

Pulmonary Eosinophilia

Patterns of eosinophilic lung injury and their differential diagnosis

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Learning objectives

- Identify the three categories of pulmonary eosinophilia.
- Connect eosinophilic recruitment to acute and chronic lung injury patterns.
- Interpret BAL, radiographic distribution, and histologic findings in context.
- Distinguish idiopathic eosinophilic pneumonia from secondary etiologies.

The unifying feature is tissue eosinophilia

- Pulmonary eosinophilia comprises relatively uncommon clinical–pathologic entities.
- The shared finding is eosinophilic infiltration within pulmonary tissue.
- Clinical tempo, distribution, accompanying injury, and causation distinguish the entities.

An eosinophil-rich lung is a pathologic pattern—not by itself an etiologic diagnosis.

Pulmonary eosinophilia in the interstitial disease context

Pulmonary eosinophilia is discussed within diffuse interstitial (restrictive) lung diseases.

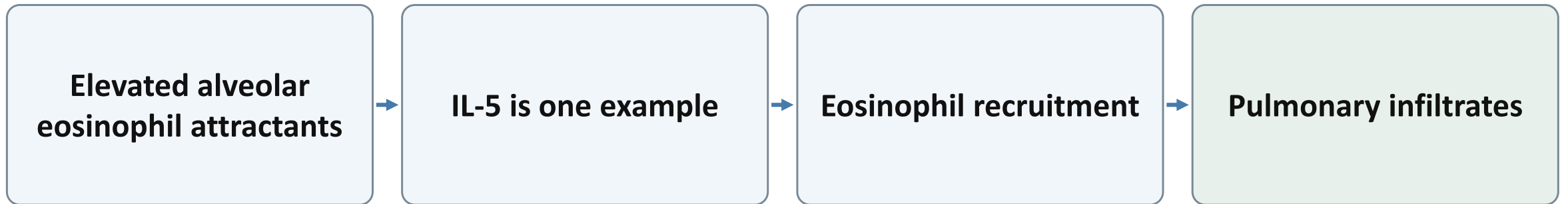
- The designation groups several distinct patterns rather than a single disease.
- Acute disease may involve widespread alveolar injury.
- Chronic disease can involve septa, alveolar spaces, and organization.

Acute alveolar injury

Septal inflammation

Airspace inflammation

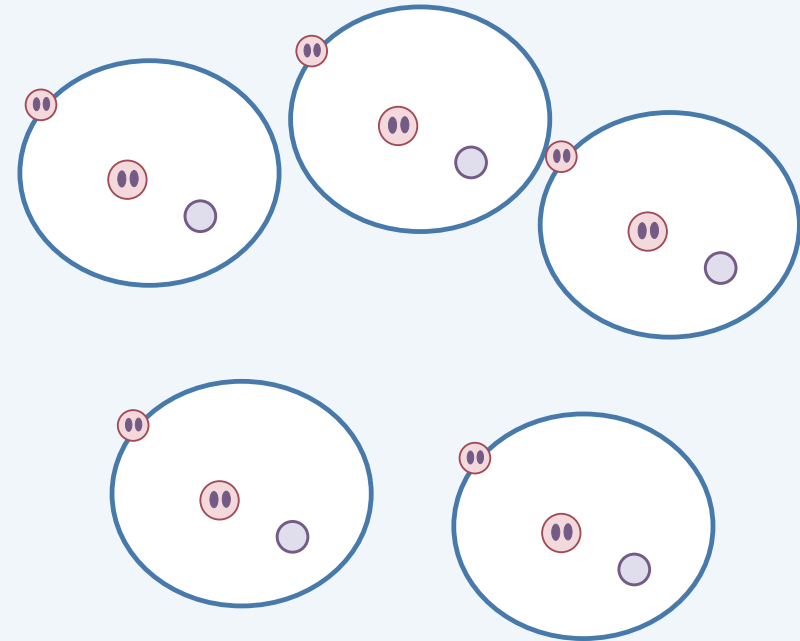
IL-5 contributes to eosinophil recruitment



Elevated intra-alveolar eosinophil-attracting signals, including IL-5, contribute to recruitment;
Other mechanisms are not represented.

Two compartments help organize morphology

- Alveolar septa are the walls between airspaces.
- Alveolar spaces represent the luminal air-containing compartment.
- Chronic eosinophilic pneumonia may show eosinophils and lymphocytes in both compartments.



Schematic — not a histology image

Three major diagnostic groups

Acute eosinophilic pneumonia

Rapid illness; Respiratory failure

Diffuse infiltrates; BAL eosinophilia

Secondary pulmonary eosinophilia

Infectious, allergic, or associated diseases

Etiologic or syndromic context matters

Idiopathic chronic eosinophilic pneumonia

Peripheral consolidation

Exclude alternative causes

Tempo and pattern guide the first branch

Feature	Acute eosinophilic pneumonia	Chronic eosinophilic pneumonia
Tempo	Rapid onset	Chronic clinical syndrome
Imaging emphasis	Diffuse infiltrates	Peripheral consolidation
Prominent morphology	Diffuse alveolar damage + eosinophils	Eosinophils/lymphocytes in septa + airspaces

Why identify an underlying cause?

