Волгоградский государственный медицинский университет

Кафедра патологической анатомии

LECTURE: Diseases of Kidneys

The Kidney

- The kidney serves as the principal regulator of the fluid and electrolyte content of the body. This task is accomplished by the complex filtering mechanism of the glomerulus and the selective tubular reabsorption of the solutes from the filtrate.

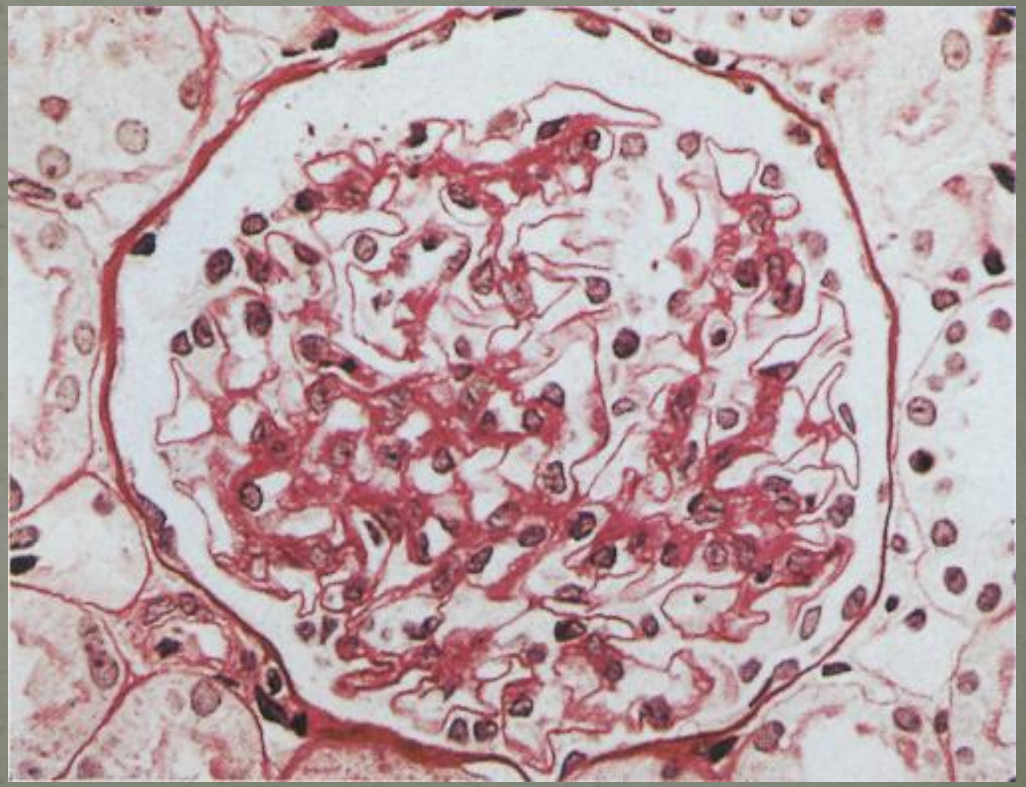
The Kidney
Glomerulus

- The kidney is also an endocrine organ, secreting renin,which regulates sodium metabolism and blood pressure, and erythropoietin, a hormone, that stimulates production of red blood cells by the bone marrow.

The Kidney
Glomerulus

- The glomerulus is a specialized tuft of capillaries with an arteriole at either end.

Light micrograph of a normal glomerulus. The periodic acid-Schiff stain highlights the delicate basement membranes and mesangial matrix.



The Kidney
Glomerulus

- Glomerular Basement Membrane.

The glomerular basement membrane is functionally and chemically distinct. It is approximately 300 nm in thickness and has three definable layers by electron microscopy:

1) a central dense zone, the lamina densa;

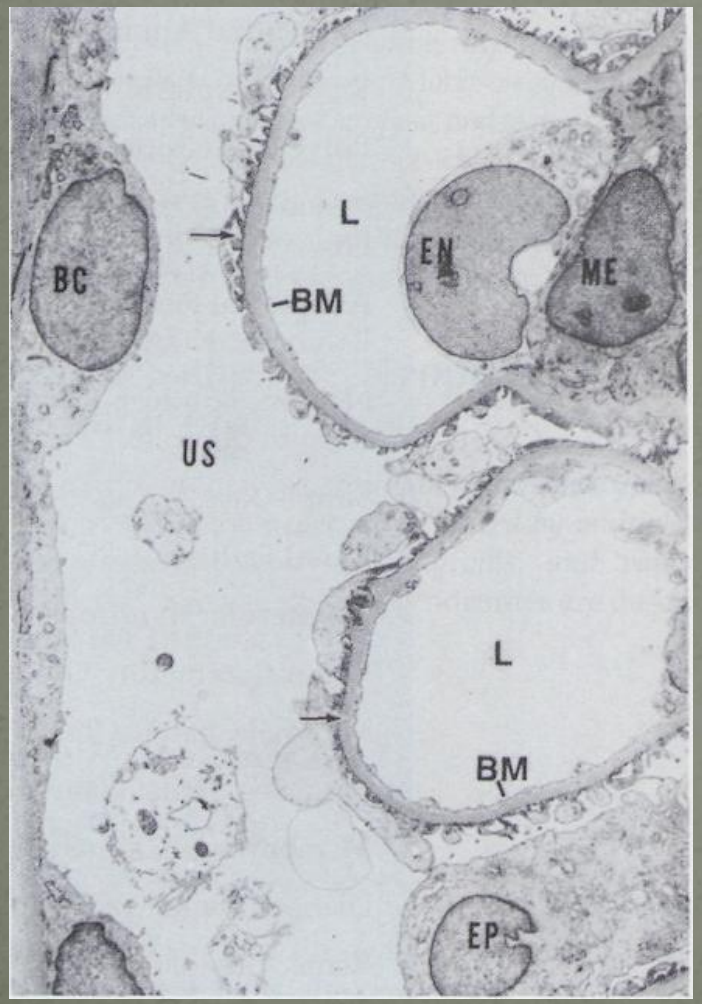
2) a paler lamina rara interna; and

3) a lamina rara externa.

The Kidney
Glomerulus

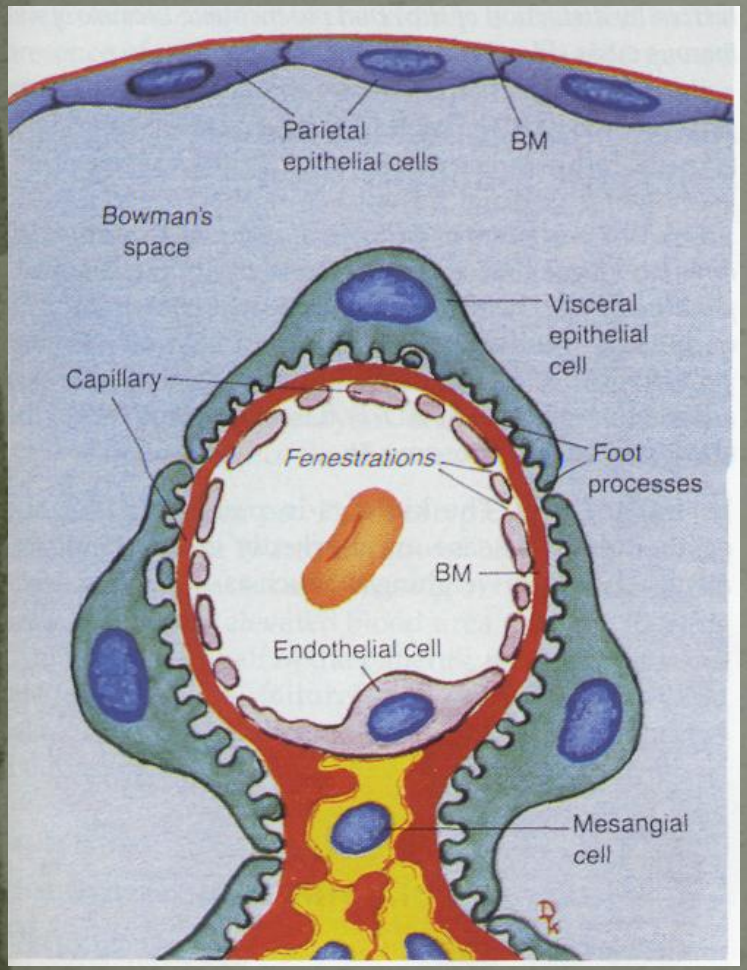
- The glomerular basement membrane has a strong negative charge owing to the presence of the polyanionic proteoglycan heparan sulfate. This property allows charge-selective filtration of electrically neutral and cationic molecules.

The Kidney
Glomerulus



Electron micrograph of a normal glomerulus. The normal glomerular capillary is covered by epithelial cells (EP), **with foot processes *(arrows)* in contact with the basement membrane (BM). The endothelial cell** (EN) **has large pores and surrounds the capillary lumen (L). The mesangial cell (ME) is bordered by the endothelial cell on the luminal surface and by the stalk basement membrane on the lateral ar-eas. *BC,* Bowman capsule; US, urinary space.**

The Kidney
Glomerulus



Normal glomerulus. The relation of the different glomerular cell types to the stroma is illustrated using a single glomerular loop. The entire outer aspect of the glomerular **basement** membrane (BM; peripheral loop and stalk) is **covered by** the epithelial cell foot processes. The outer **portions** of the endothelial cell, which surrounds the capillary **lumen,** are in contact with the inner surface of the basement **membrane,** whereas the central part is in contact with the **mesangial** cell of the stalk. The relationship of the mesan**gial** cell to **its** stroma is unique to the glomerulus and has **not yet** been entirely clarified.

The Kidney
Glomerulus

- *Endothelial Cells.*

- Glomerular capillaries have a fenestrated endothelial layer. The fenestrate are too large to influence capillary permeability, however, and the endothelium probably plays a largely passive role in filtration.

The Kidney
Glomerulus

- Epithelial Cells

- Glomerular epithelial cells line Bowman's space. The visceral epithelial cells rest on the basement membrane and send cytoplasmic projections, termed *foot processes*, onto it.

The Kidney
Glomerulus

- Mesangium. The glomerulus is supported by a mixed cellular and stromal network that is collectively termed the mesangium. Important functions of the mesangium include
- 1) endocytosis and processing of plasma proteins;
- 2) modulation of glomerular filtration by the contractility of mesangial cells;
- 3) generation of vasoactive agents (e.g. prostaglandins and cytokines).

Classification of primary glomerulopathies

Glomerulonephritis (inflammatory glomerulopathies)

- **Acute GN**
- **Rapidly progressive (subacute) GN**
- **Chronic GN:**

I Minimal glomerular changes - immunopositive
II Membranous
III Mesangial
· Mesangiomembranous
· Mesangioproliferative:
- Berger's disease
· Mesangiocapillary (membrane-proliferative)
type 1 (glomerulonephritis with subendothelial deposits)
type 2 (dense deposits disease)
type 3 (GN with subendothelial and subepithelial deposits)
IV Fibroplastic

Non-inflammatory glomerulopathies

- Hereditary glomerulopathy
- Disease of thin basement membranes
- Membranous glomerulopathy (nephropathy)
- Lipoid nephrosis (minimal change disease)
- Focal segmental glomerular sclerosis / hyalinosis

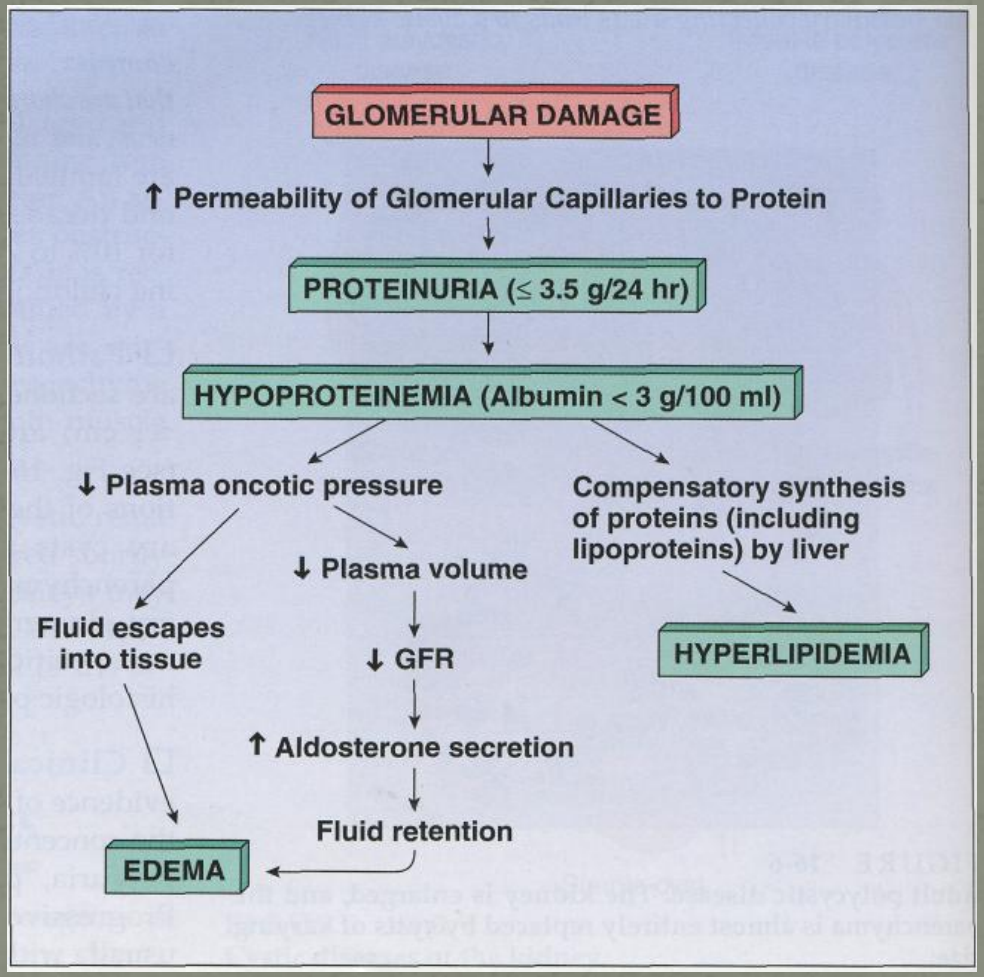
Secondary glomerulopathies

- Diabetic glomerulosclerosis
- Amyloid nephropathy
- Paraproteinemic nephropathy (with multiple myeloma, Waldenstrom macroglobulinemia, cryoglobulinemia)
- Lupus nephropathy
- Glomerulonephritis associated with bacterial endocarditis
- Glomerulonephritis in Schönlein – Genoch disease

The Kidney
GLOMERULAR SYNDROMES

- **Nephrotic syndrome is characterized principally by heavy proteinuria (>3.5 g of protein per 24 hours) together with hypoproteinemia (hypoalbuminemia), peripheral edema, and hyperlipidemia**

The Kidney
GLOMERULAR SYNDROMES



Pathophysiology of the nephrotic syndrome

The Kidney
GLOMERULAR SYNDROMES

There are major causes of nephrotic syndrome in both adults and children. The vast majority of the cases of "pure“ nephrotic syndrome (those without features of the nephritic syndrome) result from glomerular diseases within the category of noninflammatory glomerulopathies.

The Kidney
GLOMERULAR SYNDROMES

Major Causes of the Nephrotic Syndrome

Adults

Membranous nephropathy (30%)
Glomerulopathies associated with systemic diseases (20-30%)
Epithelial cell (minimal change) disease (20%)
Focal segmental glomerulosclerosis (10-20%)
Membranoproliferative glomerulonephritis (5%)
***Other* primary glomerulopathies (5%)**

Children

Epithelial cell (minimal change) disease (70%)
Focal segmental glomerulosclerosis (10%)
Membranous nephropathy (5-10%)
***Glomerulopathies associated with systemic diseases* (5-10%)**
Other primary glomerulopathies (5%)

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GLOMERULAR SYNDROMES

Nephritic syndrome is characterized by

(I) hematuria,

(II) proteinuria (<3.5 g/d),

(III) oliguria, and

(IV) decreased glomerular filtration rate (with resulting elevations in blood urea nitrogen and serum creatinine values).

(V) hypertension and edema

The Kidney
GLOMERULAR SYNDROMES

Salt and water retention often results in hypertension and edema. In clear contrast to the nephrotic syndrome, the nephritic syndrome is most often associated with inflammatory glomerular diseases (glomerulonephritis).

The Kidney
GLOMERULAR SYNDROMES

Signs and Symptoms of Nephritic Syndrome

Hematuria (gross or microscopic)
Decreased urine output (oliguria)
Elevated blood urea nitrogen and serum creatinine levels
Hypertension
Proteinuria (< 3.5 g per 24 hours), with or without edema

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Minimal Change Glomerulopathy

Minimal change glomerulopathy is a glomerular disorder that is characterized clinically by the nephrotic syndrome and pathologically by fusion of the visceral epithelial foot processes.

Minimal change nephrotic syndrome (also known as epithelial cell disease or lipoid nephrosis) is largely a disorder of children, in whom it is the major cause of nephrotic syndrome.

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NONINFLAMMATORY GLOMERULOPATHIES

Minimal Change Glomerulopathy

However, the disease also occurs in adults with a significant frequency (20% of adults with nephrotic syndrome). Most children with the disease are boys who initially present before the age of 6 years.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Minimal Change Glomerulopathy

Pathogenesis.: Neither the cause nor the pathogenesis of minimal change nephrotic syndrome is understood. Because of the association between the onset of nephrotic syndrome and an allergic history, and also because the disease sometimes follows infection or exposure to allergens, involvement of the immune system has been postulated.

