

# Pyelonephritis and Urinary Tract Infection

- Is defined as inflammation affecting the tubules, interstitium, and renal pelvis.
- It occurs in two forms.
  - a. Acute pyelonephritis
  - b. Chronic pyelonephritis

Note: Pyelonephritis is a serious complication of *urinary tract infections* that affect the bladder (cystitis)

# Etiology and Pathogenesis

- More than 85% of cases of urinary tract infection are caused by the gram-negative bacilli that are normal inhabitants of intestinal tract.
- The most common is *Escherichia coli*, followed by *Proteus*, *Klebsiella*, and *Enterobacter*.

- There are two routes by which bacteria can reach the kidneys:

(1) From the lower urinary tract (ascending infection) and is the most common cause of clinical pyelonephritis.

(2) Through the bloodstream (hematogenous infection)

# Mechanism of ascending infection

- Normal human bladder and bladder urine are sterile; therefore, a number of steps must occur for renal infection to occur:
  1. The first step is the *colonization of the distal urethra and introitus* (in the female) by coliform bacteria and the colonization is influenced by the degree of bacterial adherence to urethral mucosal epithelium

2. *From the urethra to the bladder,*

- Organisms gain entrance during urethral catheterization .
- **In the absence of instrumentation, *urinary infections are much more common in females, due to***
  - a. the shorter urethra in females,
  - b. The absence of antibacterial properties found in prostatic fluid

### **3. From bladder to kidneys**

#### **A. Urinary tract obstruction and stasis of urine.**

- Urinary tract infection is frequent among patients with lower urinary tract obstruction, such as may occur with benign prostatic hypertrophy, tumors, or calculi
- However, outflow obstruction or bladder dysfunction results in incomplete emptying and residual urine (stasis) and this favors infection of bladder

*- Infection of the bladder leads infection , irritation and incompetence of vesicouretral valvw and this allows infected bladder urine to relax into the ureters and then to renal pelvis (acquired vesicouretral reflux)*

## B. Congenital vesicouretral reflux

- Due a congenital absence or shortening of the intravesical portion of the ureter, such that the ureter is not compressed during micturition.



## **4. Intrarenal reflux.**

- Vesicoureteral reflux also affords a ready mechanism by which the infected bladder urine can be propelled up to the renal pelvis and deep into the renal parenchyma through open ducts at the tips of the papillae (intrarenal reflux).

- Intrarenal reflux is most common in the upper and lower poles of the kidney, where papillae tend to have flattened or concave tips rather than the convex pointed type present in the midzones of the kidney

# 1.Acute Pyelonephritis

- Is a suppurative inflammation of the kidney caused by bacterial and sometimes viral infection,

## Morphology

- The hallmarks of acute pyelonephritis are interstitial suppurative inflammation, and intratubular aggregates of neutrophils .
- The glomeruli are relatively resistant to the infection. except in candidal infection

# complications of acute pyelonephritis

## 1. Papillary necrosis

## 2. Pyonephrosis

- is seen when there is total or almost complete obstruction, particularly when it is high in the urinary tract, so pus fills the renal pelvis, calyces, and ureter with pus.

## **Clinically Features.**

*1 -Causes of Acute pyelonephritis*

*- is usually associated with*

*1. Vesicoureteral reflux*

*2. Pregnancy.*

*3. Gender and age.*

- After the first year of life and up to around age 40 years, infections are much more frequent in females.

- With increasing age the incidence in males rises as a result of prostatic hypertrophy and instrumentation.

4. Diabetes mellitus•

5. Immunosuppression

- Acute pyelonephritis usually presents with
  - a. a sudden onset of pain at the costovertebral angle with fever and malaise.
  - b. Indications of bladder and urethral irritation, such as dysuria, frequency, and urgency.
  - c. The urine contains many leukocytes (pyuria) derived from the inflammatory infiltrate, but pyuria does not differentiate upper from lower urinary tract infection.

