

Acute pyelonephritis

Routes of infection · suppurative morphology · complications · clinical correlation

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraihsakhri.com

Learning objectives

- Distinguish ascending infection from hematogenous renal seeding.
- Explain the anatomic basis of reflux-associated renal infection.
- Recognize the characteristic neutrophilic tubular and interstitial lesions.
- Differentiate papillary necrosis, pyonephrosis, and perinephric abscess.
- Integrate symptoms, pyuria, urine casts, culture, and clinical course.

Acute pyelonephritis is a tubulointerstitial infection

Tubules

Tubular lumina provide a route for neutrophils and infection through the nephron.

Interstitium

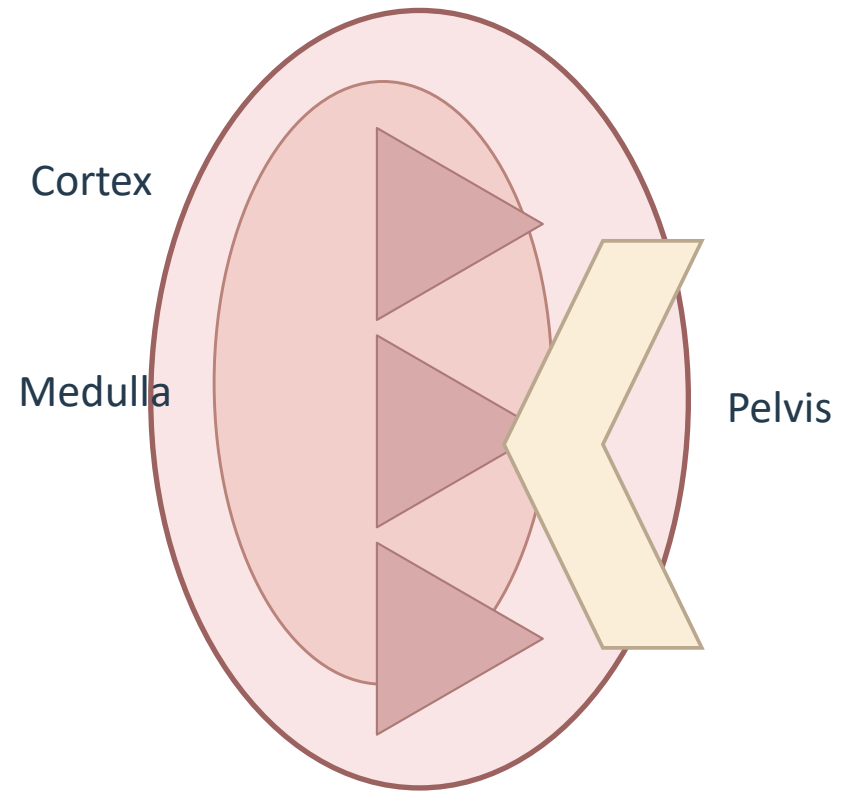
Neutrophil-rich inflammation produces focal suppuration and abscesses.

Renal pelvis

Infection of the collecting system links urinary infection with renal parenchymal injury.

The affected renal compartments

- Pyelonephritis involves tubules, interstitium, and the renal pelvis.
- Injury is not initially a primary glomerular disease.
- In severe cases, surrounding structures may also be destroyed.



Simplified renal anatomy

Definition: Suppurative inflammation of the kidney

Core lesion

Acute pyelonephritis is an infectious, neutrophil-rich inflammation involving renal tubules and interstitium, usually linked to ascending urinary infection.

Two entry routes reach the kidney

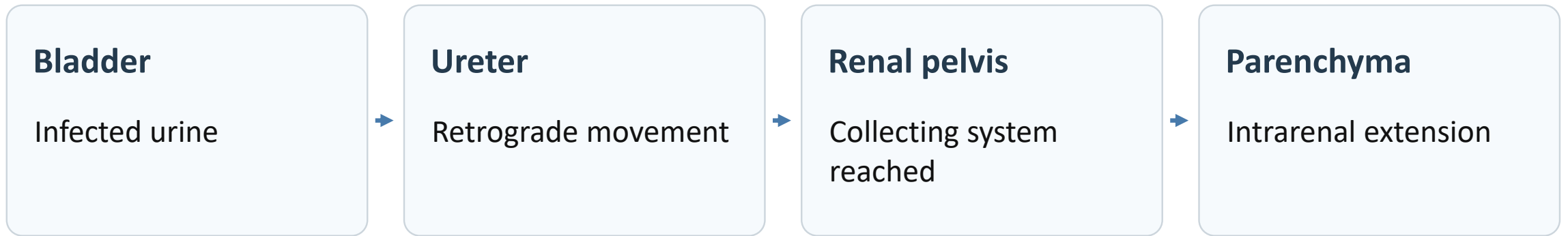
Ascending route — common

Lower urinary infection can ascend via the ureters, particularly with vesicoureteral and intrarenal reflux.

Hematogenous route — less common

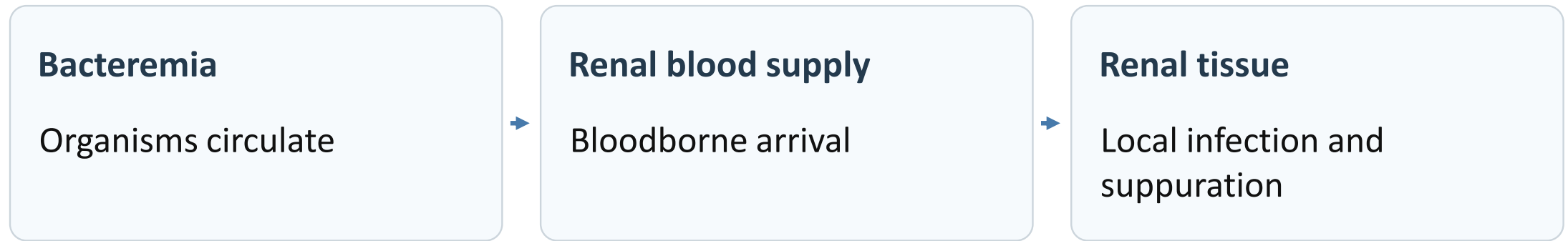
Organisms reaching the bloodstream can seed the kidney during bacteremia.

Ascending infection follows a urinary pathway



Vesicoureteral reflux and intrarenal reflux promote delivery of infected urine to renal tissue.

