

Acute and Chronic Cystitis

Etiology • Morphology • Clinicopathologic interpretation

Bladder inflammation and its clinical consequences

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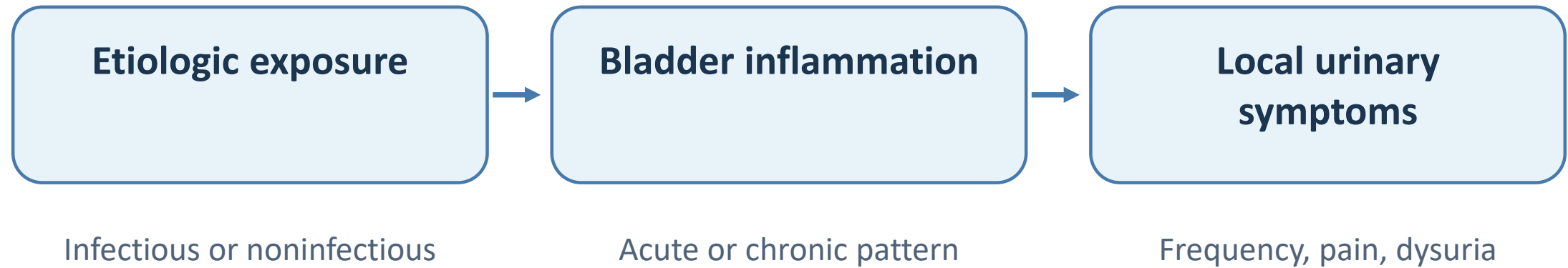
Learning objectives

Interpret bladder inflammation through its cause, tissue reaction and symptoms.

- Identify documented infectious organisms and predisposing conditions.
- Distinguish acute neutrophilic from chronic mononuclear inflammation.
- Relate hemorrhagic, emphysematous and iatrogenic forms to their causes.
- Explain how urinary stasis and ascending infection alter clinical significance.

Cystitis involves the urinary bladder

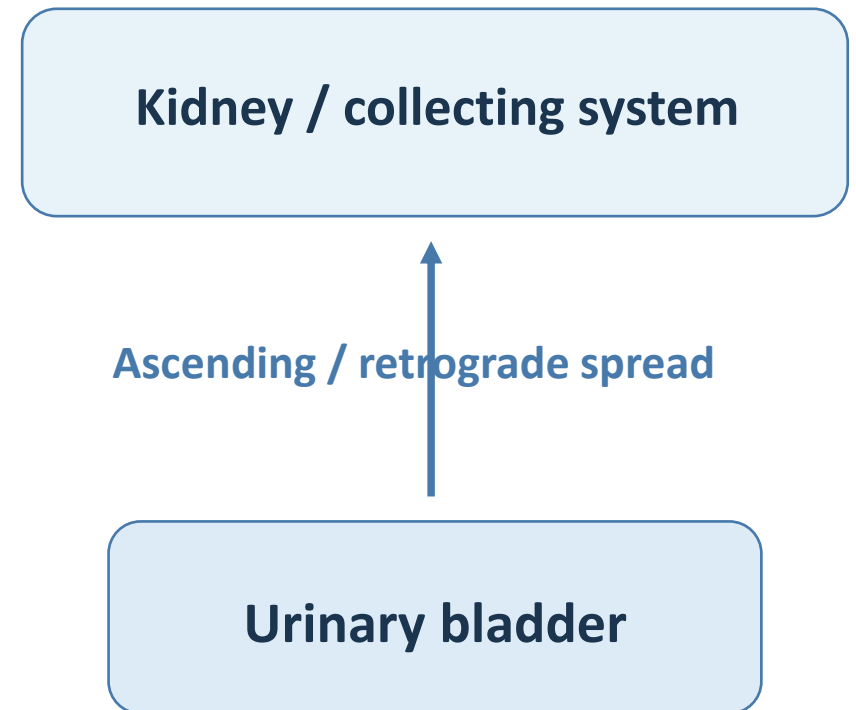
The core lesion is inflammation centered in the bladder.



The bladder is a gateway to the upper tract

Bladder infection may precede bacterial pyelonephritis.

- Bacterial infection can spread retrogradely from bladder toward kidney.
- The consequence extends beyond local symptoms when infection ascends.
- The kidney and collecting system are the downstream sites of spread.



Acute versus chronic: The cellular distinction

Inflammatory cell predominance is the fundamental morphologic contrast.

Acute cystitis

- Hyperemic mucosa
- Neutrophilic inflammatory infiltrate
- Exudate may accompany injury

Chronic cystitis

- Persistence of bacterial infection
- Mononuclear infiltrates
- Chronic inflammatory tissue response