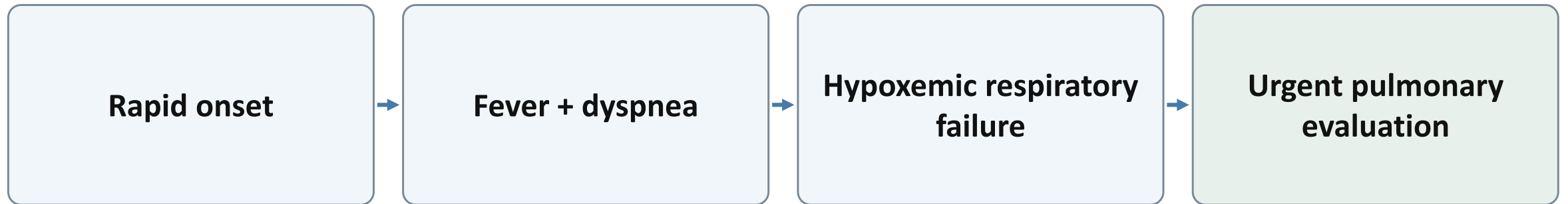
- Pulmonary eosinophilia also occurs with infections, drug allergy, and associated airway or systemic diseases.
- A secondary cause changes the interpretation of an eosinophilic infiltrate.
- The term idiopathic chronic eosinophilic pneumonia requires other causes to be excluded.

Cause-finding is part of the diagnostic classification, not an optional afterthought.

Acute eosinophilic pneumonia: Defining syndrome

- An acute pulmonary illness for which the cause is unknown.
- Characterized by rapid fever and dyspnea.
- The severe presentation includes hypoxemic respiratory failure.
- Eosinophilia is demonstrated in alveolar lavage fluid and tissue.

Rapid presentation is a major clue



The source describes a rapidly developing syndrome. No specific number of symptom-days is provided.

Acute disease combines symptoms and organ failure

AEP is more than eosinophilic infiltration: The clinical presentation includes hypoxemic respiratory failure.

- Fever signals an acute illness.
- Dyspnea is a dominant pulmonary symptom.
- Respiratory failure marks substantial physiologic impact.

Fever

Dyspnea

Hypoxemia

Chest radiograph: Diffuse infiltrates

Observed radiographic pattern

Diffuse pulmonary infiltrates are described in acute eosinophilic pneumonia.

Diffuse does not identify the cause by itself.

Interpretation

Combine the radiograph with tempo and BAL findings.

The contrasted pattern in the chronic form is peripheral consolidation.

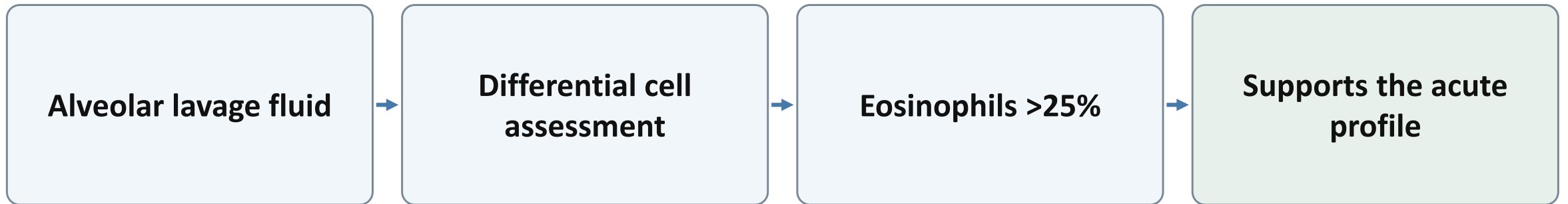
BAL: Eosinophils greater than 25%

>25%

- A value above 25% is specifically described for acute eosinophilic pneumonia.
- It provides direct evidence of an eosinophil-rich alveolar inflammatory process.
- Do not automatically transfer this source-specific statement to every category.