Hematogenous spread bypasses the ascending route



Hematogenous seeding as an alternative, less common route.

Enteric gram-negative bacilli predominate in UTIs

Microbial context

More than 85% of urinary tract infections are attributable to enteric gram-negative bacilli, which commonly originate from intestinal flora.

Escherichia coli is the leading organism

Most common

E. Coli is identified by Robbins as the leading urinary tract pathogen.

Clinical relevance

Persistent or recurrent renal infection can involve E. Coli or other organisms.

Other important enteric urinary pathogens

Proteus

Enteric gram-negative organism listed in the pyelonephritis pathogenesis section.

Klebsiella

Enteric gram-negative organism associated with urinary infection.

Enterobacter

Another enteric genus included in the source organism list.

Fungal and mycobacterial infection are possible

Typical bacterial disease

Neutrophilic, suppurative tubulointerstitial inflammation.

Fungal infection

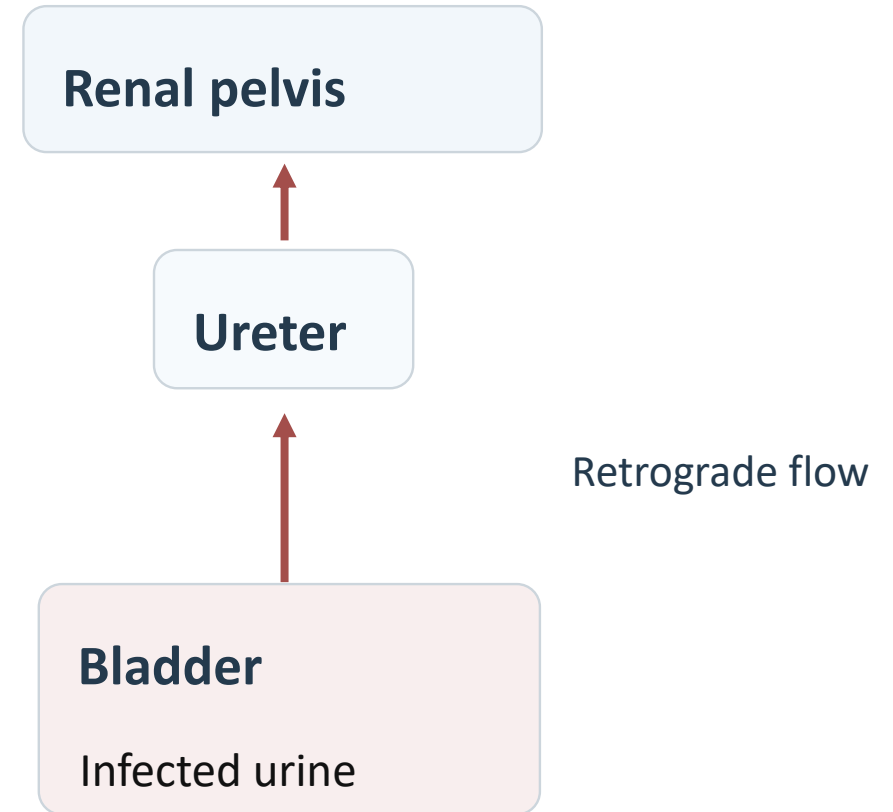
Candida-associated infection may elicit a granulomatous interstitial response.

Mycobacterial infection

Less common infectious cause; Granulomatous responses merit consideration.

Vesicoureteral reflux promotes upward spread

- Normally, urine passes from ureters into the bladder.
- Vesicoureteral reflux permits retrograde passage of bladder urine toward the renal pelvis.
- Reflux provides a route for infected urine to reach the kidney.



Intrarenal reflux drives bacteria into parenchyma

Vesicoureteral reflux

Infected urine ascends into the renal pelvis.



Open papillary ducts

Reflux may pass through papillary collecting ducts.

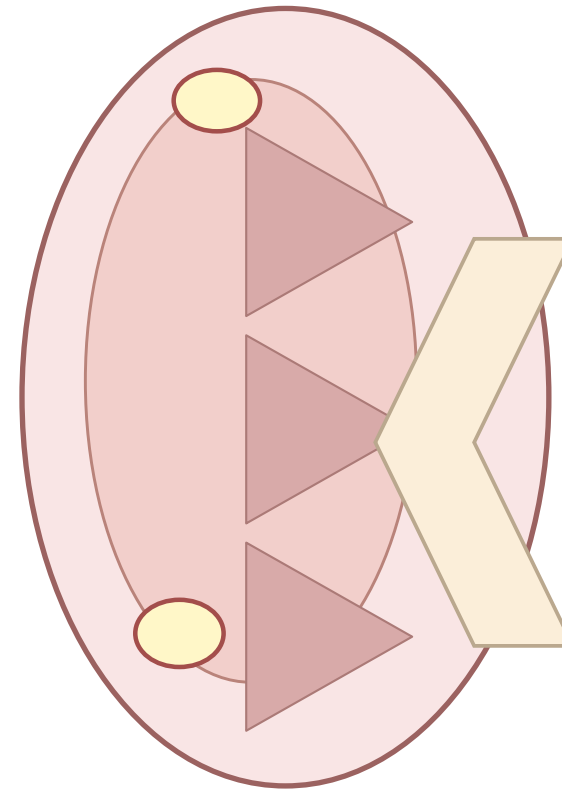


Renal tissue exposure

Organisms gain access to tubules and adjacent interstitium.

Upper and lower poles are vulnerable to intrarenal reflux

- Reflux is particularly frequent at the renal poles.
- Polar papillae commonly have flattened or concave tips.
- Open ducts at these papillae permit retrograde entry into renal parenchyma.



Polar reflux-prone sites
Simplified renal anatomy

Lower urinary infection does not imply pyelonephritis

Without significant upward reflux	With reflux and intrarenal spread
Infection may remain in the bladder.	Bacteria can reach the renal pelvis and papillary ducts.
Cystitis or urethritis may predominate.	Tubulointerstitial inflammation may develop.
Persistent bacteriuria alone does not establish renal involvement.	Renal signs and nephron-derived casts support upper tract involvement.

Urinary obstruction is a major predisposition

Outflow resistance

Congenital or acquired lesions impair drainage.

