GLOMERULONEPHRITIS and NEPHROTIC SYNDROME

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### Manifestation of Nephrotic and Nephritic Features by Glomerular Diseases

<table>
<thead>
<tr>
<th>Disease</th>
<th>Nephrotic features</th>
<th>Nephritic features</th>
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<tbody>
<tr>
<td>Minimal change</td>
<td>++++</td>
<td>-</td>
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<tr>
<td>Membranous</td>
<td>++++</td>
<td>+</td>
</tr>
<tr>
<td>Diabetic glomerulopathy</td>
<td>++++</td>
<td>+</td>
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<tr>
<td>Amyloidosis</td>
<td>++++</td>
<td>+</td>
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<tr>
<td>FSGS</td>
<td>+++</td>
<td>++</td>
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<tr>
<td>MPGN</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Proliferative GN</td>
<td>++</td>
<td>+++</td>
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<tr>
<td>Acute post infectious GN</td>
<td>+</td>
<td>++++</td>
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<tr>
<td>Crescentic GN</td>
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### Nephrotic Syndrome

- Nephrotic range proteinuria: Urine excretion of >3.5g protein/1.73m² in 24 hours.
- Nephrotic syndrome: Nephrotic range proteinuria, edema, hypoalbuminemia, hyperlipidemia/lipiduria, and hypercoagulable.

### Major Causes of Nephrotic Syndrome

- Primary Renal Disease
  - Membranous nephropathy (MN)
  - Focal glomerulosclerosis (FSGS)
  - Minimal change disease (MCD)
  - IgA nephropathy (IgA)
  - Membranoproliferative (MPGN)
Nephrotic Syndrome

- DYSPROTEINEMIAS
  - Multiple Myeloma
  - Immunotactoid/Fibrillary GN
  - Light chain deposition disease
  - Heavy chain deposition disease
  - Amyloid

- INFECTIONS
  - HIV (FSGS)
  - Hepatitis B (MN)
  - Hepatitis C (MPGN)
  - Syphilis (MN)
  - Malaria (MN)
  - Schistosomiasis (MN)
  - Tuberculosis (Amyloidosis)
  - Leprosy (MN)

- MALIGNANCY
  - Solid adenocarcinomas (lung, breast, colon, thyroid-MN)
  - Lymphomas (MCD)

- DRUGS OR TOXINS
  - NSAIDS (MCD, MN)
  - Gold, Pencillamine, Probenecid, Mercury, Captopril (MN)
  - Heroin (FSGS)

Nephrotic Syndrome

Case 1

- 36 y/o female with no significant PMH referred to her PCP from an urgent care center for further work up of leg swelling.
- Patient had noticed gradually increasing leg swelling over one week and over the weekend had worsening and presented to an urgent care center.
- She was told that she had 4+ protein in her urine dipstick, labs with normal renal function but low albumin and was asked to follow up with her PCP. She was started on Lasix 40mg daily.

Evaluation of Nephrotic Syndrome in Adults

- History
  - Family, occupational and drug and toxin exposure, recent infections
- Physical:
  - Usual recommendations for age (Pap-smear, endoscopies, Chest X-ray, mammogram, stool exam)
- Laboratory:
  - CBC, CHEM 19, lipid profile, Urinalysis, Urine micro (oval fat bodies), 24 hour urine protein
- Consider systemic diseases
  - Fluorescein angiography for DM, ANA for SLE

What to do Next?

???
Nephrotic Syndrome

- **Renal ultrasound:**
  - Evaluate for anatomical abnormalities and kidney size.
  - The presence of atrophic smooth kidneys (<9 cm) suggests CKD, which is usually irreversible.
  - Large kidneys (>13 cm) can be associated with diabetes nephropathy, amyloid or lymphoma infiltration, HIV associated nephropathy, or other GN or interstitial nephritis.

- If >50 y/o or if initial evaluation raises suspicion, consider malignancy and order:
  - Serum protein electrophoresis
  - Serum immunoelectrophoresis
  - Urine protein electrophoresis
  - Abdominal fat pad biopsy

- Consider infection: Hepatitis B/C, HIV, syphilis

- Renal biopsy:
  - distinguish primary glomerular disease
  - diagnosis of unsuspected secondary glomerular disease
  - determine disease severity

Urine Microscopy

Under polarized light, oval fat bodies in urine demonstrate the "Maltese cross" appearance.