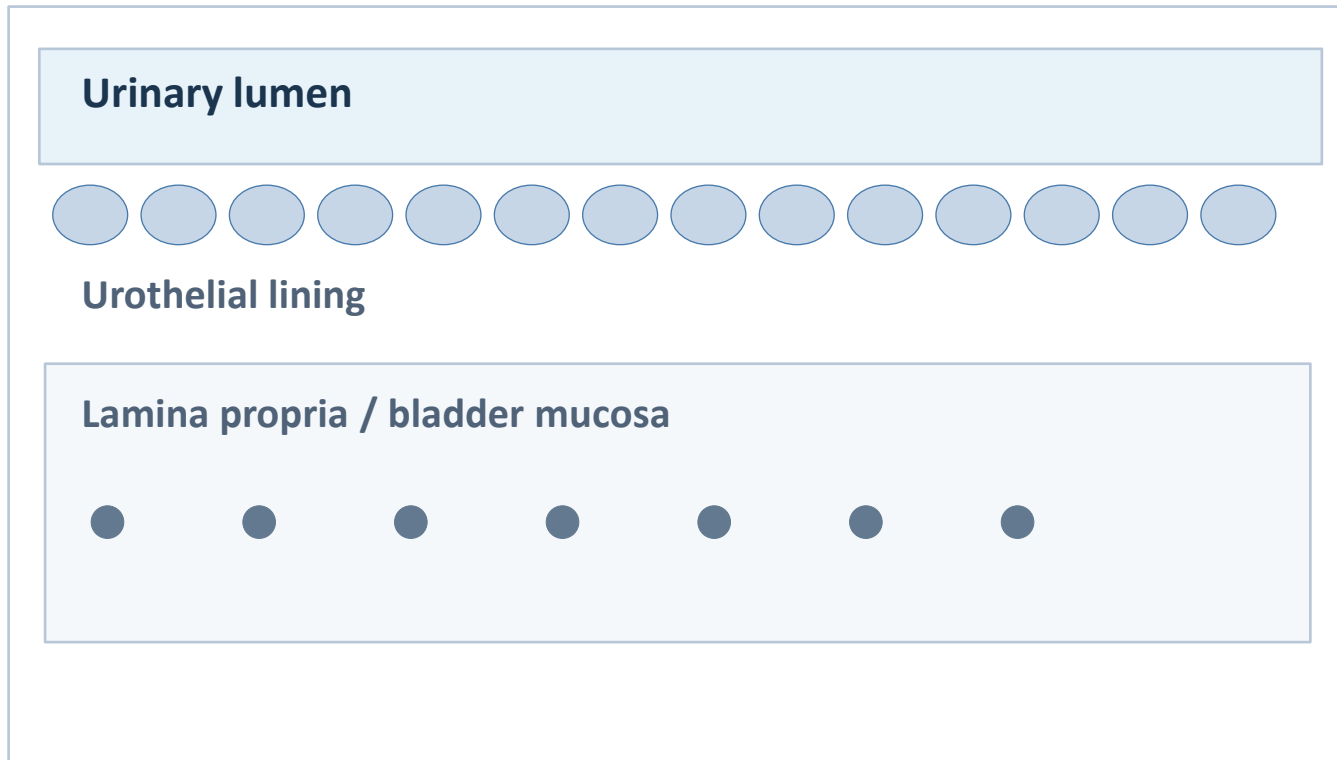
Cause and duration are different axes

Acute/chronic pattern and infectious/noninfectious cause should be assessed separately.

Clinical dimension	Potential states	Source-grounded example
Tissue pattern	Acute chronic	Neutrophilic vs mononuclear
Cause	Infectious noninfectious	Coliforms vs radiation
Visible complication	Hemorrhagic emphysematous	BK virus vs gas-forming bacteria

Bladder wall compartments

The mucosa and underlying wall are sites of the inflammatory reaction.



- The urothelial lining faces the urinary lumen.
- Bladder mucosa can be hyperemic and inflamed.
- Follicular cystitis produces follicles in mucosa and underlying wall.

Conceptual schematic — not a histologic section

Predisposition is multifactorial

Host, flow and procedural factors can facilitate cystitis.

Mechanical

Calculi; Urinary obstruction

Systemic

Diabetes; Immune deficiency

Procedural

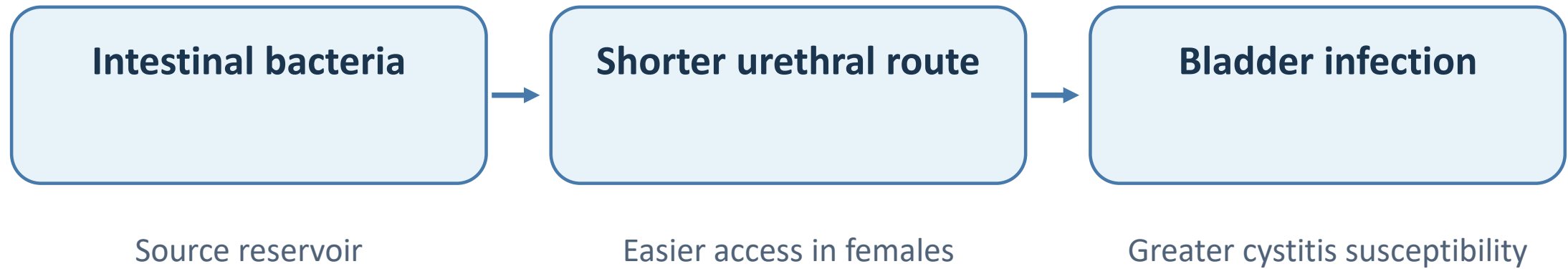
Instrumentation

Anatomic

Shorter female urethra

Female anatomy modifies exposure

A shorter urethra facilitates entry of intestinal bacteria.



Bladder calculi: A structural risk

A calculus is a recognized predisposing lesion.

Predisposing lesion

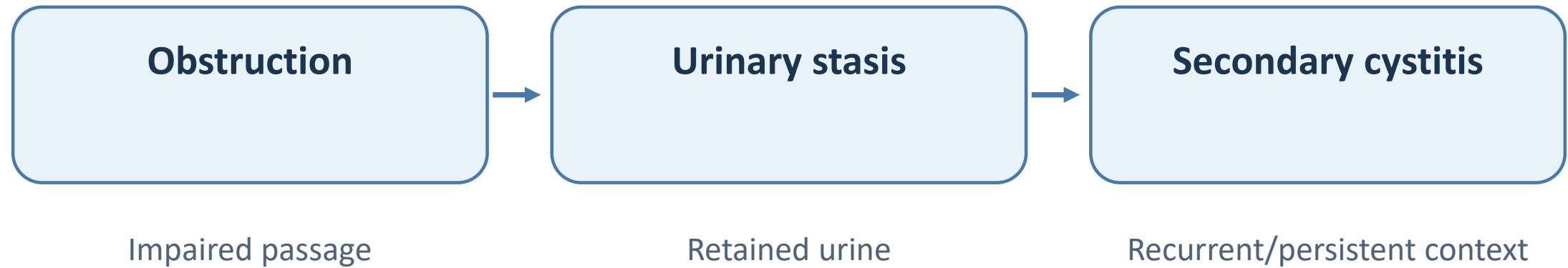
- Bladder calculi appear in the risk-factor list.
- Calculi can coexist with urinary stasis.

Consequence to consider

- Cystitis may be secondary rather than isolated.
- Recognizing the underlying lesion changes clinical interpretation.

Obstruction and urinary stasis

Bladder outflow obstruction can sustain the setting for infection.



Immune deficiency broadens the etiologic context

Impaired host defenses appear among the major risk factors.

Host condition

- Immune deficiency predisposes to cystitis.
- Immunosuppression is noted with fungal cystitis.

Microbial implication

- Consider organisms beyond common coliforms.
- Candida and rarely cryptococcal agents are specifically described.

Risk factors should not be viewed in isolation

Multiple types of predisposition may coexist in one patient.

Domain	Documented factor	Teaching question
Mechanical	Calculus; obstruction	Is urinary stasis present?
Host	Diabetes; immune deficiency	Is susceptibility increased?
Procedural	Instrumentation	Did an intervention precede symptoms?
Anatomic	Shorter female urethra	Is ascending entry easier?

Coliform bacteria dominate infectious cystitis

The source ranks E. Coli first among common coliform causes.

