

Urinary Bladder **Inflammation**

Cystitis: causes, tissue response, and clinicopathologic significance

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraisakhri.com

Learning objectives

- Distinguish infectious from noninfectious inflammatory disorders of the bladder.
- Correlate acute and chronic cystitis with characteristic tissue responses.
- Recognize the clinicopathologic features of interstitial cystitis and malakoplakia.
- Explain why urinary stasis and ascending infection affect disease persistence.

Bladder inflammation comprises several disease patterns

Infectious cystitis

Bacteria predominate;
Several viral, fungal, and
parasitic causes are
recognized.

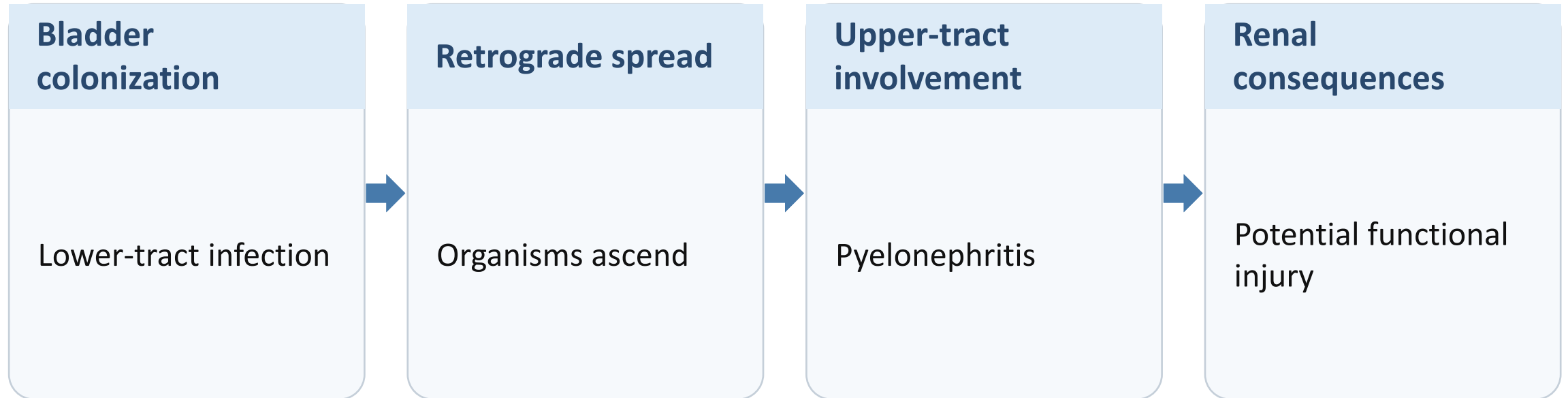
Noninfectious injury

Iatrogenic, radiation-
associated, follicular and
eosinophilic forms occur.

Distinctive disorders

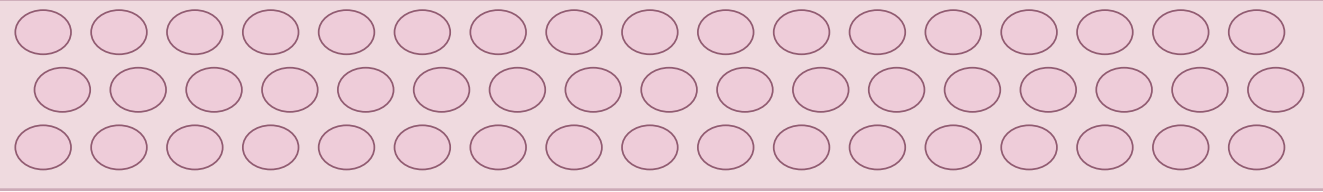
Bladder pain syndrome and
malakoplakia have special
clinical or morphologic
profiles.

Why bladder infection has consequences beyond the bladder

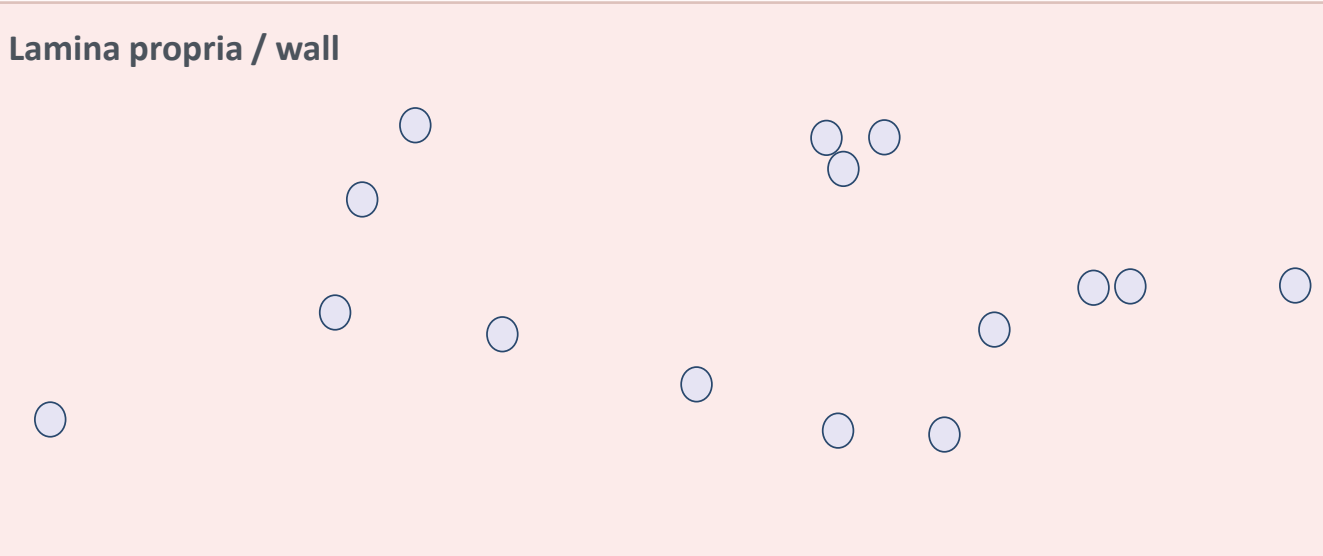


A simplified map of bladder-wall inflammatory responses

Urothelium



Lamina propria / wall



SCHEMATIC — not a histologic specimen

Surface urothelium

Exposed mucosa

Underlying wall

Site of inflammatory infiltrate

Persistence

Changes cellular composition

Etiology and morphology are separate classification axes

Cause / context	Representative phenotype
Coliform bacterial infection	Acute or persistent chronic cystitis
Systemic chemotherapy / irradiation	Iatrogenic inflammatory or hemorrhagic injury
Chronic infection with phagocyte dysfunction	Malakoplakia
Unexplained bladder-associated pain syndrome	Interstitial cystitis / bladder pain syndrome

