GLOMERULONEPHRITIS (GN) FOR THE NON- NEPHROLOGIST

DISCLOSURES

- Non-Declaration Statement: I have no relevant relationships with ineligible companies to disclose within the past 24 months. (Note: Ineligible companies are defined as those whose primary business is producing, marketing, selling, re-selling, or distributing healthcare products used by or on patients.)

OBJECTIVES

- Define and describe the major subtypes of GN
- Highlight the common clinical presentation of GN
- Using the KDIGO guidelines, review treatment options for GN discussing pros and cons of treatment modalities

WHAT IS GLOMERULONEPHRITIS (GN)?

Injury to the glomerulus

Pathological mechanisms may include:

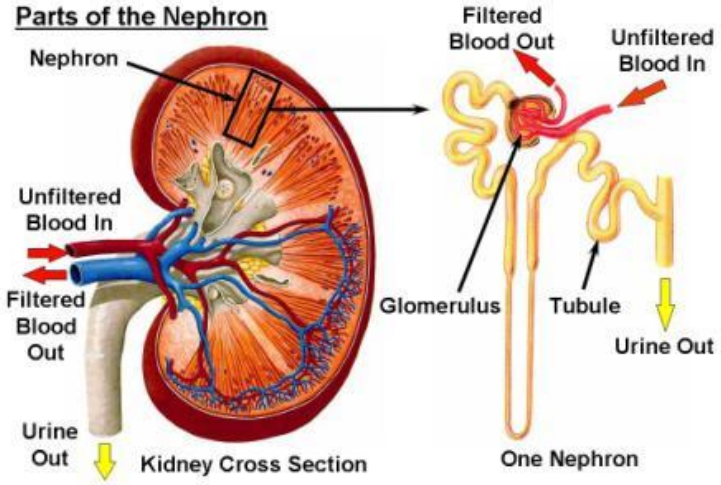
- Immune complexes directed at various glomerular antigens or deposited in the glomerulus from circulation
- Complement deposition
- Inflammatory cell deposition
- Necrosis and eventual sclerosis (scarring)

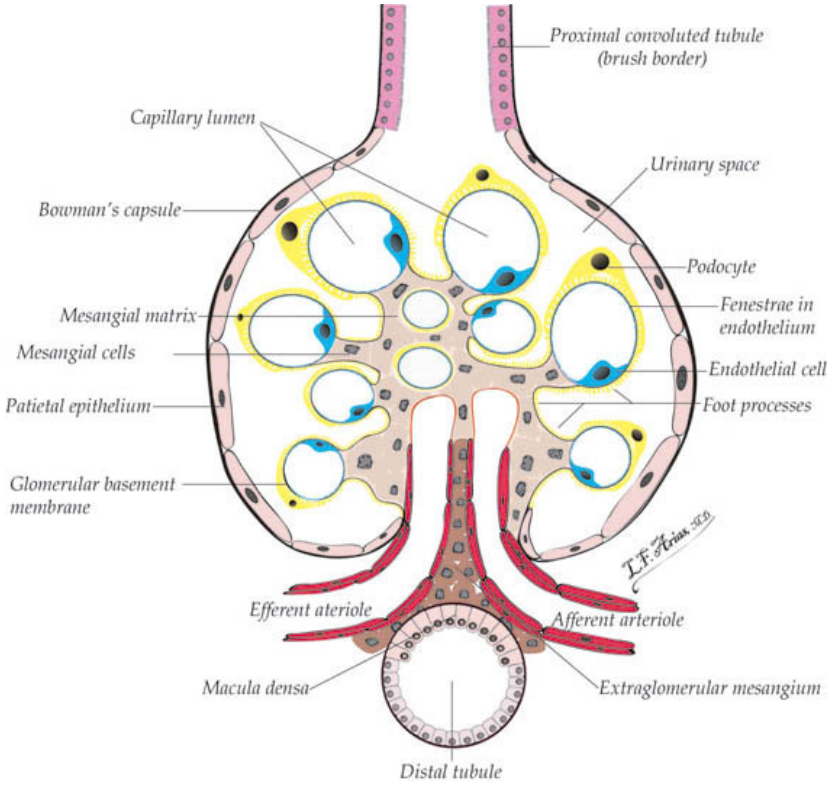
Glomerular injury can be

- primary disease (IE: FSGS)
- systemic process (IE: Lupus)

3rd most common cause of ESRD

ANATOMY REVIEW





TYPES OF GN

Primary (Problem is **IN** the kidney)

- Minimal Change Disease (MCD)
- Focal Segmental Glomerulosclerosis (FSGS)
- Membranous Nephropathy (MN)
- Anti-glomerular basement membrane (anti-GBM) GN
- IgA Nephropathy (IgAN)
  - Asian but seeing AA now
- Membranoproliferative Glomerulonephritis (MPGN)*

*can be considered primary or secondary

Secondary (Systemic)

- Post-infectious GN
  - Strep, E. Coli, Salmonella
- Lupus Nephritis
  - More common in the AA patient
- Renal Vasculitis: Pauci-immune focal and segmental necrotizing GN
  - *Gets nephrology excited*
- Membranoproliferative Glomerulonephritis (MPGN)*
- HIVAN
- Hepatitis C

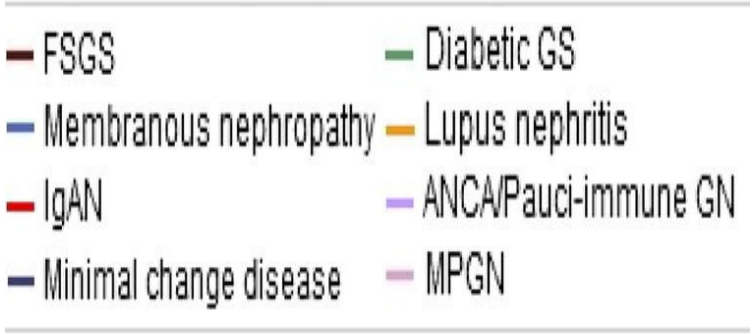
TYPES OF GN

But you don't care….