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NONINFLAMMATORY GLOMERULOPATHIES

Minimal Change Glomerulopathy

In experimental models, heavy proteinuria has been related to a loss of polyanionic sites on the glomerular basement membrane. The loss of these sites allows anionic proteins, particularly albumin, to pass easily through the normal barrier.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Minimal Change Glomerulopathy

Pathology: By definition, the light microscopic appearance of glomeruli in patients with minimal change nephrotic syndrome (epithelial cell disease) is essentially normal. However, there is often some irregular prominence of the epithelial cells, and minor degrees of mesangial enlargement are common.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Minimal Change Glomerulopathy

Electron microscopic examination of the glomeruli reveals total effacement of the epithelial cell foot processes, with the basement membrane being covered by a sheet of cytoplasm. Scanning electron microscopy shows that loss of foot processes results from their retraction into the parent epithelial cell bodies rather than from actual fusion. Numerous microvilli protrude from the surface of the epithelial cells.

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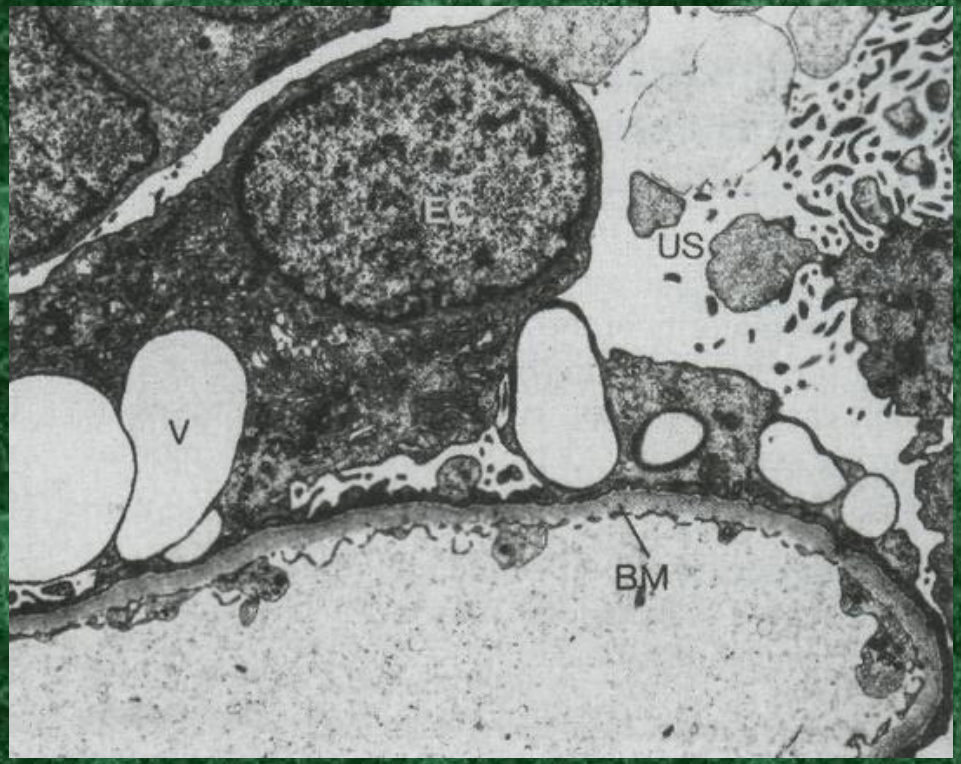
NONINFLAMMATORY GLOMERULOPATHIES

Minimal Change Glomerulopathy



Epithelial cell disease. In this electron micrograph, the epithelial cells (EC) display foot process effacement, "villous" hyperplasia, numerous vacuoles (V). BM, basement membrane; *US,* urinary space.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Minimal Change Glomerulopathy

Clinical Features: Most patients with epithelial cell disease show complete remission of proteinuria within 8 weeks of the initiation of corticosteroid therapy. However, after the withdrawal of corticosteroids, approximately half of these patients suffer intermittent relapses for as long as 10 years. Each relapse is responsive to corticosteroid therapy, however, and there is no tendency to progress into chronic renal failure.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Minimal Change Glomerulopathy

Death from infection was frequent before antibiotics and corticosteroids became readily available. A fatal outcome or progression to renal failure is now exceptional. Thus, in the absence of complications, the longterm outlook for patients with epithelial cell disease today is probably no different from that of the general population.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis

Focal segmented glomerulosclerosis ***refers to a malady in which some (focal) glomeruli exhibit segmental areas of sclerosis in the capillary tufts whereas others appear to be normal.*** **The majority of these cases occur in the context of nephrotic syndrome in children, accounting for 10% of such cases. In adults, 10% to 20% of the cases of nephrotic syndrome also exhibit focal segmental glomerulosclerosis.**

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis

Pathogenesis: Focal segmental glomerulosclerosis represents a morphologic pattern that is common to at least three circumstances:

Idiopathic. The majority of the cases of focal segmental glomerulosclerosis are of unknown cause.

Major features that distinguish idiopathic focal segmental glomerulosclerosis from the typical case of epithelial cell disease are (1) the poor response to corticosteroids, (2) the poorly selective nature of the proteinuria, (3) the presence of focal sclerosing lesions early in the disease, and (4) an unfavorable clinical course.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis

Decreased Renal Mass. Focal segmental glomerulosclerosis occurs in conditions characterized by a decrease in functional renal mass (e.g., unilateral nephrectomy or agenesis) or in various types of acquired renal disease.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis

The ratio of renal mass to body mass is also decreased in patients with morbid obesity, a condition in which the most frequent cause of nephrotic syndrome is focal segmental glomerulosclerosis.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis

As a result, increased glomerular capillary pressure and filtration injure the cells of the glomerulus and lead to capillary thrombosis, microaneurysms, mesangial enlargement, and subendothelial hyaline deposits.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis

These lesions cause focal segmental glomerulosclerosis which may progress to sclerotic obliteration of the affected glomeruli. Low-protein diets and certain drugs that ameliorate the hemodynamic disturbances in the kidney protect, at least to some extent, against the development and progression of glomerular injury.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis

Healed Focal Glomerular Lesions. The separation of focal segmental glomerulosclerosis from healed lesions of other focal glomerular disorders (e.g., focal glomerulonephritis, microscopic polyarteritis, malignant hypertension) on morphologic groundsalone is difficult.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

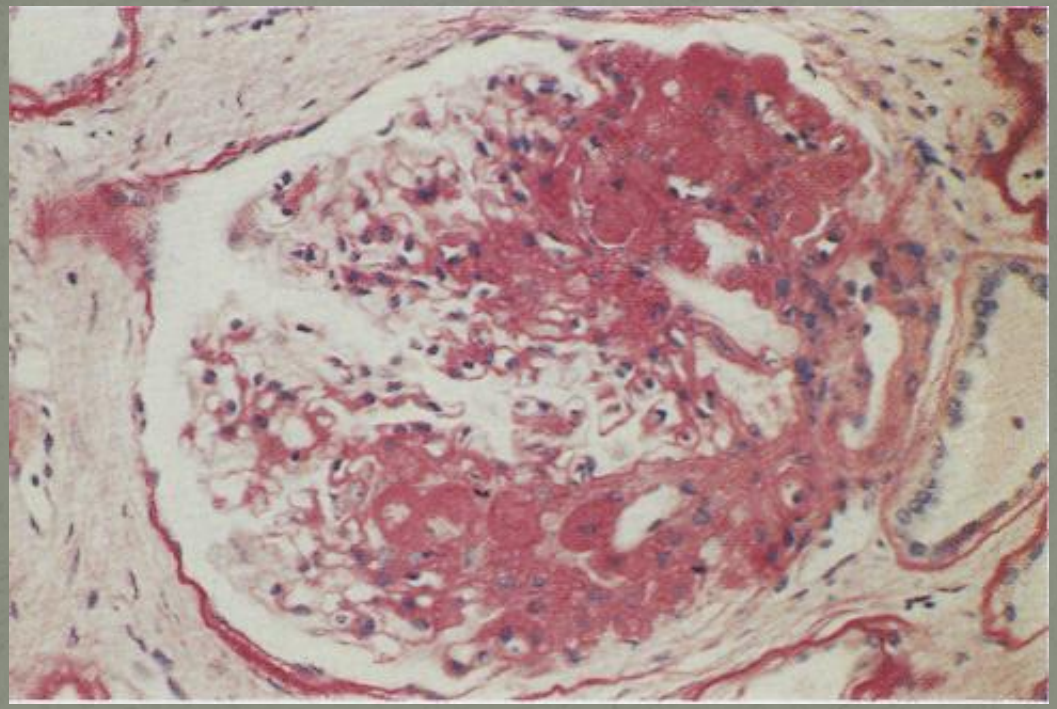
Focal Segmental Glomerulosclerosis

Pathology: By light microscopy, varying numbers of glomeruli show segmental areas of capillary loop obliteration, initially in the juxtamedullary glomeruli.

The Kidney

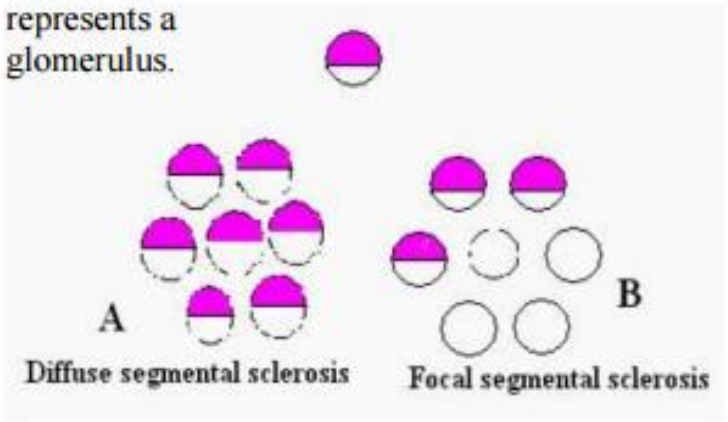
NONINFLAMMATORY GLOMERULOPATHIES

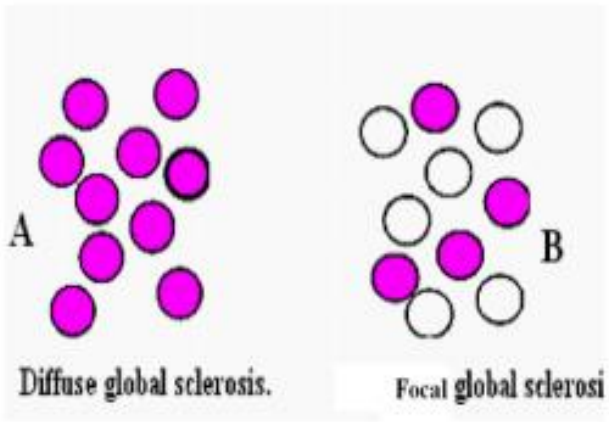
Focal Segmental Glomerulosclerosis

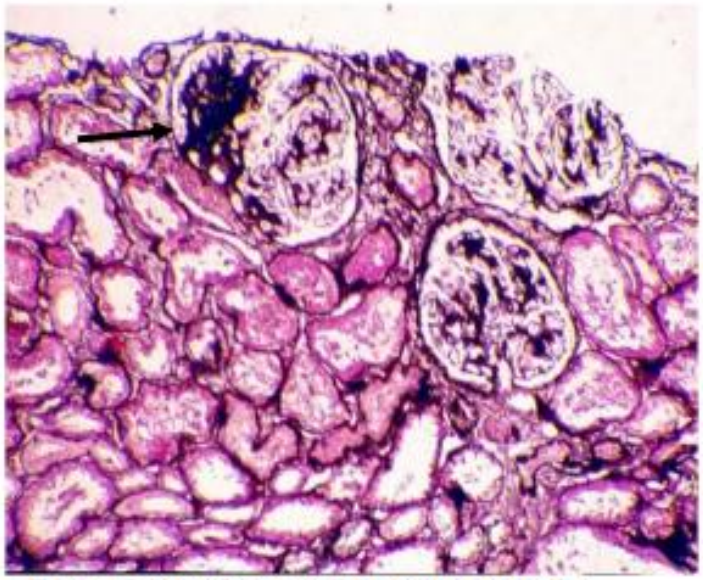


Focal segmental glomerulosclerosis. A periodic acid-Schiff stain shows perihilar areas of segmental sclerosis and adjacent adhesions to the Bowman capsule.

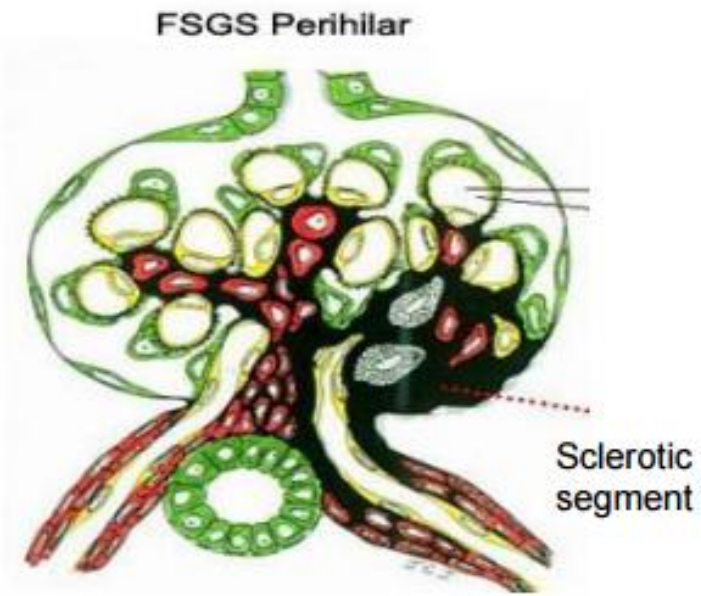
Focal segmental glomerulosclerosis







One of the glomeruli is segmentally sclerotic (arrow)



The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis

Adhesions to the Bowman capsule are seen adjacent to these lesions. The mesangium is hypercellular, and lipid-containing foam cells are often found within it. The frequent accumulation of a periodic acid-Schiff (PAS)-positive material in the affected areas produces a lesion referred to as hyalinosis.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis

Uninvolved glomeruli appear to be entirely normal, although on occasion, a diffuse, global mesangial hypercellularity is superimposed on all the glomeruli.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

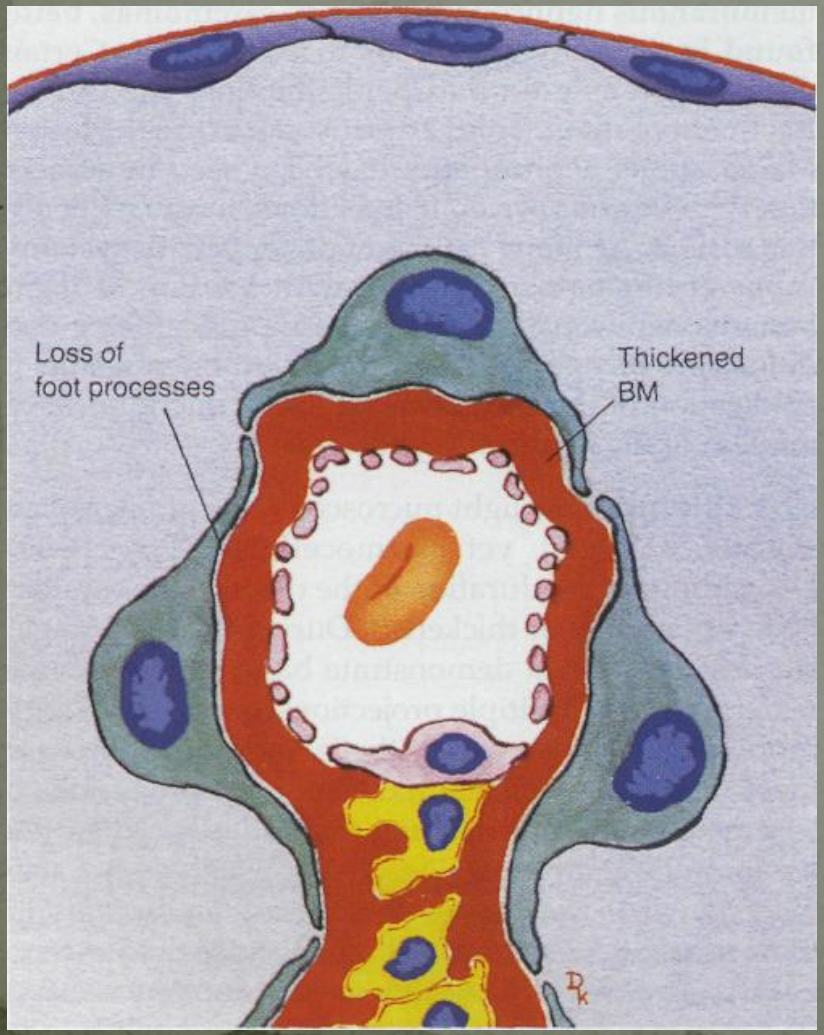
Focal Segmental Glomerulosclerosis

By electron microscopy, diffuse effacement of the epithelial cell foot processes is identical to the lesion of epithelial cell disease. In addition, folding and thickening of the basement membrane.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis



Focal segmental glomerulosclerosis. This disorder typically displays epithelial cell change and basement membrane thickening. Epithelial cells show effacement of the foot processes and distention of the cytoplasm. The basement membrane is thickened and folded. The glomeruli located deep within the cortex are the earliest to demonstrate these pathognomonic changes

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis

Immunofluorescence studies show trapping of immunoglobulin (Ig) M and C3 in the segmental areas of sclerosis and hyalinosis. IgG, C4, and Clq are less frequently found. This trapping of immune proteins is nonspecific and, presumably, is not related to the pathogenesis of this disease.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis

Clinical Features: Most patients with the diagnosis of focal segmental glomerulosclerosis present with the insidious onset of proteinuria and the nephrotic syndrome. Many of these patients are hypertensive, and microscopic hematuria is frequent.