Escherichia coli

Most common

Proteus

Other common agents

**Klebsiella /
Enterobacter**

Additional coliforms

Escherichia coli: Principal named organism

E. Coli is the most common etiologic agent section.

Source statement

- E. Coli heads the listed coliform causes.
- Intestinal organisms have a route to the female urinary bladder.

Diagnostic discipline

- Commonest is not the same as exclusive.
- Morphology is described as nonspecific; Organism identity is not established histologically.

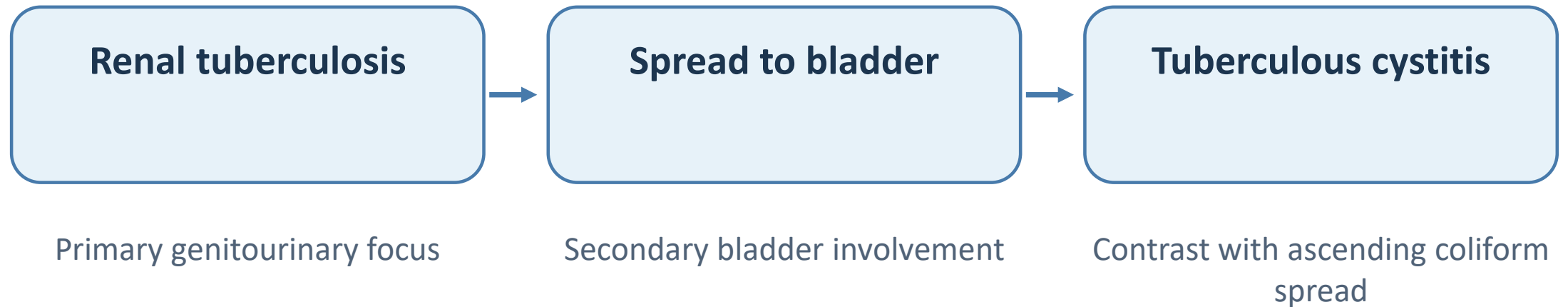
Infectious causes extend beyond coliforms

A bacterial default should not obscure fungal, parasitic and viral causes.

Category	Named agents	Clinical or morphologic context
Coliforms	E. coli, Proteus, Klebsiella, Enterobacter	Common causes
Fungi	Candida albicans; rare cryptococcal agents	Immunosuppression / prolonged antibiotics
Parasites	Schistosoma haematobium	Specific endemic regions
Viruses	Adenovirus, BK virus	May be hemorrhagic
Other	Chlamydia; Mycoplasma	Additional potential causes

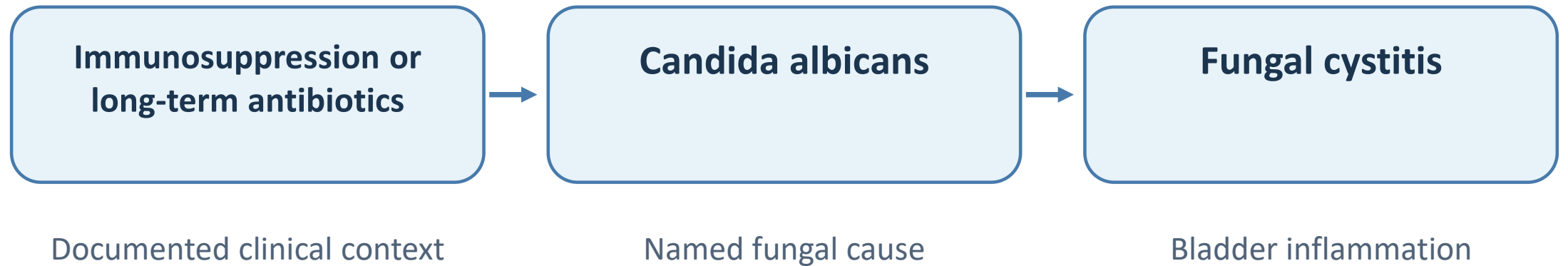
Tuberculous cystitis has a different direction

Bladder tuberculosis is generally a sequel to renal tuberculosis.



Candida cystitis in susceptible hosts

Candida albicans is emphasized in selected clinical settings.



Schistosoma haematobium: Geographic context

Geography changes the differential etiology of cystitis.

Parasite

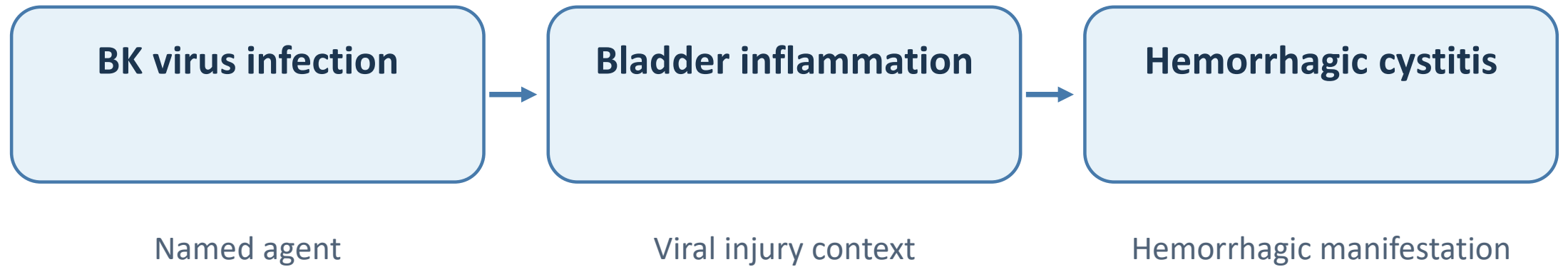
- Schistosoma haematobium can cause bladder inflammation.
- This is a parasitic—not coliform—etiology.

Clinical context

- Important in parts of Africa and the Middle East.
- Exposure context matters in assessing etiology.

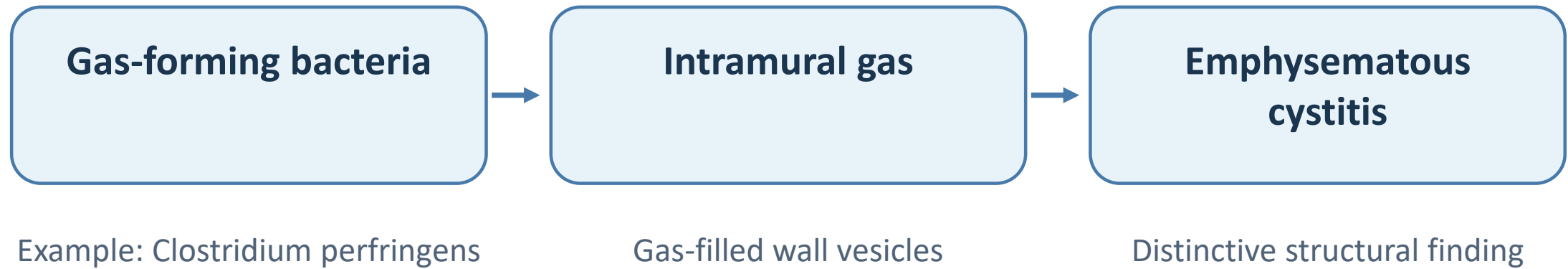
BK virus and hemorrhagic cystitis

BK virus is explicitly associated with hemorrhagic cystitis.



