Pulmonary Eosinophilia

:Understanding the Spectrum from Transient Infiltrates to Chronic Disease

2026.7.11

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1. Definition & Classification of Pulmonary Eosinophilia

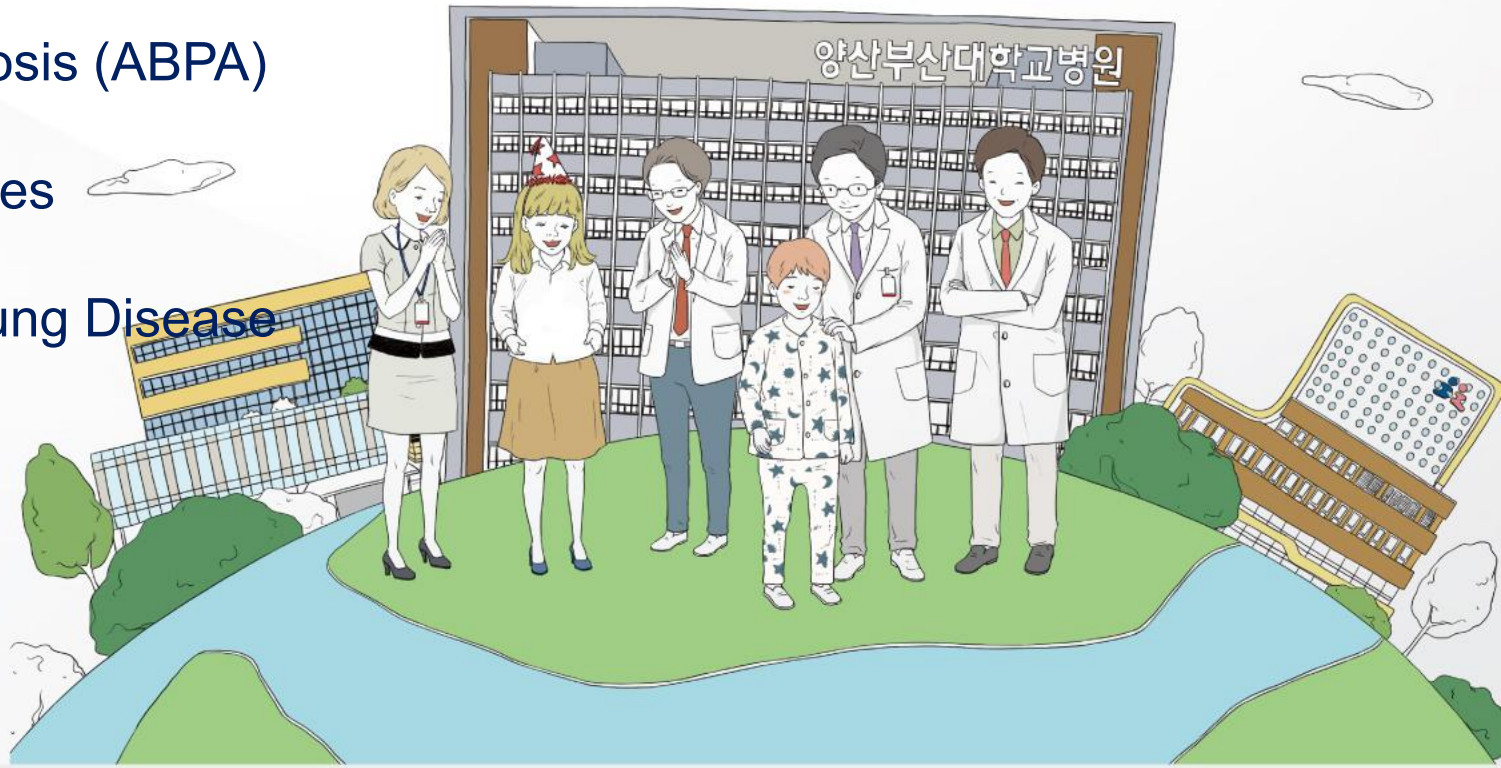
2. Idiopathic Eosinophilic Pneumonia (IAEP & ICEP)

3. Allergic Bronchopulmonary Aspergillosis (ABPA)

4. Secondary Eosinophilic Lung Diseases

5. Practical Approach to Eosinophilic Lung Disease

6. Take-home Messages



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Definition

Eosinophilic Lung Disease

- Diffuse parenchymal lung disease characterized by the prominent infiltration of Eosinophilic infiltration of the lung interstitium and alveolar spaces
- Preservation of normal lung architecture
- Considered part of the ILD spectrum
- Typically responds rapidly to systemic corticosteroids
- Minimal or no residual lung damage after treatment

Definitions of eosinophilia

Table 1
Definitions of eosinophilia in clinical practice

Terminology	Eosinophil Blood Count	Reference
Blood eosinophilia	>0.5 × 10^9/L (500/μL), or >6% in differential counts	Benson et al,[5] 2022
Blood hypereosinophilia	>1.5 × 10^9/L (1500/μL) on 2 examinations at least 1 mo apart	Valent et al,[1] 2023
Mild blood eosinophilia	0.5–1.5 × 10^9/L (500–1500/μL)	Valent et al,[1] 2023
Moderate blood eosinophilia	1.5–5 × 10^9/L (1500–5000/μL)	Valent et al,[1] 2023
Severe blood eosinophilia	>5 × 10^9/L (5000/μL)	Valent et al,[1] 2023
Alveolar eosinophilia	Differential cell count of at least 25% eosinophils at bronchoalveolar lavage	Cottin,[2] 2023

Strongly suggestive of eosinophilic lung disease

- Blood eosinophils ≥1,500 cells/μL + Pulmonary infiltrates
- BAL eosinophils >40%

Classification of Eosinophilic Lung Diseases

Table 2
Classification of the eosinophilic interstitial lung diseases in clinical practice

	Acute Onset	Chronic Onset
Only pulmonary manifestations		
Idiopathic	Idiopathic acute eosinophilic pneumonia	Idiopathic chronic eosinophilic pneumonia
Identified cause or trigger	Löffler syndrome Acute eosinophilic pneumonia induced by smoking or airborne dust Acute eosinophilic pneumonia due to medications Acute eosinophilic pneumonia due to illicit drugs Acute eosinophilic pneumonia due to infections	Allergic bronchopulmonary aspergillosis and related syndromes Eosinophilic pneumonias of parasitic origin Eosinophilic pneumonias of other infectious causes Eosinophilic pneumonias due to radiation therapy
With systemic manifestations		
Idiopathic	Eosinophilic granulomatosis with polyangiitis Eosinophilic vasculitis	Hypereosinophilic syndromes
Identified cause or trigger	DRESS Parasitic infection	

Diagnostic algorithm for EP

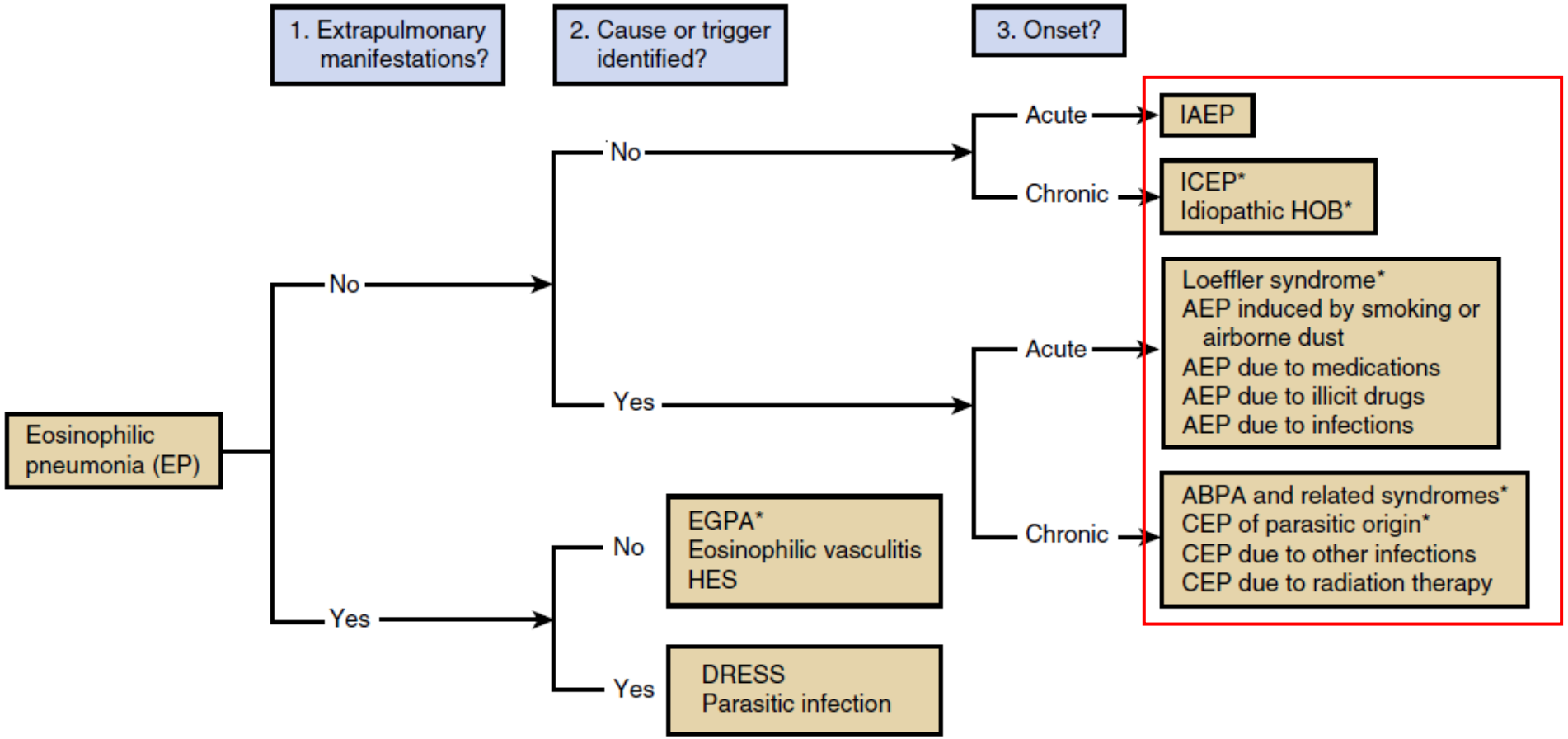

Figure 96.1 Diagnostic algorithm for eosinophilic pneumonia. The initial diagnostic question is whether there are extrapulmonary manifestations. *Conditions commonly associated with asthma or airflow obstruction. ABPA, allergic bronchopulmonary aspergillosis; AEP, acute eosinophilic pneumonia; CEP, chronic eosinophilic pneumonia; DRESS, drug reaction with eosinophilia and systemic symptoms; EGPA, eosinophilic granulomatosis with polyangiitis; HES, hypereosinophilic syndrome; HOB, hypereosinophilic obliterative bronchiolitis; IAEP, idiopathic acute eosinophilic pneumonia; ICEP, idiopathic chronic eosinophilic pneumonia.

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IAEP

Typical patient

- Young adults (mean age ~30 years)
- Male predominance
- Usually non-asthmatic

Smoking-related Triggers

- 2/3 of patients have a smoking history
- New/increased smoking
- Smoking re-initiation
- Passive smoking
- E-cigarettes

Other triggers

- Environmental/occupational inhalational exposure
- Drugs
- Infections (parasites, fungi, viruses)

Pathophysiology of AEP

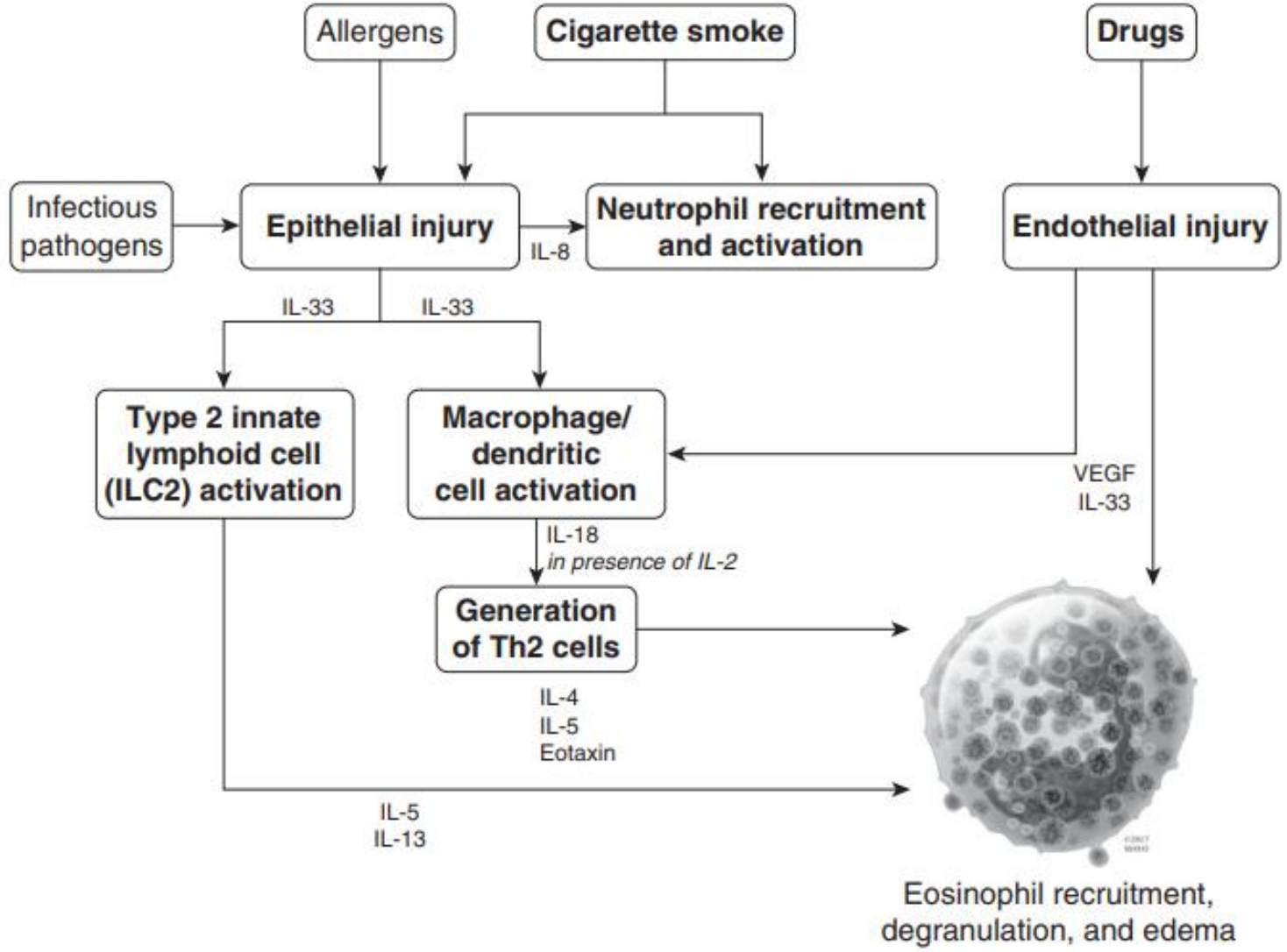


Figure 1. A proposed model for the pathogenesis of acute eosinophilic pneumonia. IL-33 may be

Am J Respir Crit Care Med . 2018 Mar 15;197(6):728-736.

