

Upper Airways

Inflammatory Lesions

Pharyngitis • Tonsillitis • Poststreptococcal pathology

Dr Abu Suraih Sakhri

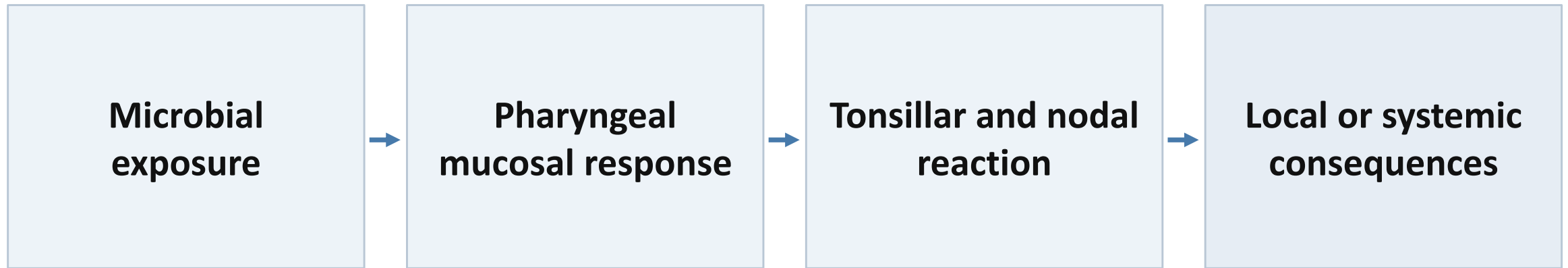
MBBS, MD (Pathology)

www.abusuraihsakhri.com

Learning outcomes

- Distinguish viral from bacterial patterns of pharyngeal inflammation.
- Explain tonsillar enlargement, crypt exudates, and pseudomembranes.
- Connect group A streptococcal pharyngitis to systemic sequelae.
- Apply morphology and etiology to differential diagnosis.

Nasopharyngeal inflammation: The scope



The same infection can produce mucosal, tonsillar and regional lymph-node findings.

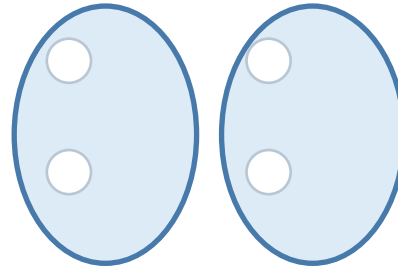
The connected mucosal–lymphoid unit

Pharyngeal mucosa



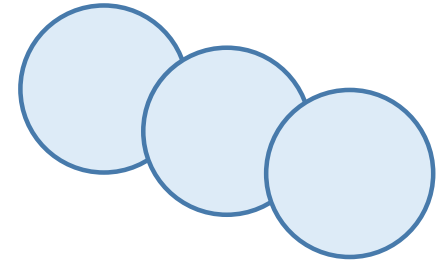
Inflammatory erythema and edema

Palatine tonsils



Reactive lymphoid enlargement

Regional nodes



Reactive enlargement

Schematic — three sites can respond to the same respiratory infection.

Pharyngitis: Mucosal involvement

- Inflammation affects pharyngeal mucosa during respiratory infection.
- The visible tissue response includes erythema and edema.
- Adjacent lymphoid tissue commonly responds with enlargement.

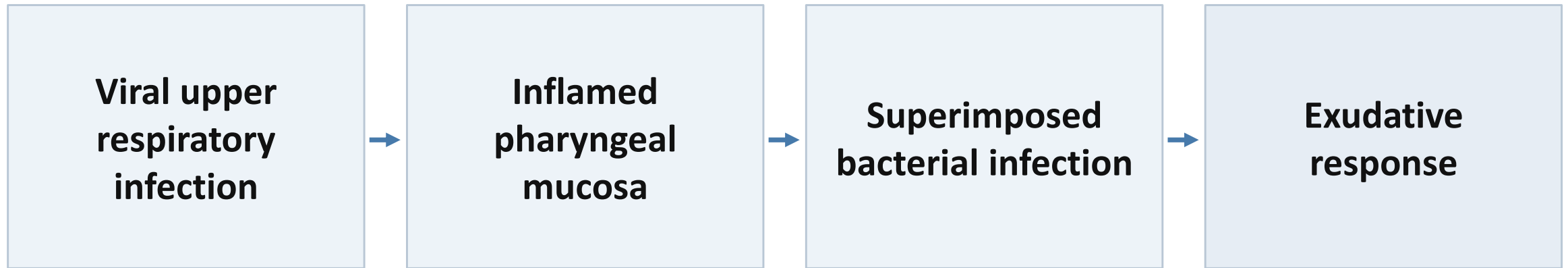
Tonsillitis: Lymphoid-tissue involvement

- Tonsils enlarge during a reactive inflammatory response.
- Bacterial infection may add crypt-centered exudation.
- Pharyngeal mucosal findings and tonsillar findings often coexist.

Two major infectious patterns

Viral pattern	Bacterial pattern
Most frequent context	Primary infection or superinfection
Diffuse erythema and edema	Exudate may be prominent
Reactive tonsils and nodes	Crypt-centered suppuration possible

Primary and secondary bacterial infection



Secondary infection is one possible route; Bacteria can also initiate illness directly.

