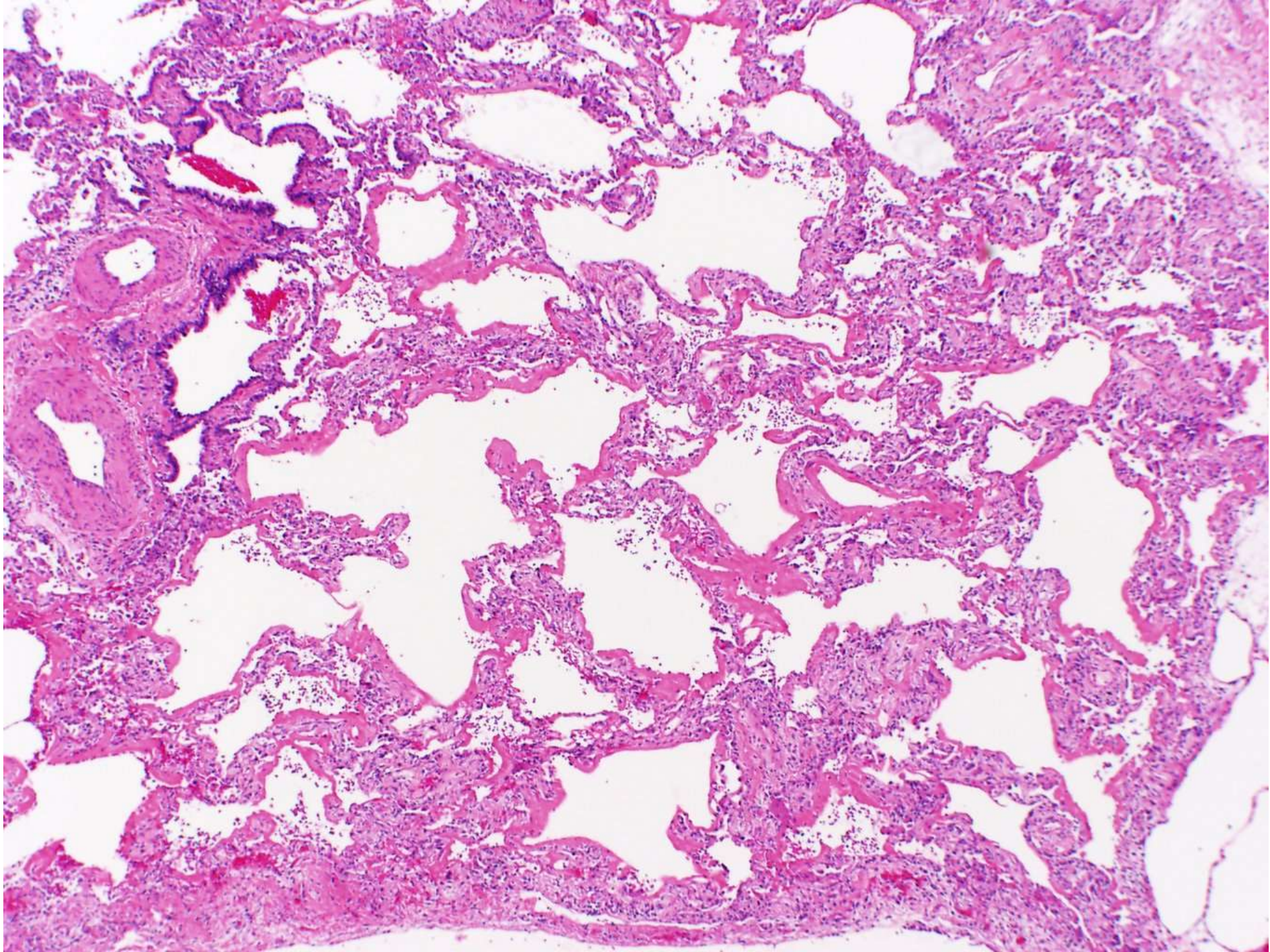
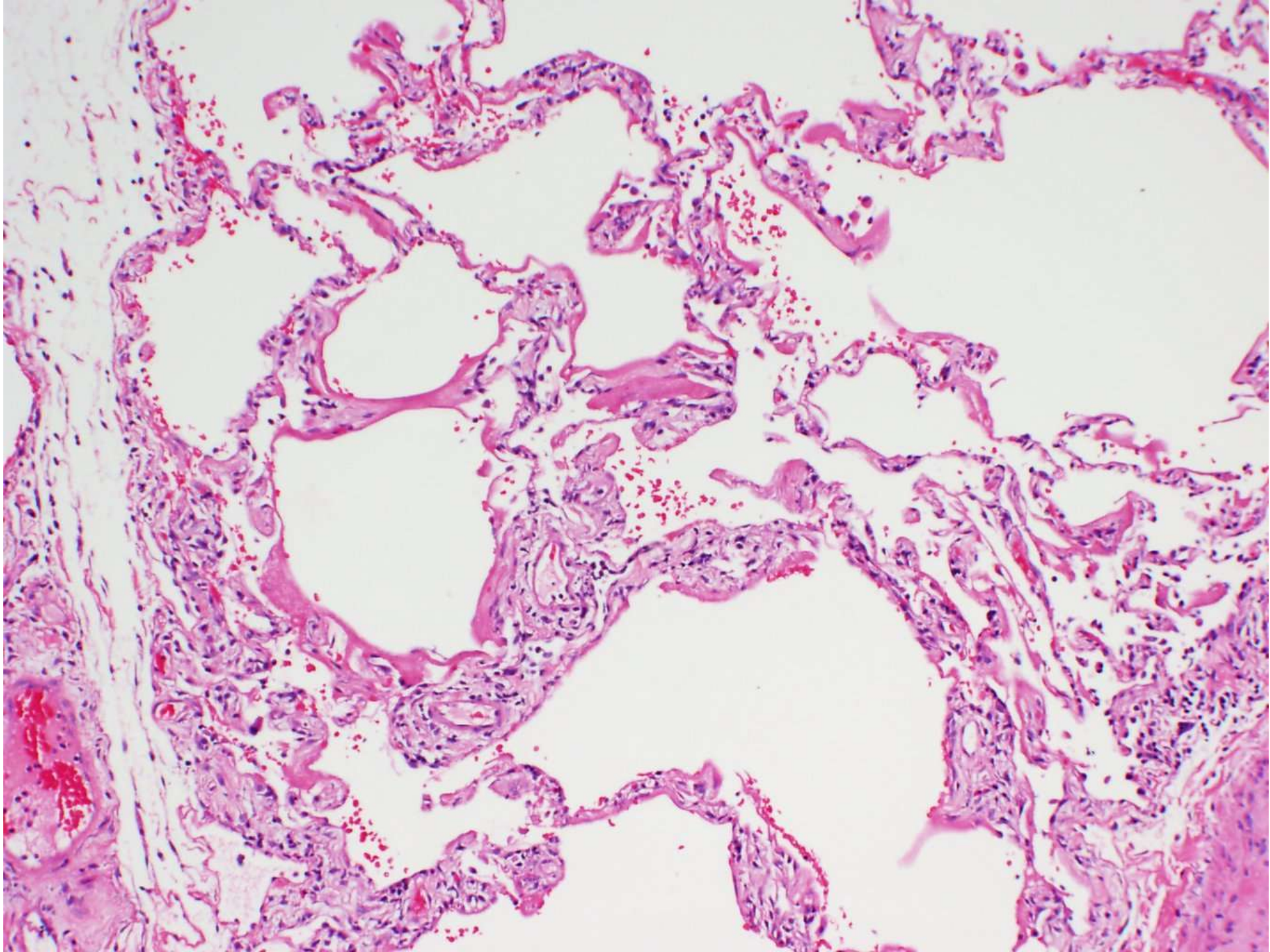
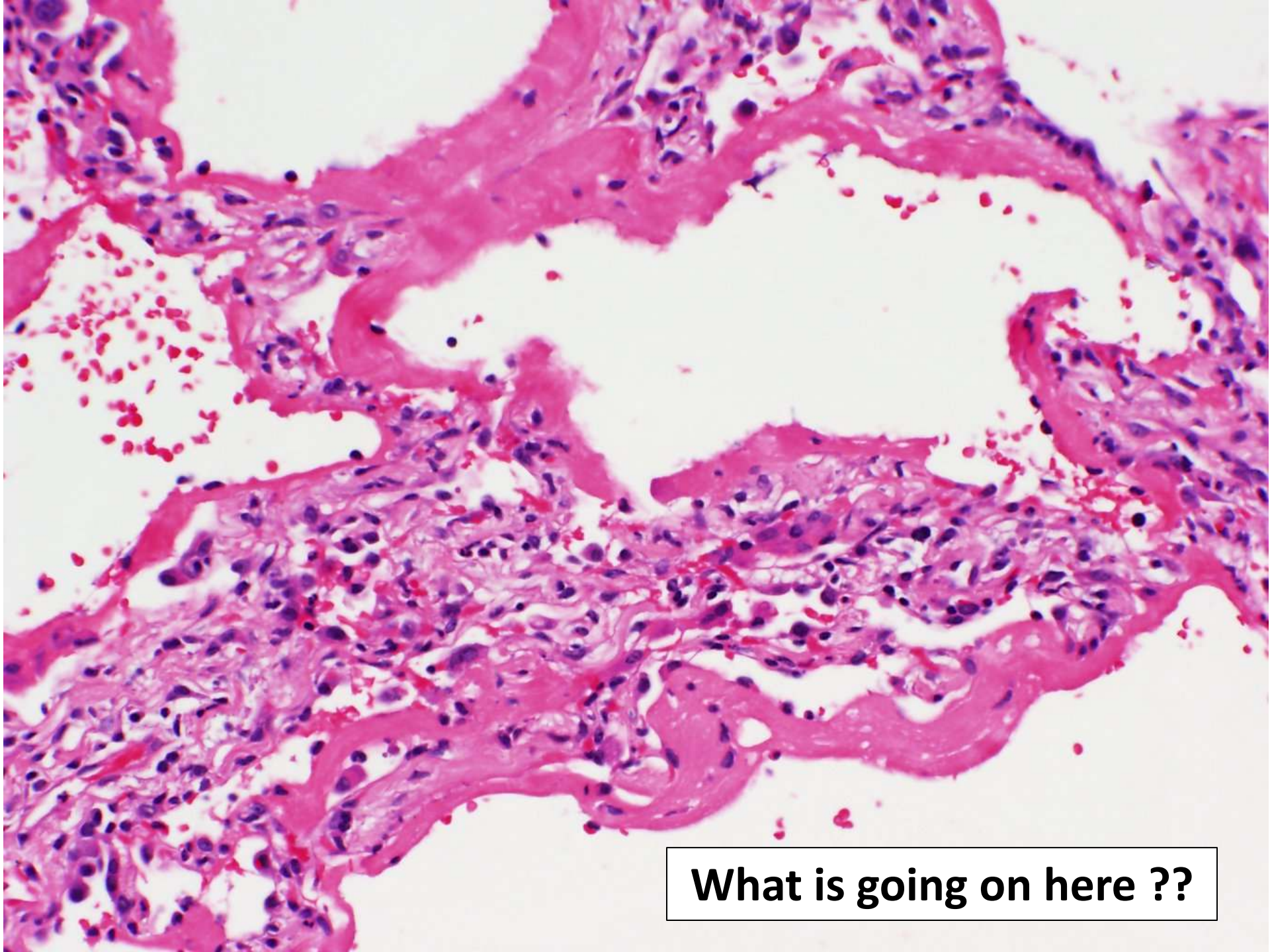
Case1 (TV95-455)



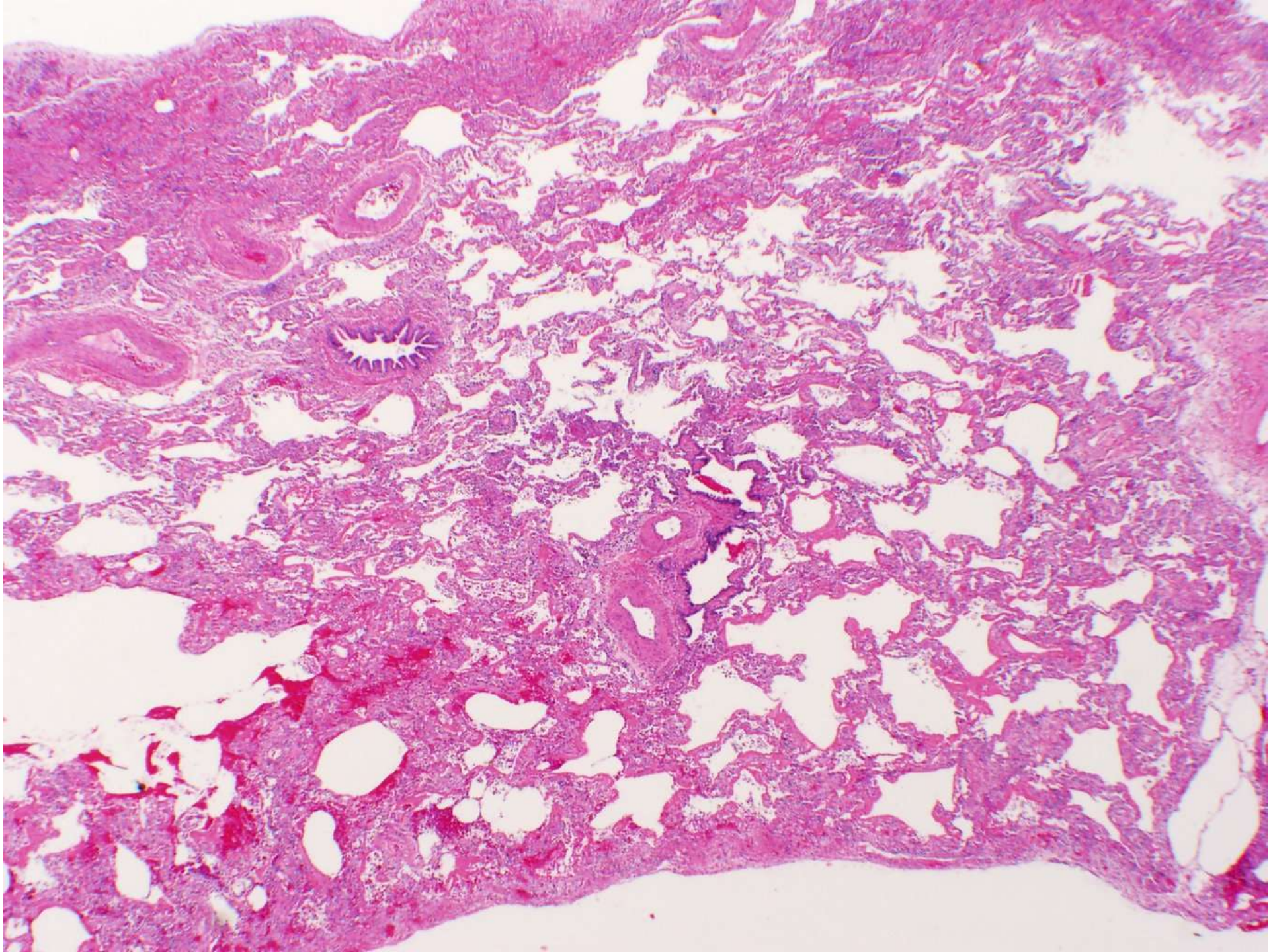



What is going on here ??

Question 1 TV95-455

- The changes shown are best described as:

1. Amyloid in diffuse alveolar septal amyloidosis
2. Fibrin in acute fibrinous and organizing pneumonia (AFOP)
3. Hyaline membranes in diffuse alveolar damage (DAD)
4. Fibrosis usual interstitial pneumonia (UIP)
5. None of the above



Here is the history: TV95-455

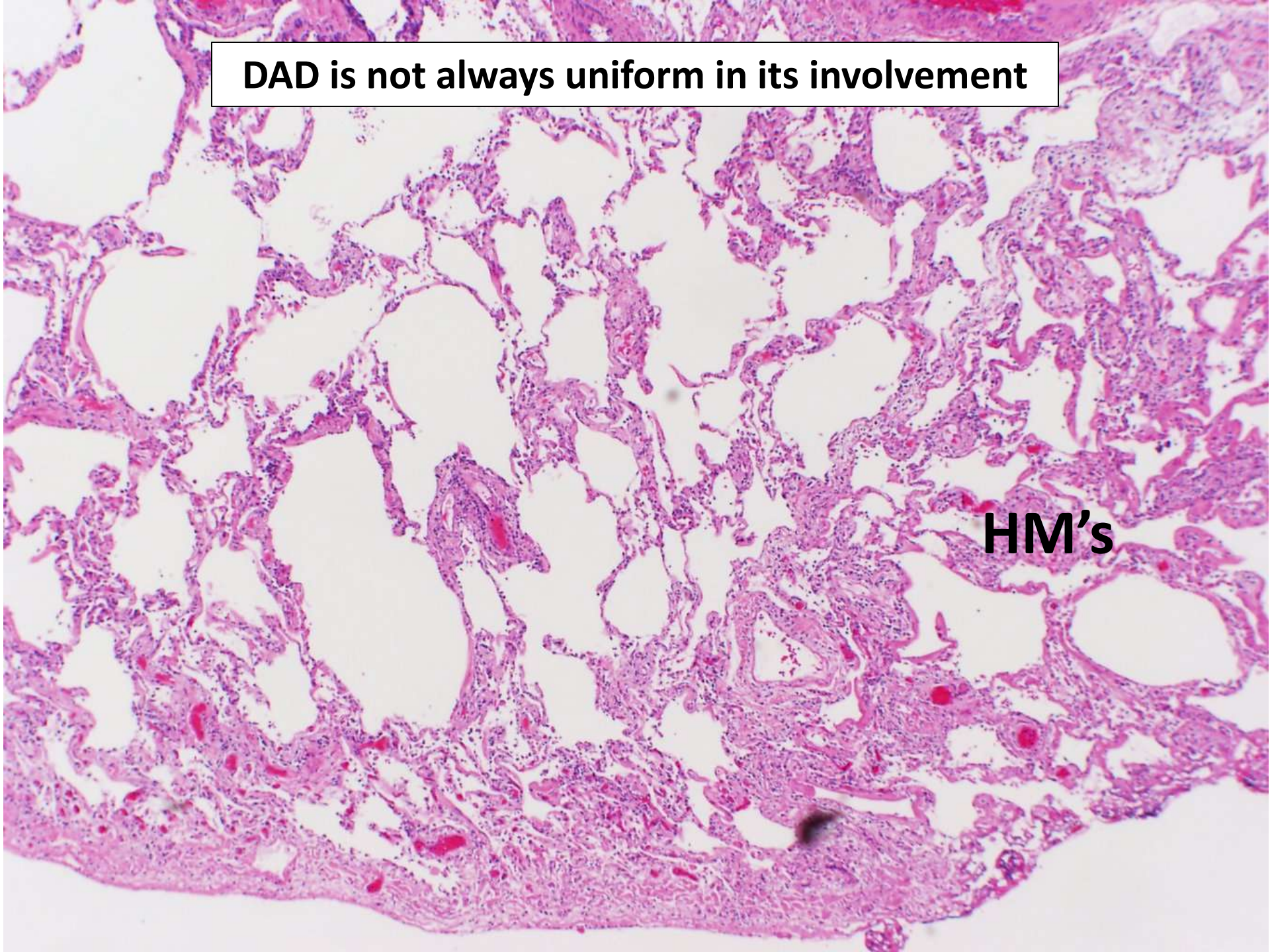
- 70F with RA
- On methotrexate
- One week of dyspnea and progressive respiratory failure