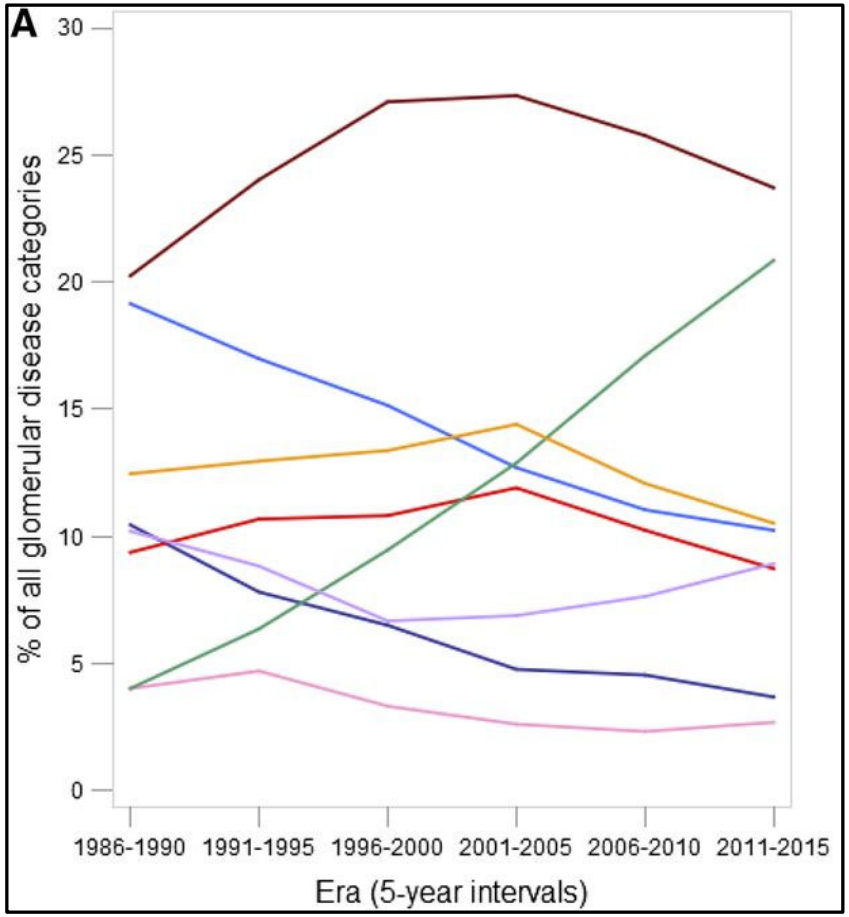
- This is very exciting to nephrology only
- You need to know:
  - Causes 25-30% of ESRD
  - Presentation Scenario
  - Age at presentation
- This is often a disease of the young….

(I consider anyone under 40 to be young)

Epidemiology 1986-2015

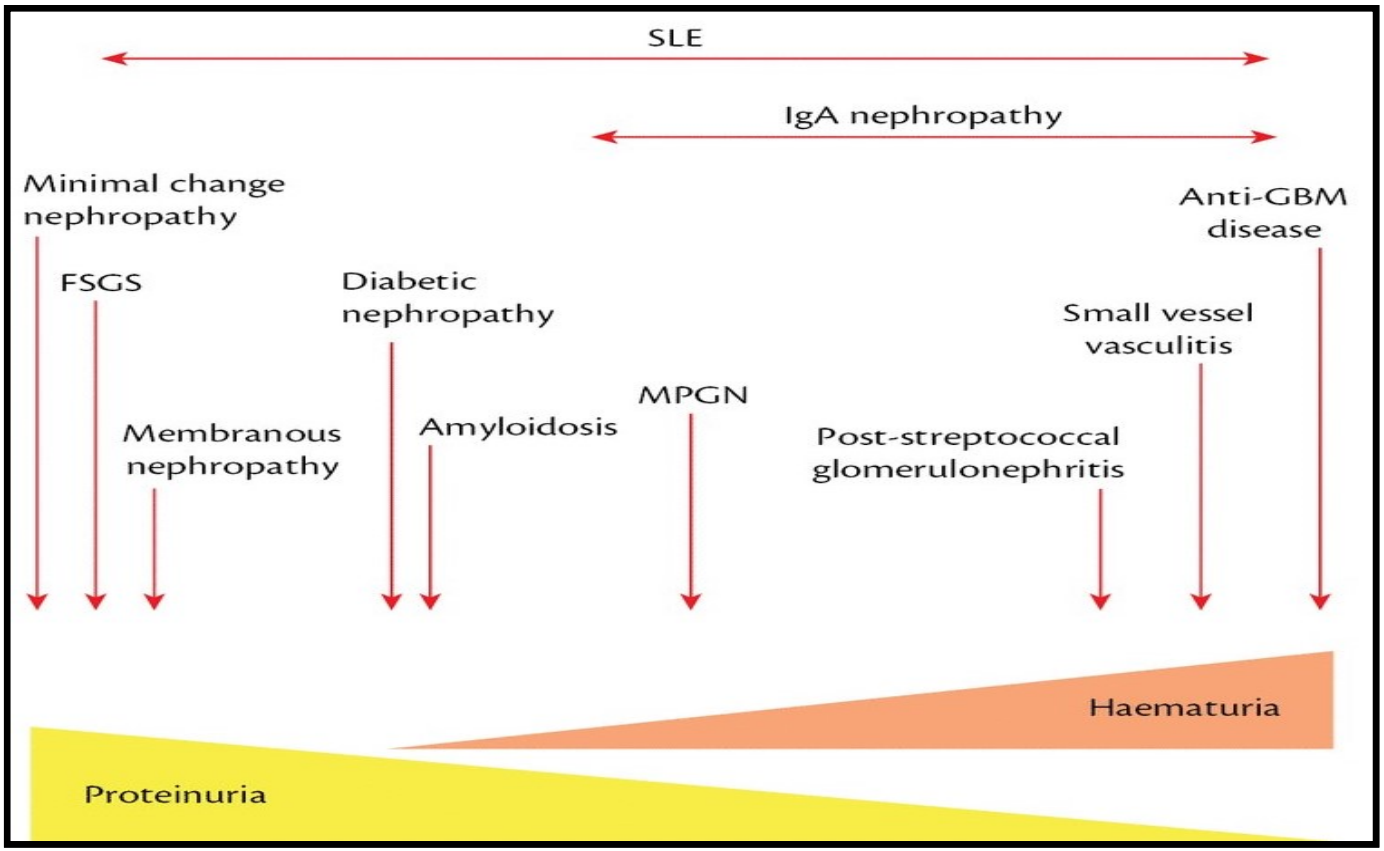


Focal Segmental glomerulosclerosis **(FSGS)**
Diabetic glomerulosclerosis **(GS)**
IgA nephropathy **(IGAN)**
Anti-neutrophilic cytoplasmic antibodies **(ANCA)**
Membranoproliferative GN **(MPGN)**



