Al-Mustaqbal University College

Pathophysiology 3rd stage

Disorders of Renal System

Part 2

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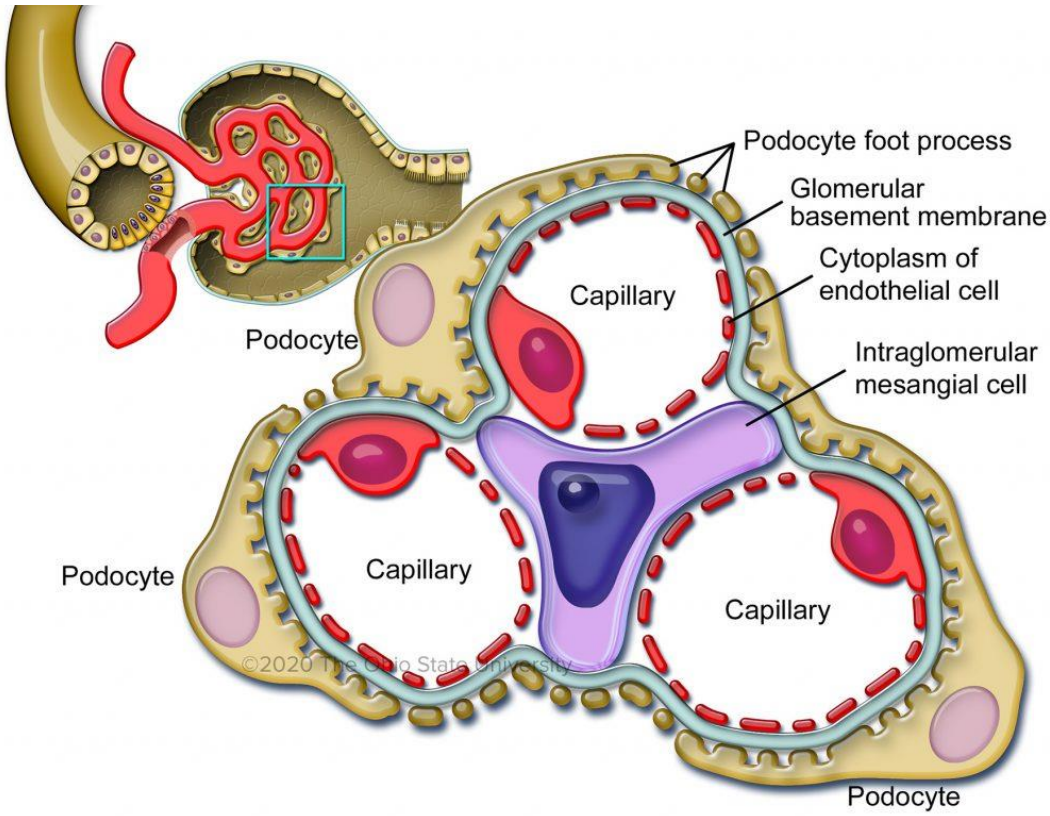
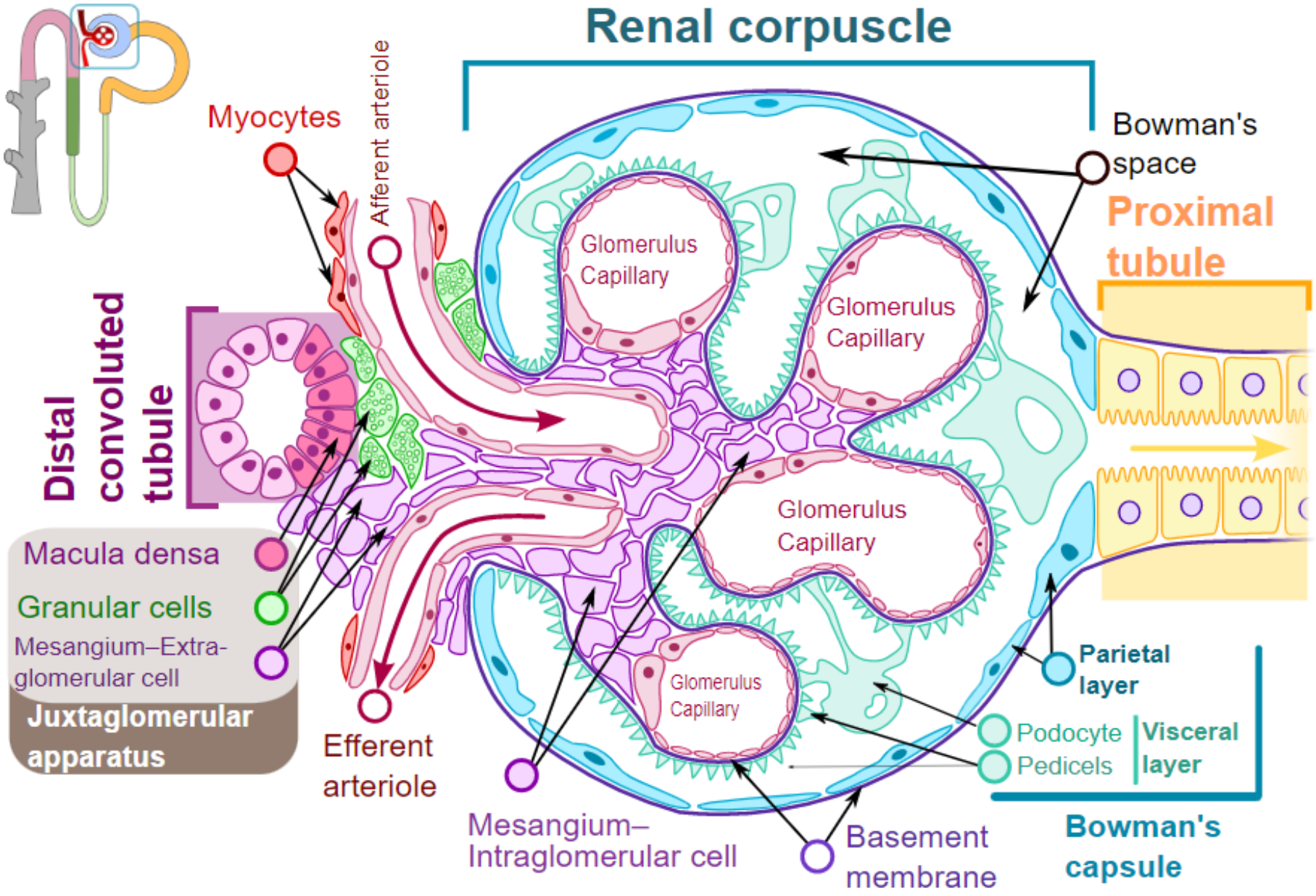
DISORDERS OF GLOMERULAR FUNCTION

