Acute and Chronic Glomerulonephritis

LECTURE IN INTERNAL MEDICINE FOR V COURSE STUDENTS

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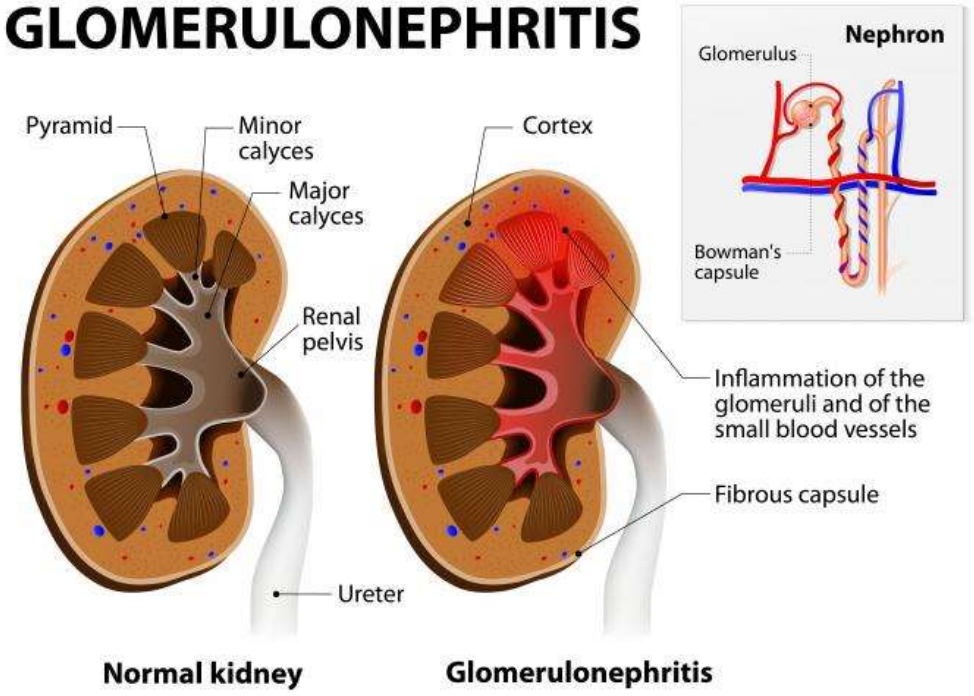
Plan of the Lecture

- Definition
- Epidemiology
- Risk factors
- Etiology
- Mechanisms
- Classification
- Clinical investigation
- Diagnosis
- Treatment
- Prognosis
- Prophylaxis
- Abbreviations
- Diagnostic and treatment guidelines



http://cdn1.medicalnewstoday.com/content/images/articles/167/167252/glomerulonephritis.jpg

Definition

Glomerulonephritis (glomerular nephritis , GN) denotes group of primary and secondary acute and chronic kidney diseases (usually affecting both kidneys) mostly characterised by immune mediated inflammation in the glomerular capillaries and the glomerular basement membrane (GBM), that may present with isolated hematuria and/or proteinuria, or as a nephrotic syndrome, a nephritic syndrome, a hypertensive syndrome and their combinations associated with progressive kidney failure, high cholesterol and anemia.

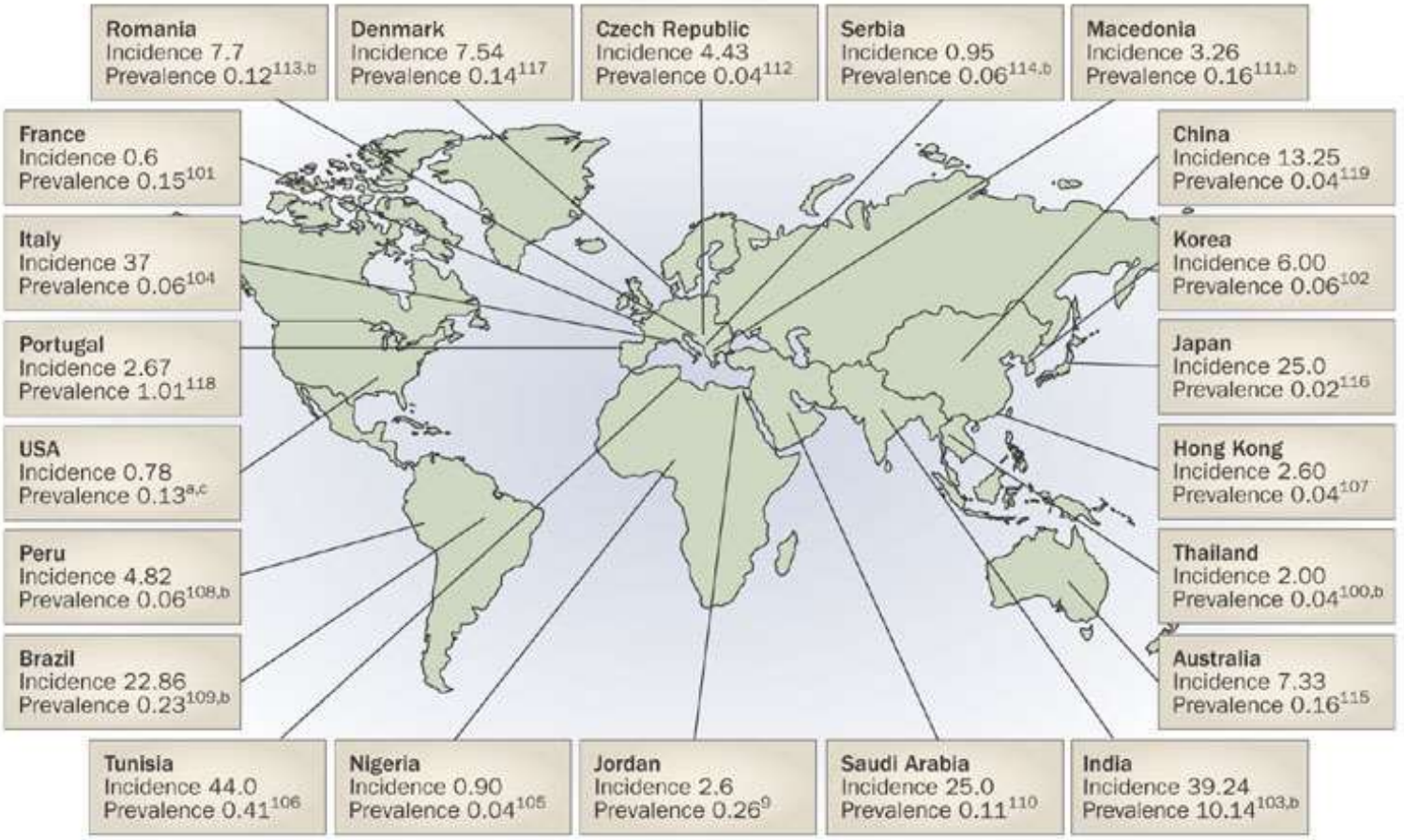
http://bestpractice.bmj.com/best-practice/monograph/207/basics.html

Epidemiology

- For every patient with clinically apparent GN, approximately 5 to 10 patients have undiagnosed subclinical disease.
- Incidence rates of primary GN vary between 0.2/100,000/year and 2.5/100,000/year.
- Focal segmental glomerulosclerosis is the most common cause of GN, especially among black patients.
- Membranous nephropathy (MN) used to be the most common biopsy diagnosis in adult patients.
- Idiopathic MN is more common in white men >40 years of age.
- MN is associated with lupus in young women and with hepatitis B in children.
- The incidence of IgA nephropathy is at least 2.5/100,000/year in adults; this disease can exist subclinically.

https://online.epocrates.com/diseases/20723/Glomerulonephritis/Epidemiology http://www.ncbi.nlm.nih.gov/pubmed/21068142