Image from archives of The Internet Pathology Laboratory For Medical Education by Edward C. Klatt MD, University of Utah Health Sciences Center, Salt Lake City, Utah

Case 2

- 28 y/o Caucasian male with DM-1, HTN, dyslipidemia presenting to PCP to establish care.

- DM-1 diagnosed at age 10. He was compliant with insulin until 20 years of age. Had not seen a PCP in a few years. Insulin refilled through doc in the box/ ER visits.

- Complaints of decreased vision in L eye and also leg swelling which started 2 months ago. Frothy urine+

- Physical examination- BP 160/90, decreased BS at the bases and 2+ leg edema.


Question: Case 2
Most likely cause of proteinuria in this patient:

a) Multiple myeloma
b) Diabetic nephropathy
c) Amyloidosis
d) HIV
Diabetic Nephropathy

- DM most common cause of nephrotic syndrome in adults in USA.
- 35% of all patients with either type 1 or type 2 diabetes develop nephropathy after 25 to 30 years of diabetes.

Clinical Stages of Diabetic Nephropathy

<table>
<thead>
<tr>
<th>Stage</th>
<th>GFR</th>
<th>Years after diagnosis</th>
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<tbody>
<tr>
<td>1. Hyperfiltration</td>
<td>Supernormal</td>
<td>0</td>
</tr>
<tr>
<td>2. Microalbuminuria</td>
<td>High normal to normal</td>
<td>5-15</td>
</tr>
<tr>
<td>3. Proteinuria</td>
<td>Normal to decreasing</td>
<td>10-20</td>
</tr>
<tr>
<td>4. Progressive Nephropathy</td>
<td>Decreasing</td>
<td>15-25</td>
</tr>
<tr>
<td>5. ESRD</td>
<td>&lt;15ml/min</td>
<td>20-30</td>
</tr>
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</table>

Therapy of Incident and Overt Diabetic Nephropathy

- Blood pressure control
  - Goal <130/80
- Renoprotection with Renin-Angiotensin System blockade
  - HOPE and LIFE
  - IRMA2 and MARVAL
  - RENAAL and IDNT
  - DETAIL and ONTARGET
- Glycemic control
- Dietary protein restriction (to 0.8mg/kg/day)
- Lipid management

Case 3

18 y/o female goes to her family doctor with complaints of leg swelling. No other relevant history. Physical examination remarkable for 2+ pitting edema. BP 130/80. Labs: creat 0.8, BUN 12, Alb 1.7g/dl, Urinalysis 4+ protein, no cells or casts. Renal biopsy under light microscopy shows normal tissue.

Most appropriate treatment:
- a) Prednisone and cyclosporine
- b) Prednisone
- c) Mycophenolate mofetil (MMF)
- d) Prednisone and MMF

Minimal Change Disease

- Most common idiopathic
- Secondary Minimal Change Disease
  - Drugs: NSAIDS, lithium, interferon
  - Atopy/Allergies (bee stings, post-immunizations etc)
  - Neoplasia: Hodgkins Disease, non-hodgkins lymphoma, CLL
  - DM, SLE

- Minimal Change Disease

  - Common in children. Incidence decrease as patients get older.
  - Idiopathic or secondary
  - Pure nephrotic syndrome with normal renal function.
  - Normal kidney on biopsy. EM with diffuse loss of podocyte foot process.
  - Treatment: Prednisone 1mg/kg/day or 2mg/kg QOD. (min 4 weeks and max 16 weeks)
  - Once remission, taper over 6 months.
  - Secondary agents- contraindication to steroids, steroid dependent and resistant pts and frequent relapers.
Case 4

24 y/o AAM presents with progressive ankle and leg swelling. No PMH. No significant family history.
Physical exam - BP 120/70, wt 301lbs, 2+ edema
Labs - BUN 20, Creat 1.1, Alb 2.1, UA 4+ protein.
24 hour urine protein 12 gm/day
Serological work up negative
Renal US – large echogenic kidneys

Question: Case 4

What is the renal biopsy most likely going to show?

a) Minimal change disease
b) IgA nephropathy
c) FSGS
d) Membranous nephropathy

Focal Segmental Glomerulosclerosis

- Idiopathic or secondary
- Nephrotic range proteinuria
- Glomerulosclerosis in focal/segmental pattern
- Foot process effacement+
- No immune complexes
- Most common in African Americans.

