Glomerular Diseases

By: Mahmoud Abu Znaid, MD.

Nephrologist and Internal Medicine Specialist

Dr. MAHMOUD ABUZNAID
د.محمود أبو زنيد

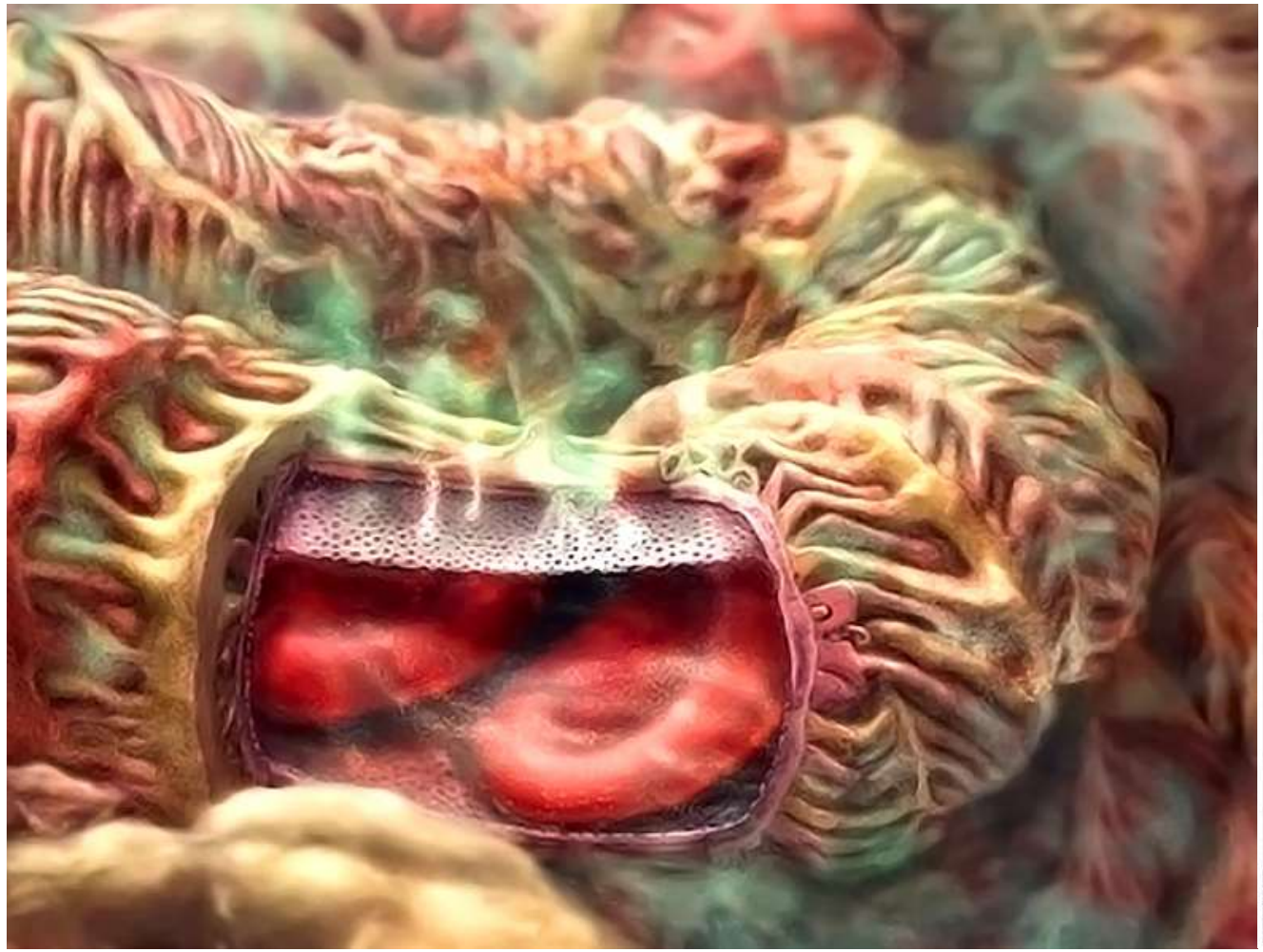
Basic Information

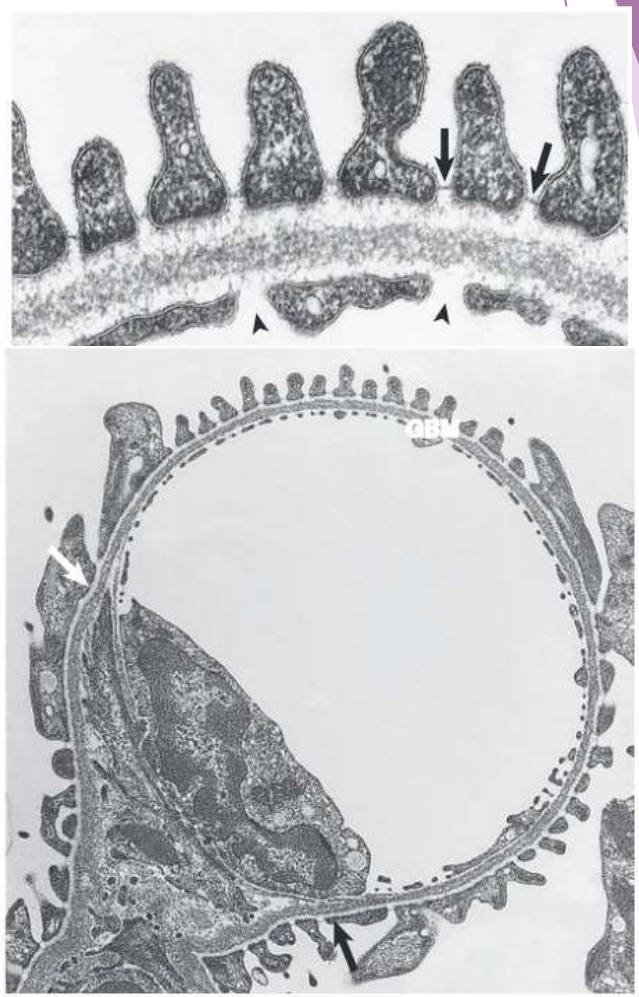
- Glomerular disease includes a group of disorders in which the **glomerular filtration barrier** is altered.
- The resultant change in filtration may be accompanied by **proteinuria** (in nephrotic disorders) or **hematuria** (in nephritic disorders), or **both**.
- Familiarity with the causes of **nephrotic** and **nephritic** disorders will guide the **history**, **physical**, and **laboratory** examination of patients presenting with abnormal urinary sediment.

Basic Information

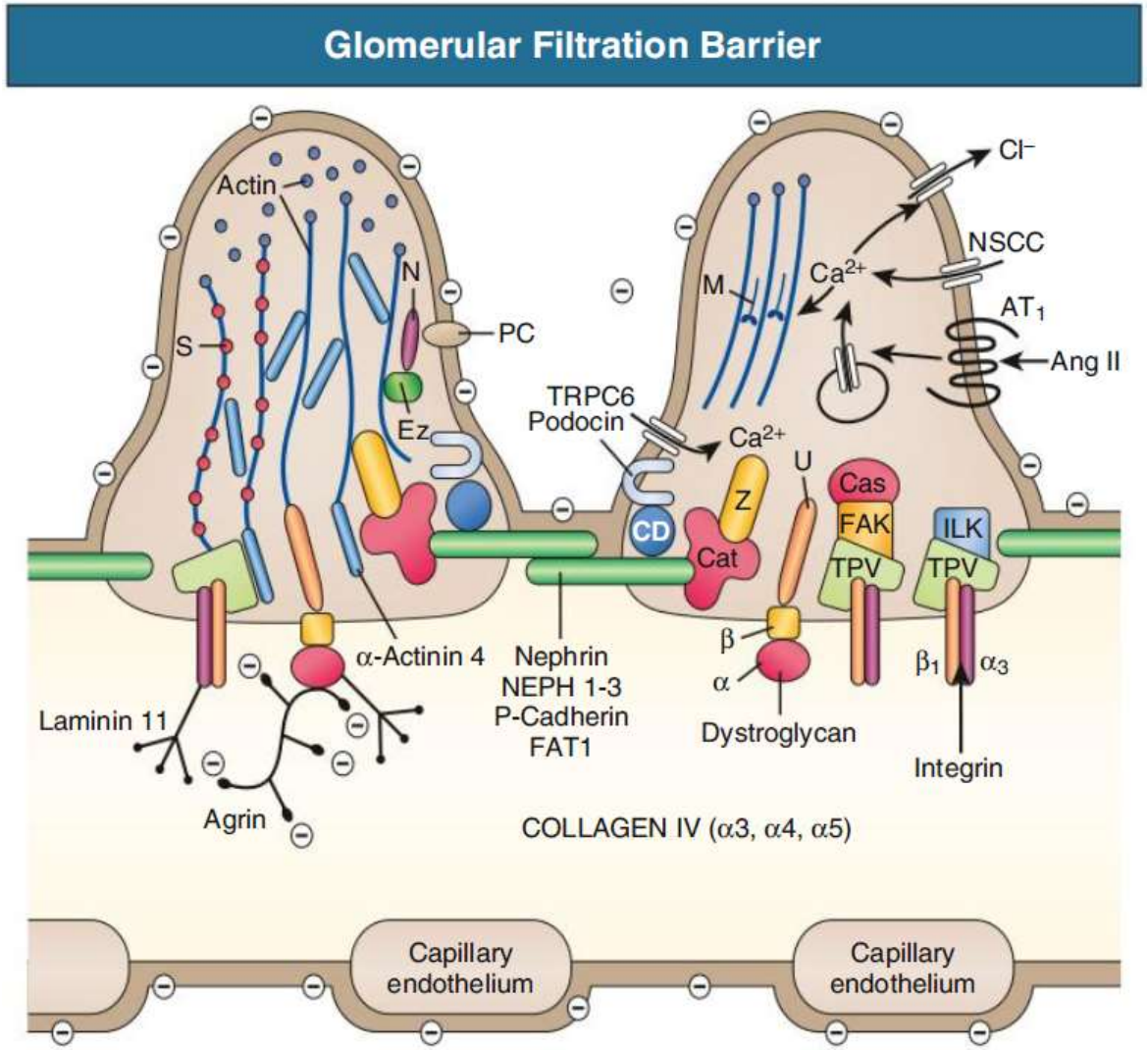
- Glomerular disease refers to disorders of the **glomerular filtration barrier** that occur either as a **primary disorder** or as a result of **other diseases** toxins, or infections
- The glomerular filtration barrier may be affected so that proteinuria results; if the proteinuria is **greater than 3.5 g/24 hr** (nephrotic-range proteinuria), a nephrotic syndrome may result
- If glomerular filtration barrier is affected so that **hematuria** results (often accompanied by proteinuria), the disorder is classified as nephritic (glomerulonephritis [GN])
- Additional clinical features typically accompany nephrotic and nephritic disorders
- **Significant overlap may exist** among disorders that result in nephrotic syndrome and GN