The Kidney

NONINFLAMMATORY GLOMERULOPATHIES

Focal Segmental Glomerulosclerosis

When focal segmental glomerulosclerosis is detected soon after the onset of nephrotic syndrome, it is probably progressive at all ages. Most patients suffer an uninterrupted decline in renal function for a period of as long as 10 years.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy

- ***Membranous glomerulopathy is a frequent cause of nephrotic syndrome in adults and results from the accumulation of immune complexes in the subepithelial zone of glomerular capillaries.*** **In fact, this condition is the most frequent cause of nephrotic syndrome in adults (30% of cases). Although the disease has been associated with many precipitating factors, most cases are idiopathic.**

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy

- **Pathogenesis:** **Membranous nephropathy is believed to result from the deposition of immune complexes from the circulation or the formation in situ of immune complexes within the capillary walls. Electron-dense immune complex deposits have been demonstrated by electron microscopy, and immunoglobulins and complement have been shown by immunofluorescence.**

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy

- Most patients have the primary, idiopathic form of membranous nephropathy. However, a number of associated conditions predispose to the development of "secondary" membranous nephropathy.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy

- In such cases, the immune complexes within the glomerular capillary walls may contain endogenous antigens (e.g., tumor antigens) or antigens derived from an infectious agent.
- In adults, one of the most frequent associations of membranous nephropathy is with carcinomas, being found in as many as 10% of these patients.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy

- Certain drugs, such as gold and penicillamine, used in the treatment of rheumatoid arthritis, can cause the lesion. Membranous nephropathy has been seen in association with various systemic infections, most frequently hepatitis B.

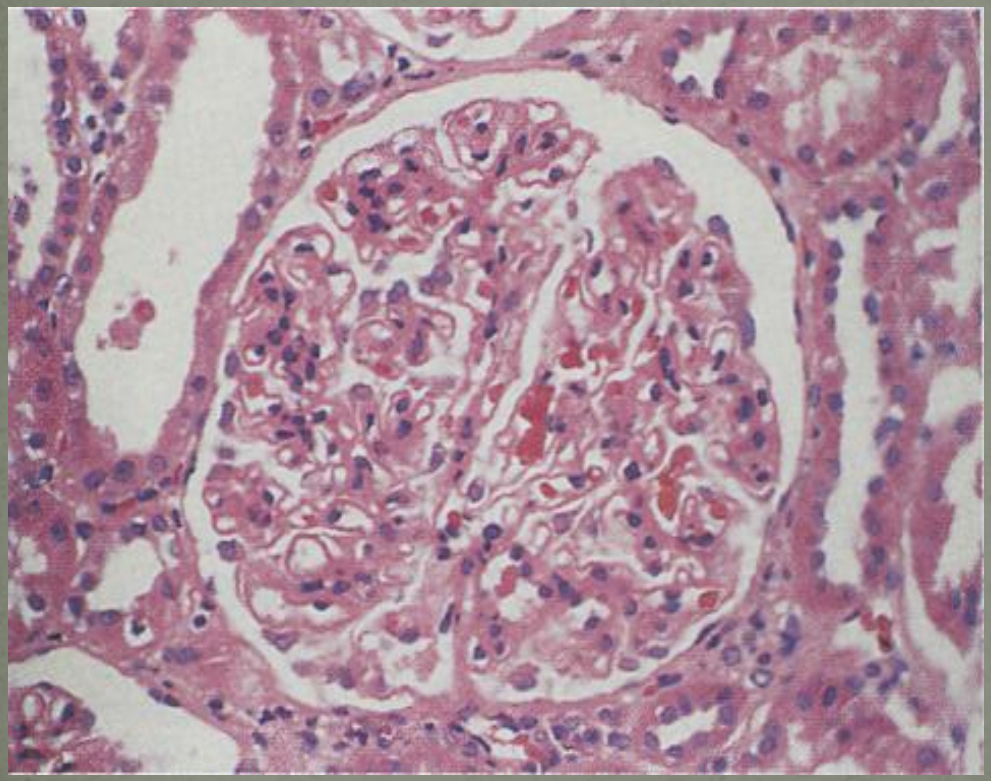
The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy

- As many as 10% of patients with systemic lupus erythematosus present with a lesion of membranous nephropathy. When these predisposing conditions are recognized, their treatment often results in eradication of the membranous lesion and clinical remission of the nephrotic syndrome.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy

- Pathology: By light microscopy, the glomeruli are slightly enlarged yet normocellular. Depending on the duration of the disease, the capillary walls are normal or thickened.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy



Membranous glomerulopathy. The glomerulus is slightly enlarged and shows diffuse thickening of the capillary walls. There is no hypercellulariry.

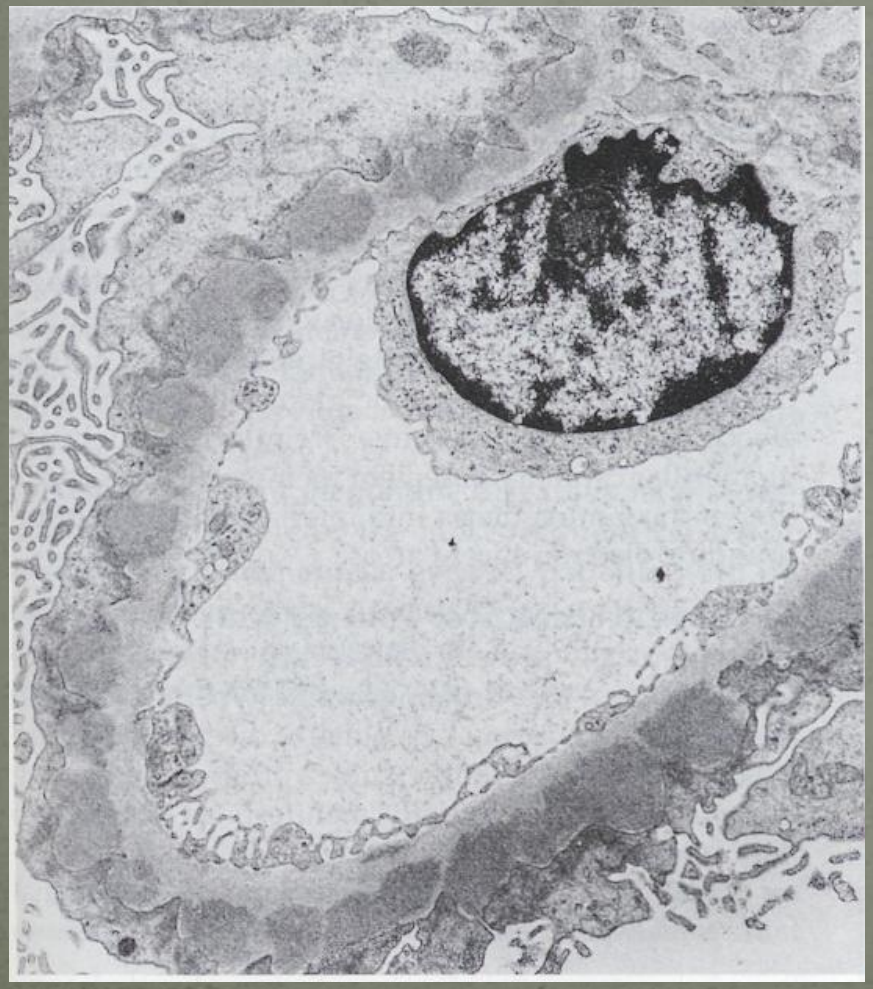
The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy

- During the early stages, silver stains, which demonstrate basement membrane material, reveal multiple projections, or spikes, of argyrophilic material on the epithelial surface of the basement membrane.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy

- Such spikes represent projections of basement membrane material that is deposited around the immune complexes which do not stain with silver. As the disease progresses, the capillary lumina are encroached on, and glomerular obsolescence eventually ensues.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy

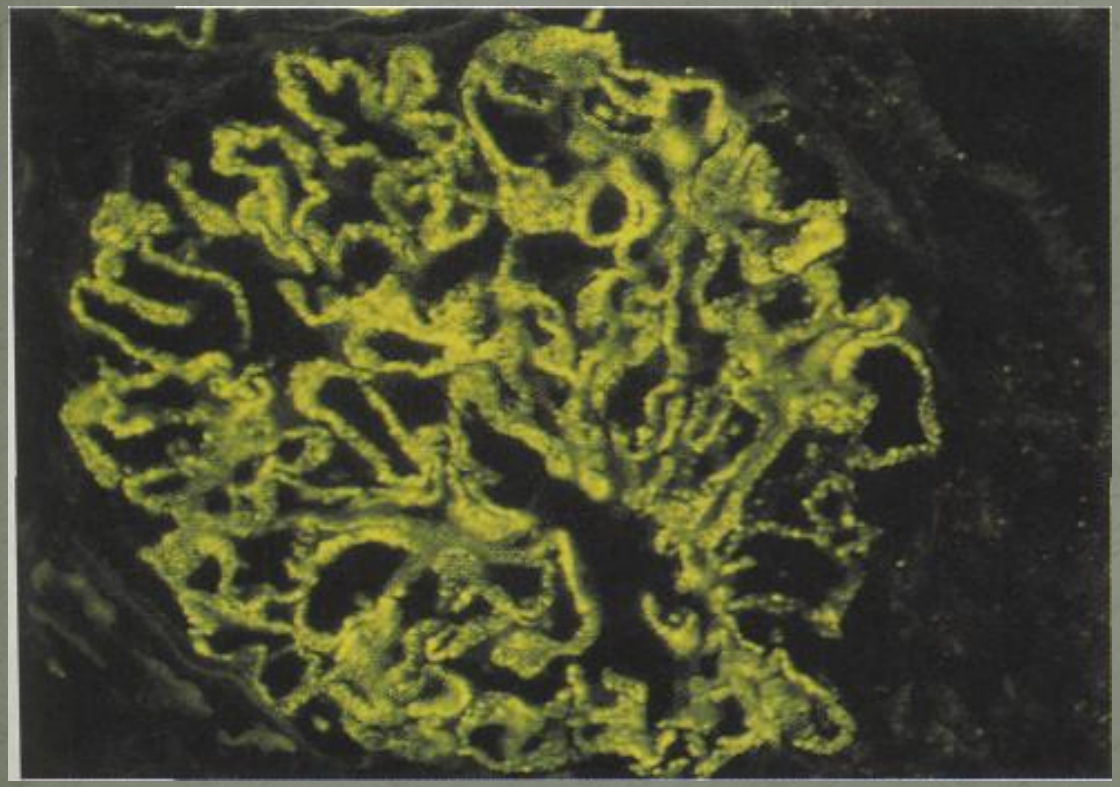


Stage II membranous glomerulopathy. An electron micrograph shows deposits of electron-dense material, with intervening delicate projections of basement membrane material.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy

- Immunofluorescence studies reveal diffuse, *granular* deposition of IgG and C3 in the glomerular capillary loops. Mesangial deposits are not typically seen in patients with the idiopathic variety of membranous nephropathy, but they are frequently seen in the membranous lesion of systemic lupus erythematosus.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy



Membranous glomerulopathy. Immunofluorescent microscopy shows granular deposits of immunoglobulin G outlining the glomerular capillary loops.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Membranosus Glomerulopathy

- Clinical Features: At one end of the clinical spectrum, patients have spontaneous remissions, whereas at the severe end, progressive renal failure ensues within 10 to 15 years. In the middle of these extremes, many patients have persistent proteinuria, with normal renal function, for many years.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Diabetic Glomerulosclerosis

- ***Diabetic glomerulosclerosis (Kimmelstiel-Wilson disease)*** *embraces the glomerular changes seen in patients with diabetes, which result in proteinuria and progressive renal failure.* Only the glomerular lesion of diabetes mellitus is discussed here; a more general discussion of diabetes.

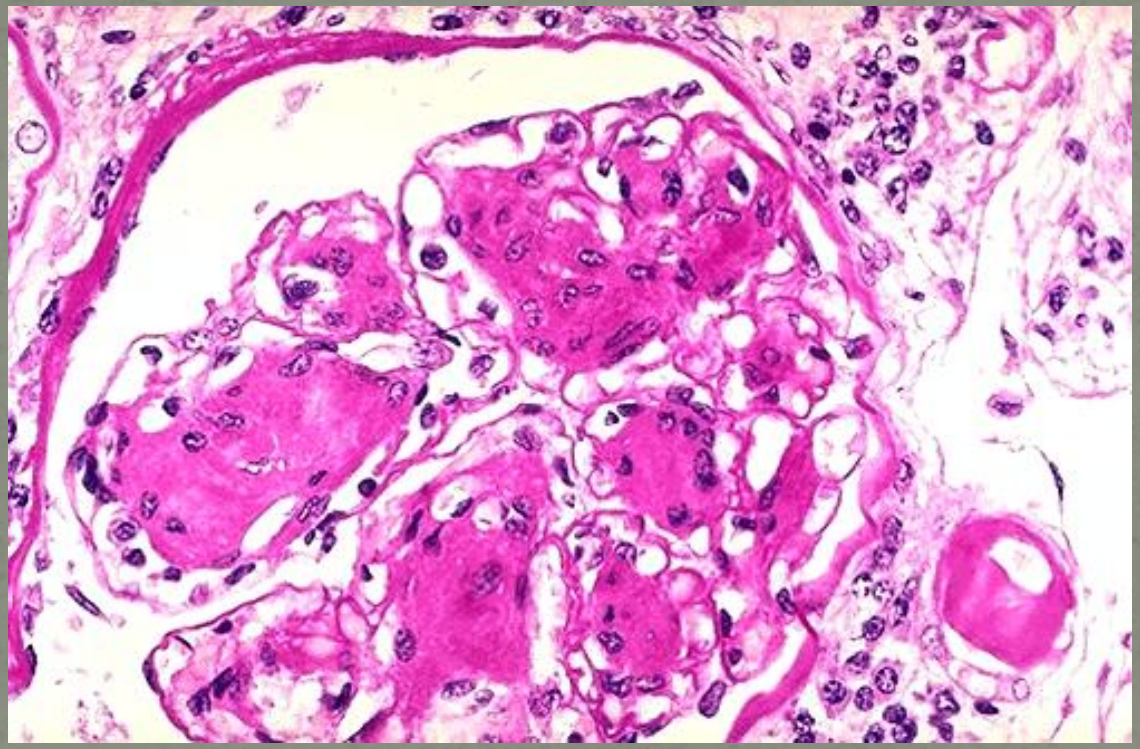
The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Diabetic Glomerulosclerosis

- Pathogenesis: The alterations in diabetic glomerulosclerosis are an expression of the diabetic microangiopathy that occurs in many systemic small arteries, arterioles, and capillaries.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Diabetic Glomerulosclerosis

- As in the systemic vessels, glomerulosclerosis results from progressive accumulation of basement membrane material, a process that, in turn, produces enlargement of the glomeruli. The pathogenesis appears to be related to the severity and duration of hyperglycemia.