## Note:

- The finding of leukocyte casts, typically rich in neutrophils (pus casts), indicates renal involvement, because casts are formed only in tubules.
- Uncomplicated acute pyelonephritis follows a benign course, disappear within a few days after appropriate antibiotic therapy
- The superimposition of *papillary necrosis* may lead to acute renal failure.



# Chronic Pyelonephritis and Reflux Nephropathy

- Is a disorder in which chronic tubulointerstitial inflammation and scarring involve the calyces and pelvis
- Note:  
Only chronic pyelonephritis and analgesic nephropathy affect the calyces, making pelvocalyceal damage an important diagnostic clue.

# classification

## 1. *Reflux nephropathy.*

- Is by far the more common form of chronic pyelonephritic scarring.
- Reflux nephropathy occurs early in childhood as a result of superimposition of a urinary infection on congenital vesicoureteral reflux and intrarenal reflux.

- Reflux may be unilateral or bilateral; and if bilateral it may lead to chronic renal failure.

## 2. *Chronic obstructive pyelonephritis.*

- Recur-rent infections superimposed on obstructive lesions lead bouts nflammation and scarring, resulting in chronic pyelonephritis.
- The disease can be
  - a. bilateral, as with posterior urethral valves, resulting in renal insufficiency unless the anomaly is corrected,
  - b. or unilateral, as occurs with calculi

# III.Urolithiasis

# Types of stones in the urinary tract

## 1. Calcium oxalate stones

- 1..in 5% of patients are associated with *hypercalcemia* and *hypercalciuria*, such as occurs
  - a. with hyperparathyroidism,
  - b. sarcoidosis, and other hypercalcemic states.

2. In About 55% have *hypercalciuria without hypercalcemia*. And this is caused by several factors, including
  - a. Hyperabsorption of calcium from the intestine (absorptive hypercalciuria),
  - b. an intrinsic impairment in renal tubular reabsorption of calcium (renal hypercalciuria)

3. 20% of calcium oxalate stones are associated with increased uric acid secretion (*hyperuricosuric calcium nephrolithiasis*), with or without hypercalciuria.

Note: The mechanism of stone formation in this setting involves “nucleation” of calcium oxalate by uric acid crystals in the collecting ducts.



4. Five percent are associated with *hyperoxaluria*, either
  - a. Hereditary (primary oxaluria) or,
  - b. More commonly, acquired by intestinal overabsorption in patients with enteric diseases. and this is called enteric hyperoxaluria, also occurs in vegetarians, because much of their diet is rich in oxalates.).

## *II. Magnesium ammonium phosphate stones*

- Are formed largely after infections by bacteria (e.g., *Proteus* ) that convert urea to ammonia.
- The resultant alkaline urine causes the precipitation of magnesium ammonium phosphate salts.
- These are the largest type of stones called staghorn stones calculi that occupy large portions of the renal pelvis .

### *III. Uric acid stones*

- Are common in individuals with hyperuricemia, such as
  - a. Gout,
  - b. and diseases involving rapid cell turnover, such as the leukemias. However,
  - c. *more than half of all patients with uric acid calculi have neither hyperuricemia nor increased urinary excretion of uric acid.*

Note: In this group, it is thought that an unexplained tendency to excrete urine of pH below 5.5 may predispose to uric acid stones, because uric acid is insoluble in acidic urine.

#### *IV. Cystine stones*

- Are caused by **genetic defects in the renal reabsorption of amino acids**, including cystine, leading to cystinuria.

- It can therefore be appreciated that
  - a. Increased concentration of stone constituents,
  - b. Changes in urinary pH,
  - c. Decreased urine volume,
  - d. and the presence of bacteria influence the formation of calculi.

- *However, many calculi occur in the absence of these factors;*
- *conversely, many individuals with hypercalciuria, hyperoxaluria, and hyperuricosuria often do not form stones.*
- it has therefore been postulated that stone formation is enhanced by a *deficiency in inhibitors of crystal formation in urine.*

- The inhibitors is long, include
  - a. pyrophosphate,
  - b. diphosphonate,
  - c. citrate,
  - d. and a glycoprotein called *nephrocalcin*.