, C1q	Mesangial IgA ± IgG/C3
Morphologic range	Six lupus classes	Mesangial → proliferative → crescentic
Associated disease	Tubulointerstitial and vascular lesions	Dermal small-vessel vasculitis
Clinical concern	Activity, extent, scarring	Adult severity and crescents

Applied case: Multisystem IgA-associated disease

- A child develops purpura on the legs and buttocks, abdominal pain, and hematuria.
- Renal biopsy shows mesangial proliferation and predominant mesangial IgA deposits.
- Question: What syndrome best unifies the extrarenal and renal manifestations?

Systemic clinical pattern

Skin

Purpuric lesions

Gut

Pain • bleeding

Joints

Arthralgia

Kidney

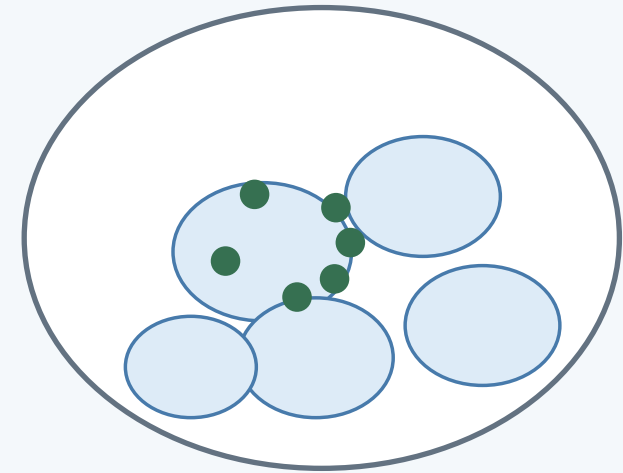
Hematuria ±
syndrome

The four-organ pattern supports the systemic diagnosis.

Case interpretation: Henoch–Schönlein purpura

- Purpura with abdominal symptoms and hematuria supports the systemic syndrome.
- Mesangial IgA staining closely links the renal lesion with IgA-associated nephropathy.
- Renal severity still depends on the observed injury pattern, especially diffuse proliferation or crescents.

IgA-associated mesangial pattern



Mesangial IgA deposits

Schematic drawing — not a histologic specimen.

The crucial tissue discriminators

- Deposit predominance: Lupus often has IgG with several other reactants; HSP has mesangial IgA.
- Distribution: Mesangial versus capillary wall and focal versus diffuse.
- Injury state: Active necrosis/proliferation versus established sclerosis.

Immunofluorescence patterns

Lupus nephritis

IgG; Often also IgA, IgM, C3, C1q

Henoch–Schönlein purpura

Mesangial IgA $\pm$ IgG/C3

Integrated pathology checklist

- Identify the systemic syndrome and renal clinical manifestations.
- Use LM to describe cell proliferation, crescents, necrosis, membrane thickening, and scarring.
- Use IF and EM to map immune deposits and integrate extraglomerular lesions.

Pathology interpretation sequence

Distribution

Focal versus diffuse

Compartment

Mesangial vs capillary wall

Activity

Necrosis / crescents

Chronicity

Glomerular sclerosis

Core concepts to retain

- Systemic lupus erythematosus produces diverse renal immune-complex lesions, formally described in six classes.
- Deposit localization, extent, and inflammatory activity explain key patterns of clinical presentation.
- Henoch–Schönlein purpura links a systemic vasculitic syndrome to predominant mesangial IgA deposition.

Immune-complex glomerular injury

Systemic autoimmunity



Glomerular deposits



Inflammatory response



Capillary injury

Causal framework summarized from the described deposits and lesions.

Reference and scope

- Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th edition (2026), The Kidney.
- Primary teaching section: Glomerular Lesions Associated With Systemic Diseases, including lupus nephritis and Henoch–Schönlein purpura.
- The textbook figure series depicts proliferative lesions, wire loops, ultrastructural deposits, and granular IgG staining.