Eosinophils in bronchoalveolar lavage fluid

BAL evaluates the alveolar inflammatory population



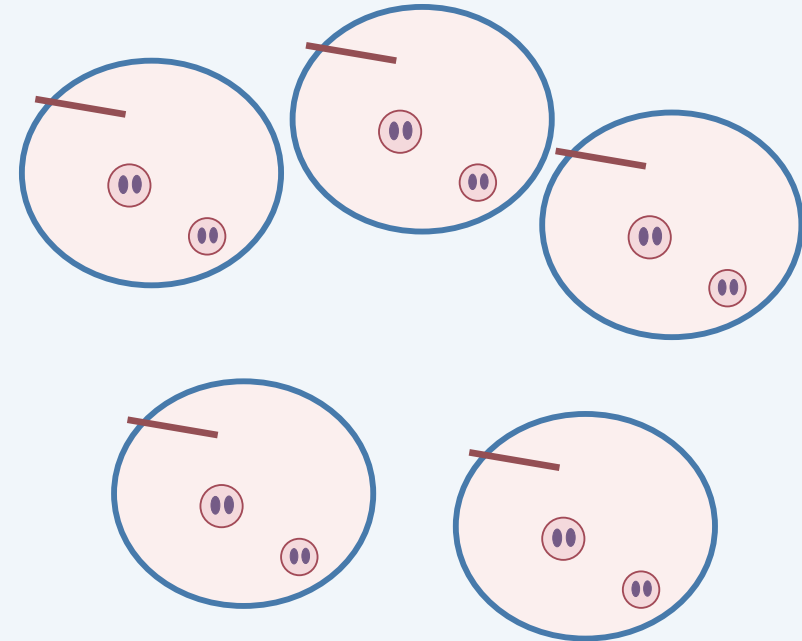
The percentage belongs to the source description of acute eosinophilic pneumonia; It does not stand alone as a complete cause assessment.

Acute histology: Diffuse alveolar damage

- Histologic examination shows diffuse alveolar damage.
- Numerous eosinophils accompany the alveolar injury.
- The co-occurrence of widespread injury and eosinophilic inflammation is the morphologic hallmark in the excerpt.

Acute injury: Inflammation and damage together

- The relevant pattern is diffuse alveolar damage with many eosinophils.
- Diffuse injury is concordant with severe hypoxemic respiratory failure.
- This editable schematic is conceptual; No microscopic specimen is implied.



Schematic — not a histology image

Integrating acute evidence

Clinical

Rapid fever and dyspnea

Hypoxemic respiratory failure

Investigation

Diffuse infiltrates

BAL eosinophils >25%

Histopathology

Diffuse alveolar damage

Numerous eosinophils

AEP responds promptly to corticosteroids

- A prompt corticosteroid response characterizes the described acute clinical course.
- Therapeutic response complements clinical and morphologic characterization.
- Treatment response should be interpreted alongside clinical, BAL, and tissue evidence.

A response to steroids is not a substitute for correct classification of the eosinophilic syndrome.

Hypothetical acute case: Identify the pattern

Teaching vignette (hypothetical)

A patient develops fever and rapidly worsening dyspnea followed by hypoxemic respiratory failure. Chest radiography demonstrates diffuse infiltrates. BAL contains 31% eosinophils.

Which category best matches the combined clinical, radiologic, and lavage findings?

Rapid progression

Diffuse infiltrates

BAL above 25%

Acute case: Confirm the reasoning

Supporting findings

Acute fever, dyspnea, respiratory failure

Diffuse pulmonary infiltrates

BAL eosinophils >25%

Expected additional evidence

If examined, histology may show diffuse alveolar damage with many eosinophils.

Prompt corticosteroid responsiveness is part of the acute clinical description.

The acute pattern does not erase etiologic assessment

- The acute idiopathic category is described as of unknown cause.
- Secondary eosinophilia has multiple recognized causal associations.
- Tempo and BAL findings should be interpreted alongside the patient's infectious, drug, allergic, and systemic contexts.

The differential remains broader than the acute syndrome label.

Secondary eosinophilia: A category of causes

Infectious

Parasitic

Fungal and bacterial

Allergic / inflammatory

Drug allergies

Hypersensitivity pneumonitis

Associated syndromes

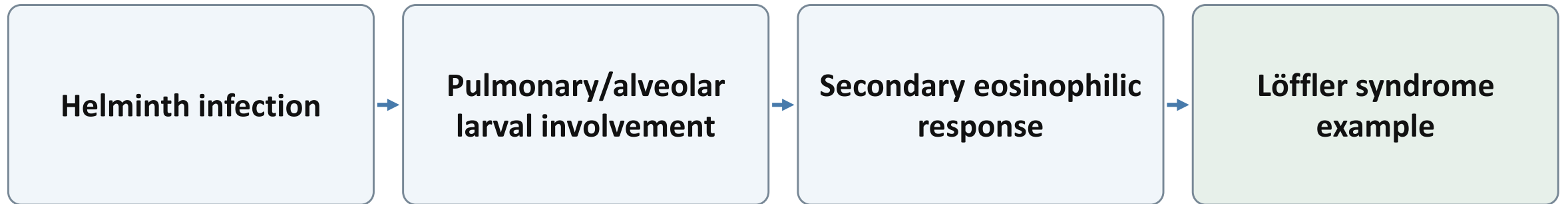
Asthma, ABPA

Churg–Strauss vasculitis

Infection is a major secondary branch

- Parasitic, fungal, and bacterial infections are recognized secondary causes.
- The source does not attribute one universal microscopic pattern to all these infections.
- Infectious context therefore has to be assessed separately from tissue eosinophilia.