The glomeruli are tufts of capillaries that lie between the afferent and efferent arterioles.

The glomerular membrane is composed of three layers: an endothelial layer lining the capillary, a basement membrane, and a layer of epithelial cells forming the outer surface of the capillary and lining Bowman's capsule



The capillary membrane is selectively permeable: it allows water,
electrolytes, and
dissolved particles, such as glucose
and amino acids,
to leave the capillary and enter Bowman's space and prevents larger particles, such as
plasma proteins
and blood cells, from leaving the blood.


Renal corpuscle
Myocytes
Afferent arteriole
Bowman's space
Proximal tubule
Glomerulus Capillary
Glomerulus Capillary
Glomerulus Capillary
Glomerulus Capillary
Distal convoluted tubule
Macula densa
Granular cells
Mesangium–Extra-glomerular cell
Juxtaglomerular apparatus
Efferent arteriole
Parietal layer
Podocyte
Pedicels
Visceral layer
Mesangium–Intraglomerular cell
Basement membrane
Bowman's capsule

Mechanisms of Glomerular Injury

Glomerulonephritis, an inflammatory process that involves glomerular structures.

The disease may occur as a primary condition or it may occur as a secondary condition.

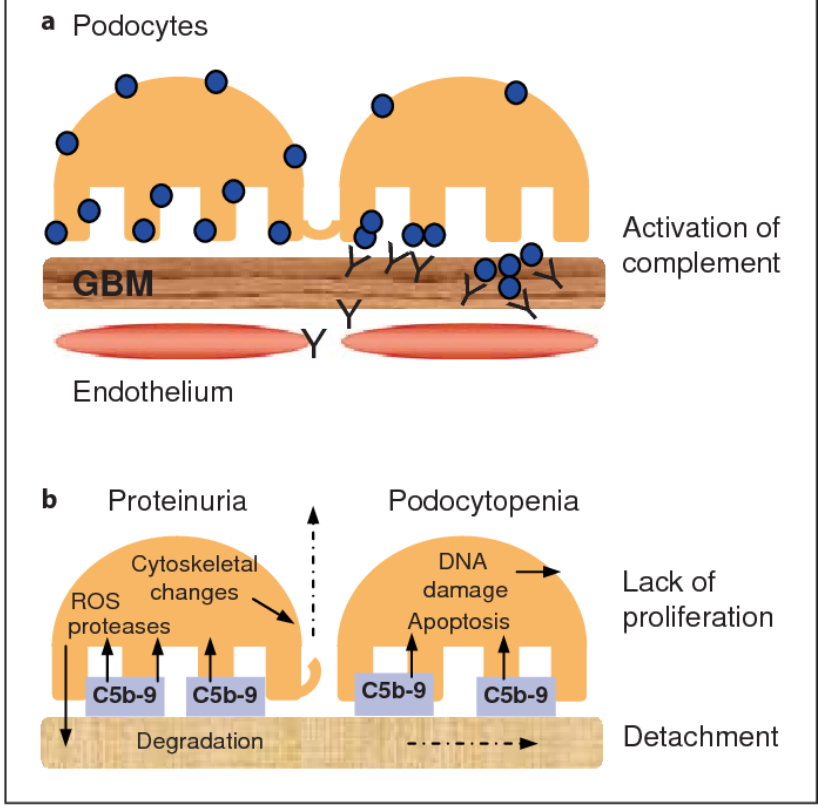
most cases of primary and many cases of secondary glomerular disease probably have an immune origin.

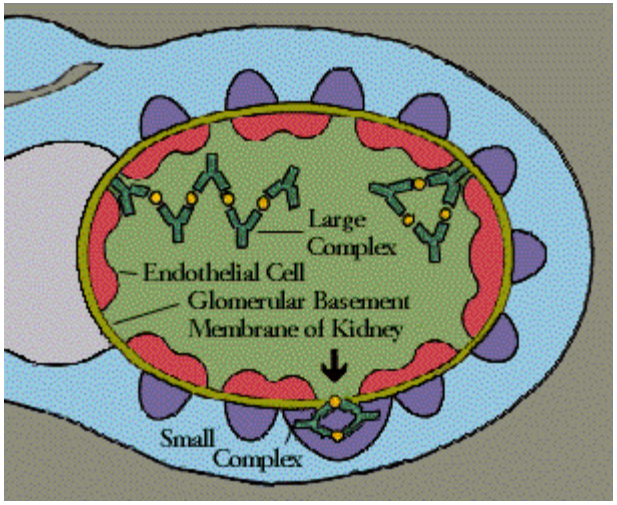
The cellular changes that occur with glomerular disease include proliferative, sclerotic, and membranous changes.

Two types of immune mechanisms have been implicated in the development of glomerular disease:

injury resulting from antibodies reacting with fixed glomerular antigens,

and injury resulting from circulating antigen-antibody complexes that become trapped in the glomerular membrane.





1- Acute Glomerulonephritis

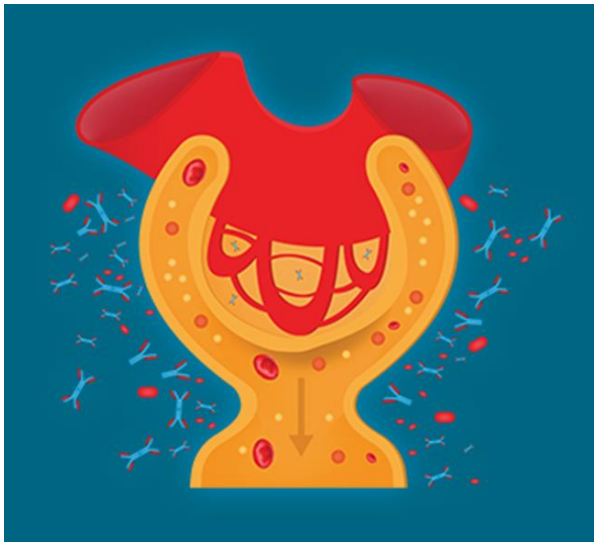
A sudden inflammation of the glomerulus is called acute glomerulonephritis.

Acute inflammation of the glomerulus occurs as a result of deposition of antibody-antigen complexes in the glomerular capillaries.