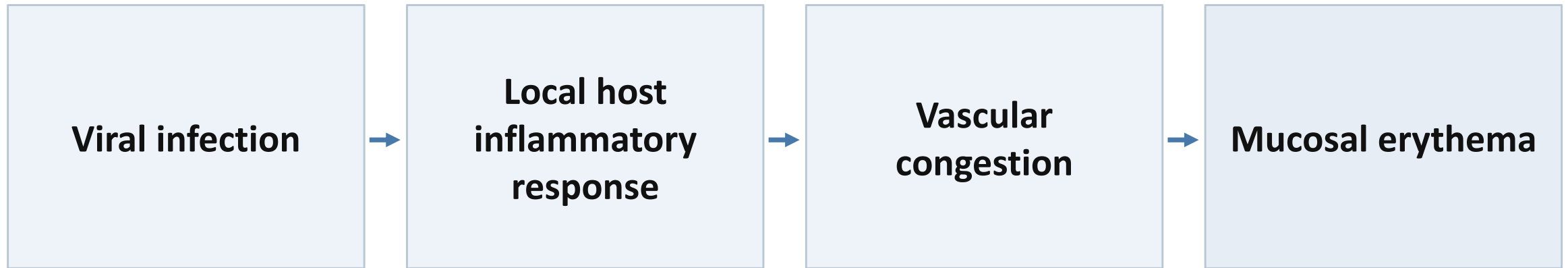
Viruses dominate the etiologic list

- Rhinoviruses, echoviruses, and adenoviruses are emphasized as common agents.
- Influenza strains and respiratory syncytial virus account for additional cases.
- The shared histologic outcome is a host mucosal inflammatory reaction.

Viral agents: Named etiologic groups

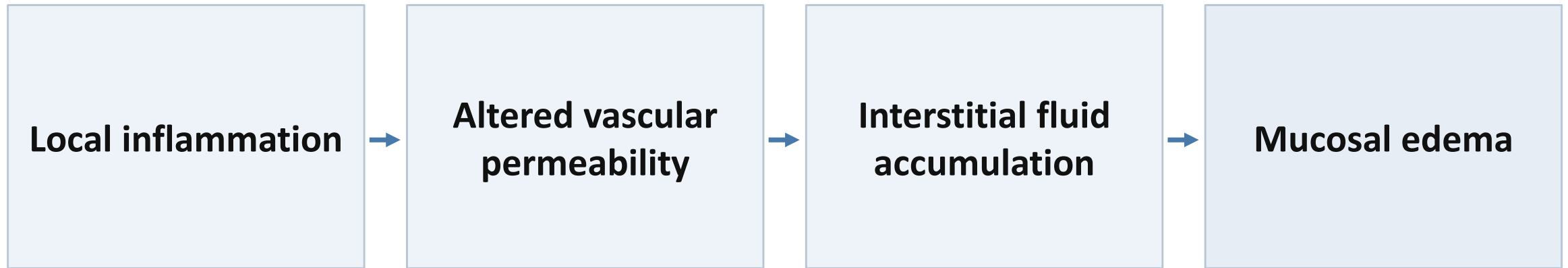
Highlighted as common	Also implicated
Rhinoviruses	Influenza viruses
Echoviruses	Respiratory syncytial virus
Adenoviruses	Multiple strains and presentations

Viral disease: Why the pharynx is red



Visible redness represents a vascular component of the inflammatory response.

Viral disease: Why mucosa swells



Edema is extracellular fluid accumulation within the inflamed mucosa.

Erythema and edema are linked findings

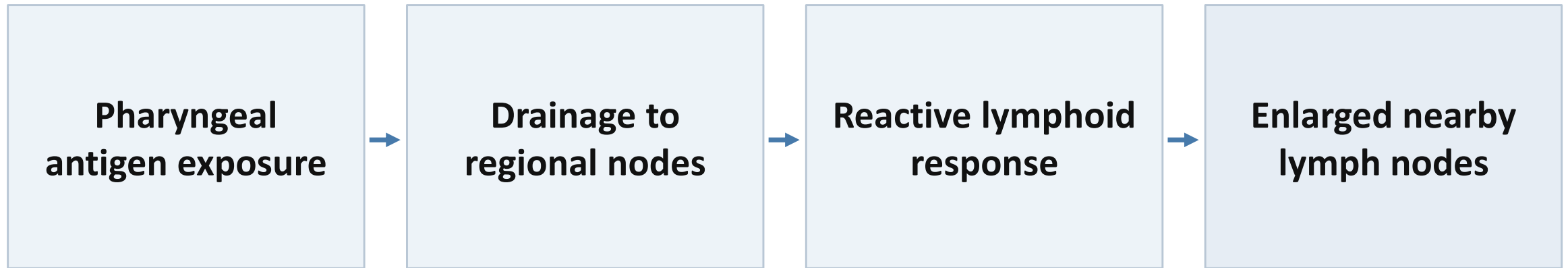
Erythema	Edema
Dominant visible change: redness	Dominant visible change: swelling
Reflects vascular congestion	Reflects fluid in interstitium
Distributed over mucosa	Contributes to tissue fullness

Reactive enlargement of the tonsils



Enlargement reflects an immune response rather than proof of a specific pathogen.