Parasitic infection: A specifically named association



The named helminths are Ascaris, hookworm, and Strongyloides; This diagram organizes the given association.

Löffler syndrome is secondary pulmonary eosinophilia

- Löffler syndrome is classified as secondary pulmonary eosinophilia.
- It describes invasion of alveoli by larvae of specified helminths.
- This infectious mechanism distinguishes Löffler syndrome from the idiopathic chronic category.

Three helminth groups are explicitly named

Ascaris

Larval alveolar invasion

Löffler syndrome association

Hookworms

Larval alveolar invasion

Löffler syndrome association

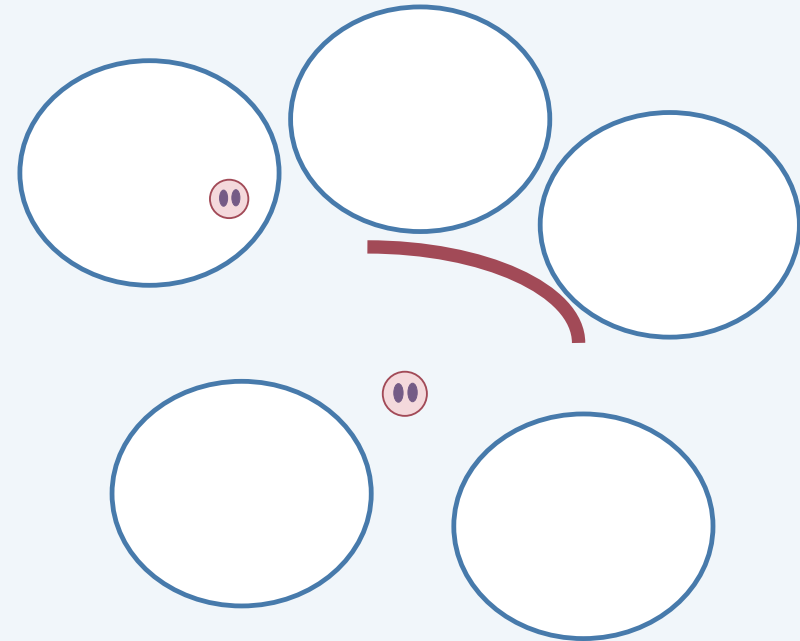
Strongyloides

Larval alveolar invasion

Löffler syndrome association

Larval involvement is the key morphologic idea

- In Löffler syndrome, helminth larvae enter/invade alveolar spaces.
- Pulmonary eosinophilia occurs in association with this invasion.
- The diagram is a schematic of association, not a parasitology micrograph.



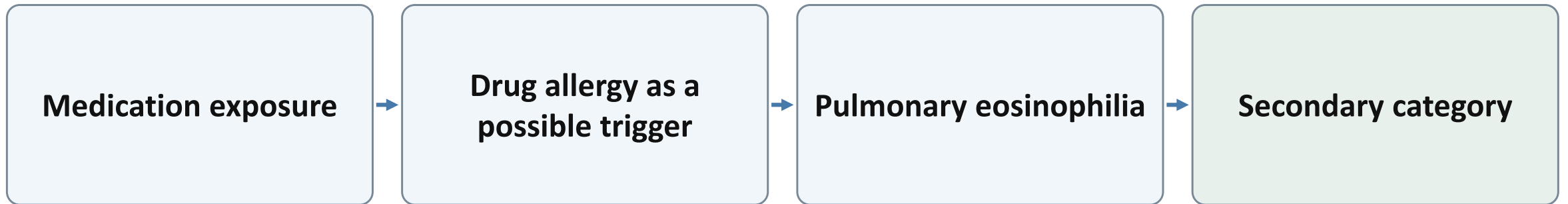
Schematic — not a histology image

Fungal and bacterial infection can also be associated

- Fungal infection appears in the Robbins list of causes.
- Bacterial infection also appears in the list.
- The excerpt does not identify a single pathogen, test, or diagnostic cutoff for either group.

Do not invent organism-specific rules from a general etiologic list.

Drug allergy belongs in the etiologic history



Drug allergy is a recognized context for secondary eosinophilia. The scheme shows classification, not a verified drug-specific pathway.

Hypersensitivity pneumonitis: Classification nuance

Pulmonary eosinophilia section

Lists hypersensitivity pneumonitis among secondary associations.

Adjacent hypersensitivity pneumonitis section

Typical subacute HP interstitial infiltrates feature lymphocytes, plasma cells, macrophages; Eosinophils are described as rare. A shared etiologic listing does not imply eosinophils dominate HP morphology.