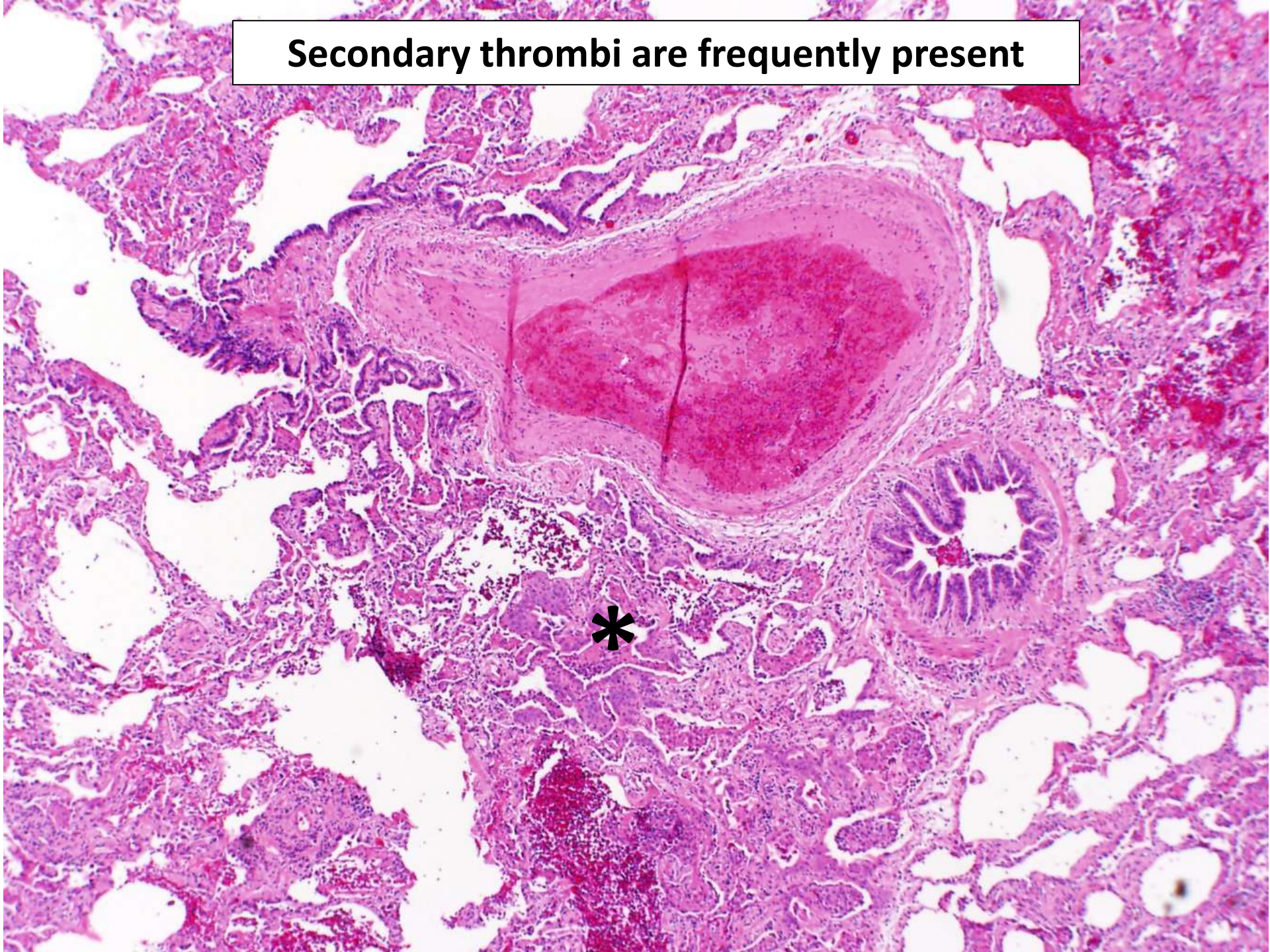
Question 2 TV95-455

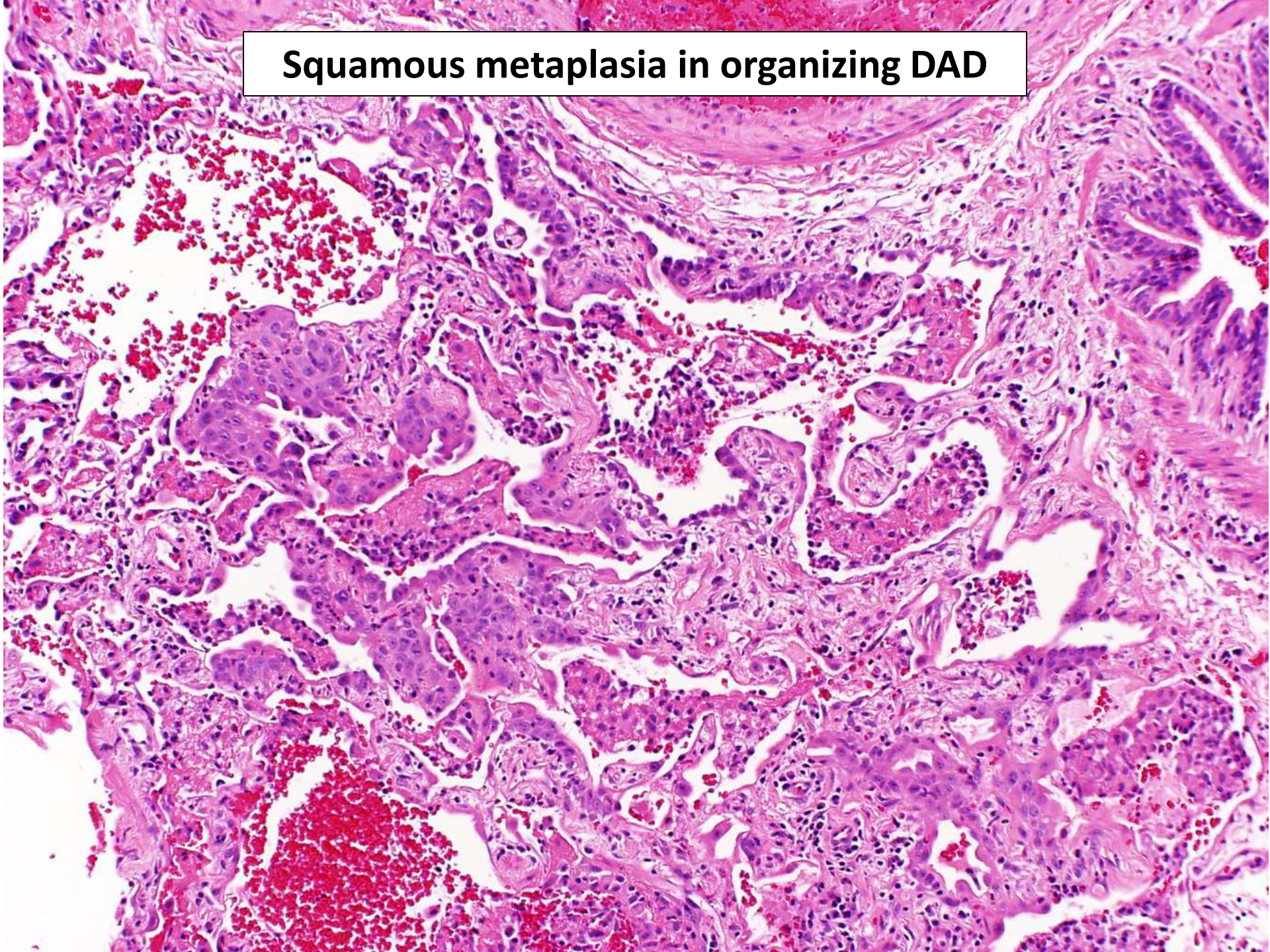
- The most likely cause of the changes seen in the biopsy is:

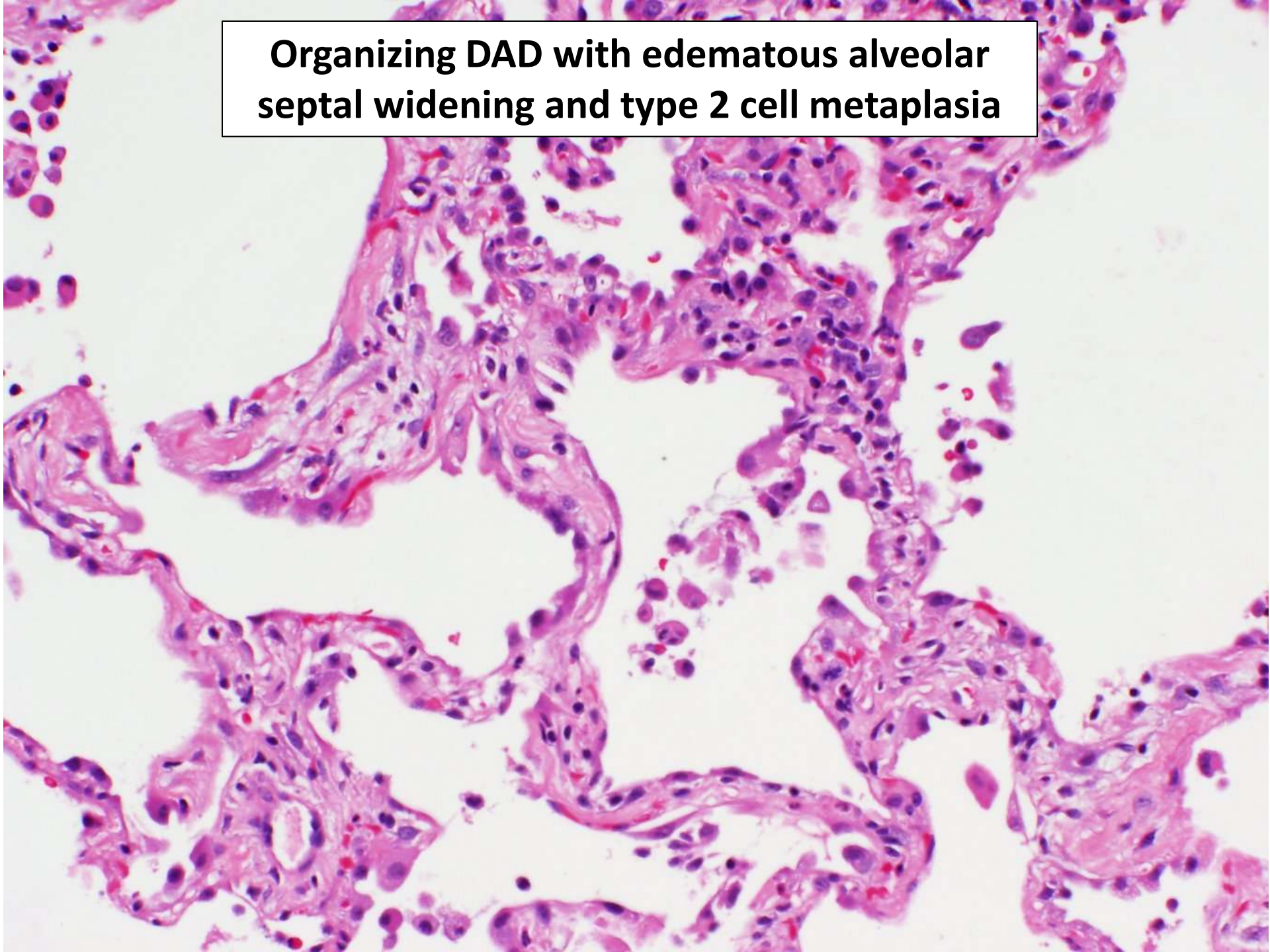
1. RA related DAD
2. Methotrexate reaction
3. Intercurrent influenza
4. Pneumocyustis pneumonia
5. Cannot be determined from the histology

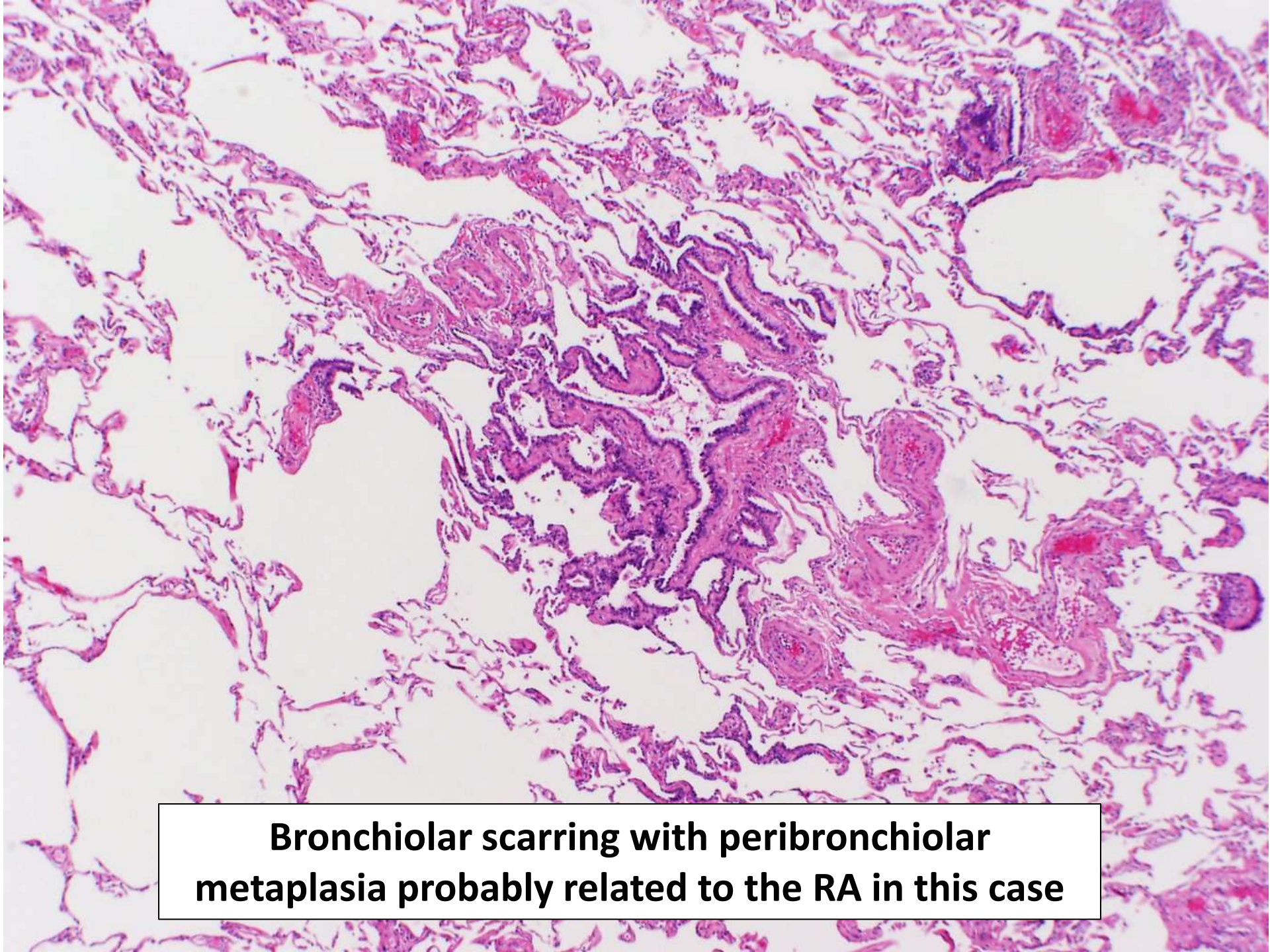
Additional findings in this case


DAD is not always uniform in its involvement
HM's


Secondary thrombi are frequently present
*


Squamous metaplasia in organizing DAD


Organizing DAD with edematous alveolar septal widening and type 2 cell metaplasia


Bronchiolar scarring with peribronchiolar metaplasia probably related to the RA in this case

Final Diagnosis TV95-455

- DAD c/w MTX toxicity…by exclusion
  - Cultures and special stains negative for organisms
  - Time course of illness c/w drug reaction
  - RA-related DAD remains in the D/D

Case included in: Imokawa S et.al. Methotrexate pneumonitis: review of the literature and histopathologic findings in 9 patients. ERJ 2000; 15: 373-381.

Injury Patterns in Methotrexate Pneumonitis
(from Pneumotox.com)

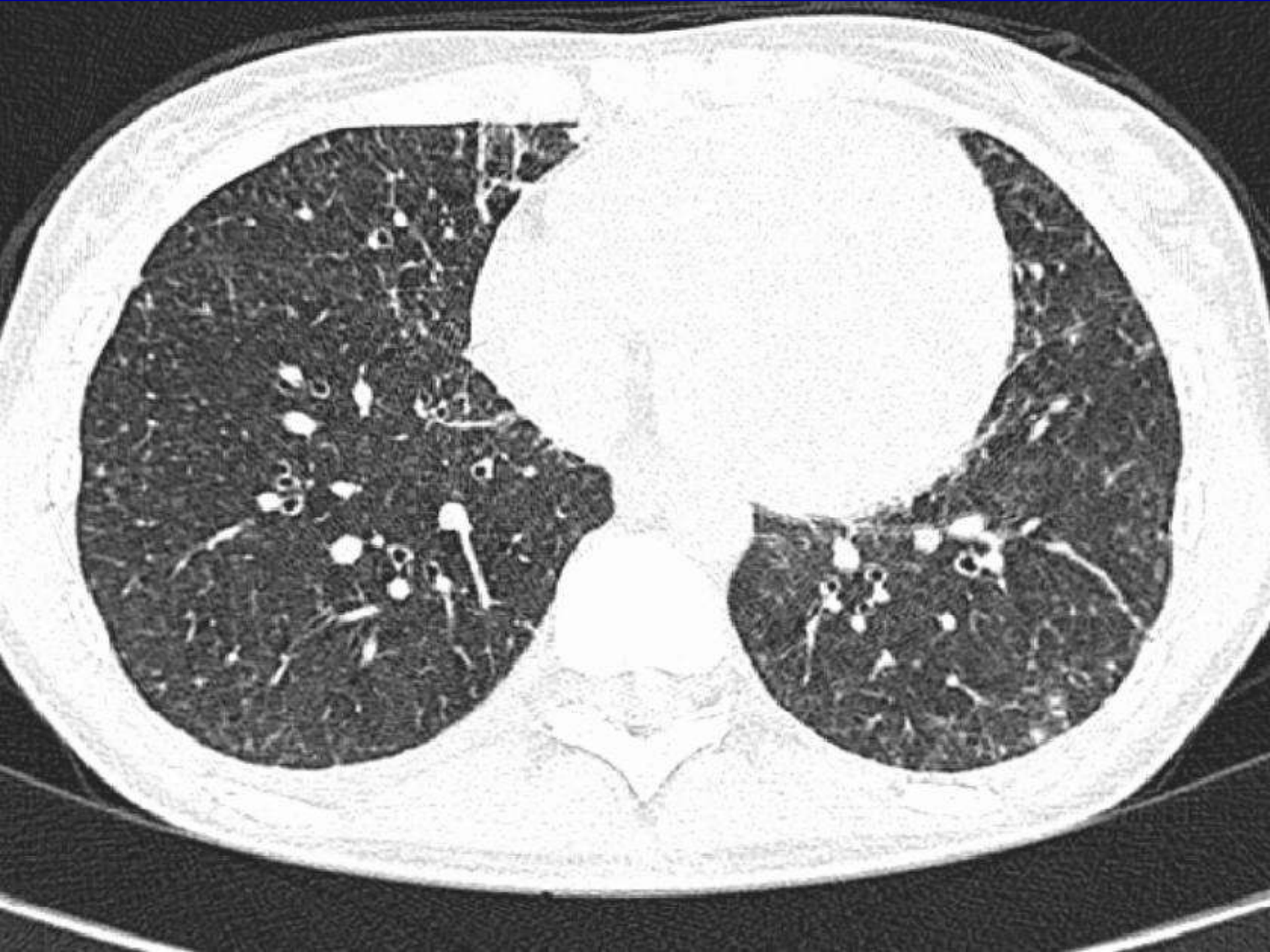
I.a	Acute pneumonitis/ILD
I.b	Subacute pneumonitis/ILD
I.d	Organizing pneumonia (OP/BOOP)
I.g	Pulmonary fibrosis
I.h	Subclinical parenchymal opacities
I.l	Diffuse alveolar damage (DAD)
I.m	ILD with a granulomatous component
I.q	Pulmonary nodulosis
I.v	Subclinical changes in lung function
I.w	Rapidly progressive pulmonary fibrosis
II.a	Noncardiogenic pulmonary edema (NCPE)
II.b	ARDS
III.a	Diffuse alveolar hemorrhage

…and many more clinical presentation patterns

Case No 2

Current symptoms

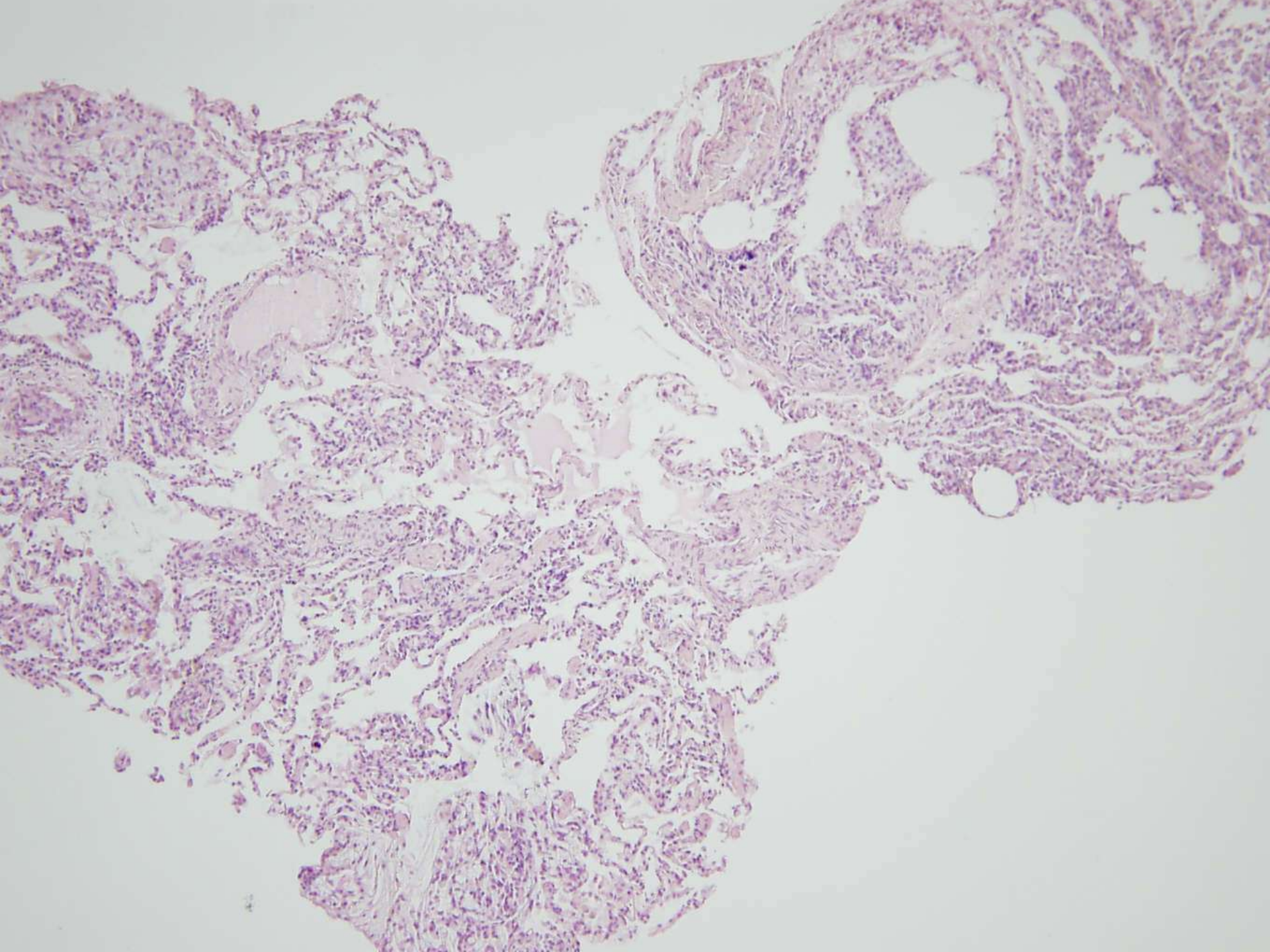
- 24-year-old lady; nurse in pneumology
- Smoker (20 daily) for 10 y
- Five-month-long unspecific troubles
- Night sweat, weight loss, fatigue, SOB
- Inflammatory markers - ATB
- US slight hepatosplenomegalia
- HIV neg; Mantoux neg
- RTG – bilateral fluidothorax, diffuse bilateral small nodules - CT