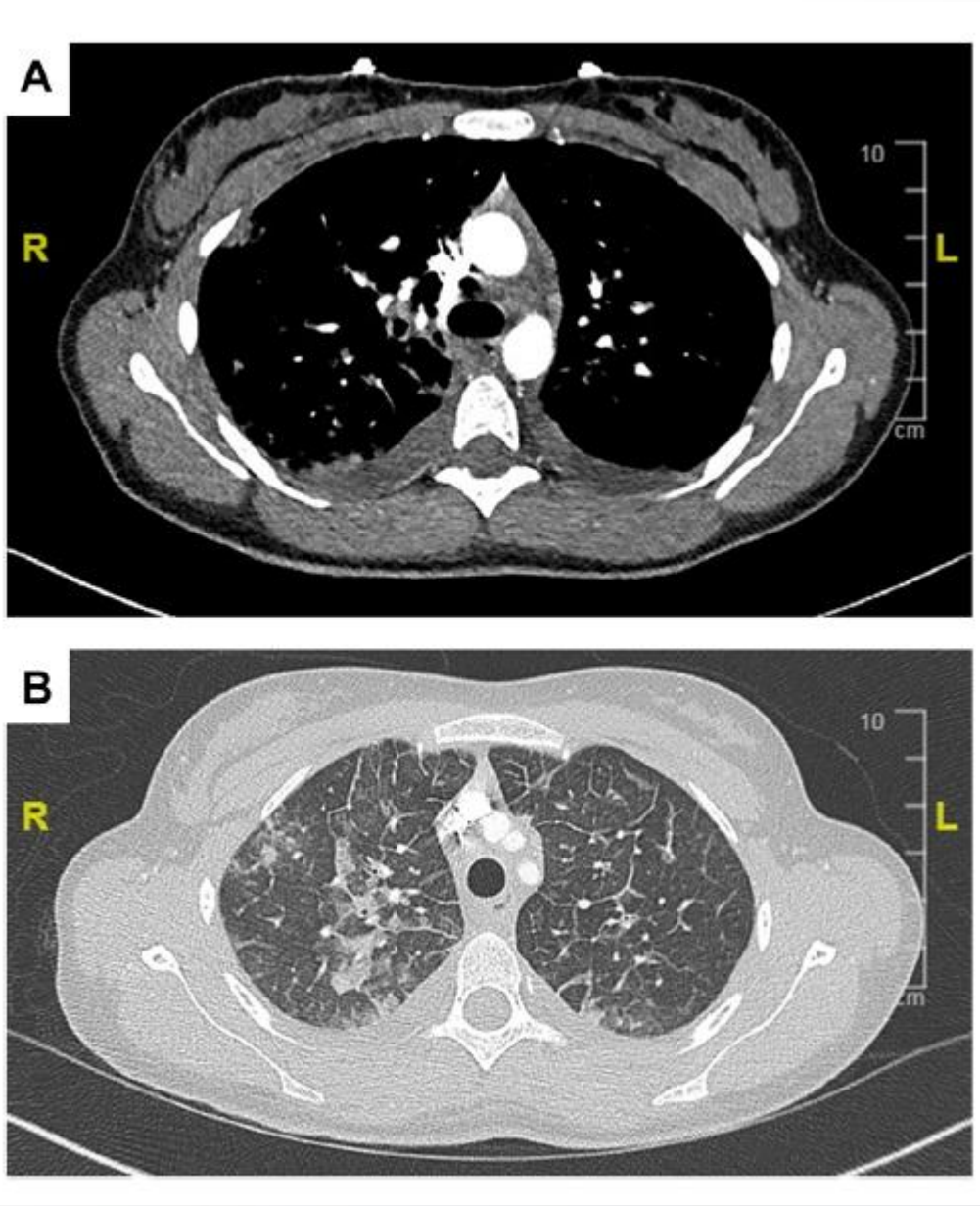
IAEP

Symptoms

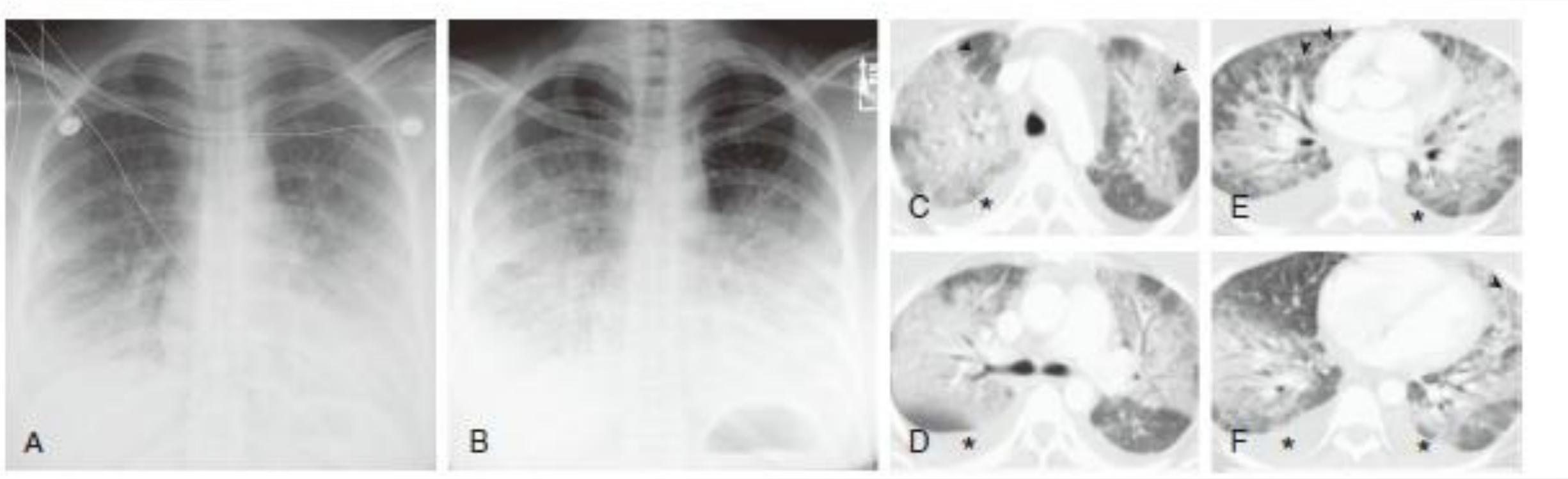
- Acute onset (<7 days)
- Dyspnea + Fever
- Cough
- Pleuritic chest pain
- Acute respiratory failure

Imaging Finding

- Diffuse bilateral GGO
- Septal thickening
- Pleural effusion
- Consolidation
- May mimic pulmonary edema



IAEP



Murray & Nadel's Textbook of Respiratory Medicine, 7th ed. Elsevier, 2022.

IAEP

Blood

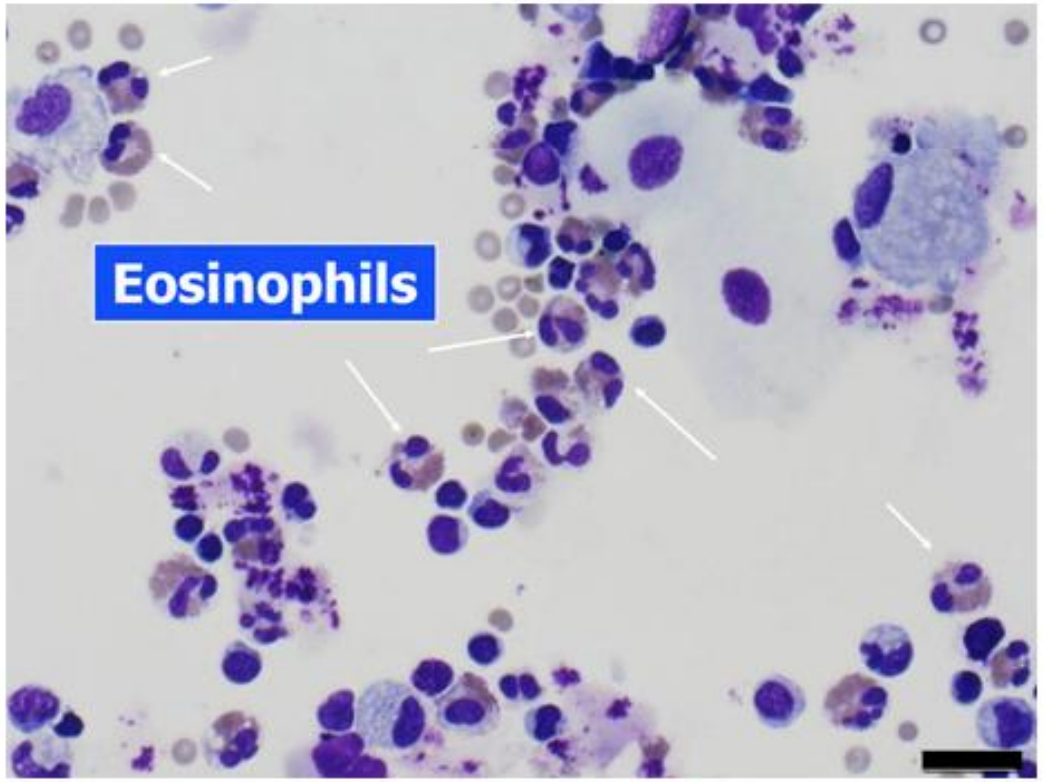
- Peripheral eosinophils often normal initially
- Eosinophilia develops within 1–7 days

BAL

- Eosinophils >25% (diagnostic)
- BAL culture usually sterile

Pulmonary Function

- Severe hypoxemia
- Mild restrictive pattern
- ↓ DLCO
- ↑ A–a oxygen gradient



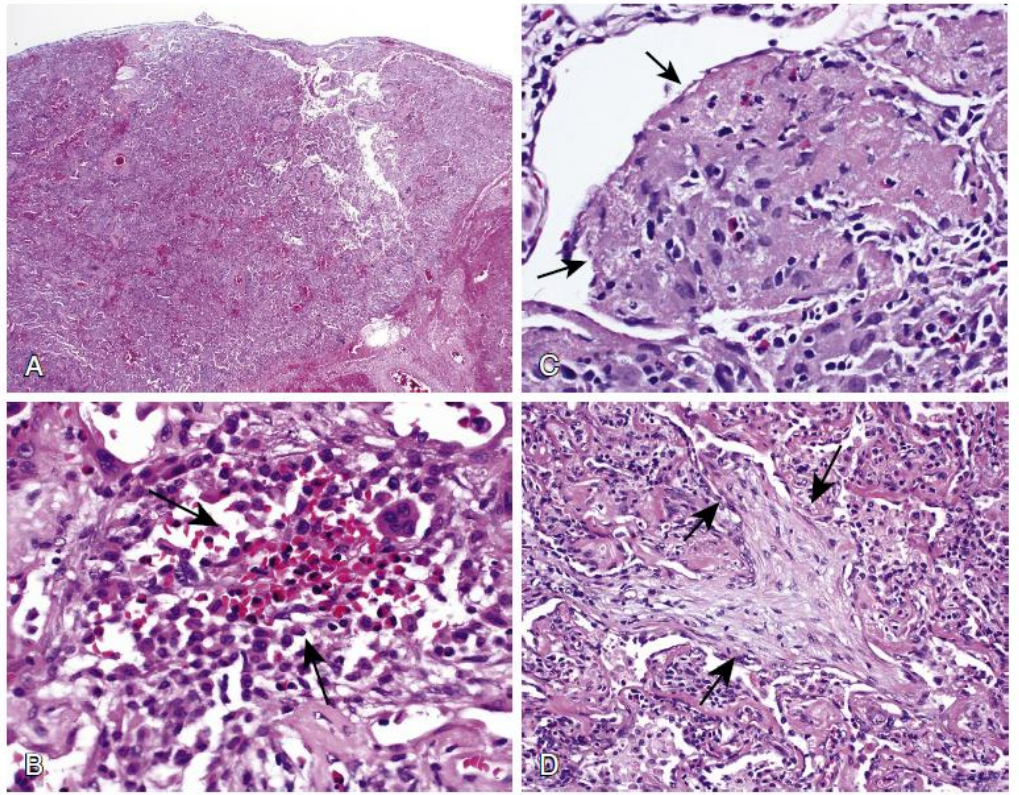
IAEP

Pathology

- Lung biopsy rarely required(performed only when the diagnosis is uncertain)
- Diffuse intra-alveolar eosinophilic infiltration
- Interstitial edema

Diagnosis

- BAL eosinophils >25% (key diagnostic finding)
- Exclude infection
- Search for smoking, inhalational, drug, or infectious causes



Clin Chest Med . 2025 Dec;46(4):667-684., Murray & Nadel's Textbook of Respiratory Medicine, 7th ed. Elsevier, 2022.