Complexes usually develop 7 to 10 days after a pharyngeal or skin streptococcal infection (poststreptococcal glomerulonephritis) but may follow any infection.

Inflammatory reactions in the glomeruli cause complement activation and mast-cell degranulation, leading to increased blood flow, increased glomerular capillary permeability, and increased glomerular filtration. Plasma proteins and red blood cells leak through the damaged glomeruli.

Acute glomerulonephritis usually resolves with specific antibiotic therapy, especially in children.



2- Chronic Glomerulonephritis

Chronic glomerulonephritis is the long-term inflammation of the glomerular cells. It may occur as a result of unresolved acute glomerulonephritis, or it might develop spontaneously.

Common causes include diabetes mellitus and long-standing hypertension.

Clinical Manifestations

All types of glomerulonephritis are associated with

Decreased urine volume.

Blood in the urine (brownish-colored urine), either gross or subtle.

Fluid retention.

Diagnostic Tools

Hematuria as measured by urinalysis.
Red blood cell casts in the urine.
Proteinuria greater than 3 to 5 g/day. (Normal < 150 mg/day)
Decreased GFR as measured by creatinine clearance.
If the condition is caused by acute poststreptococcal glomerulonephritis, antistreptococcal enzymes, such as antistreptolysin-O and antistreptokinase, will be present.

Complications

Renal failure may develop.

Treatment

antibiotic therapy, corticosteroids for immunosuppression, anticoagulants to decrease fibrin deposits, ACE inhibitors .

Renal Failure

Renal failure is the loss of function in both kidneys.

Stages of chronic kidney disease

Stage 1: Kidney damage including abnormalities in blood or urine tests or in imaging studies) with normal or near-normal glomerular filtration rate, at or above 90 mL/min

Stage 2: Glomerular filtration rate between 60 and 89 mL/min (approximately 50% of normal), with evidence of kidney damage.

Stage 3: Glomerular filtration rate between 30 and 59 mL/min (25 to 50% of normal).

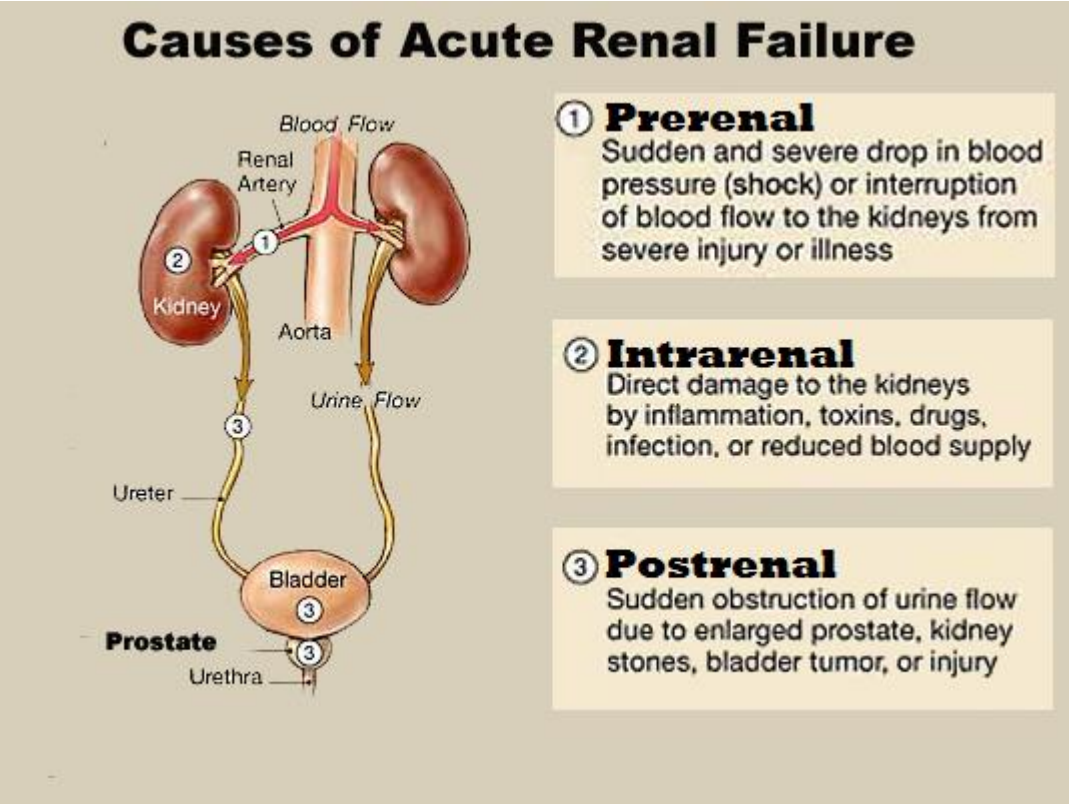
Stage 4: Glomerular filtration rate between 15 and 29 mL/min