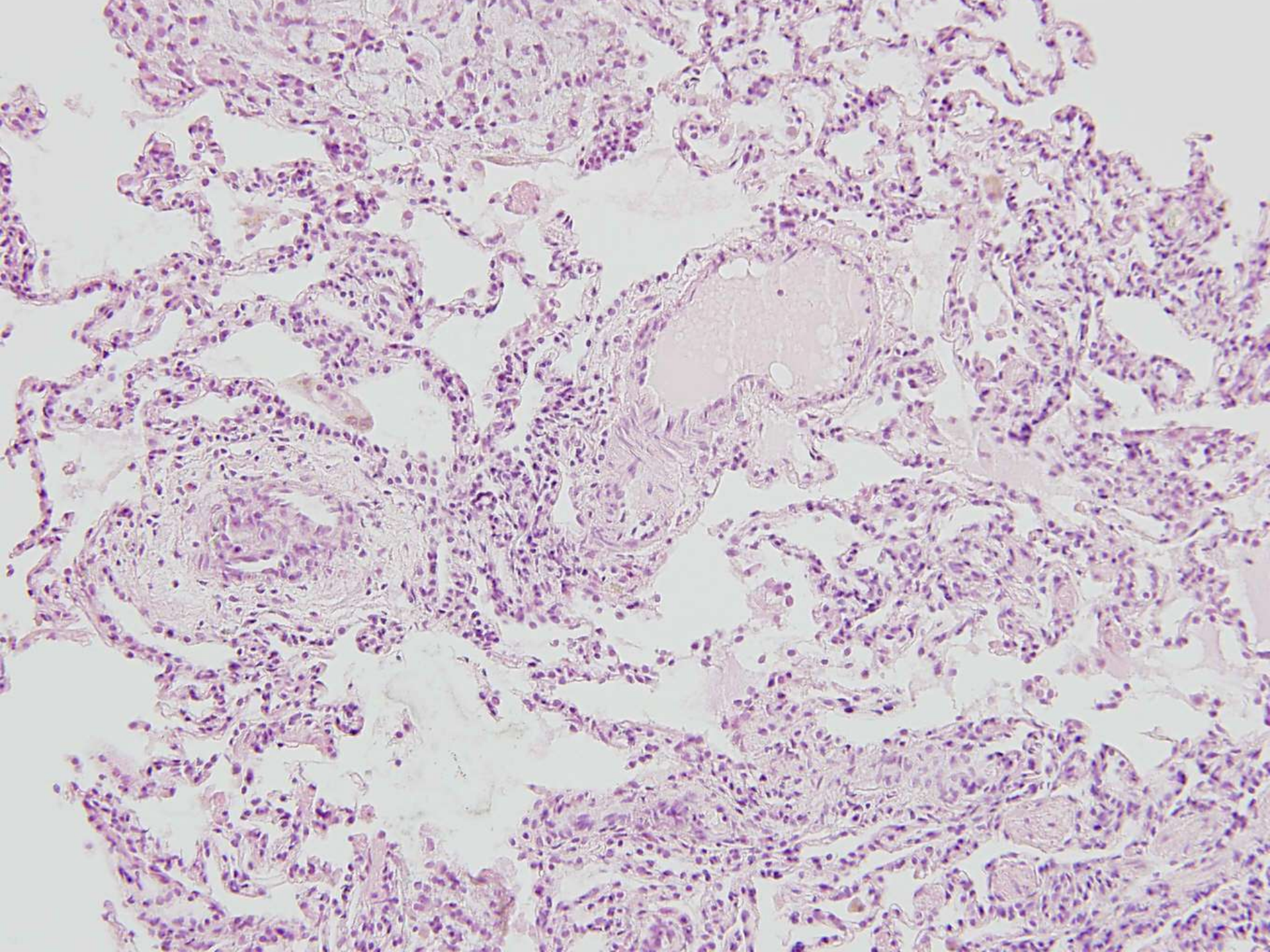


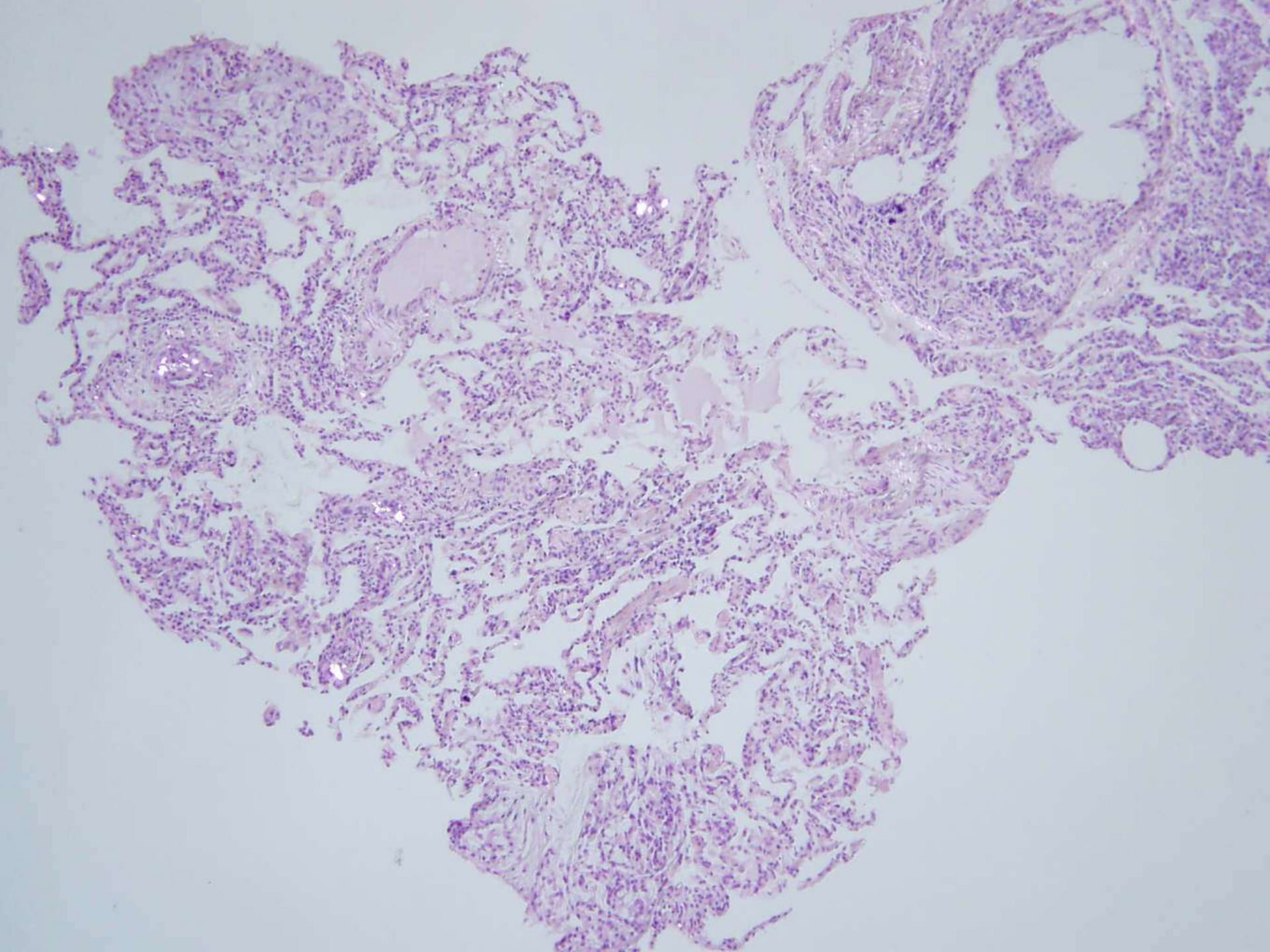
Dif.dg

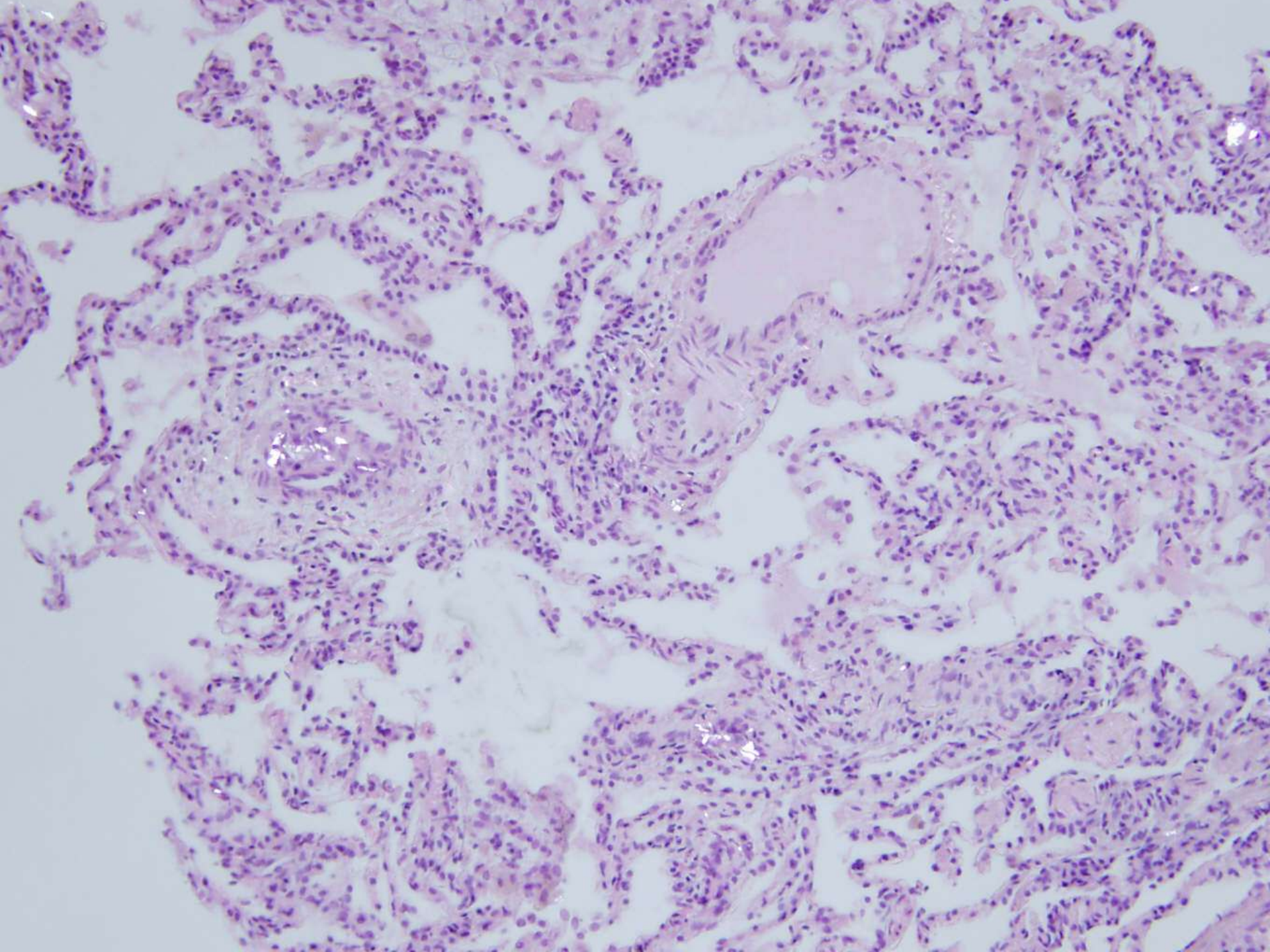
- Sarcoidosis II. grade
- TBC
- Tumor metastasis

- Bronchoscopy BB and TBB







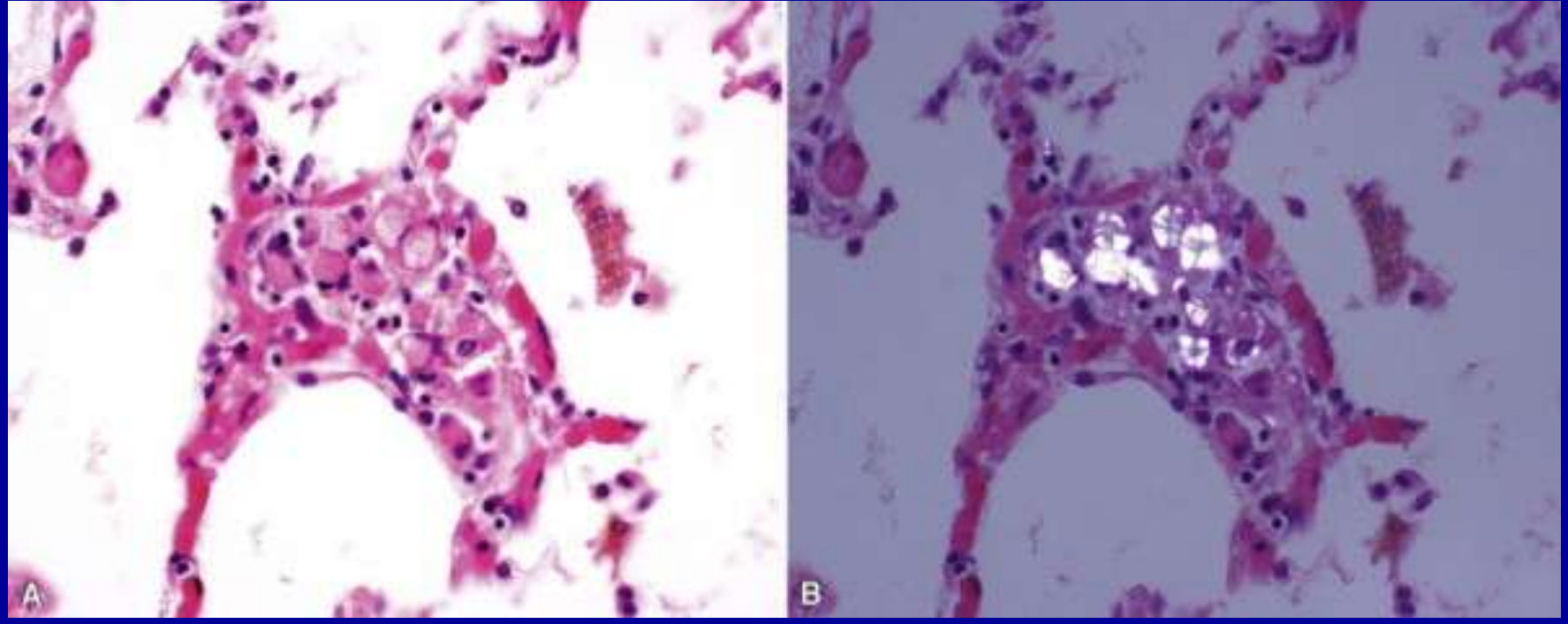


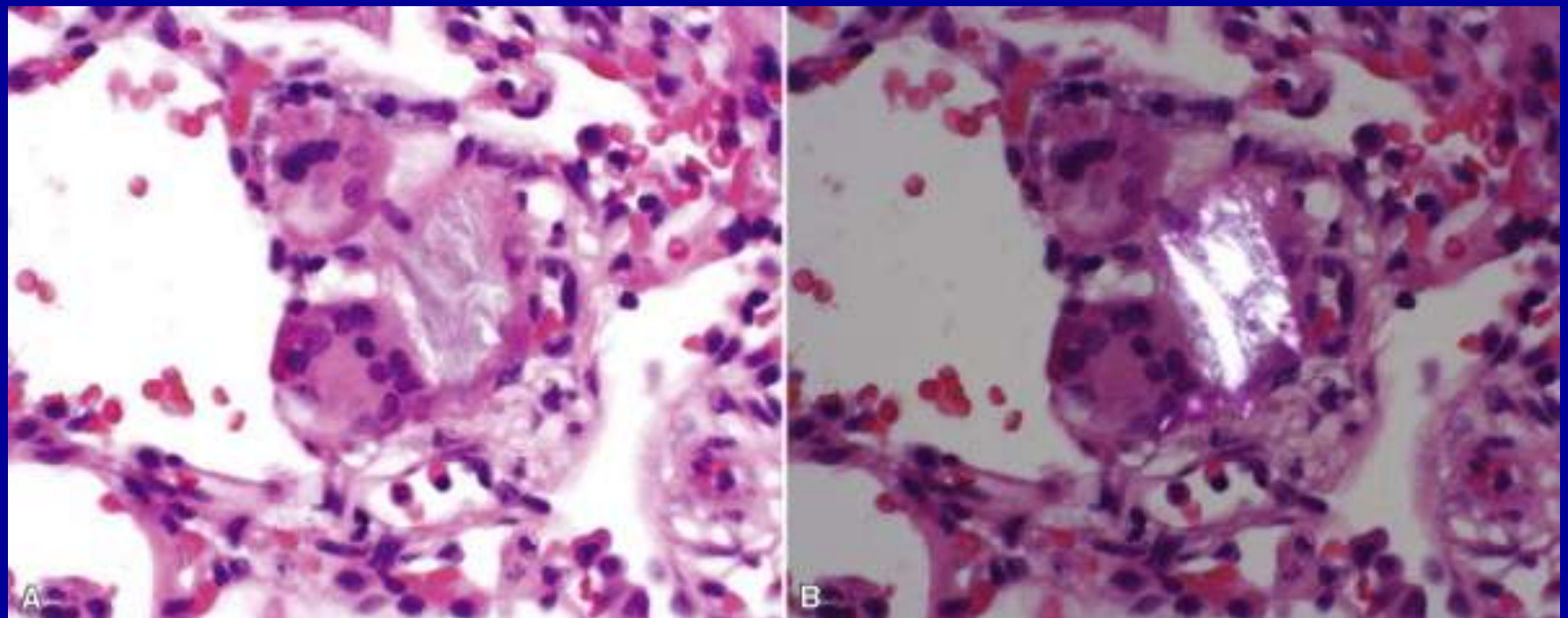
DIAGNOSIS ????

1. Sarcoidosis
2. TBC
3. Generalisation of cancer
4. Mycotic infection
5. i.v. talcosis
6. GPA

I.V. talcosis due to drug abuse

„filler embolism“


A
B


A
B

Pulmonary complications i.v. drug

- Infections (incl. HIV) and infection-related diseases
- Pneumonia
- Abscess
- Septic embolism
- ARDS (DAD)
- Pulmonary hypertension
- Interstitial fibrosing process (ILD)
- Massive fibrosis
- Emphysema

BEWARE OF NURSES!