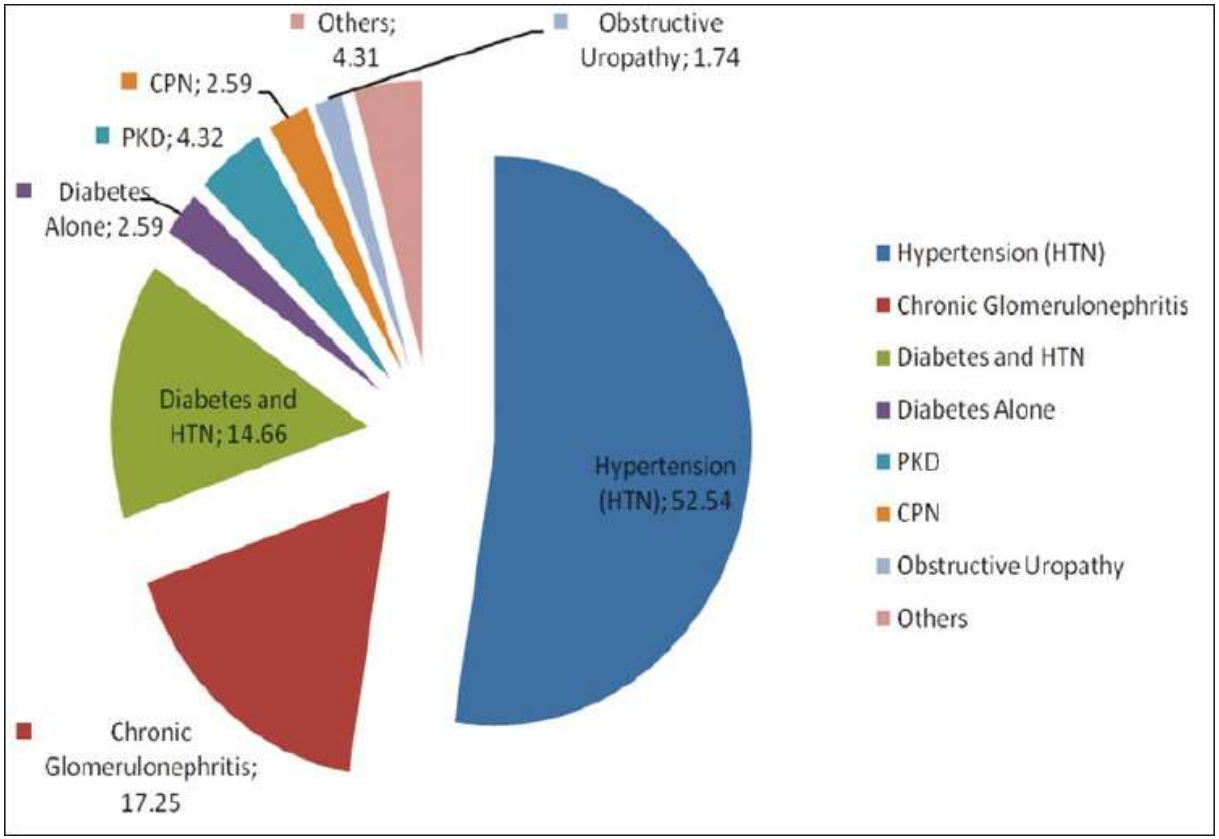
Epidemiology
(Acute Postinfectious Glomerulonephritis Worldwide)



Incidence is given as cases per year; prevalence is given as cases per 100,000 population. [a] Data from children. [b]Data from adults. [c]Data provided by Dr Tray Hunley, Division of Pediatric Nephrology, Vanderbilt Children's Hospital, Nashville, TN, USA.

http://www.nature.com/nrneph/journal/v5/n5/images/nrneph.2009.44-f1.jpg

Epidemiology
(Chronic GN and chronic kidney disease)



Etiology of chronic kidney disease **(**CKD): HTN = Hypertension, CGN = Chronic Glomerulonephritis, DM = Diabetes Mellitus, PKD = Polycystic Kidney Disease, CPN = Chronic Pyelonephritis, OU = Obstructive Nephropathy

http://www.annalsafrmed.org/article.asp?issn=1596-3519;year=2012;volume=11;issue=1;spage=21;epage=26;aulast=Chijioke

Risk Factors

- Disorders of the lymphatic system.
- Blood disorders.
- Family or personal history of cancer or malignant tumors.
- Exposure to chemicals such as toxic hydrocarbon solvents.
- Recurring strep infections or skin abscesses.
- Viral infections.
- Heart infections.
- Specific diseases (amyloidosis, vasculitis, polyarthritis, Goodpasture syndrome, IgA nephropathy, NSAIDs, lupus nephritis, Henoch-Schonlein purpura, etc.).

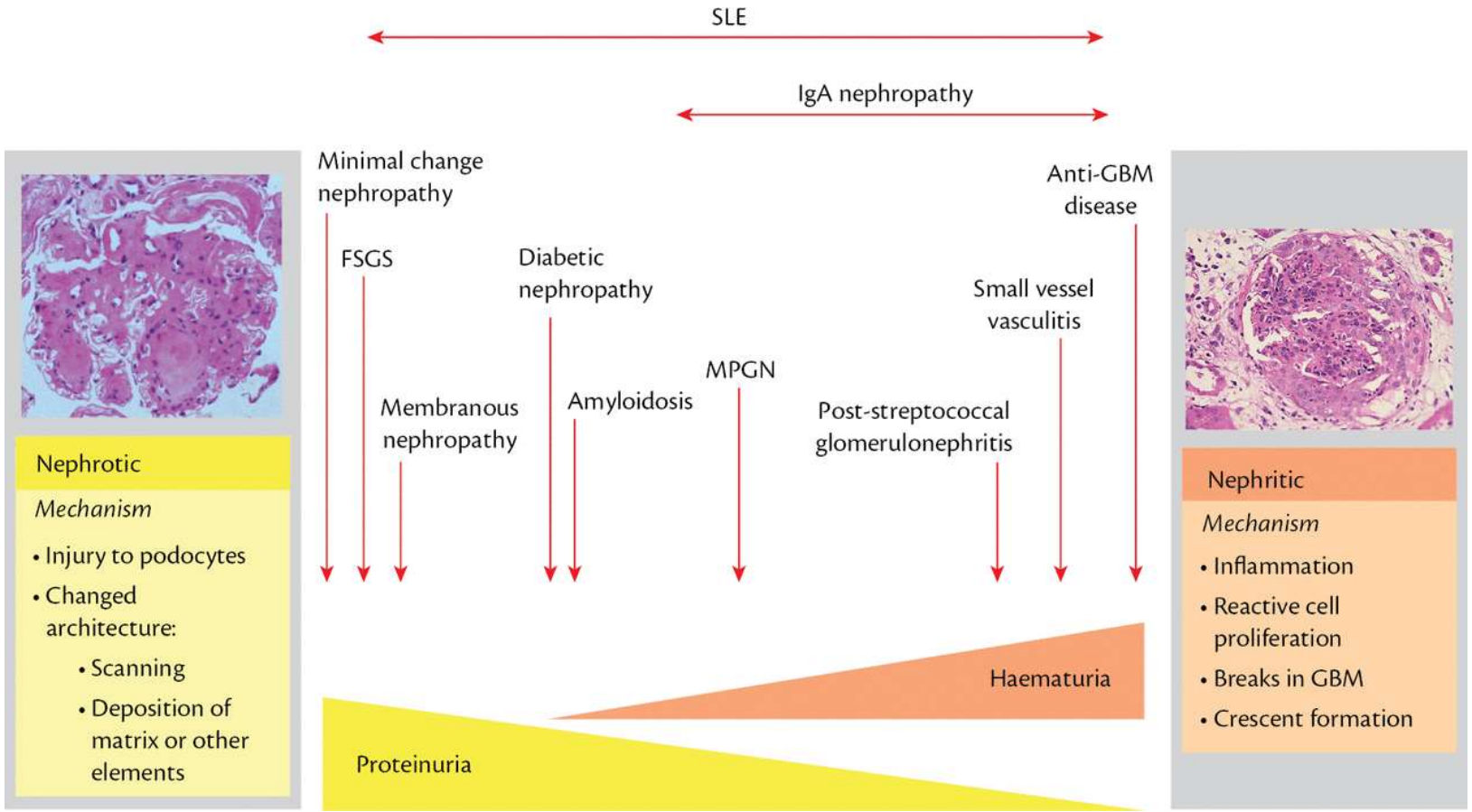
http://www.home-remedies-for-you.com/glomerulonephritis/causes.html

Etiology

- Renal-limited glomerulopathy or glomerulopathy-complicating systemic disease.
- Inflammation due to leukocyte infiltration, antibody deposition, and complement activation.
- Other causes:
  - Infections (e.g., group A beta-hemolytic *Streptococcus*, other bacterial infections, viruses).
  - Systemic inflammatory (e.g., SLE, rheumatoid arthritis).
  - Drugs (e.g., penicillamine, NSAIDs, captopril, heroin).
  - Metabolic disorders (diabetes mellitus, thyroiditis).
  - Malignancy (e.g., lung and colorectal cancer, Hodgkin lymphoma)
  - Hereditary (Fabry disease, Alport syndrome, etc.).
  - Deposition diseases (amyloidosis, light chain deposition disease).