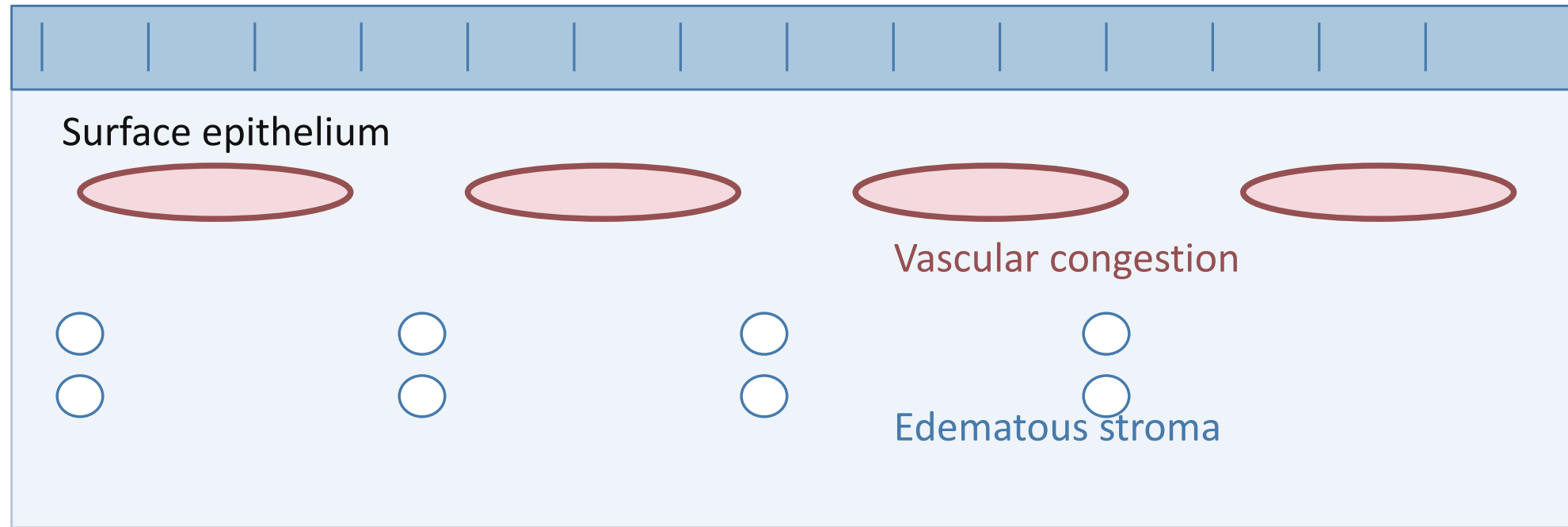
Regional lymphadenopathy in pharyngitis



Cervical node enlargement can accompany both viral and bacterial pharyngeal disease.

Viral pattern: Integrated morphology

Original native schematic of an inflamed mucosal surface



Morphology is a response, not a viral test

- Several viruses can produce similar mucosal erythema and edema.
- Reactive enlargement does not by itself identify the pathogen.
- Etiologic classification requires more than gross morphology.

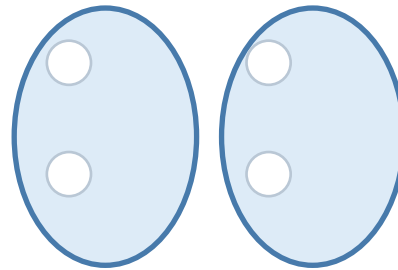
Anatomy of the visible response

Pharyngeal mucosa



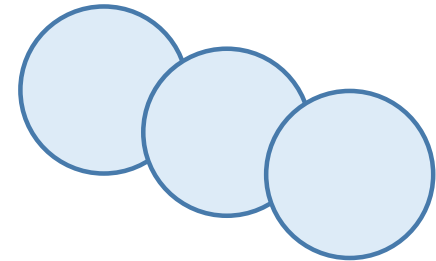
Inflammatory erythema and edema

Palatine tonsils



Reactive lymphoid enlargement

Regional nodes



Reactive enlargement

Schematic — three sites can respond to the same respiratory infection.

Bacterial pharyngitis: Two routes

Primary bacterial disease	Secondary bacterial process
Bacteria initiate inflammation	Follows a viral infection
May cause tonsillitis directly	Superimposed inflammatory change
Exudate may develop	May accentuate exudation

β -Hemolytic streptococci predominate

- The upper-airway source identifies β -hemolytic streptococci as the most frequent bacterial pathogens.
- Group A Streptococcus pyogenes is a key cause of streptococcal pharyngitis.
- Late cardiac and renal sequelae make this infection consequential.

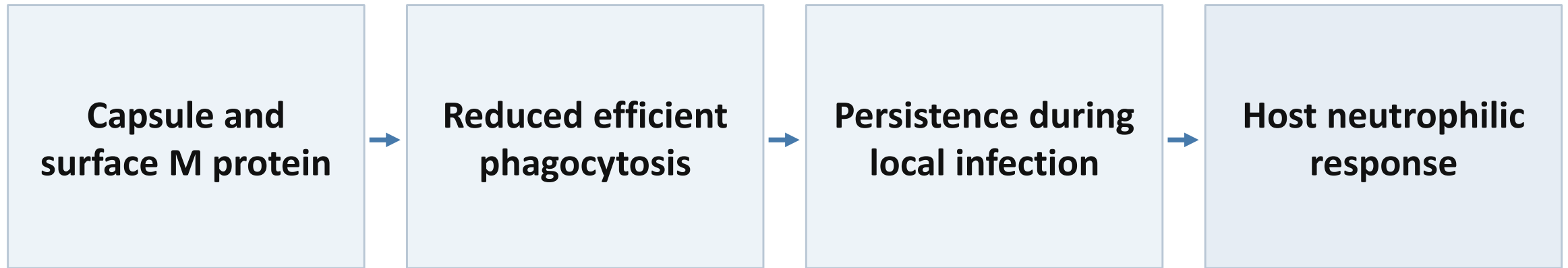
Other bacterial causes remain possible

- Staphylococcus aureus is named as another potential pathogen.
- Other bacterial species can also infect the pharynx or tonsils.
- An exudative surface alone does not establish bacterial species.