Secondary FSGS

- Drugs: Pamidronate, Heroin abuse, Cyclosporine
- Infections: HIV (HIVAN: collapsing FSGS)
- Oligonephronia: after partial nephrectomy, vesico-ureteric reflux nephropathy, renal cortical necrosis
- Sickle cell disease
- Morbid Obesity
- Hereditary glomerular structural disorders
- Lupus nephritis
- HTN

Focal Segmental Glomerulosclerosis

- Collapsing FSGS (subtype)
  - Pamidronate
  - Ischemia
  - Calcineurin inhibitor
  - HIV (HIVAN)
  - Parvovirus
  - Idiopathic

FSGS Treatment

- Exclude secondary FSGS
- Prednisone 1mg/kg/d OR 2mg/kg QOD (min 4 weeks and max 16 weeks).
- After CR, taper over 6 months.
- Second line agents (CNI)- steroid resistant or CI to steroids
- Remission with treatment determine prognosis: partial/complete remission have a 10yr kidney survival of 65-90%
Case 5

45 y/o male s/p donor nephrectomy presents to PCP for yearly exam. Complaints of foamy urine and occasional leg swelling. Family history significant for Polycystic kidney disease in mother and brother, Colon cancer in father.

BP 150/84, 1+ edema.

Labs: Hb 11.4, BUN 22, Creat 1.3, UA 3+ protein. No casts, no cells.

24 hour urine protein 12gm/day.

ANA, HIV, Hepatitis panel, RPR, SPEP, UPEP negative

Question: Case 5

Renal biopsy shows membranous nephropathy.

What will be the next step?

a) Start steroids  
b) Genetic testing for PKD  
c) Colonoscopy  
d) Doppler US to rule out DVT

Membranous Nephropathy

- Idiopathic most common (possible antigen found)
- Secondary causes:
  - Drugs (gold, Pencillamine, NSAIDS, captopril)
  - Infections (Hep B and C, syphilis, parasites)
  - Malignancy (lung, stomach, breast, bladder)
  - Auto immune (SLE, RA, primary biliary cirrhosis)
- Commonest cause in Caucasian adults
- Peak incidence 30-50 yrs, Males> females.
- 80% nephrotic ; 20% asymptomatic; 50% hematuria
- HTN not common (30%)
- Increased risk of thromboembolism (15-40%)

Membranous Treatment

- First 6 months, OK to monitor with ACE/ARB, diet modification, BP <130/80.
- Exclude secondary causes
- Start therapy only in pts with NS and at least one of the following:
  - Upro >4gm/day despite ACE/ARB x 6 months
  - Severe symptoms from NS
  - SCr risen by 30% over 6-12m but eGFR >30.
- Consider anticoagulation in pts with iMN and NS with serum alb <2.5g/dl and additional risks for thrombosis.

Glomerulonephritis

Idiopathic Membranous Nephropathy

- M-Type Phospholipase A2 Receptor as Target Antigen in Idiopathic Membranous Nephropathy
    - Serum samples from 26 of 37 patients (70%) with idiopathic but not secondary membranous nephropathy specifically identified a 185-60 glycoprotein in nonreduced glomerular extract.
    - A majority of patients with idiopathic membranous nephropathy have antibodies against a conformation-dependent epitope in PLAR.
    - PLAR is present in normal podocytes and in immune deposits in patients with idiopathic membranous nephropathy, indicating that PLAR is a major antigen in this disease.
Glomerulonephritis

- Azotemia
- Hypertension
- Edema
- Hematuria
- Proteinuria (~500mg/d to 3g/d)