Predisposing factors increase the likelihood of cystitis

Impaired drainage

Obstruction and urinary stasis

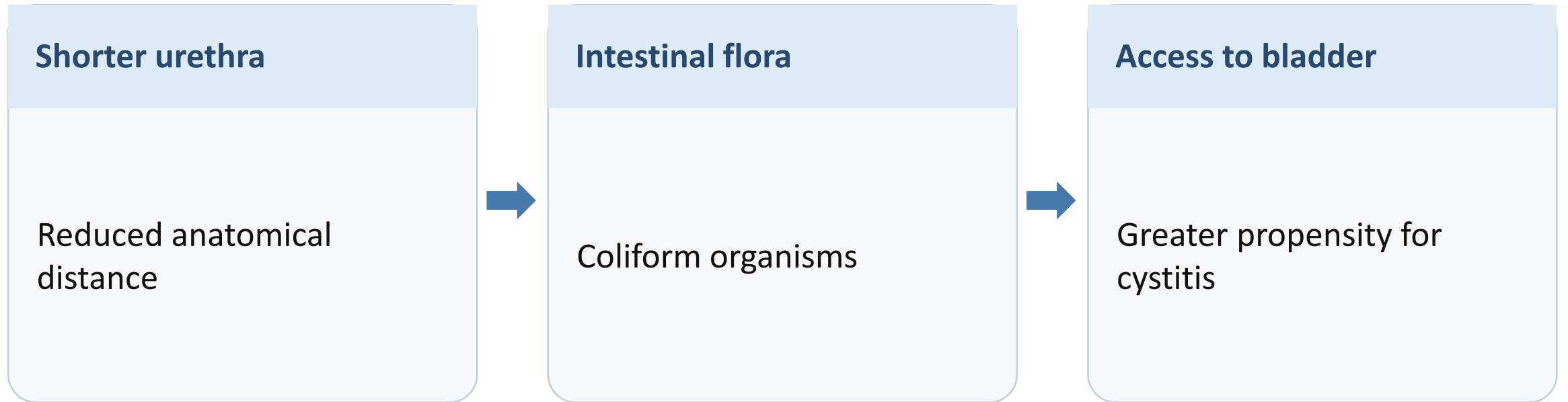
Local perturbation

Calculi and instrumentation

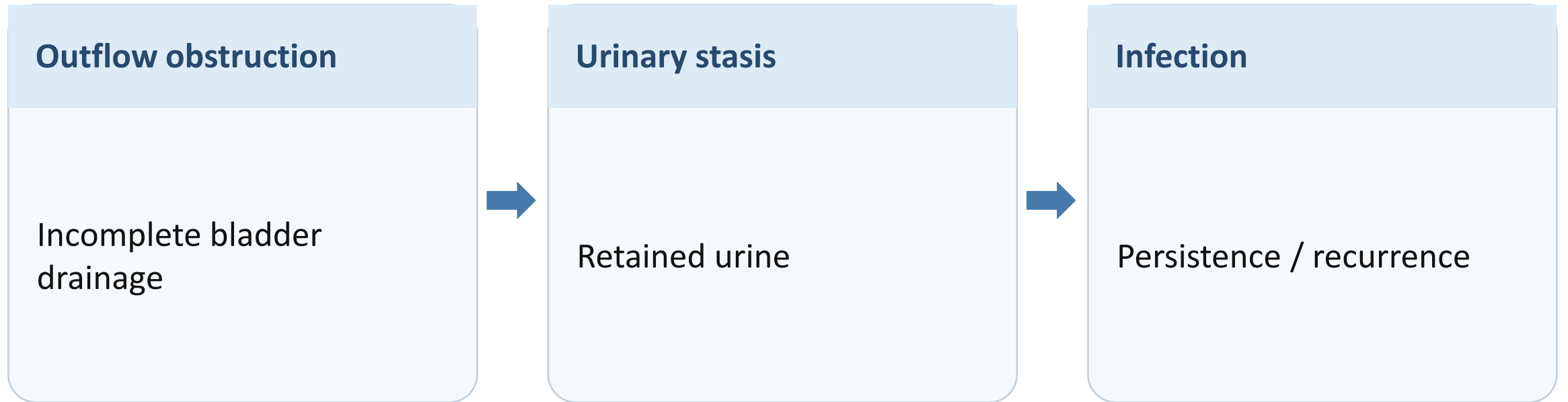
Host factors

Diabetes mellitus and
immune deficiency

Female anatomy favors access of enteric bacteria



Urinary outflow obstruction creates a persistent setting



Benign prostatic enlargement may underlie cystitis

Primary process

Prostatic hypertrophy can cause urinary outflow obstruction.

Bladder effect

A residual, poorly emptied bladder favors urinary stasis.

Interpretive lesson

Cystitis may be secondary rather than the primary disorder.

Cystocele and bladder calculi create mechanical risk

Cystocele

An anatomic abnormality associated with urinary stasis.

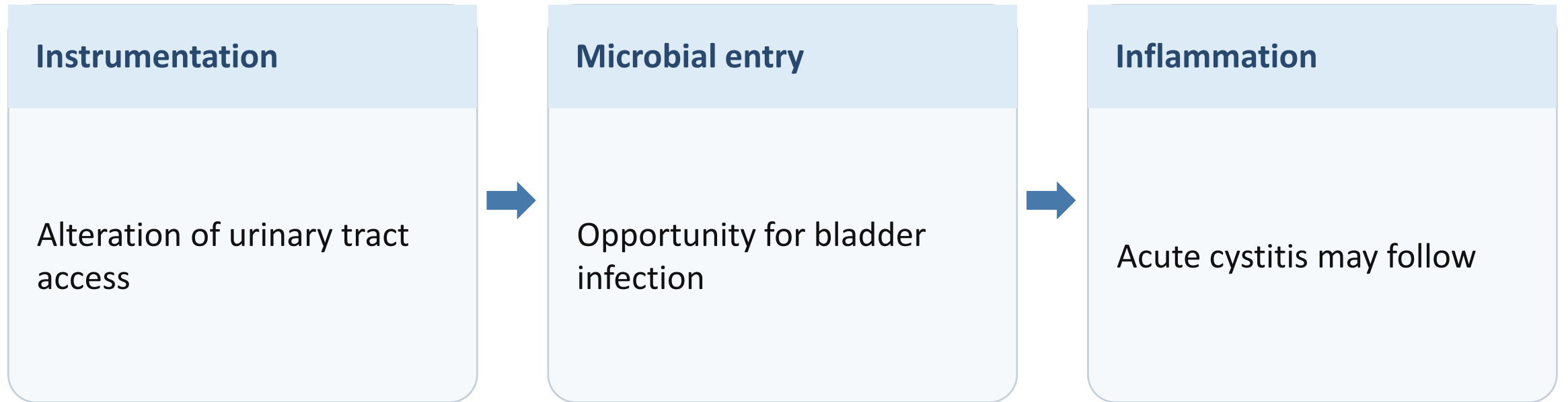
Calculi

Listed both as a predisposing factor and as a primary lesion.

Practical significance

A structural abnormality can explain repeated inflammatory episodes.

Instrumentation is a distinct predisposing factor



Host conditions modify infectious susceptibility

Diabetes mellitus

A recognized predisposing condition for cystitis.

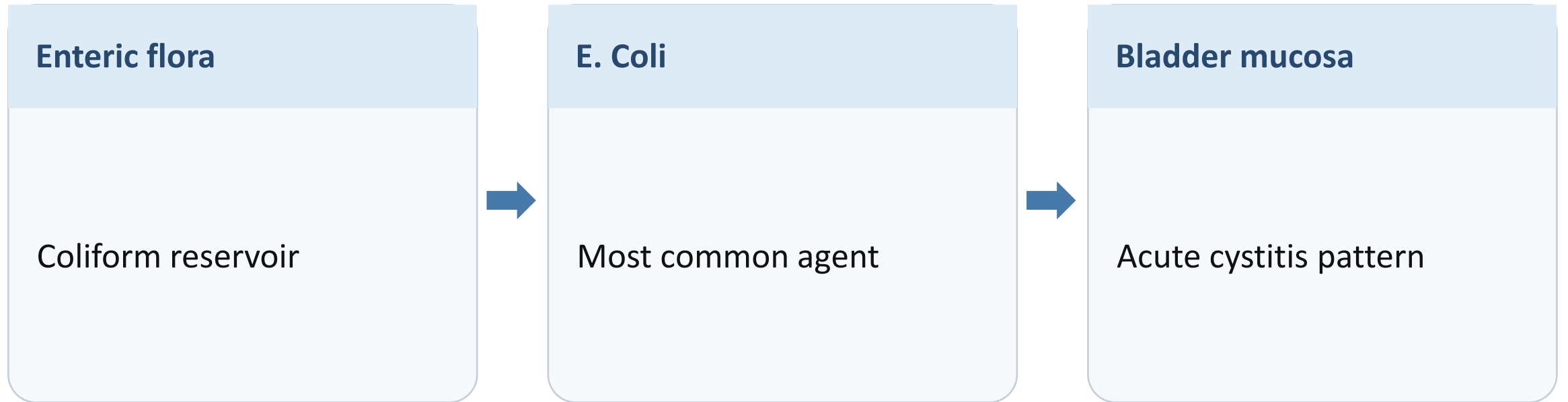
Immune deficiency

Broadens the range of plausible pathogens.

Prolonged antibiotics

A context in which fungal cystitis can be encountered.

Escherichia coli leads the common bacterial causes



Other coliform organisms must remain in the differential

Proteus

Named among common etiologic agents after E. Coli.

Klebsiella

Another important coliform pathogen.

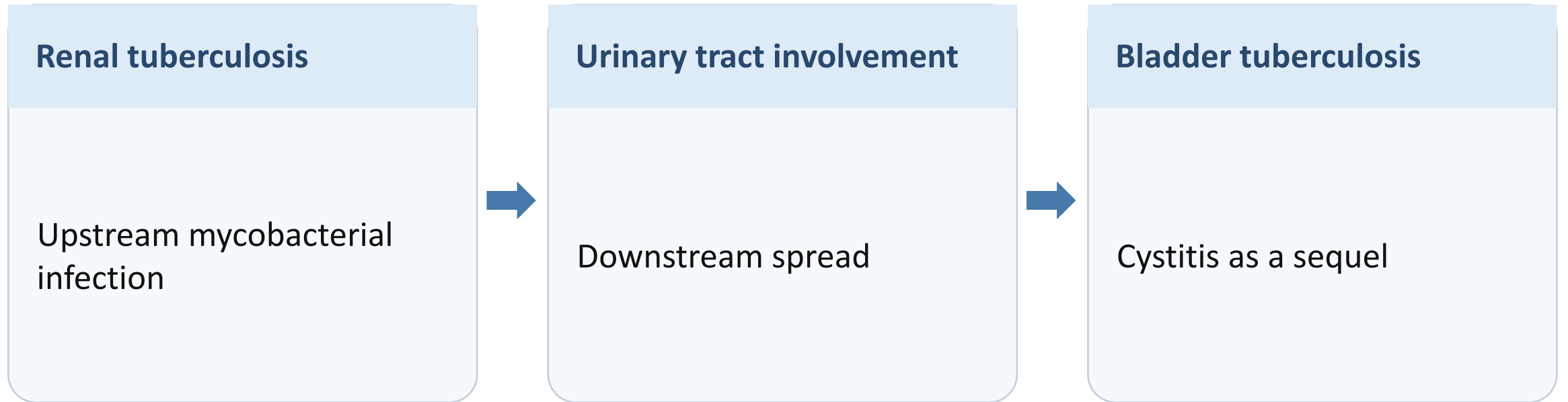
Enterobacter

Included in the common bacterial spectrum.