Emphysematous cystitis: Gas in the wall

Gas-forming bacteria can create vesicles within the bladder wall.



Hemorrhagic is a phenotype, not a pathogen

Several mechanistically different exposures can lead to hemorrhagic cystitis.

Cause or exposure	Pathology association	Category
Adenovirus	Hemorrhagic cystitis may occur	Viral
BK virus	Hemorrhagic cystitis may occur	Viral
Cyclophosphamide	May cause hemorrhagic cystitis	Drug-associated

Most cystitis shows nonspecific inflammation

The tissue response alone often does not establish etiology.

Morphologic description

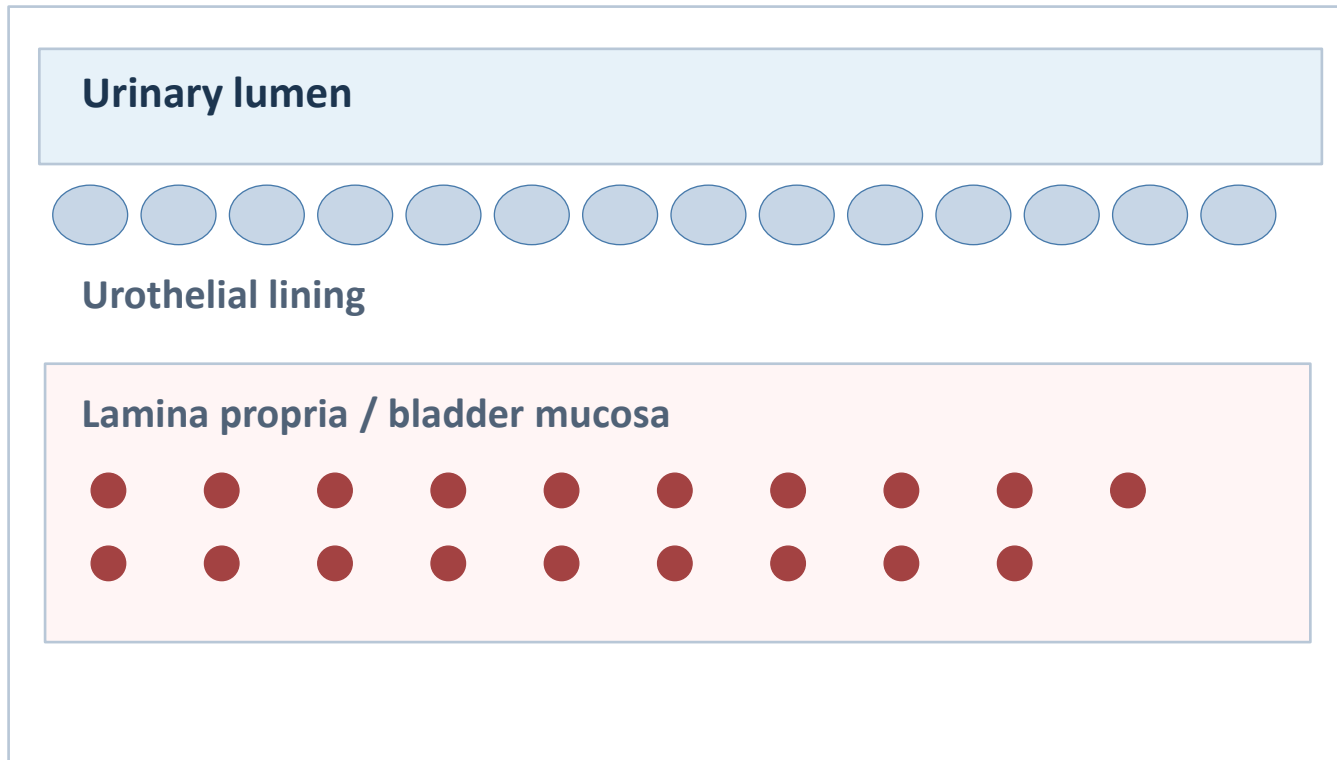
- Acute and chronic inflammation is often nonspecific.
- Etiologic agents can differ despite similar tissue reactions.

Interpretive limit

- Use morphologic pattern to describe injury.
- Integrate the organism and predisposing context separately.

Acute cystitis: Hyperemic mucosa

Vascular congestion is part of the acute morphologic picture.