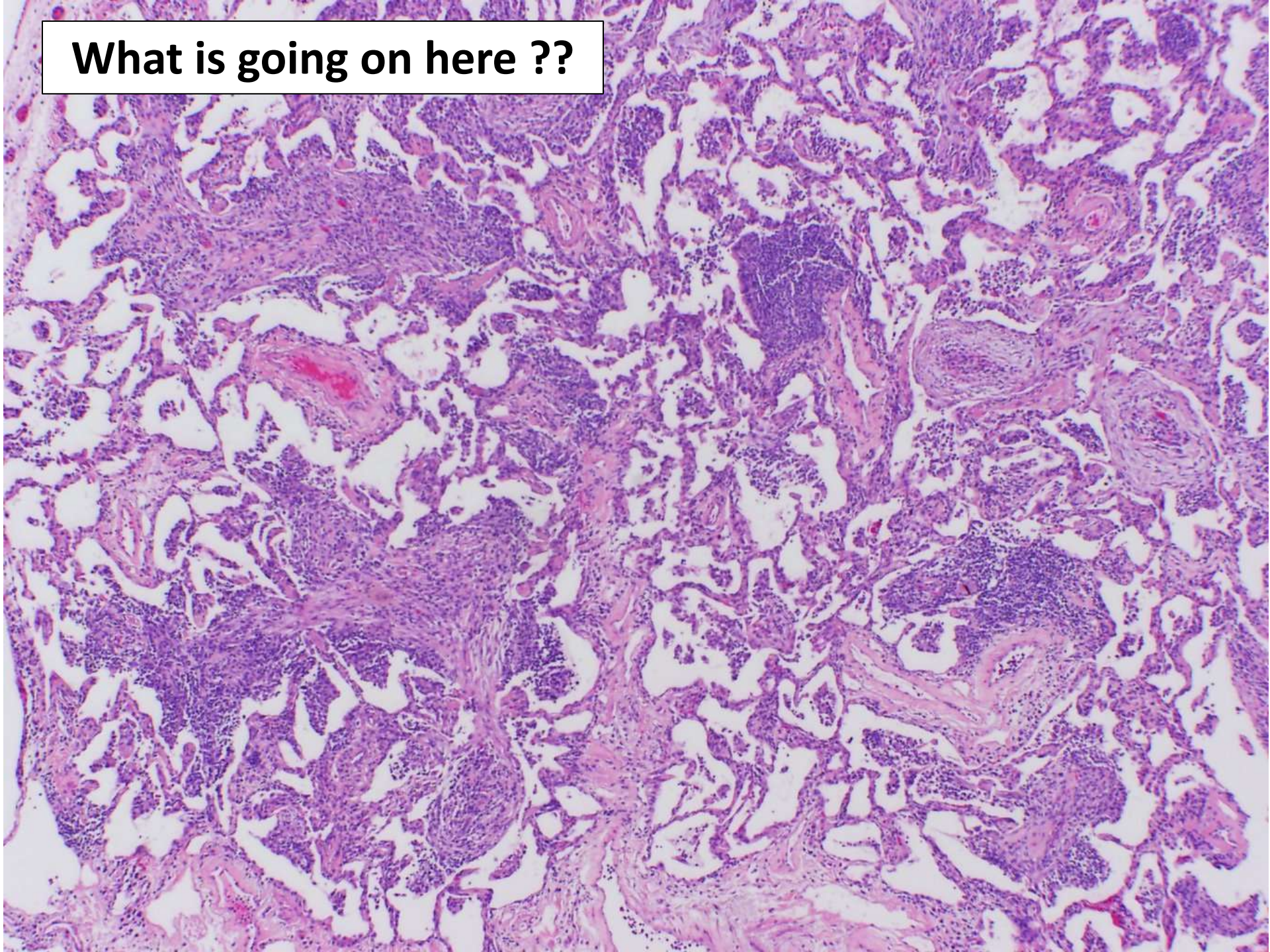
- crime story based on TBB

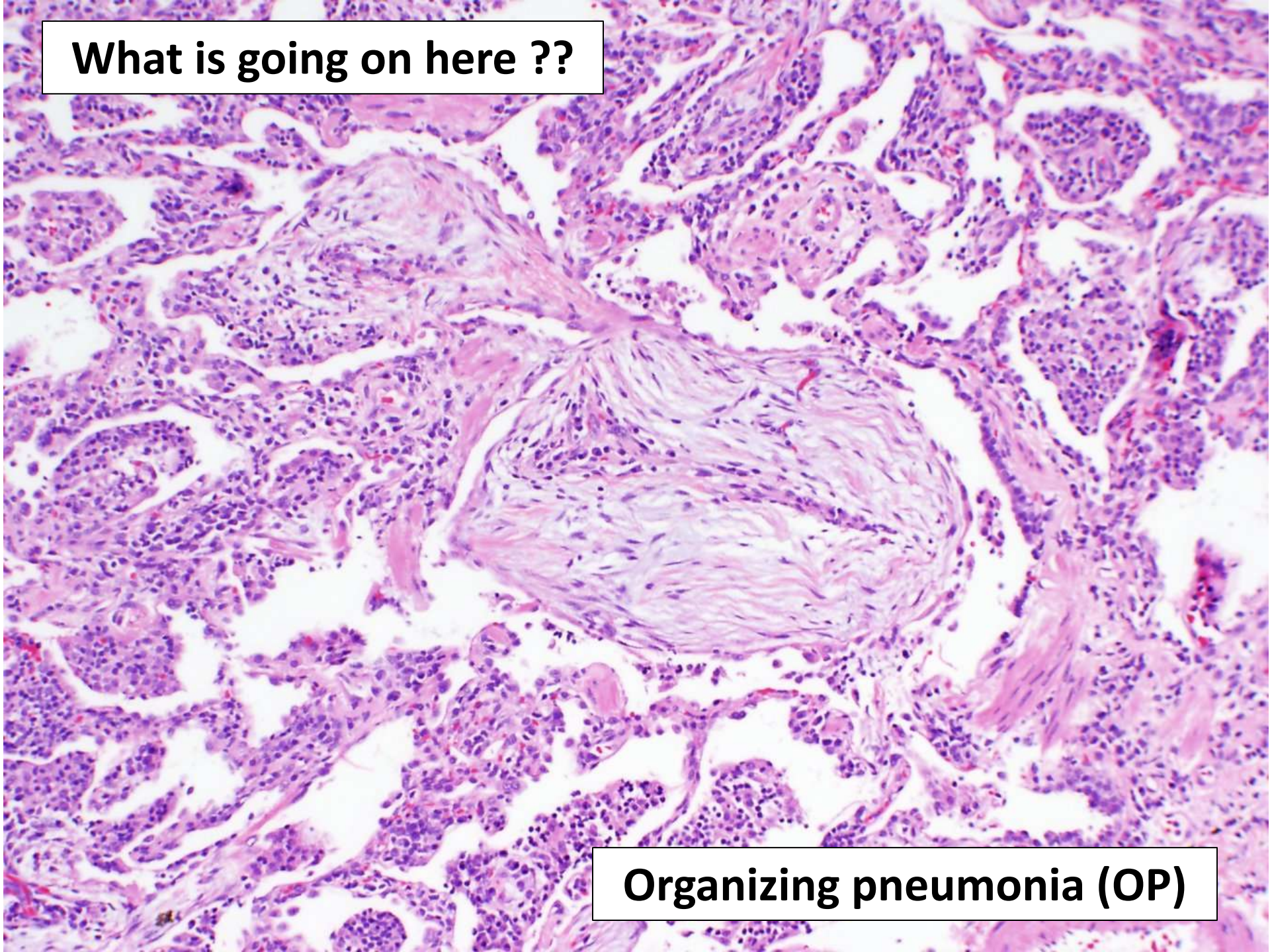
Pathologists - use easy and cheap methods!

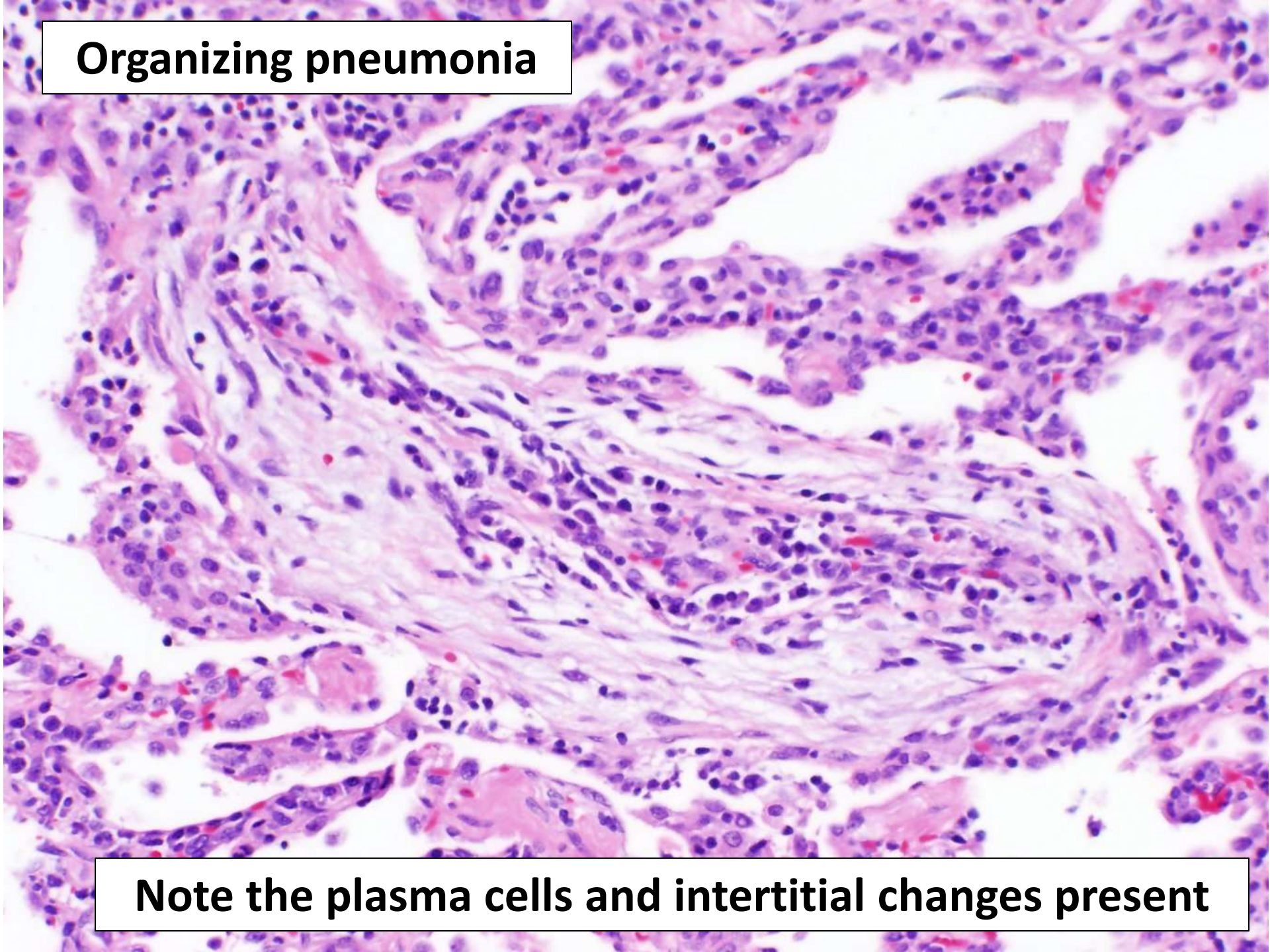
Polarization

Deeper level

Case 3 (TV07-196)


What is going on here ??


What is going on here ??
Organizing pneumonia (OP)


Organizing pneumonia
Note the plasma cells and intertitial changes present

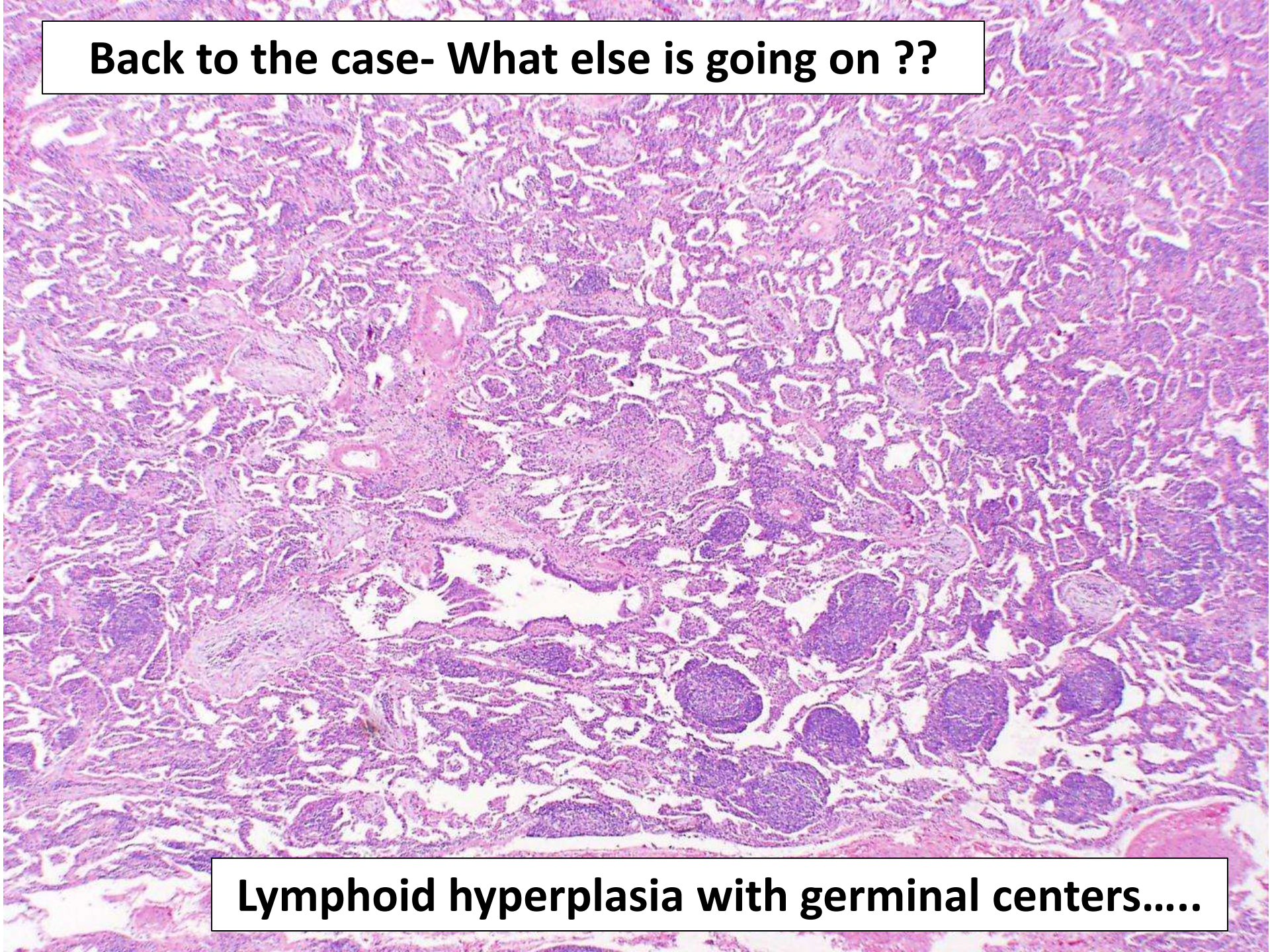
Question 1 TV07-196

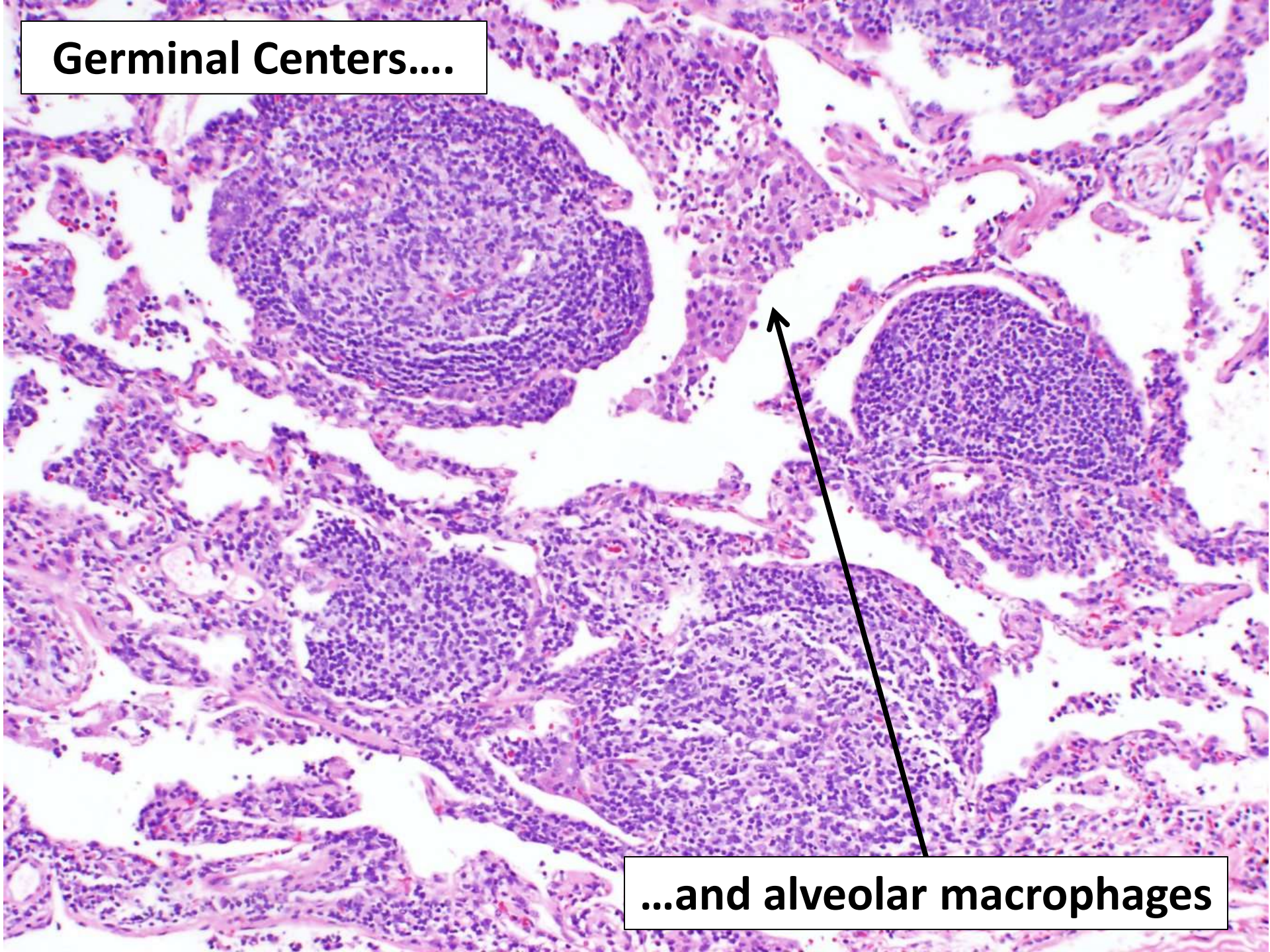
- Organizing pneumonia may be seen in:
  1. Localized nodules
  2. Drug reactions
  3. Organizing infections
  4. Idiopathic interstitial pneumonias
  5. All of the above

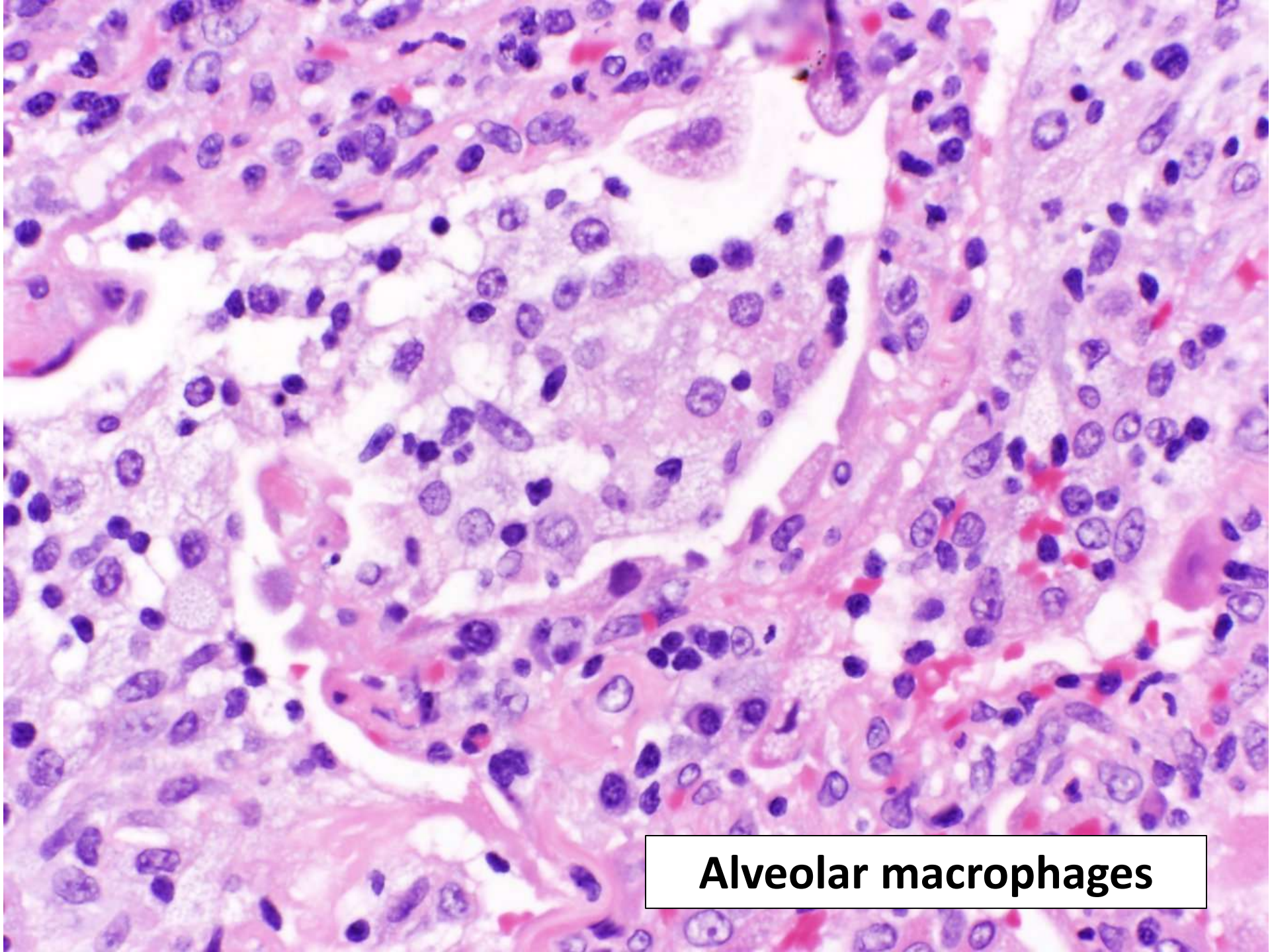
Organizing pneumonia is a very common nonspecific pattern in biopsy material

Here is some history: TV07-196

- 68M with a history of cardiac disease on Rx
- Nonsmoker
- Several weeks of progressive cough and dyspnea
- Patchy ground glass infiltrates on CT scan
- Surgical lung biopsy performed


Back to the case- What else is going on ??
Lymphoid hyperplasia with germinal centers…..