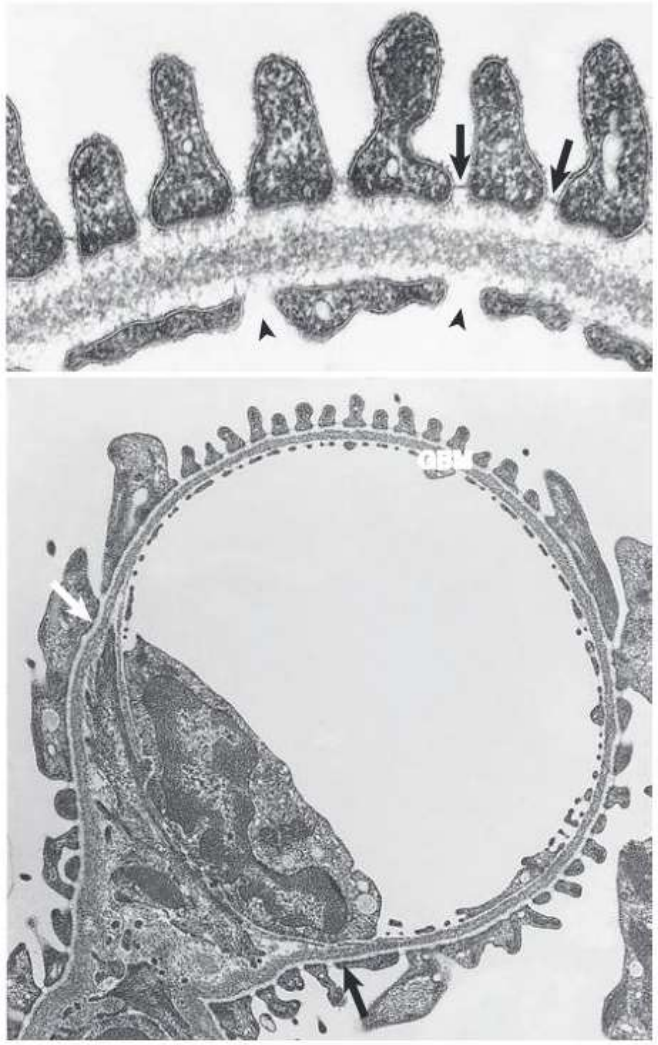
Glomerular Filtration Barrier





Glomerular Filtration Barrier





Nephrotic Syndrome

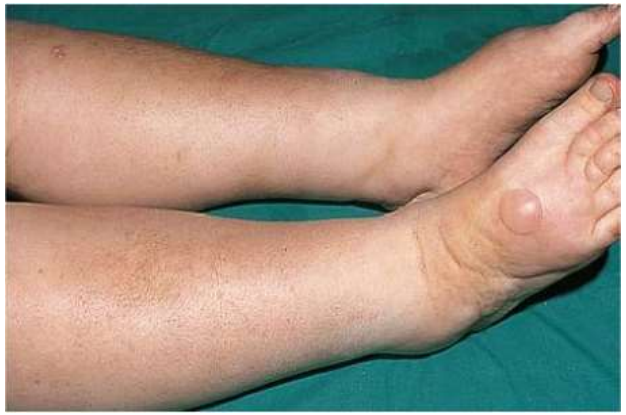
Basic Information

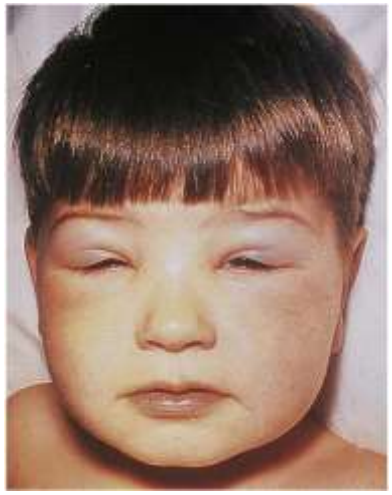
- General features of nephrotic syndrome Nephrotic syndrome is defined by the presence of:
  - Proteinuria (>3.5 g/24 hr)
  - Hypoalbuminemia (<3.0 g/dL)
  - Edema results from hypoalbuminemia
  - Hyperlipidemia
  - Lipiduria
- Features of specific causes of nephrotic syndrome all may be:
  - Idiopathic
  - related to secondary causes

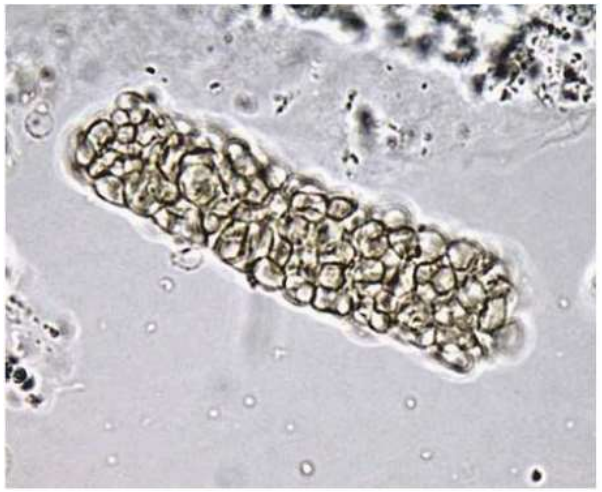
Basic Information

- **Blood pressure is variable** depending on the underlying disease
- Usually **normal** in minimal change disease and membranous nephropathy
- Frequently **elevated** in focal segmental glomerulosclerosis (FSGS)
- Urinary loss of anticoagulant proteins (e.g., protein C, protein S, antithrombin III) may result in a hypercoagulable state
- **Renal vein thrombosis** may occur with any cause of nephrotic syndrome but is most common with **membranous GN**
- **Renal vein thrombosis** often presents as sudden onset worsening of renal insufficiency, worsening proteinuria, hematuria, or flank pain
- Loss of immunoglobulins may result in a relative immune-deficient state

Clinical Presentation







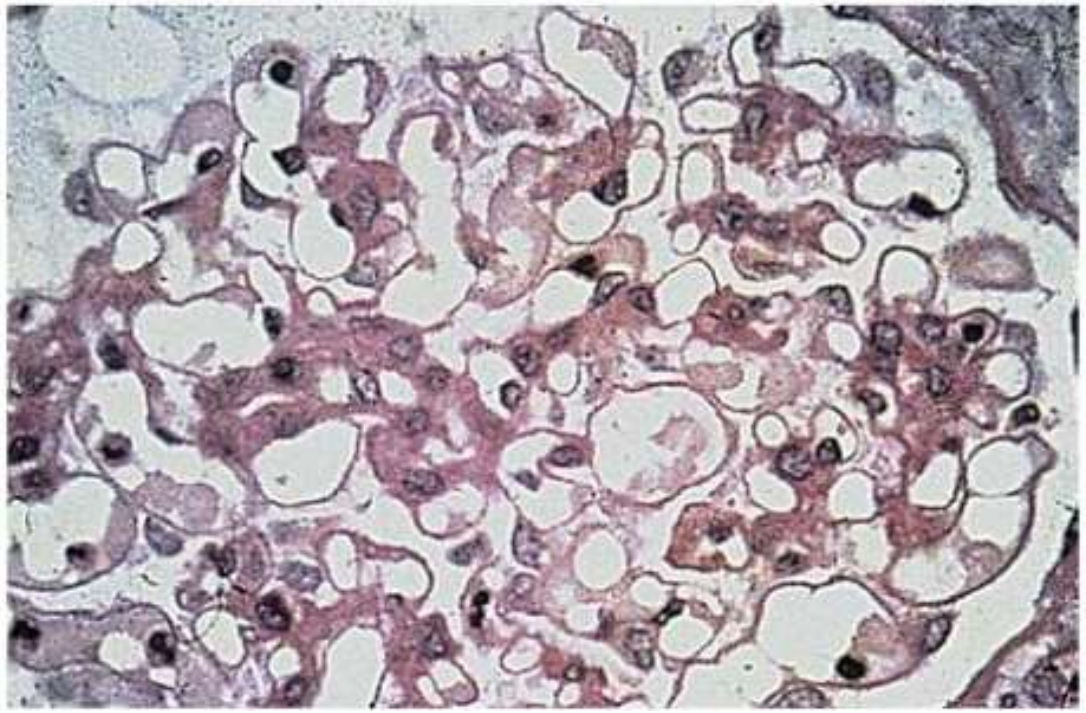
Minimal change disease

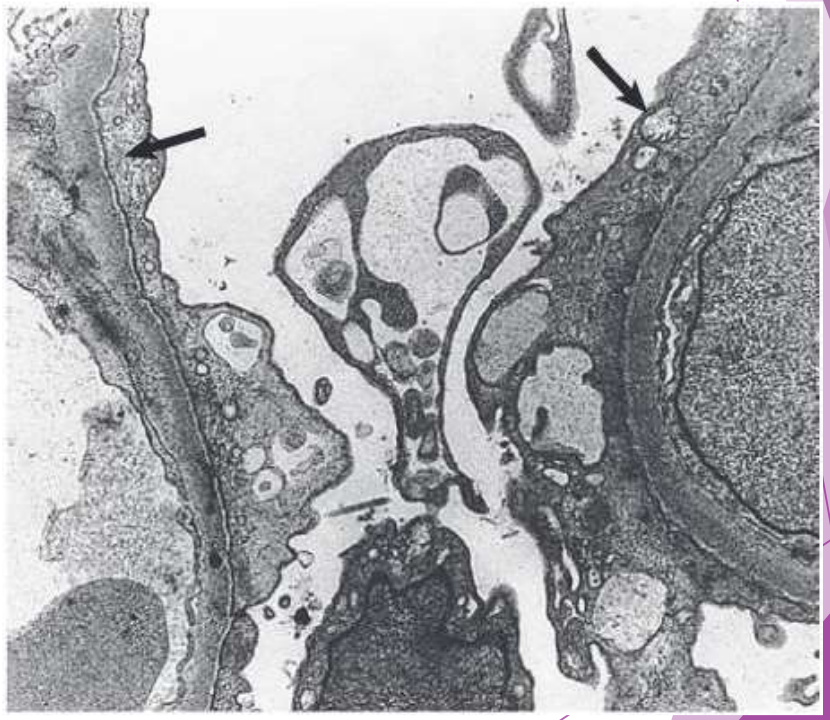
- **Rapid onset**; patient is normal one day and has edema the next
- Glomerular filtration rate **(GFR) remains normal**
- **Massive proteinuria** common (>4 g/24 hr)
- Pathologic hallmark is **normal-appearing glomeruli on microscopy** and generalized **loss of foot processes of podocytes on electron microscopy**