IAEP

Treatment

- Systemic corticosteroids (first-line)
- Prednisone 30 mg/day (Higher doses for severe cases)
- Rapid response
  - Clinical improvement within 3 days
  - Treatment duration: 2 weeks
  - Imaging & PFT normalize within 1 month

Prognosis

- Excellent prognosis with prompt treatment
- Relapse is rare
- Usually occurs after re-exposure to smoking
- Smoking cessation is essential

ICEP

Typical patient

- Female predominance (mean age ~45 years)
- Non-smokers
- Asthma (~67%)
- Atopy (~50%)

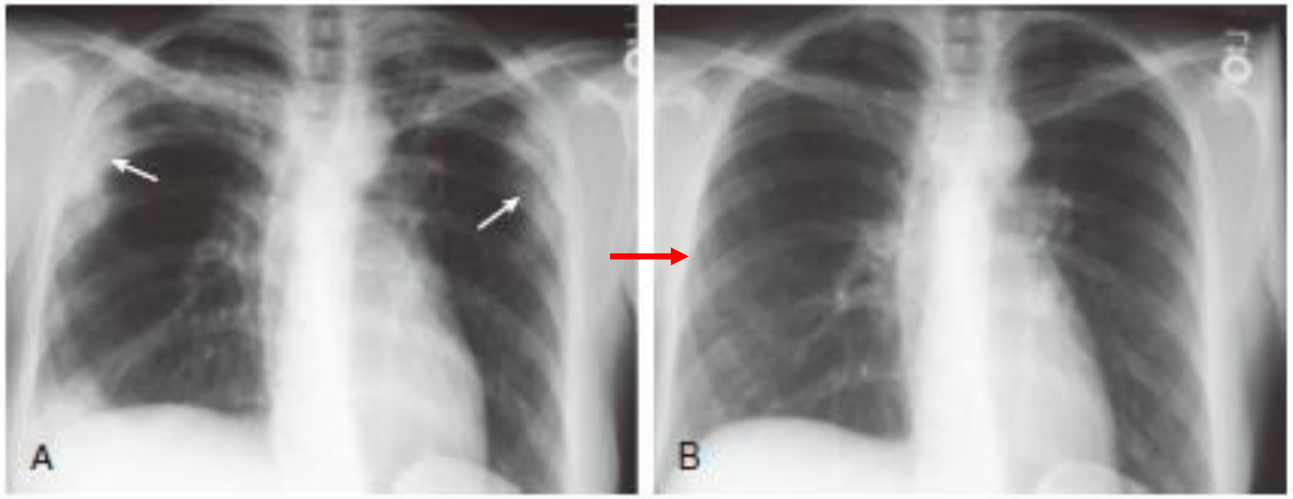
Symptoms

- Subacute onset (>2–4 weeks)
- Dyspnea
- Cough (~90%)
- Malaise / weight loss
- Respiratory failure is uncommon

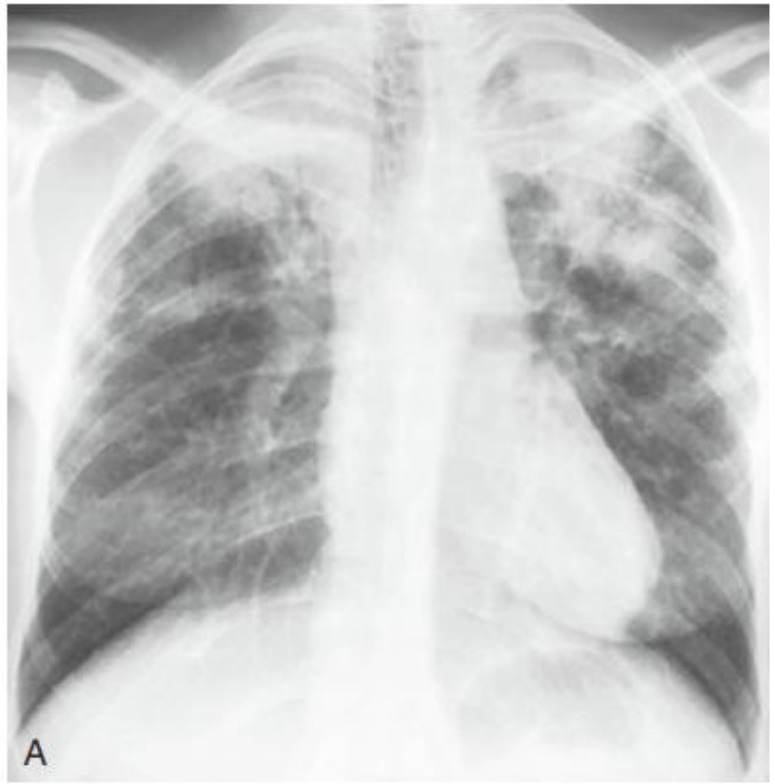
ICEP

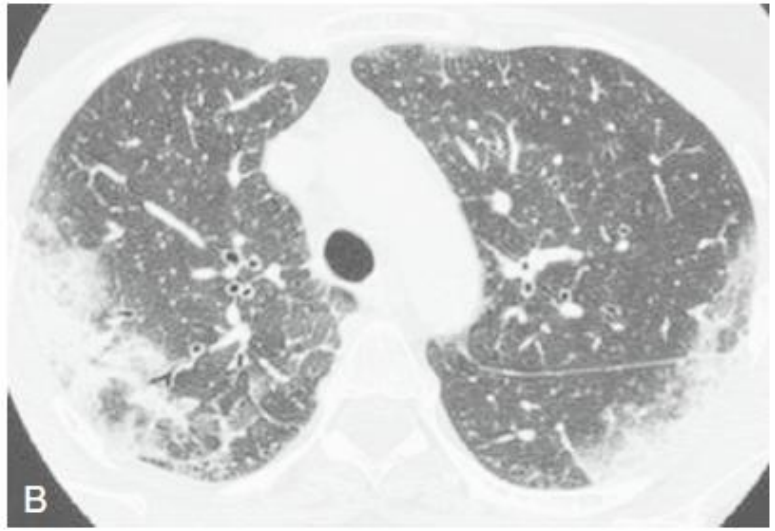
Imaging Finding

- Bilateral peripheral consolidations ± GGOs
- Upper lobes & subpleural predominance
- Migratory pulmonary opacities (25%)
- Rapid radiologic response to corticosteroids



Before treatment **After corticosteroids**





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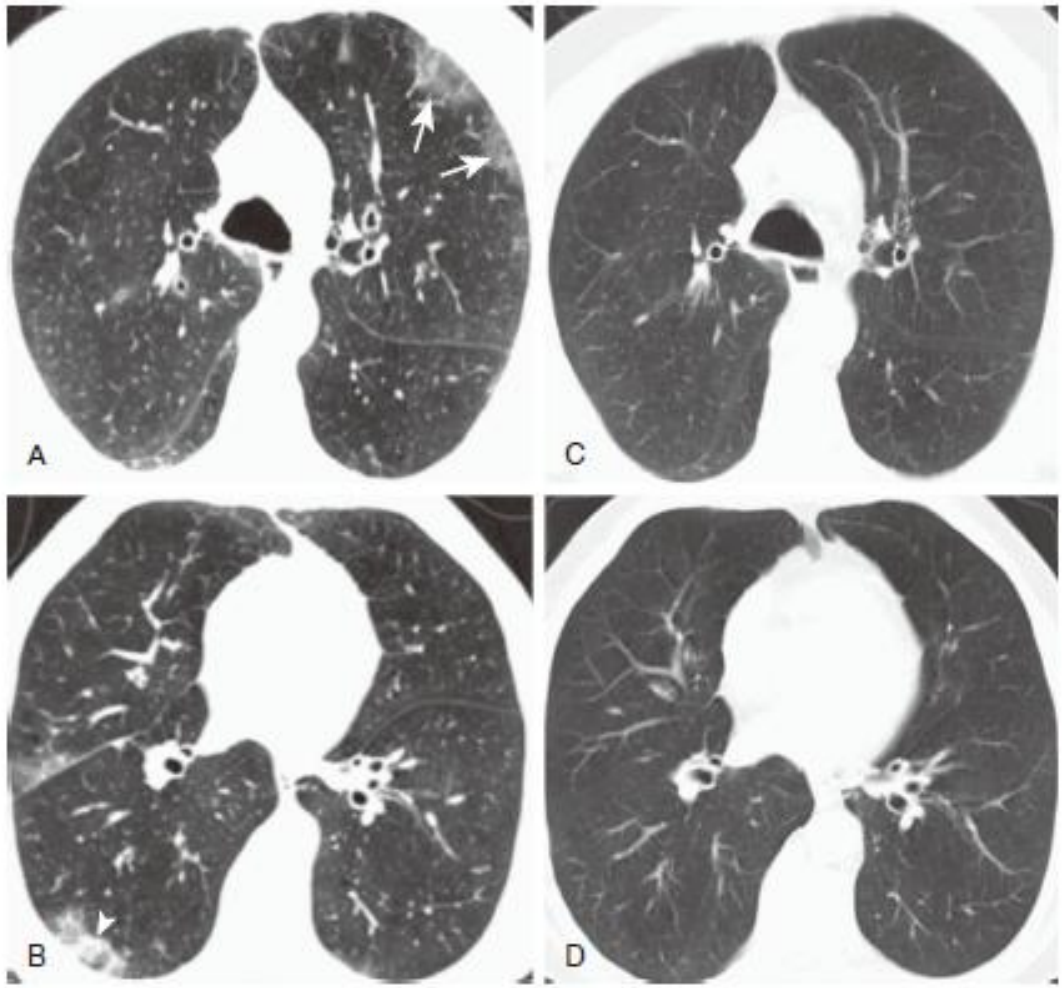
ICEP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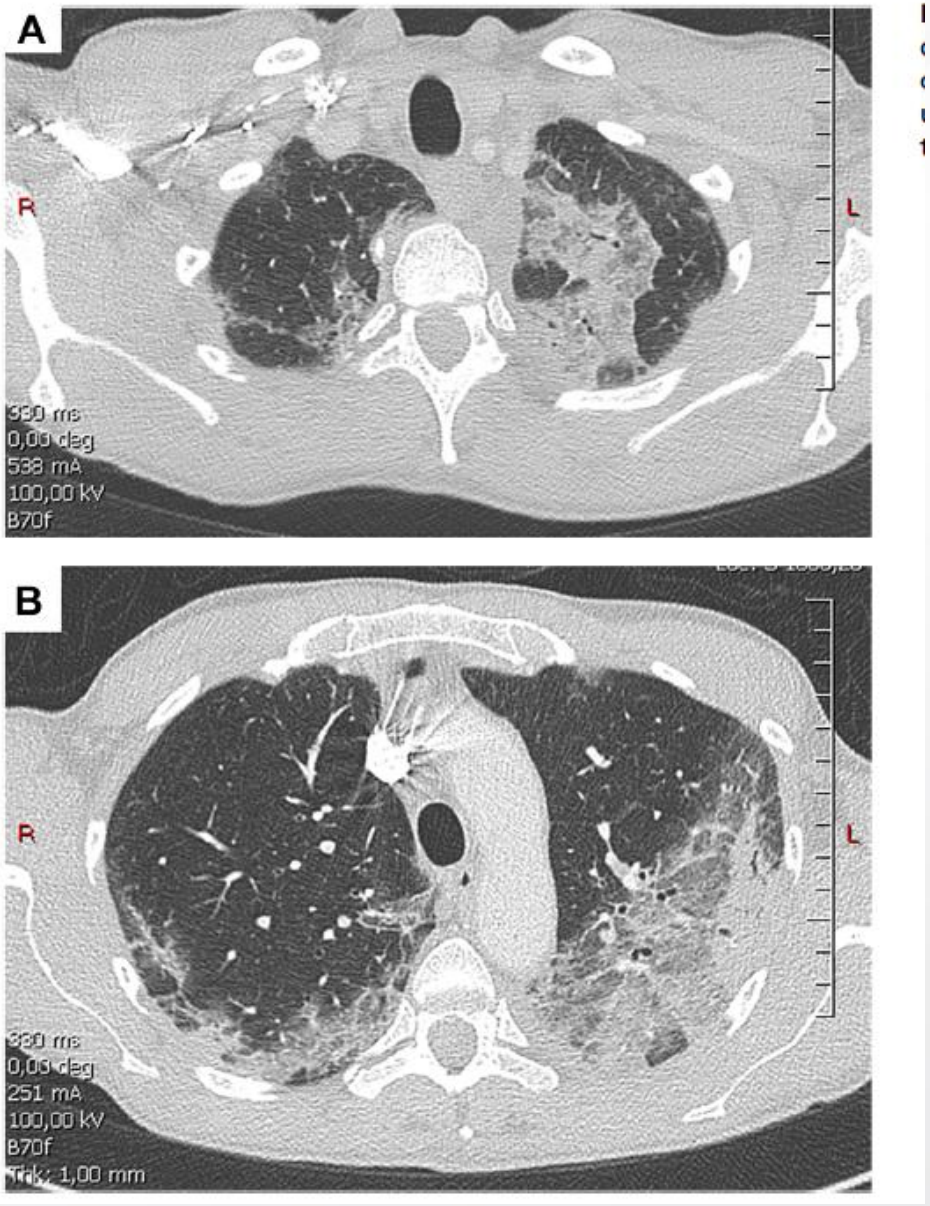
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ICEP