Asthma is a recognized associated condition

- Asthma is among the associated conditions for secondary pulmonary eosinophilia.
- The association alone does not classify the pneumonia as idiopathic.
- Documenting asthma belongs to the background clinical assessment.

Allergic bronchopulmonary aspergillosis

- Allergic bronchopulmonary aspergillosis (ABPA) appears as a secondary association.
- An eosinophilic lung infiltrate alone does not establish an ABPA diagnosis.
- The name should prompt focused etiologic consideration rather than a conclusion based only on eosinophilia.

Churg–Strauss syndrome is a vasculitic association

- Churg–Strauss syndrome is among the associated disorders.
- It describes this disorder as a form of vasculitis.
- The lung findings should be considered in the broader systemic disease context.

An eosinophilic lung infiltrate alone cannot establish systemic vasculitis.

A practical secondary-cause inventory

Assessment domain	Named Robbins contexts
Infection	Parasitic; fungal; bacterial
Medication / allergy	Drug allergies
Inflammatory disease	Hypersensitivity pneumonitis
Associated disease	Asthma; ABPA; Churg–Strauss syndrome

Secondary vs idiopathic: The causal distinction

Secondary pulmonary eosinophilia

There is an infectious, allergic, inflammatory, or syndromic association.

Examples are specified by the source.

Idiopathic chronic eosinophilic pneumonia

The characteristic chronic pattern may be present.

Diagnosis requires exclusion of other causes of pulmonary eosinophilia.

Hypothetical case: An infectious clue

Teaching vignette (hypothetical)

A pulmonary eosinophilia workup identifies helminth larvae involving alveoli. The parasite belongs to one of the *Ascaris*, hookworm, or *Strongyloides* groups.

Why is “idiopathic chronic eosinophilic pneumonia” the wrong first label?

**Demonstrated larval
involvement**

Infectious cause identified

Secondary class

Hypothetical case: A medication clue

Teaching vignette (hypothetical)

A patient with eosinophilic lung infiltrates has a clinically relevant history of suspected medication allergy. No claim is made here that the medication caused the infiltrates.

Which etiologic category must remain under consideration?

Medication history

Possible allergic trigger

Cause not yet proven

Idiopathic chronic eosinophilic pneumonia

- The chronic idiopathic form has a characteristic clinicopathologic pattern.
- It typically produces focal areas of consolidation at the lung periphery.
- The process involves eosinophils and lymphocytes within alveolar septa and airspaces.
- Other causes must be excluded before assigning the diagnosis.

Peripheral consolidation is a distinctive clue

Anatomic distribution

Focal areas of consolidation occur at the periphery of the lungs.

This differs from the diffuse radiographic infiltrates described in the acute form.

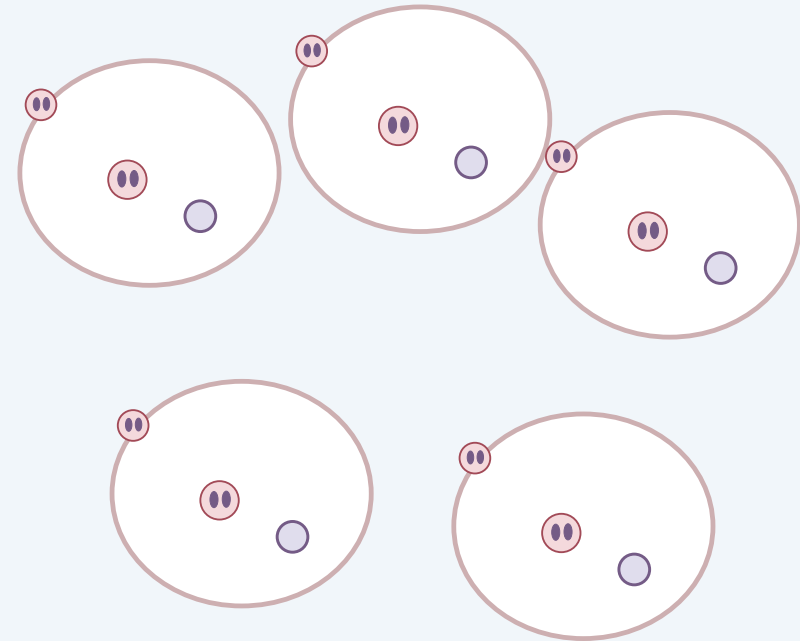
Diagnostic interpretation

Location is useful pattern information, not a standalone proof of etiology.

Integrate distribution with symptoms, inflammation, and exclusions.

Chronic morphology spans two compartments

- Eosinophils and lymphocytes infiltrate alveolar septal walls.
- The same inflammatory populations may occupy alveolar spaces.
- This combined distribution accounts for the alveolar and interstitial components.



Schematic — not a histology image

Eosinophils and lymphocytes coexist

The chronic form is not described as an eosinophil-only lesion.