Non-coliform and unusual infections are recognized

Category	Representative infectious agents
Mycobacterial	Tuberculosis
Fungal	Candida albicans; rarely Cryptococcus
Parasitic	Schistosoma haematobium
Viral	Adenovirus; BK virus
Other infectious agents	Chlamydia; Mycoplasma

Tuberculous cystitis usually follows renal tuberculosis



Candida cystitis has particular host associations

Etiologic agent

Candida albicans is specifically cited.

Host setting

Immunosuppression increases plausibility.

Antibiotic context

Long-term antibiotics are another setting highlighted.

Schistosoma haematobium is geographically relevant

Organism

Schistosoma haematobium

Geography

Important in certain African and Middle Eastern countries

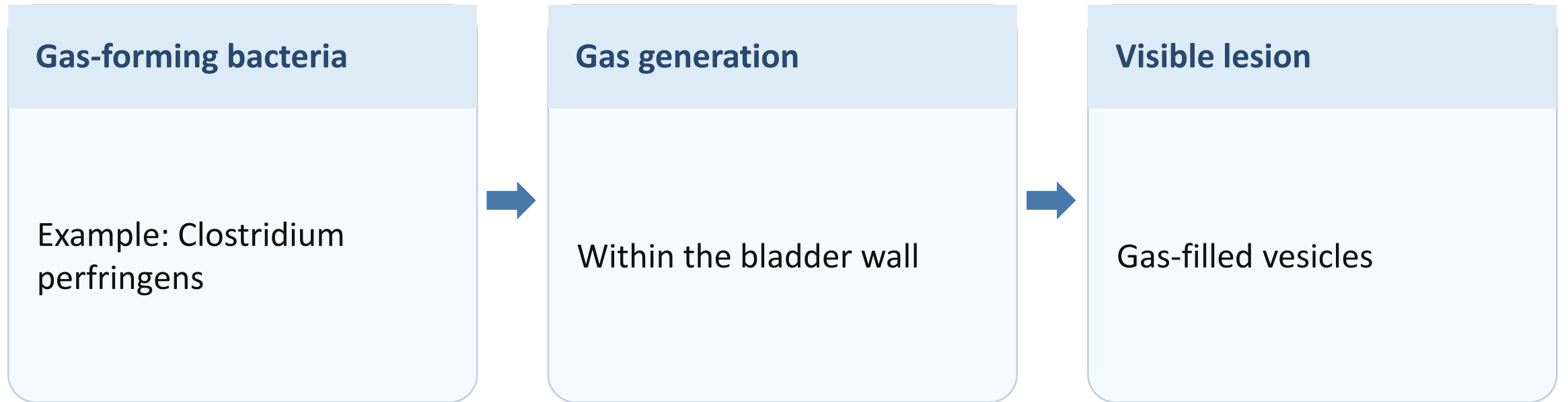
Pathology implication

A parasitic etiology belongs in the infectious differential

Viral infections can produce hemorrhagic cystitis



Emphysematous cystitis contains gas in the bladder wall

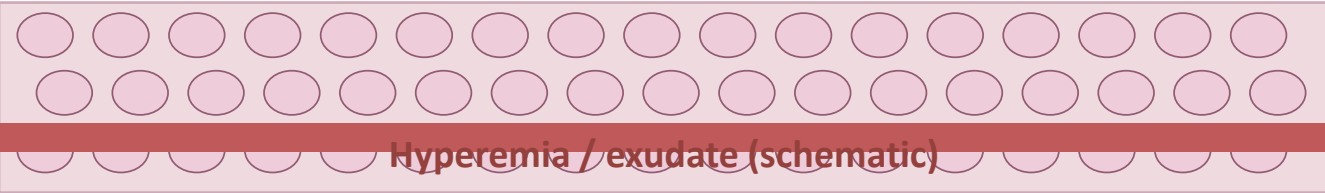


Hemorrhagic cystitis has infectious and treatment-related causes

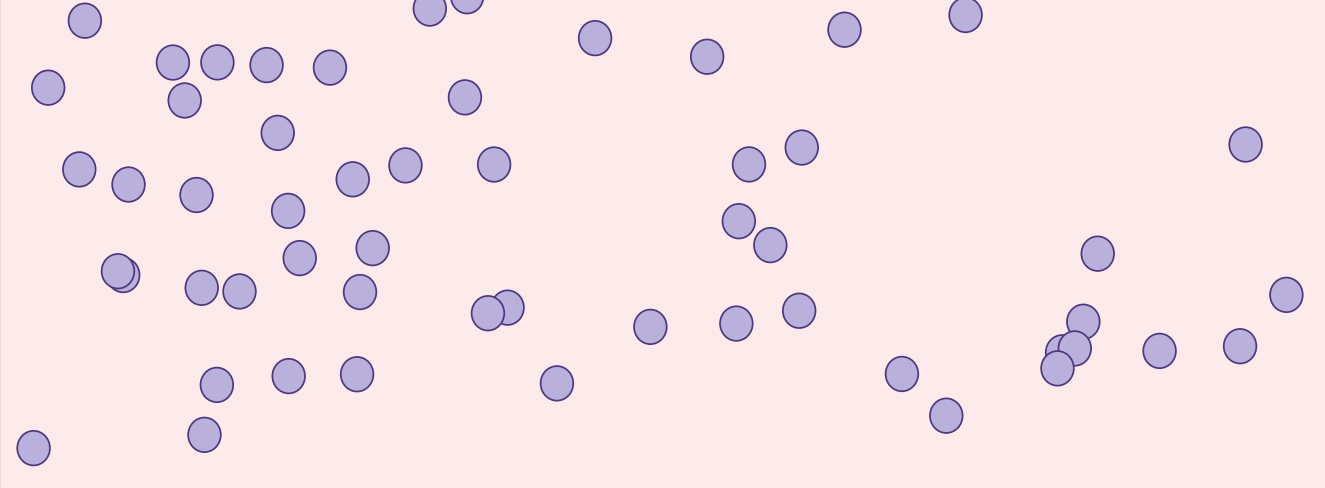
Mechanism	Representative associations
Viral injury	Adenovirus; BK virus
Chemotherapy-related injury	Cyclophosphamide
Other noninfectious damage	Radiation-related bladder injury is also recognized