Minimal change disease

- **Typical Age of Onset:** <12 years or mid-60s
- **Associated Diseases:**
  - NSAIDs
  - Lymphoma
  - Bee sting
  - Drugs: Gold, Penicillamine, etc.
- **Pathologic Findings:**
  - Light microscopy: Normal
  - Immunofluorescence: Normal
  - Electron microscopy: Diffuse podocyte foot process effacement

Minimal change disease





Minimal change disease

- **Treatment:**
  - Corticosteroids
  - Cyclosporin or cyclophosphamide or mycophenolate mofetil for relapse
- **Response:**
  - Children respond within 2 wk
  - Adults respond in 4–8 wk, but often relapse

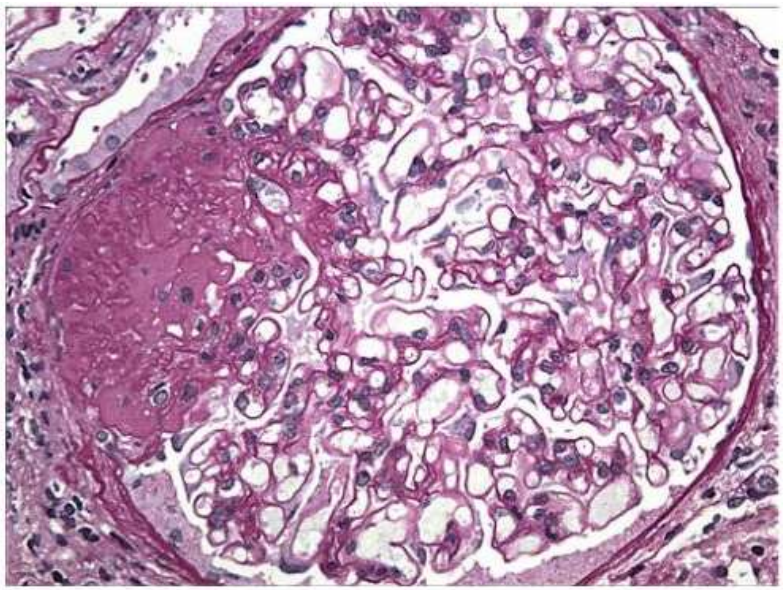
Focal segmental glomerulosclerosis (FSGS)

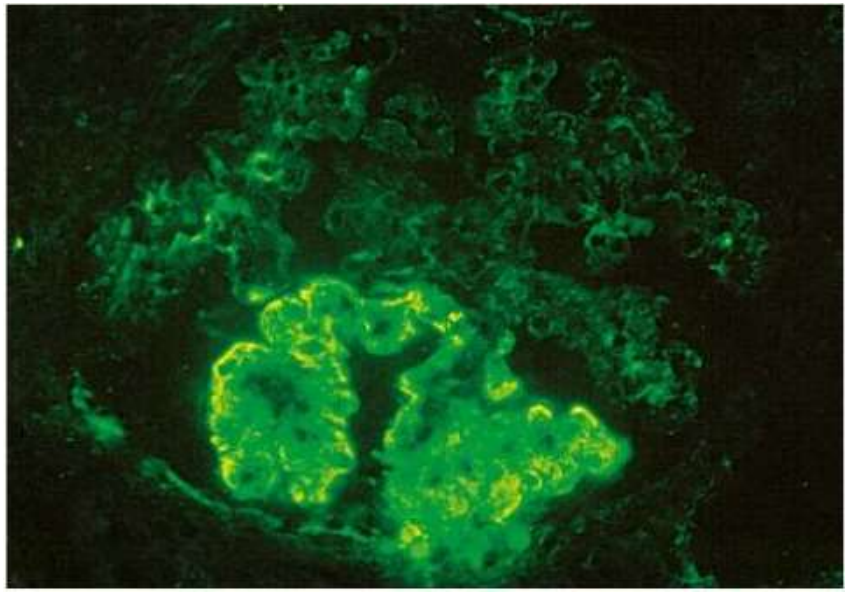
- "Classic" FSGS common in **African Americans**
- Slow onset with progressive decrease in GFR if untreated
- Creatinine normal, but may be elevated at presentation (elevated creatinine indicates poorer prognosis)
- Proteinuria not massive (2–4 g/24 hr)
- Includes findings seen with hyperfiltration injury (secondary FSGS)
- Human immunodeficiency virus (HIV)–associated nephropathy (HIVAN) presents with a collapsing FSGS (vs. classic FSGS)
  - Characterized by a very rapid onset of renal failure (over months) with massive proteinuria
  - Collapsing FSGS may occasionally be seen in non–HIV-infected patients

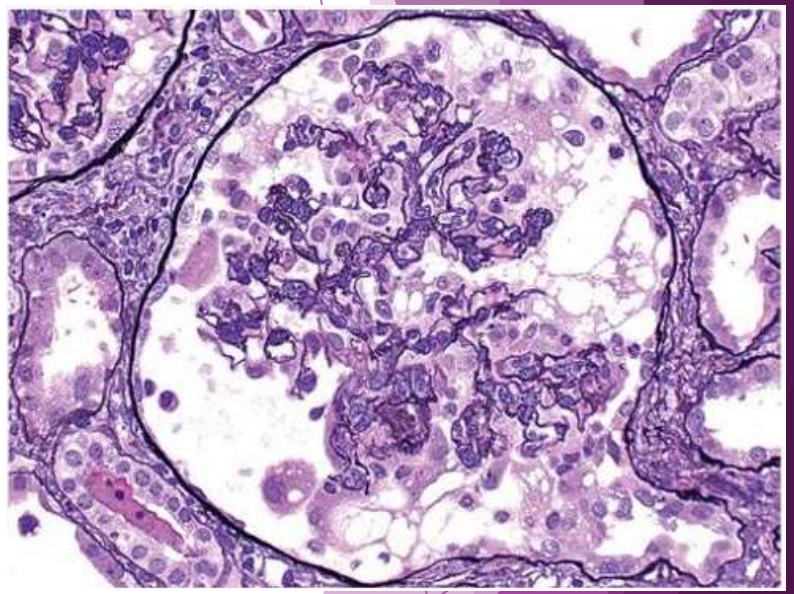
Focal segmental glomerulosclerosis (FSGS)

- **Typical Age of Onset:** Early teens to mid-30s
- **Associated Diseases:**
  - Hyperfiltration (seen in morbid obesity; may result from nephron loss due to other causes)
  - HIV (collapsing variant)
  - Heroin nephropathy
- **Pathologic Findings:**
  - Light microscopy: Focal and segmental glomerulosclerosis
  - Immunofluorescence: May show nonspecific IgM deposition
  - Electron microscopy: Foot process effacement Collapsing variant seen on light microscopy in those with HIV

Focal segmental glomerulosclerosis (FSGS)







Focal segmental glomerulosclerosis (FSGS)

- **Treatment:**
  - Corticosteroids + ACE-I
  - Cyclosporin or mycophenolate mofetil may be added to steroids
- **Response:**
  - 40–60%
  - Response takes 4–13 mo

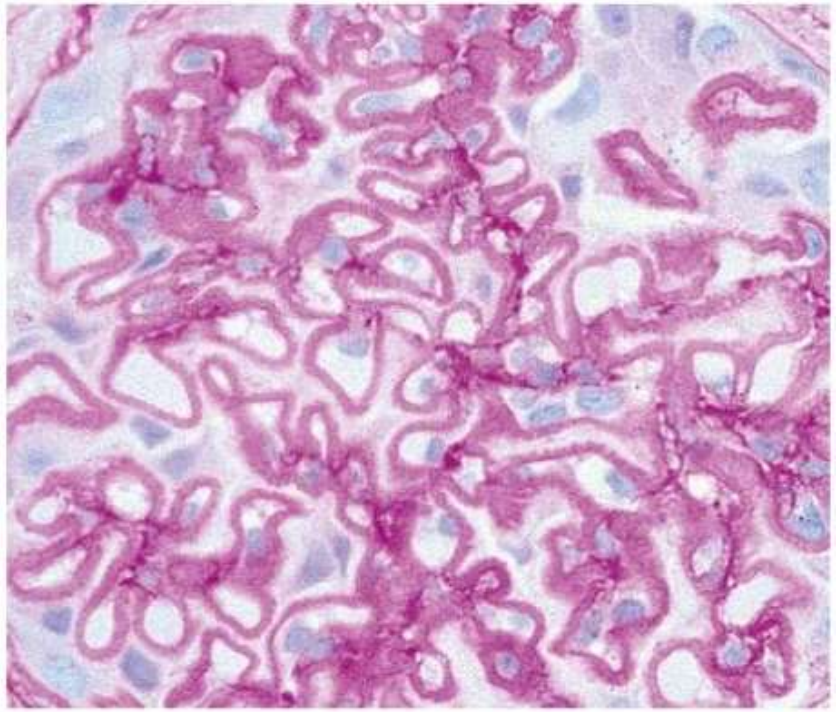
Membranous GN

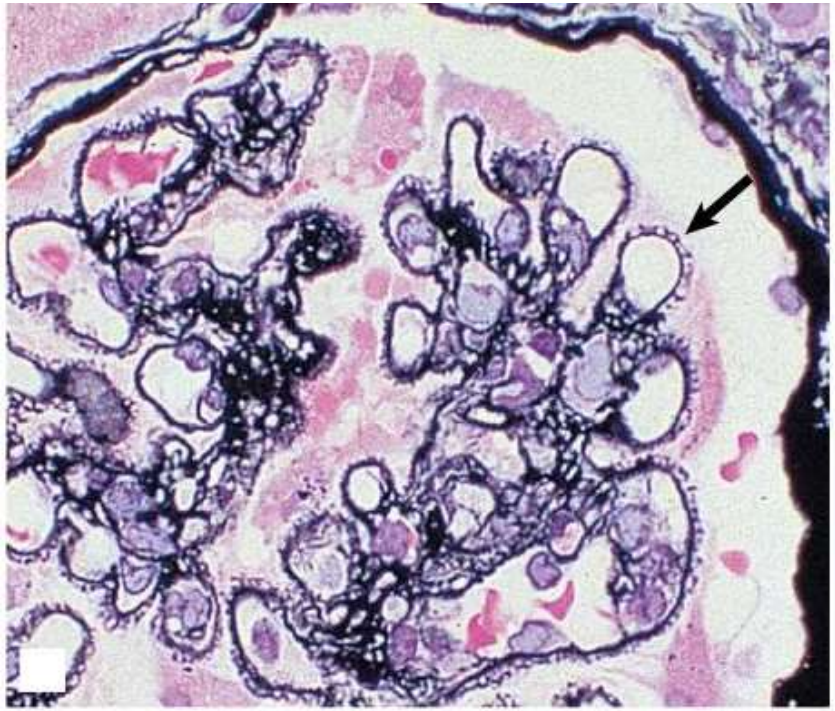
- Was the most common cause of nephrotic syndrome in adults now **FSGS** is most common cause of nephrotic syndrome
- Associated with presence of **anti-phospholipase A2 (PLA2R) antibodies**
- Slow onset with loss of GFR, usually over years
- Spontaneous remission in one third of patients
- Creatinine often normal; if creatinine deteriorates rapidly, consider renal vein thrombosis
- Proteinuria varies from subnephrotic range to massive (>20 g/24 hr)