Dysmorphic Red Cells


SEROLOGIC WORK UP FOR NEPHRITIC SYNDROME

<table>
<thead>
<tr>
<th>Test</th>
<th>Results/Implications</th>
</tr>
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<tbody>
<tr>
<td>C3, C4, CH50</td>
<td>Usually inversely related to disease activity</td>
</tr>
<tr>
<td>ANA, Anti-ds DNA</td>
<td>SLE (membranous lupus may not have any evidence of lupus)</td>
</tr>
<tr>
<td>ANCA TITERS</td>
<td>Granulomatosis With Polyangiitis (Wg)</td>
</tr>
<tr>
<td></td>
<td>Microscopic Polyangiitis And Churg Strauss</td>
</tr>
<tr>
<td>Streptozyme test (95%), 80% Skin Infec</td>
<td>PSGN</td>
</tr>
<tr>
<td>Anti-GBM Antibody</td>
<td>Anti-GBM Dx, Good Pastures</td>
</tr>
<tr>
<td>Cryoglobulins</td>
<td>Mixed Essential Cryoglobunemia</td>
</tr>
<tr>
<td>Hepatitis Panel</td>
<td>MPGN, IgA</td>
</tr>
<tr>
<td>Blood CX</td>
<td>PSGN</td>
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Major Causes of Acute Nephritis

<table>
<thead>
<tr>
<th>Low Complement Levels</th>
<th>Normal Complement Levels</th>
</tr>
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<tbody>
<tr>
<td>SLE</td>
<td>PAN</td>
</tr>
<tr>
<td>Cryoglobulinemia</td>
<td>Wegener’s Granulomatosis</td>
</tr>
<tr>
<td>Subacute bacterial endocarditis</td>
<td>HSP/ IgA nephropathy</td>
</tr>
<tr>
<td>Shunt nephritis</td>
<td>Hypersentivity vasculitis</td>
</tr>
<tr>
<td>Acute post streptocccal GN</td>
<td>Goodpasture’s syndrome</td>
</tr>
<tr>
<td>MPGN</td>
<td>Visceral abscess</td>
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<tr>
<td></td>
<td>Idiopathic RPGN</td>
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Glomerulonephritis

- Kidney biopsy: Useful for establishing or confirming diagnosis and determine the degree of inflammation and fibrosis
- Sometimes, absence of findings are helpful (absence of immune deposits suggests vasculitis)

Rapidly Progressive Glomerulonephritis

- The most severe form of the nephritic syndromes.
- Renal failure develops over the course of a few days to weeks.
- Usually presents as proteinuria <3 g/d and hematuria with dysmorphic RBCs and/or red cell casts, with or without signs of systemic vasculitis.
- A specific finding on kidney biopsy is crescent formation.
- RPGN can progress to end-stage renal disease in most untreated patients within a period of weeks to months
Glomerulonephritis

- **Type 1:** Anti-GBM disease
  - Goodpasture’s syndrome.
  - IgG + in IF and has a smooth, diffuse, linear pattern
  - Serologic testing for anti-GBM in patient serum is often positive.

- **Type 2:** Immune complex glomerulonephritis
  - Mesangial IgA deposits in IgA nephropathy
  - ASO antibodies and subepithelial humps in PSGN
  - ANA and subendothelial deposits in lupus nephritis
  - Circulating cryoglobulins and intraluminal “thrombi” in mixed cryoglobulinemia.

- **Type 3:** Pauci-immune
  - Necrotizing glomerulonephritis but few or no immune deposits by immunofluorescence or EM.
  - Majority ANCA positive (~ 75% to 80% myeloperoxidase)

- **Type 4:** Double-antibody-positive disease
  - Has features of both types 1 and 3.

Treatment of Glomerulonephritis

- **Treatment:**
  - Induction phase
  - Maintenance phase if remission achieved (return of normal renal function, absence of proteinuria, and no systemic manifestations)

Case 6

- 32 y/o female presents to ER with dyspnea x 2 days with hemoptysis. 1 week ago, she finished a course of levofloxacin prescribed by her internist for flu-like symptoms. No other PMH.
- Physical examination: BP 110/80, HR 104, SpO2 94% on RA. Unremarkable systemic exam.
- Labs: hgb 8.0, wbc 6.0, BUN 32, creat 2.5, UA large blood, 1+ protein, >50 rbcs, 1 rbc cast, 3-4 granular casts.