Stage 5: End-stage renal failure; glomerular filtration rate of less than 15 mL/min

3- Acute Renal Failure

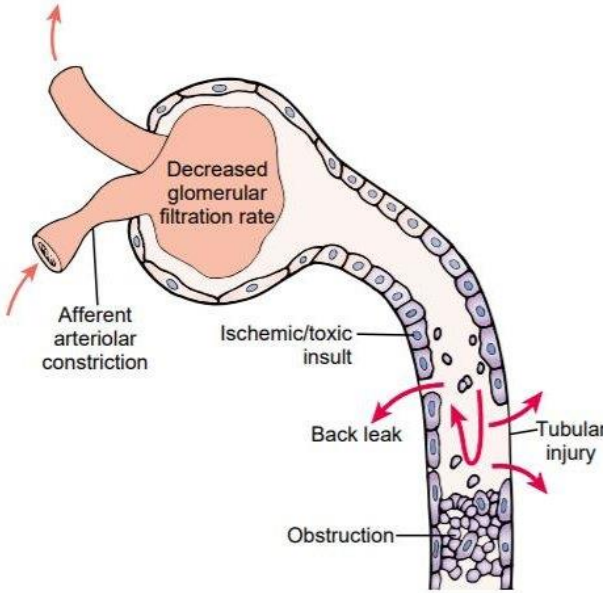
Causes of acute renal failure have been separated into three general categories:
prerenal,
intrarenal,
and postrenal.

Identifying the cause of acute renal failure is accomplished by a study of the patient's history and the quantity and quality of his or her urine.



Symptoms

- Oliguria may occur, especially if the failure is caused by ischemia or by obstruction. oliguria results from decreased GFR
- –Toxic tubular necrosis may be non-oliguric (high output) and is associated with the production of an adequate volume of dilute urine.

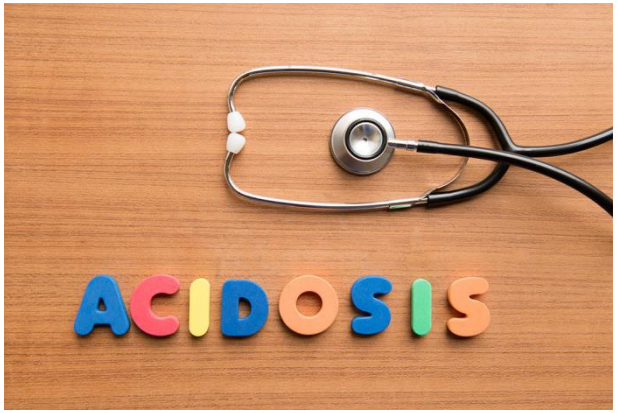


Diagnostic Tools

- A good history identifies precipitating causes of renal failure.

- Laboratory finding of azotemia (increased nitrogenous compounds in the blood), and elevated BUN and creatinine confirm diagnosis.

- Laboratory findings of hyperkalemia (increased potassium in the blood) and acidosis are common.

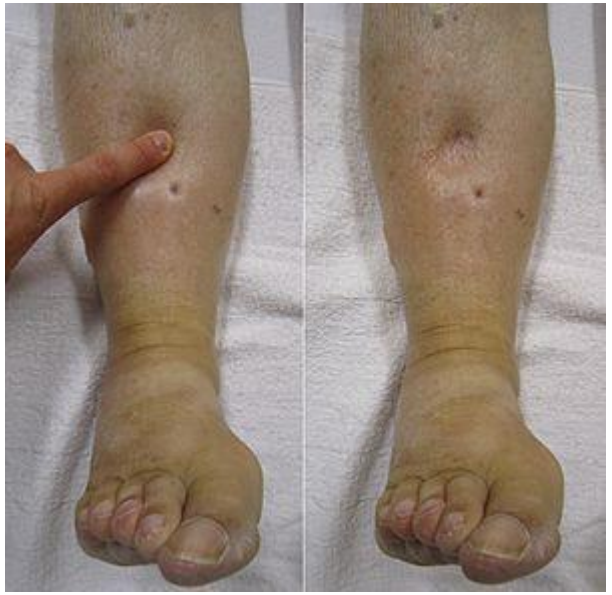


Complications

Fluid retention from non-functioning kidneys may lead to edema, congestive heart failure, or water intoxication.