Germinal Centers….
…and alveolar macrophages

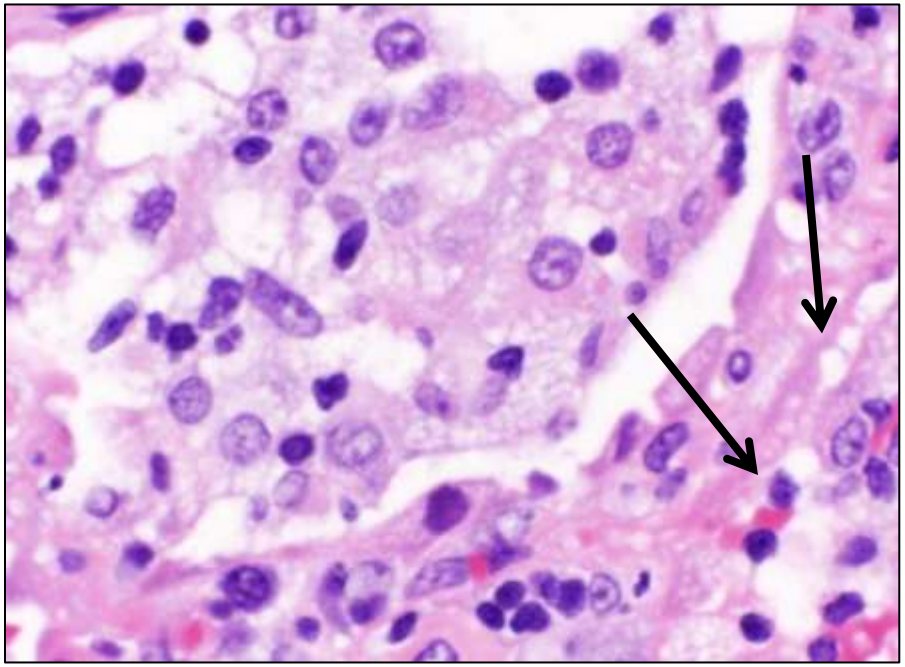

Alveolar macrophages

Question 2 TV07-196

- What diagnosis do you like at this point?
  1. Connective disease
  2. Cryptogenic organizing pneumonitis (COP)
  3. Organizing influenza pnemonia
  4. Drug reaction
  5. None of the above

TV07-196

- 68M with a history of cardiac disease on Rx…
  …which was long term amiodarone



Diagnosis: Changes consistent with amiodarone toxicity

- Note the foamy changes in the macrophages and type 2 cells

Lung Injury Patterns with Amiodarone
(from Pneumotox.com)

I.a	Acute pneumonitis/ILD
I.b	Subacute pneumonitis/ILD
I.c	Pulmonary infiltrates and eosinophilia (PIE)
I.d	Organizing pneumonia (OP/BOOP)
I.f	Acute fibrinous organizing pneumonia (AFOP)
I.g	Pulmonary fibrosis
I.h	Subclinical parenchymal opacities
I.k	Lung nodule or nodules
I.l	Diffuse alveolar damage (DAD)
I.r	Amiodarone pneumonitis
I.s	A mass or masses
I.u	Relapsing or migrating pneumonitis/pneumonia (see also Id)
I.w	Rapidly progressive pulmonary fibrosis

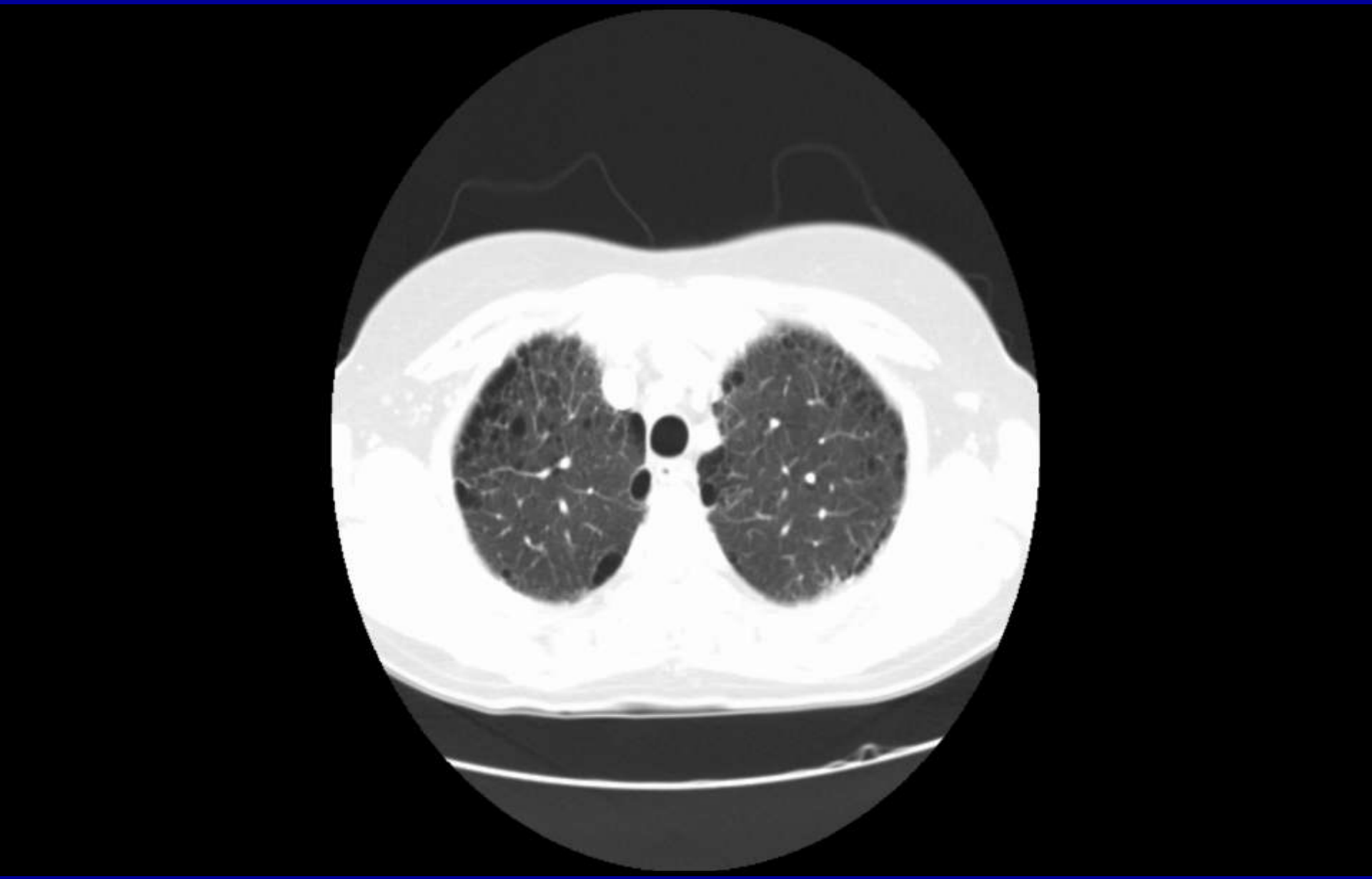
And many more clinical presentation patterns are listed.

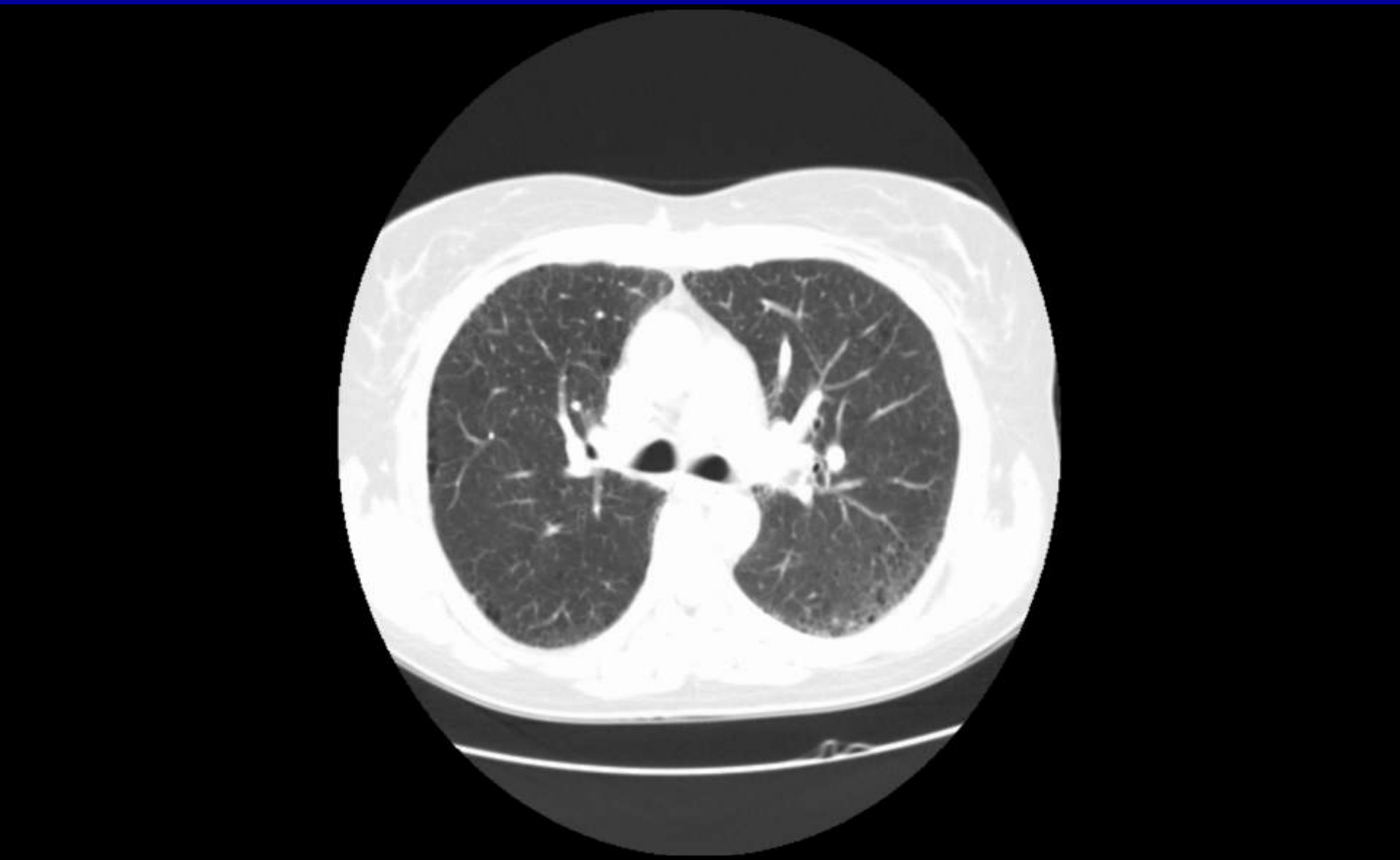
Case presented included in: Larsen BT, et.al. Lymphoid hyperplasia and eosinophilic pneumonia as histologic manifestations of amiodarone-induced lung toxicity. Am J Surg Pathol. 2012; 36:509-16.

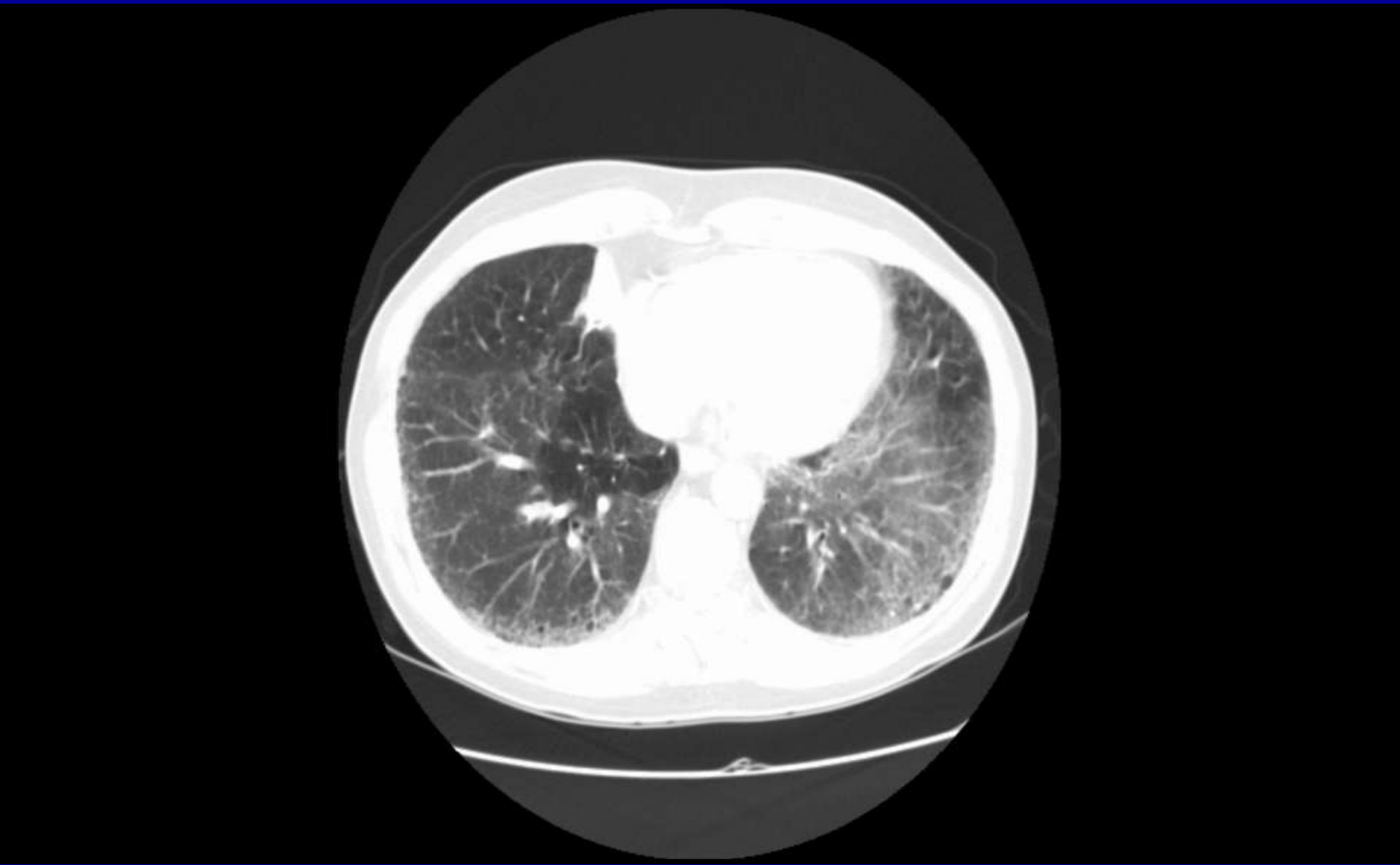
Case No 4

Current symptoms

- 42-year-old lady; waitress
- Smoker (20 daily) for 30 y
- 14 y ago hysterectomy for „cancer“
- Five-month-long unspecific troubles
- Cough (morning), expectoration, SOB
- Allergy: straw, grass, herbs
- Bronchoscopy + TBB – not specific
- BALF: slight lymphocytosis, eosinophilia
- RTG – diffuse bilateral small nodules - CT