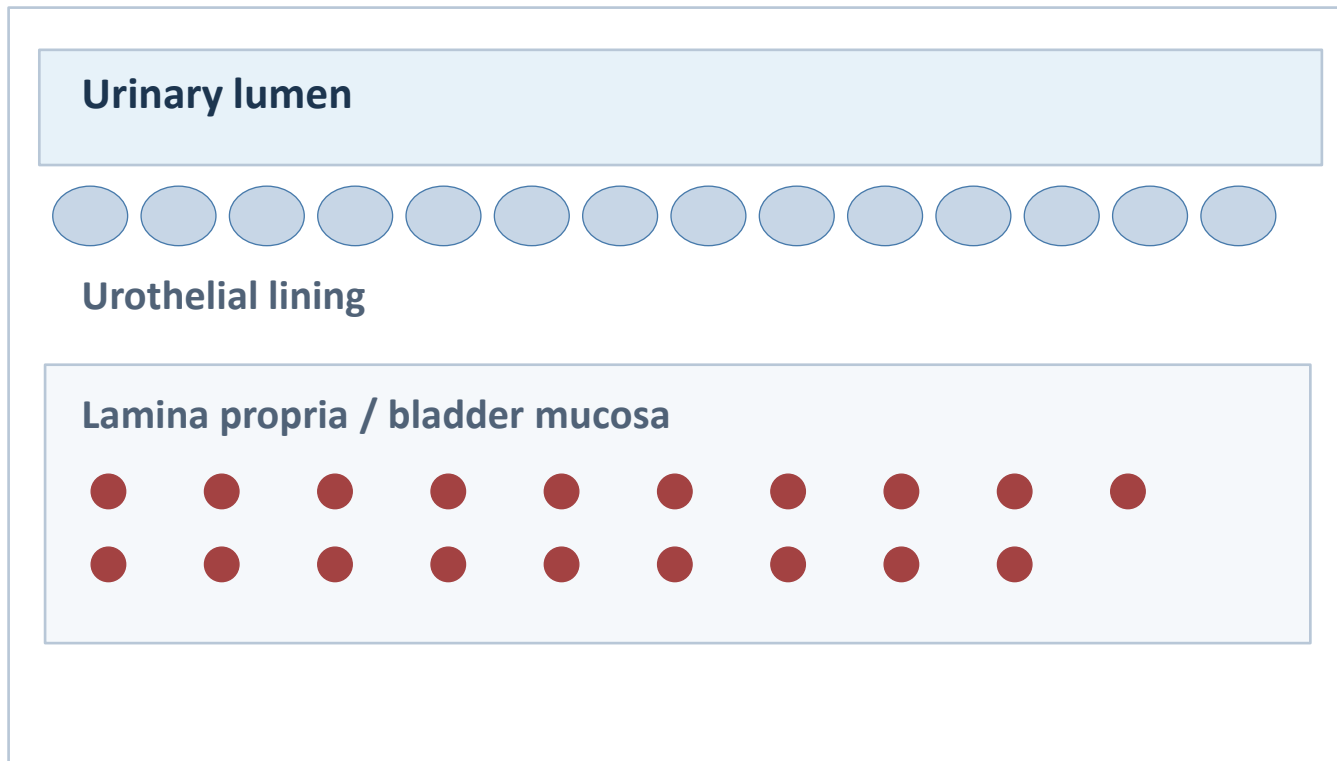


- The bladder mucosa becomes hyperemic.
- This accompanies acute inflammatory infiltration.
- Hyperemia alone is not agent-specific.

Conceptual schematic — not a histologic section

Acute cystitis: Neutrophilic infiltrate

Neutrophils are the signature inflammatory cells in acute disease.

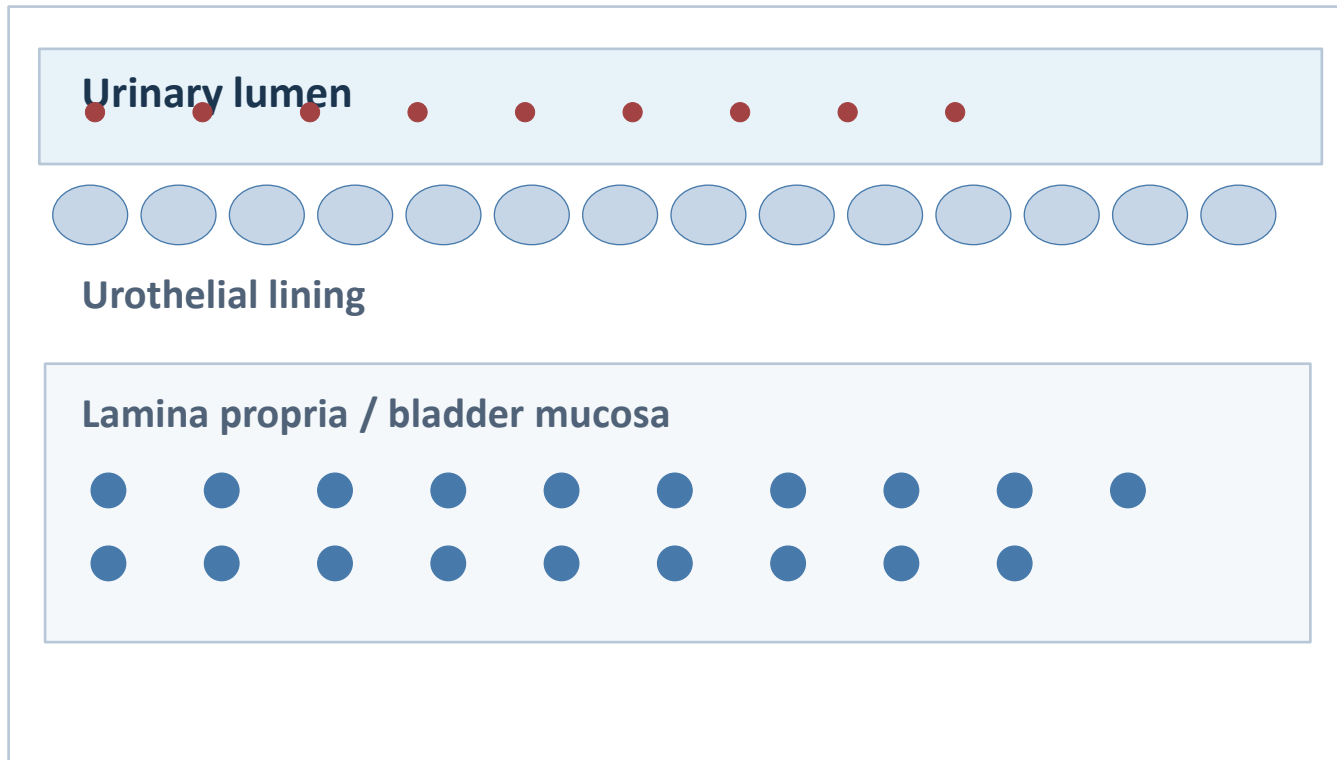


- Neutrophilic cells infiltrate the bladder mucosa.
- This characterizes the acute inflammatory reaction.
- The microscopic description is not linked to a unique bacterium.

Conceptual schematic — not a histologic section

Acute cystitis: An exudate may be present

Some acute cases also show an inflammatory exudate.

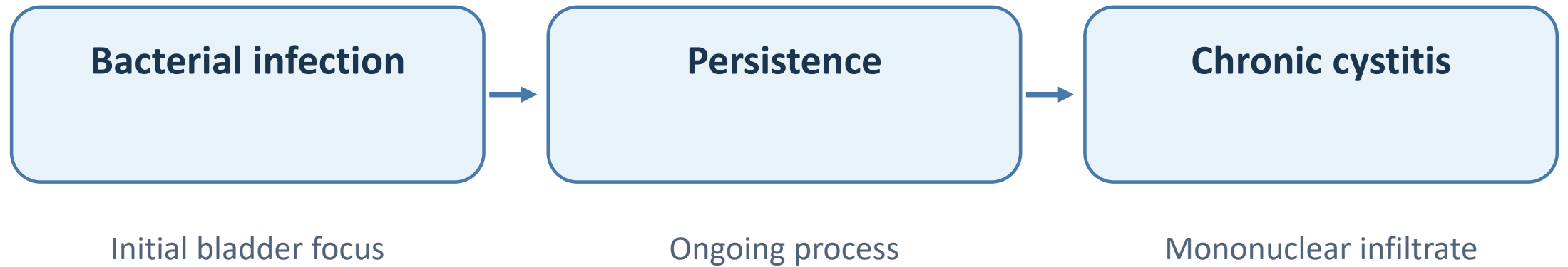


- Exudate is described as an occasional associated feature.
- Its presence does not replace the tissue-based assessment.
- The degree or composition is not quantified in the source.