Diabetic Glomerulosclerosis



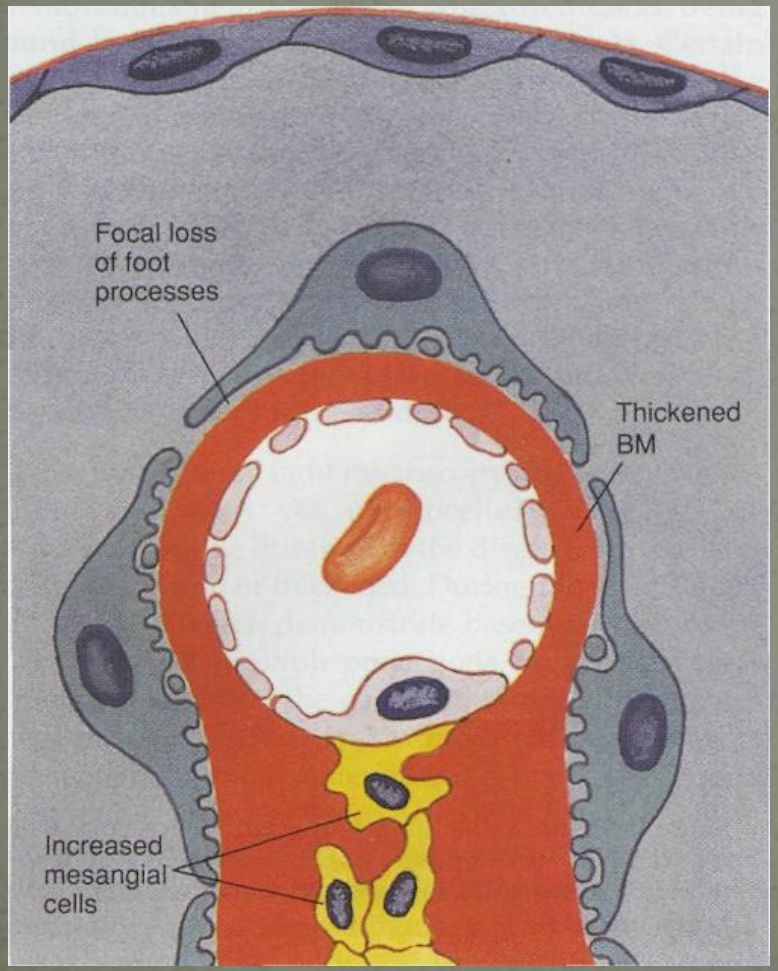
The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Diabetic Glomerulosclerosis

- The reasons for proteinuria in patients with diabetic glomerulopathy are not clear, but they may relate, in part, to nonenzymatic glycosylation of proteins making up the glomerular filtration barrier, including the basement membrane components. This chemical reaction leads to changes in the charge of these proteins and in the filtration properties of the glomerulus.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Diabetic Glomerulosclerosis

- **Pathology:** Early thickening of the glomerular basement membrane is followed by a diffuse widening of the mesangial areas, with the accumulation of a PAS-positive matrix. The glomerulus becomes enlarged and may appear hypercellular.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Diabetic Glomerulosclerosis



Diabetic glomerulosclerosis. The glomerular tuft is enlarged and displays a thickened basement membrane that retains a normal texture and density, with smooth inner and outer contours. Focal effacement of the epithelial cell foot processes is common. Accumulation of basement membrane-line stroma in the mesangium parallels diffuse widening of the basement membrane.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Diabetic Glomerulosclerosis

- Diffuse glomerulosclerosis refers to enlarged glomeruli with expanded mesangial areas and diffusely thickened basement membranes.
- Nodular glomerulosclerosis describes single or multiple nodules in the glomeruli. These are rounded, homogeneous, and eosinophilic masses in centrilobular areas With time, the nodules become acellular, in which case only a rim of peripheral mesangial nuclei is visible.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Diabetic Glomerulosclerosis

- Insudative changes occur in both the adjacent arterioles and the glomerular tufts. **These are manifested by hyaline arteriolosclerosis, which in patients with diabetes uniquely involves both the afferent and efferent arterioles.**

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Diabetic Glomerulosclerosis

- **Clinical Features:** Diabetic glomerulosclerosis is the leading cause of endstage renal disease in the United States, accounting for one-third of all patients with chronic renal failure. Proteinuria in patients with diabetic glomerulosclerosis is initially mild and may remain so, although patients with nephrotic-range proteinuria usually progress to renal failure within 6 years. Hematuria is usually not present.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Renal Amyloidosis

- ***Renal amyloidosis*** *refers to the deposition in the kidneys of diverse extracellular proteins that have common morphologic properties, stain with specific dyes, and have a characteristic appearance under polarized light when stained with certain dyes.* Amyloidosis is associated with many different diseases, and the protein material (amyloid) may be deposited in a variety of tissues.

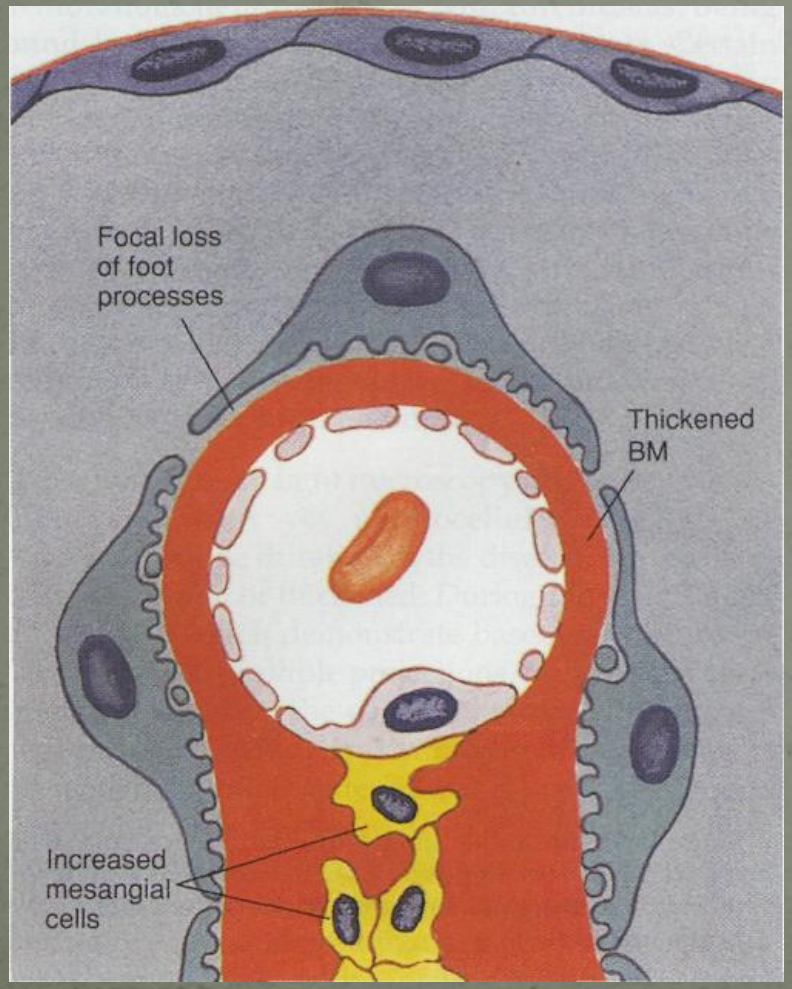
The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Renal Amyloidosis

- Glomerular involvement is a prominent feature in most cases of systemic amyloidosis. In 60% of the patients, proteinuria is severe enough to produce nephrotic syndrome. Severe infiltration of the glomeruli and blood vessels by amyloid results in renal failure.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Renal Amyloidosis

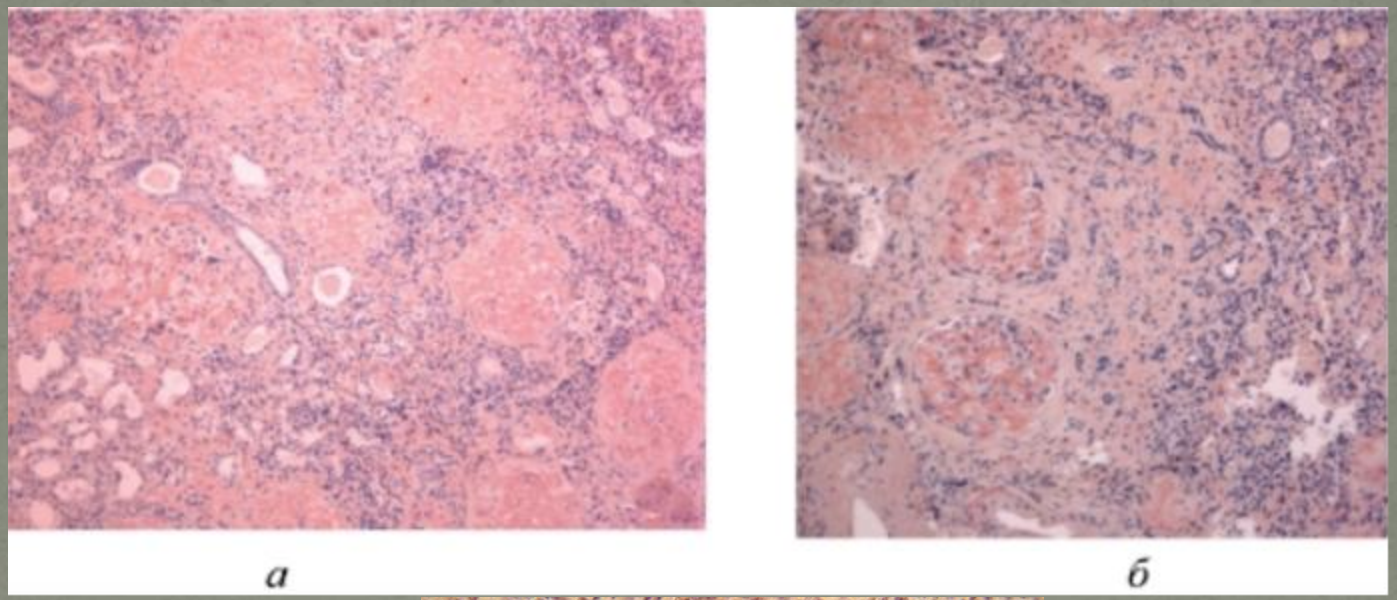
- Pathology: Amyloid deposition initially tends to be mesangial, thereby producing diffuse mesangial widening without hypercellularity. However, it progressively spreads to obliterate capillary lumina.

The Kidney
NONINFLAMMATORY GLOMERULOPATHIES
Renal Amyloidosis



The glomerular tuft is enlarged and displays a thickened basement membrane. Accumulation of amyloid in the mesangium and vascular wall transforms the glomerulus into rounded and nonfunctional structure.

Renal Amyloidosis



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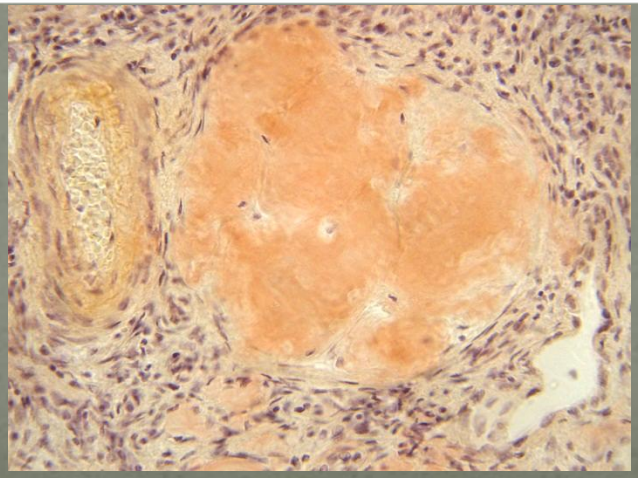


The Kidney
Glomerulonephritis

- *Glomerulonephritis refers to inflammatory lesions of the glomerulus that are characterized histologically by hypercellularity of the glomeruli and clinically by nephritic syndrome*. Occasionally, proteinuria may predominate.

The Kidney
Glomerulonephritis

Pathogenetic Mechanisms

- Because of their importance in both experimental and human glomerular disease, immunologically mediated processes have been studied extensively. Immunologic injury can be divided into four basic
- types:
- Trapping of circulating immune complexes
- In situ immune complex formation
- Activation of the alternative pathway of complement
- Cell-mediated processes

The Kidney
Glomerulonephritis

Circulating Immune Complex Nephritis

- Circulating immune complex nephritis results from the glomerular trapping of circulating antigen/antibody complexes. In such disorders, the glomerulus can be considered an innocent bystander, involved only because of filtration by glomerular capillaries.

The Kidney
Glomerulonephritis

- Aggregates of immune complexes may penetrate the glomerular basement membrane and are then trapped in a subepithelial location. Their presence is confirmed on electron microscopy by the presence of subepithelial "humps." With immunofluorescence, a peripheral and granular staining with antisera directed against IgG and C3 is noted.

The Kidney
Glomerulonephritis

- Alternatively, circulating immune complexes usually do not penetrate the glomerular basement membrane and often localize in the subendothelial zone or mesangium.

The Kidney
Glomerulonephritis

- Antigens in clinically important forms of circulating immune complex nephritis may be either exogenous or endogenous. Examples of immune complex nephritis induced by exogenous antigens include bacterial antigens in glomerulonephritis associated with streptococcal infections and bacterial endocarditis and viral antigens in glomerulonephritis induced by hepatitis B.

The Kidney
Glomerulonephritis
In Situ Immune Complex Formation

- The glomerulus may be damaged by the binding in situ of circulating antibody to an antigen already deposited in the glomerular basement membrane. The best-known example of this is Goodpasture syndrome, in which a subunit of the globular domain of type IV collagen in the glomerular basement membrane acts as an endogenous antigen to which circulating antibodies bind.

The Kidney
Glomerulonephritis

- Immunofluorescence shows a linear localization of IgG along the basement membrane. The combination of antigen with antibody results in activation of complement, and a rapidly progressive glomerulonephritis usually ensues.

The Kidney
Glomerulonephritis
Alternative Complement Pathway

- The alternative complement pathway is important in the pathogenesis of a form of membranoproliferative glomerulonephritis. The alternative pathway for complement activation is also thought to be important in focal glomerulonephritis caused by deposition of IgA.

The Kidney
Glomerulonephritis

Cell-Mediated Immunity

- Although there is no direct evidence that cell-mediated processes cause any specific form of human glomerulonephritis, occasional cases of immunologically mediated disease occur in which the accumulation of mononuclear cells and the absence of immunoglobulins suggest a delayed-type (cell-mediated) reaction.

The Kidney
Glomerulonephritis

- Once immune complexes have localized within a glomerulus, a number of secondary pathogenetic mechanisms appear to play a role in effecting immunologic injury.

The Kidney
Glomerulonephritis

- **Complement** may produce glomerular injury through two distinct mechanisms. The terminal components of complement (C5b-9) form the membrane-attack complex that directly produces cell injury at the level of the plasma membrane.

The Kidney
Glomerulonephritis

- Neutrophils are attracted to the glomerulus in exudative forms of glomerulonephritis, such as in acute postinfectious glomerulonephritis and anti-GBM antibody disease. The chemotactic actions of specific complement components, particularly C5a, are responsible.

The Kidney
Glomerulonephritis

- Monocytes and macrophages infiltrate the glomerulus in many forms of renal disease and contribute to the cellularity of glomerular tufts and the surrounding crescents. Activated macrophages release a number of cytokines and growth factors that are probably important in tissue damage.

The Kidney
Glomerulonephritis

- Activation of the coagulation cascade is important in some forms of glomerular injury. Fibrin can usually be demonstrated in Bowman's space early during the formation of crescents and is probably responsible, in part, for the subsequent epithelial cell proliferation and crescent formation.

The Kidney
Glomerulonephritis

- Platelets may be activated by sensitized mast cells and basophils on exposure to antigen or immune complexes. Growing evidence suggests that platelet-derived, polycationic proteins disturb the anionic charge of the glomerular wall and contribute to its damage.

The Kidney
Acute Postinfectious Glomerulonephritis

- ***Acute postinfectious glomerulonephritis*** *is characterized clinically by the sudden onset of nephritic syndrome and morphologically by diffuse hypercellularity of glomeruli.* The disease is a sequel to infection with a variety of agents (e.g., staphylococci, pneumococci, spirochetes, and viruses), but the most frequent association is with certain strains of **group A**
- **β-hemolytic streptococci *(S. pyogenes)*.**

The Kidney
Acute Postinfectious Glomerulonephritis

- Acute glomerulonephritis most commonly affects children, and although it is not seen as frequently in developed countries now as in the past, it remains one of the most common renal diseases of childhood.

The Kidney
Acute Postinfectious Glomerulonephritis

- **Pathogenesis:** The exact mechanism by which streptococcal infection produces the characteristic proliferative changes in the glomeruli is still not completely characterized, although the similarities with the experimental model of acute serum sickness suggest that the disease is caused by glomerular localization of immune complexes generated by an antibody response to circulating antigens.

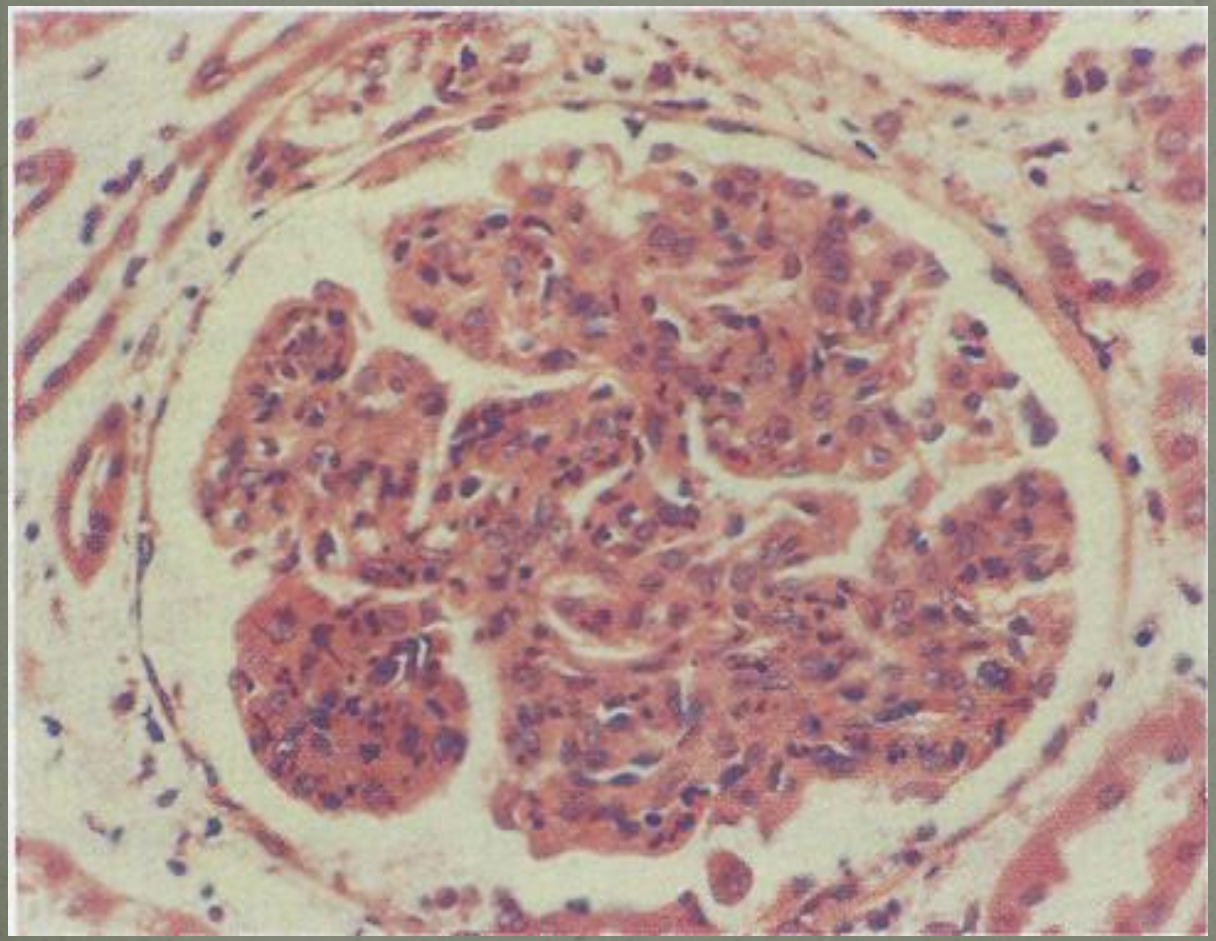
The Kidney
Acute Postinfectious Glomerulonephritis

- Circulating immune complexes are demonstrable in half of the patients with acute poststreptococcal glomerulonephritis. Evidence also suggests the formation of glomerular immune complexes in situ, between trapped bacterial antigens and circulating antibodies.