Question: Case 6

Most appropriate step would be:

- a) Start oral steroids, admit for renal biopsy
- b) Pulse with solumedrol and initiate plasmapheresis and cyclophosphamide treatment
- c) Admit for pneumonia, initiate iv abx after blood cultures
- d) Check IgA levels

Glomerulonephritis

- **Pulmonary renal syndrome**
- Goodpasture’s, ANCA vasculitis, lupus nephritis, post infectious, cryoglobulinemic
- Goodpasture’s
  - Crescentic GN
  - Bimodal (30yrs, 60yrs)
  - Male > female
  - Caucasians > AA
  - Associated with HLA DR2, DR4 and DRw15
  - Risk factors - hydrocarbon exposure, cigarette smoking, metallic dust, cocaine, influenza infection
  - Prodrome in 20-60%
Goodpasture's (Anti-GBM)
• Anti GBM autoantibodies react with target antigen of the noncollagenous domain of the alpha chain of type IV collagen.
• CXR - interstitial shadowing
• CT scan and measuring DLCo, bronchoscopy; consider in the work up.
• Therapy: plasmapheresis with plasma exchange x 2 weeks; methyl prednisone pulse 1gm/day x 3 days followed by prednisone 1mg/kg and cyclophosphamide 2-3 mg/kg po qday.

Case 7
19 y/o female nursing school student presents to student health center with complaints of bright red urine. She has been having an URI for the past two days with rhinorrhea, sore throat and low grade fever. No similar episodes in the past. Family history pertinent for father with IgA nephropathy.

Question: Case 7
Her diagnostic evaluation should include all of the following EXCEPT:
- a) Renal biopsy
- b) Serum IgA levels
- c) Renal panel and Urinalysis with microscopy
- d) Serological work up for secondary causes

IgA Nephropathy (Berger's Disease)
• Commonest cause of nephritis in the world
• Males > females
• Second and 3rd decade peak occurrence
• Asian predominance
• Spectrum - asymptomatic microscopic hematuria to RPGN
• Synpharyngitic - 24-48 hours after URI (2-3 weeks in PSGN)
• Mesangial proliferation in light microscopy on biopsy

IgA Nephropathy
• Familiar aggregation: 55% of IgA patients had a relative with IgA nephropathy.
• Clinical predictors of poor outcome
  - Absence of gross hematuria
  - Higher creatinine on presentation
  - Males
  - Older presentation
  - HTN
  - Heavy proteinuria
• Treatment variable and depends on the clinical presentation.
• Nephritic or nephrotic, or both.

Lupus Nephritis
• 40% of SLE patients
• Predominantly affects women of child bearing age.
• Spectrum variable and biopsy necessary.
• Look for hematuria/proteinuria in SLE pts.
• Hypertension usually associated with proliferative lesions.
• Anti ds-DNA and complements correlate with disease activity.
Lupus Nephritis

- Class I, Minimal lupus glomerulonephritis (LGN)—normal urine or microscopic hematuria
- Class II, Mesangial proliferative LGN—microscopic hematuria and/or low-grade proteinuria
- Class III, Focal proliferative LGN—nephritic urine sediment and subnephrotic proteinuria
- Class IV, Diffuse proliferative LGN—nephritic and nephrotic syndromes, hypertension, azotemia
- Class V, Membranous LGN—nephrotic syndrome
- Class VI, Sclerosing disease—HTN and decreased renal fx

Timing of Initial Kidney Biopsy for Lupus Nephritis

- Protein excretion greater than 500 mg/day.
- An active urinary sediment with hematuria (five or more red blood cells per high-power field, most of which are dysmorphic) and cellular casts.

Repeat SLE Kidney Biopsy?

- Increasing proteinuria—new or worsening; to rule out concomitant membranous component.
- An active sediment and a rapidly rising serum creatinine—rule out crescentic disease that requires more aggressive initial therapy.
- Slowly rising serum creatinine along with urine sediment—to distinguish active proliferative LN from advanced sclerosing LN (class VI).
- Suspicion of possible renal disease unrelated to lupus (eg, drug-induced acute interstitial nephritis).

Lupus Nephritis Kidney Biopsy

- Avoid biopsy in patients with successfully treated diffuse proliferative disease who develop recurrent active sediment
- Especially if it represents recurrent proliferative disease.
- No additional value to aid treatment

Treatment of Lupus nephritis

**Mesangial LN**
- Minimal mesangial (class I)
- Mesangial proliferative (class II)

**Treatment**
- No specific therapy
- May require treatment if proteinuria is >1g/d. Consider prednisone in low-to-moderate doses (ie, 20-40 mg/day) for 1-3 months, with subsequent taper.