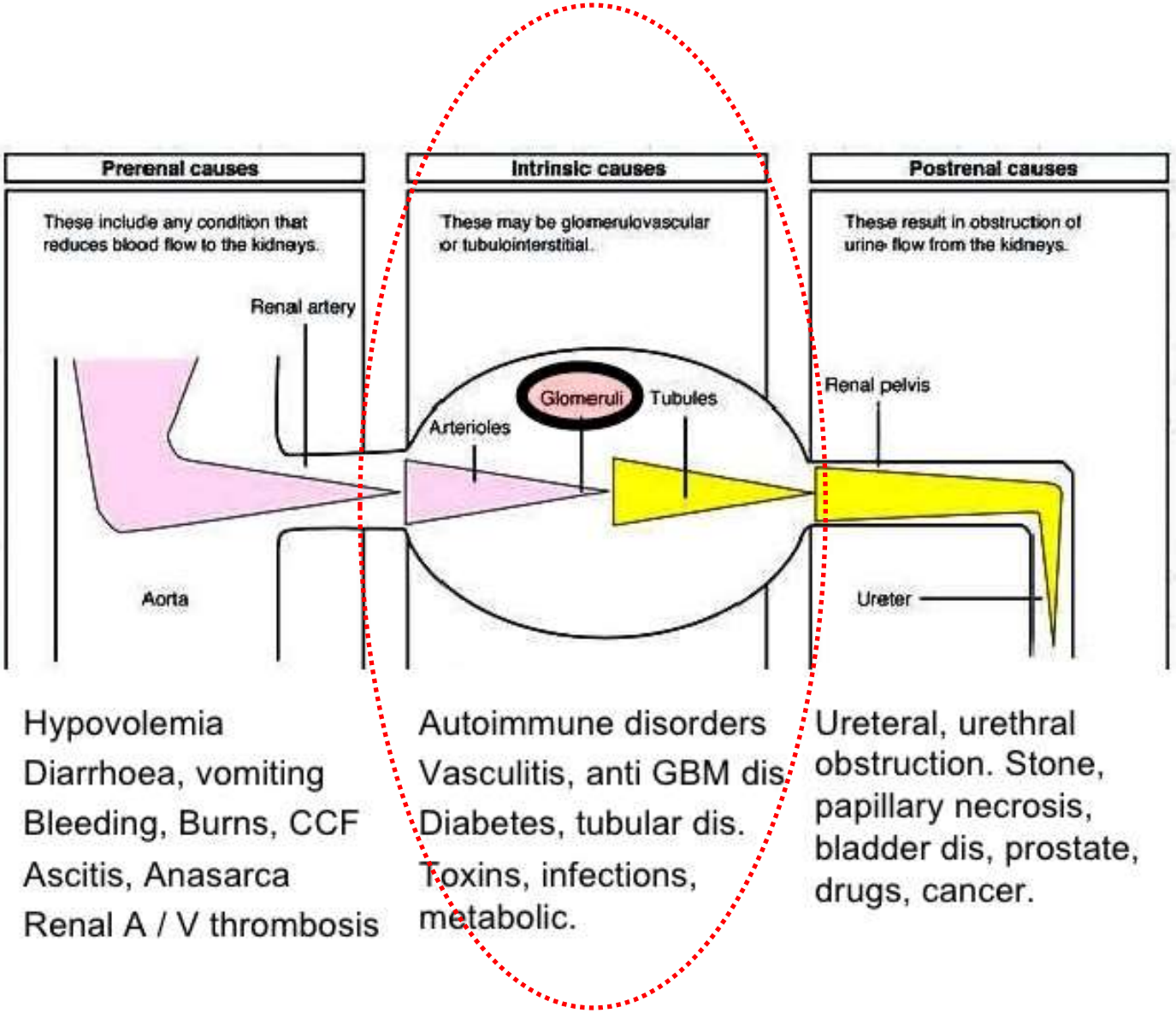
https://online.epocrates.com/diseases/20721/Glomerulonephritis/Definition

Etiology
(The Spectrum of Glomerular Diseases)



http://oxfordmedicine.com/doc/10.1093/med/9780199592548.001.0001/med-9780199592548-graphic-185-full.gif

Etiology
(Causes of Renal Disease and GN)



http://image.slidesharecdn.com/cpc-4-4-1-ren-gn-pathlec-view-091013211218-phpapp02/95/pathology-of-glomerulonephritis-29-728.jpg?cb=1255468487

Mechanisms
(Key Position of Immunologic Response)

- Most cases of GN are due to an immunologic response to a variety of different etiologic agents.
- The immunologic response, in turn, activates a number of biological processes (e.g., complement activation, leukocyte recruitment, and release of growth factors and cytokines) that result in glomerular injury and inflammation.
- GN may be isolated to the kidney (primary glomerulonephritis) or be a component of a systemic disorder (secondary glomerulonephritis).
- Humoral (also referred to T helper cell 2-regulated) immune response to a variety of inciting agents results in immunoglobulin deposition and complement activation within the glomeruli.

http://emedicine.medscape.com/article/239278-overview#a3

Mechanisms
(Acute GN)

- Glomerular lesions are the result of glomerular deposition or in situ formation of immune complexes.
- On gross appearance, the kidneys may be enlarged up to 50%.
- Histopathologic changes include swelling of the glomerular tufts and infiltration with polymorphonucleocytes.
- Immunofluorescence reveals deposition of immunoglobulins and complement.
- Except in poststreptococcal glomerulonephritis (PSGN), the exact triggers for the formation of the immune complexes are unclear.
- In PSGN, involvement of derivatives of streptococcal proteins has been reported.

http://emedicine.medscape.com/article/239278-overview#a3

Mechanisms
(Chronic GN: Reduction in Nephron Mass)

- Reduction in nephron mass from the initial injury reduces the glomerular filtration rate (GFR), that leads to hypertrophy and hyperfiltration of the remaining nephrons and to the initiation of intraglomerular hypertension with further glomerulosclerosis and nephron loss.
- In early renal disease, a substantial decline in the GFR may lead to only slight increases in serum creatinine levels.
- Azotemia (i.e., a rise in blood urea nitrogen (BUN) and serum creatinine levels) is apparent when the GFR decreases to less than 60-70 mL/min.

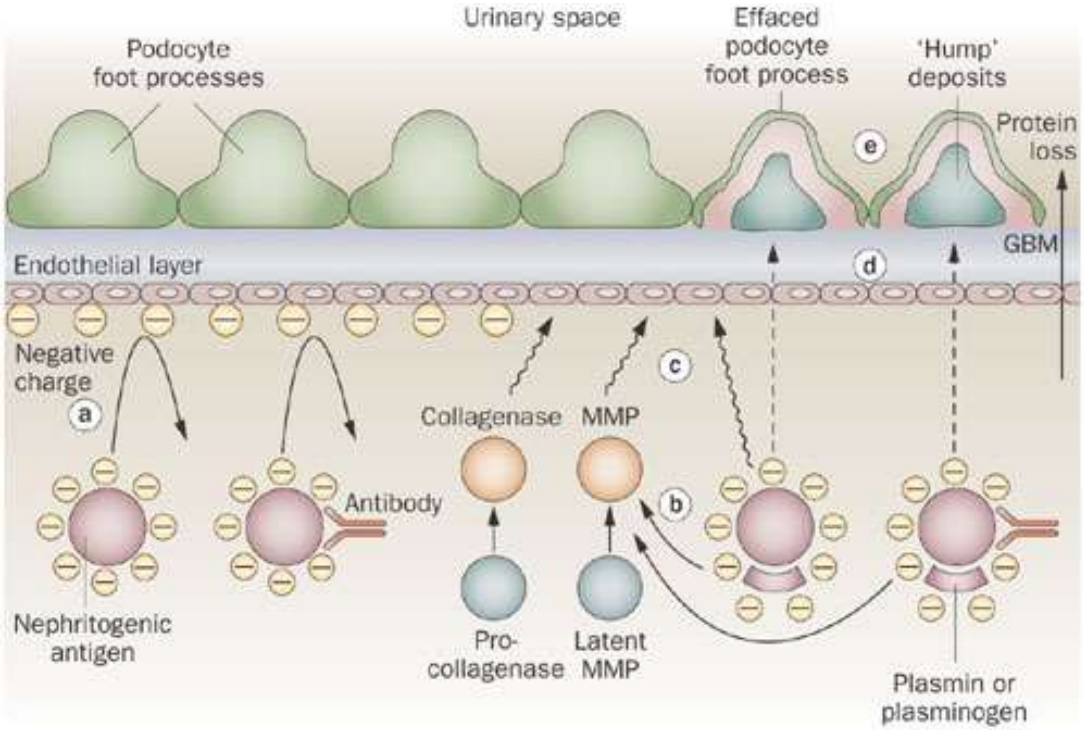
http://emedicine.medscape.com/article/239392-overview#a4