Conceptual schematic — not a histologic section

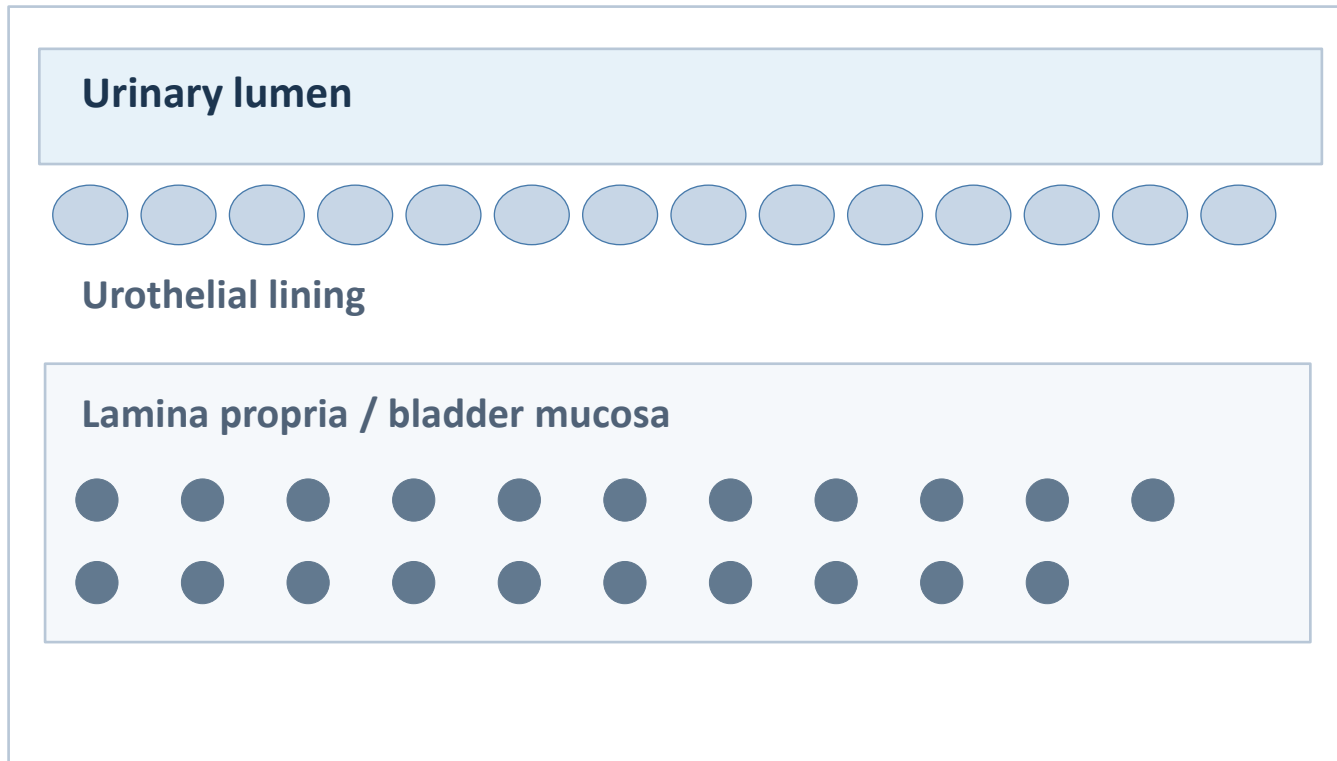
Chronic cystitis follows persistent infection

Persistent bacterial infection can produce a chronic tissue reaction.



Chronic cystitis: Mononuclear infiltrates

The chronic inflammatory pattern is characterized by mononuclear cells.



- Mononuclear inflammatory infiltrates predominate.
- This differs from acute neutrophilic inflammation.
- The description remains etiologically nonspecific.

Conceptual schematic — not a histologic section

Comparing acute and chronic histomorphology

Inflammatory-cell composition provides the clearest source-based distinction.

Feature	Acute cystitis	Chronic cystitis
Mucosal vascular response	Hyperemia described	Not specified
Predominant cells	Neutrophils	Mononuclear cells
Exudate	May be present	Not specified
Etiologic interpretation	Nonspecific	Persistence can lead to chronicity

Interpretation exercise: Cellular phenotype

Microscopic description should precede etiologic speculation.

Hypothetical teaching vignette

A bladder sample is described as showing mucosal hyperemia and a prominent neutrophilic infiltrate.

Questions for interpretation

- Which inflammatory pattern is supported?
- Does that pattern identify E. Coli by itself?
- What associated tissue feature may also be seen?

Interpretation: Acute inflammatory phenotype

Hyperemia and neutrophils support acute cystitis, not a species diagnosis.

- The supported morphologic pattern is acute cystitis.
- Neutrophils are the principal distinguishing inflammatory cells.
- An associated exudate may be present.
- Nonspecific morphology cannot identify the causal organism.

Iatrogenic cystitis: Treatment-related injury

Some noninfectious forms follow medical intervention.

Systemic chemotherapy

Can cause iatrogenic cystitis

Pelvic irradiation

Can cause iatrogenic cystitis

Cyclophosphamide

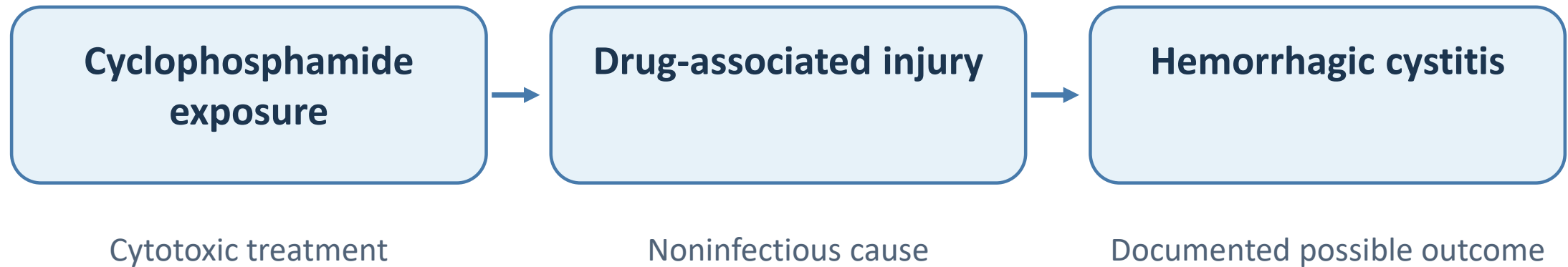
May cause hemorrhagic cystitis

Radiation effect

May be acute or chronic

Cyclophosphamide can produce hemorrhage

A cytotoxic drug may cause hemorrhagic bladder inflammation.