The Kidney
Acute Postinfectious Glomerulonephritis

- Pathology: By light microscopy, diffuse enlargement and hypercellularity of the glomeruli are present during the first 3 weeks after the onset of acute glomerulonephritis.

The Kidney
Acute Postinfectious Glomerulonephritis

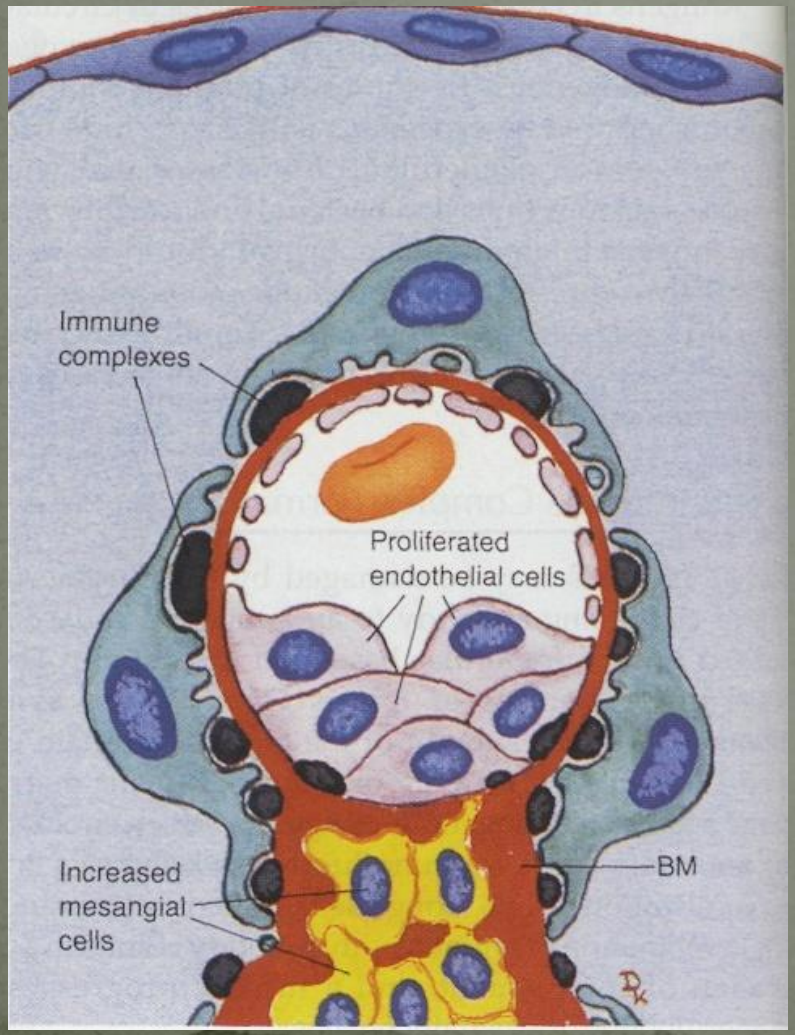


Acute poststreptococcal glomerulonephritis. The glomerulus of a patient who developed glomerulonephritis after a streptococcal infection is hypercellular because of the proliferation of endothelial and mesangial cells and infiltration by neutrophils.

The Kidney
Acute Postinfectious Glomerulonephritis

- The hypercellularity is caused by the proliferation of both endothelial and mesangial cells and the infiltration of neu-trophils and monocytes. Crescents may be present but are usually sporadic and segmental. Tubulointerstitial damage and inflammation occur in parallel with the glomerular changes.

The Kidney
Acute Postinfectious Glomerulonephritis



Postinfectious glomerulonephritis. Trapping of immune complexes in a subepithelial pattern ("lumpy-bumpy") is seen together with focal effacement of the foot processes. Less prominent subendothelial immune complexes are associated with endothelial cell proliferation and are related to increased capillary permeability and narrowing of the lumen. Frequently, proliferation of mesangial cells and a thickened mesangial basement membrane result in widening of the stalk and conspicuous trapping of immune complexes.

The Kidney
Acute Postinfectious Glomerulonephritis

- The characteristic ultrastructural features of acute postinfectious glomerulonephritis are subepithelial humps, and these deposits are invariably accompanied by mesangial and subendothelial deposits. The humps are variably sized, dome-shaped deposits that are situated on the epithelial aspect of the basement membrane.

The Kidney
Acute Postinfectious Glomerulonephritis

- The characteristic morphologic features of acute inflammation usually resolve by 8 weeks after the onset of nephritis. Generally, those patients who recover completely do so both clinically and histologically by 3 years after onset.

The Kidney
Acute Postinfectious Glomerulonephritis

- **Clinical Features:** The primary infections may be in the pharynx or, especially in hot and humid environments, the skin. The nephritic syndrome typically begins abruptly, with oliguria, hematuria, facial edema, and hypertension. Usually, there is a depression of the serum C3 level during the acute syndrome.

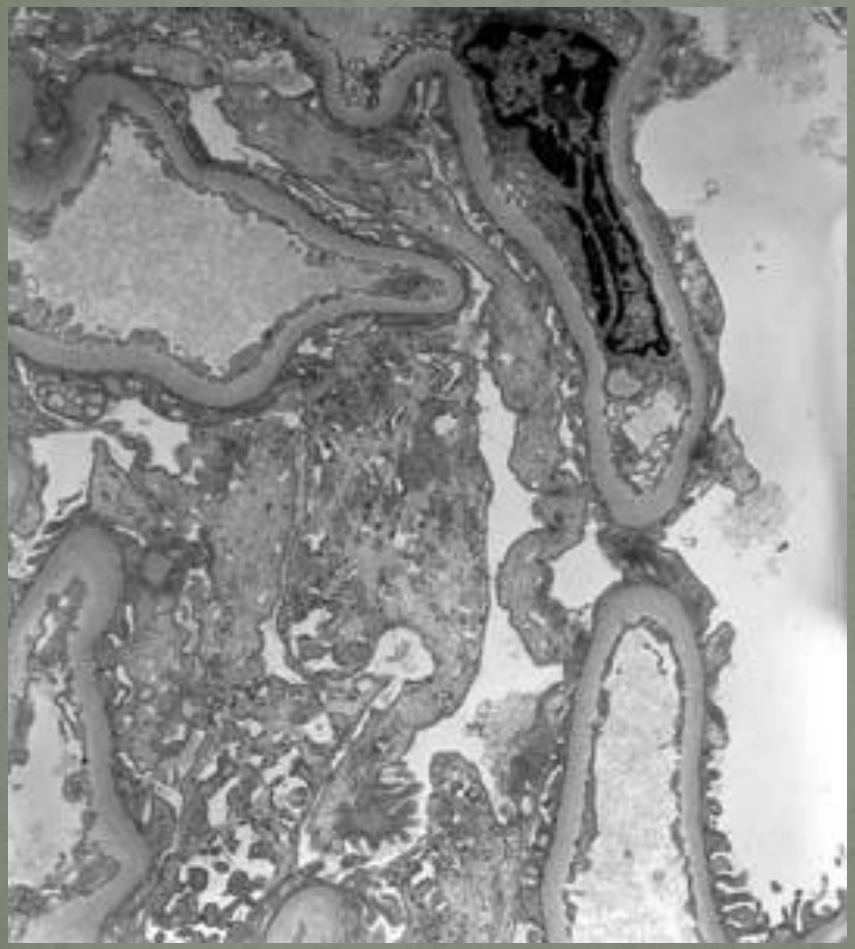
The Kidney
Acute Postinfectious Glomerulonephritis

- This returns to normal within 1 to 2 weeks, however, as does the clinical condition of most patients. However, in a minority of patients, an abnormal urinary sediment persists for years after the acute episode.

The Kidney
Acute Postinfectious Glomerulonephritis

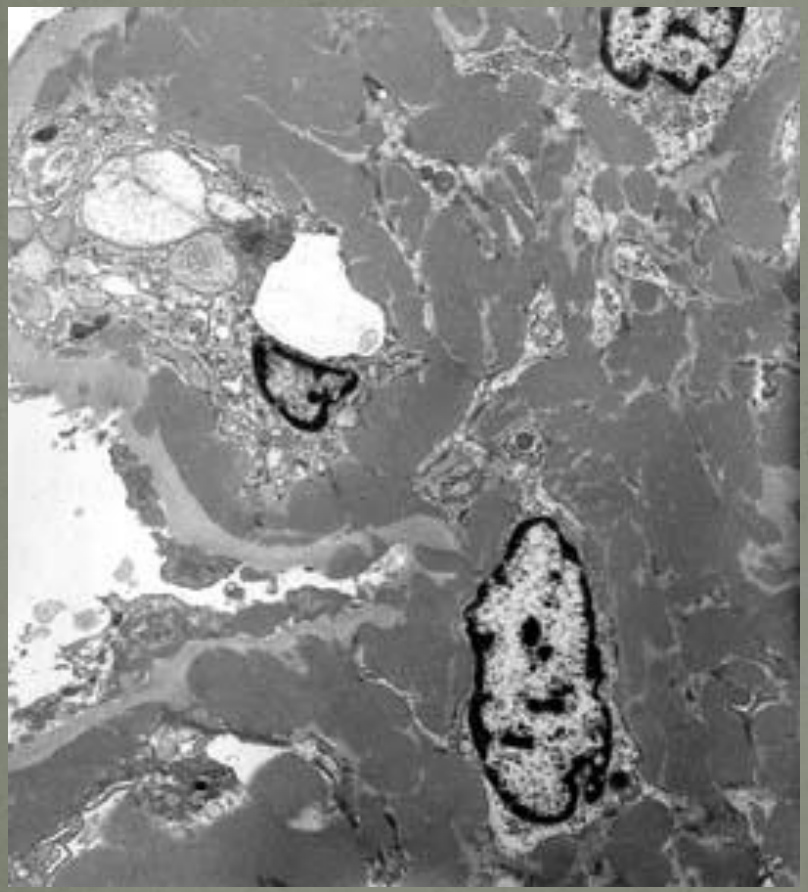
- Complete recovery, particularly in children, is the rule, although some adults with initially severe disease manifest persistent renal dysfunction.

Anti-GBM antibody disease (crescentic glomerulonephritis)



- Anti-GBM antibody disease represents 2% to 30% of cases of diffuse extracapillary proliferative (crescentic) glomerulonephritis. When accompanied by alveolar hemorrhage and hemoptysis, it is termed "Goodpasture's syndrome." Immune-type electron dense deposits are not found here, as is the case with anti-GBM disease.

Diffuse proliferative glomerulonephritis with immune complex deposition



- Diffuse proliferative glomerulonephritis with immune complex deposition is often associated with nephritic syndrome and is associated with a variety of disorders, including infection, SLE, IgA nephropathy, and others. By electron microscopy, endothelial and mesangial cells are swollen with closure of glomerular capillary loops. Here we see numerous electron dense immune-type mesangial deposits. Deposits in this distribution may be characteristic of the condition when associated with SLE or IgA nephropathy.

Membranoproliferative Glomerulonephritis

Age: Can affect any age.

Causes: Idiopathic or Secondary (Viral infection e.g. hepatitis, paraneoplastic syndrome, SLE and drugs).

Clinical picture: : Nephrotic syndrome and in some cases nephritis.

Microscopy:

- **Light microscopy:** Glomerli: Diffuse thickening of the capillary basement membranes and increased mesangial matrix and cellularity resulting in prominent mesangial lobulation.

  Tubules and interstitium: Variable degrees of chronic changes.

- **Immunofluorescence** Mesangial and capillary deposits of IgM, IgG and C3.

- **Electron microscopy** There are 3 types according to the cause of thickening of basement membrane.

  1. **Typel:** Subendothelial immune complex deposits and mesangial interposition between the basement membrane and endothelial cells.
  2. **Type II:** Dense deposits disease. There is replacement of the lamina densa by electron dense material. Its nature is not exactly known.
  3. **Type III:** Subendothelial and subepithelial deposits.

Prognosis: Progression to renal failure.

Proliferative glomerulonephritis

Age: Any age may be affected.

Causes: Idiopathic and secondary (viral, bacterial (post streptococcal), , subacute bacterial endocarditis, SLE, vasculitis).

Clinical picture: Nephrotic syndrome or nephritis.

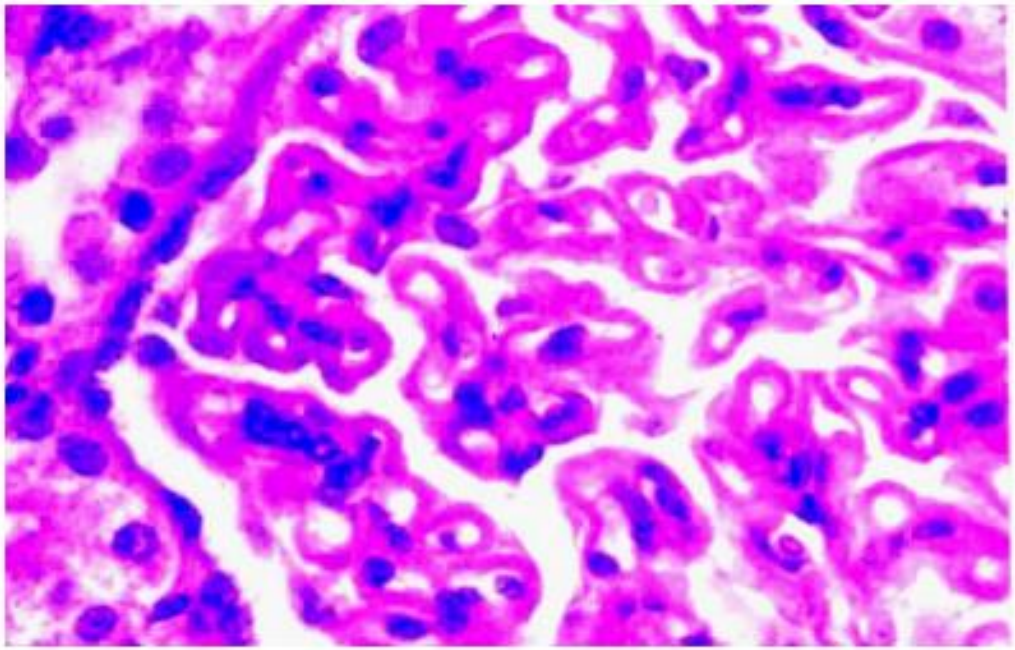
Microscopy:

- **Light microscopy:** Glomeruli: Mesangial proliferative focal or diffuse.