Mechanisms
(Chronic GN: Reduction in in the Glomerular Filtration Rate)

- Decreased production of erythropoietin, thus resulting in anemia.
- Decreased production of vitamin D, resulting in hypocalcemia, secondary hyperparathyroidism, hyperphosphatemia, and renal osteodystrophy.
- Reduction in acid, potassium, salt, and water excretion, resulting in acidosis, hyperkalemia, hypertension, and edema.
- Platelet dysfunction, leading to increased bleeding tendencies.
- Accumulation of toxic waste products (uremic toxins) affects virtually all organ systems.
- Uremia occurs at a GFR of approximately 10 mL/min.

http://emedicine.medscape.com/article/239392-overview#a4

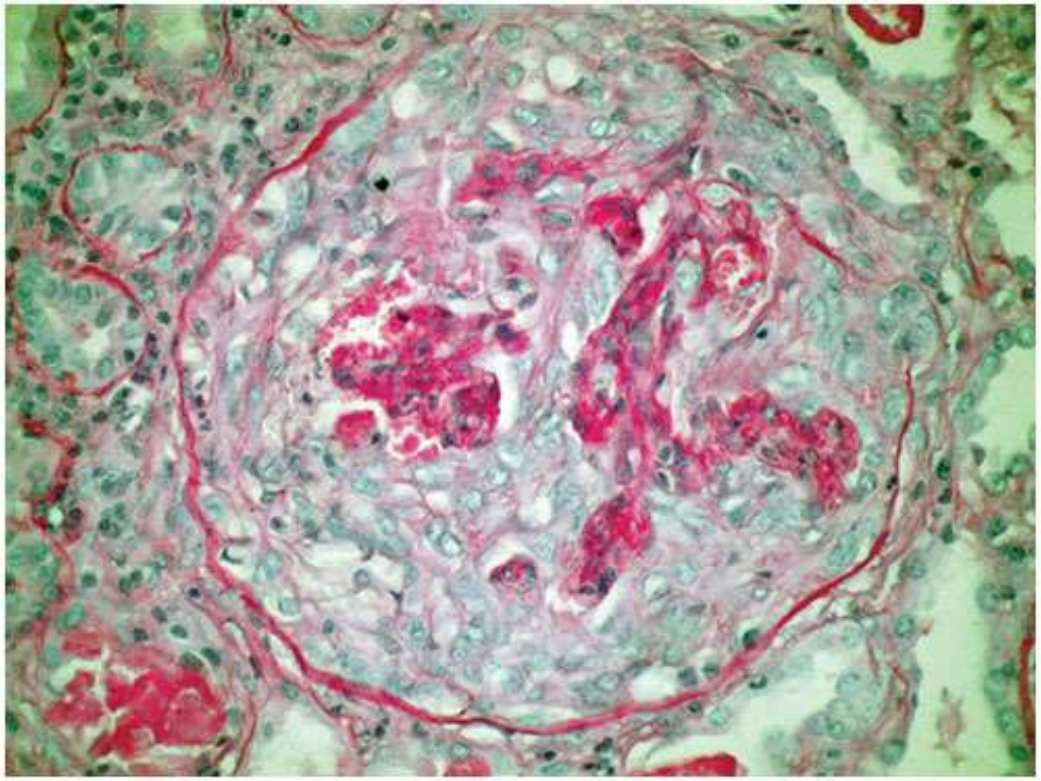
Mechanisms
(Possible Pathogenic Mechanism of Poststreptococcal GN)



Abbreviations: GADPH - streptococcal glyceraldehyde phosphate dehydrogenase, GBM - glomerular basement membrane, MMP - matrix metalloproteinase, SpeB - streptococcal cationic proteinase exotoxin B.

http://www.nature.com/nrneph/journal/v5/n5/fig_tab/nrneph.2009.44_F5.html

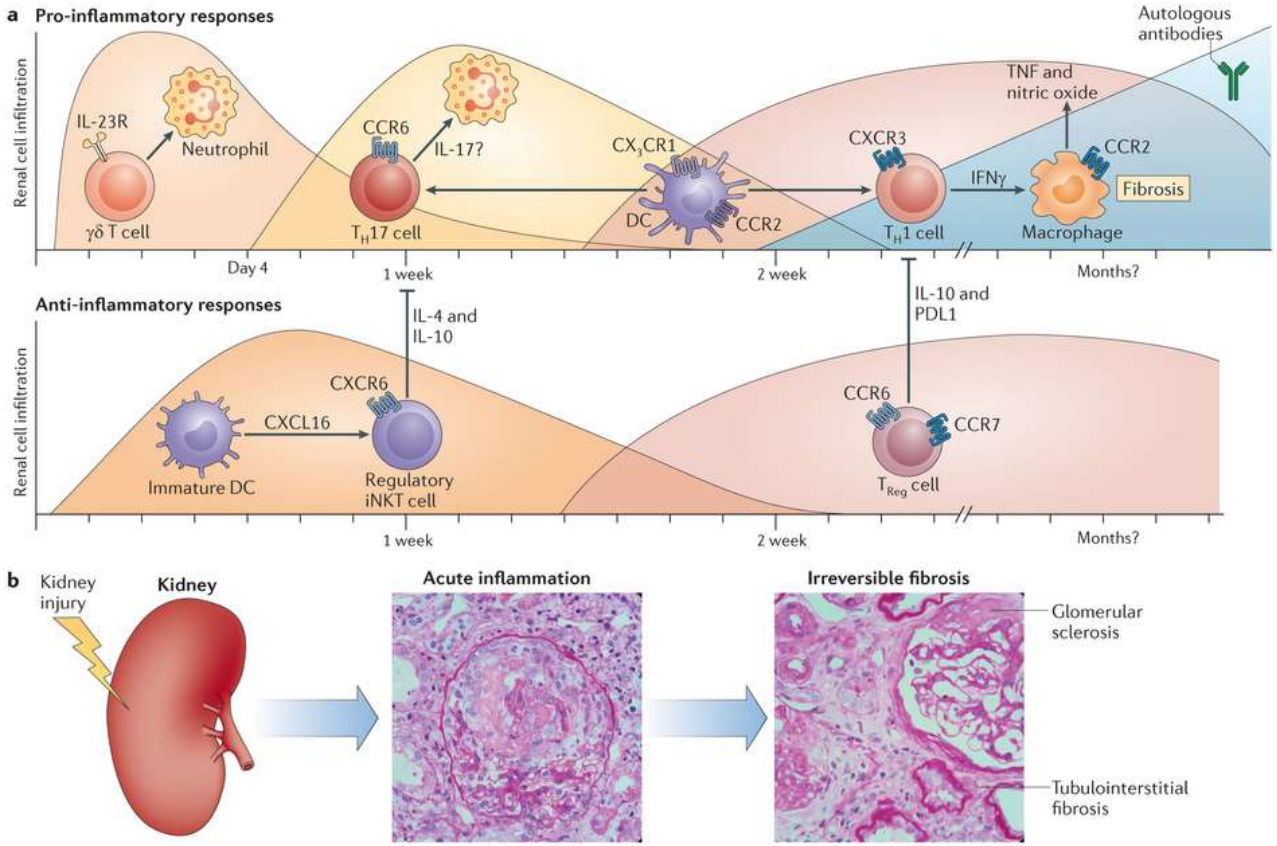
Mechanisms
(Focal Necrotizing GN)