Michelle M. O'Shaughnessy et al. CJASN 2017;12:614-623

THE SPECTRUM OF GLOMERULAR DISEASE



COMMON PATIENT PRESENTATIONS

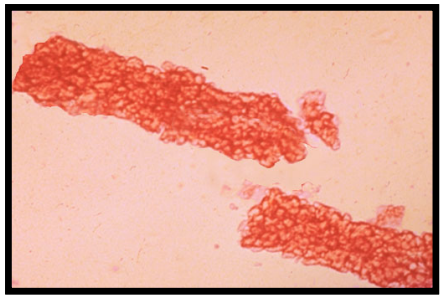
While Nephrology feels that nephrotic vs nephritic is not useful (everything in life is a continuum)
Boards still insist on the distinction…

Acute Nephrotic Presentation:

- Proteinuria >~3.5 grams/day
- Edema
- Hypoalbuminemia
- Hyperlipidemia

Acute Nephritic presentation:

- Proteinuria
- Hypertension
- Hematuria (gross or microscopic)
- RBC Casts



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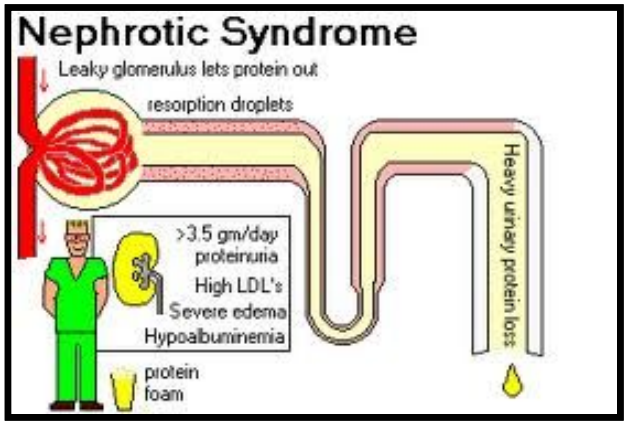

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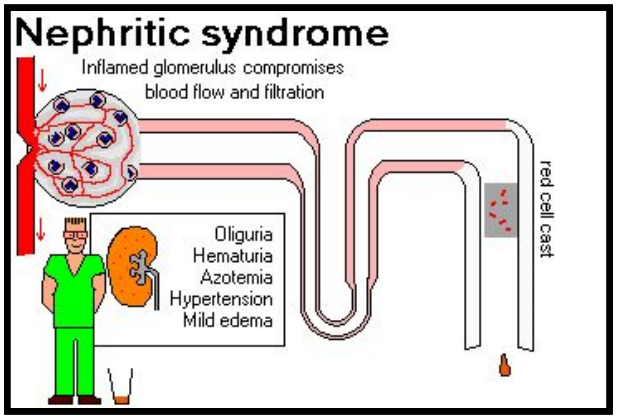


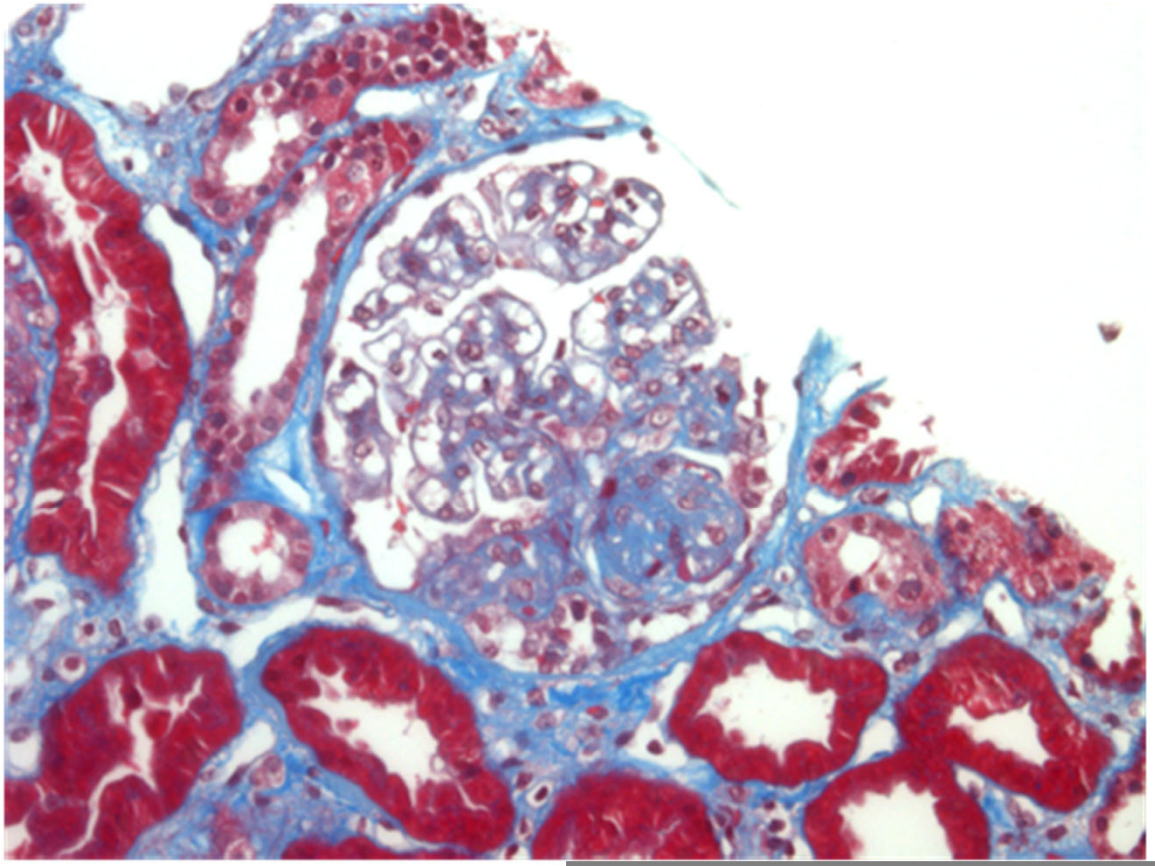
GN DIAGNOSIS

Primary GN:

- Requires histopathological diagnosis with **Kidney Biopsy**

Secondary GN:

- Serum studies can strongly support the diagnosis
- **Kidney Biopsy** often done to confirm diagnosis or refine prognosis



Glomerulus with prominent segmental sclerosis responsible for proteinuria (20X magnification, Masson's Trichrome Stain)

TREATMENT AFTER BIOPSY CONFIRMS DIAGNOSIS

Medication	Advantages	Side Effects
Corticosteroids	**MOST COMMON** Potent in inducing and maintaining remission in a variety of GNs	Psychiatric disturbances, diabetes, cataracts, fractures
CNIs	Most nephrologists comfortable with medication since we use it in transplant	Long-term nephrotoxicity, HTN, may reduce proteinuria by vasoconstriction rather than directly addressing underlying disease
Cyclophosphamide		Infertility, bone marrow toxicity, secondary malignancies nausea, hemorrhagic cystitis
Mycophenolate Mofetil (MMF, Cellcept)	Steroid-sparing agent with relatively well-tolerated side effect profile	Diarrhea, cytopenias, not safe in pg
Azathioprine (AZA, Imuran)	Relatively safe in pregnancy compared with MMF	Cytopenias, hepatotoxicity, secondary malignancies, GI disturbance
Rituximab	6-month dosing reduces need for patient compliance	Infusion reaction, cytopenias, GI disturbance, immunosuppression lasting 6 months after single dose

TREATMENT AFTER BIOPSY DIAGNOSIS

Hyperlipidemia
- Statins
- If resolving nephrotic syndrome, no statins needed

Immunosuppressed state
- Pneumocystis prophylaxis for patients on high dose steroids
- TB, HBV testing prior to immunosuppressive treatment
- No live vaccines!

Hypercoagulable state
- Anticoagulation not typically used
- Exception: Membranous patients with serum albumin <2.5g/dL and additional risk factors

Diet
- Low Na for BP control, edema

HTN
- ACEi/ARB for both HTN & proteinuria
- Diuretics (loop +/- amiloride) for edema

Special considerations on high-dose steroids:
- Pneumocystis prophylaxis
- Warn of effects on glucose control, need to adjust meds/insulin if diabetic
- Fracture risk: ensure adequate Ca++ & Vit D
- Consider GI prophylaxis

WHEN TO TREAT VS WHEN TO MONITOR…

Treatment does not always guarantee cure and multiple courses often needed

Persistent, nephrotic range proteinuria or rapidly progressive GN will almost certainly result in ESRD and warrants ***an attempt*** at medical therapy

Sometimes, there are other diseases….

- Recurrent/persistent proteinuria does not always reflect relapse of the primary GN

If the risks/side effects outweigh benefit:

- Advanced CKD/ESRD (SCr>3.5mg/dL) and/or small, atrophic kidneys on ultrasound
- Relatively stable kidney function with conservative measures
- Short life expectancy (another DX)

MEMBRANOUS NEPHROPATHY

Presentation:

- Usually nephrotic range proteinuria
- Can be due to autoimmune, infectious, malignant
- High risk for PE
- More common in Caucasians

Pathology:

- Diffuse glomerular basement membrane (GBM) thickening
- Sub-epithelial immune complex deposits

Treatment:

- *Rule of thirds*
  - 1/3 spontaneously remit
  - 1/3 with persistent proteinuria
  - 1/3 progress to ESRD
- Observation (6 months) if:
  - Stable proteinuria <4g/day, preserved renal function

MINIMAL CHANGE DISEASE (MCD)

Presentation:

- Usually nephrotic range proteinuria
- Patient feels fine but + edema
- More common in kids (edema anywhere, scrotal)
- HTN, hyperlipidemia

Pathology:

- Normal appearance by light microscopy
- Diffuse foot process effacement

Treatment:

- Corticosteroids (high dose) X 4 weeks with slow taper
- Most respond within 8 weeks
- Frequent relapse
- Treat hyperlipidemia, HTN

FOCAL SEGMENTAL GLOMERULOSCLEROSIS (FSGS)

Alonzo Mourning

Dayna Stephens

Presentation:

- No symptoms
- More common in AA males
- High SCr
- ±HTN

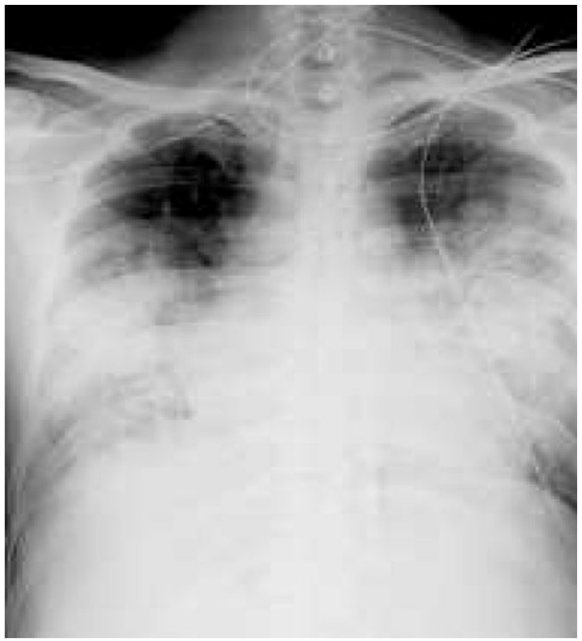
Pathology:

- Focal (% of kidney), Segmental (portion of glomeruli) Glomerular (in the glomerulus) Sclerosis (dead area)
- Biopsy can miss the 'bad area' and looks like MCD on biopsy! (*'missed FSGS'*)

Treatment:

- Blood pressure control with ACEi/ARB
- Steroids typically only employed for primary FSGS with active nephrotic syndrome
- Cyclosporine or CNIs (calcineurin inhibitors) in steroid resistant cases
- Transplant

ANTI-GLOMERULAR BASEMENT MEMBRANE (ANTI-GBM)



http://www.sjkdt.org/articles/2013/24/4/images/SaudiJKidneyDisTranspl_2013_24_4_783_113886_u3.jpg

Presentation:

- Usually lungs/kidney hemorrhage
- Occurs in teenage years and >50 y/o
- Commonly referred to as 'Goodpastures'
- Rapidly progressive, often fatal

Pathology:

- Antibodies against the glomerular basement membrane
- Often associated with crescent formation

Treatment:

- Cyclophosphamide + corticosteroids + plasmapheresis
- Transplant deferred until anti-GBM antibodies undetectable x 6 months
- Due to high fatality rate, start RX while awaiting diagnosis!