Streptococcus pyogenes: Organism context

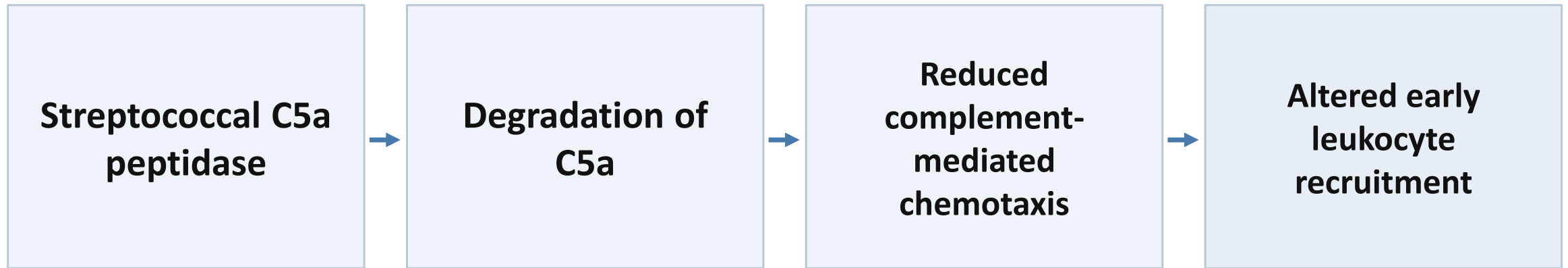
- Group A β -hemolytic streptococcus; Gram-positive cocci in pairs or chains.
- M protein protects against phagocytosis; A capsule also contributes to immune evasion.
- The organism causes both local suppurative disease and postinfectious immune syndromes.

Streptococcal immune evasion



Immune-evasion factors make streptococcal persistence biologically plausible.

C5a peptidase: Another virulence factor



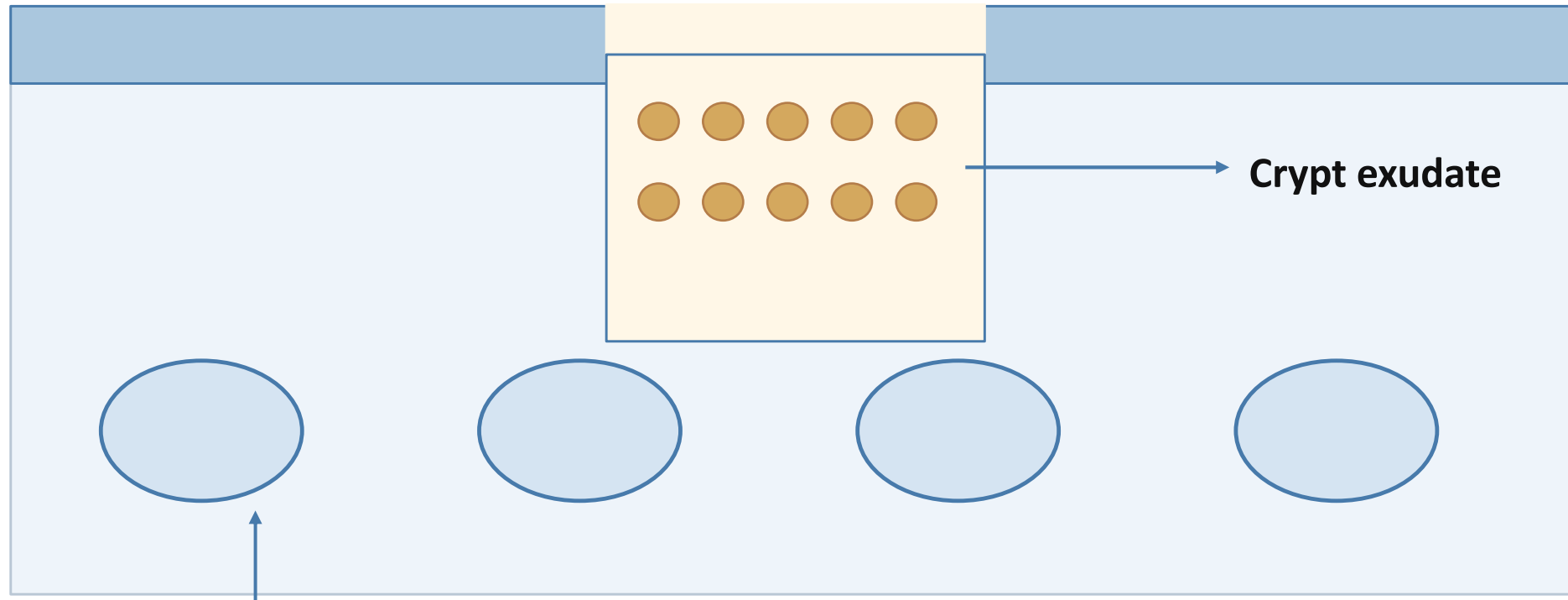
Degradation of C5a affects recruitment signals and should not be confused with bacterial invasion.

Streptococcal infection can be suppurative

- Diffuse neutrophilic inflammation in streptococcal infection.
- Pharyngeal lesions can feature punctate abscesses of tonsillar crypts.
- Cervical lymphadenopathy may accompany the local process.

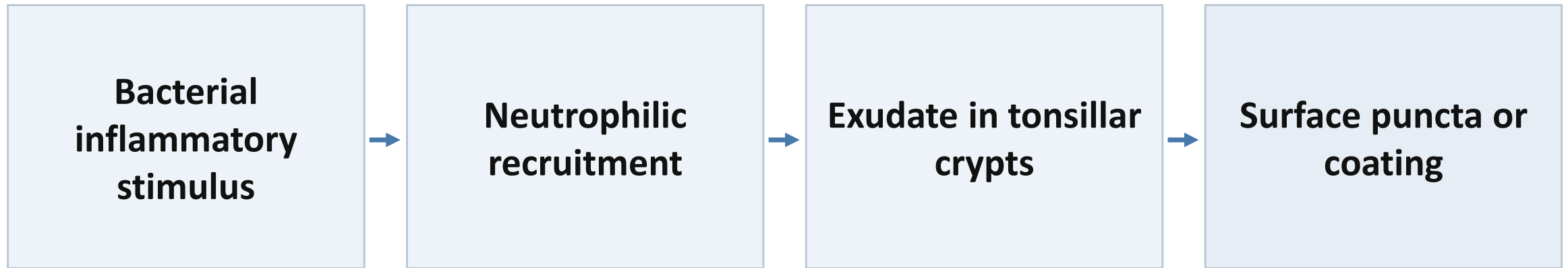
The tonsillar crypt is a key locus

Schematic — crypt lumen and underlying reactive lymphoid tissue



Reactive lymphoid tissue

Why tonsillar exudate becomes visible



Puncta on the tonsil reflect exudate reaching the crypt openings.