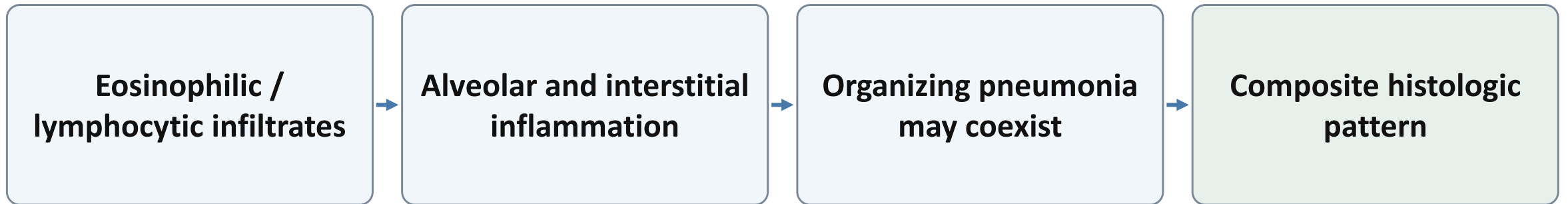
- Inflammatory infiltrates include lymphocytes as well as eosinophils.
- Both cell types occur within septal walls and airspaces.
- The cell mixture helps define the stated histologic pattern.

Eosinophils

Lymphocytes

Septal + airspace

Organizing pneumonia may accompany chronic disease



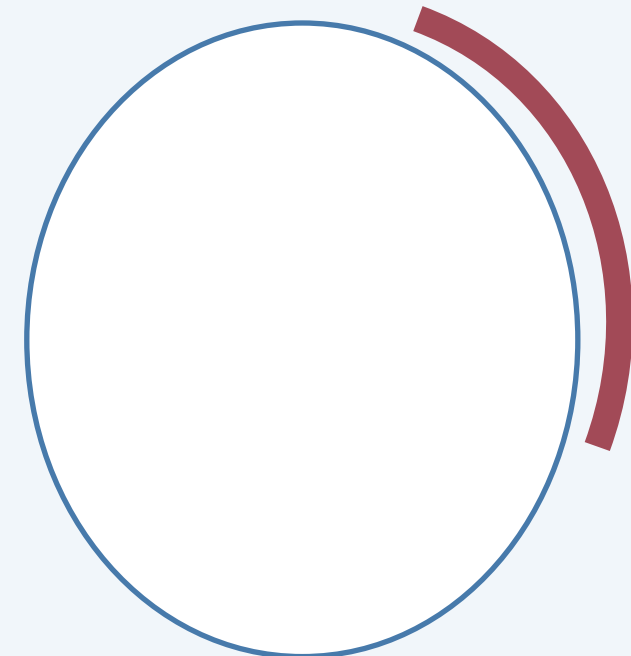
Organizing pneumonia is often present in the chronic form. This flow is an interpretative grouping, not proof that one event causes the next.

Interstitial fibrosis may also be present

- Interstitial fibrosis often accompanies chronic eosinophilic pneumonia.
- The fibrosis constitutes an additional tissue-remodeling feature.
- The excerpt does not specify fibrosis stage, magnitude, or prognostic quantification.

Consolidation reflects the distribution of inflammatory injury

- The characteristic described gross/radiologic distribution is peripheral.
- Underlying alveolar and septal infiltrates accompany the areas of consolidation.
- The lung silhouette is a conceptual distribution map, not a patient CT or radiograph.



Peripheral focus

Schematic distribution

Chronic symptoms: Respiratory complaints

- Cough is one of the clinical complaints.
- Dyspnea is also typical of the chronic presentation.
- The pulmonary symptoms are accompanied by constitutional manifestations in the source.

Chronic symptoms: Constitutional illness

Fever

Systemic symptom

Listed

Night sweats

Systemic symptom

Listed

Weight loss

Systemic symptom

Listed

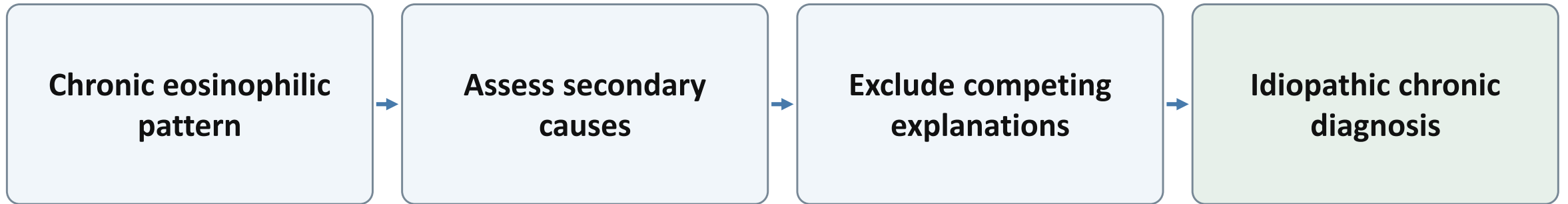
Chronic symptom cluster must be integrated

Pulmonary findings	Systemic findings	Morphologic context
Cough	Fever	Septal + airspace inflammation
Dyspnea	Night sweats	Peripheral consolidation
—	Weight loss	Organizing pneumonia / fibrosis often present

Corticosteroid responsiveness in chronic disease

- Chronic cough, fever, sweats, dyspnea, and weight loss may respond to corticosteroid therapy.
- A prompt corticosteroid response is also described in the acute category.
- Therefore, steroid response is shared information and cannot by itself identify which category is present.