LUPUS NEPHRITIS

Nick Cannon with Sydney the Kidney

Presentation:

- Usually known lupus
- SLE more common in female AA population
- Next to impossible to see 'butterfly rash' in AA patient
- Treat underlying SLE regardless of renal manifestations
- Should be co-managed with nephrology
- Typical presentation, proteinuria within 3y of diagnosis
- Bad prognostic factor, abnormal SCr in early disease

Pathology:

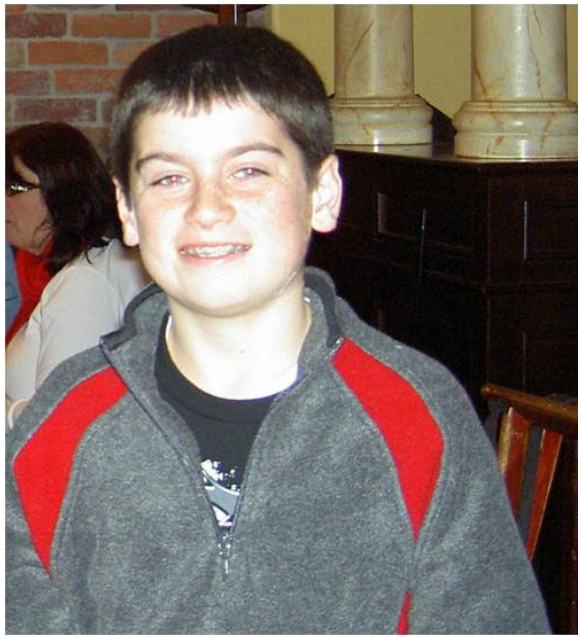
- 6 different subclasses based on underlying pathology
- Disease severity ranges from Class I (no light microscopy abnormalities) to Class VI (sclerosis of >90% of the glomeruli)

Treatment:

- **DEPENDENT ON BIOPSY CLASSIFICATIONS!!**

- Class I, II, VI:
  - conservative treatment
- Class III, IV, V:
  - immunosuppressive induction
  - maintenance

POST-INFECTIOUS GN



Presentation:

- Usually hematuria but it can be significant
- History of infectious disease
- *More common in kids*

Pathology:

- Often not done, with DX made by history
- Diffuse, proliferative lesions or 'humps' with sub-endothelial immune deposit

Treatment:

- Treat underlying infection
  - Ex: Post-streptococcal GN with penicillin
- Supportive care

IGA NEPHROPATHY

Presentation:

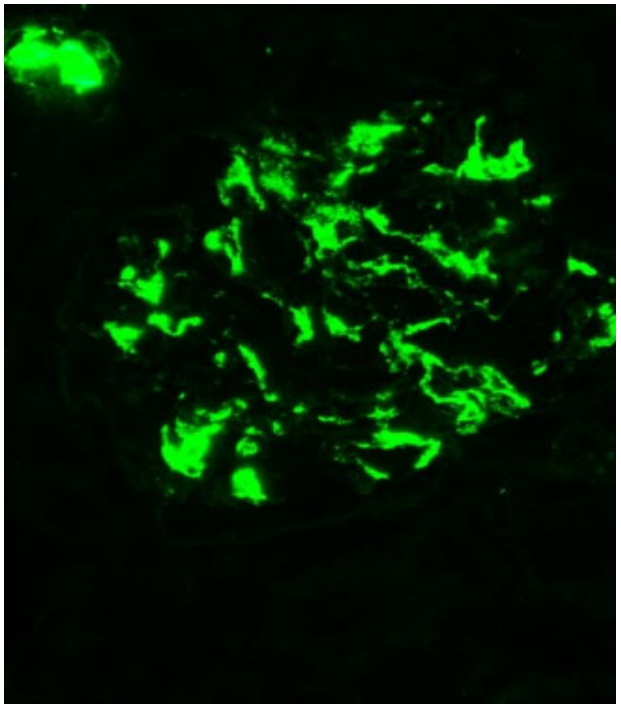
- Classically intermittent hematuria but it can be significant amount
- More common in the Asian population but this may be a sampling bias

Pathology:

- IgA deposits in the mesagium
- Florescence on electron microscopy

Treatment:

- Risk factors for progressive disease warranting consideration of active treatment:
  - proteinuria >1g/day
  - uncontrolled HTN
  - increased serum creatinine
- Steroids can be effective, but risks usually outweigh benefits
- BP and proteinuria control with ACEi/ARB



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MEMBRANOPROLIFERATIVE GN (MPGN)

Presentation:

- Onset is insidious
- Approximately 80% of patients describe edema
- Patients may present with nonspecific complaints:
  - anorexia
  - malaise
  - fatigue
- Some patients may present with asymptomatic proteinuria