The formation of crescents and the glomerular capillary necrosis (hematoxylin and eosin stain; original magnification 400).

http://www.nature.com/nrneph/journal/v5/n5/fig_tab/nrneph.2009.51_F1.html

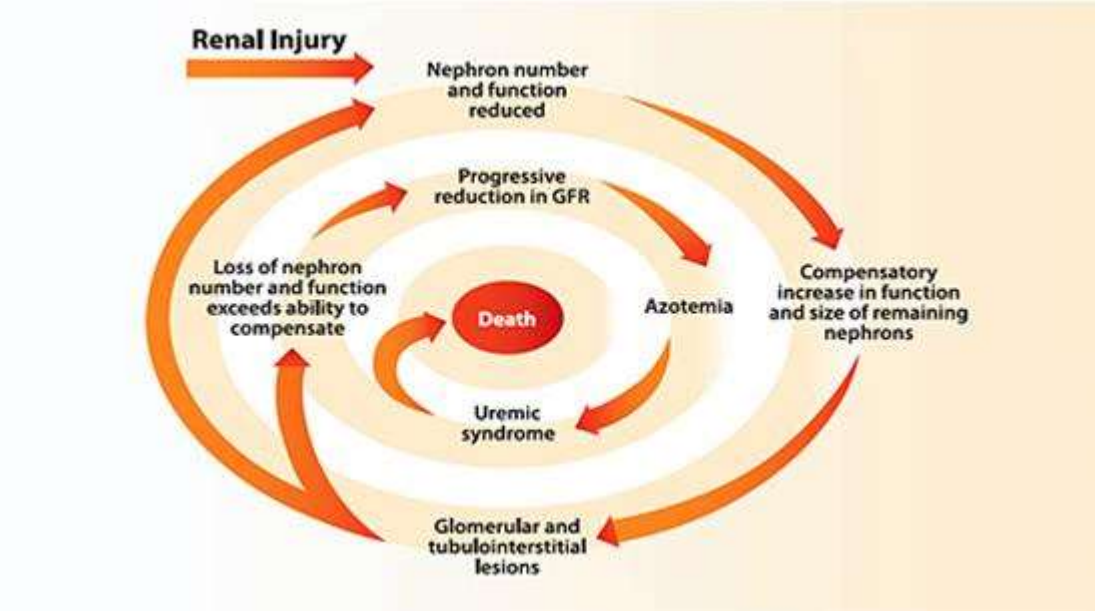
Mechanisms
(Focal Necrotizing GN)



The time-dependent changes in the pro-inflammatory and anti-inflammatory functions of leukocyte subsets during the course of crescentic GN.

http://www.nature.com/nri/journal/v13/n10/full/nri3523.html

Mechanisms
(Chronic Renal Failure can Result from a GN)



All forms of renal failure are characterized by a reduction in the GFR, reflecting a corresponding reduction in the number of functional nephrons.

http://intranet.tdmu.edu.ua/data/kafedra/internal/magistr/classes_stud/English/First%20year/Clinical%20Pathophysiology%20of%20Diseases/05.%20CP%20of%20kidney.htm

Classification
(International Classification of Diseases (ICD))

XIV Diseases of the genitourinary system.

N00-N08 Glomerular diseases.

- N00 Acute nephritic syndrome .
- N01 Rapidly progressive nephritic syndrome.
- N02 Recurrent and persistent hematuria.
- N03 Chronic nephritic syndrome.
- N04 Nephrotic syndrome .
- N05 Unspecified nephritic syndrome.
- N06 Isolated proteinuria with specified morphological lesion.
- N07 Hereditary nephropathy, not elsewhere classified.
- N08 Glomerular disorders in diseases classified elsewhere.

http://apps.who.int/classifications/icd10/browse/2016/en#/N00.2

Classification
(Primary and Secondary GN)

- Primary: the pathologic process is limited to the kidney and not part of a systemic disease manifestation (postinfectious GN, IgA nephropathy, antiglomerular basement membrane (anti-GBM) GN, idiopathic crescentic GN).
- Secondary: systemic lupus erythematosus (SLE), Henoch-Schonlein purpura, Wegener granulomatosis, microscopic polyangiitis, cryoglobulinemia, thrombotic microangiopathies, deposition diseases (amyloidosis, light chain deposition disease), malignancies (Hodgkin lymphoma, lung and colorectal cancer).

https://online.epocrates.com/diseases/20721/Glomerulonephritis/Definition

Classification
(Nephrotic/Nephritic)

Nephrotic syndrome (nephrotic-range proteinuria, hypoalbuminemia, hyperlipidemia, and edema):

- deposition diseases,
- minimal change disease,
- focal and segmental glomerulosclerosis,
- membranous nephropathy,
- membranoproliferative GN.

Nephritic syndrome (hematuria, subnephrotic-range proteinuria, and hypertension (HTN)):

- IgA nephropathy,
- postinfectious GN,
- rapidly progressive GN,
  - vasculitis,
  - anti–glomerular basement membrane (anti-GBM) GN.

https://online.epocrates.com/diseases/20721/Glomerulonephritis/Definition

Classification
(Nephritic and rapidly progressive GN (RPGN))

- Granular immune deposits (immune complex mediated GN).
- Linear immune deposits (anti-GBM GN).
- Pauci-immune GN.

https://online.epocrates.com/diseases/20721/Glomerulonephritis/Definition

Classification
(Severity)

- Mild: asymptomatic isolated hematuria or proteinuria <2 g.
- Moderate to severe: symptomatic proteinuria, hematuria, and reduced GFR (nephrotic and nephritic syndromes, and rapidly progressive GN).

https://online.epocrates.com/diseases/20736/Glomerulonephritis/Diagnostic-Criteria

Clinical Investigation
(Symptoms)

Symptoms depend on whether patient have the acute or chronic form, and the cause and may include:

- pink or cola-colored urine from red blood cells in urine (hematuria),
- foamy urine due to excess protein (proteinuria),
- high blood pressure (HTN),
- fluid retention (edema) with swelling evident in face, hands, feet and abdomen,
- fatigue from anemia or kidney failure.

http://www.mayoclinic.org/diseases-conditions/glomerulonephritis/basics/symptoms/con-20024691

Clinical Investigation
(Post-Streptococcal Glomerulonephritis)

https://i.ytimg.com/vi/bjwTnwvuY6E/hqdefault.jpg

Clinical Investigation
(Edema In Acute Renal Failure)

Clinical Investigation
(Sings)

- Asymptomatic proteinuria (150 mg - 3 g per day).
- Microscopic hematuria: >2 red blood cells (RBCs) per high power field in spun urine.
- Macroscopic hematuria: brown or smoky urine.
- Nephrotic syndrome: proteinuria >3.5 g/day, hypoalbuminemia less than 3.5 g/dL, edema, hypercholesterolemia and lipiduria.
- Nephritic syndrome: abrupt onset with oliguria, hematuria, proteinuria, azotemia, edema and hypertension.
- Rapidly progressive glomerulonephritis (RPGN): proteinuria, hematuria and renal failure developing over days to a week.
- Chronic GN: proteinuria, hypertension, renal failure and smooth contracted kidneys on ultrasonography (USG) examination.

http://www.apiindia.org/medicine_update_2013/chap128.pdf

Clinical Investigation
(Modalities)

- About half of the people with acute GN have no symptoms. If symptoms do occur, the first to appear are tissue swelling, low volume dark urine.
- In RPGN weakness, fatigue, and fever are the most frequent early symptoms. These patients have edema and usually produce very little urine.
- Because chronic GN usually causes only very mild or subtle symptoms, it goes undetected for a long time in most patients . The disease may progress to kidney failure.

http://www.merckmanuals.com/home/kidney-and-urinary-tract-disorders/kidney-filtering-disorders/glomerulonephritis-nephritic-syndrome