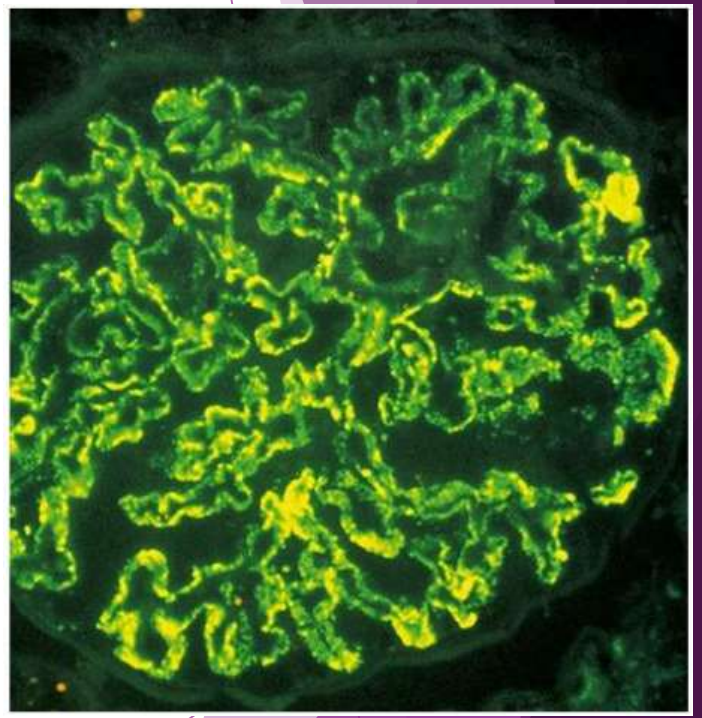
Membranous GN

- **Typical Age of Onset:** Mid-30s to mid-60s
- **Associated Diseases:**
  - Adenocarcinoma (breast, bowel, lung)
  - Hepatitis B
  - Systemic lupus erythematosus
  - Drug reaction (NSAID)
- **Pathologic Findings:**
  - Light microscopy: Thickened capillary loops
  - Immunofluorescence: Deposition of immunoglobulin and complement
  - Electron microscopy: Subepithelial immune complex deposition

Membranous GN







Membranous GN

- **Treatment:**
  - ACE-I for all patients
  - Low risk for progression (proteinuria <4 g/day + creatinine clearance >80 mL/min):
    - Observation with close follow-up
  - Moderate risk for progression (persistent proteinuria between 4 g/day and 8 g/day + creatinine clearance >80 mL/min):
    - Corticosteroids + Rituximab
    - Corticosteroids + cytotoxic agents
  - High risk for progression (persistent proteinuria >8 g/day and/or abnormal or declining creatinine clearance):
    - Corticosteroids + cytotoxic agents
- **Response:**
  - Variable, dependent on prognostic indicators
  - One third remit spontaneously without treatment—treatment decisions should be based on persistent proteinuria on 24-hr urine collections

Secondary causes of nephrotic syndrome

Diabetes

- **Diabetic nephropathy** is the **most common** cause of nephrotic proteinuria
- Earliest evidence of diabetic nephropathy is:
  - **microalbuminuria (Moderately increased)** (30–300 mg albumin/24 hr), which then progresses to
  - **albuminuria (severely increased)** (>300 mg/24 hr) and may progress to
  - **nephrotic-range proteinuria**
- Once overt proteinuria has set in, renal function inevitably declines
- Average time to end-stage renal disease (ESRD) after onset of proteinuria is 10 years
- Progression to ESRD may be delayed by angiotensin- converting enzyme (ACE) inhibitors or angiotensin receptor blockers

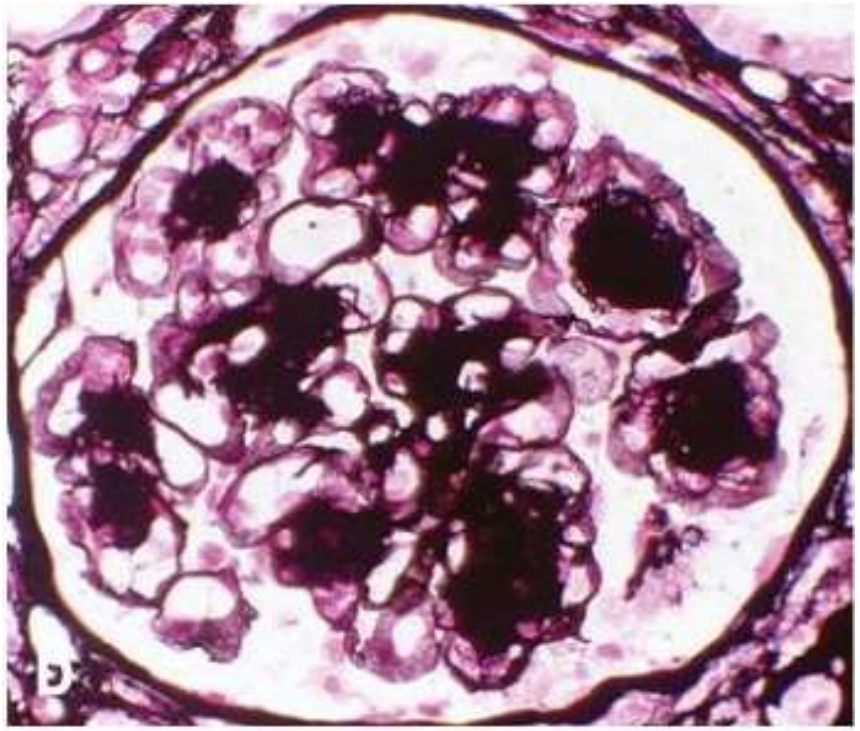
Diabetes

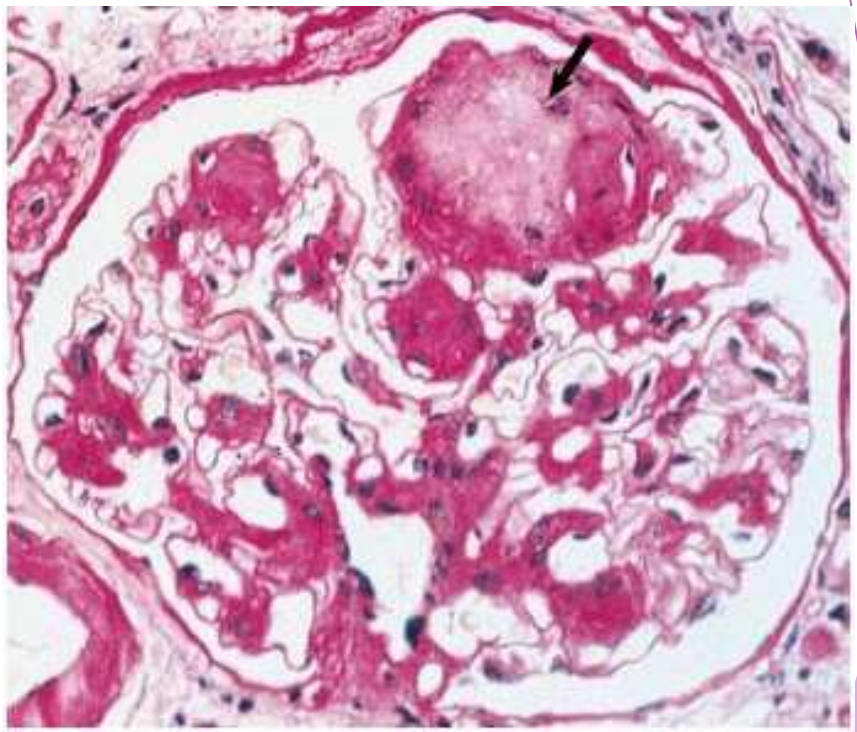
- Diabetic nephropathy **rarely develops before 10 years duration** of diabetes
- **Type 1 diabetics** with nephropathy **95%** also have **retinopathy**
- In a patient with type 1 diabetes and nephropathy, the absence of retinopathy should prompt consideration of nondiabetic etiology of proteinuria/nephropathy
- **Type 2 diabetics** with nephropathy **50%** to **75%** have **retinopathy**
- Nephropathy in the absence of retinopathy in a patient with type 2 diabetes is therefore more common than with type 1 diabetes, but should still prompt consideration of other causes of nephropathy
- If retinopathy is present in patients with type 2, almost 100% have nephropathy

Diabetes

- All diabetic patients with **proteinuria should be evaluated for other systemic diseases** (e.g., hepatitis B and C, systemic lupus erythematosus [SLE], and monoclonal gammopathy) because up to **20% have either primary or superimposed nondiabetic cause of proteinuria**
- Clinical scenarios in which to consider renal biopsy in diabetics
  - Presence of proteinuria with less than 10 years duration of diabetes
  - Presence of significant hematuria or red blood cell (RBC) casts on urinalysis
  - Absence of retinopathy in patients with type 1 diabetes and possibly type 2 diabetes
  - Any clinical or laboratory evidence of other systemic disease