Alterations in electrolytes and pH may cause uremic encephalopathy.

If the hyperkalemia is severe (6.5 mEq/L), dysrhythmia and muscle weakness may occur.



Chronic renal failure is the progressive, relentless destruction of renal structure.

Chronic renal failure can result from virtually any of the diseases described in this chapter.

In addition, analgesic nephropathy, the destruction of the renal papillae related to the daily use of analgesic medications for many years, may lead to chronic renal failure in susceptible individuals.

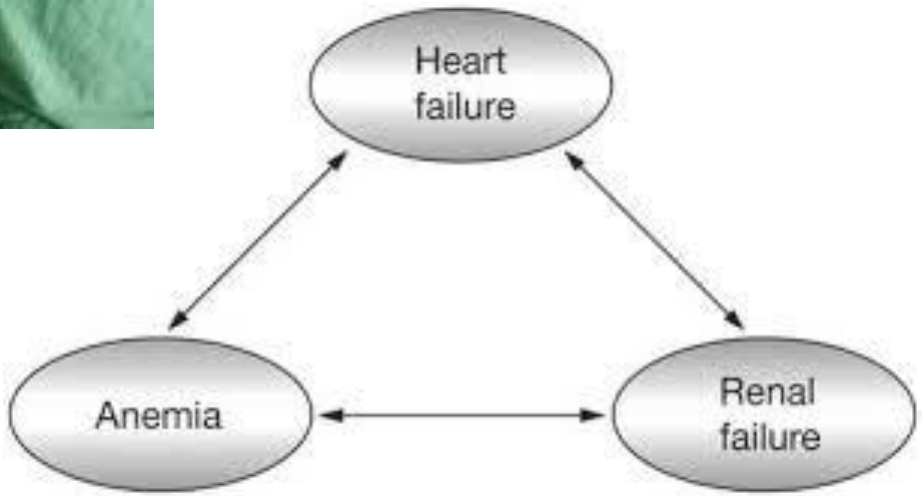
Regardless of the cause, unremitting deterioration of the kidneys occurs as indicated by a progressive fall in GFR.

Until renal function has decreased to less than 25% of normal, clinical manifestations of chronic renal failure may be minimal as surviving nephrons take over the functions of those lost.

As more nephrons progressively die, remaining ones have an increasingly difficult job, which leads to their own damage and eventual death.

Failure of adequate production of erythropoietin by the kidneys frequently leads to anemia and a resultant fatigue that negatively impacts quality of life.

In addition, chronic anemia leads to decreased tissue oxygenation which leads to activation of the sympathetic nervous system and an increase in cardiac output and consequently congestive heart failure.



Clinical Manifestations

- In stage 1 renal failure, no symptoms may be apparent.
- As disease progresses, reduced production of erythropoietin causes chronic fatigue, and early signs of tissue hypoxia and cardiovascular compromise may develop.
- As disease progresses, polyuria (increased urine output) occurs as the kidneys are unable to concentrate the urine.
- During the final stages of renal failure, urine output decreases because of low GFR.

Diagnostic Tools

- Radiographs or ultrasound will show small, atrophied kidneys.
- Serum BUN, creatinine, and GFR will be abnormal.
- Hematocrit and hemoglobin are reduced.
- Plasma pH is low.
- An elevated respiratory rate indicates respiratory compensation for metabolic acidosis.

Complications

With progression of renal failure,
volume overload,
electrolyte imbalance,
metabolic acidosis,
azotemia, and uremia occur.

Hypertension, anemia, osteodystrophy, hyperkalemia, uremic encephalopathy, and pruritus (itching) are common complications.

cardiorenal anemia syndrome,
Congestive heart failure may develop.
Without treatment, coma and death result.

Reference: Corwin , Pathophysiology, 3rd Edition