Laboratory Findings

- Marked peripheral eosinophilia
- BAL eosinophils >40%
- Elevated CRP / Total IgE *(non-specific)*

Pulmonary Function

- Airflow obstruction (~50%)
- Restrictive defect (~50%)
- Mild hypoxemia
- Persistent airflow limitation despite treatment

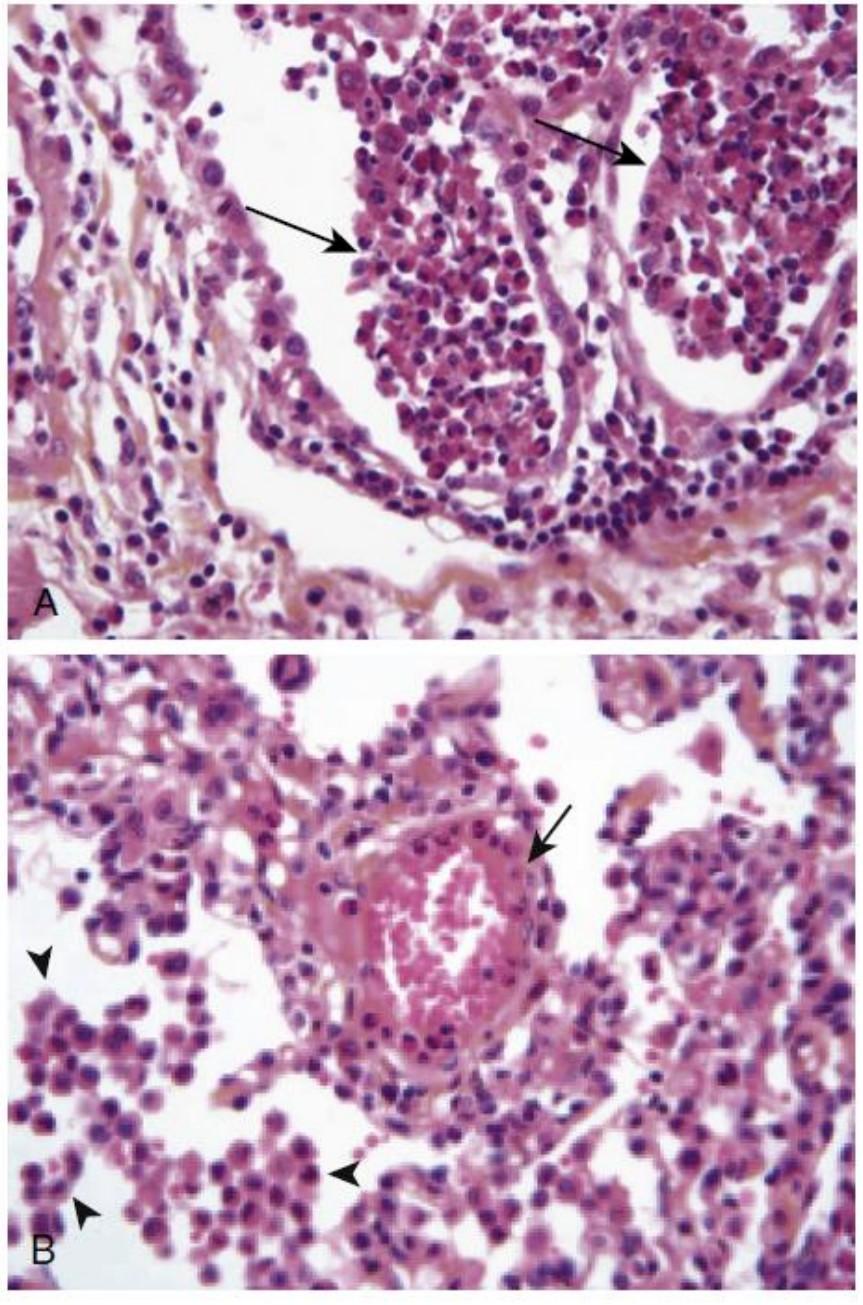
ICEP

Pathology

- Lung biopsy rarely required
- Eosinophilic infiltration of the alveoli and interstitium
- Lung architecture is preserved

Diagnosis

- Exclude secondary causes (drugs, parasites, fungi, toxins)
- Typical imaging + blood eosinophilia
- BAL eosinophils >40%



Clin Chest Med . 2025 Dec;46(4):667-684., Murray & Nadel's Textbook of Respiratory Medicine, 7th ed. Elsevier, 2022.

ICEP

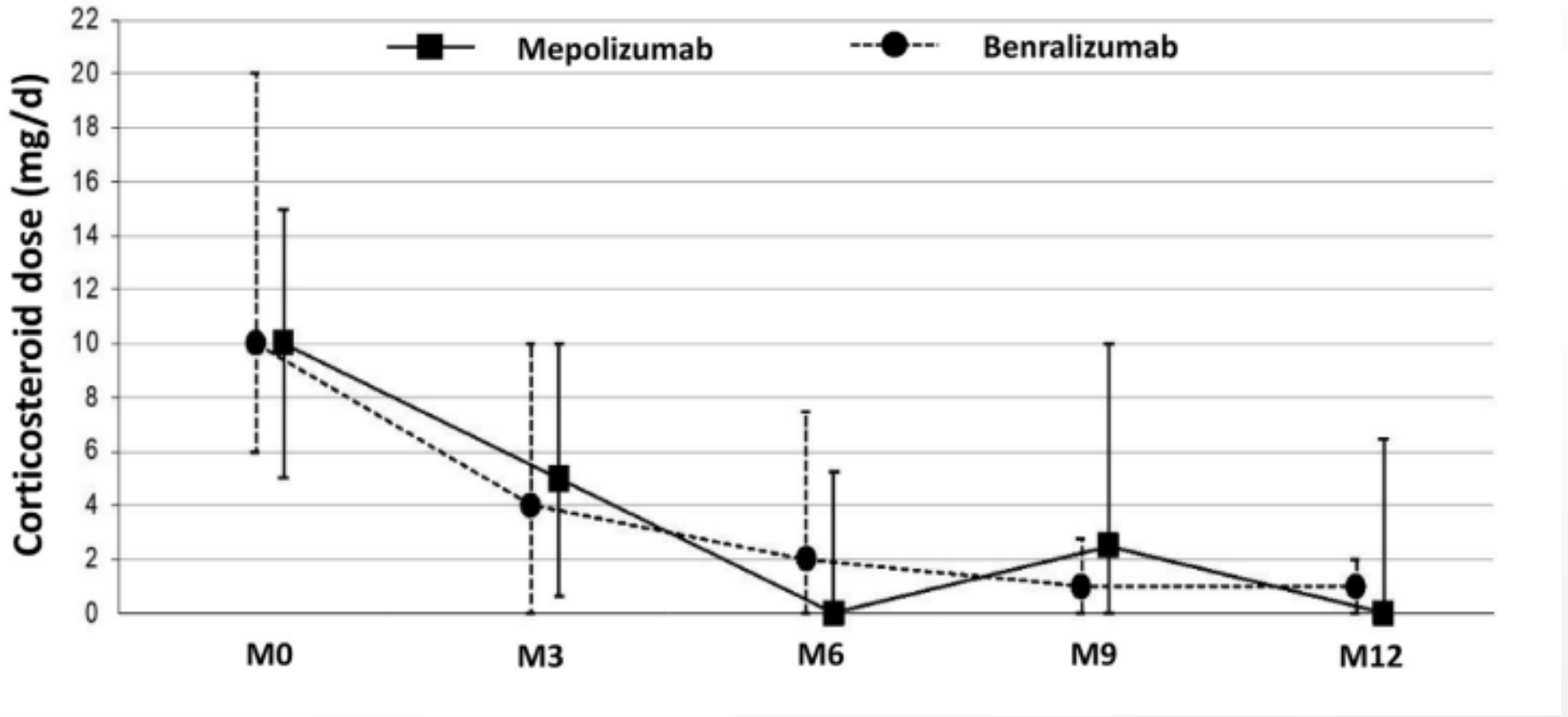
Treatment

- Oral glucocorticoids (first-line)
- Rapid response:
  - Clinical improvement within 48 hours
  - Radiographic improvement within 1 week
- Gradual taper over ~3 months due to the high risk of relapse

Prognosis

- Relapse occurs in >50% during tapering or after discontinuation
- Relapses usually respond well to prednisone
- ICS not effective for relapse prevention
- Mepolizumab/Benralizumab may be considered in selected relapsing or steroid-dependent patients

Biologics for Relapsing ICEP



- French multicenter retrospective cohort study
- 29 patients with relapsing and/or steroid-dependent ICEP
- Evaluated the efficacy and steroid-sparing effect of mepolizumab or benralizumab

IAEP vs ICEP

	AEP	CEP
Onset	Acute (<1 month, usually <7 days)	Subacute (>2–4 weeks)
Typical patient	Young male	Middle-aged female
Asthma	Rare	Common (~67%)
Smoking history	~2/3 smokers, often recent initiation	Usually non-smoker
Respiratory failure	Common	Rare
Blood eosinophils	Often normal initially (delayed)	Present at diagnosis
BAL eosinophils	> 25% (marked)	> 40%
Imaging	Diffuse bilateral GGO, septal thickening, pleural effusion	Peripheral consolidation ± GGO
Relapse	Rare	Common (>50%)
Steroid response	Dramatic, rapid	Excellent but relapses on taper — may need months

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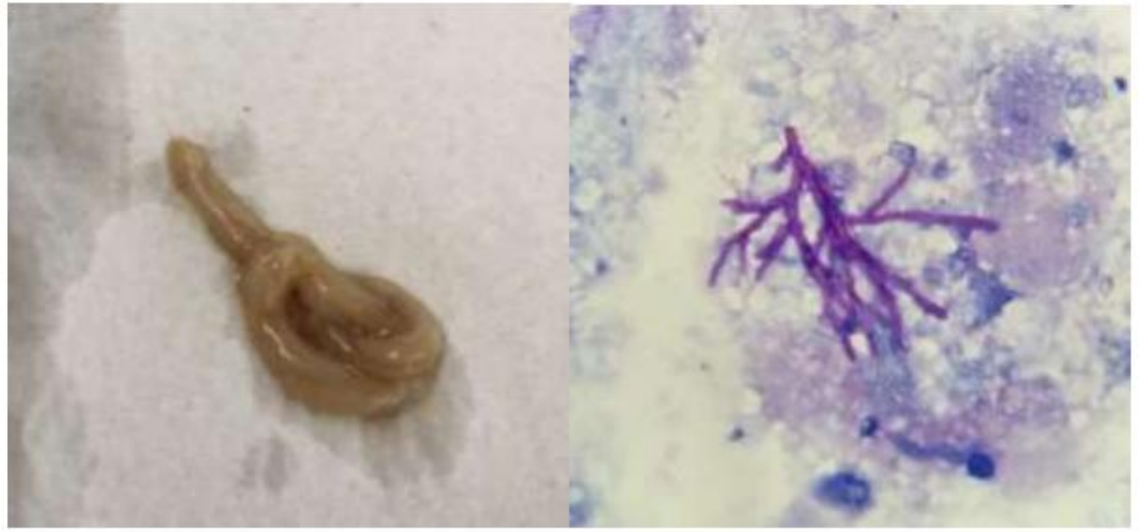
ABPA

Typical patient

- Occurs almost exclusively in patients with chronic airway diseases
  - Asthma (ABPA in ~1–2% of asthma patients)
  - Cystic fibrosis (ABPA in ~7–10% of CF patients)
  - Rare in COPD
- Caused by hypersensitivity to *Aspergillus fumigatus*
- Type I (IgE-mediated) and Type III hypersensitivity with Th2 immune response

Symptoms

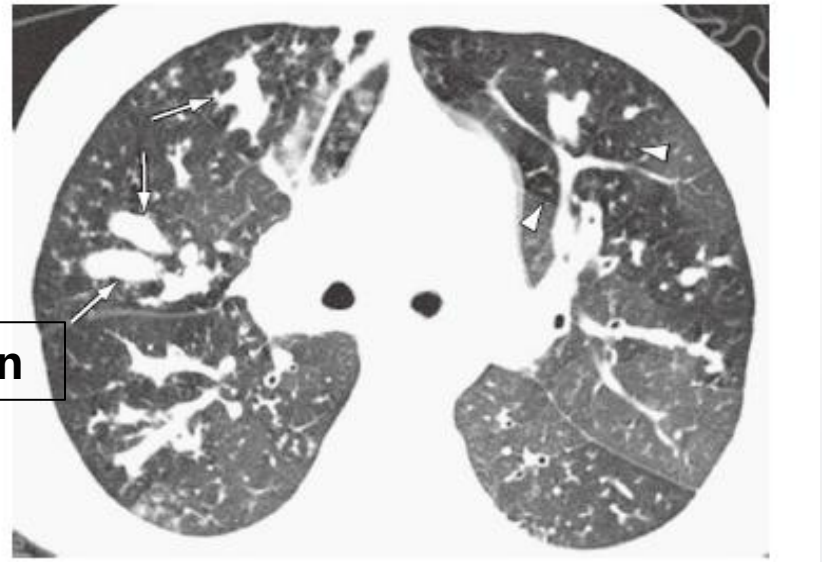
- Chronic/recurrent cough
- Wheezing
- Brown mucus plugs
- Recurrent exacerbations