Diabetes

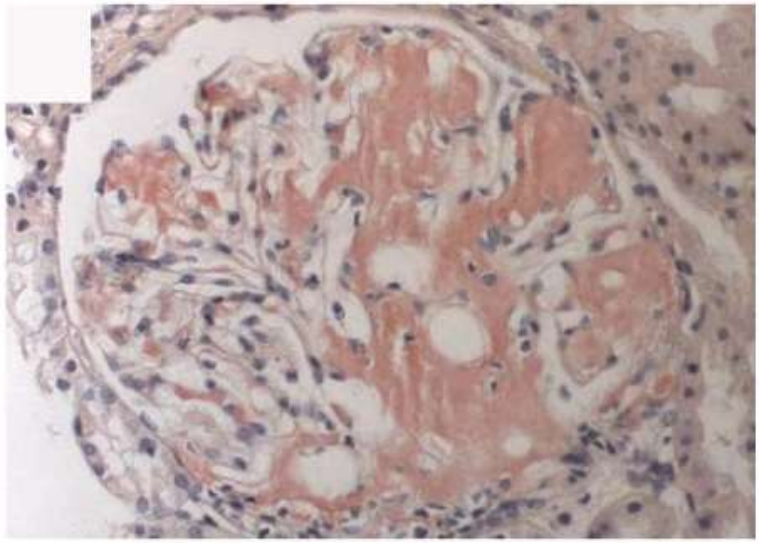


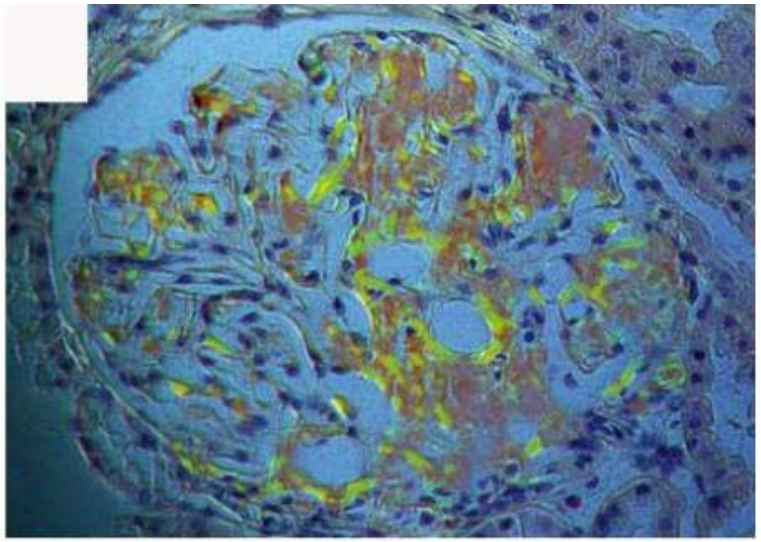


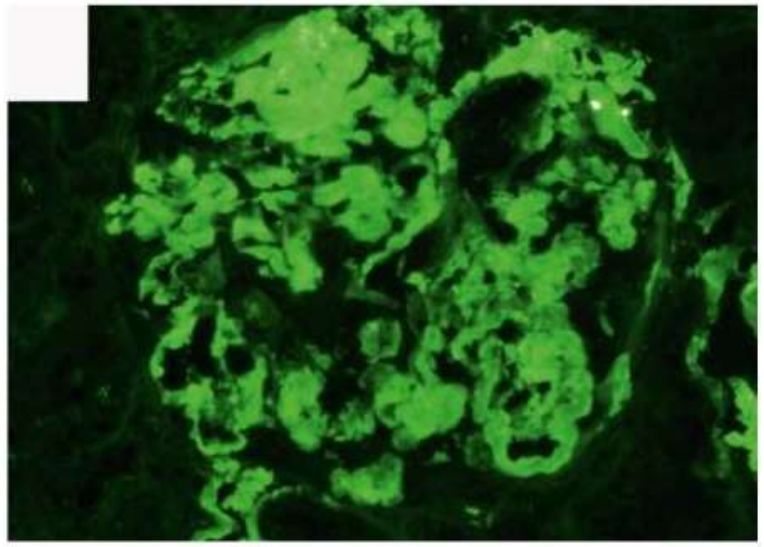
Amyloidosis

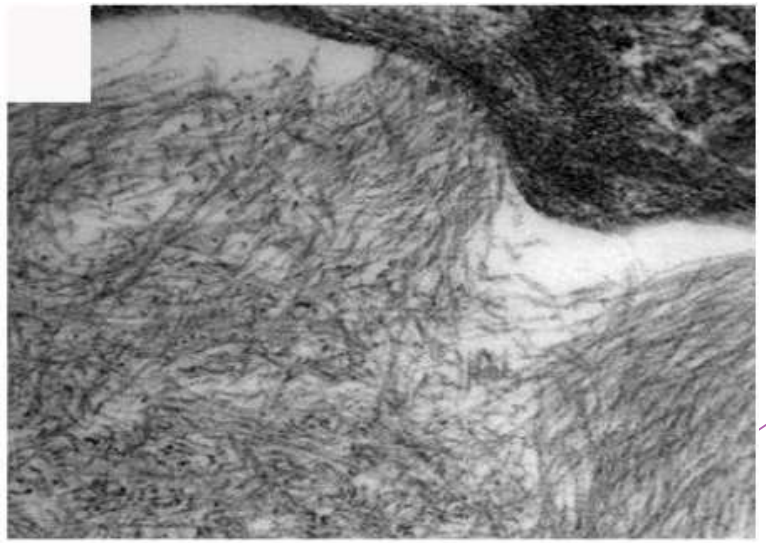
- Systemic disease with extracellular deposition of **amyloid** in various organs
- Most common is **AL amyloid (primary amyloidosis)** with deposits of light-chain immunoglobulin
- **Secondary (AA amyloid)** associated with chronic inflammatory or infectious states
- AL amyloid is caused by plasma cell dyscrasia with overt multiple myeloma in 20% of cases
- 90% have monoclonal light chains (Bence Jones proteins) in urine or blood
- Proteinuria is the consistent feature with associated renal insufficiency
- Nephrotic syndrome in patient older than 50 years— should have a high index of suspicion for AL amyloid
- Generally **poor prognosis**, but may respond to chemotherapy

Amyloidosis









Diagnosis of Nephrotic disorders

- **Serologic evaluation:** antinuclear antibody, hepatitis B surface antigen, anti-hepatitis C antibody, serum and urine protein electrophoresis, HIV antibody, complement levels
- **Kidney biopsy needed** in most patients
- All patients with rapidly rising creatinine, biopsy is urgent
- Diabetics with consistent time course (>10 years' disease), no other systemic disease or serologic abnormalities, and presence of retinopathy may not require biopsy for diagnosis
- Nephrotic patients with amyloid diagnosed by biopsy of other organ (e.g., heart, skin, or bone marrow) may not need kidney biopsy

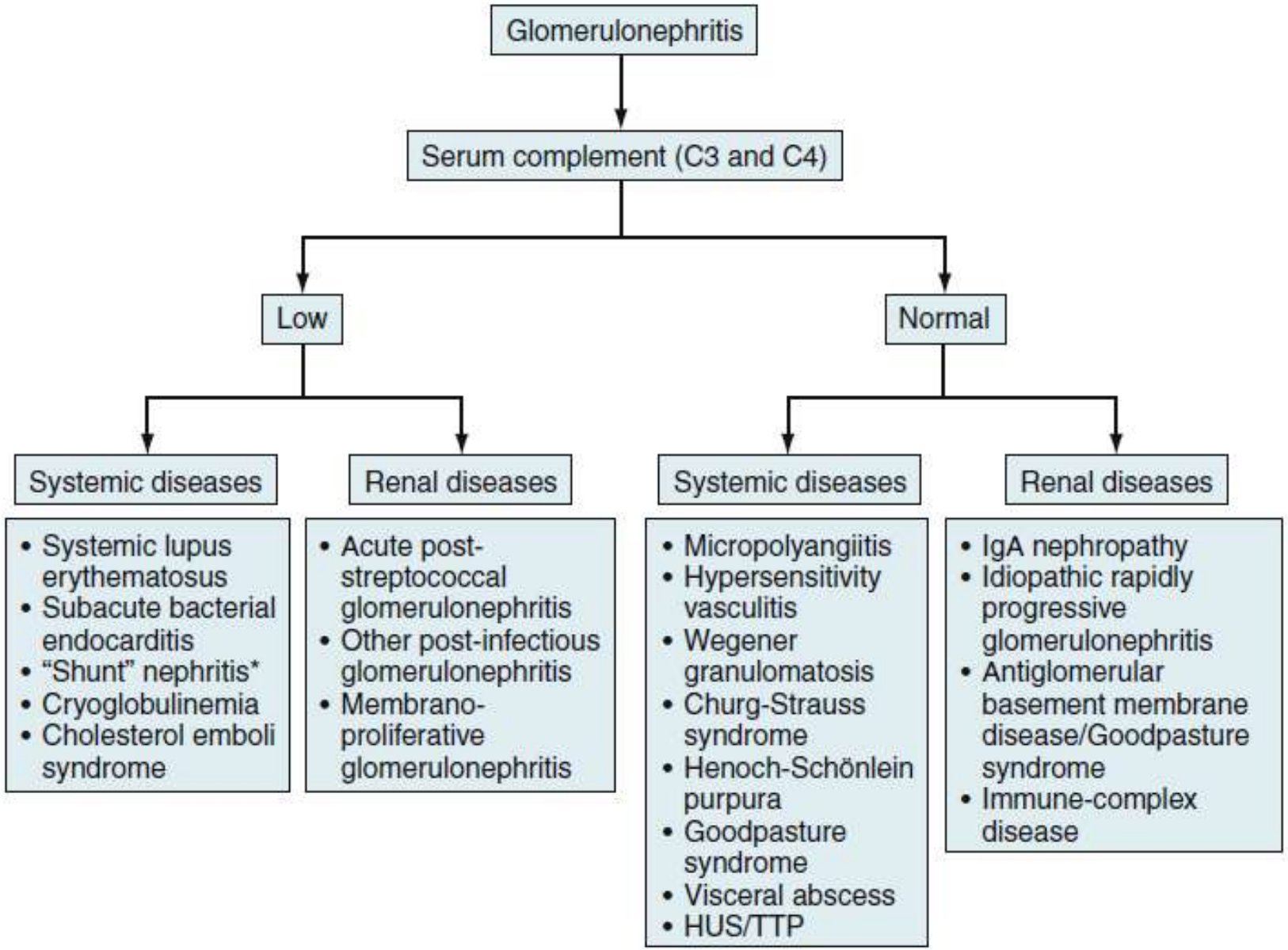
Treatment of Nephrotic disorders

- When nephrotic syndrome is secondary to a systemic disease, treat the underlying disorder
- Additional Reno protective measures:
  - Strict blood pressure control (<130/80 mm Hg)
  - Use of ACE inhibitors or angiotensin receptor blockers
  - Salt restriction and diuretics when indicated
  - Protein restriction
  - Anticoagulation when indicated
  - Aggressive control of cardiovascular risk factors
  - Diabetes control essential in the diabetic patient

Nephritic Disorders

Basic Information

- General features of nephritic disorders (GN)
  - Nephritic disorders are **commonly associated with systemic diseases**
  - Patients typically present with **deteriorating renal function**, mild to moderate **proteinuria**, and an **active urine sediment** (RBCs and RBC casts in the urine)
  - **Hypocomplementemia** suggests lupus nephritis, postinfectious GN, mebranoproliferative GN or cryoglobulinemic GN (present 60–80% of the time in these disorders)
  - Patients may report dark urine **Blood pressure is typically elevated**; peripheral edema may be seen (which may progress to include ascites or pleural effusions)
  - Urine output may be normal or oliguric