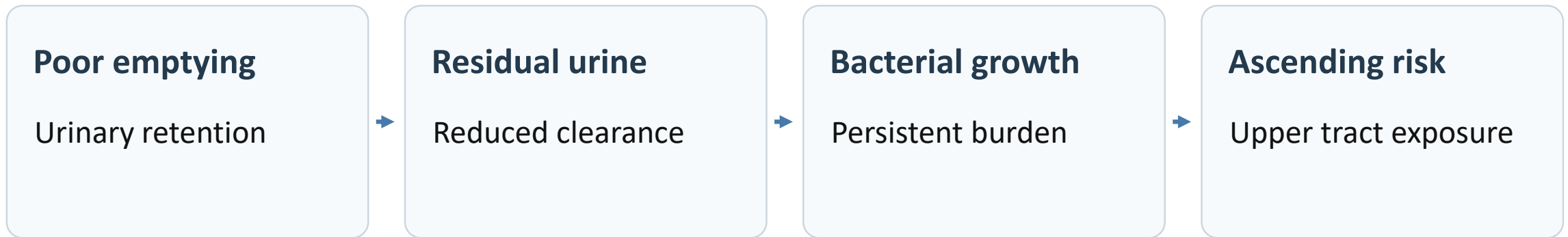
Urinary stasis

Residual urine favors continued bacterial growth.

Clinical consequence

Unrelieved obstruction increases the risk of severe or recurrent pyelonephritis.

Residual urine favors persistence of infection



Urinary stasis with obstruction and with bladder atony due to neurologic injury.

Instrumentation increases susceptibility

- Urinary catheterization is a common form of instrumentation.
- Instrumentation can facilitate urinary infection.
- Patients with repeated instrumentation may have additional predisposing conditions.

Instrumentation

Urinary catheterization



Bacterial access

Urinary infection



Additional risks

Predisposing conditions

Pregnancy is a recognized risk context

Urinary infection risk

Bacteriuria may arise during pregnancy.

Clinical significance

Without treatment, some pregnant patients with bacteriuria develop symptomatic urinary infection.

Interpretation

Pregnancy is a predisposing clinical context, not a distinct morphologic subtype.

Sex and age modify susceptibility

Life stage	Predisposing pattern
Infancy	Congenital urinary tract abnormalities may be detected, including in male infants.
After infancy to approximately 40 years	Urinary infections are more frequent in females.
Older age	Male incidence rises with prostatic enlargement and instrumentation.

Diabetes creates multiple susceptibility mechanisms

Host susceptibility

Greater propensity for infection.

Bladder dysfunction

Neurogenic bladder can promote impaired emptying.

Instrumentation

More frequent urinary procedures may add exposure.

Immunodeficiency increases clinical risk

Acquisition

Immunosuppression or immunodeficiency is listed among predisposing conditions.

Disease severity

Complicated infection may lead to repeated episodes of septicemia.

Preexisting renal lesions can amplify risk

Prior renal abnormality

Renal scarring or anatomic distortion may obstruct urinary flow.



Urinary stasis

Impaired drainage facilitates persistence of bacteria.



Subsequent renal infection

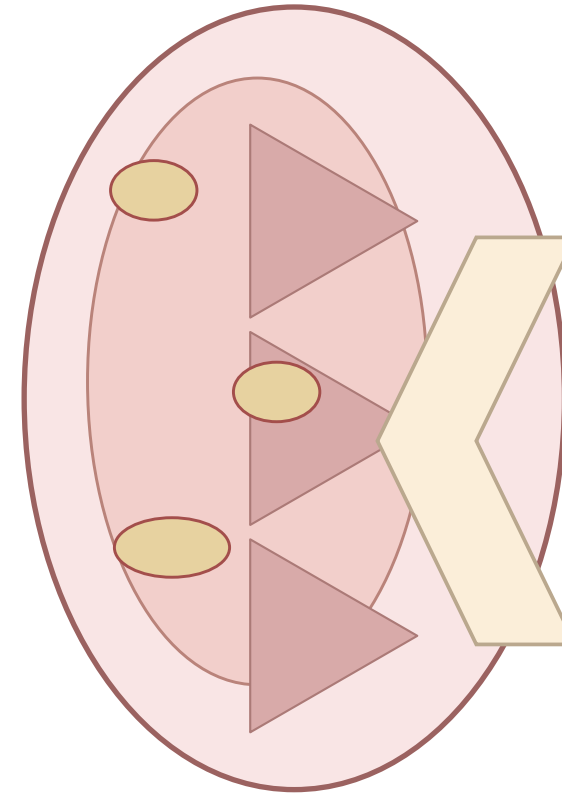
The predisposition can sustain recurrent upper urinary infection.

Risk factors converge on impaired urinary defense

Predisposing factor	Principal link to renal infection
Reflux	Ascending passage of infected urine
Obstruction / residual urine	Stasis and bacterial persistence
Instrumentation	Urinary tract exposure
Diabetes / immunodeficiency	Greater susceptibility or severe infection
Pregnancy / age-related changes	Clinical context favoring urinary infection

Gross lesions are patchy and suppurative

- Gray-white cortical foci may represent inflammation and abscess formation.
- Lesions may be discrete or confluent.
- Morphologic distribution reflects locally destructive infection.



Simplified renal anatomy

Abscesses may coalesce into wedge-like areas

Discrete microabscesses

Focal neutrophil collections initially involve tubules and interstitium.

Larger wedge-like lesions

Coalescent suppuration may extend through a sector of renal tissue.

Either one kidney or both may be involved

Unilateral disease

A single kidney may display localized focal suppuration.

Bilateral disease

Infection may involve both kidneys.