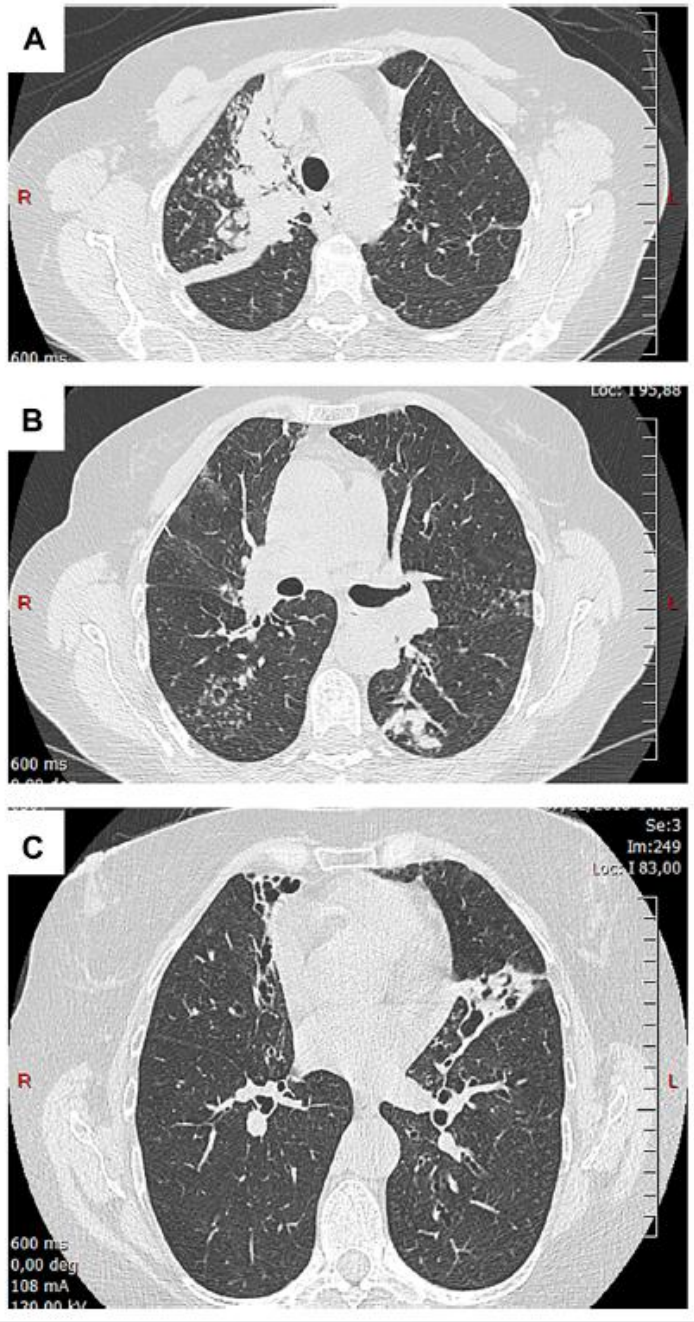
ABPA

Imaging Finding

- Central bronchiectasis (upper lobe predominance)
- Mucoid impaction (Finger-in-glove sign)
- Bronchial wall thickening
- GGO / consolidation
- Centrilobular nodules / tree-in-bud pattern



Finger-in-glove sign



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Diagnostic criteria for ABPA (2024 ERS/ISHAM)

진단기준	내용

선행 조건	• 기저질환
	  • Asthma
	  • Cystic fibrosis
	  • COPD
	  • Bronchiectasis
	• 또는 ABPA를 시사하는 임상·영상 소견

필수 항목	• A. fumigatus 특이 IgE ≥ 0.35 kUA/L • Total IgE ≥ 500 IU/mL

추가 항목(2가지 이상)	• fumigatus 특이 IgG 양성
	• 혈액 호산구 ≥500 cells/μL
	• 흉부 CT에서 ABPA에 합당한 소견
	  • Central bronchiectasis
	  • Mucus plugging
	  • High attenuation mucus

Clin Chest Med . 2025 Dec;46(4):667-684., Murray & Nadel's Textbook of Respiratory Medicine, 7th ed. Elsevier, 2022.

2024 ERS/ISHAM guideline

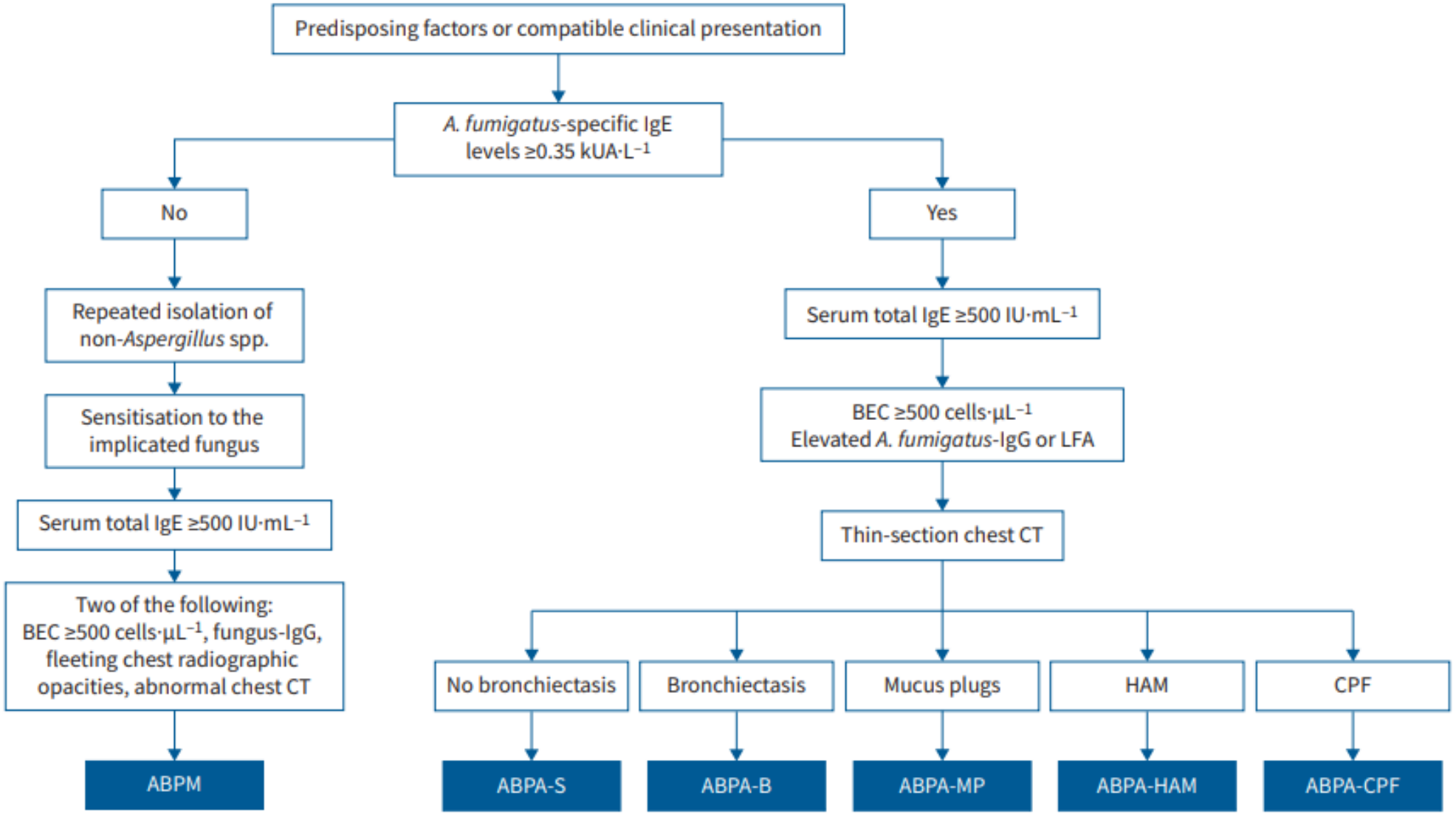
TABLE 1 Revised International Society for Human and Animal Mycology (ISHAM)-ABPA working group consensus criteria for diagnosing allergic bronchopulmonary aspergillosis (ABPA)

Predisposing conditions (asthma, cystic fibrosis, chronic obstructive lung disease, bronchiectasis) or [a]compatible clinico-radiological presentation
Essential components
[b]*A. fumigatus*-specific IgE ⩾0.35 kUA·L^{-1}
[c]Serum total IgE ⩾500 IU·mL^{-1}
Other components (any two)
[d]Positive IgG against *A. fumigatus*
Blood eosinophil count ⩾500 cells·µL^{-1} (could be historical)
Thin-section chest computed tomography consistent with ABPA (bronchiectasis, mucus plugging and [e]high-attenuation mucus) or fleeting opacities on chest radiograph consistent with ABPA
Important considerations
[a]Expectoration of mucus plugs, finger-in-glove and fleeting opacities on chest radiograph, lung collapse, and others.
[b]A positive type 1 skin test is acceptable when *Aspergillus*-IgE is unavailable.
[c]Serum total IgE <500 IU·mL^{-1} may be acceptable if all other criteria are fulfilled.
[d]*A. fumigatus*-specific IgG can be detected using lateral flow assays or enzyme immunoassays. The cut-offs for *A. fumigatus*-specific IgG must be developed for specific populations (*e.g.* ⩾27, ⩾60 and ⩾40 mgA·L^{-1} for India, Japan and the UK, respectively). In the absence of population-specific cut-offs, we suggest using manufacturer recommendations.
[e]High-attenuation mucus is pathognomonic of ABPA and confirms ABPA diagnosis even if all other criteria are not fulfilled.
Elevated IgE against rAsp f1, f2 and f4 supports the diagnosis of ABPA and could be used as another component for diagnosing ABPA.

A. fumigatus: *Aspergillus fumigatus*; rAsp: recombinant *A. fumigatus*.

Eur Respir J. 2024 Apr 4;63(4):2400061.

Diagnostic algorithm for ABPA



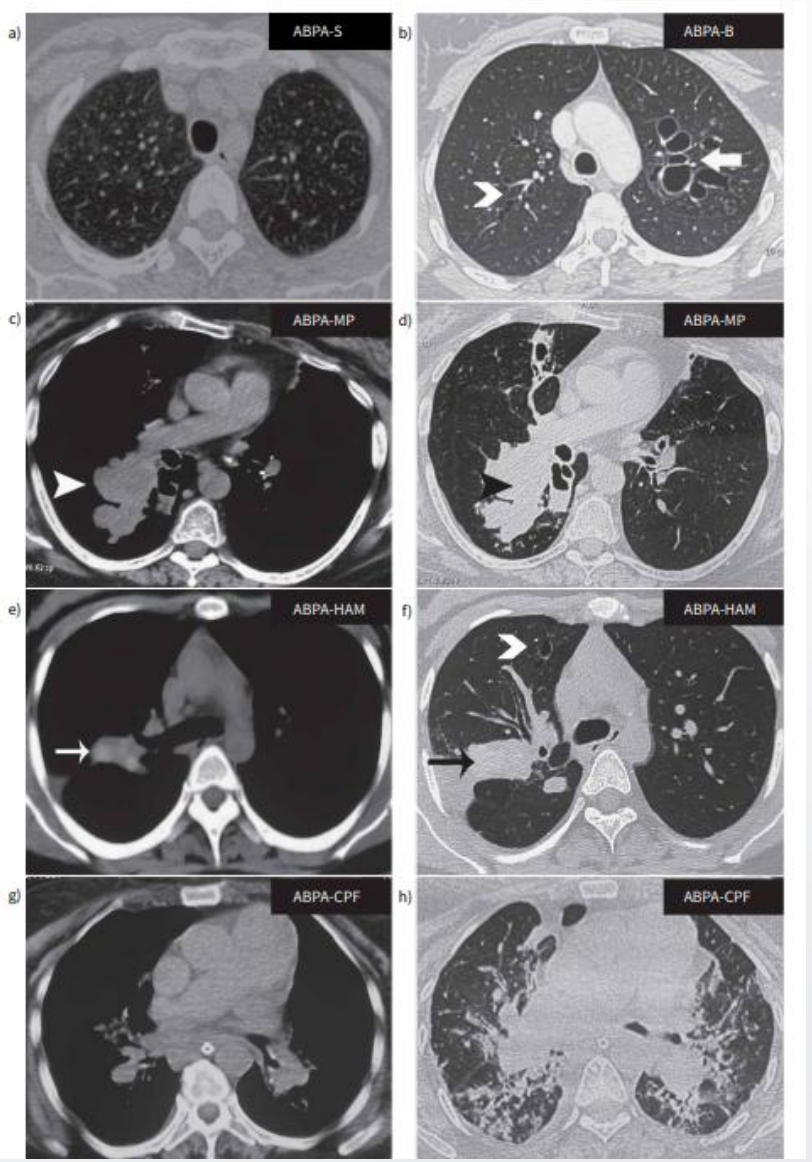
Eur Respir J. 2024 Apr 4;63(4):2400061.