Glomerulonephritis
Serum complement (C3 and C4)
Low
Normal
Systemic diseases
Renal diseases
Systemic diseases
Renal diseases
• Systemic lupus erythematosus
• Subacute bacterial endocarditis
• "Shunt" nephritis*
• Cryoglobulinemia
• Cholesterol emboli syndrome
• Acute post-streptococcal glomerulonephritis
• Other post-infectious glomerulonephritis
• Membrano-proliferative glomerulonephritis
• Micropolyangiitis
• Hypersensitivity vasculitis
• Wegener granulomatosis
• Churg-Strauss syndrome
• Henoch-Schönlein purpura
• Goodpasture syndrome
• Visceral abscess
• HUS/TTP
• IgA nephropathy
• Idiopathic rapidly progressive glomerulonephritis
• Antiglomerular basement membrane disease/Goodpasture syndrome
• Immune-complex disease

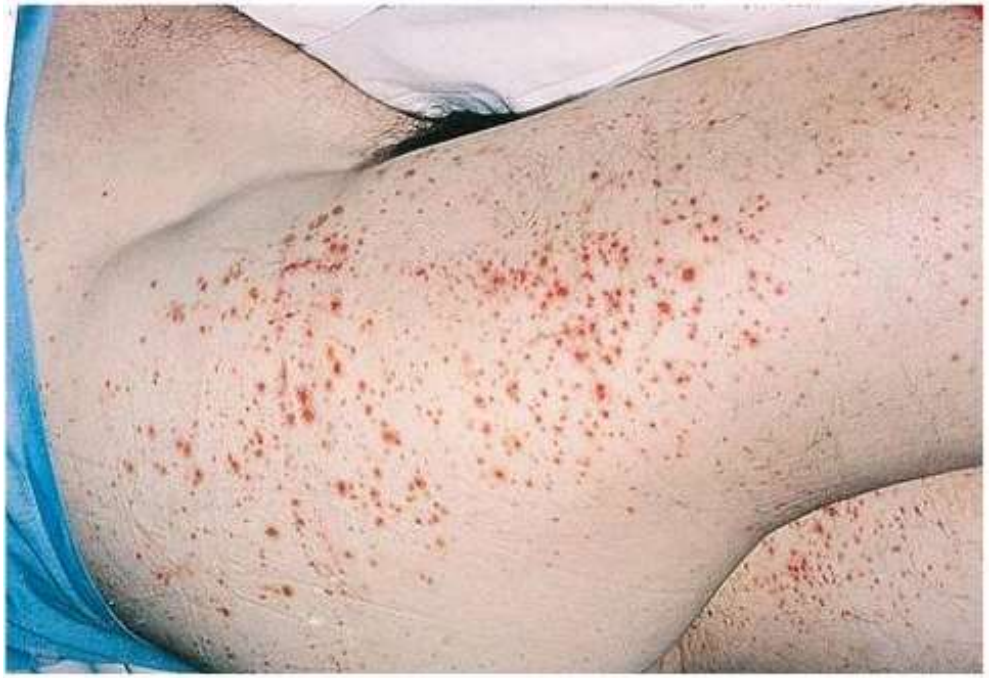
Clinical Presentation

- When evaluating the patient with a diagnosis of nephritis, consideration should be given to a search for a **systemic**, **infectious**, or **postinfectious** cause because GN is often secondary to another disorder
- Systemic diseases associated with GN Frequently present with **rapid-onset renal insufficiency** in the setting of immune dysregulation
- Immunofluorescence (IF) of the kidney biopsy sample is essential in defining the underlying process:
  - Immune complex GN (immune complexes seen on IF)
  - Pauci-immune GN (no immune staining, hence the term pauci-immune)
  - Anti–glomerular basement membrane (anti-GBM) disease (linear GBM staining)
  - Systemic lupus erythematosus (SLE): A secondary cause of proliferative or membranous GN

Henoch-Schönlein purpura (HSP)

- **Manifestations often include:**
  - purpuric rash
  - Arthralgias
  - abdominal pain
  - renal involvement
- Commonly follows upper respiratory infection Immunoglobulin A (IgA) deposition seen in renal mesangial cells (looks exactly like IgA nephropathy)
- Uncommon in adults—**more common in children**; predominates in males

Henoch-Schönlein purpura (HSP)



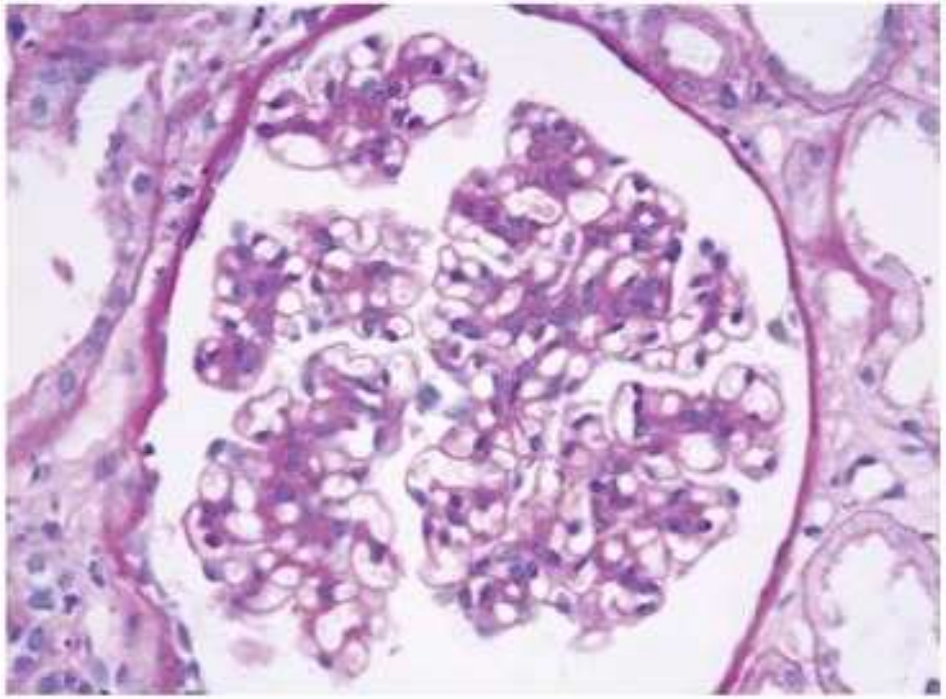
Henoch-Schönlein purpura (HSP)

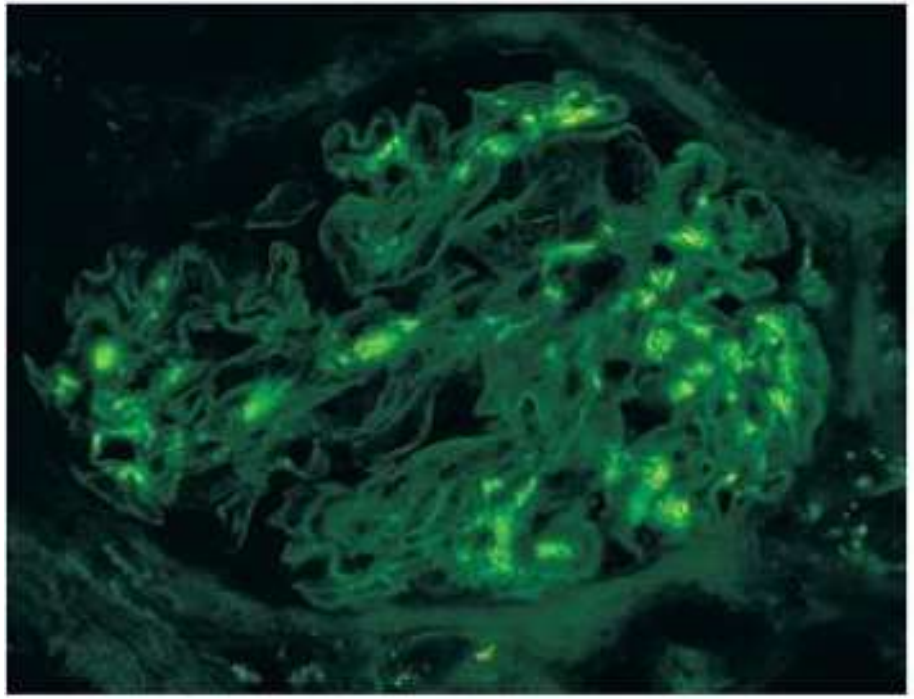
- **Treatment:**
  - Supportive care
  - Fluids
  - Pain relief
  - Hospitalize for: Inadequate oral, intake GI bleeding, Renal failure
  - Glucocorticoids for severe disease (unproven efficacy, but reasonable)
- **Response:**
  - Excellent prognosis
  - Roughly one third relapse within 4 mo
  - Acute morbidity: Due to GI bleeding
  - Chronic morbidity: Renal involvement; 10% develop ESRD

IgA nephropathy (Berger disease)

- **Most common cause of GN** in adults (although uncommon in African Americans)
- Classic presentation is gross **hematuria following upper respiratory infection** (majority of patients)
- Others present with persistent, microscopic hematuria long after viral upper respiratory infection has been forgotten; less than 10% have a nephrotic presentation
- **Mesangial IgA deposition** is seen on biopsy (establishes the diagnosis)
- In contrast to HSP, rash is not seen and course is usually less aggressive

IgA nephropathy (Berger disease)





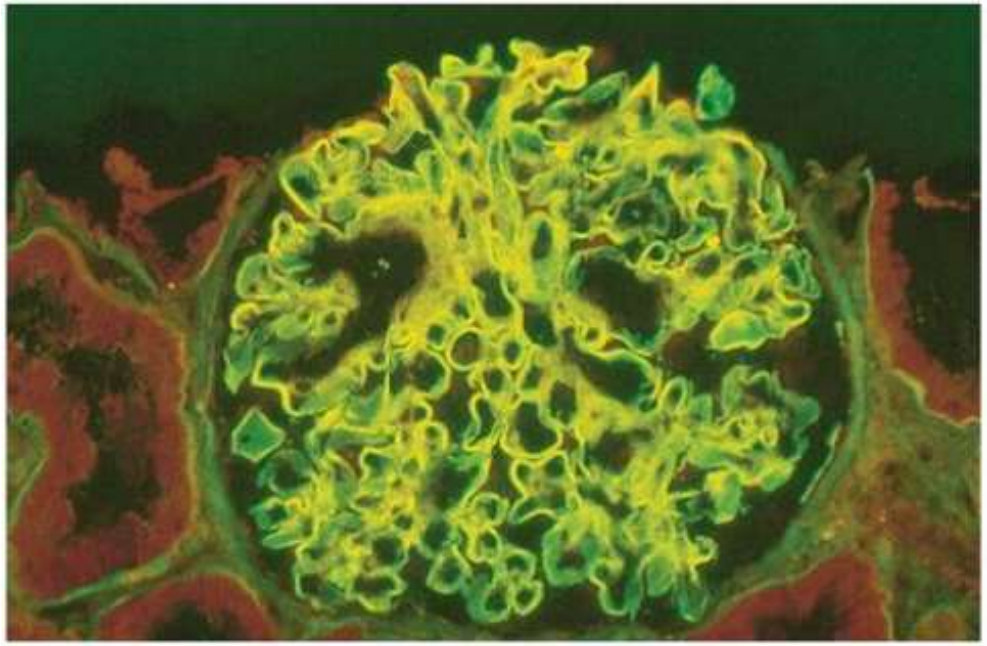
IgA nephropathy (Berger disease)