Distribution is variable

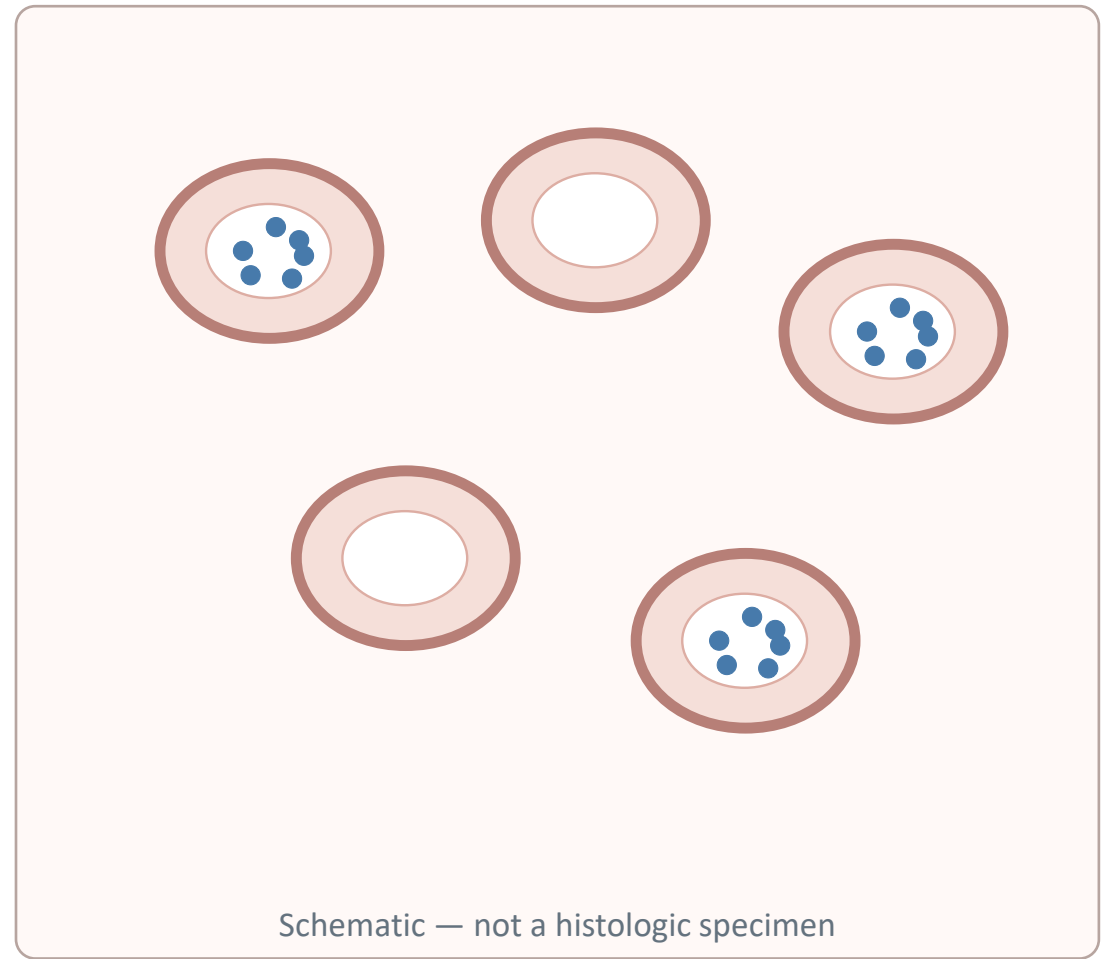
Neither laterality nor gross extent alone defines the underlying route.

Four histologic hallmarks of acute pyelonephritis

Hallmark	Microscopic implication
Interstitial suppuration	Neutrophil-rich inflammation within renal interstitium
Intratubular neutrophils	Collections of neutrophils in tubular lumina
Neutrophilic tubulitis	Neutrophils infiltrating tubular epithelium
Tubular injury	Epithelial injury associated with acute inflammation

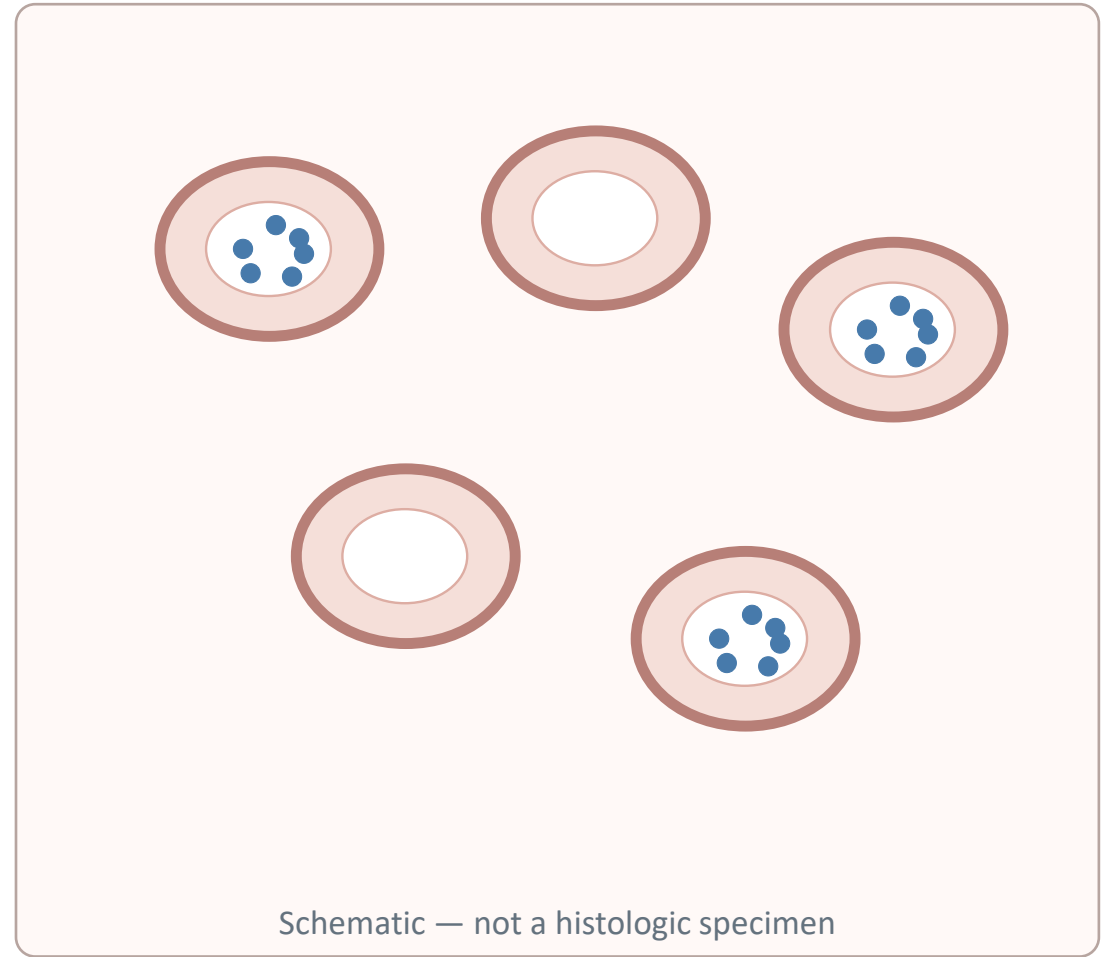
Early inflammation may be confined to tubules

- The earliest neutrophilic infiltrate may be limited to tubular lumina.
- Tubular lumina can act as pathways for infection.
- Interstitial spread follows when infection extends beyond affected tubules.