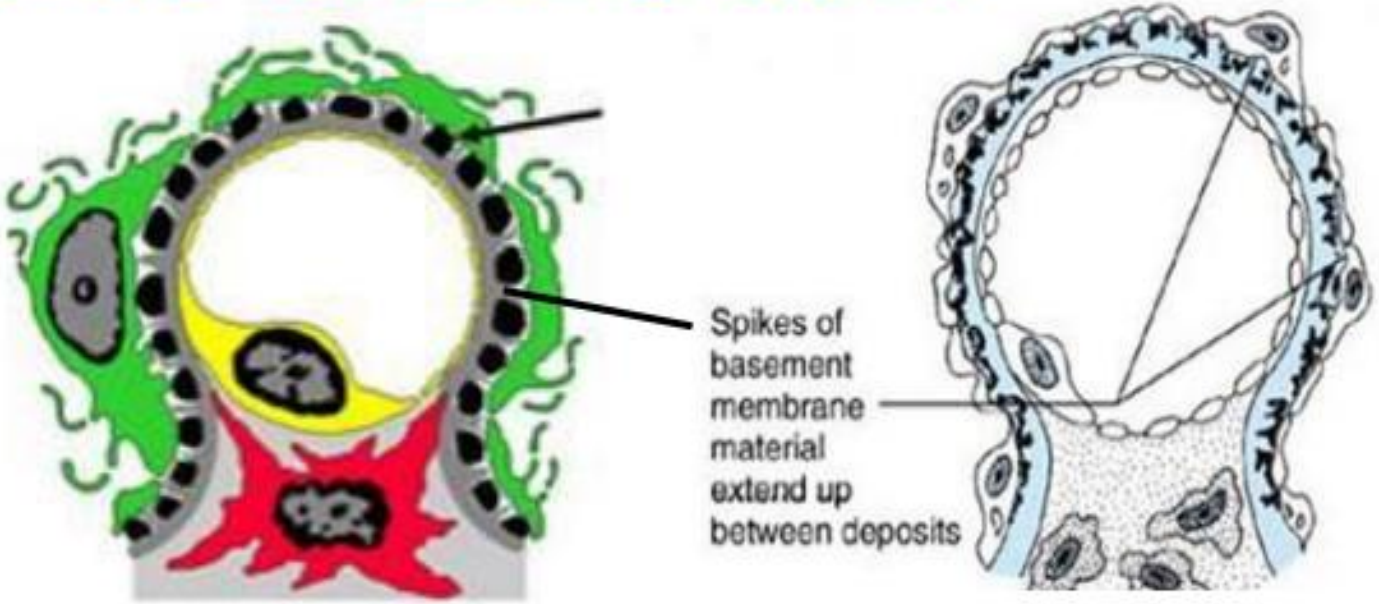
Tubules and Interstitium: Variable degrees of tubular atrophy and interstitial fibrosis.

- **Immunofluorescence:** Variable + ve or -ve.
- **Electron microscopy:** Mesangial proliferation focal or diffuse.

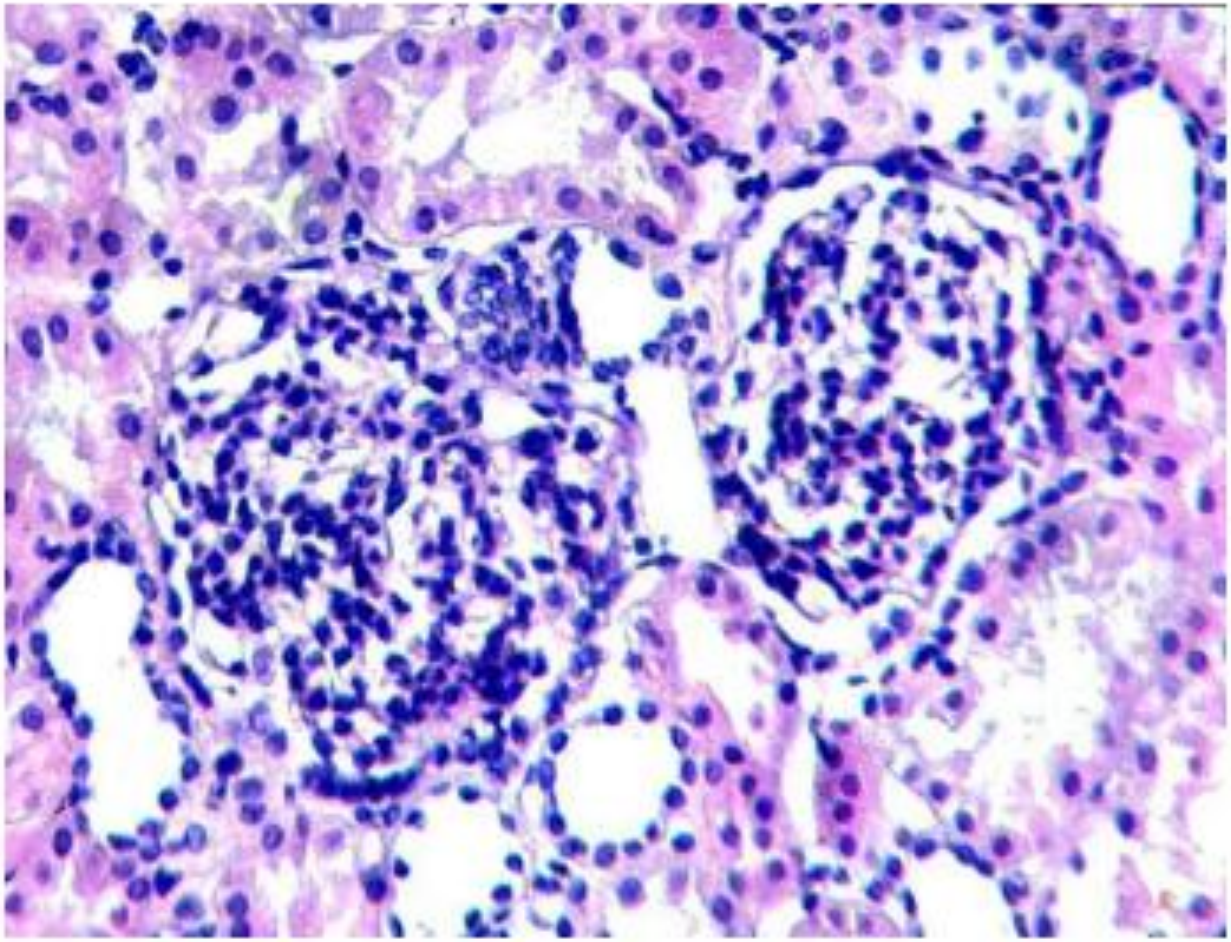
Prognosis: Recovery in some mild cases: Others may progress to renal failure.



Light microscopy. Diffuse hyaline thickening of basement membranes. No associated hypercellularity



Diffuse mesangial proliferation



Crescentic Glomerulonephritis

Age: Any age may be affected.

Causes: Idiopathic and secondary (Post streptococcal, Good Pasture's syndrome and vasculitis).

Clinical picture: Severe form of nephritis, acute renal failure, or nephritic syndrome.

Microscopy:

- **Light microscopy:**

Glomeruli: Epithelial crescents result from: Proliferation of parietal epithelial cells lining the Bowman's capsule, fibroblasts, histocytes and cells of unknown origin.

Tubules and Interstitium: Variable degrees of tubular atrophy and interstitial fibrosis.

- **Immunofluorescence:** Varies according to the etiology:

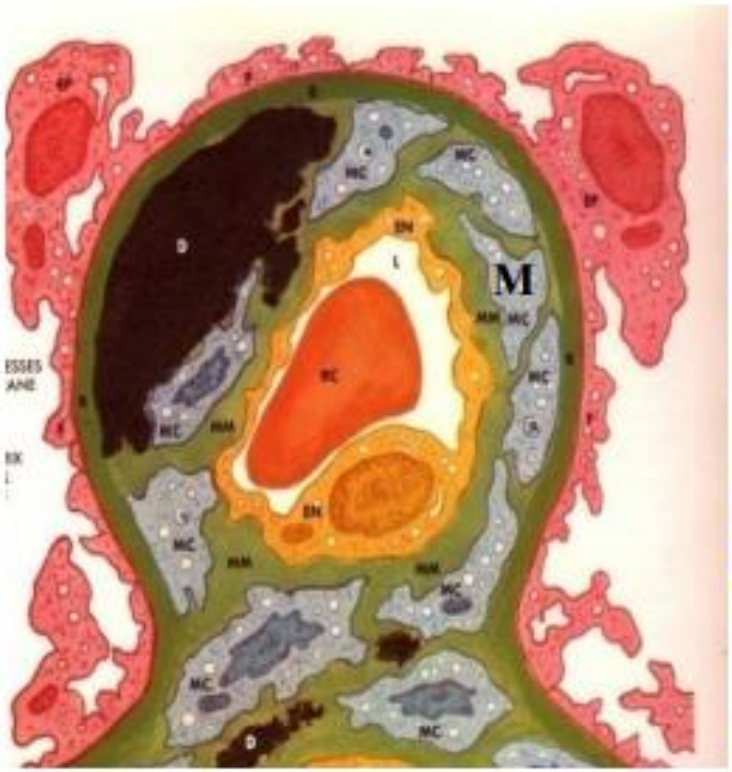
1- Granular Ig deposits if the cause is immune complex.

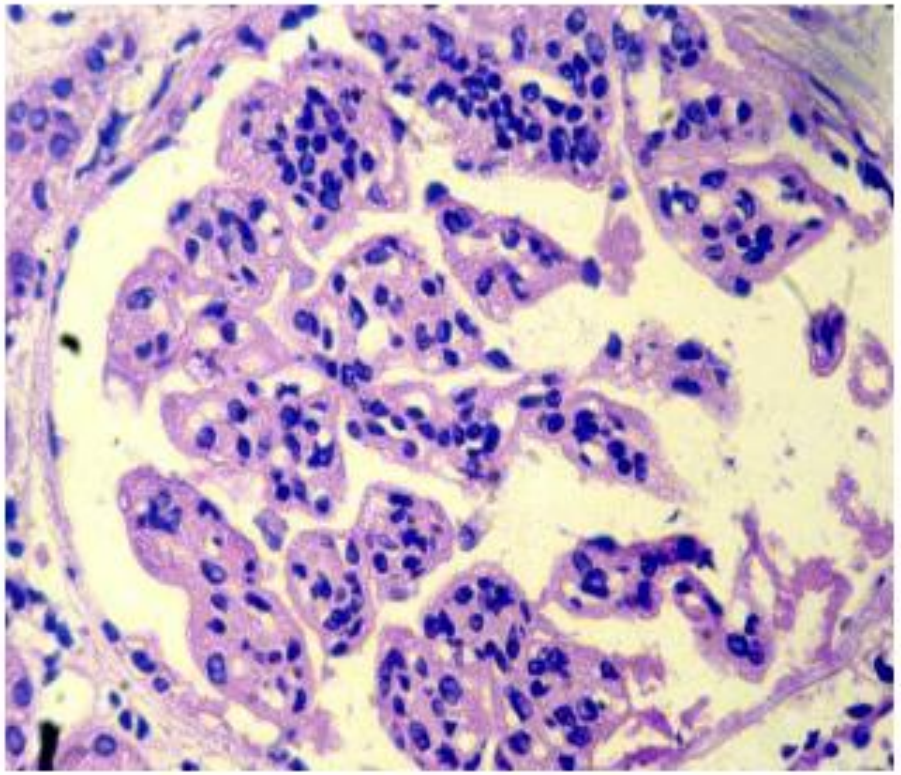
2- Negative if the cause is non immunologic

3- Linear deposits of IgG along the basement membranes in cases of Good Pasture's syndrome.

- **Electron microscopy:** Fusion of foot processes, crescents and immune complex deposits

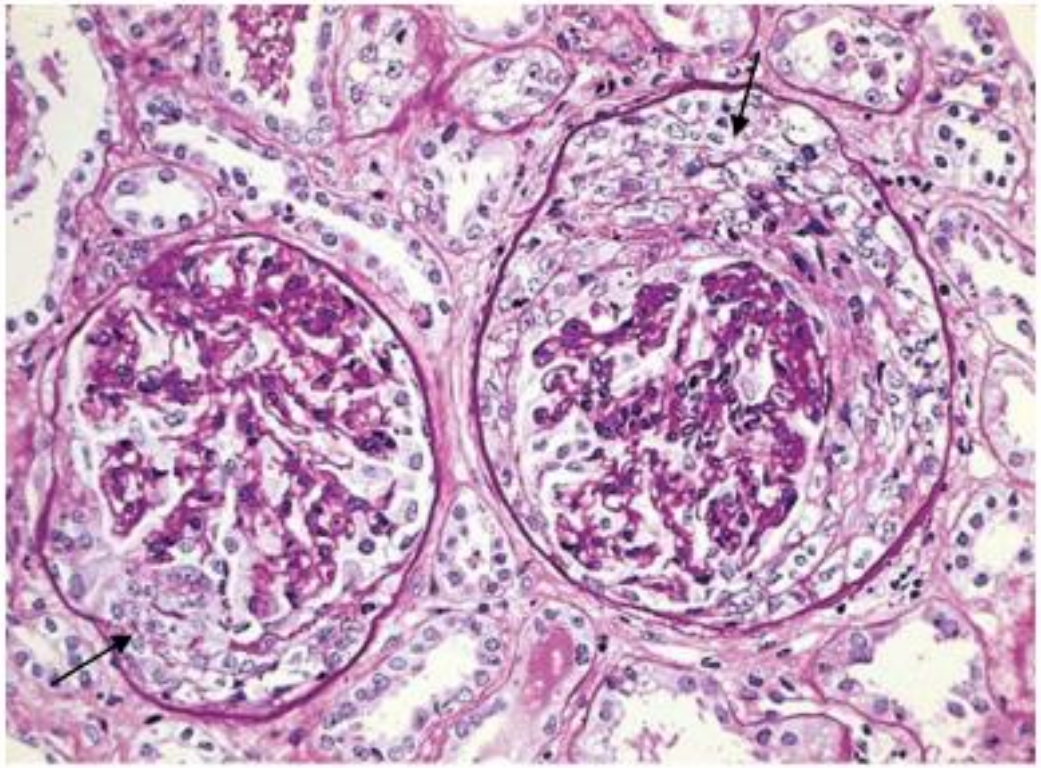
Prognosis: Rapid progression to chronic renal failure.





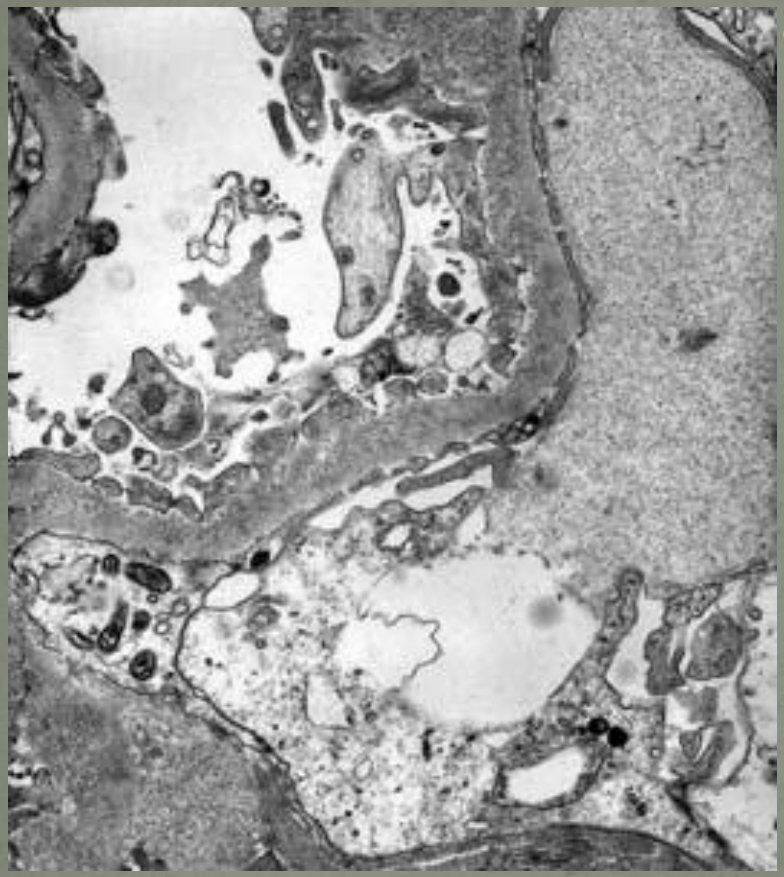
MPGN type :Thickening of basement membrane is due to subendothelial deposits (**D**) and mesangial interposition (**M**).

Membranoproliferative glom. nephritis (MPGN): Thickening of the capillary basement membranes and increased mesangial matrix and cellularity resulting in prominent mesangial lobulation.



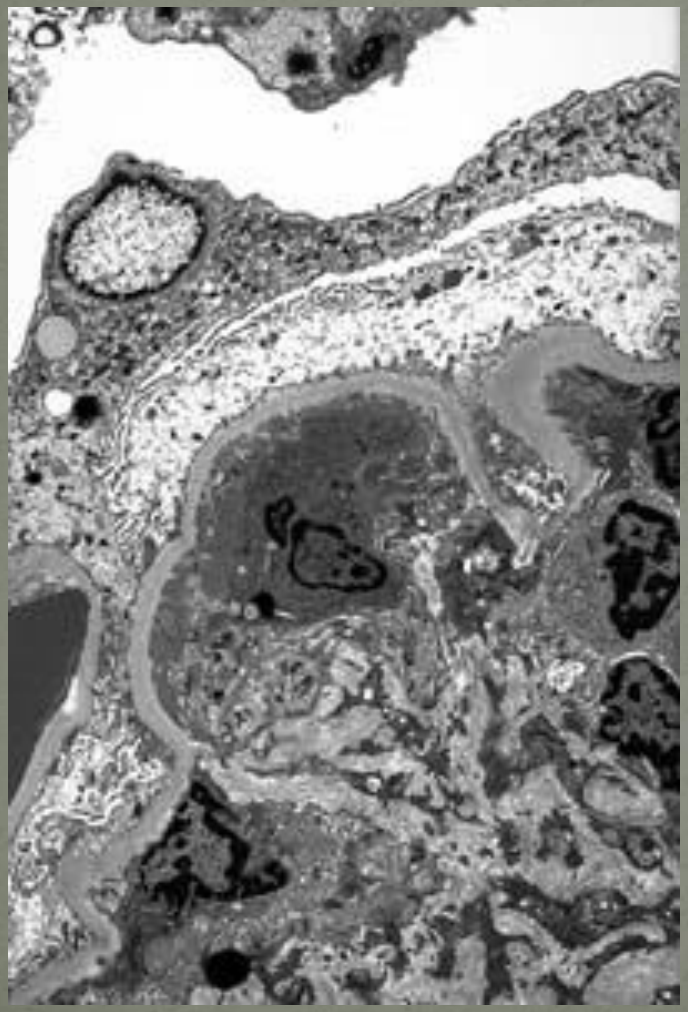
Crescentic GN: Epithelial crescents (arrows) result from: Proliferation of parietal epithelial cells lining the Bowman's capsule, fibroblasts, histocytes and cells of unknown origin.