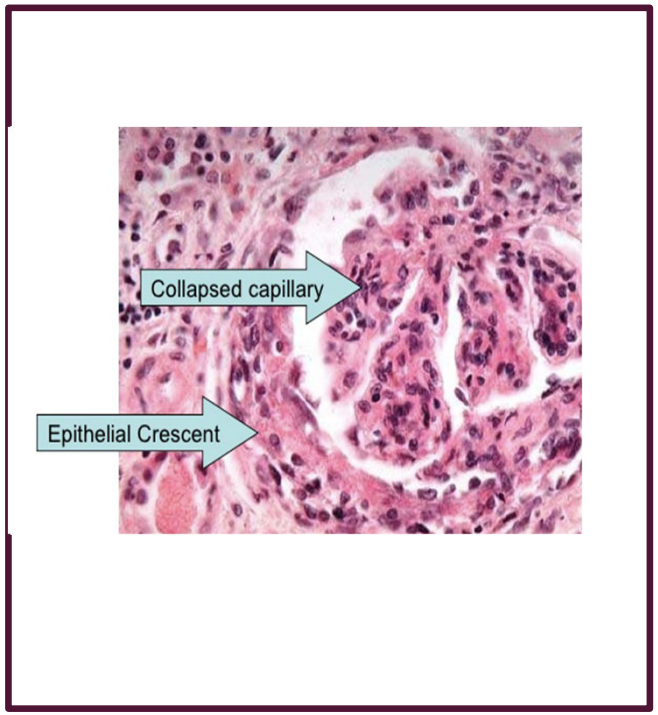
Pathology:

- Expert nephropathologist evaluation to ID specific subtype of proliferative lesions
- Location and appearance of deposits can vary depending on the cause
- Deposits can be composed of complement alone and/or immunoglobulin

Treatment:

- Limited data
- Observation reasonable in non-nephrotic patient with stable proteinuria
- Steroids + cyclophosphamide or MMF for rapidly progressive disease
- Most of us refer to expert tertiary care center (NIH, Stanford, Hopkins, Mayo)

RENAL VASCULITIS: PAUCI-IMMUNE FOCAL & SEGMENTAL NECROTIZING GN



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Presentation:

- Hematuria + signs of small vessel vasculitis (diffuse skin lesions, lung hemorrhage, etc)
- Encompasses a group of diseases characterized by necrotizing inflammation of small blood vessels
- If little or no deposition of immune complexes referred to as *pauci-immune*
- Common names: Wegener's, microscopic polyangiitis, and Churg-Strauss syndrome; each one has slight variations but essentially are small vessel vasculitis

Pathology:

- Pauci-immune (little deposit) focal, segmental necrotizing, crescentic
- Often ANCA + (Anti-neutrophil cytoplasmic antibodies)

Treatment:

- Poor outcomes (body attacking itself) without RX
- Aggressive treatment with:
  - steroids
  - cyclophosphamide
  - rituximab
- Plasmapheresis for severe disease (rare)
- Even with GFR<10ml/min, 57% remission with RX!!!!!!

VIRAL AND BACTERIAL GN

Presentation:

- Disease dependent IE: HIV, Hep C, Hep B, *E. coli 0157:H7* (food borne), Salmonella, etc
- *E.Coli 0157* presents as bloody diarrhea that has resolved. Most children fully recover from their bowel illness without developing HUS (hemolytic uremic syndrome). However, a small percentage will become pale and have less energy, due to the progression to HUS. Their urine output may also decrease, but a loss of color in the skin is the most striking symptom

Pathology:

- Rarely done; Hep B/C may be found incidentally on biopsy
- Diagnosis often via serum assays

Treatment:

- Treat underlying disease (HIV, Hep C, Hep B)
- HUS may require dialysis, 10% death rate

GLOMERULONEPHRITIS (GN)

- Seems complicated because many diagnoses are uncommon
- 3rd most common cause of ESRD after Diabetes and HTN
- Almost all will need a biopsy so involve nephrology early
- Can be primary or secondary
- Think of it as a puzzle and you

  won't tear your hair out!

CASE STUDIES

SADIE

58 y/o presents to ED with 'failure to thrive'
PMH: long-standing lupus
PE: BP 188/101, 1+ edema feet, RRR, HR 80, states everything tastes like metal
Labs: BUN 102mg/dL, SCr 4.8mg/dL (eGFR 11)

Which of the following would suggest GN?

A. Red Blood Cell (RBC) Casts
B. Hyaline Casts
C. White Blood Cell (WBC) Casts
D. Muddy Brown Casts

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13 y/o male
PMH: sore throat week previously w/N&V
Dark 'coke' colored urine, brought to ED
Labs: Na 132mEq/L, K 5mEq/L, BUN 80mg/dL, SCr 2.6mg/dL, bicarb 16mEq/L
UA: Dip 2+ blood, 1+ protein, RBC casts on micro

What does Brendan have?

A) Pauci-immune GN
B) Post-strep GN
C) IgA nephropathy
D) I have absolutely no idea….

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What is the next step for Brendan?

A) CBC
B) ASO titer
C) Kidney biopsy
D) IV fluids

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On Boards, this is referred to as a 2nd Generation question
You had to know the diagnosis to determine the next step

Mike

54 y/o urology PA who practices in NOLA, notices that his urine is foamy ;'*looks like beer*'
PMH: Tourette's, HTN, HLD, prinzmetal angina (during Katrina)
Meds: losartan (Cozaar-cough with lisinopril), atorvastatin (Lipitor)
PE: 132/82, 1+ edema to knees

What is the cause of Mike's urine changes?

A) Beer Potomania
B) Nephrotic syndrome
C) Medication-induced
D) Cardio-renal syndrome

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Meds: losartan (Cozaar-cough with lisinopril), atorvastatin (Lipitor)
PE: 132/82, 1+ edema to knees
Labs: SCr 1.2, 3+ protein on urine dip

What is the next step in the workup for Mike?

A) Repeat labs
B) Send for kidney biopsy
C) 24-hour urine collection
D) Renal ultrasound

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Nephrology does a 24 hour urine that comes back with 1.86gm/24 hours; **nephrotic range proteinuria *(1860mg/24H)*** and schedules a kidney biopsy

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C) Minimal Change Disease
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Mike's biopsy shows **Minimal Change Disease (MCD)** with little to no change on kidney biopsy (thus, the name)

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What is the treatment for MCD?

A) Diet and exercise
B) Strict management of HTN
C) Steroids
D) Strict management of hyperlipidemia
E) All of the above

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CJ

27 y/o Asian male with sudden onset of hematuria
PMH: URI earlier in the week
Meds: none
Labs: pending

What is the most likely diagnosis?