Neutrophils can aggregate inside tubular lumina

- Intratubular aggregates are a core feature of acute pyelonephritis.
- Collections reflect neutrophil recruitment to infected tubular spaces.
- Urinary leukocyte casts may derive from this intrarenal inflammatory compartment.



Neutrophilic tubulitis indicates epithelial invasion

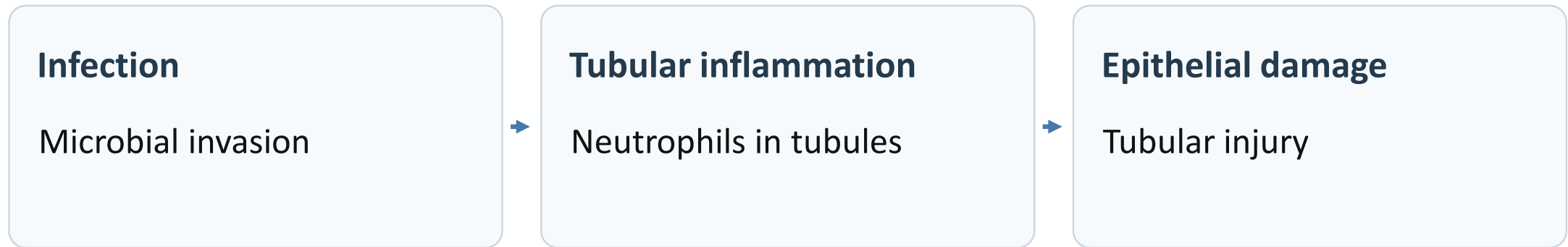
Finding

Neutrophils are present in or traverse the tubular epithelium.

Pathologic meaning

The tubule is directly involved, not merely surrounded by interstitial inflammation.

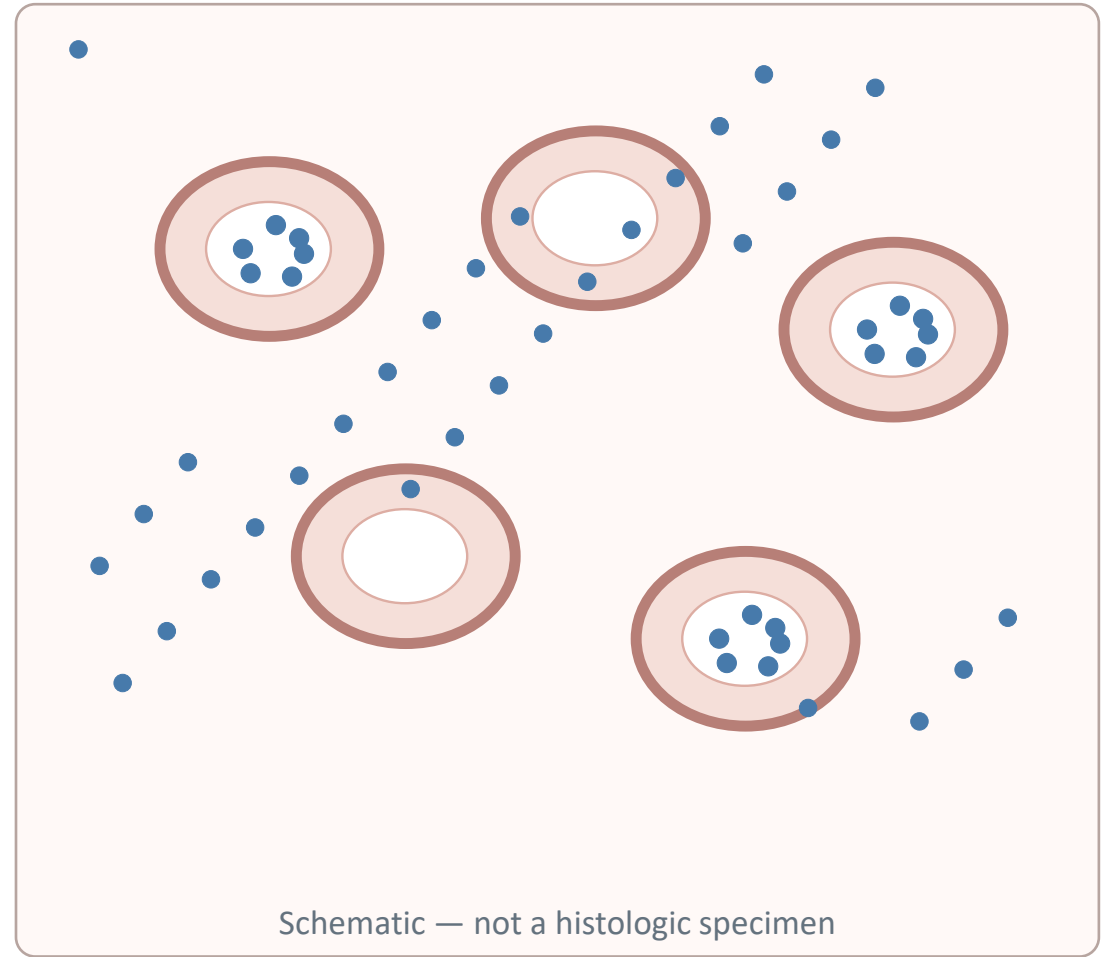
Tubular injury accompanies neutrophilic infiltration



Tubular injury is one of the four defining microscopic hallmarks emphasized by Robbins.