**Proliferative LN**
- Proliferative Lupus (Class III and Class IV)

**Treatment**
- Induction (IV Cyclophosphamide or PO Mycophenolate)
- Maintenance (PO Mycophenolate or Azathioprine)
- Supportive care
### Treatment of Lupus Nephritis

**Membranous LN**
- Membranous (Class V)

**Treatment**
- Non immunosuppressive therapy (diet, BP)
- Immunosuppressive Rx if proteinuria >3.5g/d despite above rx or worsening renal function.
- Prednisone + IV Cyclophosphamide or Cyclosporine

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### Membranoproliferative GN

- Nephrotic/Nephritic syndrome
- Primary MPGN
- Secondary MPGN
  - Bacterial/Viral infection
    - HCV and HBV
  - Cirrhosis
  - Autoimmune: SLE and Sjogren's
  - Cryoglobulinemia
  - Malignancy: Leukemia/Lymphoma
  - Diabetes
  - Sickle cell disease
  - Light chain deposits
  - HIV

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### Thrombotic Microangiopathies

- TTP-HUS
  - Idiopathic
  - Secondary
    - HUS 2/2 e coli 0157:H7
- Antiphospholipid Antibody Syndromes
  - Lupus associated
  - Unrelated to lupus
- Malignant hypertension
- Scleroderma renal crisis
- HIV
- Pre-eclampsia
- Medications/drugs

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### Pregnancy and Lupus Nephritis

- Previous history of lupus nephritis or active lupus nephritis associated with higher rates of maternal and fetal complications
- Delay pregnancy until the disease is inactive for at least six months
- Medication adjustments:
  - Discontinue ACE-, Methotrexate and Mycophenolate
  - Continue Hydroxychloroquine
  - Switch to Azathioprine

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### MPGN Treatment

- Treat secondary causes
- Steroids
  - If good response continue supportive care such as ace inhibitor
- Cytoxan
- Rituxan

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TTP Diagnosis

- **Hemolytic anemia**
  - Schistocytes 2 or more on 100x field, so >1% of RBC
- **Thrombocytopenia**
- Neurologic changes
- Fever
- Acute renal failure (often anuric)

General Principles of Treatment

- **Disease-specific treatment** and **symptomatic and supportive** treatment of proteinuria, hypertension, hyperlipidemia, and control of edema.
- **Proteinuria**-treated with ACE- or ARBs to reduce proteinuria to <1 g/d.
- **Control of blood pressure**: with a goal of <130/80 mmHg. In patients with proteinuria, ACE inhibitors or ARBs should be the first-line therapy.

General Principles of Treatment

- Control of edema or volume overload requires the use of **dietary salt and water restriction** in conjunction with diuretics.
- Treatment of **hyperlipidemia**. Most patients with nephrotic syndrome should be on statin therapy to prevent coronary and other atherosclerotic long-term complications.

Differential Diagnosis of Nephrotic Syndrome

- **Nephrotic Glomerular Diseases**
  - FSGS
  - Membranous
  - Minimal change
  - Amyloidosis
  - Diabetes
- **Nephritic/Nephrotic**
  - Lupus nephritis
  - MPGN/Cryoglobulinemia
  - IgA nephropathy

Differential Diagnosis: Nephritic Syndrome

- ANCA assoc Wegener’s, Microscopic polyangitis, Churg-Strauss.
- Anti-GBM: Goodpasture’s, Anti-GBM
- Post-strep GN
- MPGN/Cryoglobulinemia
- IgA nephropathy
- Lupus nephritis
- Endocarditis
- Henoch-Schonlen purpura
- TTP/HUS

- Some on both Nephritic/Nephrotic
References

• The Diagnosis of glomerular Diseases; Acute Glomerulonephritis and The nephrotic syndrome. Michale P Madaio, Md and John T Harrington: Arch Intern med. 2001; 161:25-34
• The Internet Pathology Laboratory For Medical Education by Edward C. Klatt MD, University of Utah Health Sciences Center, Salt Lake City, Utah.
• Primer On Kidney Diseases

• Answer Key:

<table>
<thead>
<tr>
<th>CASE #</th>
<th>ANSWER</th>
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<tbody>
<tr>
<td>Case 2</td>
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<td>Case 3</td>
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<td>Case 4</td>
<td>c</td>
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<td>Case 5</td>
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<td>Case 6</td>
<td>b</td>
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