A. Henoch-Schonlein purpura (HSP)
B. Bladder cancer
C. Post-strep GN
D. Immunoglobulin A nephropathy (IgA)

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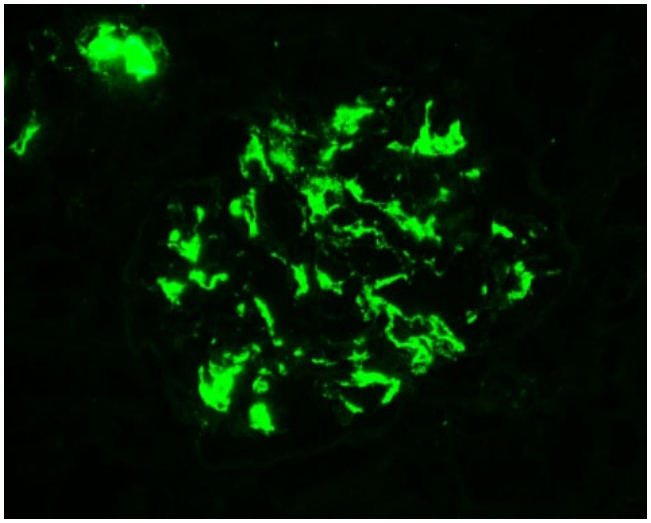

What is the treatment?

A. Steroids
B. Depends on the urine
C. ACE/ARB
D. No treatment needed

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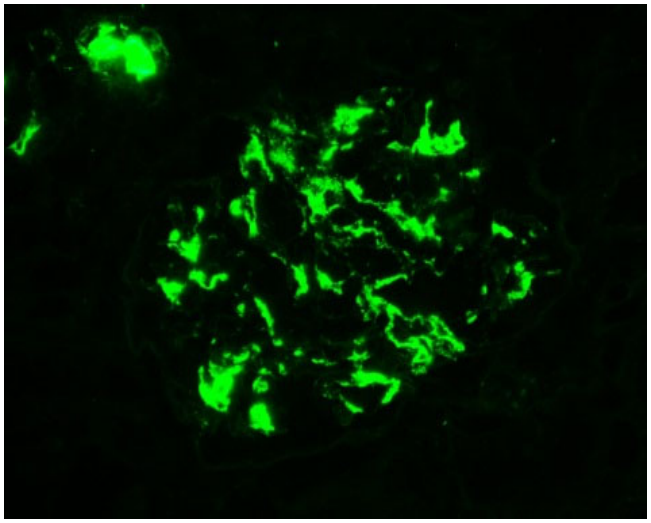
27 y/o Asian male with sudden onset of hematuria
You suspect IgA and send him for a biopsy
Your biopsy returns-The glow from the immunoflorescence shows a beautiful staining pattern…classic IgA staining
With more in-depth questioning, he admits this is not his first episode



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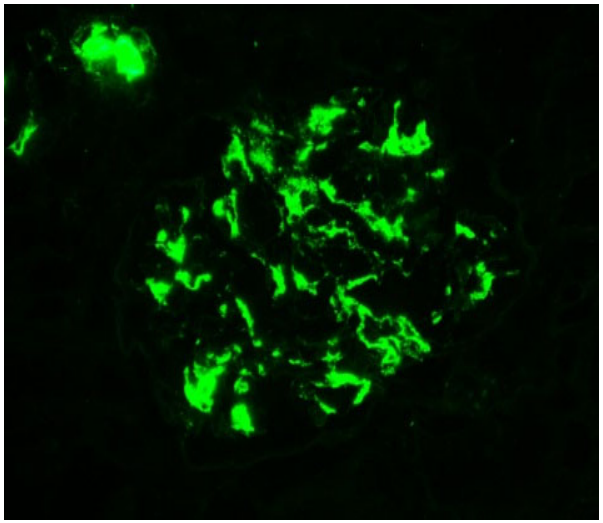

CJ

27 y/o Asian male with sudden onset of hematuria

You suspect IgA and send him for a biopsy

Your biopsy returns-The glow from the immunoflorescence shows a beautiful staining pattern…classic IgA staining

With more in-depth questioning, he admits this is not his first episode

Natural history is very slow progression to ESRD

Poorer prognosis: older age, HTN, high BMI, low GFR, high uric acid level

If high proteinuria, poorest prognosis so attempt to treat

No treatment is all that effective

Steroids are used but ± results in trials

Fish oil may help but ± results in trials

MMF (mycophenolate mofetil) in trials

Sara

10 y/o female presents to ED with severe abdominal pain, bloody diarrhea (resolving), in town for family reunion with multiple picnics

PE: writhing child w/bloody stool, no vomiting, very pale, looks sick

Labs: WBC 14.5K mg/dL, SCr 4.5 mg/dL, Hgb 9 mg/dL

Admitted to ICU

What is the Diagnosis?

A. Post Strep GN
B. Membrano-proliferative GN
C. Viral GN
D. HUS

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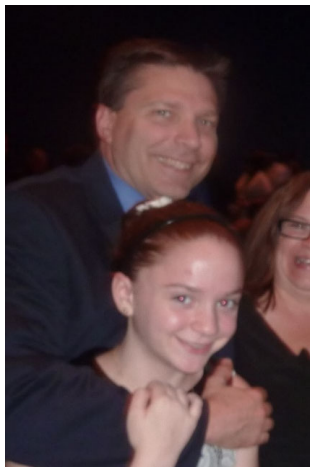

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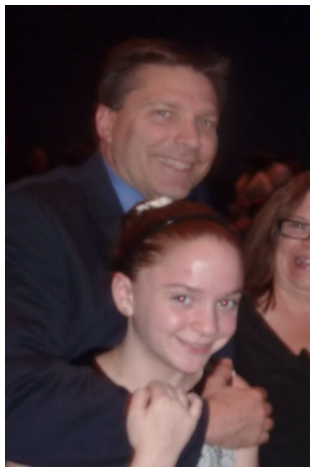
Admitted to ICU, SCr continues to increase (10.8 mg/dL), multi-organ failure with need for multiple transfusions, CRRT, on dialysis for >1mo, succumbs to the disease

Could this have been prevented?