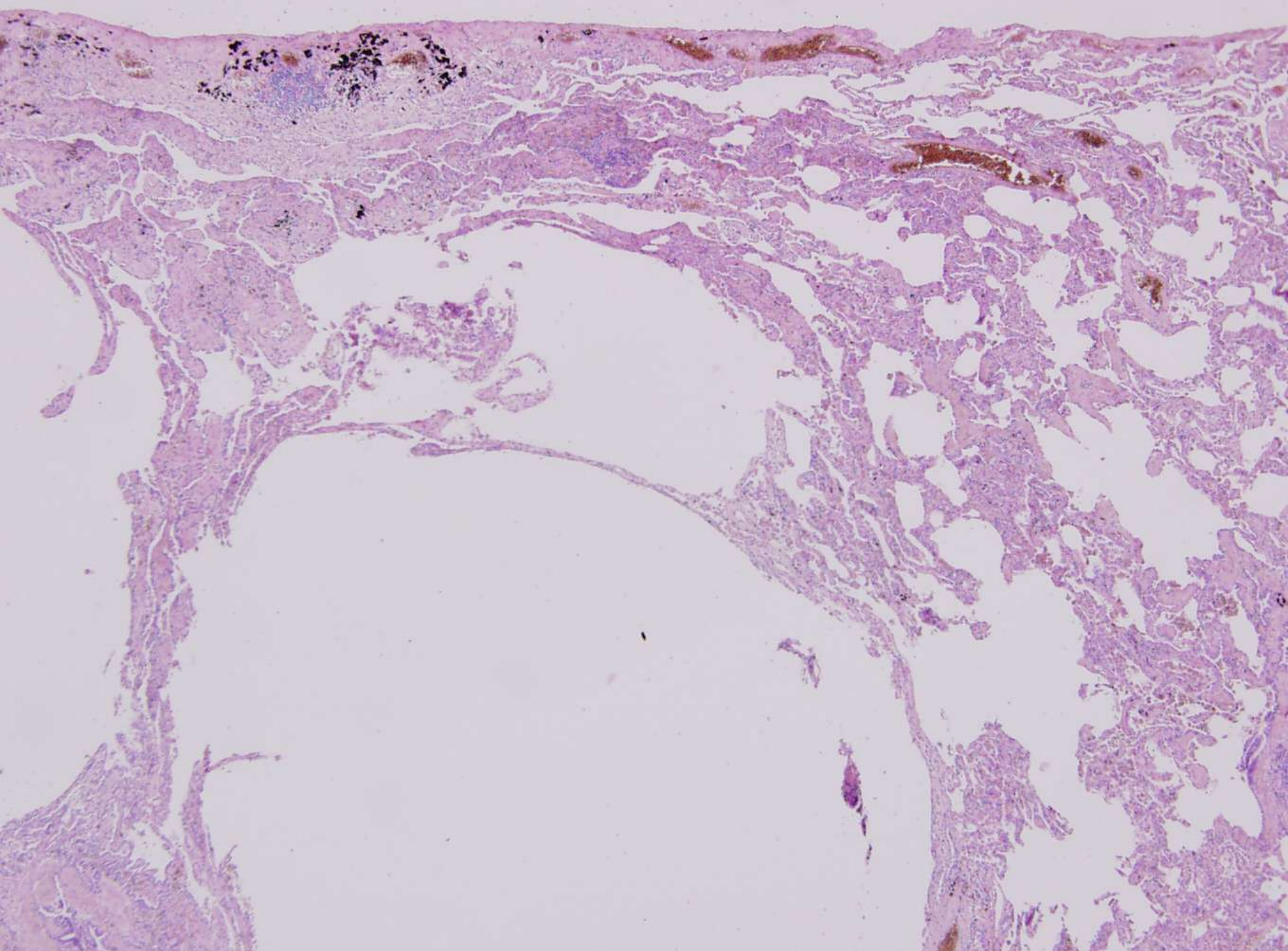


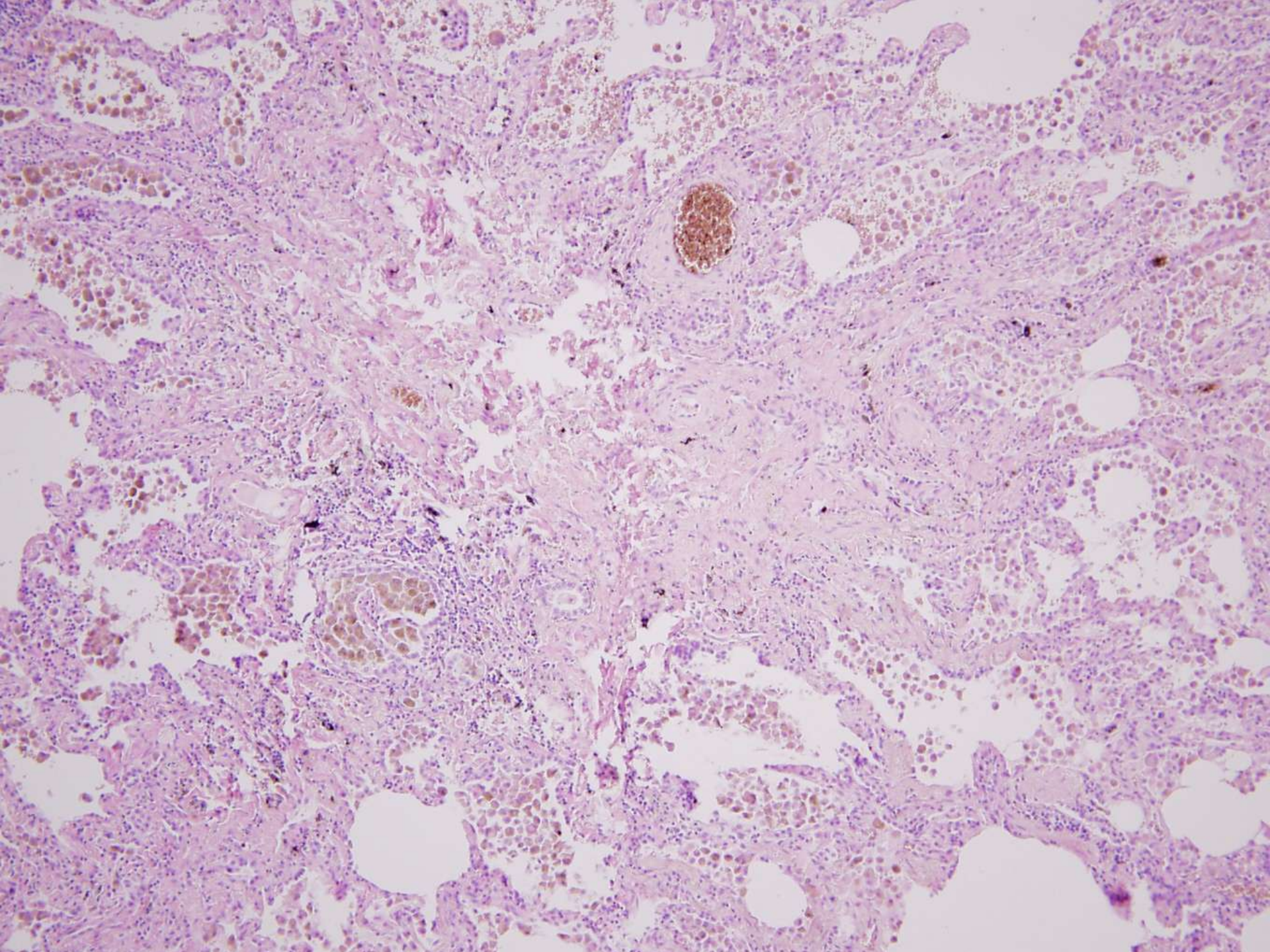


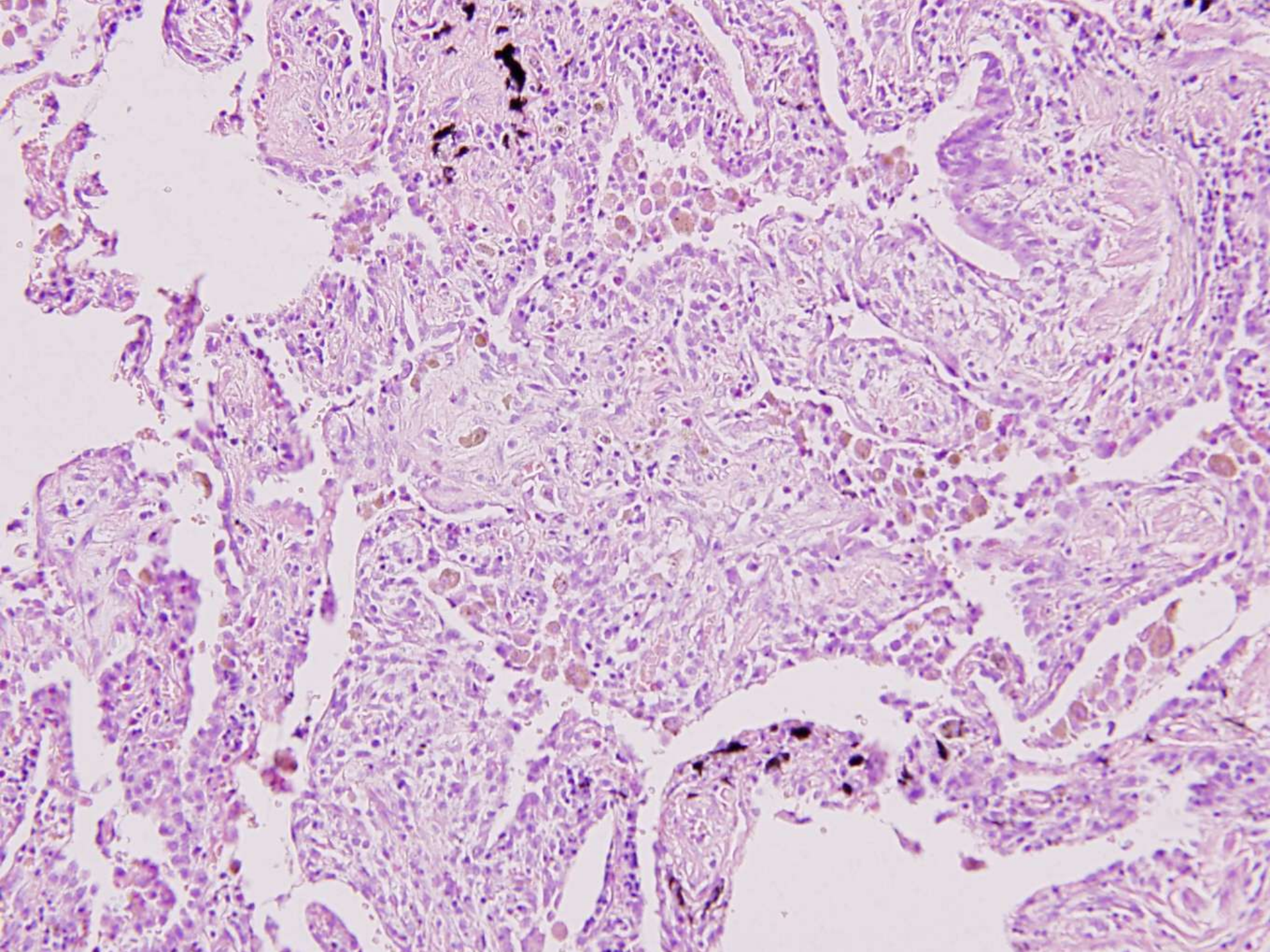
Dif.dg

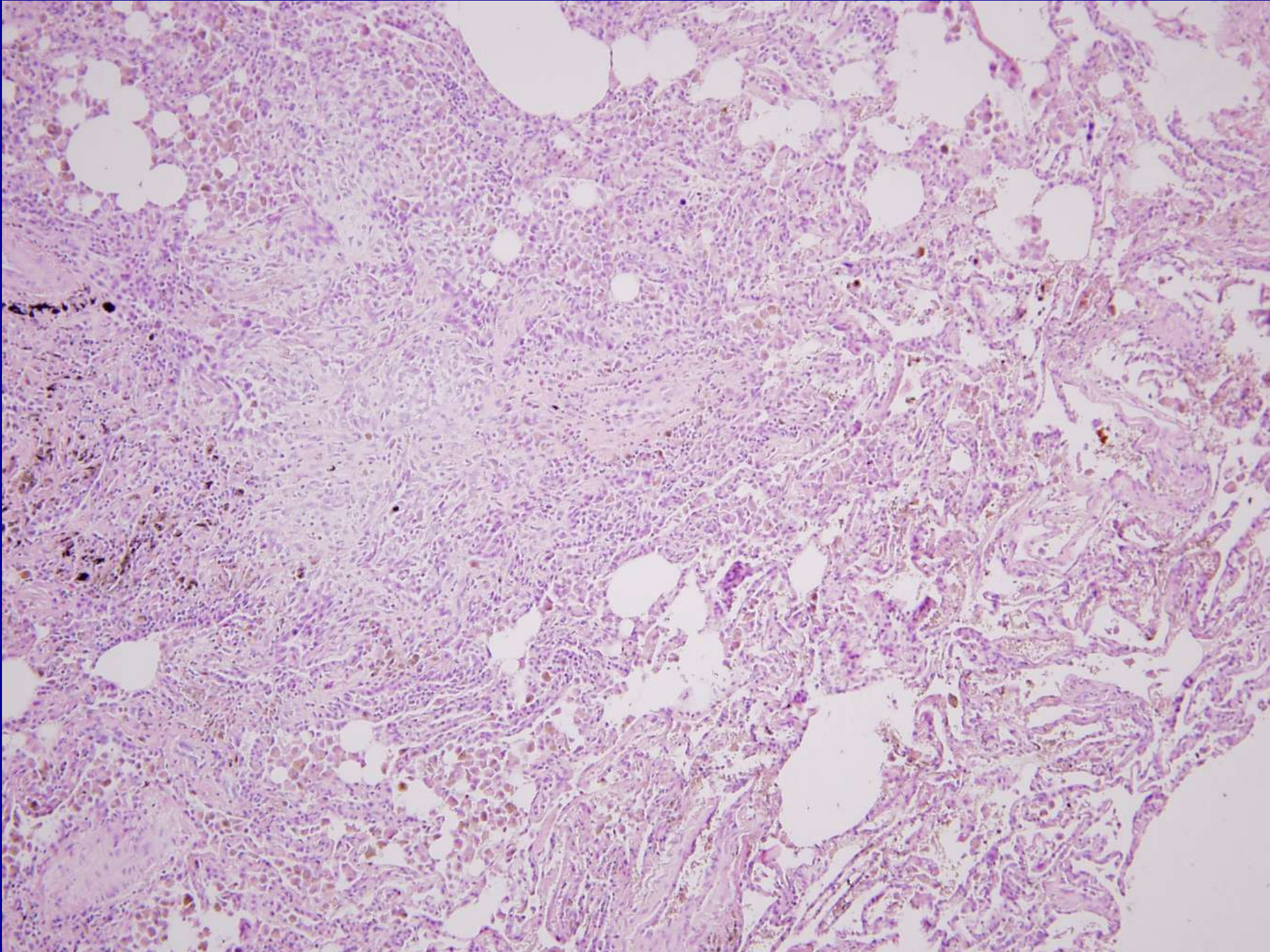
- ILD - HP
- TBC
- Tumor metastasis

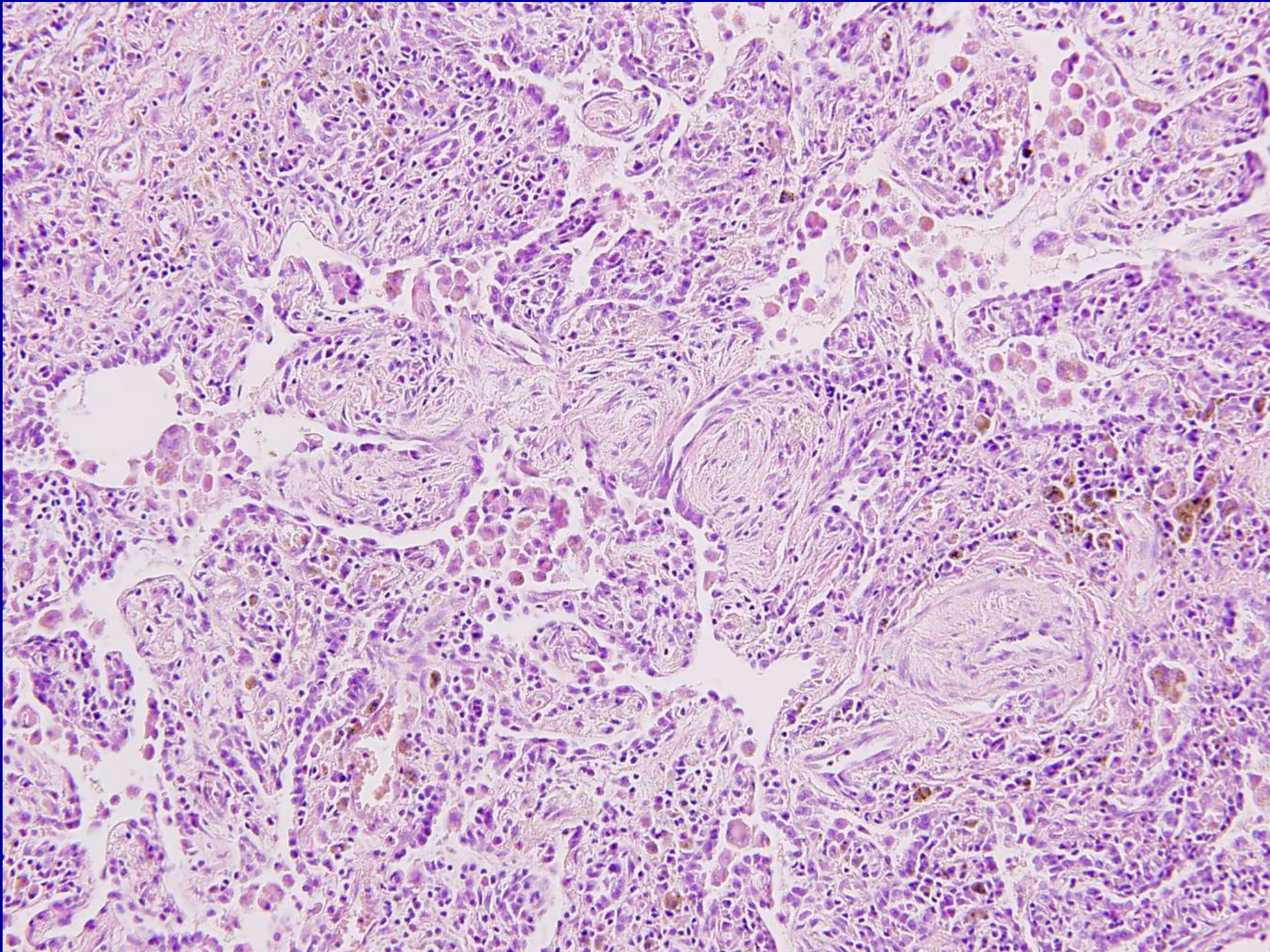
- VATS surgical biopsy

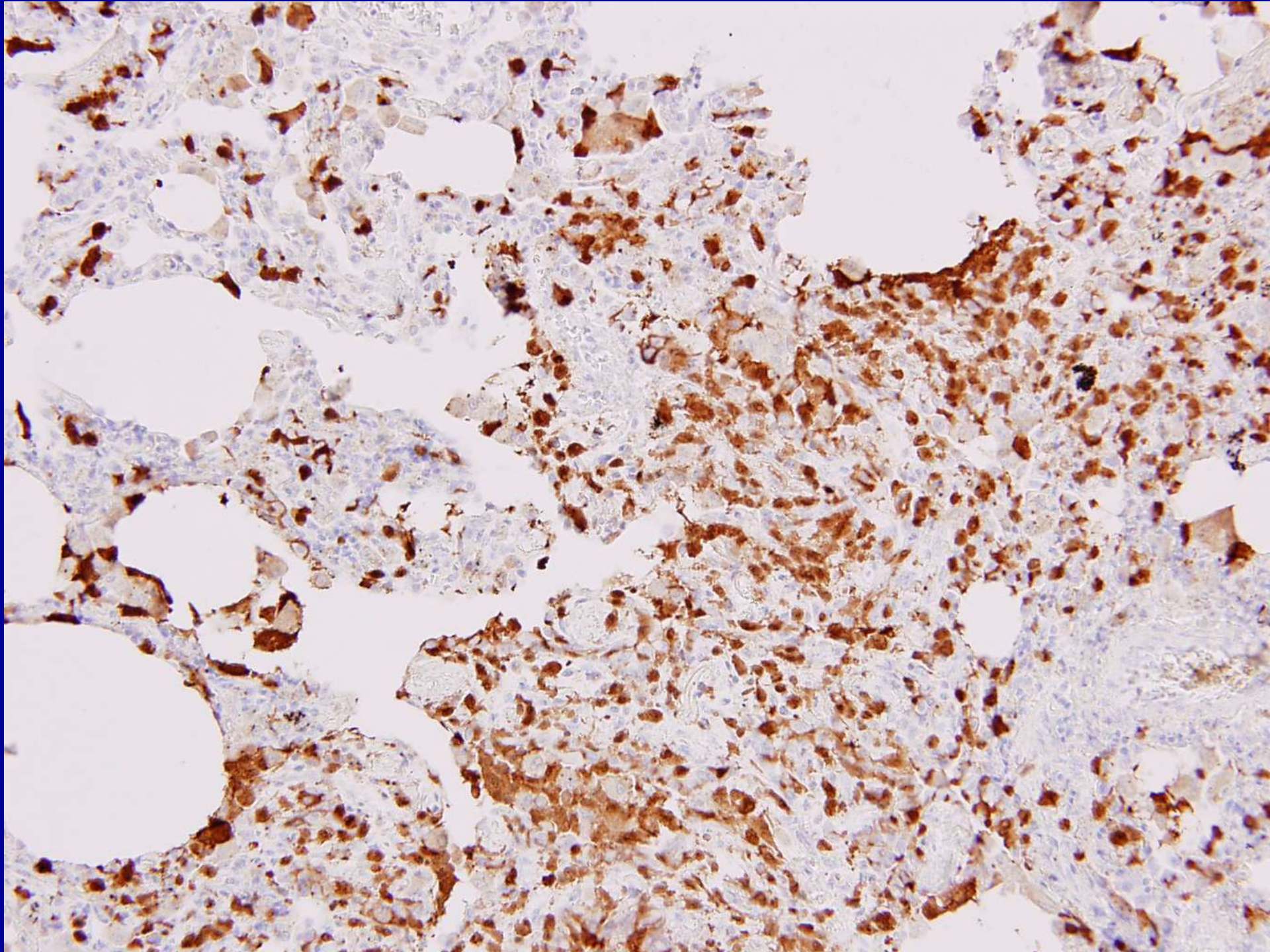


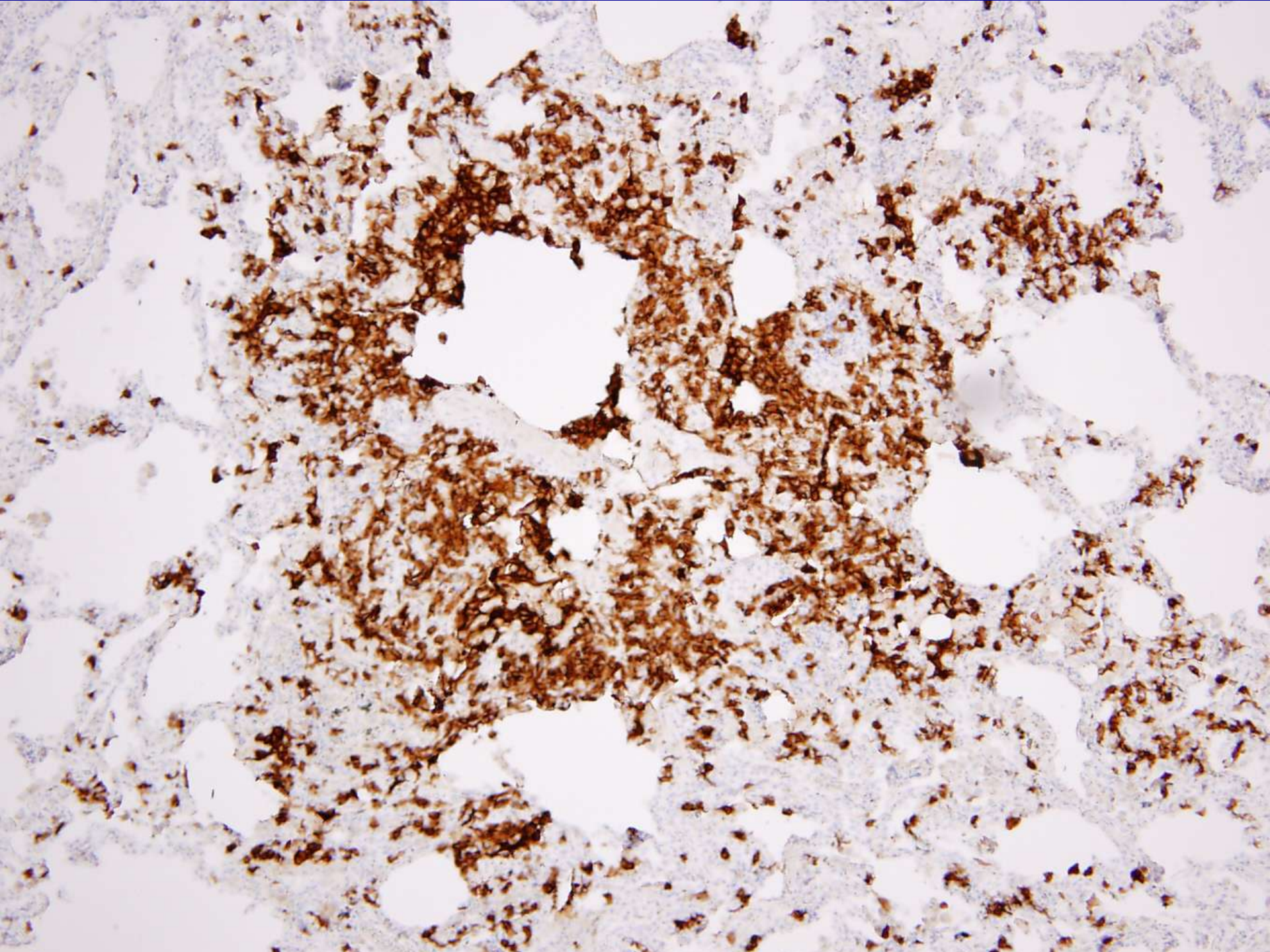












DIAGNOSIS ????