Clinical Investigation
(Complications)

- Acute kidney failure.
- Chronic kidney disease (CKD).
- High blood pressure.
- Nephrotic syndrome.

http://www.mayoclinic.org/diseases-conditions/glomerulonephritis/basics/complications/con-20024691

Diagnosis

- Clinical data.
- Urine examination.
- Blood tests:
  - complete blood cell count (CBC),
  - inflammatory markers and special tests:
    - anti-streptolysin O (ASLO),
    - anti-neutrophil cytoplasmic antibodies (ANCA),
    - Anti-GBM antibody ,
    - complement levels,
    - antinuclear antibodies,
- Imaging.
- Biopsy of the kidney.

https://en.wikipedia.org/wiki/Glomerulonephritis#Signs_and_symptoms

Diagnosis
(Clinical Data)

- Clinical features vary depending on the etiology and may include one or a combination of the following: hematuria (macroscopic or more commonly microscopic), proteinuria, and edema (characteristic of nephrotic syndrome).
- Hypertension may or may not be present, it is uncommon.
- Patients may have features of the underlying disorder, for example:
- joint pain, rash, and hemoptysis in vasculitis,
- fever and sore throat in streptococcal infections,
- jaundice in hepatitis B and C,
- weight loss in malignancies,
- stigmata of IV drug use.

https://online.epocrates.com/diseases/20731/Glomerulonephritis/Diagnostic-Approach

Diagnosis
(Laboratory tests: 1)

- A urinalysis and urine microscopy is generally the first test, and further testing is prompted on the basis of the results.
- Other initial recommended tests include GFR and creatinine evaluation, 24-hour urine collection, CBC, metabolic profile, and lipid profile.
- Urinalysis and renal function tests show hematuria and proteinuria. In complicated disease GFR and creatinine may suggest reduced renal function.

https://online.epocrates.com/diseases/20731/Glomerulonephritis/Diagnostic-Approach

Diagnosis
(Laboratory tests: 2)

- Proteinuria, measured by 24-hour urine collection, is generally <3.5 g/day, but if it is >3.5 g/day, patients are classified as having nephrotic-range proteinuria and may have full nephrotic syndrome (hyperlipidemia, hypoalbuminemia, edema, nephrotic-range proteinuria).
- Hematuria is characterized by dysmorphic RBCs and formation of RBC casts that are best seen in freshly prepared urine sediments.

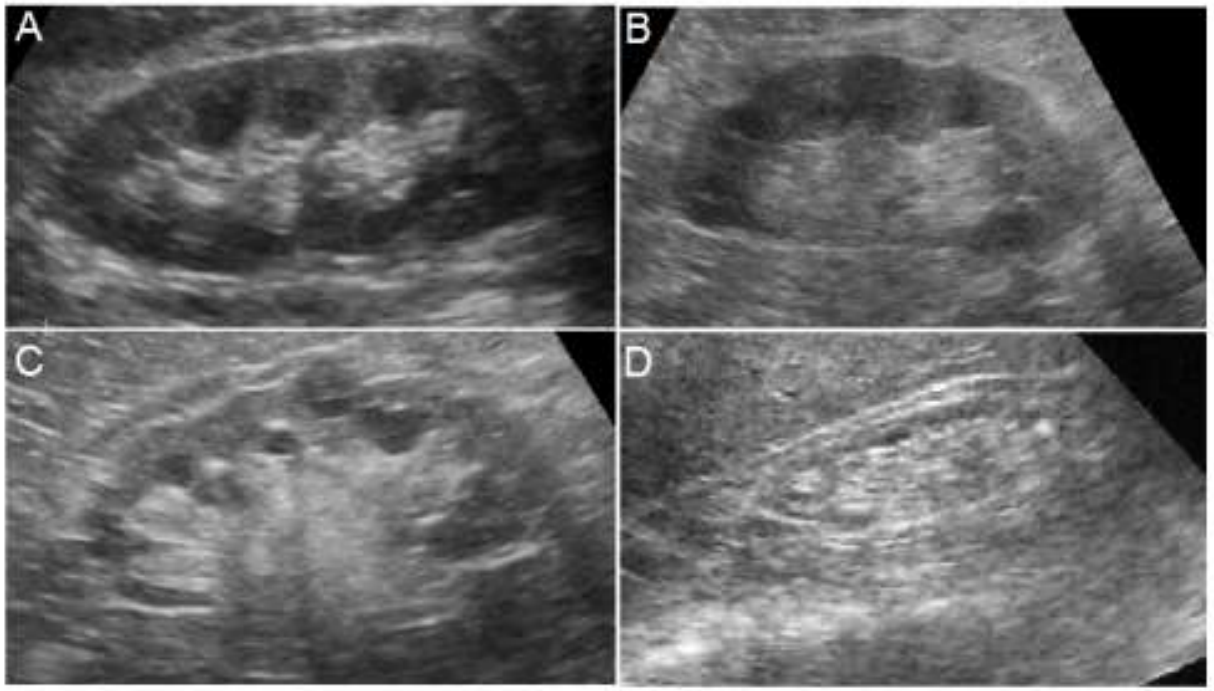
https://online.epocrates.com/diseases/20731/Glomerulonephritis/Diagnostic-Approach

Diagnosis
(Laboratory tests: 3)

- Anemia, hyperglycemia (if diabetic), hyperlipidemia (nephrotic picture), and hypoalbuminemia (nephrotic picture) may also be evident from the CBC, and from metabolic and lipid profiles.
- If urinalysis indicates GN, subsequent tests are ordered to determine the etiology and hence to guide the treatment. Specific serologic testing for systemic causes include erythrocyte sedimentation rate (ESR), complement, ANCAs, anti-GBM antibodies, monoclonal protein on serum or urine electrophoresis, antistreptococcal antibodies, circulating cryoglobulin, HIV serology, hepatitis B virus serology, hepatitis C virus serology, and drug toxicology screen.

https://online.epocrates.com/diseases/20731/Glomerulonephritis/Diagnostic-Approach

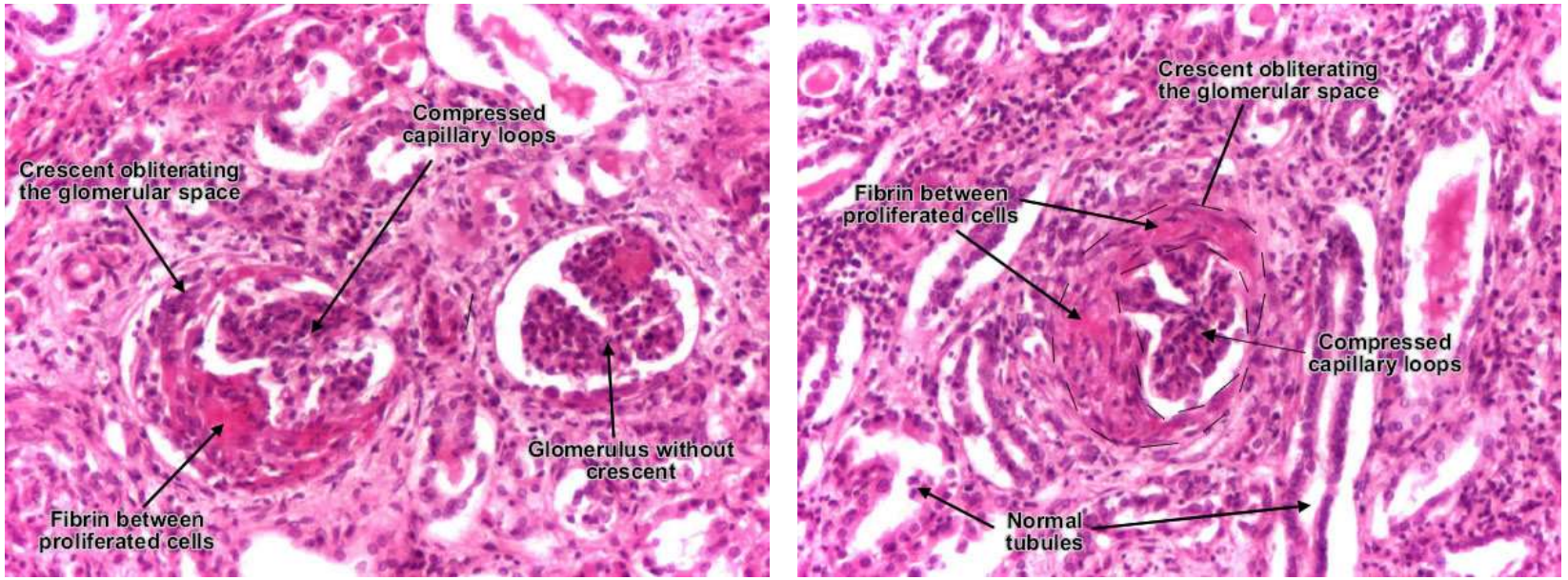
Diagnosis
(Ultrasound)