Acute cystitis begins with mucosal hyperemia

Urothelium



Lamina propria / wall



SCHEMATIC — not a histologic specimen

Dominant change

Hyperemic mucosa

Inflammatory response

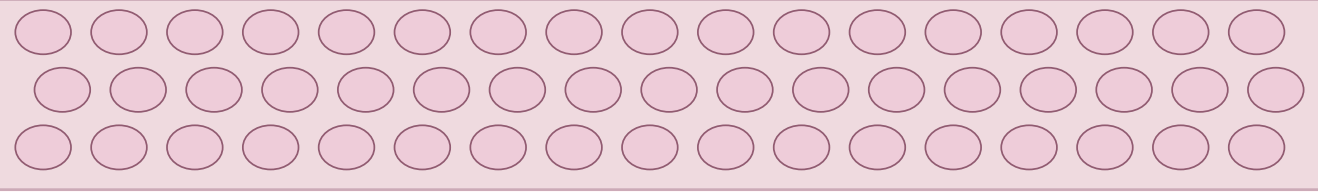
Neutrophil recruitment

Possible additional finding

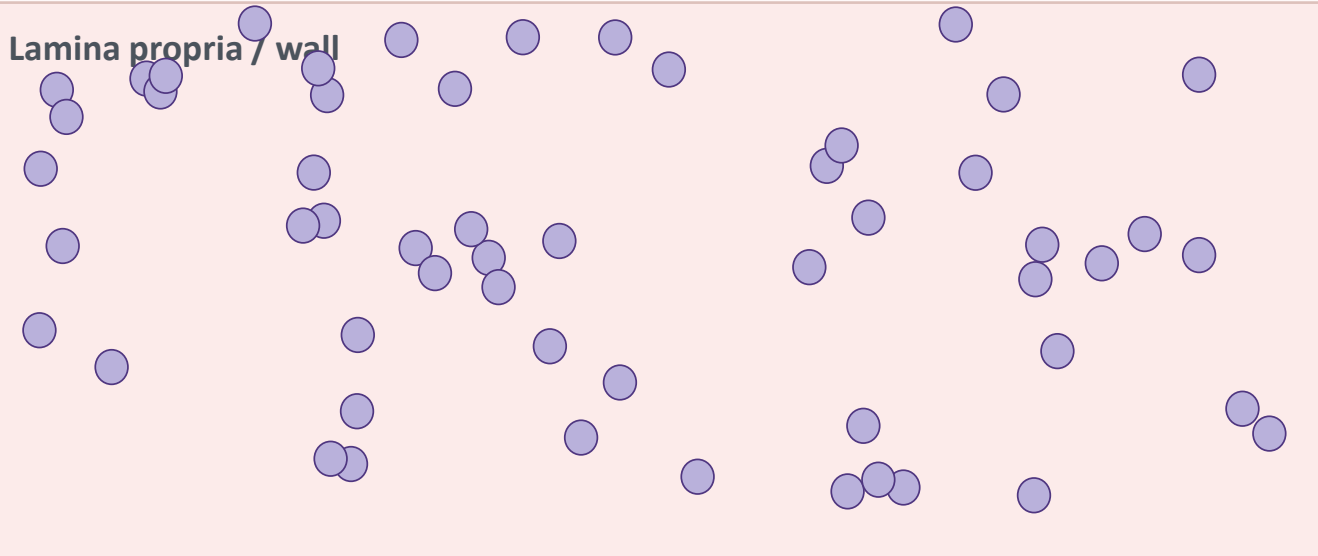
Surface exudate

Neutrophils are the principal acute inflammatory cells

Urothelium



Lamina propria / wall



SCHEMATIC — not a histologic specimen

Tissue finding

Neutrophilic infiltrate

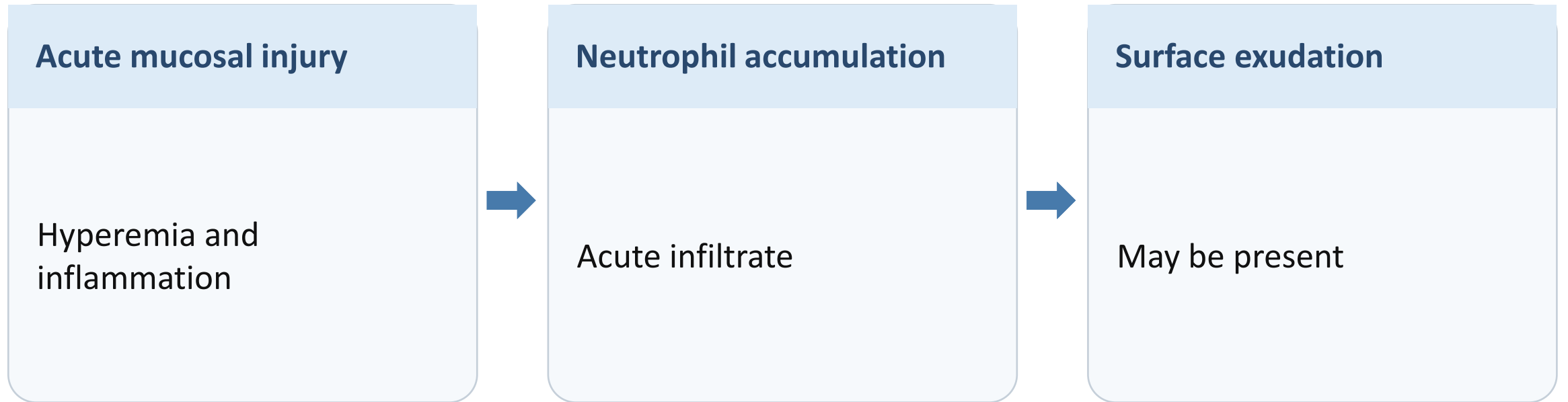
Interpretation

Acute inflammatory response

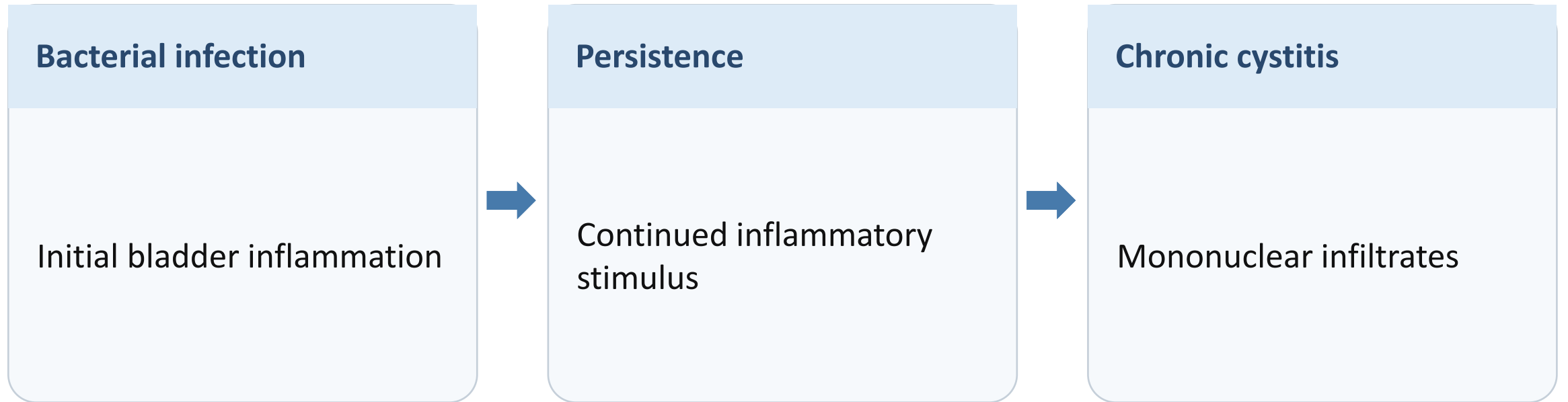
Etiology caveat

Neutrophils alone do not speciate a pathogen

Exudate may accompany acute mucosal inflammation

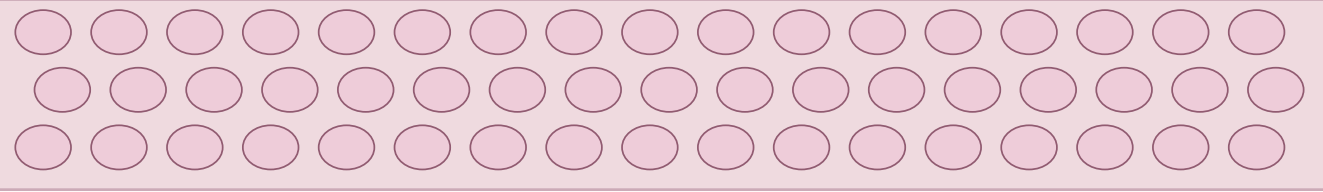


Persistent infection may yield chronic cystitis

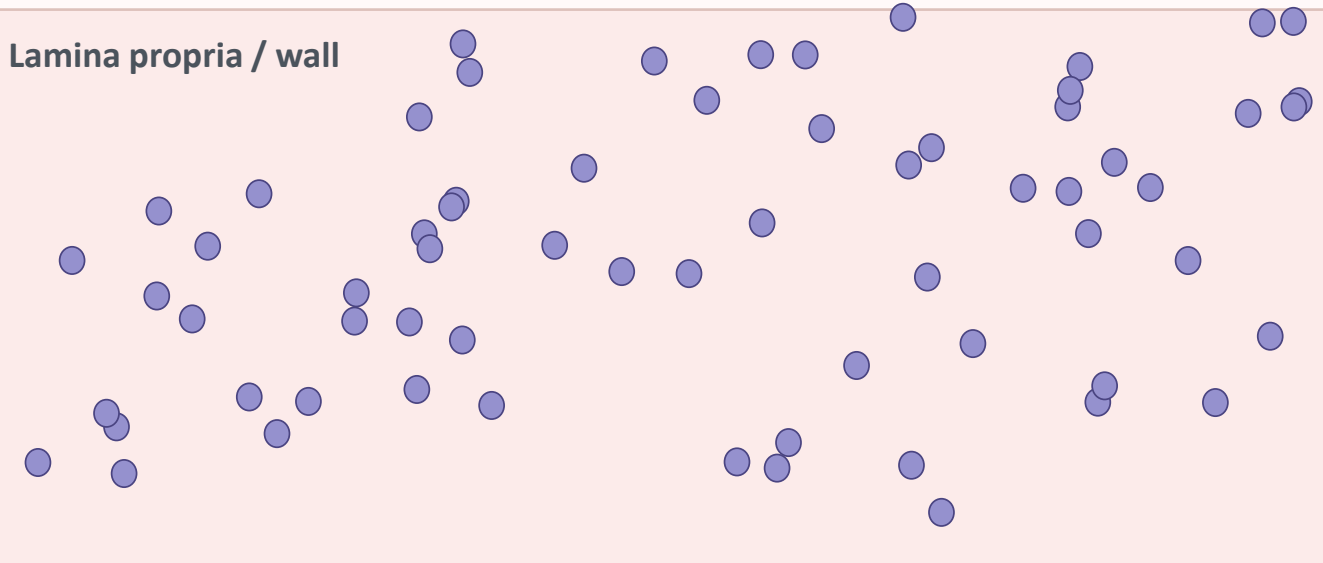


Chronic cystitis is associated with mononuclear cells

Urothelium



Lamina propria / wall



SCHEMATIC — not a histologic specimen

Dominant infiltrate

Mononuclear inflammatory cells

Setting

Persistent bladder inflammation

Limit

Often histologically nonspecific

Acute and chronic histologic patterns differ

Feature	Acute cystitis	Chronic cystitis
Key cellular infiltrate	Neutrophils	Mononuclear cells
Mucosal appearance	Often hyperemic	Variable / nonspecific
Associated exudate	May occur	Not defining in the text
Typical underlying context	Recent injury or infection	Persistent inflammatory stimulus

The microscopy of most cystitis is nonspecific

Acute pattern

Neutrophils and vascular congestion support acute injury.