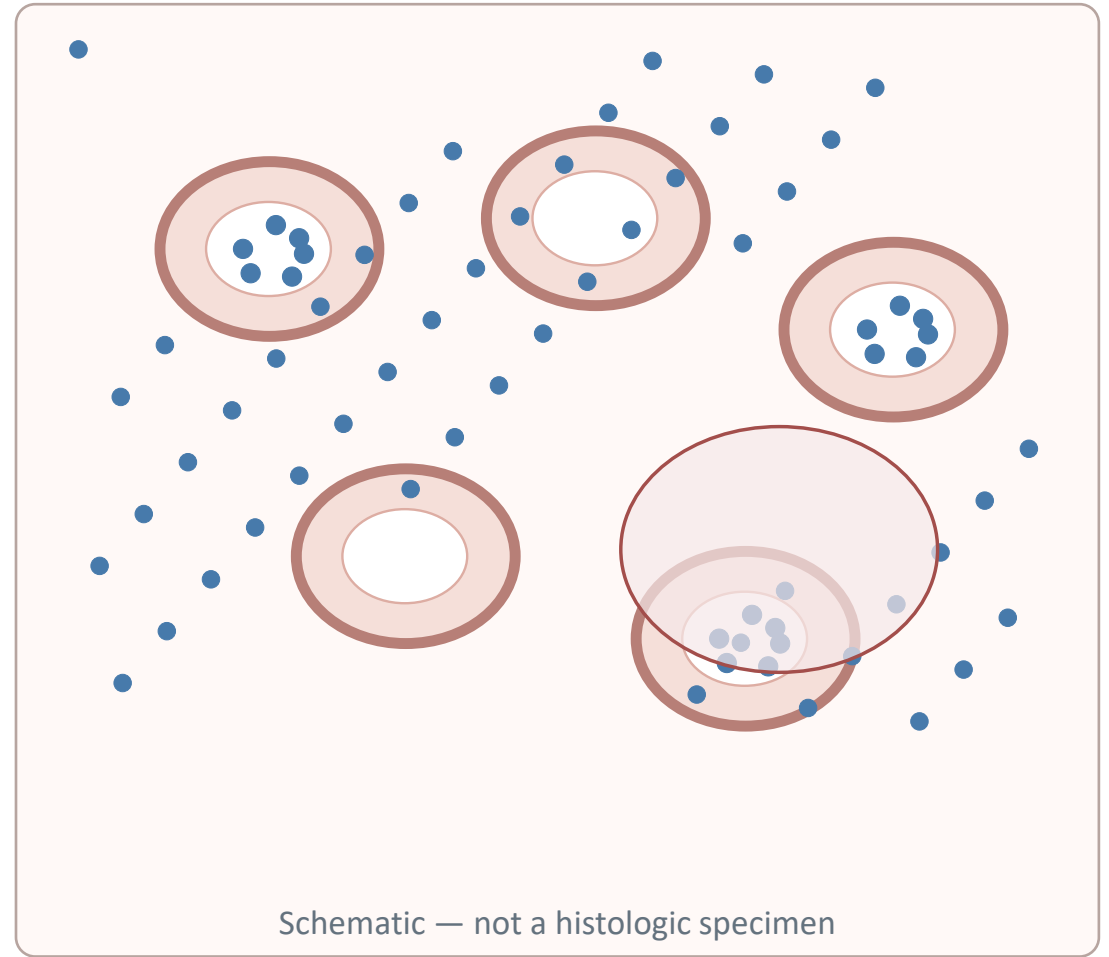
Interstitial spread produces suppurative inflammation

- Neutrophils extend from involved tubules into surrounding interstitium.
- Interstitial collections alter and destroy normal tubular architecture.
- Focal suppuration can form abscesses.



Abscess formation can destroy involved tubules

- Focal abscesses comprise collections of suppurative inflammatory cells.
- Progressive infection causes loss of tubules within the abscess.
- This differs from mere luminal neutrophils in early disease.



Glomeruli are relatively resistant early in disease

Early or moderate disease

Predominant damage involves renal tubules and interstitium.

Glomeruli

Glomerular structures are often spared initially.

Interpretation

The characteristic morphologic emphasis is tubulointerstitial, not glomerulonephritic.

Extensive infection can eventually destroy glomeruli

Severity-dependent extension

Relative glomerular sparing is not absolute. With sufficiently extensive renal destruction, glomeruli may also be involved.

Fungal pyelonephritis may be granulomatous

Typical acute bacterial pattern	Fungal-associated pattern
Suppurative interstitial inflammation	Granulomatous interstitial inflammation may occur
Tubular neutrophils and tubulitis	Inflammatory morphology may depart from the typical neutrophilic pattern
E. Coli and other enteric organisms predominate	Candida is a fungal example provided

Morphologic progression: From tubules to abscess

Tubular neutrophils

Inflammatory cells are initially concentrated in renal tubules.



Interstitial extension

Suppuration spreads into surrounding renal interstitium.



Focal destruction

Abscesses destroy tubules; Severe disease may extend further.

Morphology must be interpreted at the correct scale

Examination	Characteristic observation
Gross kidney	Patchy gray-white suppurative cortical areas, sometimes wedge-like
Microscopy — tubules	Intraluminal neutrophil aggregates, tubulitis, tubular injury
Microscopy — interstitium	Suppurative inflammation and focal abscesses
Severe disease	Extensive tubular destruction and possible glomerular involvement

Three major complications

Papillary necrosis

Ischemic coagulative necrosis of renal papillae.

Pyonephrosis

Pus accumulates in an obstructed renal collecting system.

Perinephric abscess

Suppuration extends through the capsule into perinephric tissue.

Papillary necrosis has characteristic risk factors

Diabetes

A major setting for papillary necrosis complicating infection.

Sickle cell disease

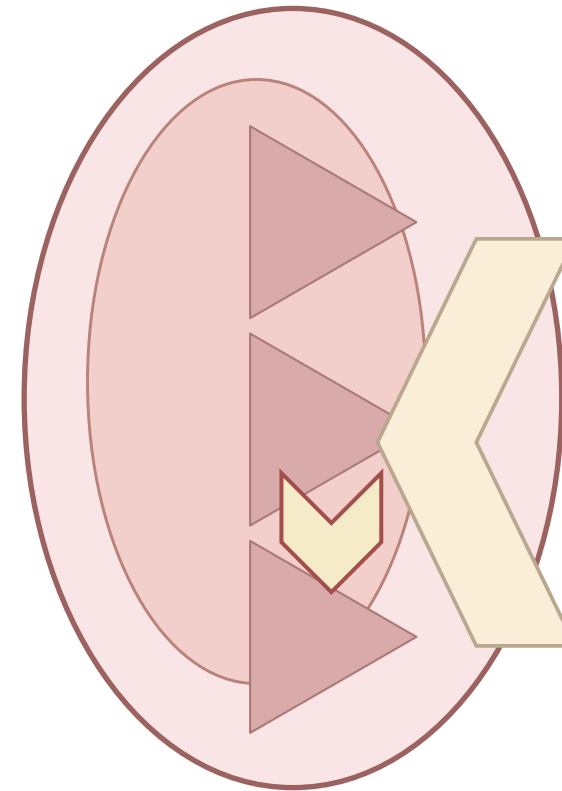
Another important predisposing condition.