Radiological Classification in ABPA

TABLE 4 Revised International Society for Human and Animal Mycology (ISHAM)-ABPA working group recommendations for radiological classification for allergic bronchopulmonary aspergillosis/mycosis (ABPA/M)

Serological ABPA (ABPA-S)	ABPA with no bronchiectasis
ABPA with bronchiectasis (ABPA-B)	ABPA with radiological evidence of bronchiectasis
ABPA with mucus plugging (ABPA-MP)	ABPA with mucus plugging but without high-attenuation mucus; patients with both bronchiectasis and mucus plugging will be classified as ABPA-MP
ABPA with high-attenuation mucus (ABPA-HAM)	ABPA with high-attenuation mucus
ABPA with chronic pleuropulmonary fibrosis (ABPA-CPF)	ABPA with two or more of the following: pulmonary fibrosis, fibro-cavitary lesions, fungal ball and pleural thickening

Consolidation, centrilobular nodules (with a tree-in-bud appearance), atelectasis and mosaic attenuation are other common radiological findings seen in ABPA that can occur in isolation or in the presence of ABPA-B, ABPA-MP and ABPA-HAM. In patients with ABPA-CPF, chronic pulmonary aspergillosis complicating ABPA should be excluded.



High-Attenuation Mucus (HAM)

- Hyperdense mucus on non-contrast CT
- Pathognomonic for ABPA
- Strongly supports the diagnosis, even if some criteria are not fulfilled

ABPA

Treatment

- Oral prednisolone or itraconazole
- Prednisolone + itraconazole for recurrent exacerbations
- Voriconazole or posaconazole if itraconazole is not tolerated
- Bronchoscopy for mucus plug removal (selected patients)

Biologic Therapy

- Anti-IgE: Omalizumab
- Anti-IL-5/IL-5R: Mepolizumab, Benralizumab
- Anti-IL-4R / TSLP: Dupilumab, Tezepelumab
- Exacerbations & steroid use ↓
- Refractory or steroid-dependent ABPA

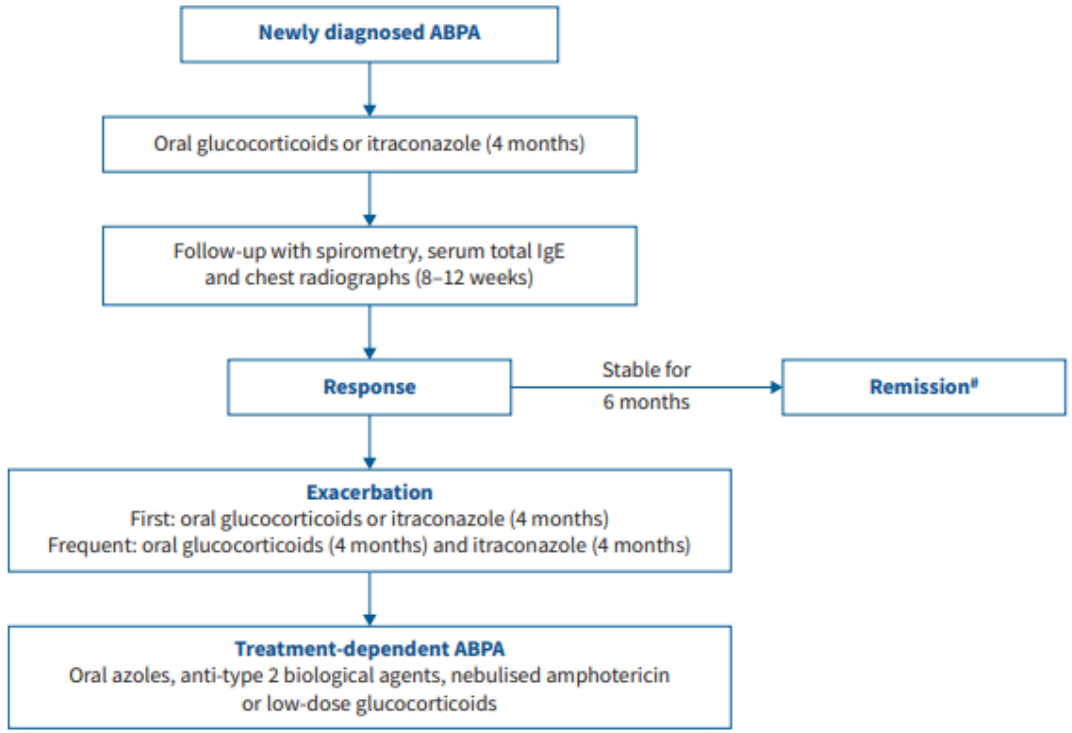
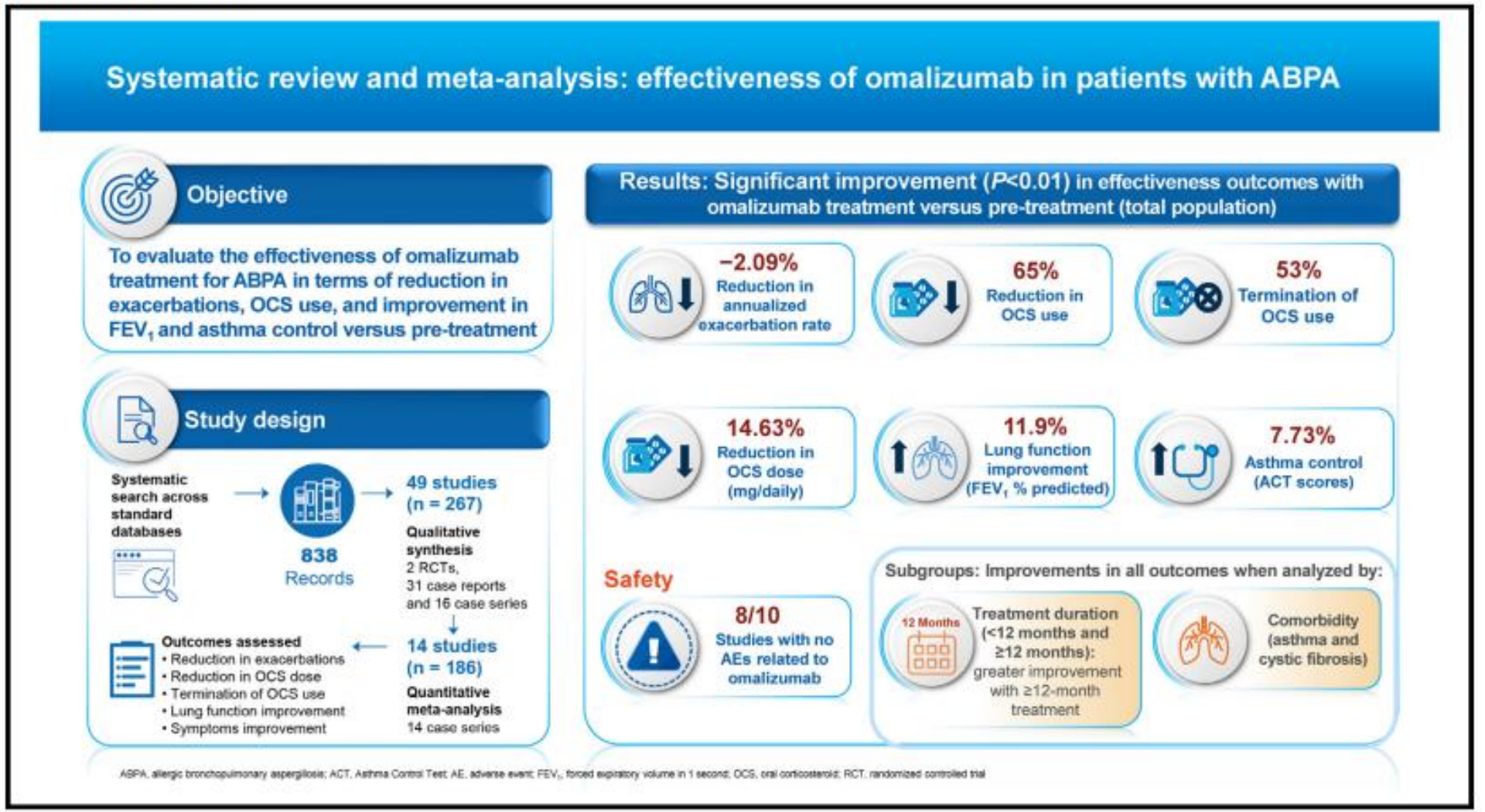


FIGURE 5 Stagewise management of patients with allergic bronchopulmonary aspergillosis/mycosis (ABPA/M).
#: remission may be achieved on antifungal azoles or biological agents, or spontaneously after therapy.

Effectiveness of biologics in ABPA

GRAPHICAL ABSTRACT



J Allergy Clin Immunol Pract . 2023 Mar;11(3):896-905.

Assessment of Treatment Response in ABPA

TABLE 3 Revised International Society for Human and Animal Mycology (ISHAM)-ABPA working group recommendations for clinical classification and treatment response criteria for allergic bronchopulmonary aspergillosis/mycosis (ABPA/M)

Acute ABPA	*Newly diagnosed*: Previously undiagnosed ABPA/M meeting the diagnostic criteria (tables 1 and 2). *Exacerbation*: In a patient with diagnosed ABPA/M: • Sustained (>14 days) clinical worsening; or • Radiological worsening; and • Increase in serum total IgE by ⩾50% from the last recorded IgE value during clinical stability, along with • Exclusion of other causes of worsening. *Asthma exacerbation*: worsening respiratory symptoms for at least 48 h without immunological or radiological deterioration of ABPA/M. *Infective/bronchiectasis exacerbation*: clinical deterioration for at least 48 h with an increase in cough, breathlessness, sputum volume or consistency, sputum purulence, fatigue, malaise, fever, or haemoptysis, without immunological or radiological deterioration of ABPA/M.
Response	• Symptomatic improvement by at least 50% (on a Likert or visual analogue scale) after 8 weeks; and • Major radiological improvement (>50% reduction in radiological opacities) or decline in serum total IgE by at least 20% after 8 weeks of treatment.
Remission	• Sustained (⩾6 months) clinico-radiological improvement, off glucocorticoids; and • Lack of rise in serum total IgE by ⩾50% from the last recorded IgE value during clinical stability. *Patients on biological agents or long-term antifungal agents may also be considered in remission if they meet the above criteria.*
Treatment-dependent ABPA	• Two or more consecutive ABPA/M exacerbations, each within 3 months of stopping glucocorticoids. • Worsening respiratory symptoms AND worse imaging or rise in serum total IgE by 50% within 4 weeks of tapering oral steroids on two separate occasions.
Advanced ABPA	• Extensive bronchiectasis (⩾10 segments) due to ABPA/M on chest imaging; and • Cor pulmonale or chronic type 2 respiratory failure.

Eur Respir J. 2024 Apr 4;63(4):2400061.

TABLE 6 Summary of various drugs available for treating allergic bronchopulmonary aspergillosis/mycosis

	Dose	Adverse events	Other comments
Systemic glucocorticoids			
Prednisolone	0.5 mg·kg^{-1}·day^{-1} for 2 weeks, followed by 0.5 mg·kg^{-1}·day^{-1} for alternate days for 8 weeks; then taper by 5 mg every 2 weeks and discontinue over 3–5 months OR 0.5, 0.25 and 0.125 mg·kg^{-1}·day^{-1}, for 4 weeks each; then taper by 5 mg every 2 weeks till discontinuation	Hyperglycaemia, hirsutism, acne, osteoporosis, hypertension, cushingoid habitus, weight gain, opportunistic infections, psychosis, depression, cataracts, gastritis, HPA suppression, *etc.*	
Methylprednisolone	0.4, 0.2 and 0.1 mg·kg^{-1}·day^{-1}, each, for 4 weeks; then taper by 4 mg·week^{-1} to complete 4 months	Same as prednisolone	Itraconazole increases the plasma levels of methylprednisolone but not prednisolone
Deflazacort	0.75 mg·kg^{-1}·day^{-1} for 4 weeks; decrease by half every 4 weeks for the next 2 months; then taper by 6 mg every 2 weeks and discontinue to complete 4 months		Lesser metabolic side-effects; an ongoing trial is comparing prednisolone with deflazacort (ClinicalTrials.gov: NCT04227483)
Antifungal agents			
Conventional itraconazole capsule	400 mg·day^{-1} in two divided doses for 4 months; maximum dose 600 mg·day^{-1}; it should be administered with meals to improve absorption	Headache, gastritis, nausea, vomiting, liver toxicity, pedal oedema, heart failure, tingling or numbness, hypertension, palpitations, *etc.*	TDM should be performed (trough itraconazole levels ⩾0.5 mg·L^{-1})
Super bioavailable itraconazole capsule	260 mg·day^{-1} in two divided doses for 4 months; maximum dose 390 mg·day^{-1}; it should be given on an empty stomach	Same as conventional itraconazole	PPIs or antacids do not affect serum levels
Voriconazole capsule	400 mg·day^{-1} in two divided doses; maximum dose 600 mg·day^{-1}; it should be given on an empty stomach	Headache, hallucinations, anxiety, insomnia, rash, photosensitivity, mucositis, liver toxicity, prolonged QTc, tingling, numbness, skin cancer, *etc.*	TDM should be performed (trough itraconazole levels ⩾1 mg·L^{-1})
Posaconazole (delayed-release tablet form or suspension)	800 mg·day^{-1} (oral suspension) in two divided doses; 300 mg·day^{-1} (delayed-release tablet) once daily; better absorption with meals (tablet form) or fatty meals (suspension)	GI intolerance, ankle oedema, rash, fatigue, *etc.*	Routine TDM is not required; for better outcomes, target trough level ⩾1 mg·L^{-1}
Isavuconazole	200 mg·day^{-1} once daily; no relation to food intake	GI intolerance, ankle oedema, rash, fatigue, *etc.*	Routine TDM is not required; for better outcomes, target trough level ⩾1 mg·L^{-1}
Nebulised amphotericin B deoxycholate	10 mg twice daily 3–6 times per week	Bronchospasm, cough, fever, headache, sinusitis, *etc.*	Reconstituted solution can be stored for 1 week at 2–8°C; safe in pregnancy
Nebulised liposomal amphotericin B	25–50 mg once or twice weekly	Bronchospasm, cough, fever, sinusitis, *etc.*	Reconstituted solution can be stored for 48 h at 2–8°C; safe in pregnancy
Biological agents			
Omalizumab	Dose is based on body weight and serum total IgE values not exceeding 375 mg *s.c.* injection twice a month	Local reaction, headache, peripheral oedema, abdominal pain, arthralgia, serum sickness, anaphylaxis and TIA	
Mepolizumab	100 mg *s.c.* injection monthly	Local reaction, eczema, headache, diarrhoea, fatigue, arthralgia, herpes zoster, angioedema and anaphylaxis	
Benralizumab	30 mg *s.c.* injection 4-weekly for three doses, after that every 8 weeks	Headache, fever, angioedema, local site reaction and anaphylaxis	
Dupilumab	600 mg *s.c.* injection followed by 300 mg every 2 weeks	Diarrhoea, parasitic infections, dizziness, insomnia, arthralgia, thrombosis, skin rash, local reaction, anaphylaxis, *etc.*	
Tezepelumab	210 mg *s.c.* injection every 4 weeks	Arthralgia, local reaction, anaphylaxis and URTIs	

HPA: hypothalamic–pituitary axis; TDM: therapeutic drug monitoring; PPI: proton pump inhibitor; QTc: corrected QT interval; GI: gastrointestinal; *s.c.*: subcutaneous; TIA: transient ischaemic attack; URTI: upper respiratory tract infection.