Chronic pattern

Mononuclear cells support persistent inflammation.

Diagnostic limit

The pattern may not identify the causal agent by itself.

Bladder inflammation produces a characteristic symptom triad

Frequency

Repeated urge to void

Suprapubic pain

Pain over the bladder region

Dysuria

Pain or burning on urination

Urinary frequency is the dominant functional complaint

Mucosal inflammation

Irritative lower urinary tract response



Frequent voiding

May be very frequent in acute cases



Clinical burden

Interruption of normal daily activity

Dysuria localizes discomfort to urination

What patients describe

Burning or painful urination

Clinical context

Common alongside increased frequency

Interpretive limit

Dysuria does not establish a single microorganism

Suprapubic pain identifies bladder-region discomfort

Pain location

Lower abdomen / suprapubic region

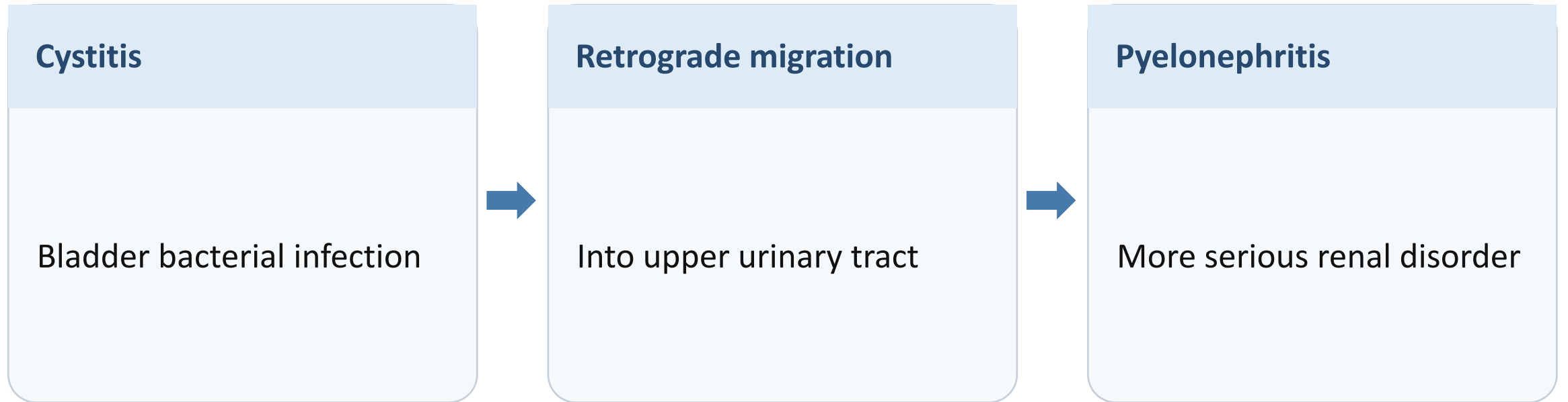
Inflammatory context

Occurs in cystitis of various causes

Comparison

Pain is prominent in bladder pain syndrome as well

The major ascending complication is pyelonephritis



Recurrent cystitis can be a marker of an underlying lesion

Outflow obstruction

Prostatic hypertrophy

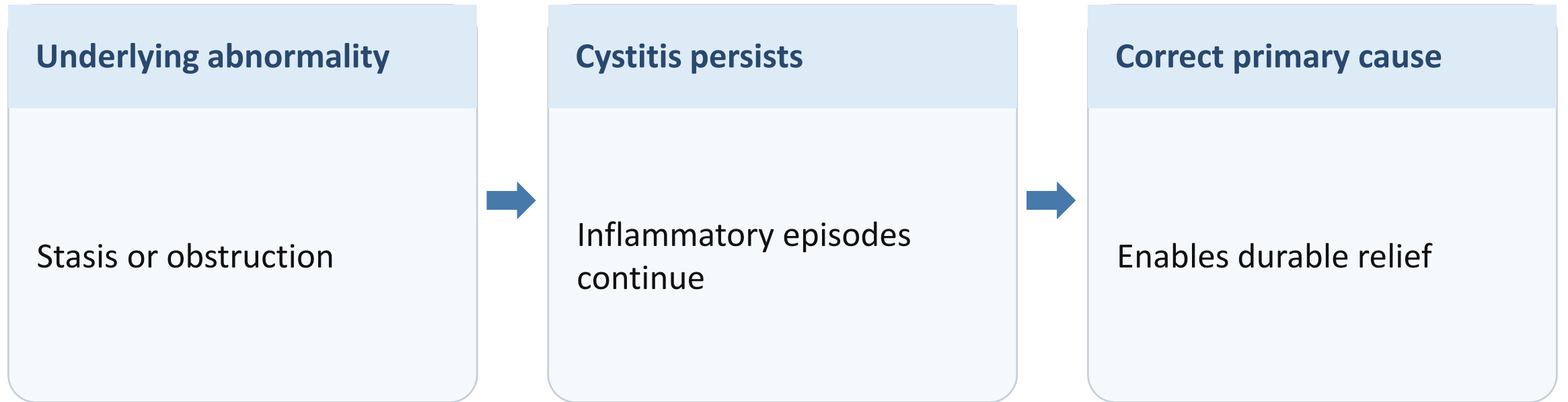
Anatomic displacement

Cystocele

Local pathological lesions

Calculi or bladder neoplasms

Addressing the primary lesion matters for resolution



Bladder inflammation is not always microbial

Iatrogenic

Systemic chemotherapy and pelvic irradiation

Organized lymphoid response

Follicular cystitis

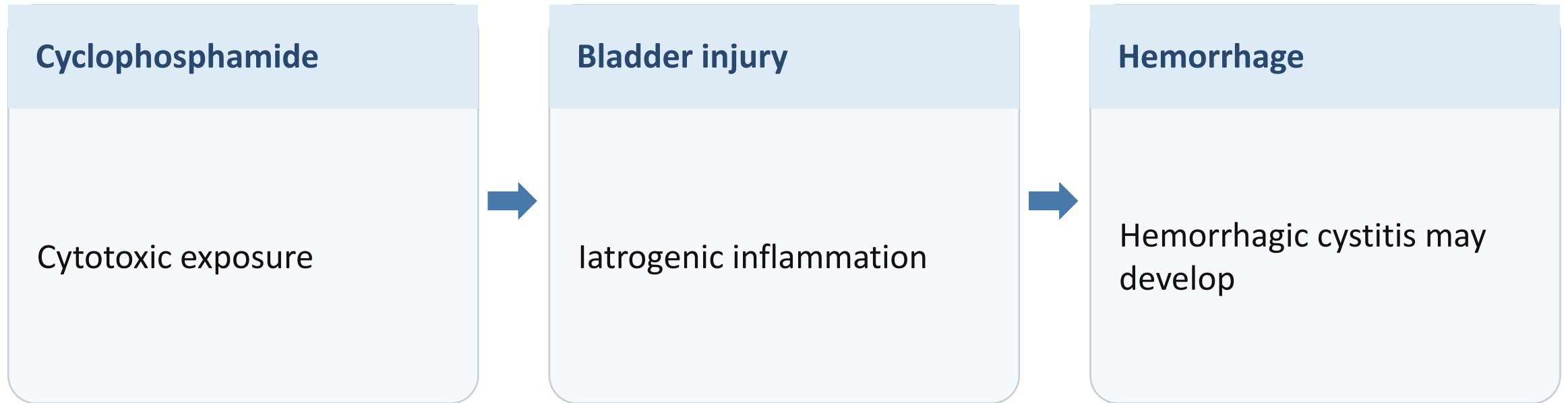
Other pattern

Eosinophilic cystitis is recognized

Treatment-related cystitis has distinct triggers

Exposure	Recognized inflammatory phenotype
Systemic chemotherapy	Iatrogenic cystitis
Cyclophosphamide	May cause hemorrhagic cystitis
Pelvic radiation	Acute and chronic radiation cystitis