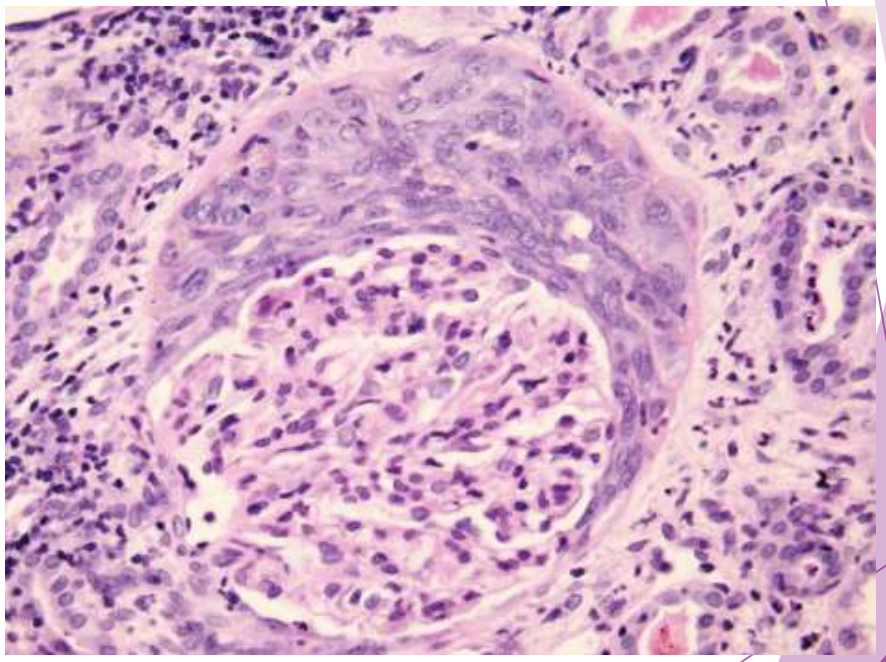
- **Treatment:**
  - Treat progessive disease (not all disease is progressive)
  - ACE inhibitors or ARBs to treat hypertension and reduce intraglomerular pressure
  - Corticosteroids may be of use in some patients
  - Fish oil (omega-3 fatty acid) use is controversial, but frequently used
  - Combined therapy with prednisone and other immunosuppressives may be needed in patients who continue to progress and in those with rapidly progressive disease
- **Response:**
  - Not all patients progress; some have stable course, some experience remission
  - In patients who progress, deterioration is usually gradual
  - Predictors of progression:
    - Elevated creatinine at diagnosis
    - Increase in blood pressure
    - Protein excretion above 500–1000 mg/24 hr
  - ESRD eventually develops in 15% at 10 yr and 20% in 20 yr

Anti–Glomerular Basement Membrane Disease and Goodpasture Disease

- Results from production of **anti-GBM antibodies**
- The presence of both lung and kidney involvement with **linear anti-GBM staining** on IF defines the syndrome
- Pathology reveals "**crescent formation**" in glomeruli; number of crescents relates to severity and prognosis
- If pulmonary disease is absent in the presence of anti-GBM antibodies, diagnosis of the kidney specific anti-GBM disease is made

Anti–Glomerular Basement Membrane Disease and Goodpasture Disease





Anti–Glomerular Basement Membrane Disease and Goodpasture Disease

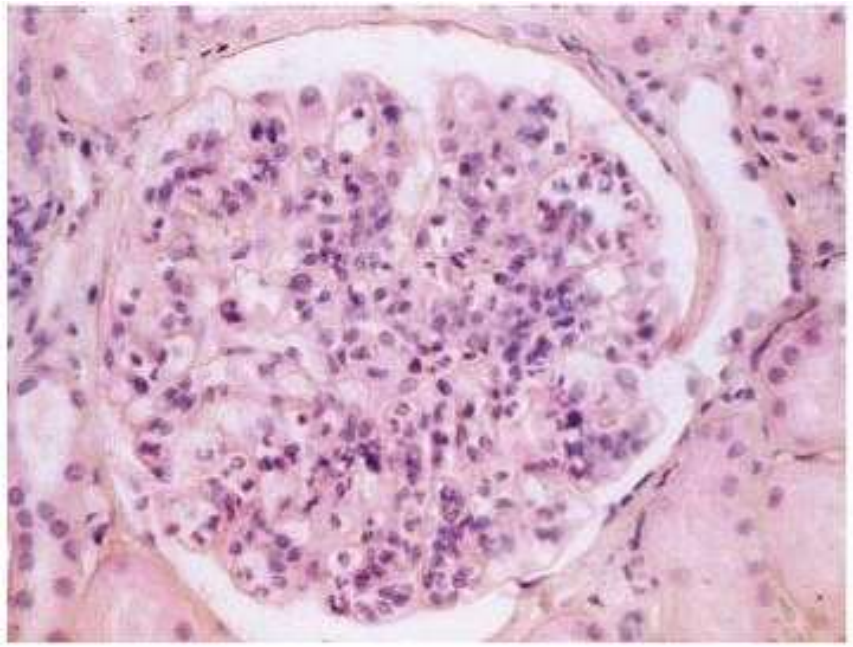
- **Treatment:**
  - Plasmapheresis (to remove anti-GBM antibodies) + corticosteroids + cyclophosphamide
  - Among those with higher creatinine requiring dialysis: Therapy less likely to be beneficial; most would still use above regimen

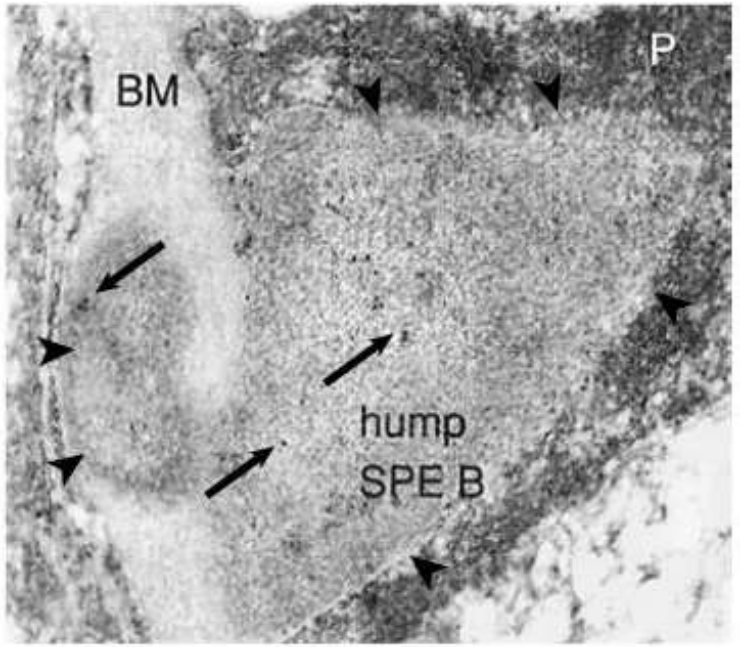
- **Response:**
  - Prognosis (patient and kidney survival) correlates with number of crescents on biopsy and level of renal insufficiency on presentation
  - Relapses are uncommon unless patient is also ANCA-positive

Infectious diseases associated with GN

- Endocarditis
- Hepatitis B and C
- Less common: syphilis, malaria
- **Postinfectious etiologies associated with GN**
  - Includes poststreptococcal GN, endocarditis-related GN, various viral infections
  - With poststreptococcal GN, patients present with **hypertension**, **oliguria**, and **elevated antistreptolysin O (ASO) antibody titers** **7 to 14 days** after throat or skin infection with group A Streptococcus
  - Certain streptococcal strains more likely to cause GN (types 12 and 49)
  - Presentation can be highly variable—from asymptomatic microscopic hematuria to florid nephritic Syndrome
- GN may also occur after staphylococcal (increasingly common in the current era) and other bacterial infections

Infectious diseases associated with GN





Infectious diseases associated with GN

- **Treatment:**
  - Immunosuppressive therapy generally not helpful
  - Rapidly progressive GN with crescents: Consider pulse steroids (though unproven efficacy)

- **Response:**
  - Prognosis generally quite good
  - Spontaneous resolution usually occurs over 3–4 wk

Membranoproliferative glomerulonephritis (MPGN)

- Clinical presentation may be **nephrotic**, **nephritic**, or **mixed**
- Most often secondary to hepatitis C infection (type I MPGN)
- Idiopathic forms of type I and type II
- Course varies depending on presentation—from slowly progressive nephrotic syndrome similar to membranous GN to a rapidly progressive GN as is seen with the necrotizing vasculitides
- Associated with cryoglobulins when related to hepatitis C (essential mixed cryoglobulinemia—type II cryoglobulins)