Urinary obstruction

Outflow impairment increases susceptibility.

Papillary necrosis: Gross location of tissue death

- One or several renal pyramids may be affected.
- Necrosis may involve papillary tips or distal portions of pyramids.
- The necrotic tissue is gray-white to yellow on cut section.



Simplified renal anatomy

Papillary necrosis shows ischemic coagulative necrosis

Architecture

Outlines of necrotic tubules remain recognizable.

Inflammation

Leukocytic response is concentrated at the boundary of viable and dead tissue.

Interpretation

Coagulative papillary injury differs from diffuse intratubular neutrophilic suppuration.

Papillary necrosis may precipitate acute renal failure

Clinical consequence

Superimposed papillary necrosis can produce acute renal failure, particularly in vulnerable patients with severe pyelonephritis.

Pyonephrosis requires severe outflow obstruction

High-grade obstruction

Drainage becomes nearly or completely blocked.



Suppurative exudate persists

Purulent material cannot leave the collecting system.

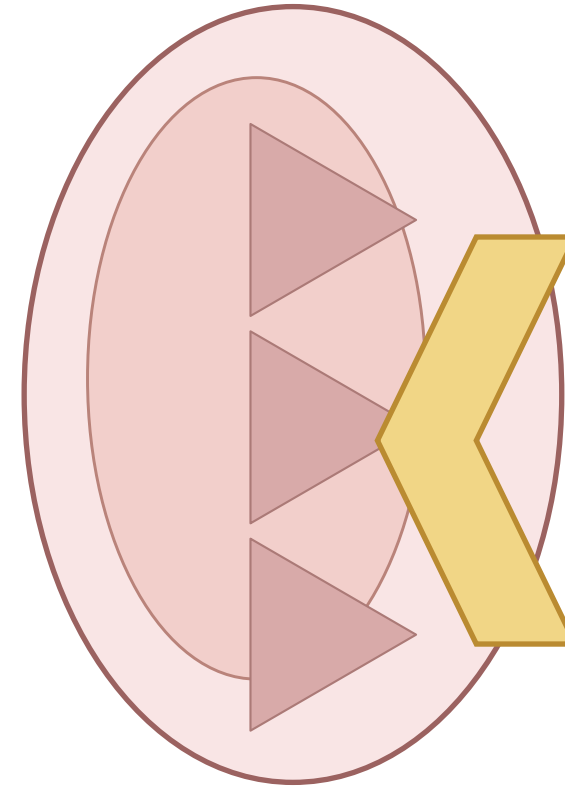


Pyonephrosis

Pelvis, calyces, and sometimes ureter fill with pus.

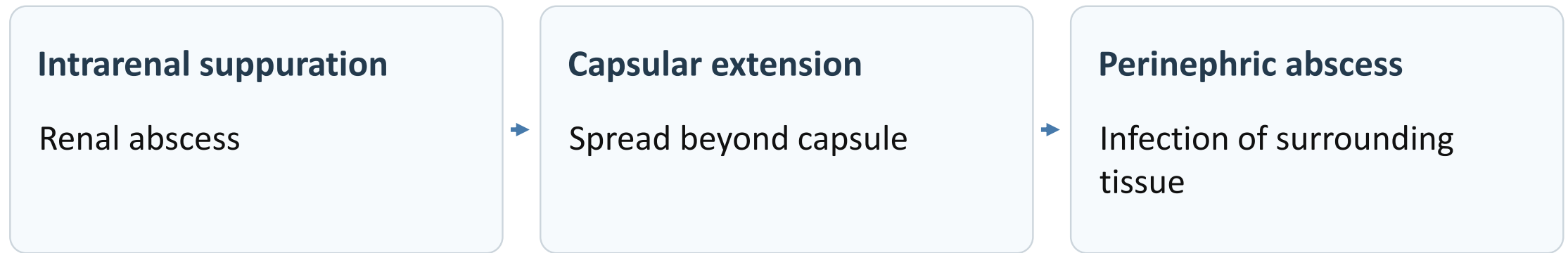
Pyonephrosis is pus within the collecting system

- The primary accumulation is within pelvis and calyces.
- The ureter may also be filled with pus.
- Retention results from near-total or total obstruction, often high in the tract.



Simplified renal anatomy

Perinephric abscess crosses the renal capsule



Anatomic location distinguishes this complication from pus confined to the collecting system.

Complications are anatomically distinct

Complication	Compartment	Defining process
Papillary necrosis	Medullary papilla	Ischemic coagulative necrosis
Pyonephrosis	Pelvis, calyces ± ureter	Pus trapped by severe obstruction
Perinephric abscess	Beyond renal capsule	Extension of suppuration into perinephric tissue

Resolution begins with a change in inflammatory cells

Active infection

Prominent neutrophilic infiltrate.



Postacute healing

Macrophages, plasma cells, and lymphocytes replace neutrophils.



Fibrous repair

Irregular cortical scars may eventually develop.

Healed lesions may produce patchy fibrous scars

Gross change

Cortical surface may show irregular fibrous depressions.

Microscopic change

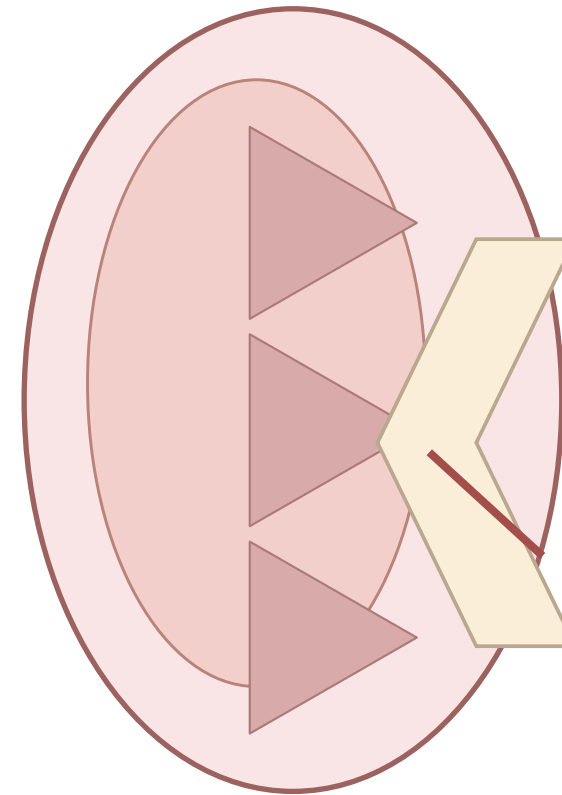
Tubular atrophy and interstitial fibrosis.