Cyclophosphamide is linked to hemorrhagic bladder injury



Radiation cystitis may be acute or chronic

Exposure

Irradiation of the bladder
region / pelvis

Timing

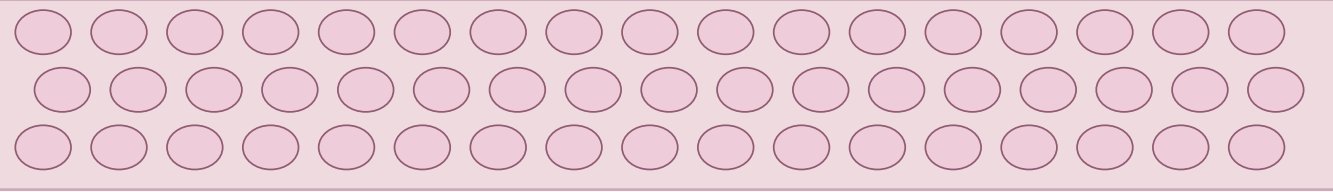
Acute or chronic
inflammatory effects

Classification

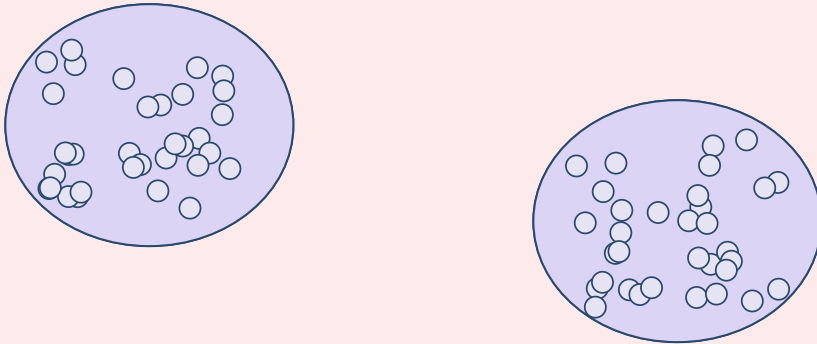
Noninfectious iatrogenic
cystitis

Follicular cystitis forms lymphoid follicles

Urothelium



Lamina propria / wall



SCHEMATIC — not a histologic specimen

Signature

Lymphoid follicles

Location

Bladder mucosa and underlying wall

Interpretation

Distinctive organized chronic inflammatory pattern

Eosinophilic cystitis belongs to the noninfectious spectrum

Textbook classification

Eosinophilic cystitis is listed.

What the chapter supplies

A brief recognition of the inflammatory variant.

Evidence boundary

Detailed etiology and diagnostic criteria are not provided in this excerpt.

Interstitial cystitis is a bladder-associated pain syndrome

Etiology

Not established

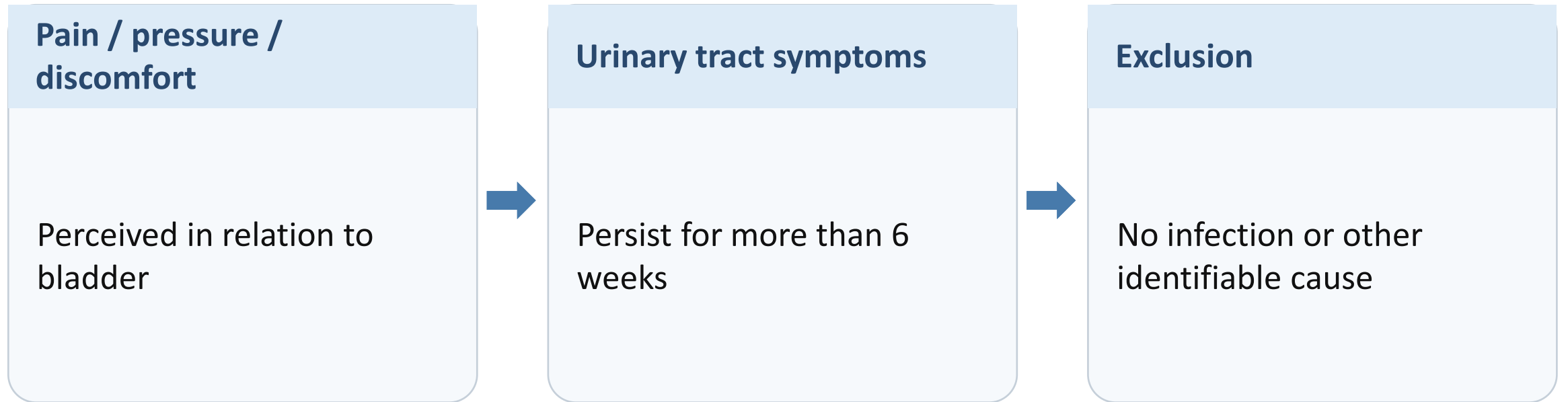
Population

Described as most frequent in females

Clinical identity

Persistent bladder-associated pain and urinary symptoms

The syndrome definition requires symptom persistence



Bladder pain syndrome has several accompanying symptoms

Pain

Intermittent, sometimes severe suprapubic pain

Irritative symptoms

Urinary frequency and urgency

Other complaints

Hematuria and dysuria may occur

Cystoscopic mucosal changes may be seen

Glomerulations

Punctate mucosal hemorrhages

Mucosal fissures

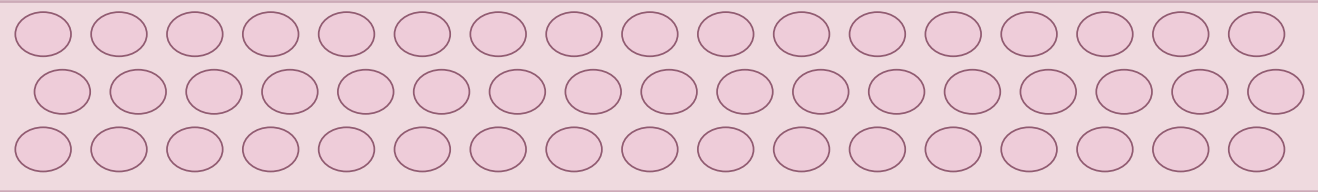
Described among typical cystoscopic observations

Interpretive context

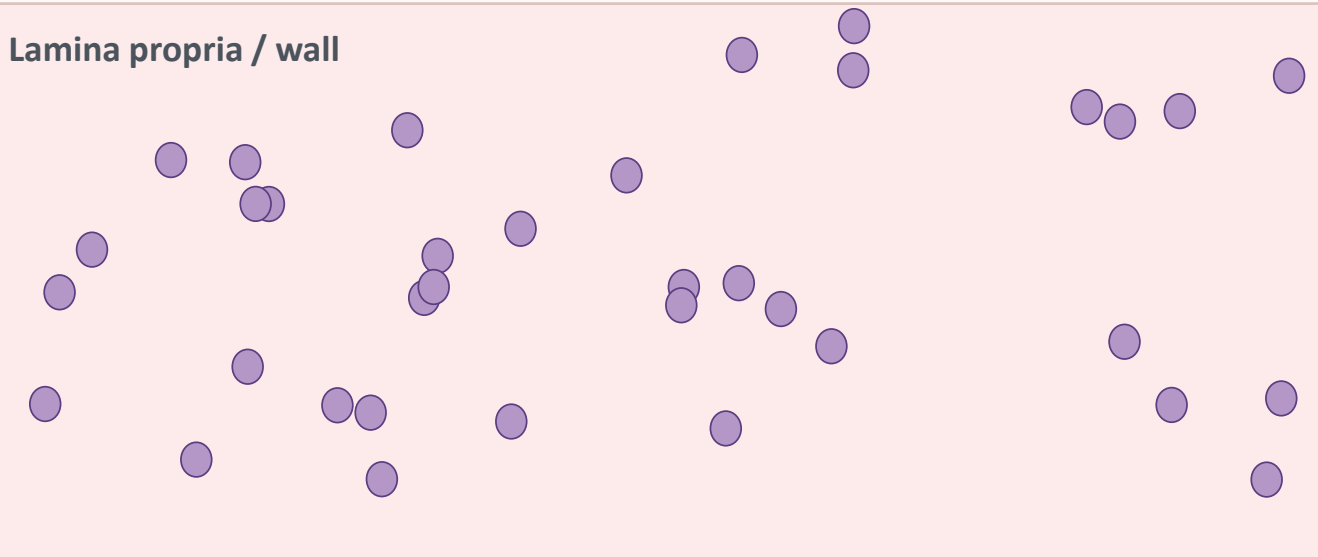
Must be correlated with symptoms and exclusion of other causes