Radiation cystitis: Acute or chronic

Radiation of the bladder region can generate either inflammatory time course.

Acute radiation cystitis

- Follows radiation to bladder region.
- Recognized noninfectious cause.

Chronic radiation cystitis

- Also follows regional irradiation.
- Chronic course is explicitly described.

Radiation and infection are distinct etiologies

Clinical context prevents an automatic assumption of bacterial infection.

Feature	Bacterial cystitis	Radiation cystitis
Initiating factor	Infectious organism	Pelvic irradiation
Time course	May become chronic with persistence	Can be acute or chronic
Inflammatory morphology	Nonspecific	Specific microscopic criteria not provided here
Implication	Evaluate infection setting	Recognize treatment exposure

Follicular cystitis: An architectural pattern

Lymphoid follicles in mucosa and wall define the named variant.



Conceptual schematic — not a histologic section

- Follicular cystitis is a described noninfectious type.
- Lymphoid follicles occur in bladder mucosa and underlying wall.
- The defining feature is their follicular organization.

Eosinophilic cystitis: Source boundary

The textbook section names this variant without providing its detailed morphology.

What is stated

- Eosinophilic cystitis is listed among noninfectious types.
- It is distinct from the acute/chronic nonspecific pattern.

What is not stated here

- No diagnostic threshold for eosinophils is supplied.
- No etiologic, immunophenotypic or treatment criteria are specified.

Infectious and noninfectious: A coherent framework

Etiology and histologic pattern require separate documentation.

Etiologic group	Documented example	Recognizable association
Common bacterial	E. coli / coliforms	Acute or persistent chronic inflammation
Other infectious	BK virus / adenovirus	Hemorrhagic presentation possible
Gas-forming infection	Clostridium perfringens example	Gas vesicles within bladder wall
Iatrogenic	Cyclophosphamide; radiation	Hemorrhagic / acute or chronic injury
Named morphologic pattern	Follicular cystitis	Lymphoid follicles in bladder wall

The clinical presentation has three core elements

The source presents frequency, suprapubic pain and dysuria as the symptom triad.

Frequency

Frequent voiding

Suprapubic pain

Lower abdominal /
bladder region

Dysuria

Pain or burning with
urination

Urinary frequency can be marked in acute disease

The source notes extremely frequent voiding in some acute cases.

Clinical observation

- In acute cystitis, urination may be required every 15–20 minutes.
- Frequency is one of the three classical symptoms.

Interpretation

- This is an illustrative severe pattern, not a diagnostic threshold.
- Frequency alone does not identify a causal organism.

Dysuria describes pain during urination

Dysuria is a symptom, not a histologic diagnosis.

Patient-experienced symptom

- Pain or burning on urination.
- One component of the symptom triad.

Pathology context

- Occurs across forms of cystitis.
- Requires correlation with the lesion and associated risk factors.

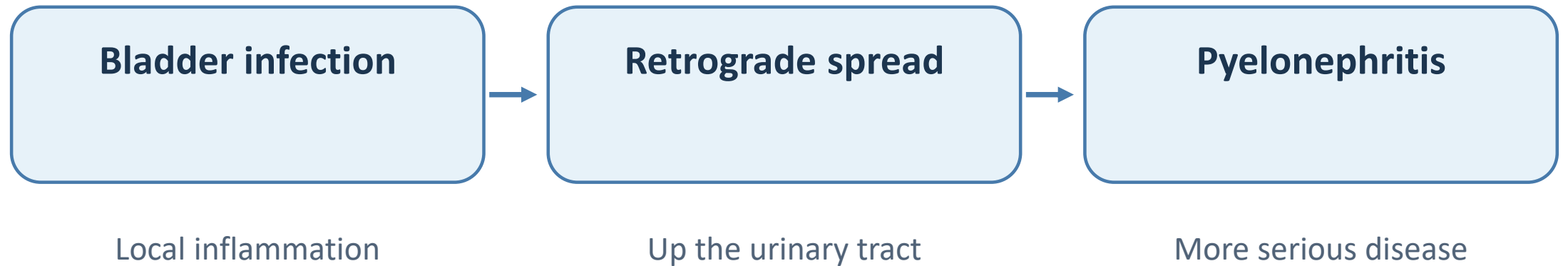
Symptoms do not uniquely identify the cause

The same symptom grouping can accompany several forms of cystitis.

Presentation or finding	Can be associated with	Not established from finding alone
Frequency + suprapubic pain + dysuria	Various forms of cystitis	Specific organism
Hemorrhagic bladder inflammation	Virus or cyclophosphamide	One exclusive mechanism
Chronic tissue infiltrate	Persistent infection	Which bacterium
Bladder-wall gas	Gas-forming bacteria	A full species diagnosis

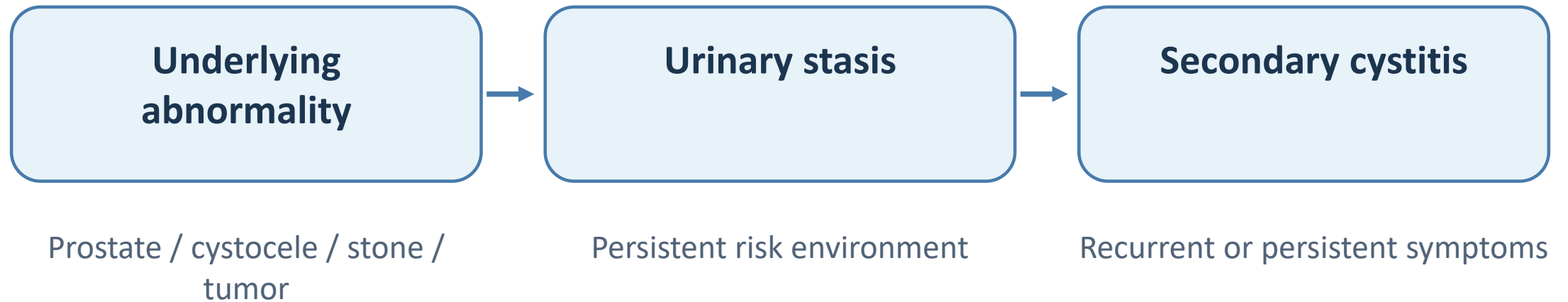
Cystitis may precede a more serious infection

Local bladder symptoms should not obscure possible renal extension.