Residual inflammatory pattern

Lymphocytes may remain in a patchy, jigsaw-like pattern around preserved areas.

Pyelonephritic scars often deform the calyces

- Healing-related fibrosis may involve the underlying pelvis and calyces.
- Pelvocalyceal deformation is an important clue to reflux-associated renal injury.
- This links a parenchymal scar to the urinary route of infection.



Simplified renal anatomy

Acute inflammation and chronic scarring are different

Acute infection	After healing / recurrent injury
Neutrophil-rich suppuration	Lymphocyte-rich chronic inflammation may persist
Tubulitis and acute tubular damage	Tubular atrophy and interstitial fibrosis
Focal renal abscesses	Irregular cortical fibrous depressions
Potentially reversible acute disease	Permanent architectural distortion can remain

The clinical syndrome often begins abruptly

Flank or costovertebral angle pain

Reflects acute renal-region involvement.

Fever and malaise

Systemic manifestations of infection.

Sudden presentation

An abrupt course is typical of uncomplicated acute disease.

Lower tract irritation may coexist

Dysuria

Painful urination may indicate concurrent lower tract involvement.

Frequency

Repeated need to void can accompany infection.

Urgency

Strong or sudden urge to urinate may occur.

Pyuria confirms inflammation, not renal localization

Diagnostic limitation

Many urinary leukocytes are compatible with urinary tract infection, but pyuria by itself cannot distinguish lower urinary tract infection from pyelonephritis.

Leukocyte casts indicate renal involvement

Renal tubules

Urinary casts are formed within tubular lumina.



Neutrophils within casts

Pus casts may contain many neutrophils.



Interpretation

Leukocyte casts support an intrarenal inflammatory process.

Urine culture establishes urinary infection

Clinical suspicion

Symptoms and urine findings suggest urinary infection.

Culture

Urine culture is used to establish the infection.

Anatomic interpretation

Clinical features and renal-derived casts help localize involvement.

Clinical and laboratory data answer different questions

Observation	Diagnostic contribution	Limitation
Fever + costovertebral pain	Raises suspicion of upper tract infection	Not an organism-specific result
Pyuria	Shows leukocytes in urine	Does not localize upper vs lower tract
Leukocyte casts	Supports intrarenal involvement	Interpret with clinical context
Urine culture	Establishes urinary infection	Does not alone show renal histology

Uncomplicated disease usually responds well

Appropriate antibiotics

Symptoms commonly subside within days after treatment begins.

Course

Uncomplicated infection generally has a favorable clinical course.

Persistence and recurrence are distinct possibilities

Persistence

Bacteria may remain in urine despite improvement of symptoms.

Recurrence

Later infections may involve new E. Coli serologic types or other organisms.

Complicated disease can cause systemic deterioration

Persistent predisposition

Obstruction, diabetes, or immunodeficiency remains.



Severe infection

Episodes may be persistent or recurrent.



Systemic consequences

Repeated septicemic episodes; Papillary necrosis may cause acute renal failure.

Clinical case 1: Localizing urinary inflammation

Vignette

An adult presents with fever, costovertebral angle pain, dysuria, and marked pyuria. Urine microscopy also shows leukocyte casts.

Question

Which finding best indicates that the kidney is involved rather than only the bladder?

Case 1 interpretation: A nephron-derived finding

Leukocyte casts

Casts form in renal tubules; Neutrophil-rich leukocyte casts therefore provide evidence of renal involvement. Pyuria alone does not localize the infection.

Clinical case 2: Obstructed suppurative infection

Vignette

A patient with severe urinary outflow obstruction develops pyelonephritis. Suppurative material accumulates within the renal pelvis and calyces because drainage is blocked.

Question

What complication is described, and what is its defining anatomic location?

Case 2 interpretation: Pyonephrosis

Pyonephrosis

Near-complete or complete obstruction prevents drainage of suppurative exudate. Pus fills the pelvocalyceal system and may extend into the ureter.

Clinical case 3: Diabetic renal papillary injury

Vignette

A patient with diabetes and severe acute pyelonephritis develops renal papillary injury. Microscopy shows necrotic papillary tissue retaining outlines of renal tubules.

Question

Which complication explains this characteristic pattern of tissue necrosis?

Case 3 interpretation: Papillary necrosis

Ischemic coagulative necrosis

Papillary necrosis is a recognized complication in diabetes, sickle cell disease, and obstruction. Preserved ghost outlines of tubules typify the necrotic papilla.

Core pathology-to-clinic synthesis

Structural finding	Interpretive or clinical link
Ascending infection with reflux	Urinary source of intrarenal infection
Tubular + interstitial neutrophils	Acute suppurative kidney infection
Leukocyte casts	Renal localization of urinary inflammation
Papillary necrosis	Risk of acute renal failure
Obstructed pus-filled collecting system	Pyonephrosis
Capsular spread	Perinephric abscess

Five central teaching points

- Ascending infection is common; Hematogenous seeding is an alternative.
- Vesicoureteral and intrarenal reflux allow access to renal tissue.
- Neutrophilic tubulitis, tubular injury, and interstitial suppuration define the lesion.
- Papillary necrosis, pyonephrosis, and perinephric abscess are distinct complications.
- Leukocyte casts indicate intrarenal involvement; Urine culture establishes infection.

Source and recommended reading

Primary source

Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. (2026). Chapter 20, The Kidney: Pyelonephritis and Urinary Tract Infection; Acute Pyelonephritis. Printed pp. 841–844.

Scope of this lecture

Source-grounded description of routes, risks, morphology, complications, healing, and diagnostic interpretation. Original editable schematics replace unavailable textbook figures; they are not diagnostic specimens.