Routine microscopy may be nonspecific in bladder pain syndrome

Urothelium



Lamina propria / wall



SCHEMATIC — not a histologic specimen

Microscopic pattern

Often nonspecific

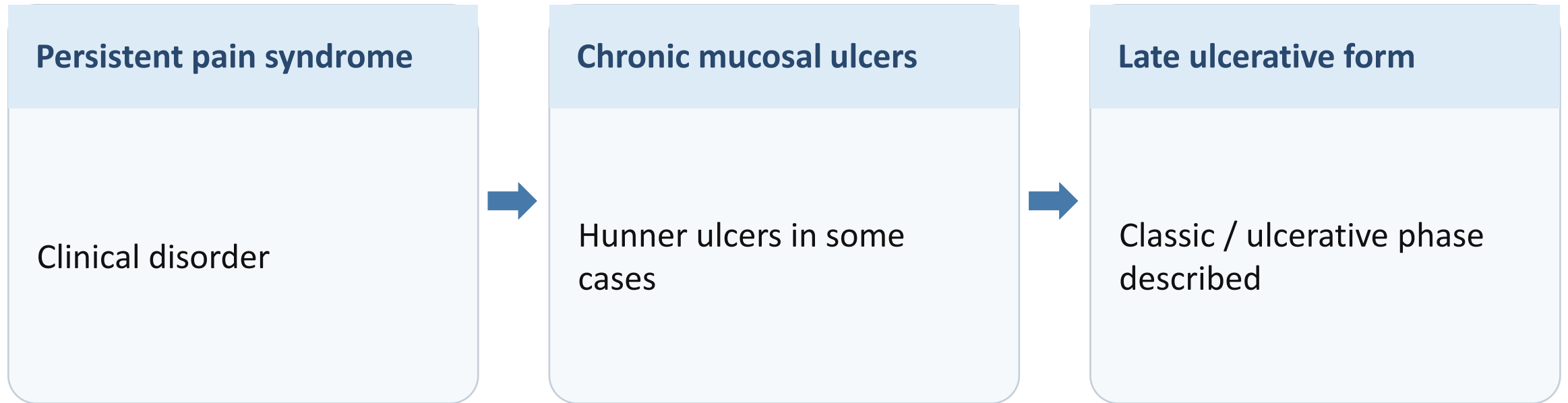
Potential observation

Increased submucosal mast cells

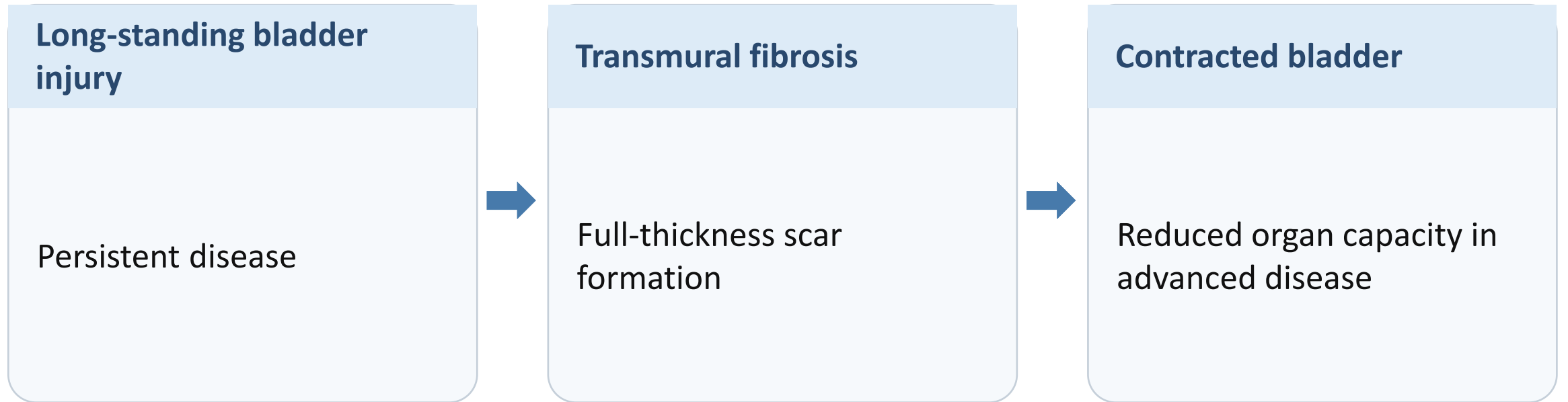
Uncertainty

Pathogenic significance remains unclear

Hunner ulcers define an ulcerative clinical-pathologic subset



Late bladder pain syndrome can cause bladder contraction



Biopsy can be important to exclude carcinoma in situ

Clinical question	Reasoning
Why consider a neoplasm?	Bladder carcinoma in situ may mimic interstitial cystitis symptoms.
What does biopsy contribute?	The main role emphasized is exclusion of carcinoma in situ.
What does routine inflammation prove?	Often little: histologic changes may be nonspecific.

Malakoplakia is a distinctive chronic inflammatory reaction

Central defect

Acquired impairment of phagocyte function

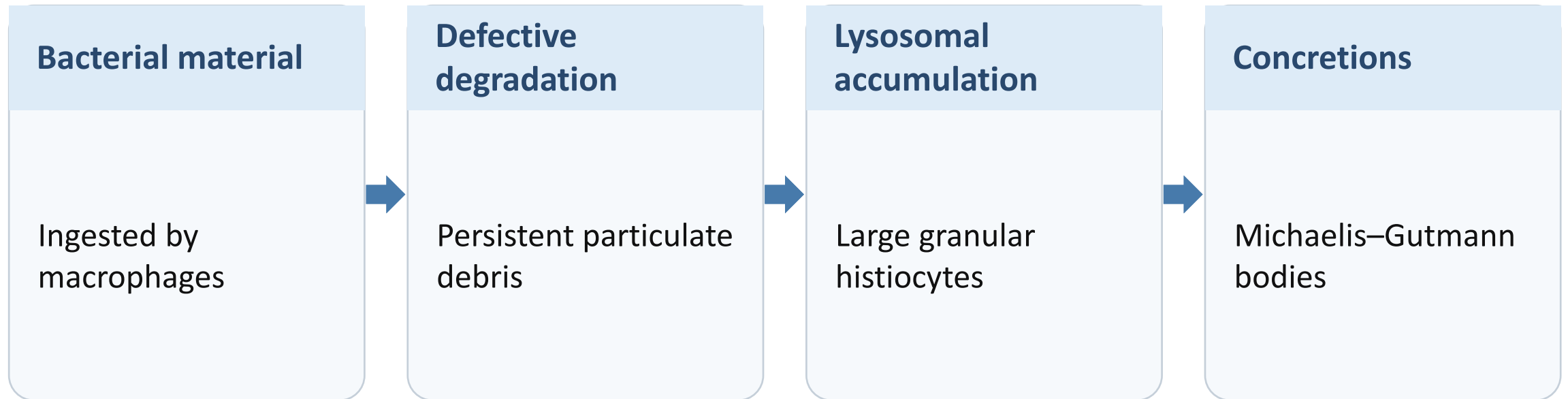
Infectious association

Chronic bacterial infection, often E. Coli

Clinical setting

More frequent in immunosuppressed individuals

Defective phagolysosomal handling underlies malakoplakia



Malakoplakia has an infection and immunosuppression setting

E. Coli

Main bacterial association described

Proteus species

Occasional alternative

Immunosuppression

Especially relevant among transplant recipients

Yellow mucosal plaques are a macroscopic clue

Cystoscopic appearance

Soft yellow, slightly raised plaques

Tissue response

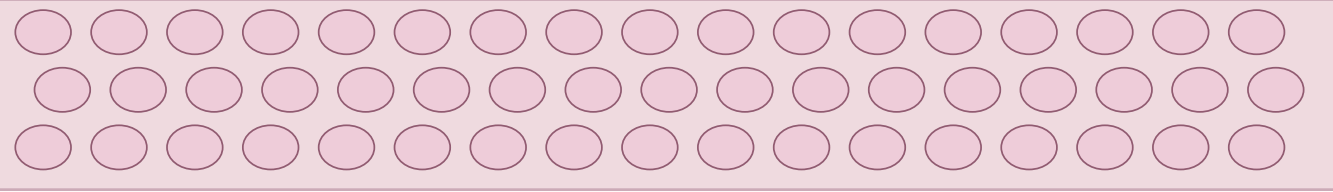
Macrophage-rich chronic inflammation

Limit

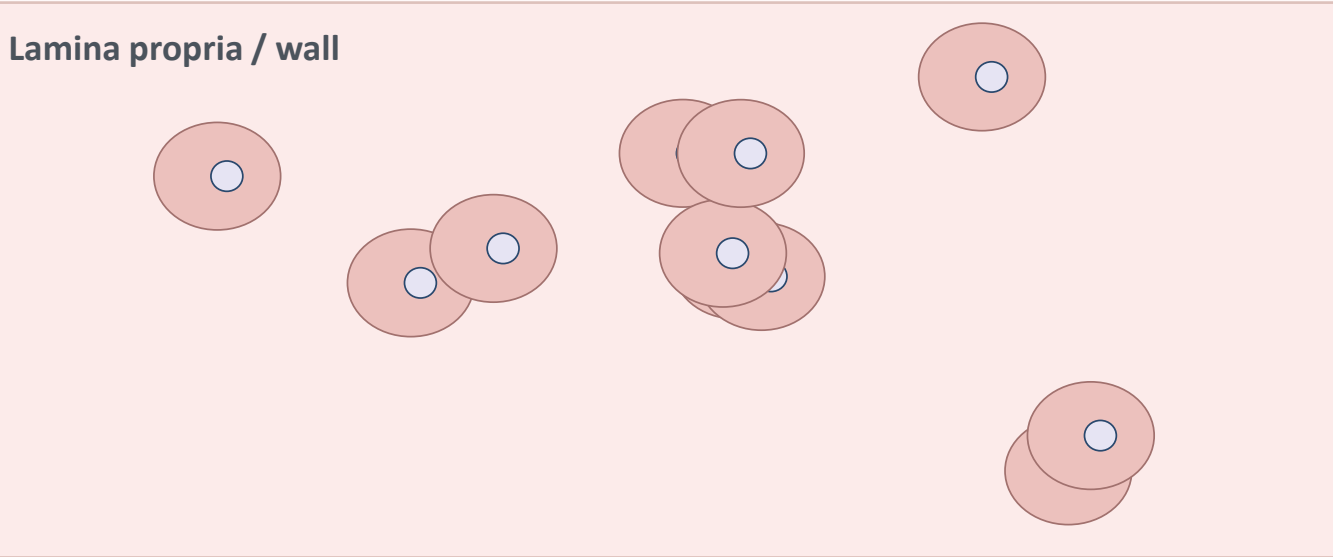
Appearance alone does not establish the characteristic histology