Exudate may coat multiple surfaces

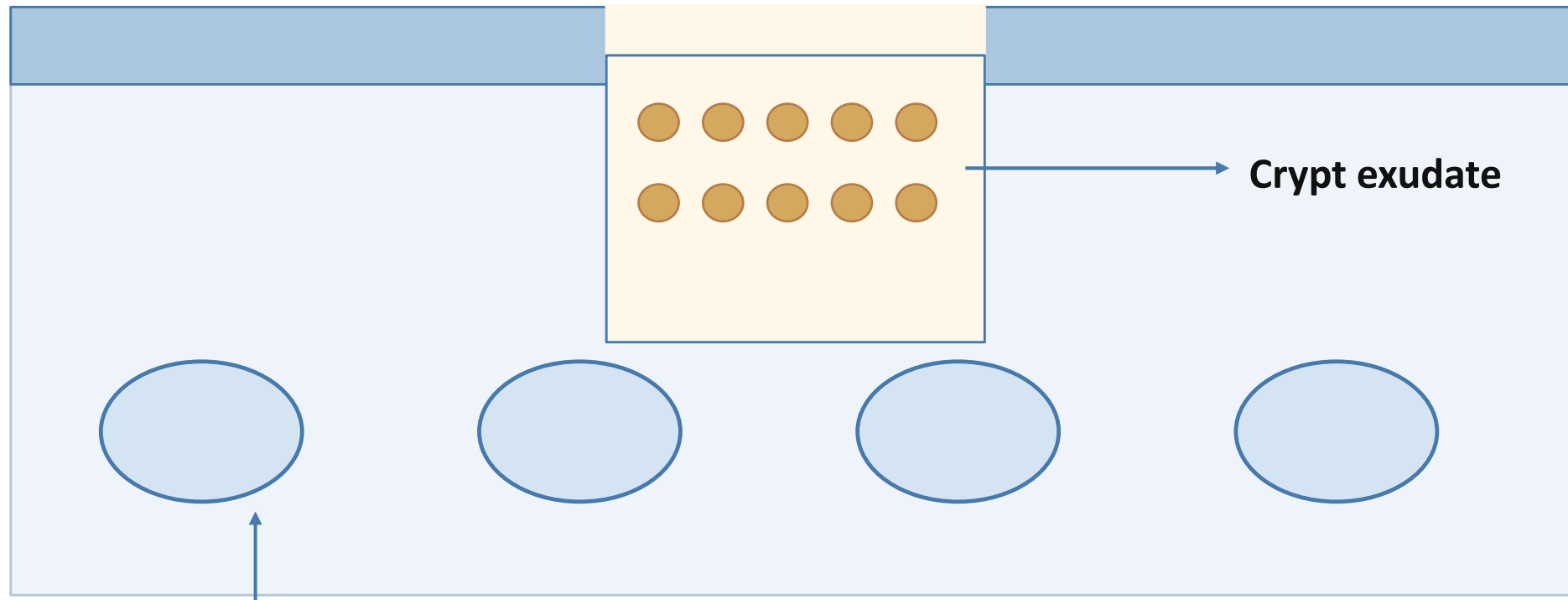
- Inflammatory material may be found on nasopharyngeal mucosa.
- Enlarged nasopalatine and palatine tonsils may also be covered.
- A visible coating should be interpreted within its anatomic distribution.

Follicular tonsillitis: Recognizable pattern

- The tonsils are enlarged by reactive lymphoid hyperplasia.
- Exudate emerging from crypts creates discrete surface dots.
- The term describes the visible architecture of a tonsillar reaction.

Tonsillar enlargement and crypt exudate

Schematic — crypt lumen and underlying reactive lymphoid tissue



Reactive lymphoid tissue

Reactive follicles are not surface exudate

Tonsillar follicular response	Crypt exudate
Reactive lymphoid hyperplasia	Inflammatory material in crypt lumen
Contributes to enlargement	Appears as punctate surface dots
Within lymphoid tissue	May accompany bacterial disease

The term “follicular tonsillitis”

- The clinical designation refers to a dotted, exudative tonsillar appearance.
- Underlying lymphoid hyperplasia contributes to organ enlargement.
- Crypt-centered surface changes should not be mistaken for lymphoid follicles themselves.

Pseudomembranes: A pattern, not a diagnosis

- “exudative membranes (pseudomembranes)” for some bacterial pharyngitides.
- A membrane-like coating can have different causes and mechanisms.
- Diphtheria warrants particular distinction when a tough membrane is present.

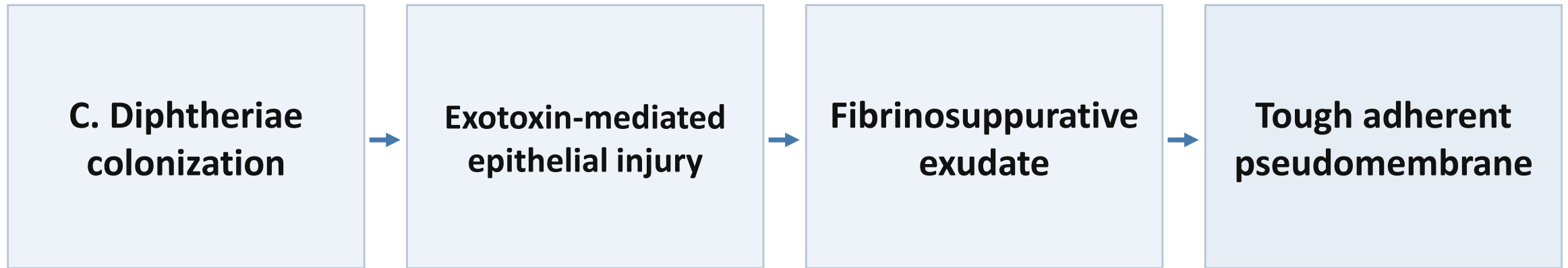
Membranous inflammation: Two examples

Nonspecific bacterial exudation	Diphtheria
Coating may form on tonsils or mucosa	Toxin-induced epithelial necrosis
Source does not assign a specific toxin	Tough fibrinosuppurative membrane
Etiology remains to be determined	May cause local bleeding and systemic toxicity

Diphtheria as an important mimic

- *Corynebacterium diphtheriae* can infect the pharynx and adjacent airway.
- A dense fibrinosuppurative exudate overlies damaged epithelium.
- Its characteristic tough membrane is a higher-risk pattern than ordinary exudation.