Chronic disease is a diagnosis of exclusion



The final exclusion requirement is explicit; The intervening evaluation logic is an educational inference, not a formal guideline algorithm.

Assess the cause before using “idiopathic”

Look for	Examples from the supplied source
Infection	Parasites / fungi / bacteria
Drug allergy	Potential medication-related eosinophilia
Associated disease	Asthma; ABPA; vasculitis
Other inflammatory context	Hypersensitivity pneumonitis

Hypothetical chronic case: Recognize the pattern

Teaching vignette (hypothetical)

A patient reports cough, dyspnea, fever, night sweats, and weight loss. Imaging shows focal peripheral consolidation. Tissue has eosinophils and lymphocytes in alveolar spaces and septal walls.

Which category is suggested, and what condition remains before the idiopathic label?

Peripheral consolidation

Mixed infiltrate

Constitutional symptoms

Chronic case: Associated histologic findings

Principal pattern

Peripheral consolidation

Eosinophils + lymphocytes in septa and airspaces

Additional findings often present

Interstitial fibrosis

Organizing pneumonia

Neither is presented here as a stand-alone diagnostic criterion.

Acute versus chronic: The tempo and severity

Domain	Acute form	Chronic idiopathic form
Tempo	Rapid onset	Chronic presentation
Core complaints	Fever, dyspnea	Cough, fever, sweats, dyspnea, weight loss
Respiratory failure	Hypoxemic failure characteristic	Not specified as defining in excerpt
Steroid response	Prompt	Symptoms respond

Acute versus chronic: Anatomic distribution

Acute eosinophilic pneumonia

Diffuse infiltrates on chest radiograph

Diffuse alveolar damage with numerous eosinophils

Idiopathic chronic eosinophilic pneumonia

Peripheral focal consolidation

Alveolar and septal lymphocytic/eosinophilic infiltrates

Acute versus chronic: Morphology comparison

Morphologic clue	Acute	Chronic
Dominant described injury	Diffuse alveolar damage	Interstitial and airspace infiltrates
Eosinophils	Many	Present with lymphocytes
Other processes	Not specified in excerpt	Organizing pneumonia / fibrosis often present

Only one numeric BAL cutoff is supplied

>25%

- The chronic description instead emphasizes clinical, radiographic, and tissue features.
- The secondary category is classified by causes and associations.
- Do not treat the acute BAL threshold as a universal criterion for every eosinophilic syndrome.

BAL eosinophils — specifically described for the acute category

Corticosteroid response is not disease-specific

Acute eosinophilic pneumonia

Prompt corticosteroid response described.

Idiopathic chronic eosinophilic pneumonia

Cough, fever, sweats, dyspnea, and weight loss respond to corticosteroids.

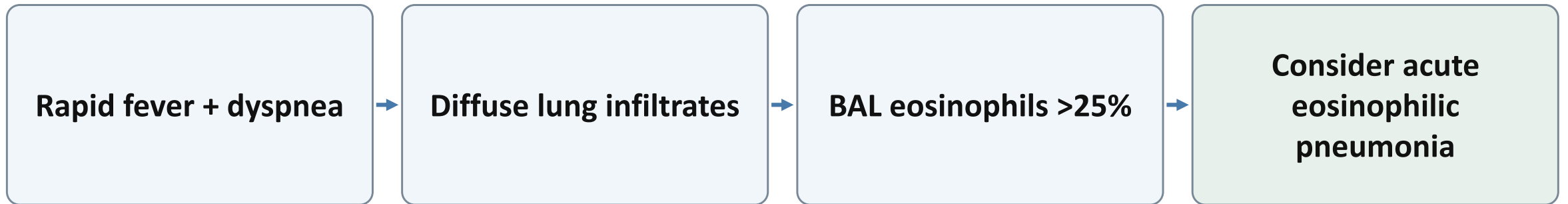
Response does not eliminate the need to exclude secondary causes.

Eosinophil-rich histology is not the whole diagnosis

- An eosinophilic infiltrate is shared by all three categories.
- Distribution, additional pathology, clinical course, and etiologic context carry discriminating information.
- The idiopathic chronic label is constrained by explicit exclusion of other causes.