A. Yes, fully cooking meat kills E.Coli 0157
B. No, even with cooking, the bacteria lives
C. Yes, if the family was vegetarian, this cannot happen
D. No, there is a 10% death rate even with RX



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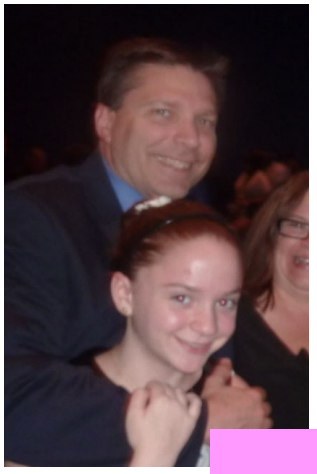

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Admitted to ICU, SCr continues to increase (10.8 mg/dL), multi-organ failure with need for multiple transfusions,

Even if meat is fully cooked, placing it on the pre-cooked plate will cause contamination!!!

E.coli 0157 is more often found in raw vegetables

E.coli 0157 is fecal to mouth transmission

Passed by swallowing water from lakes, pools

Large toddler outbreak in NE from unpasteurized apple juice and/or unpasteurized milk in toddlers

James

36 y/o AA male working as a glazer, presents w/abnormal labs after insurance physical
PMH: none
Family hx: HTN in multiple relatives
Meds: none
Labs: SCr 7.4 mg/dL (GFR 11), reck SCr 8.9mg/dL (GFR 9)

What is the most likely diagnosis?

A. Immunoglobulin A nephropathy (IgA)
B. HIV associated nephropathy (HIVAN)
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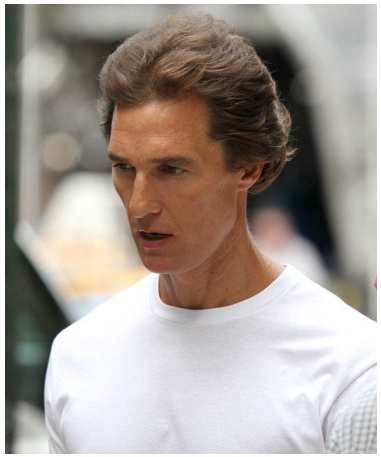

Tim

44 y/o HIV+ male presents to ID office for q 3mo visit
PMH: HIV well controlled
Meds: Stribild (elvitegravir, cobicistat, emtricitabine, temofovir), OTC vitamin
Labs: SCr 1.5 mg/dL (previous 0.7), K 4.0 meq/dL, CD4 T count 1450, undetectable HIV viral load,
Hgb 12.2 mg/dL, WBC 5.4 mg/dL

What is the most likely diagnosis?

A. Medication induced AKI
B. Focal sclerosing glomerulonephritis (FSGS)
C. Minimal change disease (MCD)
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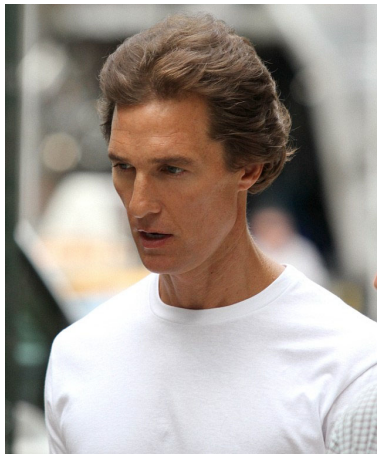

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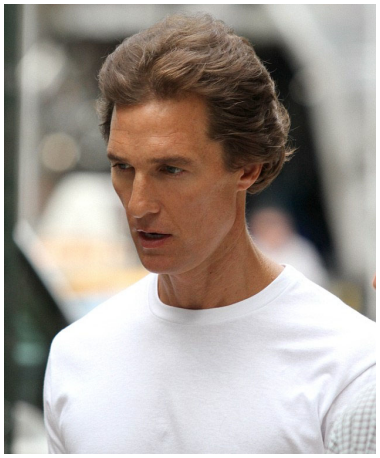

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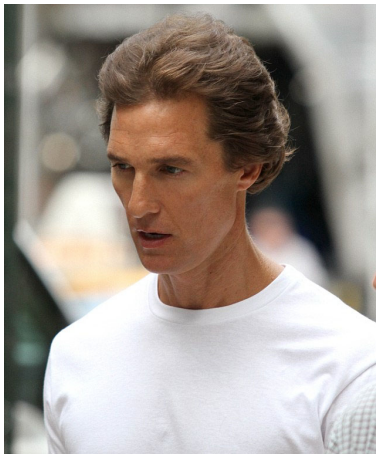
What is the treatment?

A. Steroids
B. Stopping Stribild, moving to another medication
C. None
D. Adding ACE/ARB



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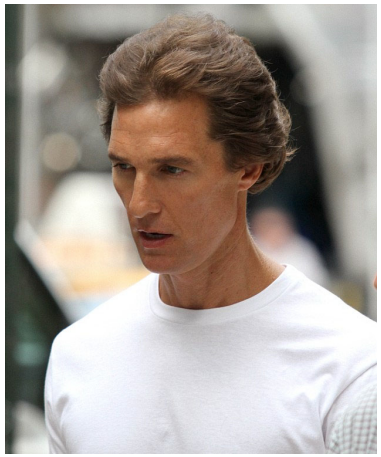

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Even without knowing the BP, you can usually add very low dose ACE/ARB as renal protection
As Tim has an undetectable viral load, changing HIV medications may be counterproductive
This may be medication-induced but it more likely chronic changes from the disease state

Carter 27 y/o male presents to ED with severe N&V

PMH: dinner with family friends at Mexican restaurant (chicken enchiladas)

Labs: WBC 11K, Hgb 13.5mg/dL mg/dL, SCr 10.8 mg/dL, K 3.4 meq/dL, micro pending

What is the most likely diagnosis?

A) Membranous Nephropathy GN
B) Post strep GN
C) Anti-Glomerular Basement Membrane GN
D) Post infectious GN

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AKI progressed to full-blown kidney failure
SCr stayed at 10.8 mg/dL with conservative care (IVF, antibiotics, Bicarb)
Dialysis for a month before resolution of AKI
Serial SCr 10.8 mg/dL, 5.88 mg/dL, now 2.0 mg/dL
Will have chronic kidney loss
Moral of the story... be careful where you eat!

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THANK YOU!

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