Morphological aspect of the kidney with acute kidney injury at B-mode renal ultrasound.

http://www.esciencecentral.org/ebooks/ultrasound-approach/renal-ultrasound-in-acute-kidney-disease.php

Diagnosis
(Biopsy of the Kidney)



Rapidly progressive ("crescentic") GN. Formation of crescents is initiated by passage of fibrin into the Bowman space as a result of increased permeability of glomerular basement membrane. Fibrin stimulates the proliferation of parietal cells of Bowman capsule, and an influx of monocytes. Rapid growing and fibrosis of crescents compresses the capillary loops and decreases the Bowman space which leads to renal failure within weeks or months. (H&E, ob. x20).

http://www.pathologyatlas.ro/rapidly-progressive-crescentic-glomerulonephritis.php

Diagnosis
(Distinguishing Acute Kidney Injury From Chronic Kidney Disease)

Finding	Comment
Prior known increase in serum creatinine	Most reliable evidence of CKD
Renal sonogram showing small kidneys	Usually CKD
Renal sonogram showing normal or enlarged kidneys	May be AKI or some forms of CKD (diabetic nephropathy, acute hypertensive nephrosclerosis, PCKD, myeloma, rapidly progressive glomerulonephritis, infiltrative diseases [eg, lymphoma, leukemia, amyloidosis], obstruction)
Oliguria, daily increases in serum creatinine and BUN	Probably AKI or AKI superimposed on CKD
Eye-band keratopathy	Probably CKD
No anemia	Probably AKI or CKD due to PCKD
Severe anemia, hyperphosphatemia, and hypocalcemia	Possibly CKD but may be AKI
Subperiosteal erosions on radiography	Probably CKD
Chronic symptoms or signs (eg, fatigue, nausea, pruritus, nocturia, hypertension)	Usually CKD

AKI = acute kidney injury; CKD = chronic kidney disease; PCKD = polycystic kidney disease.

https://www.merckmanuals.com/professional/genitourinary-disorders/chronic-kidney-disease/chronic-kidney-disease

Diagnosis
(Renal Biopsy Definitive Diagnosis of GN)

Glomerulonephritis	Site of renal injury	Clinical presentation	Serological markers
Postinfectious glomerulonephritis	Endothelial cell injury	Nephritic syndrome	Antibodies to streptococcus, low complement
IgA nephropathy	Mesangial cell injury	Nephritic syndrome	None
Anti GBM nephritis	Endothelial cell injury	Rapidly progressive GN	Anti-GBM, ANCA
Vasculitis	Endothelial cell injury	Rapidly progressive GN	ANCA
Lupus nephritis, class I	Mesangial cell injury	Mild form of GN	Anti-DNA
Lupus nephritis, class II	Mesangial cell injury	Microscopic hematuria and/or proteinuria	Anti-DNA
Lupus nephritis, class III, IV	Endothelial cell injury	Nephritic syndrome	Hypocomplementemia and elevated anti-DNA levels
Lupus nephritis, class V	Epithelial cell injury	Nephrotic syndrome	Hypocomplementemia and elevated anti-DNA levels
Minimal change disease	Epithelial cell injury	Nephrotic syndrome	None
Focal and segmental glomerulosclerosis	Epithelial cell injury	Nephrotic syndrome	None
Membranous nephropathy	Epithelial cell injury	Nephrotic syndrome with slow progression	Depends on underlying etiology
Mesangioproliferative GN	Mesangial cell injury	Nephrotic syndrome with slow progression	Depends on underlying etiology

https://online.epocrates.com/diseases/20731/Glomerulonephritis/Diagnostic-Approach

Diagnosis
(Staging CKD)

1. Normal GFR (≥ 90 mL/min/1.73 m^2) plus either persistent albuminuria or known structural or hereditary renal disease.
2. GFR 60 to 89 mL/min/1.73 m^2 .
3. GFR 30 to 59 mL/min/1.73 m^2 .
4. GFR 15 to 29 mL/min/1.73 m^2 .
5. GFR < 15 mL/min/1.73 m^2 .

GFR (in mL/min/1.73 m^2) in CKD can be estimated by: 186.3 × (serum creatinine)$^{-1.154}$×(age)$^{-0.203}$. The result is multiplied by 0.742 if the patient is female and by 1.21 if the patient is African American. For female African Americans, the result is multiplied by 0.742 × 1.21 (0.898).

https://online.epocrates.com/diseases/20731/Glomerulonephritis/Diagnostic-Approach

Diagnosis (Differential)

Condition	Differentiating Signs/Symptoms	Differentiating Tests
Nephrolithiasis	Severe pain in addition to hematuria depend on the position of the stone.	Hematuria, no dysmorphic RBC. Renal ultrasound reveals the stone.
Bladder cancer	Painless hematuria. Patients are older and with a history of smoking.	Hematuria, no dysmorphic RBCs. Diagnosis is made by cystoscopic biopsy.
Renal cancer	A triad of flank pain, fever, and hematuria is typical.	Hematuria, no dysmorphic RBCs. Imaging by CT would reveal a renal mass.
Pre- or post-renal failure	Vague generalized symptoms (fatigue, loss of appetite, and nausea) besides those of the underlying etiology.	No dysmorphic RBCs or casts. Fractional excretion of sodium is <1% in azotemia due to prerenal causes. Renal imaging shows obstructive uropathy.

https://online.epocrates.com/diseases/20735/Glomerulonephritis/Differential-Diagnosis

Treatment
(Lifestyle and Dietary Advice)

- People with chronic GN be encouraged to undertake physical activity compatible with cardiovascular health and tolerance (aiming for at least 30 min five times per week), achieve a healthy weight (body mass index 20–25 kg/m^2, according to country-specific demographics), and stop smoking.
- People with chronic GN receive expert dietary advice and information in the context of an education program, tailored to severity of CKD and the need to intervene on salt, phosphate, potassium, and protein intake where indicated.

http://www.medscape.com/viewarticle/819798_10

Treatment
(Goal of Specific Therapy)

- The goal of specific therapy for GN is to reverse the renal damage or to preserve the renal function, that is monitored by checking renal function and the degree of proteinuria.
- Most of the specific treatment include plasmapheresis, corticosteroid therapy, and immunosuppression.
- Complications such as HTN and hyperlipidemia should be managed appropriately to counteract cardiovascular events, as well as to delay progression of renal pathology.
- Treatment is patient specific and directed toward the underlying etiology and managing the complications.

https://online.epocrates.com/diseases/20741/Glomerulonephritis/Treatment-Approach

Treatment
(Mild Disease)

Patients who present with isolated hematuria, minimal or no proteinuria, and a normal GFR have a better outcome and may not need specific therapies other than treating the systemic cause (e.g., antibiotics, antivirals, withdrawal of the causative drug).

https://online.epocrates.com/diseases/20741/Glomerulonephritis/Treatment-Approach

Treatment
(Moderate to Severe Disease)

- Patients with hematuria, proteinuria, and reduced GFR are managed with specific therapies targeted at reversing the underlying etiology, for example, antibiotics in acute nephritic poststreptococcal GN.
- Nonspecific pharmacologic measures that reduce proteinuria and are also first-line considerations for controlling HTN, including angiotensin-converting enzyme (ACE) or angiotensin-II receptor antagonists.
- Edema is controlled by salt-restricted diet (1–2 g/day), oral or intravenous (IV) loop diuretics alone or combined with thiazide.
- Severe disease presenting as nephrotic syndrome is usually treated with corticosteroids and immunosuppressants.