Urinary stasis can make cystitis secondary

An underlying cause of stasis may need attention to resolve inflammation.



Prostatic hypertrophy may underlie cystitis

In a patient with obstruction, consider a primary prostatic cause.

Primary disorder

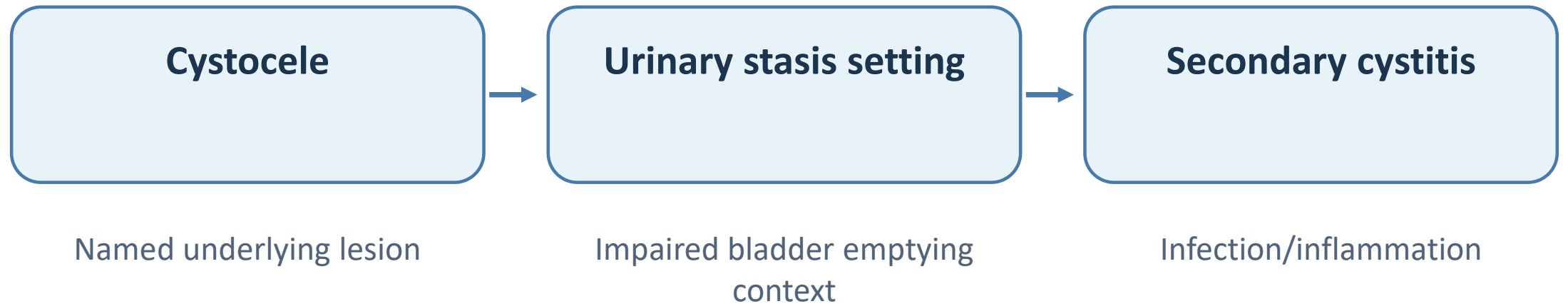
- Prostatic hypertrophy is a source-listed stasis association.
- This is a structural cause of urinary outflow difficulty.

Secondary consequence

- Cystitis may occur as a complication.
- Correcting the primary lesion may be required for relief.

Cystocele is another stasis-associated lesion

Distortion of bladder support may coexist with secondary cystitis.



Stones and bladder neoplasms are underlying lesions

Cystitis may be secondary to either a calculus or a bladder tumor.

Bladder calculus

- Listed among predisposing factors.
- Also named among lesions associated with urinary stasis.

Bladder neoplasm

- Listed as a possible underlying cause.
- Its presence should not be obscured by cystitis symptoms.

Treating cystitis requires a causal viewpoint

Persistent structural predisposition can limit relief of cystitis.



Clinical case: Acute lower-tract presentation

Apply symptoms and predisposition before naming an organism.

Hypothetical teaching vignette

A woman presents with frequent urination, suprapubic discomfort and burning on voiding. The clinical question is whether this represents bladder-centered inflammation.

Questions for interpretation

- Which three symptoms map to the source-based triad?
- What anatomic factor increases susceptibility?
- Which common bacterial group is highlighted?

Case interpretation: Symptom + host context

The triad points toward cystitis; Anatomy affects susceptibility.

- Frequency, suprapubic pain and dysuria form the characteristic triad.
- A shorter female urethra facilitates ascending access of intestinal bacteria.
- Coliforms, especially E. Coli, are common etiologic agents.
- None of these facts by itself confirms species identity.

Clinical case: Persistence with urinary stasis

An underlying lesion may explain why bladder inflammation persists.

Hypothetical teaching vignette

An older patient with known prostatic hypertrophy reports repeated bladder-region discomfort and frequent urination. A prior bladder sample showed chronic mononuclear inflammation.

Questions for interpretation

- Which predisposing mechanism is plausible?
- What microscopic pattern is described?
- Why might attention to the primary lesion be important?

Case interpretation: Chronic cystitis is secondary

Persistent inflammation may reflect a correctable structural problem.

- Prostatic hypertrophy is a named lesion associated with urinary stasis.
- Mononuclear infiltration supports a chronic inflammatory pattern.
- The primary stasis-associated lesion may need correction for relief.
- The lesion does not establish an organism without separate evidence.

Clinical case: Hemorrhagic cystitis

Different causes can converge on a hemorrhagic phenotype.

Hypothetical teaching vignette

A patient develops bladder inflammation with bleeding after exposure to a cytotoxic anticancer drug. An alternative patient with hemorrhagic cystitis has a viral infection.

Questions for interpretation

- Which drug is explicitly named in the source?
- Which two viruses may produce this pattern?
- Why does hemorrhage alone not establish the cause?

Case interpretation: Mechanism precedes label

Hemorrhage is a shared manifestation across etiologies.

- Cyclophosphamide may cause hemorrhagic cystitis.
- Adenovirus and BK virus may also produce hemorrhagic cystitis.
- Drug-associated and viral injury are different causal categories.
- Exposure and infection context are therefore essential.

Integrated clinicopathologic matrix

An etiologic hypothesis should fit susceptibility, morphology and clinical setting.

Question	Core source-based distinction	Example
Who is susceptible?	Anatomic, structural, host or procedural factor	Obstruction; immunosuppression
What is the cause?	Coliform vs other agent vs iatrogenic	E. coli; BK virus; radiation
How does tissue respond?	Acute neutrophilic vs chronic mononuclear	Hyperemia; mononuclear infiltrates
What is the phenotype?	Triad, hemorrhage, intramural gas	Dysuria; emphysematous cystitis
What sustains disease?	Primary lesion creating stasis	Prostatic hypertrophy; calculus

Core pathology concepts

Cystitis is a clinicopathologic process—not a single organism or morphologic lesion.

- Common cystitis is coliform-associated; E. Coli is the leading named agent.
- Acute inflammation shows hyperemia, neutrophils and sometimes exudate.
- Persistent infection may produce chronic mononuclear inflammation.
- The symptom triad is frequency, suprapubic pain and dysuria.
- Ascending infection and untreated urinary stasis are major clinical concerns.

Primary reference and scope

Source-based lecture; no externally updated diagnostic or treatment guidelines are incorporated.

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition (2026). Chapter 21: The Lower Urinary Tract and Male Genital System. “Acute and Chronic Cystitis,” printed page 869.
- The next textbook sections separately address interstitial cystitis / malakoplakia and metaplastic lesions; these are not taught as main entities here.