1. HP
2. Sarcoidosis
3. Generalisation of cancer
4. LCH
5. SRIF
6. COPD

Combination of smoking-related changes:

COPD

SRIF

LCH

Eur Respir J 2005; 26: 586–593
DOI: 10.1183/09031936.05.00021005

CLINICAL FORUM

Combined pulmonary fibrosis and emphysema: a distinct underrecognised entity

V. Cottin*, H. Nunes[#], P-Y. Brillet[¶], P. Delaval[+], G. Devouassoux[§], I. Tillie-Leblond[ƒ], D. Israel-Biet, I. Court-Fortune[##], D. Valeyre[#], J-F. Cordier* and the Groupe d'Etude et de Recherche sur les Maladies "Orphelines" Pulmonaires (GERM"O"P)**

ABSTRACT: **The syndrome resulting from combined pulmonary fibrosis and emphysema has not been comprehensively described.**

The current authors conducted a retrospective study of 61 patients with both emphysema of the upper zones and diffuse parenchymal lung disease with fibrosis of the lower zones of the lungs on chest computed tomography.

Patients (all smokers) included 60 males and one female, with a mean age of 65 yrs. Dyspnoea on exertion was present in all patients. Basal crackles were found in 87% and finger clubbing in 43%. Pulmonary function tests were as follows (mean±SD): total lung capacity 88%±17, forced vital capacity (FVC) 88%±18, forced expiratory volume in one second (FEV_1) 80%±21 (% predicted), FEV_1/FVC 69%±13, carbon monoxide diffusion capacity of the lung 37%±16 (% predicted), carbon monoxide transfer coefficient 46%±19. Pulmonary hypertension was present

AFFILIATIONS
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[#]Service de pneumologie, and
[¶]Service de radiologie, Hôpital Avicenne, Bobigny,
[+]Dépt de pneumologie et cardiologie, Centre Hospitalier Universitaire de Rennes,
[§]Service de pneumologie, centre

Modern Pathology (2006) 19, 1474–1479

www.modernpathology.org

Respiratory bronchiolitis-associated interstitial lung disease with fibrosis is a lesion distinct from fibrotic nonspecific interstitial pneumonia: a proposal

Samuel A Yousem

Department of Pathology, University of Pittsburgh Medical Center, Pittsburgh, PA, USA

Nine cases of clinical and radiographic chronic interstitial lung disease are presented that have features of respiratory bronchiolitis-associated interstitial lung disease, but were associated with a respiratory bronchiolitis having extensive paucicellular lamellar eosinophilic collagenous thickening of alveolar septa in a patchy, particularly subpleural distribution. Patients were middle-aged with shortness of breath, mixed obstructive and restrictive lung disease with markedly reduced diffusing capacity and radiographs demonstrating centrilobular micronodules, occasional ground glass opacities and emphysema. All were alive at follow-up. The morphology of this process raises the differential diagnosis with the fibrotic form of nonspecific interstitial pneumonia and highlights the role of cigarette smoking as a potential cause of fibrotic lung disease.
Modern Pathology (2006) **19**, 1474–1479. doi:10.1038/modpathol.3800671; published online 1 September 2006

Keywords: respiratory bronchiolitis; nonspecific interstitial pneumonia; desquamative interstitial pneumonia; cigarette smoking; tobacco use

RadioGraphics

Smoking-related Interstitial Lung Disease: Radiologic-Clinical-Pathologic Correlation[1]

TEACHING POINTS
See last page

ONLINE-ONLY CME
See www.rsna.org/education/rg_cme.html

LEARNING OBJECTIVES
After reading this article and taking the test, the reader will be able to:

■ Describe the spectrum of radiologic

Anil K. Attili, FRCR • Ella A. Kazerooni, MD, MS • Barry H. Gross, MD
Kevin R. Flaherty, MD • Jeffrey L. Myers, MD • Fernando J. Martinez, MD

Cigarette smoking is a recognized risk factor for development of interstitial lung disease (ILD). There is strong evidence supporting a causal role for cigarette smoking in development of respiratory bronchiolitis ILD (RB-ILD), desquamative interstitial pneumonitis (DIP), and pulmonary Langerhans cell histiocytosis (PLCH). In addition, former and current smokers may be at increased risk for developing idiopathic pulmonary fibrosis (IPF). The combination of lower lung fibrosis and upper lung emphysema is being increasingly recognized as a distinct clinical entity in smokers. High-resolution computed tomography

Human Pathology (2010) xx, xxx–xxx

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Original contribution

Clinically occult interstitial fibrosis in smokers: classification and significance of a surprisingly common finding in lobectomy specimens

Anna-Luise A. Katzenstein MD[a,*], Sanjay Mukhopadhyay MD[a], Conrado Zanardi MD[a], Elizabeth Dexter MD[b]

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Received 3 August 2009; revised 3 September 2009; accepted 9 September 2009

MODERN PATHOLOGY (2011), 1–11

Smoking-related interstitial fibrosis (SRIF), pathogenesis and treatment of usual interstitial pneumonia (UIP), and transbronchial biopsy in UIP

Anna-Luise A Katzenstein

Department of Pathology, SUNY Upstate Medical University, Syracuse, NY, USA

This review focuses on three selected topics of current interest that are related to chronic fibrosing lung disorders and are important for pathologists. First, the clinical and pathologic features of smoking-related interstitial fibrosis (SRIF) are highlighted. SRIF is a common finding in smokers that has striking histologic changes but only mild associated clinical manifestations. It is characterized by marked alveolar septal fibrosis composed of a distinct form of hyalinized collagen deposition. The process is present mainly in subpleural and centrilobular parenchyma and is associated with emphysema and respiratory bronchiolitis. Second, important aspects of the pathogenesis and treatment of usual interstitial pneumonia (UIP) are reviewed. The current theory proposes that UIP is caused by tiny foci of acute lung injury (manifest pathologically by fibroblast foci) that occur and recur in the interstitium over many years. Inflammation may be present as a secondary

SRIF Correlations

- Interpretation is complicated
- Presence of changes
  - Original work 60% fibrosis over 25% parenchyma
  - Kawabata 6,5% light a 21,1% heavy smokers
- Clinics:
  - Progression of COPD masks restriction (up 10%)
- HRCT:
  - Micronodules with GGO bilat
  - Progression of COPD masks interstitial changes
  - Retrospective studies – changes 23% smokers

Multidisciplinary approach

SRIF – Meaning & viewing

- Clinical picture
- HRCT pattern
- Prognosis
- Therapy

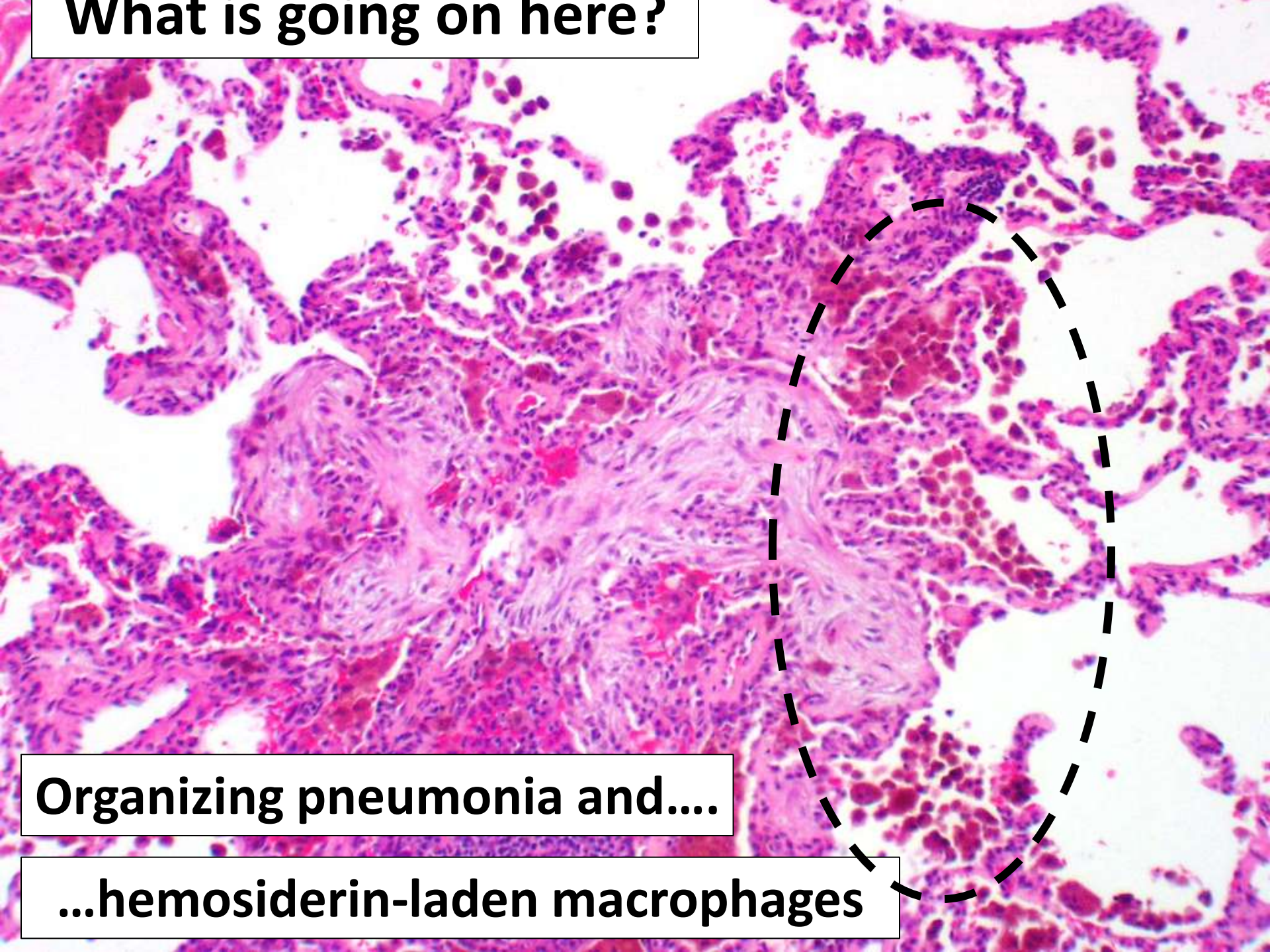
- Dif dg other fibrosing ILD
  - UIP, CPFE, fibrosing NSIP, LCH

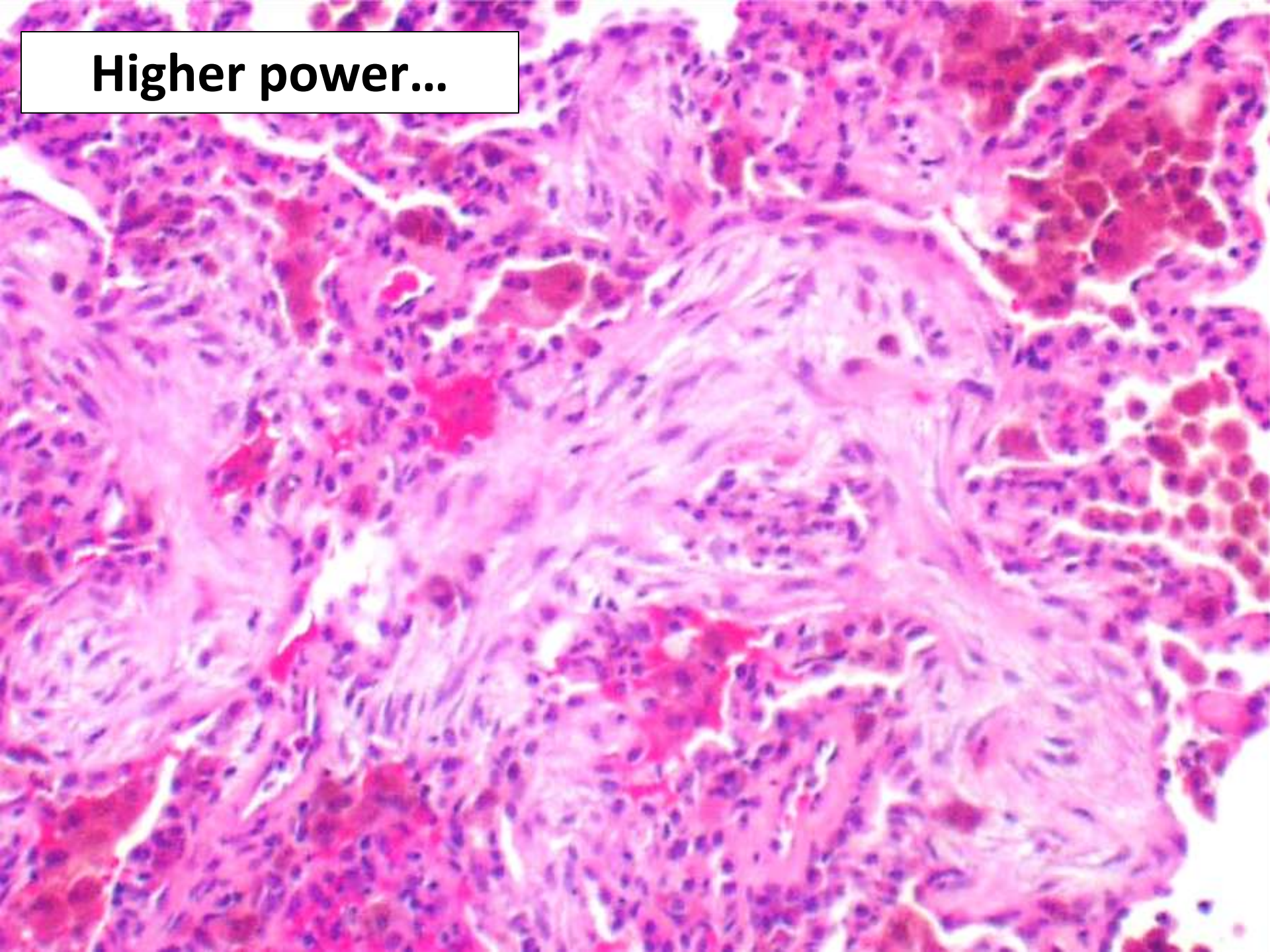
SMOKING KILLS!

…and complicates lifes and diagnosis

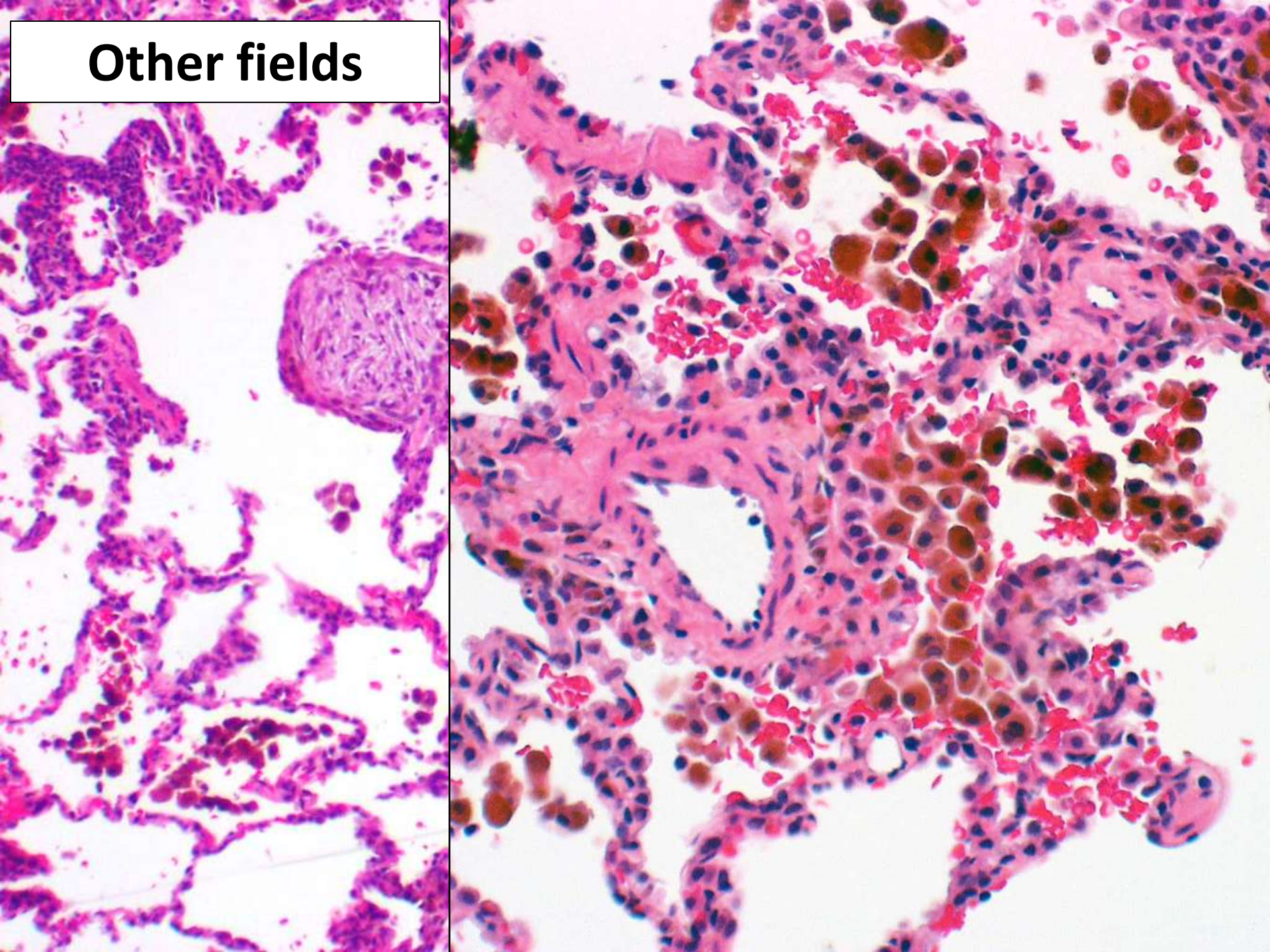
especially for pathologists

CASE 5


What is going on here?
Organizing pneumonia and….
…hemosiderin-laden macrophages


Higher power…

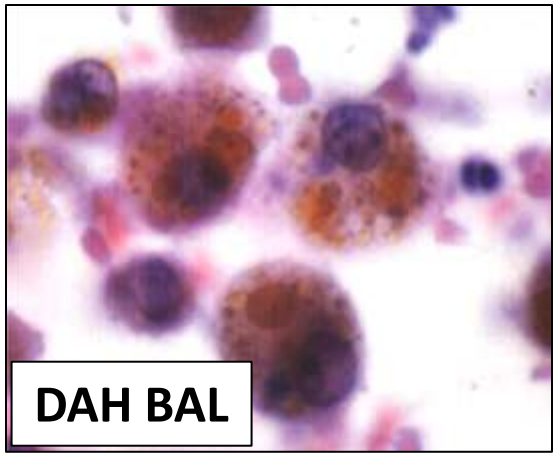
Other fields

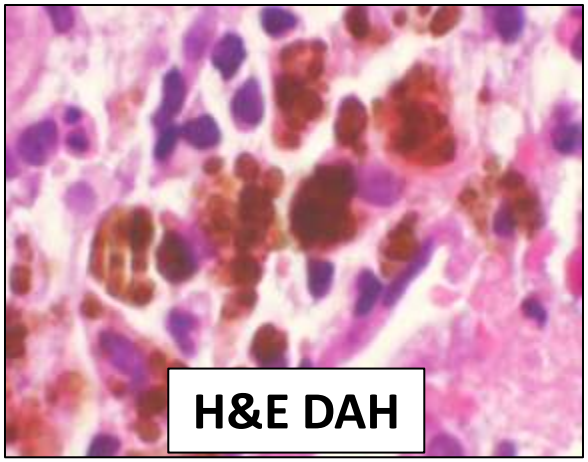


CASE 5 QUESTION 1

- Hemosiderin-laden macrophages can be seen in:
  1. Smokers
  2. Welders
  3. Alveolar hemorrhage
  4. Heart failure
  5. All of the above

Smoking related changes differ somewhat from hemorrhage





CASE 5 QUESTION 2

- The changes in CASE 3 could be seen in:
  1. Diffuse alveolar hemorrhage
  2. Organizing pneumonia in a patient with cardiac failure
  3. Organizing hemorrhagic infection
  4. Organizing pneumonia in a welder
  5. All of the above