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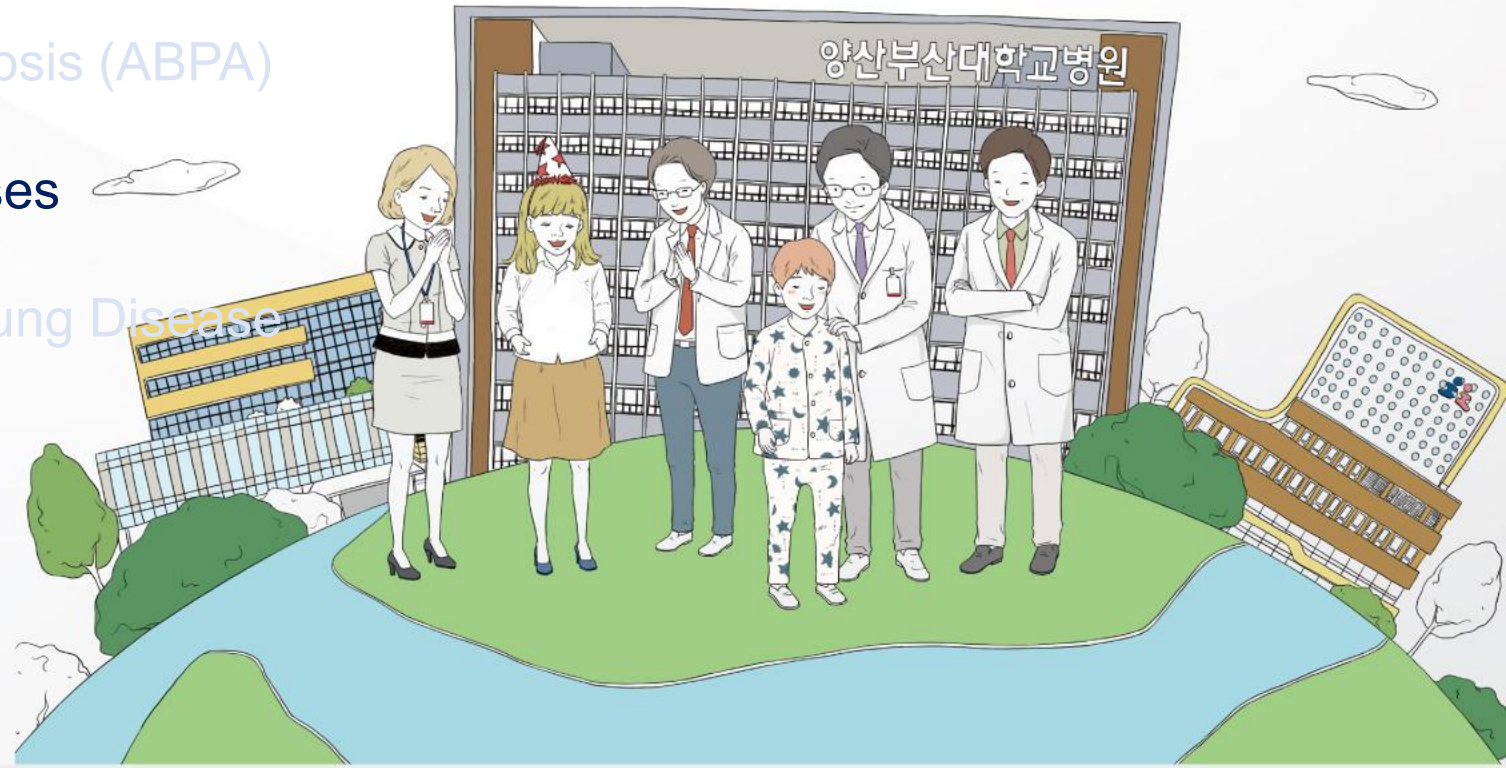
6. Take-home Messages



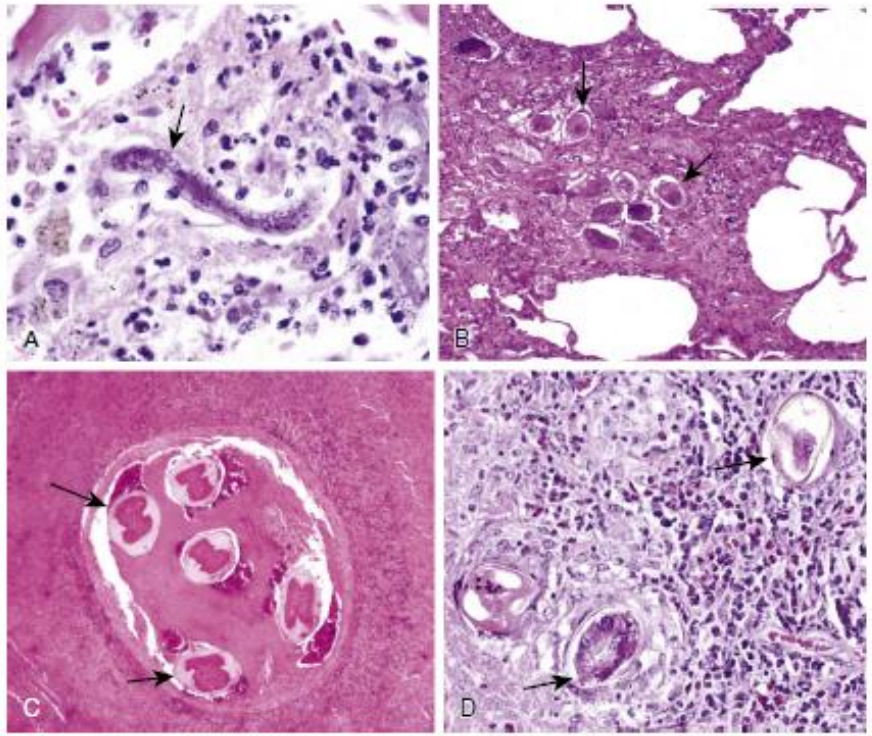
Secondary Eosinophilic Lung Diseases

Common Secondary Causes

- Parasitic infections (Most common worldwide cause of eosinophilic pneumonia)
- Drug-induced eosinophilic pneumonia
- Radiation-induced eosinophilic pneumonia

Clinical Clues

- Travel history
- Recent medication exposure
- Previous radiotherapy
- Immunocompromised host

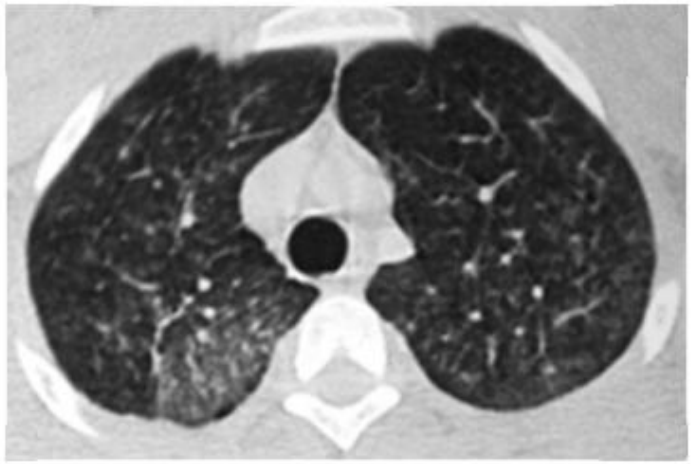


Parasites in Lung

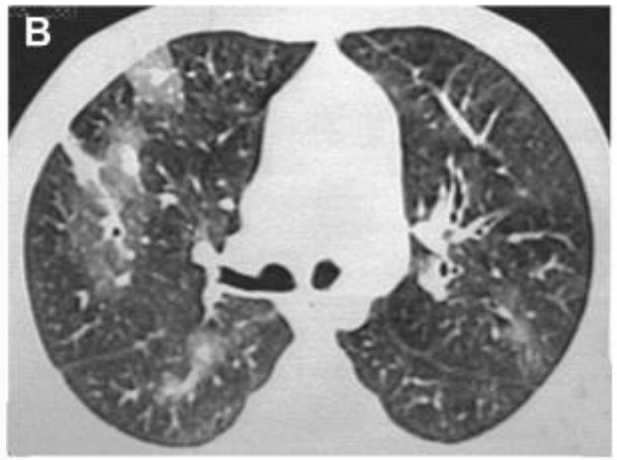
Secondary Eosinophilic Lung Diseases

Parasitic infections

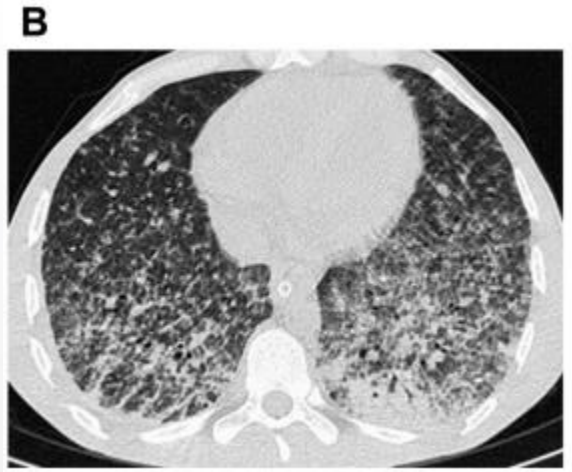
Disease	Key clue	Typical CT	Treatment
Pulmonary Ascariasis (Löffler Syndrome)	• Transient pulmonary infiltrates • mild or absent symptoms	• Migratory GGO/ Consolidation	• Supportive ±Albendazole
Toxocariasis	• Visceral larva migrans • Peripheral eosinophilia+positive ELISA	• Multiple nodules, patchy GGO	• Albendazole (5–14 days)
Strongyloidiasis	• Immunocompromised patients • Hyperinfection syndrome • Larvae detected in stool, sputum	• Diffuse GGO ±ARDS	• Ivermectin

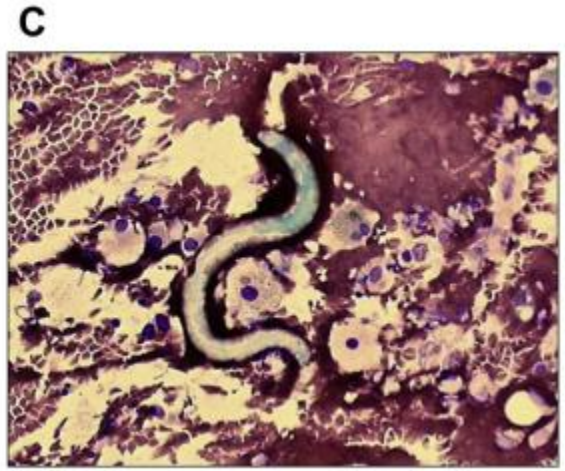


Pulmonary Ascariasis



Toxocariasis





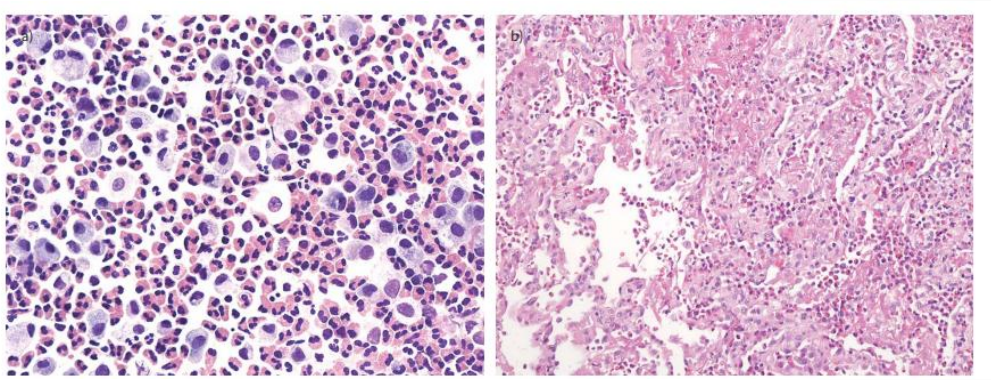
Strongyloidiasis

Clin Chest Med . 2025 Dec;46(4):667-684. Murray & Nadel's Textbook of Respiratory Medicine, 7th ed. Elsevier, 2022.