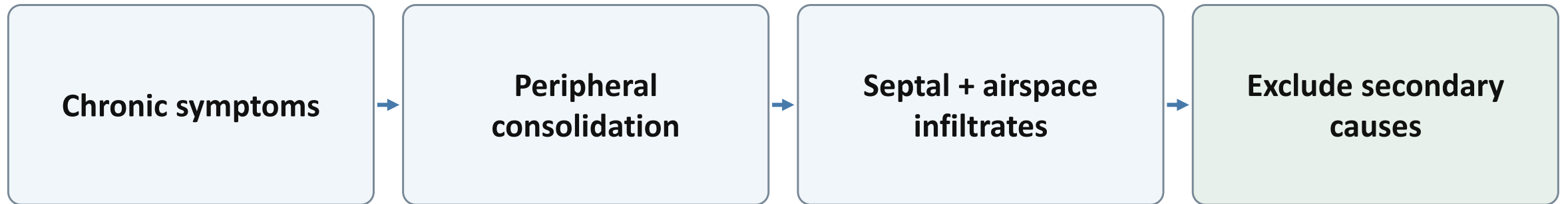
Avoid assigning a final category from eosinophils alone.

Diagnostic interpretation: Acute branch



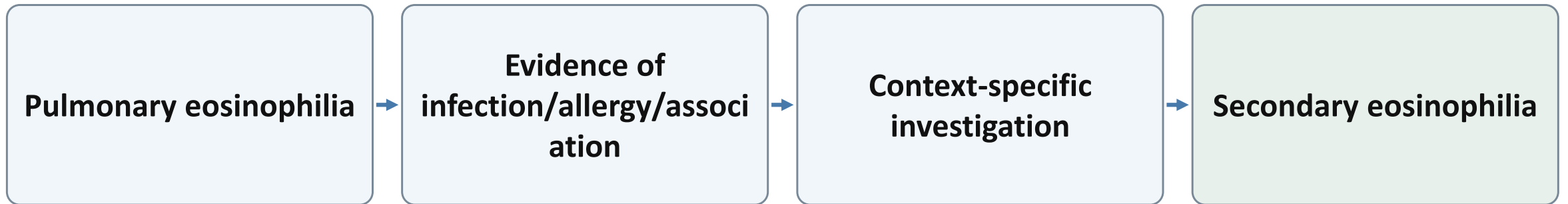
Correlate with the severe hypoxemic syndrome and the stated histologic pattern; This is a source-based teaching pathway, not a validated algorithm.

Diagnostic interpretation: Chronic branch



If competing causes are excluded, the source supports idiopathic chronic eosinophilic pneumonia.

Diagnostic interpretation: Secondary branch



The source supplies etiologic categories, not specific investigations or validated diagnostic test panels.

Three branches within one diagnostic framework

Rapid + diffuse

Fever / dyspnea / hypoxemia

Acute eosinophilic pneumonia

Context-linked

Infection / drug / associated illness

Secondary eosinophilia

Chronic + peripheral

Mixed septal/airspace infiltrates

Idiopathic only after exclusion

Knowledge check: Acute classification

Teaching vignette (hypothetical)

Rapid fever, dyspnea, hypoxemic respiratory failure, diffuse infiltrates, and BAL eosinophils above 25%.

What additional tissue finding is explicitly described for this acute category?

Acute respiratory failure

BAL eosinophilia

Diffuse imaging

Knowledge check: Cause attribution

Teaching vignette (hypothetical)

An eosinophilic pulmonary infiltrate occurs in an individual with a documented relevant helminth infection involving alveolar larvae.

Which grouping and named illustrative syndrome are appropriate?

Known parasitic infection

Larval alveolar involvement

Eosinophilic infiltrate

Knowledge check: Chronic classification

Teaching vignette (hypothetical)

Peripheral areas of consolidation accompany septal and alveolar infiltrates of lymphocytes and eosinophils; Organizing pneumonia is seen.

What prevents immediate assignment of an “idiopathic” diagnosis?

Chronic morphology

Peripheral consolidation

Cause not yet assessed

Integrative summary: Five decision anchors

- Pulmonary eosinophilia is a shared inflammatory pattern comprising distinct syndromes.
- IL-5 is one of the alveolar signals implicated in eosinophil recruitment.
- Acute disease combines diffuse infiltrates, BAL eosinophilia, and diffuse alveolar damage.
- Secondary disease demands attention to infection, drug allergy, and associated disorders.
- Chronic peripheral consolidation with mixed infiltrates requires exclusion before an idiopathic diagnosis.

Textbook reference

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition (2026). Chapter 15: The Lung. Pulmonary Eosinophilia, pp. 636–637.
- Adjacent section consulted: Hypersensitivity Pneumonitis, p. 636, for the stated nuance regarding rarity of eosinophils in typical HP morphology.
- The lecture focuses on the textbook descriptions of acute, secondary, and idiopathic chronic pulmonary eosinophilia.