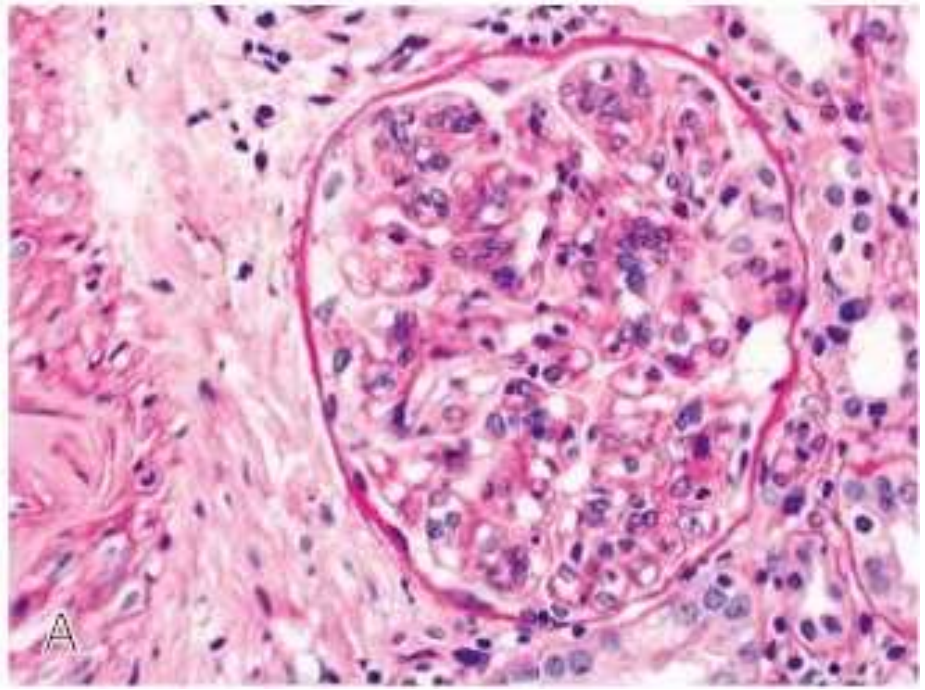
Membranoproliferative glomerulonephritis (MPGN)

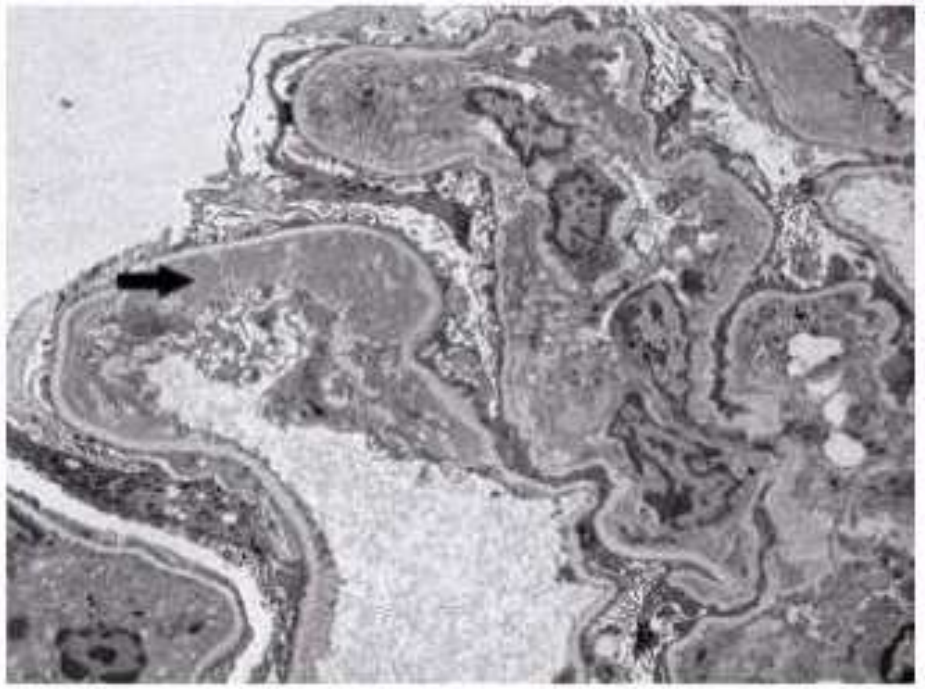
- **Typical Age of Onset:**
  - Idiopathic: 8–16 years
  - Secondary: More common in adults
- **Associated Diseases:**
  - Type I: Hepatitis C, Chronic hepatitis B, Endocarditis, Idiopathic
  - Type II (dense deposit disease): Lipodystrophy
  - Type III: Can be inherited

Membranoproliferative glomerulonephritis (MPGN)

- **Pathologic Findings:**
  - Light microscopy:
    - Glomerular lobulation, capillary wall thickening and mesangial expansion
  - Immunofluorescence
    - Type I: C3, early complement components, IgG deposits
    - Type II: ±C3, IgM
  - Electron microscopy
    - Type I: Subendothelial electron-dense deposits
    - Type II: Dense “ribbon-like” deposits along basement membrane
    - Type III: Large lucent areas in basement membrane

Membranoproliferative glomerulonephritis (MPGN)





Membranoproliferative glomerulonephritis (MPGN)

- **Treatment:**
  - Corticosteroids ± cytotoxic agents
  - Antiplatelet agents (aspirin; dipyridamole) may slow disease progression
- **Response:**
  - Spontaneous remission in <10%
  - Prolonged course of slowly deteriorating renal function seen in many

Vasculitis And Thrombotic Microangiopathy

- **Vasculitis:**
  - Wegener granulomatosis
  - Microscopic polyangiitis
  - Churg-Strauss syndrome
- **Thrombotic Microangiopathy:**
  - TTP
  - HUS
  - eclampsia

Diagnosis of Nephritic Disorders

- Clinical presentation and **testing for specific diseases** (e.g., ASO titers, hepatitis serologies) may be sufficient to diagnose likely, underlying cause of nephritic syndrome
- The evaluation and testing performed differs based on the clinical scenario
- Immunologic testing may also help narrow differential diagnosis
- Some diseases manifest with **rapidly progressive renal failure** and may mimic GN

Diagnosis of Nephritic Disorders

- Renal biopsy should be performed in the following situations:
  - Diagnosis/prognosis remains unclear
  - Rapidly progressive GN
  - Nephrotic-range proteinuria
  - Subnephrotic-range proteinuria (>2 g/day); use same
  - approach as nephrotic patient Presence of a systemic disease if accompanied by proteinuria or hematuria
  - Progressive renal insufficiency
  - Suspected acute tubular injury not improving after 3 to 4 weeks
  - Course atypical for diabetic nephropathy in diabetic patient

Treatment of Nephritic Disorders

- When GN is **secondary to a systemic disease or infection**, **treatment is aimed at the underlying disorder**
- Some patients may require dialysis therapy temporarily or indefinitely

Diagnosis of Causes of Nephrotic Syndrome			
Cause	**Typical Age of Onset**	**Associated Diseases**	**Pathologic Findings**
Minimal change disease	<12 years or mid-60s	NSAIDs Lymphoma Bee sting	Light microscopy: Normal Immunofluorescence: Normal Electron microscopy: Diffuse podocyte foot process effacement
Focal segmental glomerulosclerosis (FSGS)	Early teens to mid-30s	Hyperfiltration (seen in morbid obesity; may result from nephron loss due to other causes) HIV (collapsing variant) Heroin nephropathy	Light microscopy: Focal and segmental glomerulosclerosis Immunofluorescence: May show nonspecific IgM deposition Electron microscopy: Foot process effacement Collapsing variant seen on light microscopy in those with HIV
Membranous glomerulonephritis	Mid-30s to mid-60s	Adenocarcinoma (breast, bowel, lung) Hepatitis B Systemic lupus erythematosus Drug reaction (NSAID)	Light microscopy: Thickened capillary loops Immunofluorescence: Deposition of immunoglobulin and complement Electron microscopy: Subepithelial immune complex deposition
Membranoproliferative glomerulonephritis	Idiopathic: 8–16 years Secondary: More common in adults	Type I: Hepatitis C Chronic hepatitis B Endocarditis Idiopathic Type II (dense deposit disease): Lipodystrophy Type III: Can be inherited	Light microscopy: Glomerular lobulation, capillary wall thickening and mesangial expansion Immunofluorescence Type I: C3, early complement components, IgG deposits Type II: ±C3, IgM Electron microscopy Type I: Subendothelial electron-dense deposits Type II: Dense “ribbon-like” deposits along basement membrane Type III: Large lucent areas in basement membrane

Treatment of Idiopathic Nephrotic and Nephritic Disorders		
Disorder	**Treatment**	**Response**
Minimal change disease	Corticosteroids Cyclosporin or cyclophosphamide or mycophenolate mofetil for relapse	Children respond within 2 wk Adults respond in 4–8 wk, but often relapse
Focal segmental glomerulonephritis	Corticosteroids + ACE-I Cyclosporin or mycophenolate mofetil may be added to steroids	40–60% Response takes 4–13 mo
Membranous glomerulonephritis	ACE-I for all patients Low risk for progression (proteinuria <4 g/day + creatinine clearance >80 mL/min): Observation with close follow-up Moderate risk for progression (persistent proteinuria between 4 g/day and 8 g/day + creatinine clearance >80 mL/min): Corticosteroids + Rituximab Corticosteroids + cytotoxic agents High risk for progression (persistent proteinuria >8 g/day and/or abnormal or declining creatinine clearance): Corticosteroids + cytotoxic agents	Variable, dependent on prognostic indicators One third remit spontaneously without treatment—treatment decisions should be based on persistent proteinuria on 24-hr urine collections
Membranoproliferative glomerulonephritis	Corticosteroids ± cytotoxic agents Antiplatelet agents (aspirin; dipyridamole) may slow disease progression	Spontaneous remission in <10% Prolonged course of slowly deteriorating renal function seen in many

Treatment of Selected Nephritic Disorders		
Disorder	**Treatment**	**Response**
Henoch-Schönlein purpura	Supportive care Fluids Pain relief Hospitalize for: Inadequate oral, intake GI bleeding,Renal failure Glucocorticoids for severe disease (unproven efficacy, but reasonable)	Excellent prognosis Roughly one third relapse within 4 mo Acute morbidity: Due to GI bleeding Chronic morbidity: Renal involvement; 10% develop ESRD
Goodpasture syndrome	Plasmapheresis (to remove anti-GBM antibodies) + corticosteroids + cyclophosphamide Among those with higher creatinine requiring dialysis: Therapy less likely to be beneficial; most would still use above regimen	Prognosis (patient and kidney survival) correlates with number of crescents on biopsy and level of renal insufficiency on presentation Relapses are uncommon unless patient is also ANCA-positive
Postinfectious GN	Immunosuppressive therapy generally not helpful Rapidly progressive GN with crescents: Consider pulse steroids (though unproven efficacy)	Prognosis generally quite good Spontaneous resolution usually occurs over 3-4 wk
IgA nephropathy	Treat progessive disease (not all disease is progressive) ACE inhibitors or ARBs to treat hypertension and reduce intraglomerular pressure Corticosteroids may be of use in some patients Fish oil (omega-3 fatty acid) use is controversial, but frequently used Combined therapy with prednisone and other immunosuppressives may be needed in patients who continue to progress and in those with rapidly progressive disease	Not all patients progress; some have stable course, some experience remission In patients who progress, deterioration is usually gradual Predictors of progression Elevated creatinine at diagnosis Increase in blood pressure Protein excretion above 500–1000 mg/24 hr ESRD eventually develops in 15% at 10 yr and 20% in 20 yr

Thank You