Diphtheritic membrane: Pathogenesis



The membrane is the product of tissue necrosis plus fibrinosuppurative exudation.

Why diphtheria cannot be treated as ordinary exudate

- Sloughing of its vascularized underlying bed can cause bleeding.
- Upper-airway obstruction or asphyxiation may follow.
- Systemically distributed toxin can injure the heart and peripheral nerves.

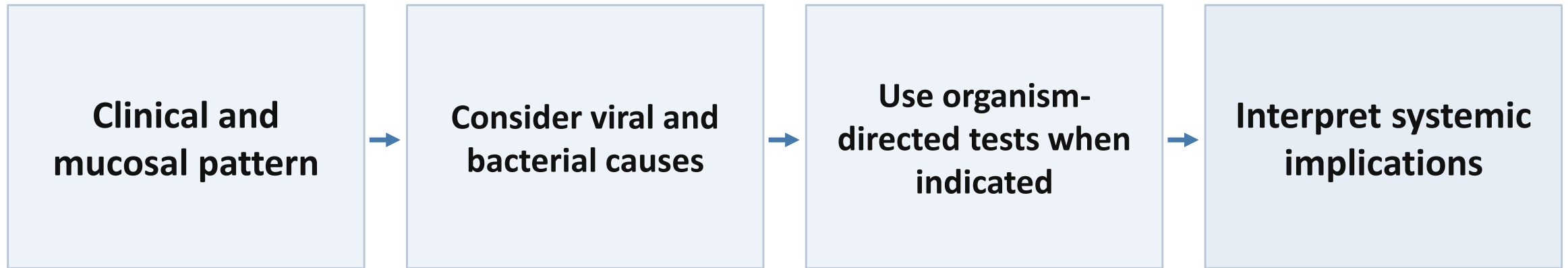
Infectious mononucleosis: Exudative mimic

- Acute pharyngitis and tonsillitis with a gray-white exudative coating.
- Associated findings include cervical lymph-node enlargement and palatal petechiae.
- The overlap reinforces that exudate is not etiologically specific.

Exudative pharyngitis: Pattern differential

Streptococcal pharyngitis	Infectious mononucleosis	Diphtheria
Crypt abscesses / tonsillar exudate	Gray-white tonsillar coating	Tough, dirty pseudomembrane
Cervical nodes may enlarge	Cervical lymphadenopathy	Toxin-mediated mucosal necrosis
Postinfectious sequelae matter	Palatal petechiae described	Risk of obstruction/systemic toxicity

The diagnostic question is etiologic



Gross features are insufficient to name the cause in many cases.

Streptococcal laboratory confirmation

- The infectious-disease chapter names throat culture as a diagnostic method.
- Rapid streptococcal antigen testing is also described for pharyngitis.
- Neither reactive tonsillar enlargement nor exudation alone identifies *S. Pyogenes*.

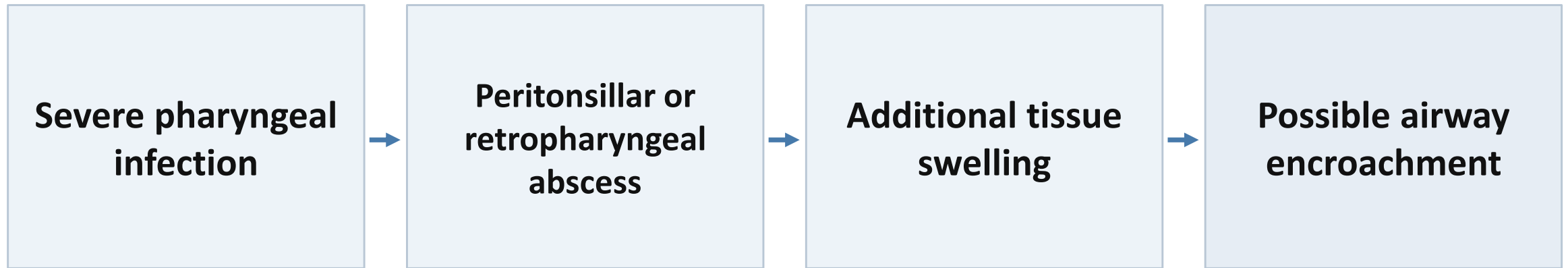
What pathology can and cannot establish

Morphology supports	Morphology alone cannot establish
Inflammation is present	Exact virus or bacterial species
Crypt-centered suppuration	Risk of later immune sequelae
Reactive lymphoid enlargement	Need for organism-specific confirmation

Airway compromise is a local complication

- Severe pharyngeal infection can produce marked swelling.
- Peritonsillar or retropharyngeal abscess formation may further narrow the airway.
- The anatomic extent of inflammation matters beyond the initiating pathogen.

Local complication pathway



Airway risk rises with the combination of inflammatory swelling and abscess location.