Fibrillary glomerulonephritis



- Fibrillary glomerulopathies include fibrillary glomerulonephritis and immunotactoid glomerulopathy (IT), which are characterized by the presence of fibrillar or microtubular deposits on ultrastructural examination, respectively.

Fibrillary glomerulonephritis



- In addition to glomerular basement membrane thickening, the mesangial matrix is always expanded.

Chronic Glomerulonephritis

Gross appearance

- Size: The kidney becomes smaller.
- Consistency: firm.
- Capsule: Adherent with decortications (It strips off with portions of the cortex).
- Outer surface: It is finely granular.
- Cut surface:
  - Cortex and medulla: Narrow with no differentiation between the two.
  - Calyces and pelvis: They are not affected.
  - Large vessels: They show evidence of hypertension i.e. thick and gaping

Microscopic picture

- **Glomeruli:** All or most of them are totally sclerotic. Few show the original glomerulopathy. Some show compensatory hypertrophy.
- **Tubules and Interstitium:** Tubular atrophy, interstitial fibrosis and chronic inflammatory cellular infiltrate.
- **Blood vessels:** Hypertensive changes.

Pyelonephritis

Definition: It is a suppurative bacterial infection of renal pelvis, calyces and interstitium.

Microorganisms: Mostly E-coli or mixed with other bacteria.

Predisposing factors:

1- Obstruction either incomplete gradual or complete intermittent. Obstruction causes: Stagnation of urine which encourages bacterial proliferation, It decreases vitality of the tissues and leads to upward spread of lower UT infection.

2- Structural abnormalities of urinary tract.

3- Diabetes mellitus.

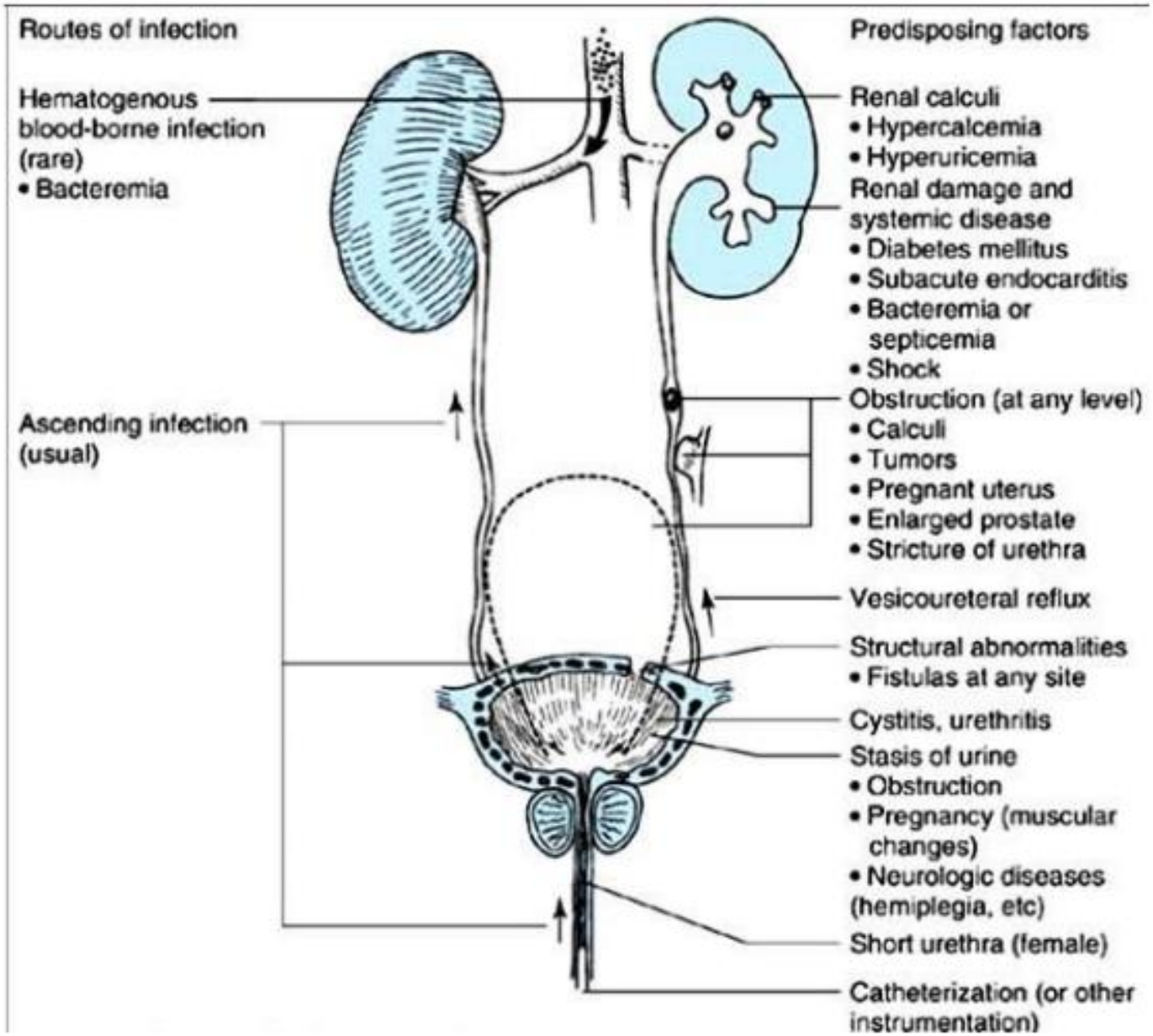
4- Instrumentation.

5- Sex: Females are more affected than males. That is because: Females have short urethra.

Effect of pregnancy: It leads to ureteric dilatation and urinary stasis due to: a- Mechanical pressure of enlarged uterus leading to obstruction b- relaxing effect of hormones on the smooth muscles.

Chronic Glomerulonephritis

Outer surface: It is finely granular



Causes of urinary tract obstruction

Mode of infection:

1- Ascending following cystitis: via lumen (in cases of vesicoureteric reflux subepithelially or periureteric lymphatics.

2- Haematogenous.

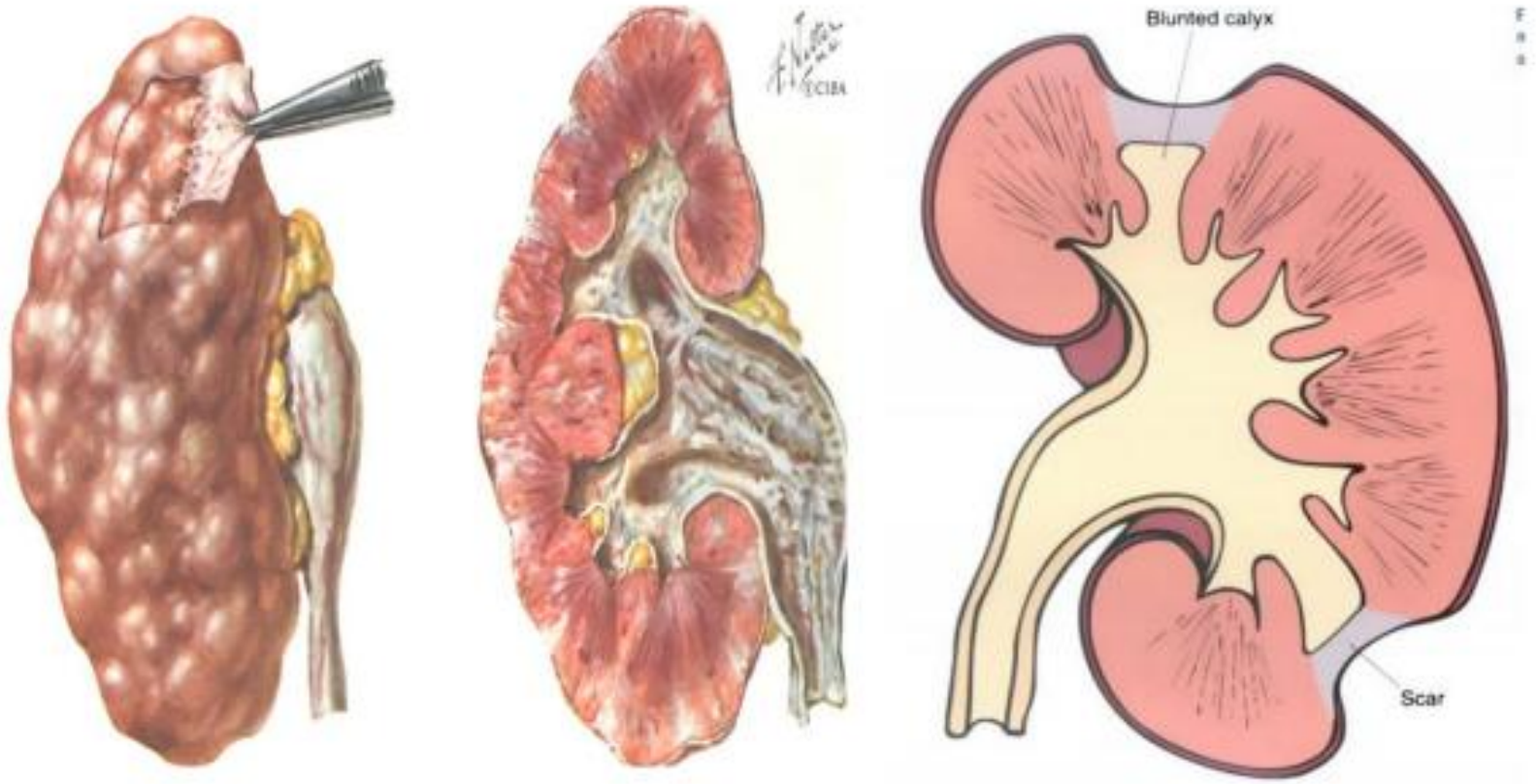
3- From the neighboring organs through the communicating lymphatics.

N.B. *the presence of pus cells in urine (pyuria) can result from infection any where in the urinary tract. However, the presence of neutrophils casts indicates Pyelonephritis. (Casts = tubular origin).

Pyelonephritis is either acute or chronic.

N/E of Pyelonephritis

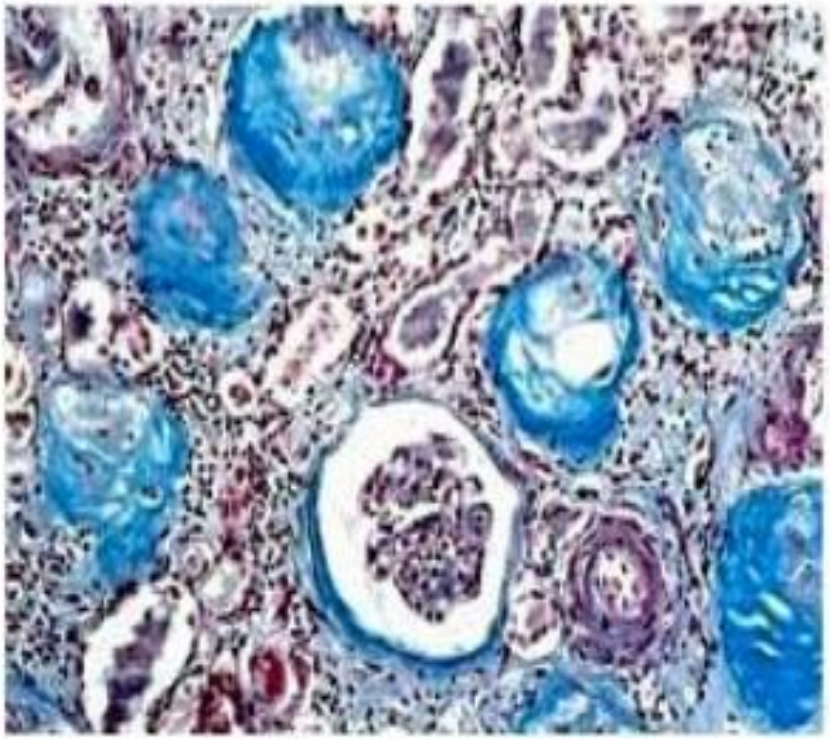
	Acute Pyelonephritis	Chronic pyelonephritis
Size	Increased	Decreased
Capsule	Strips easily	Adherent with decortication
Outer	Suppurative patchy foci surrounded by hyperemia.	Irregular coarsely granular (due to underlying coarse irregular scarring).
Cut surface - Cortex and medulla	Radial lines radiating from tips of papillae pelvis to cortex	Narrow cortex and medulla, opaque scarred areas.
- Calyces and pelvis • out lines • Mucosa • Contents	 Distorted. Hyperemic Full of pus	 Deformed by scarring Rough thick and mildly hyperemic Some pus



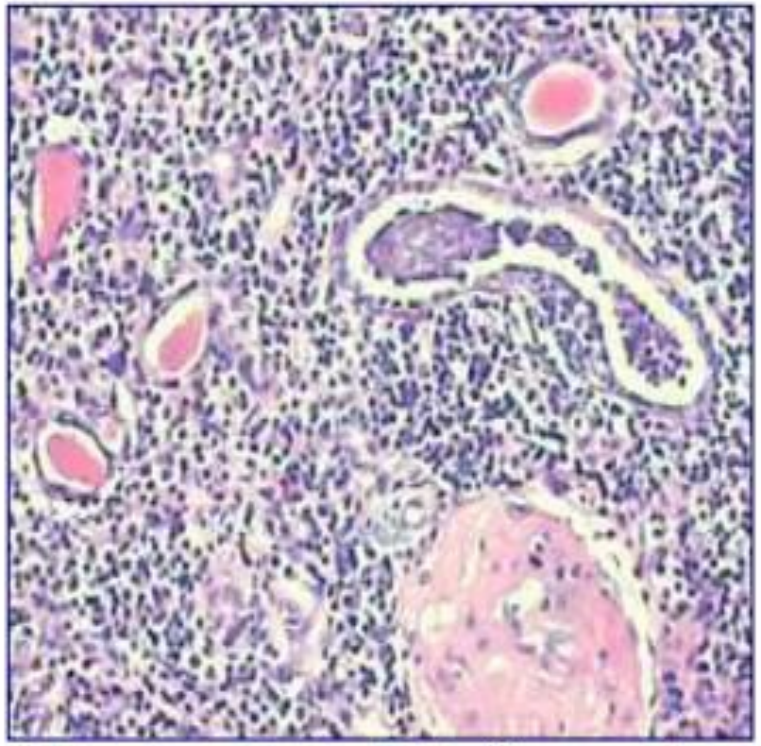
Chronic pyelonephritis . Outer surface is Irregular coarsely granular (due to underlying coarse irregular scarring).

M/E of pyelonephritis

	Acute pyelonephritis	Chronic pyelonephritis
Glomeruli	--------	Periglomerular fibrosis. Some totally sclerotic glomeruli.
Tubules	Neutrophil casts.	Groups of atrophic tubules (thyriodization i.e. dilated and lined by flattened epithelium and contain hyaline casts picture simulating thyroid follicles) Some tubules contain neutrphil casts.
Intertitium	Focal infiltration with acute inflammatory cells.	Focal fibrosis and chronic suppurative inflammation.
Blood vessels	----------	Hypertensive changes



Chronic pyelonephritis:
Periglomerular fibrosis. Other glomeruli are totally sclerotic .



Chronic pyelonephritis: Groups of atrophic tubules (thyriodization). Some tubules contain neutrphil casts. Interstitium is densely infiltrated by inflammatory cells.

The Kidney
Congenital anomalies

- **Renal Agenesis.** **Renal agenesis is the complete absence of renal tissue**. Clearly, the absence of both kidneys is not compatible with life. The majority of infants born with this anomaly are stillborn, and in most cases, the mother suffers from oligohydramnios (insufficient amniotic fluid).

The Kidney
Congenital anomalies

- Bilateral renal agenesis is often associated with other congenital anomalies, including low-set ears, receding chin, beak-like nose, and pulmonary hypoplasia. Congenital anomalies of the genitalia are also common, as are lower-limb anomalies. The pattern of malformations associated with oligohydramnios is known as **Potter syndrome.**

The Kidney
Congenital anomalies

- Unilateral renal agenesis is not a serious matter if there are no associated anomalies, because the single kidney undergoes sufficient hypertrophy to maintain normal renal function.

The Kidney
Congenital anomalies

- **Horseshoe Kidney.** Horseshoe kidney is a single, large, and midline organ that results from failure of the renal anlage to divide. The infant is born with fusion of the two kidneys, usually at the lower poles.

The Kidney
Congenital anomalies

- **Ectopic Kidney. Renal ectopia is an abnormal location of the kidney, usually in the pelvis.** Most commonly, this condition results from failure of the fetal kidney to migrate from the pelvis to the flank.