Von Hanseman histiocytes dominate the biopsy

Urothelium



Lamina propria / wall



SCHEMATIC — not a histologic specimen

Cellular finding

Large foamy macrophages

Cytoplasm

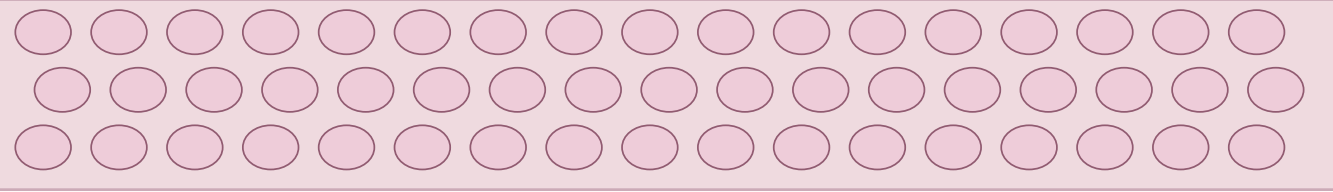
Abundant granular debris-containing material

Accompanying cells

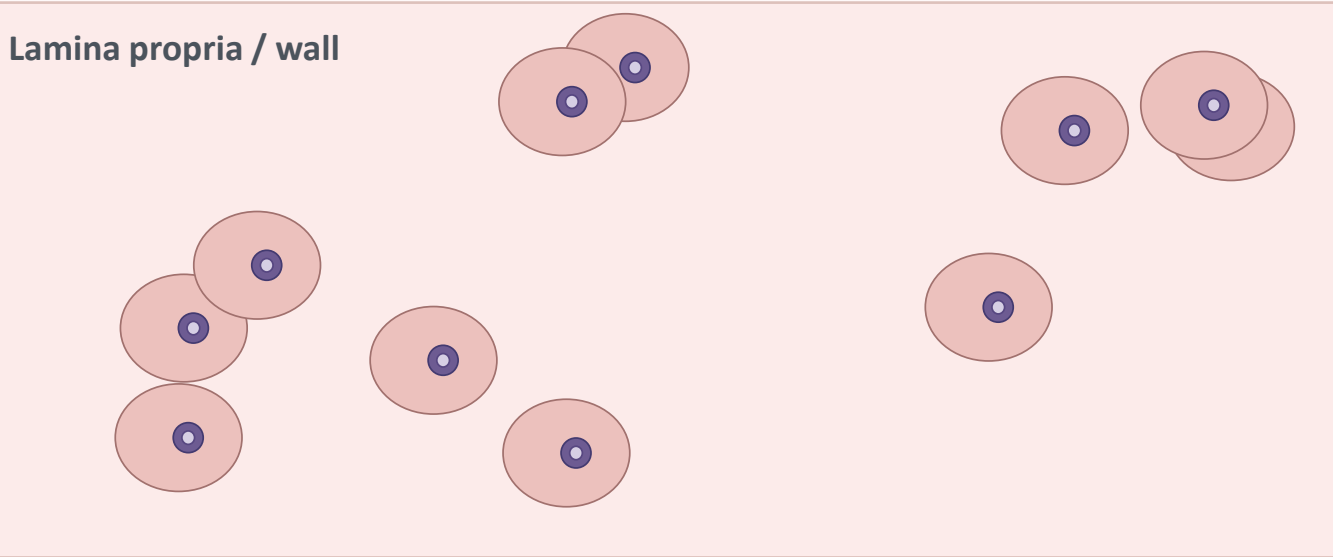
Occasional giant cells and lymphocytes

Michaelis–Gutmann bodies are the key inclusions

Urothelium



Lamina propria / wall



SCHEMATIC — not a histologic specimen

Structure

Laminated mineralized concretions

Pathogenesis

Calcium deposited in enlarged lysosomes

Localization

Characteristic inclusions in macrophages

PAS staining helps highlight the malakoplakia pattern

Macrophage cytoplasm

Granular PAS-positive material in affected macrophages

Associated inclusions

Michaelis–Gutmann bodies seen within affected cells

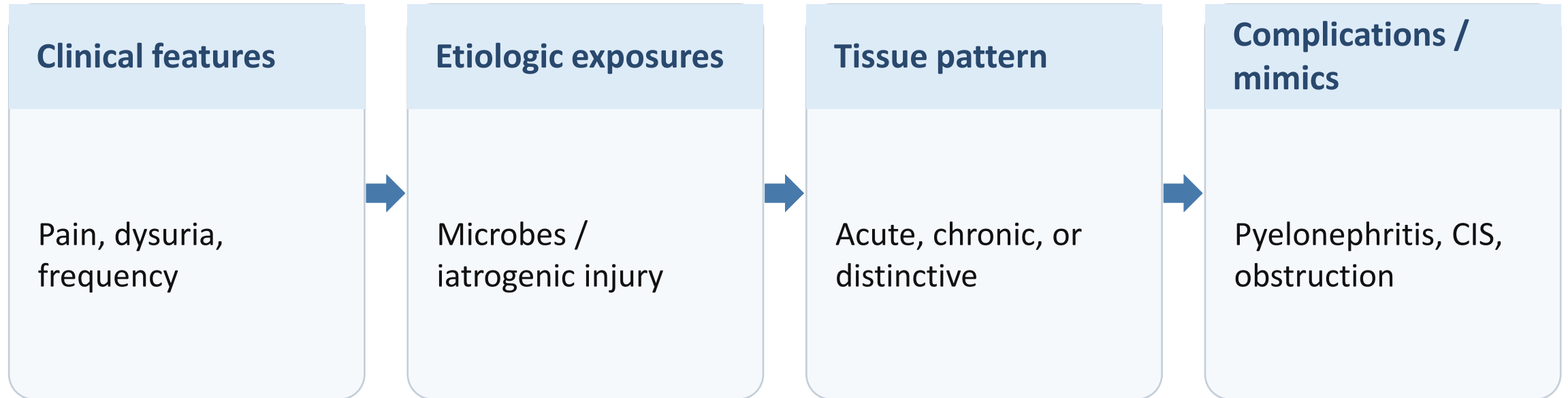
Interpretation

Evaluate morphology together with special-stain findings

Two distinctive cystitis variants have different identities

Feature	Interstitial cystitis	Malakoplakia
Defining setting	Bladder-related pain syndrome	Chronic bacterial/phagocyte dysfunction
Characteristic finding	Hunner ulcer in a subset	Michaelis–Gutmann bodies
Microscopy	Often nonspecific	Foamy von Hansemann macrophages
Key diagnostic concern	Exclude carcinoma in situ	Identify intracellular concretions

Inflammatory morphology must be interpreted in context



Teaching case: Repeated acute cystitis

Fictional vignette: Recurrent frequency, dysuria, and suprapubic pain in a patient with poor bladder emptying.

Question: Why might treating a single infectious episode be insufficient?

Reasoning: Persistent urinary stasis can sustain the underlying predisposition.

Conclusion: Evaluate and address the primary obstructive or structural lesion.

Teaching case: Chronic bladder-associated pain

Fictional vignette: Intermittent severe suprapubic pain with urgency and frequency over many weeks.

Question: Which condition merits consideration when infection and other causes are excluded?

Reasoning: Bladder pain syndrome is defined clinically by persistence and exclusion.

Diagnostic caution: Biopsy may be needed to exclude carcinoma in situ.

Teaching case: A macrophage-rich bladder lesion

Fictional vignette: An immunosuppressed patient has recurrent bacterial infection and yellow bladder plaques.

Question: Which histologic features would favor malakoplakia?

Expected: Abundant von Hanseman macrophages and Michaelis–Gutmann bodies.

Mechanistic link: Impaired phagolysosomal clearance of bacterial debris.

Integrated synthesis: Four diagnostic distinctions

Question	Most informative distinction
Is the cause infectious?	Agent spectrum, host risk and exposures
Is the response acute or chronic?	Neutrophils vs mononuclear infiltrate
Is there a special morphologic pattern?	Follicles or Michaelis–Gutmann bodies
Is an important mimic present?	Consider CIS in the bladder pain syndrome setting

Reference and scientific scope

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition (2026).
- Scope: bladder inflammation, acute and chronic cystitis, and special forms of cystitis.
- Teaching schematics are native editable shapes, not clinical specimens.
- Original textbook diagnostic figures are not reproduced in this lecture.