Secondary Eosinophilic Lung Diseases

Drug-induced eosinophilic pneumonia

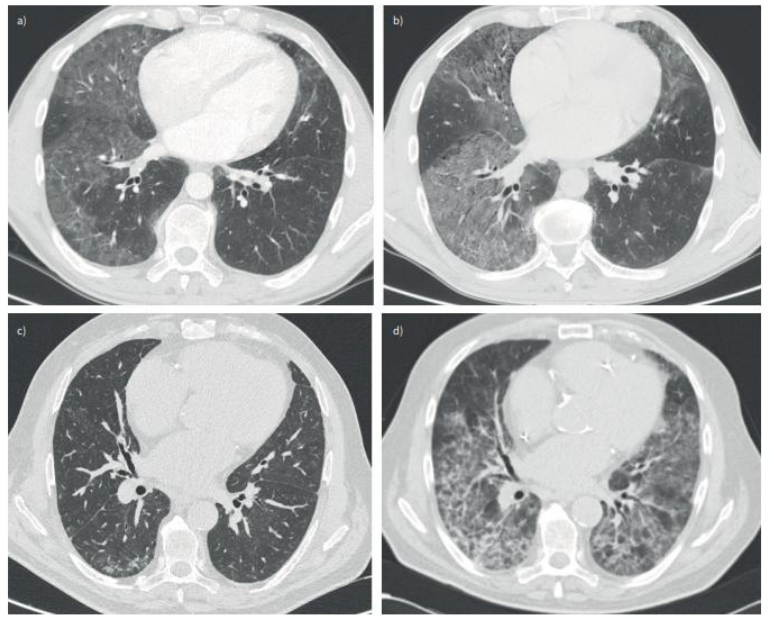
- Common causative drugs
  - Antibiotics
  - NSAIDs
  - Antiarrhythmics *(e.g., amiodarone)*
  - Immune checkpoint inhibitors



Drug-induced EP

Amiodarone-induced eosinophilic pneumonia

- AEP > CEP
- BAL eosinophils >25%
- HRCT: Diffuse GGO and consolidation
- Treatment
  - Discontinue amiodarone
  - ± Systemic corticosteroids

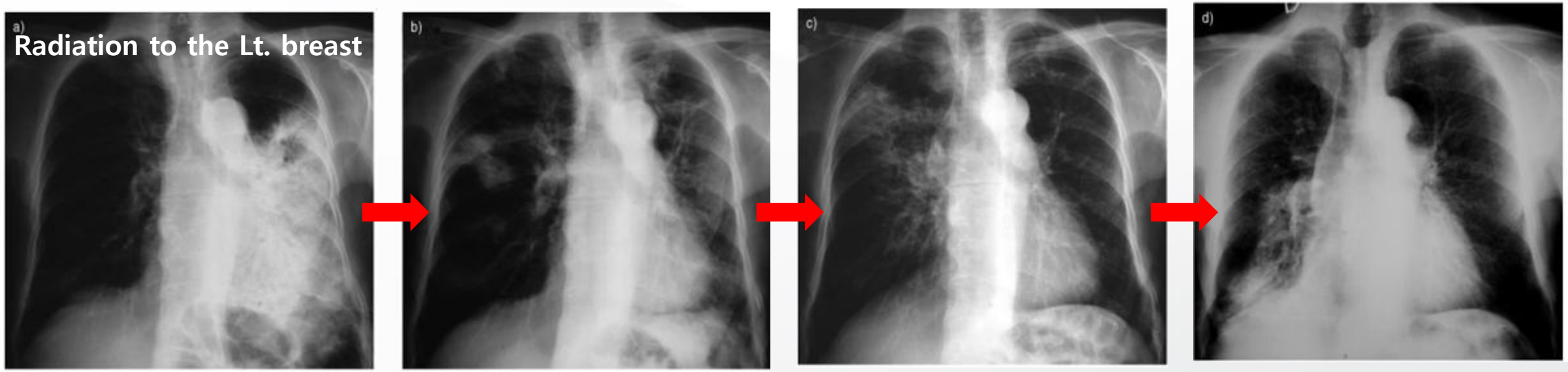


Amiodarone-induced EP

Secondary Eosinophilic Lung Diseases

Radiation-induced Eosinophilic Pneumonia

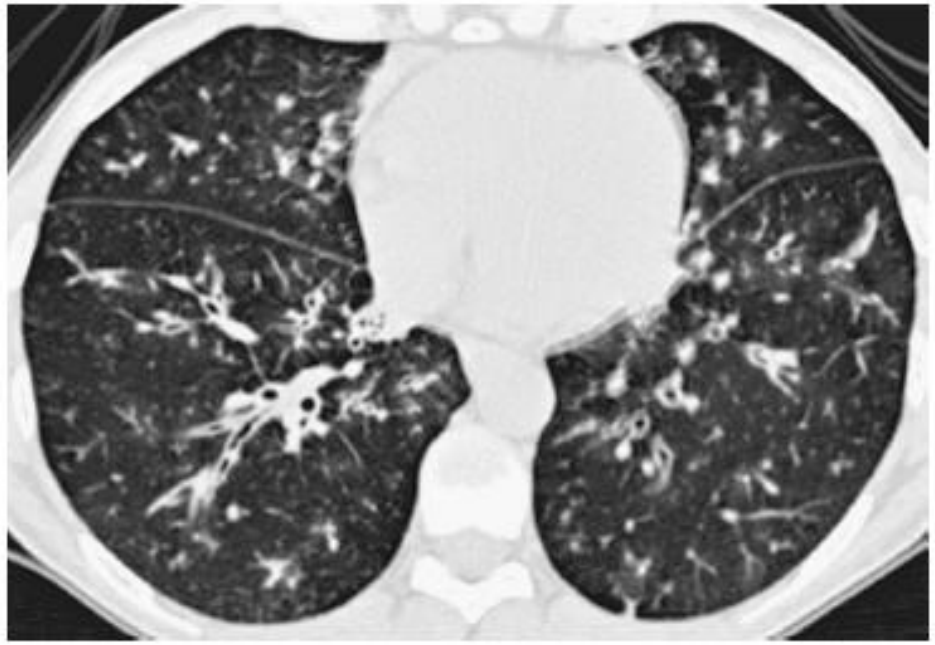
- Occur after breast cancer radiotherapy
- Usually develops 3–12 months after radiation
- Migratory pulmonary infiltrates extending beyond the irradiated lung
- Relapse after steroid withdrawal



Other eosinophilic airway disease

Hypereosinophilic Obliterative Bronchiolitis (HOB)

- Bronchiolitis with blood/BAL eosinophilia and persistent airflow obstruction
- HRCT: Centrilobular nodules and branching opacities
- Associated with EGPA, ABPA, severe asthma, or drugs
- Responds well to systemic corticosteroids



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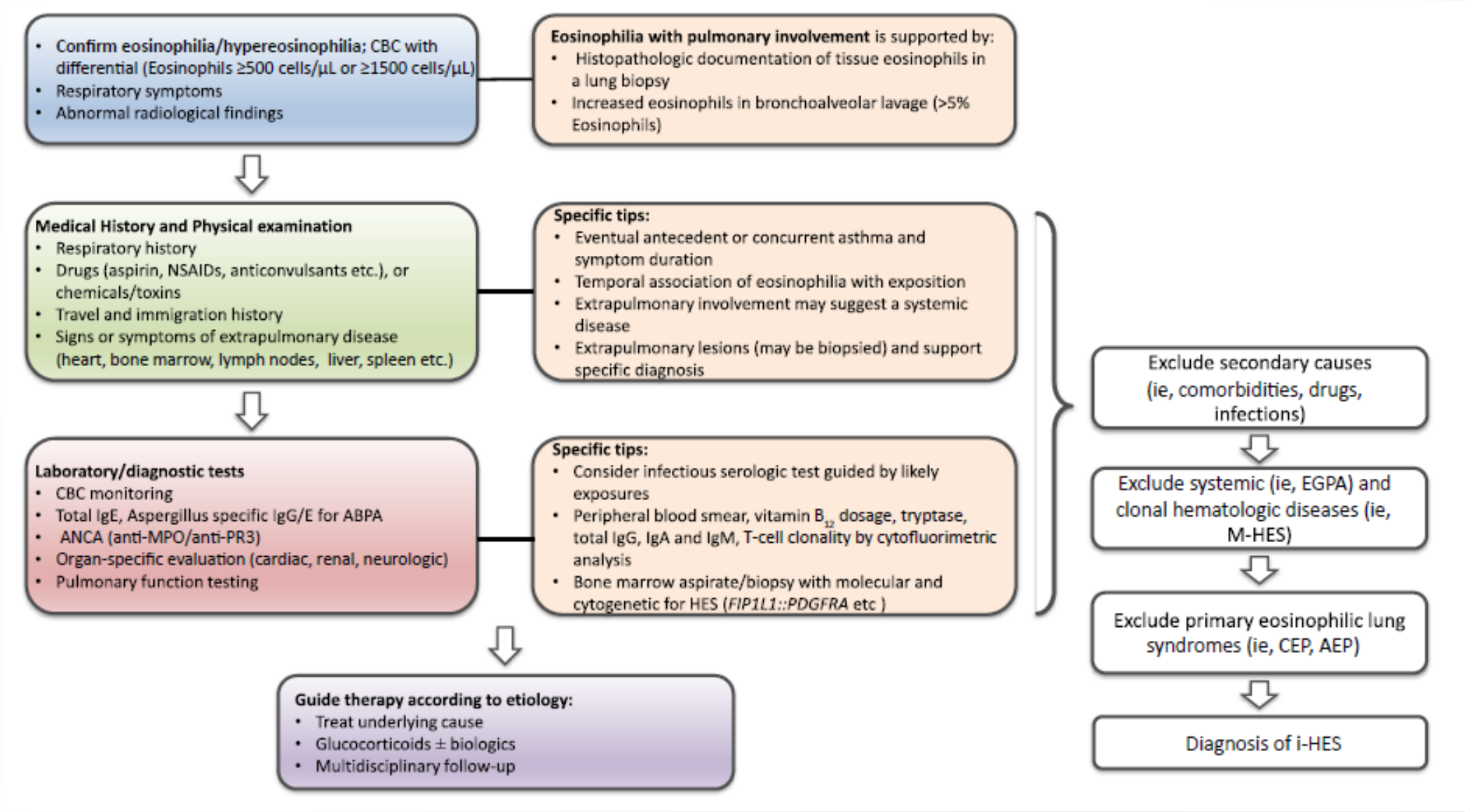
Practical Diagnostic Approach to Pulmonary Eosinophilia



J Allergy Clin Immunol Pract . 2026 Mar;14(3):542-557.

Differential diagnosis by clinical context

TABLE I. Differential diagnosis of pulmonary eosinophilia by clinical context

Clinical setting	Key clues	Most likely etiologies	First-line tests
Acute febrile illness in young adult	Rapid dyspnea, fever, diffuse infiltrates	AEP, parasitic infection, EGPA	BAL, stool exam, HRCT, ANCA
Chronic asthma with recurrent infiltrates	Wheezing, high IgE, bronchiectasis	ABPA, CEP, EGPA	Total IgE, Aspergillus IgE/IgG, ANCA, HRCT
Systemic features (fever, neuropathy, purpura)	Multisystem involvement	EGPA, HES, hematologic malignancy	CBC, ANCA, bone marrow, molecular testing
Immunosuppressed patient	High-risk exposures, severe infection	*Strongyloides* hyperinfection, fungal pneumonia	BAL, serology, cultures

CBC, Complete blood cell count.

J Allergy Clin Immunol Pract . 2026 Mar;14(3):542-557.

Key distinguishing features of major ELDs

TABLE II. Key distinguishing features of major ELDs

Disease	Onset	Blood eosinophilia	BAL eosinophilia	Imaging	Relapse	Systemic involvement
CEP	Subacute (wk)	High	>40%	Peripheral consolidations	Common	No
AEP	Acute (<1 mo)	Often absent at onset	>25%	Diffuse ground-glass, pleural effusion	Rare	No
ABPA	Subacute	Moderate-high	Variable	Central bronchiectasis, mucus plugs	Yes	Asthma, rhinosinusitis
EGPA	Subacute/chronic	High	>25%	Migratory opacities, nodules	Yes	Multisystem (neuropathy, skin, heart)
HESs	Subacute/chronic	Very high	Variable	Ground-glass opacities, nodules, pleural effusions	Variable	Multiorgan involvement
Parasitic infections	Acute/subacute	High	>25%	Transient or fixed infiltrates; nodules; pleural effusions	No	Extrapulmonary symptoms (fever, gastrointestinal, skin)

J Allergy Clin Immunol Pract . 2026 Mar;14(3):542-557.

Take-home Messages

- **Pulmonary eosinophilia**는 다양한 원인을 가진 질환군이며, **체계적인 감별진단이 핵심**이다.
- **Secondary causes를 먼저 배제한 후 특발성 호산구성 폐질환을 진단**해야 한다.
- 병력, 흉부 CT, 혈액 및 BAL 호산구를 종합적으로 해석해야 정확한 진단이 가능하다.
- **스테로이드는 대부분의 호산구성 폐질환에서 핵심 치료**이지만, 이차성 호산구성 폐질환에서는 **원인 제거가 우선**이다.
- **Biologics**는 재발성 CEP와 일부 ABPA에서 **새로운 steroid-sparing 치료 옵션**으로 제시되고 있다.

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환자 내몸

경청해 주셔서 감사합니다.

Thank you for your attention