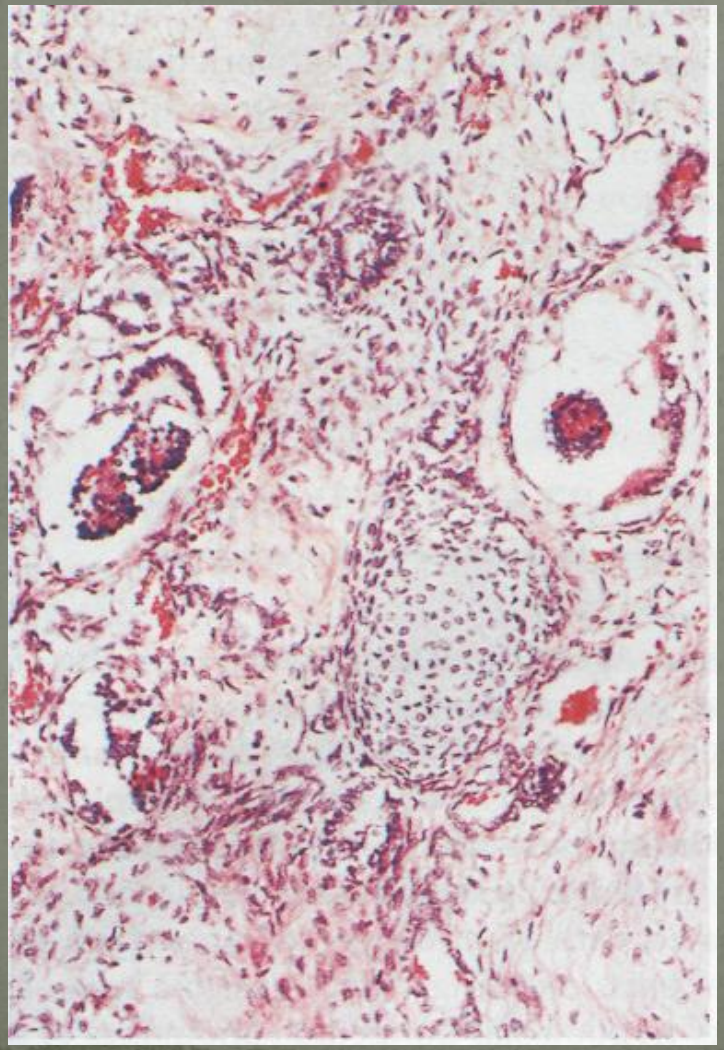
The Kidney
Renal dysplasia

- *Cystic renal dysplasia is characterized by the presence of numerous cysts in all or part of a kidney together with the persistence of abnormal structures, such as cartilage and undifferentiated mesenchyme.*

The Kidney
Renal dysplasia

- Unilateral renal dysplasia is the most common cystic disorder in children, and it is the most frequent cause of an abdominal mass in newborns. Malformations of other organs (e.g., ventricular septal defects, tracheoesophageal fistulas, and lumbosacral meningomyeloceles) occasionally occur in conjunction with renal dysplasia.

The Kidney
Renal dysplasia



Renal dysplasia. A light micrograph shows immature glomeruli, tubules, and cartilage surrounded by loose, undifferentiated mesenchymal tissue.

The Kidney
Renal dysplasia

- ***Pathology:*** The dysplastic kidney is enlarged and reveals a disorderly mass of cysts that vary in size from microscopic to several centimeters in diameter. An associated ureteral malformation, which is often obstructive, is usually found.

The Kidney
Renal dysplasia

- Histologically, dysplasia is recognized by focally dilated ducts that are lined by a cuboidal or columnar epithelium. These are surrounded by mantles of undifferentiated mesenchyme which sometimes contain smooth muscle and islands of hyaline cartilage.

The Kidney
Renal dysplasia

- ***Clinical Features:*** In most cases of cystic renal dysplasia, a palpable flank mass is discovered shortly after birth, although small, multicystic kidneys may not become apparent until many years later. Unilateral dysplasia is adequately treated by removal of the affected kidney.

The Kidney
Polycystic kidney diseases

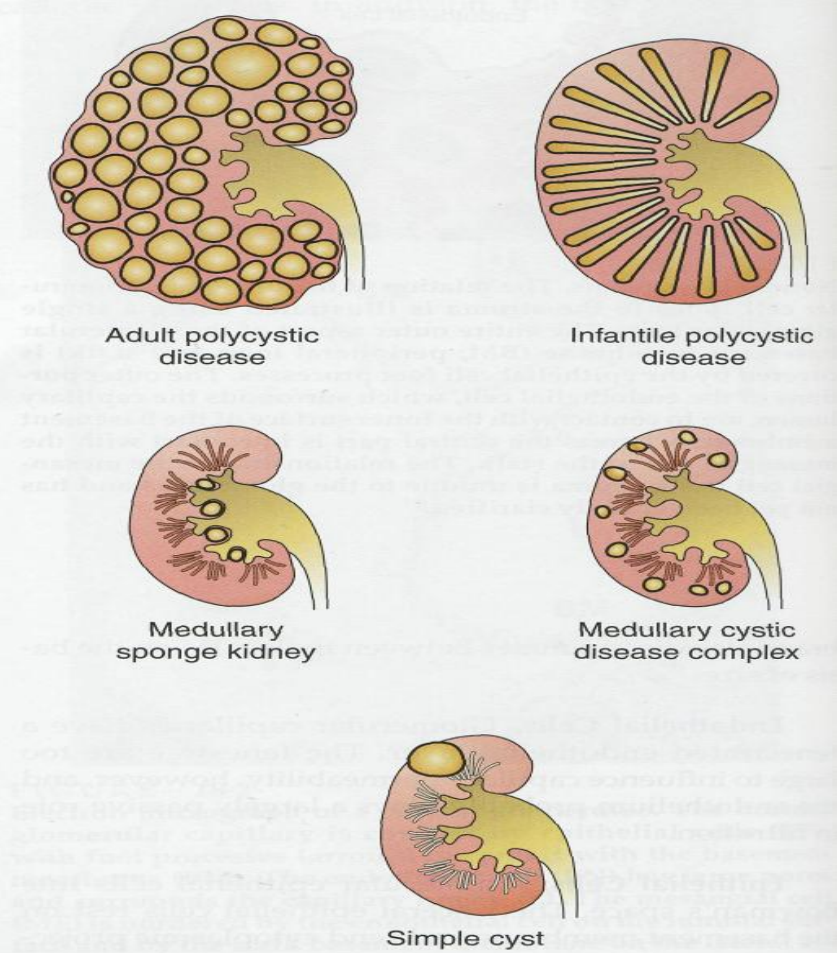


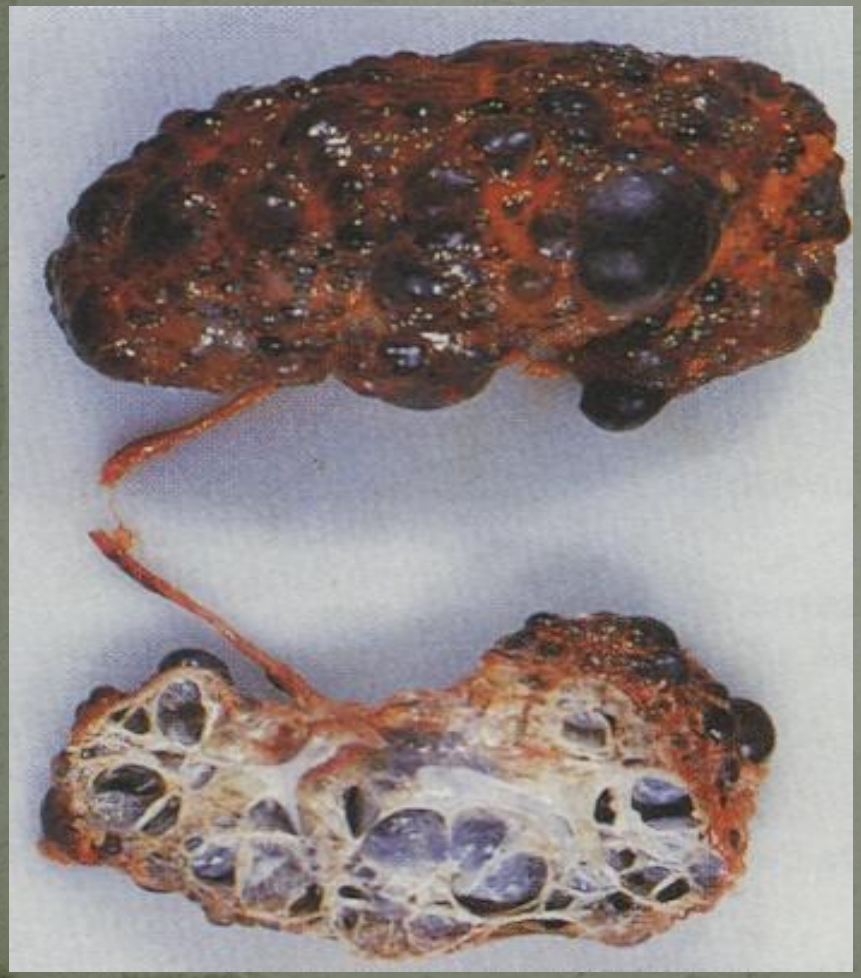
FIGURE 16-5
Cystic diseases of the kidney.

- *Polycystic kidney diseases are a heterogeneous group of congenital and acquired disorders that are characterized by distortion of the renal parenchyma because of numerous cysts.*

The Kidney
Autosomal Dominant (Adult) Polycystic Kidney Disease

- ***Adult polycystic kidney disease*** *is an autosomal dominant trait that is characterized by progressively expanding cysts.* The disorder is responsible for 10% of all end-stage kidney disease. The pathogenesis of adult polycystic kidney disease is not understood, although a defective gene *(AKPKD-1)* has been localized to the short arm of chromosome 16.

The Kidney
Autosomal Dominant (Adult) Polycystic Kidney Disease



- ***Pathology:*** The kidneys in patients with polycystic kidney disease are markedly enlarged bilaterally, each kidney weighing as much as 4500 g.

The Kidney
Autosomal Dominant (Adult) Polycystic Kidney Disease

- The external contour of the kidney is distorted by the presence of numerous cysts, which can be as large as 5 cm in diameter. These cysts are usually filled with a clear to straw-yellow fluid.

The Kidney
Autosomal Dominant (Adult) Polycystic Kidney Disease

- Microscopically, the cysts are lined by a nondescript cuboidal and columnar epithelium. As the cysts progressively expand, they exert pressure on the normal areas, thereby leading to an increasing loss of renal parenchyma.

The Kidney
Autosomal Dominant (Adult) Polycystic Kidney Disease

- One-third of the patients with adult polycystic kidney disease also have hepatic cysts lined by biliary epithelium. Fifteen percent of the patients have an associated cerebral aneurysm, and subarachnoid hemorrhage is the cause of death in many of these patients.

The Kidney
Autosomal Dominant (Adult) Polycystic Kidney Disease

- ***Clinical Features*:** Patients typically present with symptoms by the fourth decade of life. These include bilateral flank masses and passage of blood clots in the urine. Azotemia (elevated blood urea nitrogen level) is common and, in half of the patients, progresses to uremia (clinical renal failure) over a period of several years.

The Kidney
Autosomal Recessive (Infantile) Polycystic Kidney Disease

- ***Infantile polycystic kidney disease*** *is an uncommon autosomal recessive condition in which dilatation of cortical and medullary collecting ducts leads to a cystic kidney.*

The Kidney
Autosomal Recessive (Infantile) Polycystic Kidney Disease

- Pathology: In contrast to patients with adult polycystic kidney disease, the external surface of the kidney in patients with the infantile disorder is smooth, and the involvement is invariably bilateral. The fusiform cysts are dilatations of cortical and medullary collecting ducts, which have a striking radial arrangement.

The Kidney
Autosomal Recessive (Infantile) Polycystic Kidney Disease

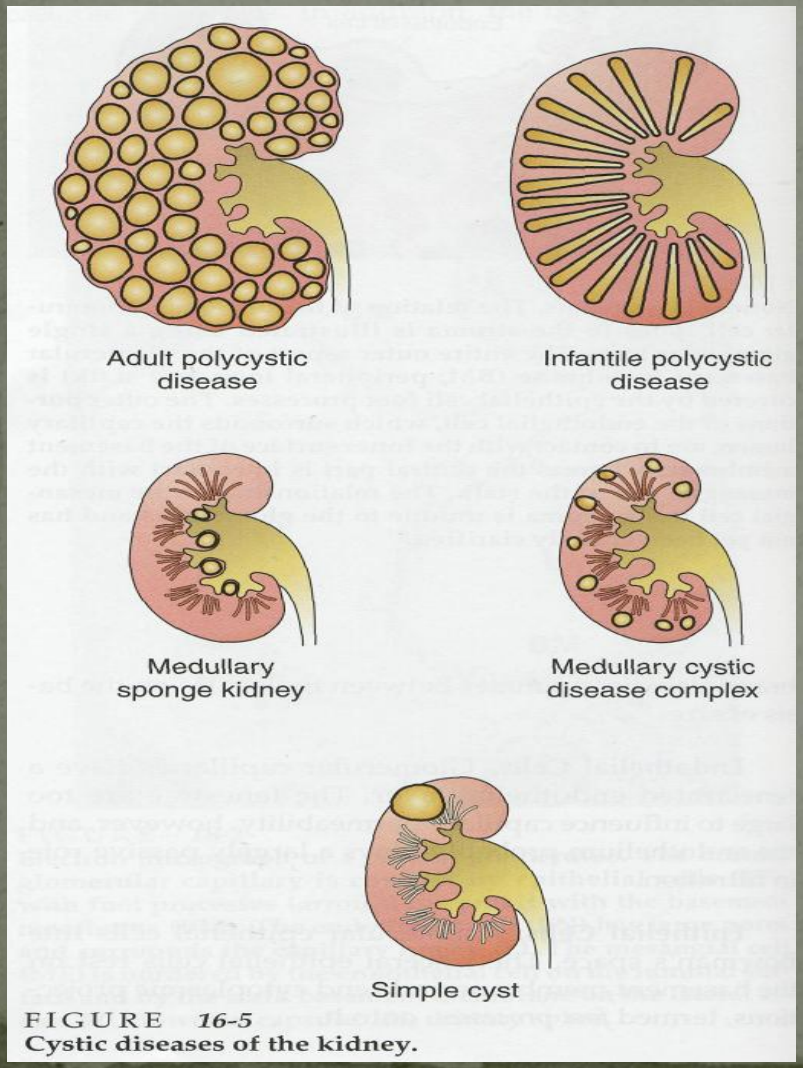


FIGURE 16-5
Cystic diseases of the kidney.

Cystic disease of the kidney.

The Kidney
Autosomal Recessive (Infantile) Polycystic Kidney Disease

- Interstitial fibrosis and tubular atrophy are common. There are usually associated liver changes, termed *congenital hepatic fibrosis*, that are characterized by the enlargement of portal areas, an increase in connective tissue, and a proliferation of bile ducts. A candidate gene has been proposed to reside on the short arm of chromosome 6.

The Kidney
Autosomal Recessive (Infantile) Polycystic Kidney Disease

- ***Clinical Features*:** Two clinical presentations occur in this condition. The more frequent is in newborns, who suffer from congenitally enlarged, spongy kidneys and renal insufficiency. Less frequently, older children present with enlarged kidneys that have small cysts, tubular atrophy, and interstitial fibrosis.

The Kidney
Nephronophthisis-Medullary Cystic Disease Complex

- *Nephronophthisis, the medullary cystic disease complex, is a group of related, autosomal recessive diseases that are characterized by renal medullary cysts, sclerotic kidneys, and renal failure.*

The Kidney
Nephronophthisis-Medullary Cystic Disease Complex

- ***Nephronophthisis, the medullary cystic disease complex,*** *is a group of related, autosomal recessive diseases that are characterized by renal medullary cysts, sclerotic kidneys, and renal failure.*

The Kidney
Nephronophthisis-Medullary Cystic Disease Complex

- Eighty-five percent of the cases are familial. Most of the patients are in the first or second decade of life, and the disease complex accounts for 10% to 20% of the cases of renal insufficiency during childhood.

The Kidney
Nephronophthisis-Medullary Cystic Disease Complex

- ***Pathology:*** The kidneys are small, and when they are sectioned, multiple, variable-size cysts (diameter, <1 cm) are seen at the corticomedullary junction These cysts arise from the distal portions of the nephron.

The Kidney
Nephronophthisis-Medullary Cystic Disease Complex

- Eventually, the corticomedullary cysts accumulate, and the remainder of the parenchyma becomes increasingly atrophic. Secondary glomerular sclerosis, interstitial fibrosis, and a nonspecific inflammatory infiltrate dominate the histologic picture.

The Kidney
Nephronophthisis-Medullary Cystic Disease Complex

- ***Clinical Features:*** These patients present with evidence of deteriorating tubular function. A defect in the concentrating ability of the tubules is reflected in polyuria, polydipsia, and enuresis (bed-wetting). Progressive azotemia and renal failure then follow, usually within 5 years of the onset of symptoms.

The Kidney
Simple Renal Cysts

- Simple renal cysts are very common acquired lesions and are found in approximately half of the population older than 50 years. They rarely produce clinical symptoms unless they are very large.

The Kidney
Simple Renal Cysts

- These cysts, which may be solitary or multiple, are found in the outer cortex, bulging the capsule, or less commonly, in the medulla. Microscopically, they are lined by a nondescript, flat epithelium.

Thank you!