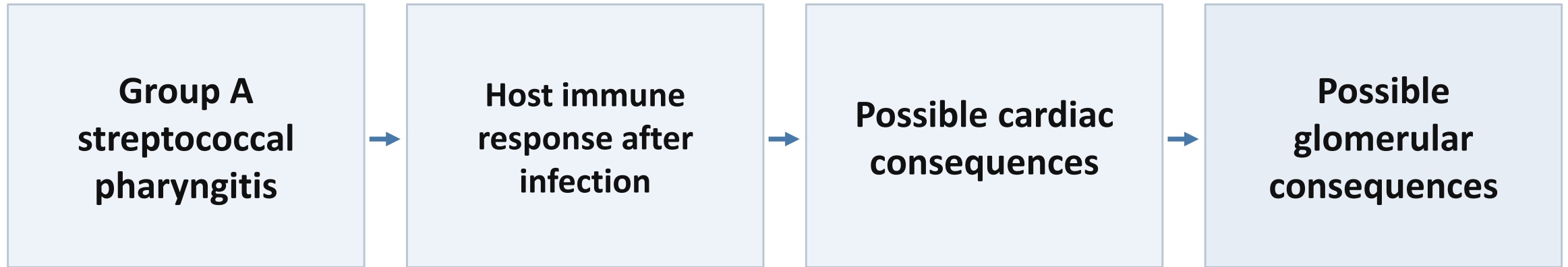
Recurrent tonsillitis and enlargement

- Whether repeated acute tonsillitis causes chronic tonsillar enlargement remains debated.
- Chronic tonsillar size cannot automatically be attributed to recurrence.
- Surgical excision may benefit selected patients despite this uncertainty.

Inflammatory enlargement versus causation

Observed finding	Causal claim requiring caution
The tonsil is enlarged	Repeated infections necessarily drive chronic hypertrophy
Reactive lymphoid hyperplasia may be present	Robbins explicitly identifies uncertainty
The enlargement is clinically relevant	Do not present causation as established

Why streptococcal sore throat matters



The local infection can be followed by organ injury after the organism has cleared.

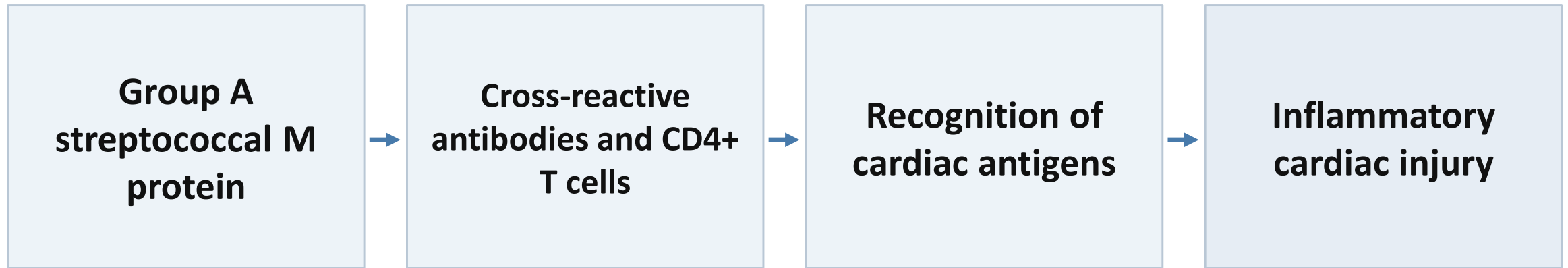
Two pathways beyond the infected pharynx

Rheumatic fever	Poststreptococcal GN
Immune cross-reactivity to host proteins	Antistreptococcal immune complexes
Often begins weeks after pharyngitis	Can follow pharyngeal or skin infection
Can produce carditis and later valve scarring	Glomerular inflammation follows

Acute rheumatic fever is postinfectious

- Robbins characterizes it as an immunologically mediated multisystem inflammatory disease.
- It classically follows group A streptococcal pharyngitis after a delay of weeks.
- The cardiac lesions are immune-mediated rather than bacterial colonization.

Rheumatic fever: Molecular mimicry



This pathway involves immune recognition of host cardiac antigens.

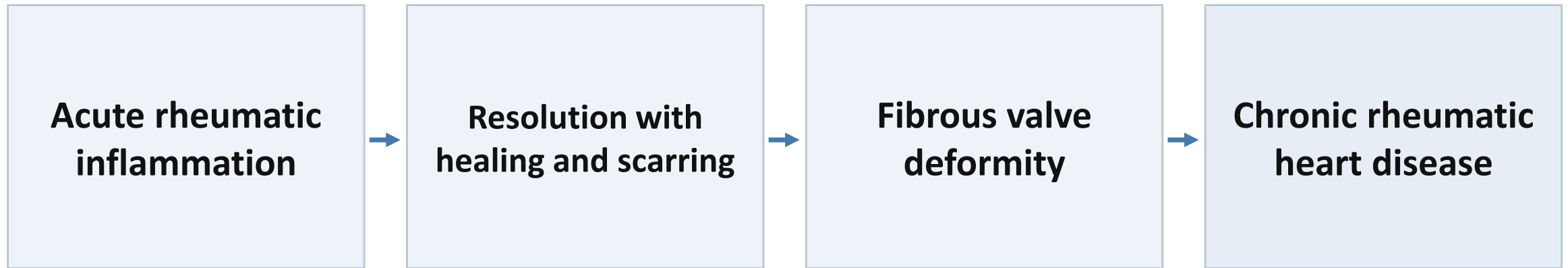
Rheumatic carditis: Tissue mechanism

- Antibody binding can activate complement and recruit Fc receptor–bearing leukocytes.
- Stimulated T cells release cytokines that activate macrophages.
- These combined processes produce cardiac inflammation in the absence of organisms.

Aschoff bodies: The cardiac lesion

- Focal myocardial inflammatory lesions contain T lymphocytes and activated macrophages.
- Anitschkow cells show elongated, ribbon-like nuclear chromatin.
- The lesion links a preceding throat infection to an immune-mediated cardiac process.