http://www.apiindia.org/medicine_update_2013/chap128.pdf

Treatment
(Anticoagulation and Statins)

- Anticoagulation with low molecular weight heparin or warfarin is mandatory if serum albumin is below 2 g/dL with one or more of the following: proteinuria greater than 10 g/day, body mass index greater than 35 kg/m2, family history of thromboembolism, congestive heart failure, recent abdominal or orthopedic surgery or prolonged immobilization.
- Statins are well tolerated and effective in correcting lipid profile, but not proven to reduce cardiovascular events or reduce decline of glomerular filtration rate (GFR).

http://www.apiindia.org/medicine_update_2013/chap128.pdf

Treatment
(Rapidly Progressive Disease (RPGN))

- Categorized into the following:
  - Antiglomerular basement membrane (linear type),
  - Immune-complex mediated (granular type): postinfectious causes, connective tissue disease, IgA nephropathy, and membranoproliferative GN,
  - Pauci-immune: Wegener granulomatosis and polyarteritis nodosa.
- Corticosteroids and immunosuppressant therapy with or without plasmapheresis (depending on the etiology) are the mainstay of treatment for severe and progressive disease presenting acutely with massive proteinuria and acute renal failure.

https://online.epocrates.com/diseases/20741/Glomerulonephritis/Treatment-Approach

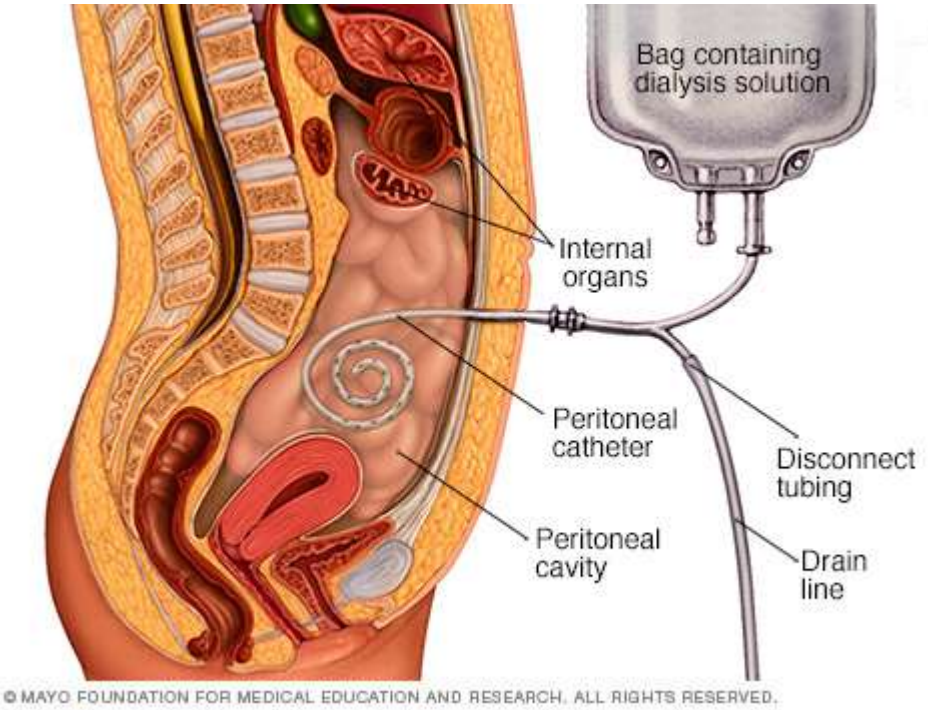
Treatment
(Definitive Diagnosis and Treatment of GN)

Glomerulonephritis	Site of renal injury	Clinical presentation	Serological markers	Treatment
Postinfectious glomerulonephritis	Endothelial cell injury	Nephritic syndrome	Antibodies to streptococcus, low complement	Antibiotics Rarely steroids
IgA nephropathy	Mesangial cell injury	Nephritic syndrome	None	Treatment of underlying cause
Anti GBM nephritis	Endothelial cell injury	Rapidly progressive GN	Anti-GBM, ANCA	Plasmapheresis, Pulse methylprednisone, cyclophosphamide
Vasculitis	Endothelial cell injury	Rapidly progressive GN	ANCA	Pulse methylprednisone, cyclophosphamide
Lupus nephritis, class I	Mesangial cell injury	Mild form of GN	Anti-DNA	No specific therapy
Lupus nephritis, class II	Mesangial cell injury	Microscopic hematuria and/or proteinuria	Anti-DNA	No specific therapy
Lupus nephritis, class III, IV	Endothelial cell injury	Nephritic syndrome	Hypocomplementemia and elevated anti-DNA levels	Pulse methylprednisone, cyclophosphamide
Lupus nephritis, class V	Epithelial cell injury	Nephrotic syndrome	Hypocomplementemia and elevated anti-DNA levels	Pulse methylprednisone, cyclophosphamide if progressive
Minimal change disease	Epithelial cell injury	Nephrotic syndrome	None	Oral steroids
Focal and segmental glomerulosclerosis	Epithelial cell injury	Nephrotic syndrome	None	Treatment of underlying cause, antiproteinuric measures, oral steroids, immunosuppressants
Membranous nephropathy	Epithelial cell injury	Nephrotic syndrome with slow progression	Depends on underlying etiology	Oral steroids and cyclophosphamide
Mesangioproliferative GN	Mesangial cell injury	Nephrotic syndrome with slow progression	Depends on underlying etiology	Oral steroids

https://online.epocrates.com/diseases/20731/Glomerulonephritis/Diagnostic-Approach

Treatment
(End-stage Renal Disease Therapy)

- Hemodialysis.
- Peritoneal dialysis.
- Renal transplantation.



http://www.mayoclinic.org/tests-procedures/peritoneal-dialysis/home/ovc-20202856 http://emedicine.medscape.com/article/239392-overview#showall

Prognosis

- Most epidemic cases follow a course ending in complete patient recovery (as many as 100%). The mortality of acute GN has been reported at 0-7%.
- Sporadic cases of acute GN often progress to a chronic GN. This progression occurs in as many as 30% of adult patients and 10% of pediatric patients. GN is the most common cause of CKD (25%).
- Approximately 15% of patients at 3 years and 2% of patients at 7-10 years may have persistent mild proteinuria.
- Generally, the prognosis is worse in patients with heavy proteinuria, severe hypertension, and significant elevations of creatinine level.
- Serum creatinine levels and the proportion of crescents were the most important predictors of developing CKD.

http://emedicine.medscape.com/article/239278-overview#showall

Prophylaxis

- There may be no way to prevent most forms of GN.
- Some interventions may be beneficial:
  - prompt treatment of a strep infection,
  - prompt treatment of a viral infection,
  - control high blood pressure
  - control blood sugar.

http://www.mayoclinic.org/diseases-conditions/glomerulonephritis/basics/prevention/con-20024691

Abbreviations

ACE - angiotensin-converting enzyme

ANCAs - anti-neutrophil cytoplasmic antibodies

ASLO - anti-streptolysin O

BUN - blood urea nitrogen

CBC - complete blood cell count

CGN - chronic glomerulonephritis

CKD - chronic kidney disease

DM - diabetes mellitus

ESR - erythrocyte sedimentation rate

ESRD - end-stage renal disease

GADPH - streptococcal glyceraldehyde

GBM - glomerular basement membrane

GFR - glomerular filtration rate

GI – gastrointestinal

GN – glomerulonephritis

HIV - the human immunodeficiency virus

HTN - hypertension

MMP - matrix metalloproteinase

NSAIDs - nonsteroidal anti-inflammatory drugs

PKD - polycystic kidney disease

PSGN - poststreptococcal glomerulonephritis

RBCs - red blood cells

RPGN - rapidly progressive GN

SLE - systemic lupus erythematosus

anti-GBM antibody - anti–glomerular basement membrane antibody

SpeB - streptococcal cationic proteinase exotoxin B

USG - ultrasonography

phosphate dehydrogenase

OU - obstructive nephropathy

Diagnostic and treatment guidelines

KDIGO Clinical Practice Guideline for Glomerulonephritis

Acute Glomerulonephritis: Evidence-based Management

Evidence-based clinical practice guidelines for rapidly progressive glomerulonephritis 2014

Acute kidney injury: prevention, detection and management