Healing may produce chronic valve disease

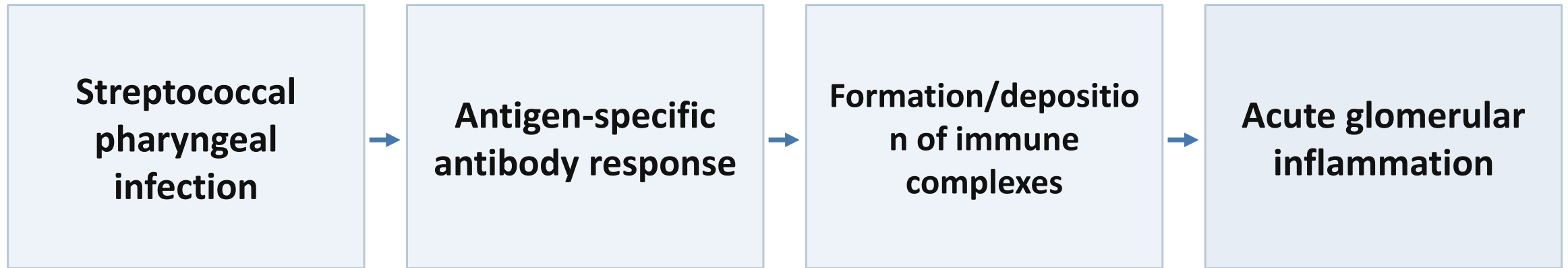


Fibrotic healing, not ongoing infection in the valve, explains deformity.

Poststreptococcal GN is immune-complex mediated

- Streptococcal antigens and antibodies form pathogenic immune complexes.
- Complement activation and leukocyte recruitment damage glomeruli.
- The disease is a late inflammatory complication, not direct bacterial invasion of glomeruli.

Glomerulonephritis: The latent interval



Delayed onset supports time required for antigen–antibody responses.

Poststreptococcal GN: Diagnostic mechanism

- A latency of approximately 1–4 weeks after infection.
- Serum complement may be reduced by complement consumption.
- Granular glomerular immune deposits support an immune-complex process.

Immune aftermath: Comparison

Rheumatic fever	Poststreptococcal GN
M-protein cross-reactivity	Microbial antigen–antibody complexes
Targets cardiac tissues	Targets renal glomeruli
May heal with fibrotic valve damage	Inflammation with complement consumption

Case 1: Diffusely inflamed pharynx

A patient with an acute upper respiratory illness has pharyngeal erythema, edema, and enlarged regional lymph nodes.

- Which broad infectious pattern is common?
- What can gross morphology identify—and what cannot it identify?

Discussion prompts; Suggested interpretation is in the presenter notes.

Case 2: Punctate tonsillar exudates

The tonsils are enlarged and show scattered pale dots emerging at the crypt openings.

- Name the tonsillar morphology.
- Explain separately the cause of enlargement and of the surface dots.

Discussion prompts; Suggested interpretation is in the presenter notes.

Case 3: Sore throat followed by carditis

Several weeks after a streptococcal throat infection, a patient develops inflammatory cardiac disease.

- Which delayed complication is relevant?
- What makes the cardiac injury immunologic rather than locally bacterial?

Discussion prompts; Suggested interpretation is in the presenter notes.

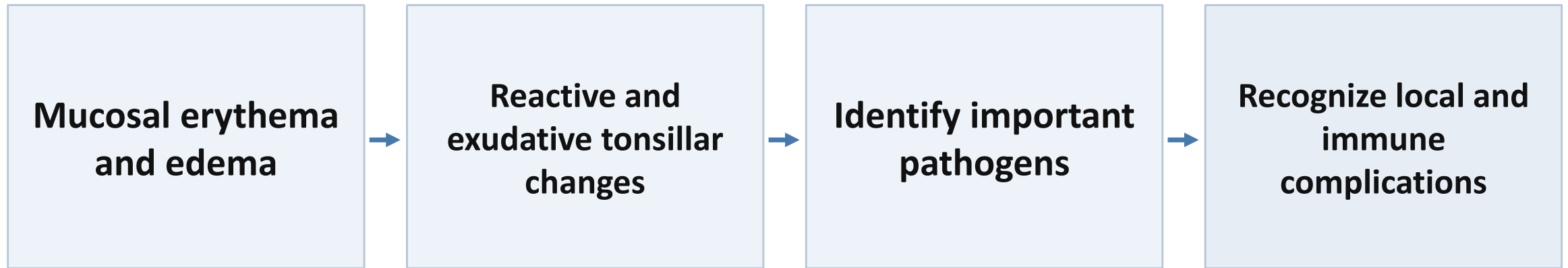
Case 4: Nephritis after infection

A child develops acute glomerular inflammation in the weeks following a pharyngeal streptococcal infection.

- Identify the postinfectious process.
- What immune deposits and complement findings support its pathogenesis?

Discussion prompts; Suggested interpretation is in the presenter notes.

Synthesis: Local lesion → systemic meaning



Observe the lesion, identify the etiology when necessary, and separate local from delayed complications.

Ten high-yield discriminators

- Viral pharyngitis is common; Erythema, edema, and reactive lymphadenopathy predominate.
- Bacterial tonsillitis may cause crypt-centered exudate and pseudomembranes.
- Follicular tonsillitis combines lymphoid hyperplasia with punctate crypt exudate.
- Streptococcal infection can precede rheumatic fever or glomerulonephritis.

Primary textbook and correlated chapters

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th ed. (2026).
- Chapter 16, Head and Neck: Nasopharynx; Inflammatory Lesions, p. 673.
- Chapter 8, Infectious Diseases: streptococci and diphtheria.
- Chapter 12, Heart: rheumatic fever; Chapter 20, Kidney: postinfectious GN.