NOW FOR SOME CASE HISTORY

- **20F with shortness of breath, hemoptysis, patchy radiologic infiltrates, and positive c-ANCA**
- **She also had hematuria**

CASE 5 QUESTION 2

- What is the best diagnosis?
  1. Diffuse alveolar hemorrhage syndrome
  2. Pulmonary-renal syndrome with alveolar hemorrhage
  3. ANCA-associated vasculitis
  4. c-ANCA positive diffuse alveolar hemorrhage
  5. Granulomatosis with polyangiitis (GPA)
     (formerly Wegener's)

Answers will differ with one's specialty and philosophy

DIFFUSE ALVEOLAR HEMORRHAGE(DAH)

- **Pulmonary hemorrhage not due to trauma, airway disease, tumors, or heart failure**
- **Usually recurrent; may be acute or chronic**
- **Typically with dyspnea, hemoptysis, airspace infiltrates, and anemia**
- **Associated renal disease common**

IMMUNOLOGIC CLASSIFICATION OF DIFFUSE ALVEOLAR HEMORRHAGE

- **ANCA-associated:** WG, MPA, pulmonary renal syndromes, isolated alveolar hemorrhage
- **Antibasement membrane antibody:** Goodpasture's syndrome, isolated alveolar hemorrhage
- **Immune complex deposition:** Collagen vascular diseases, IgA disease, pulmonary renal syndromes, isolated alveolar hemorrhage
- **Immunologic mechanism not identified:** IPH, isolated alveolar hemorrhage

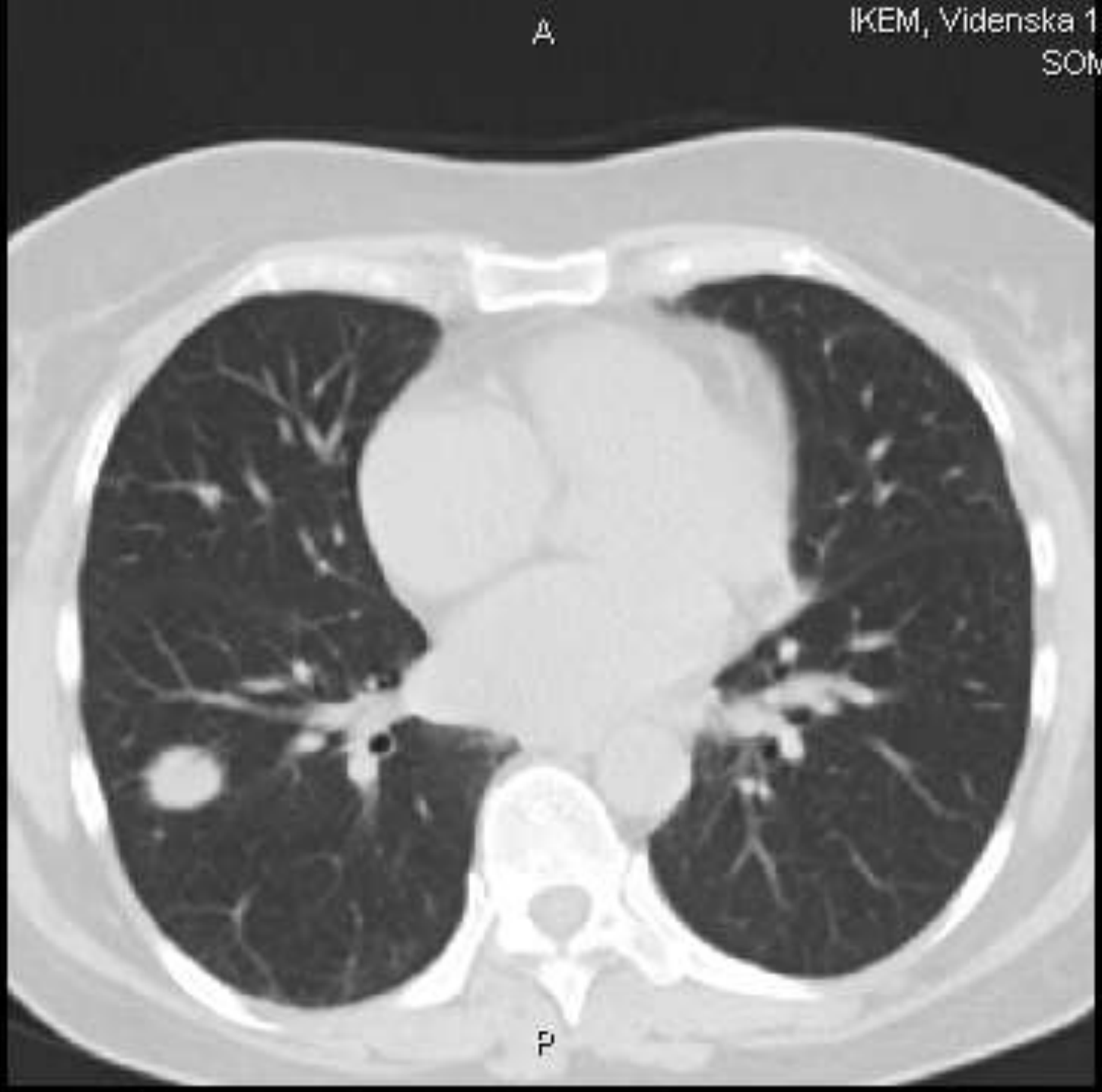
CASE 5

- **Diagnosis: ANCA-positive diffuse alveolar hemorrhage (compatible with GPA)**
- **Follow-up: The patient responded to standard therapy for GPA and was well at follow-up several years later**

Case No 6

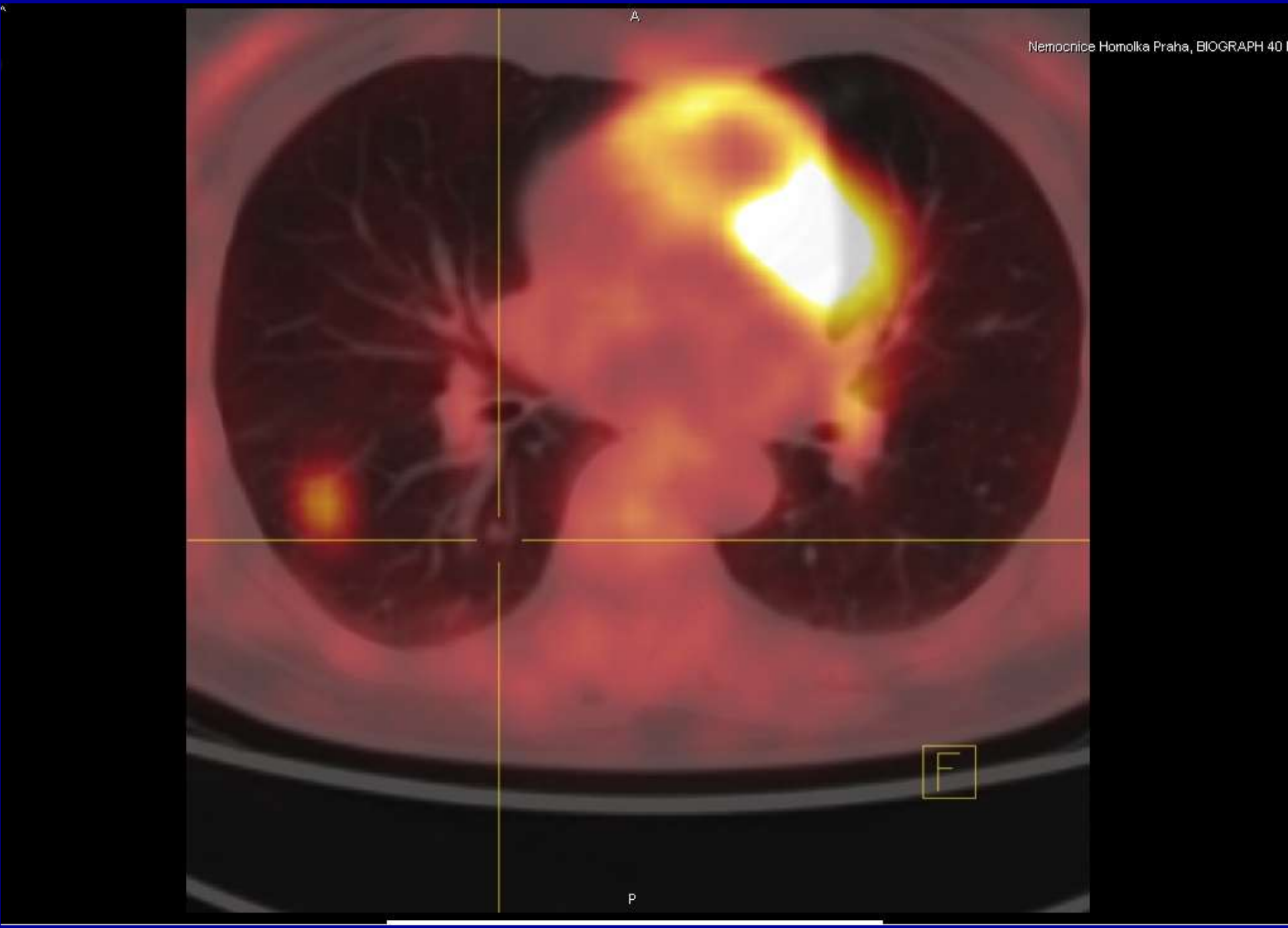
Current symptoms

- 53-year-old lady; MD
- Nonsmoker, teetotaller
- Breeding station for cats (actually 6)
- 2007 melanoma of left hand
- Unspecific dyspepsia
- CT – small cysts of liver and kidney
- BUT - S6 and near left heart ventricule two nodules with contrast accumulation


A
IKEM, Videnska 1958/9, 14
SOMATOM D
P

Clinical investigations

- PET-CT
  - Not only two nodules, but multiple bilat!
- bronchoscopy, TBB, EBUS no cancer
- Autoantibodies negative, only ANA Ig 1:80
- Oncoscreening negative


A
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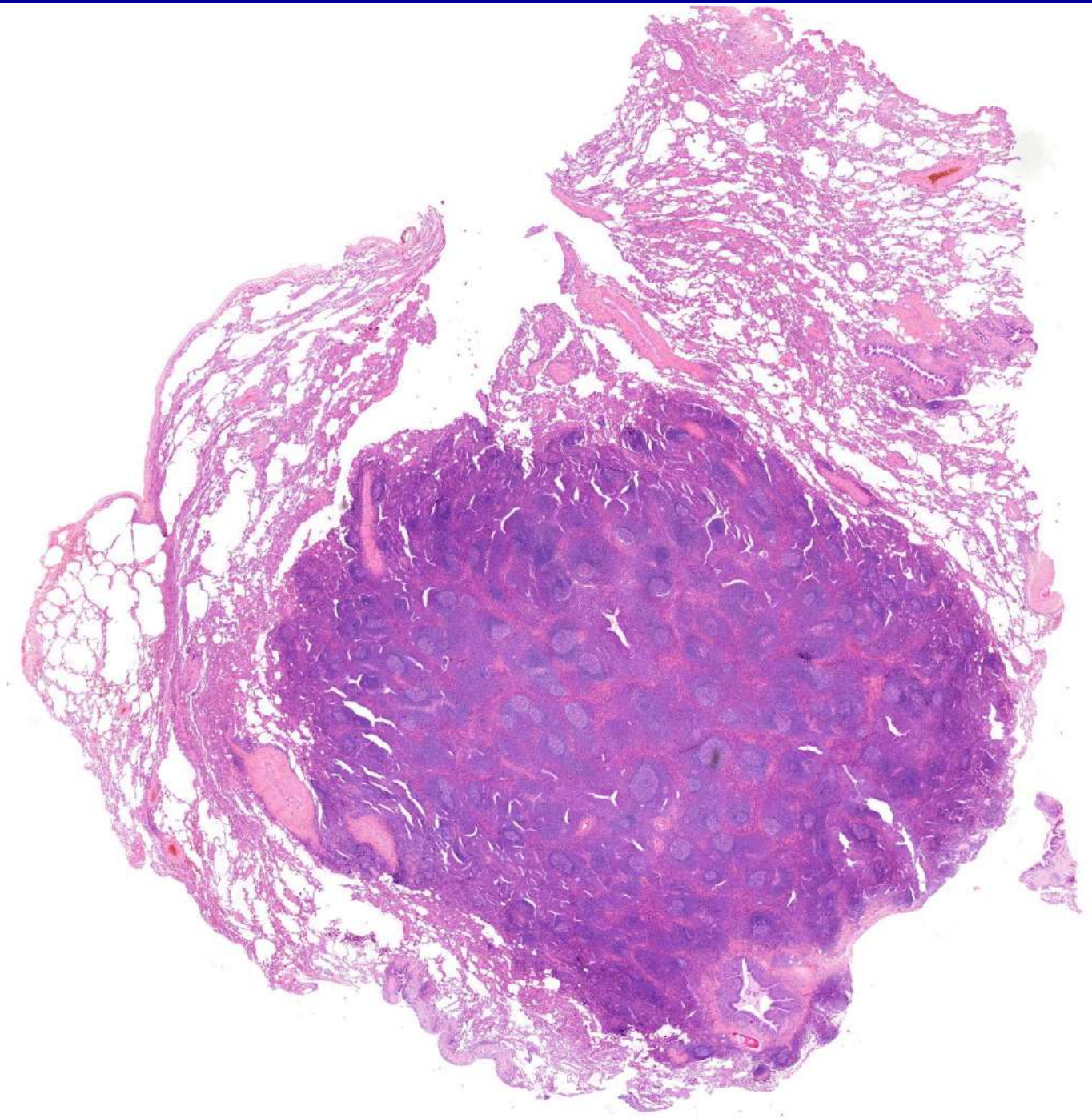
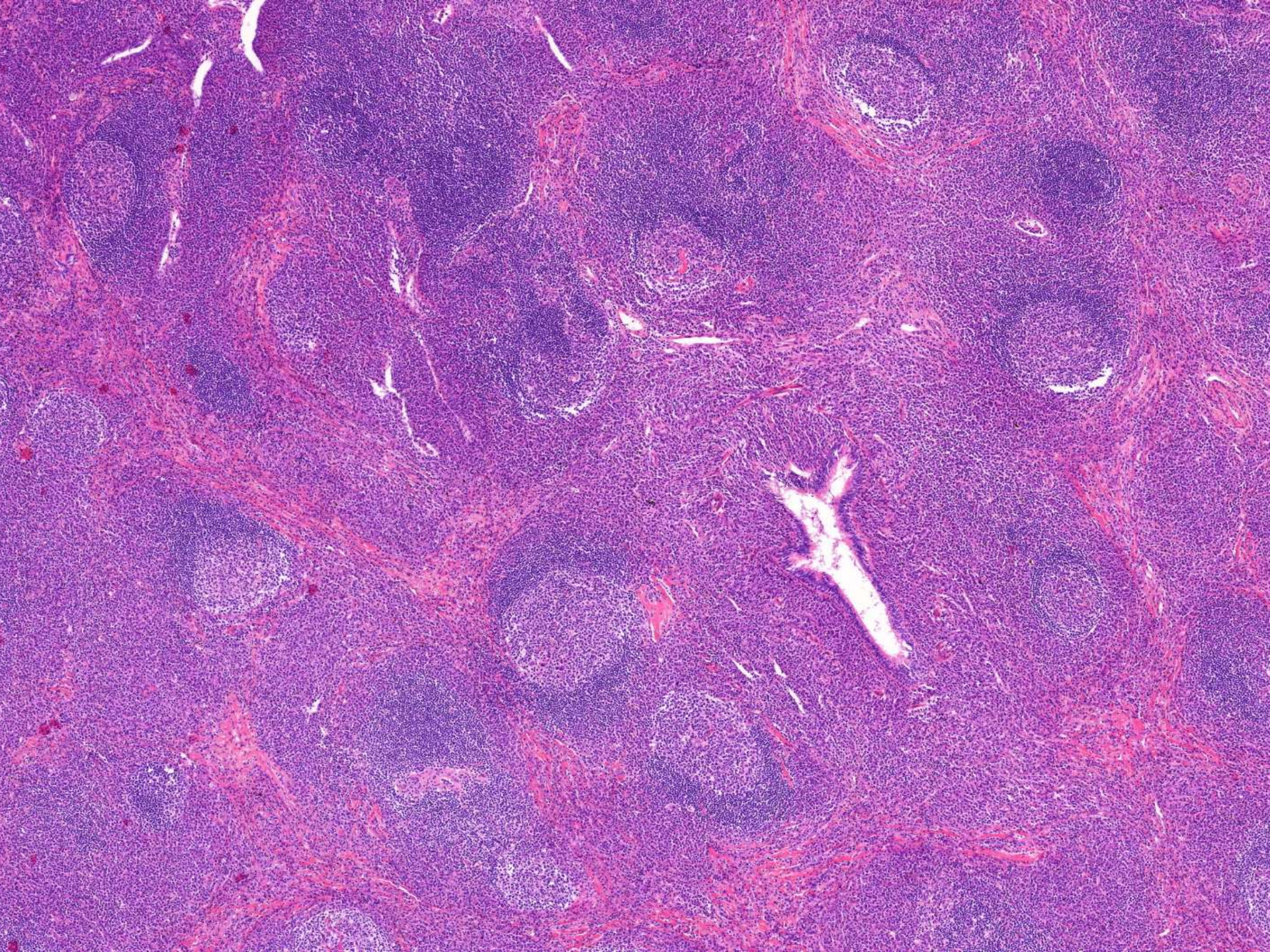
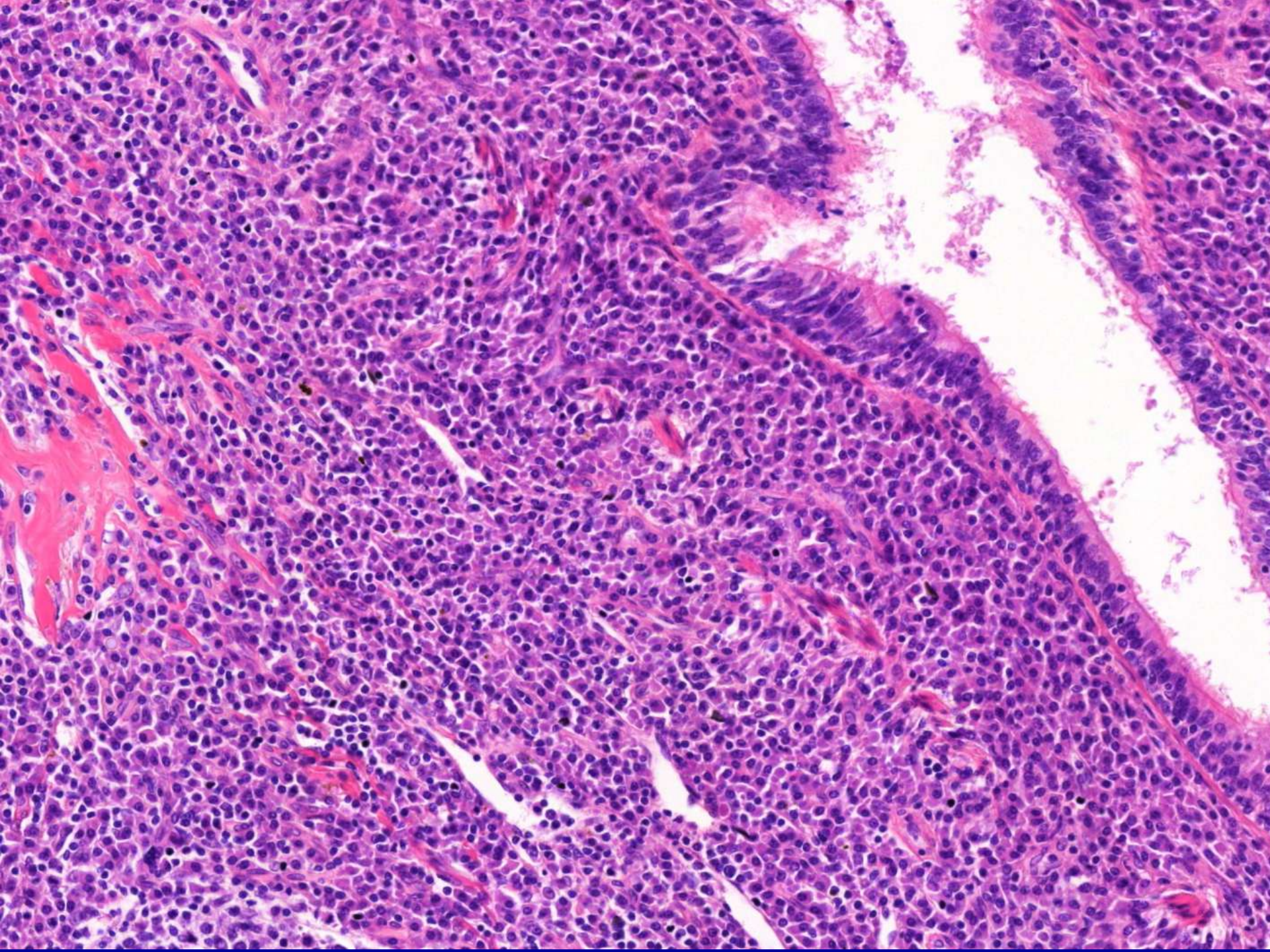
Dif. dg

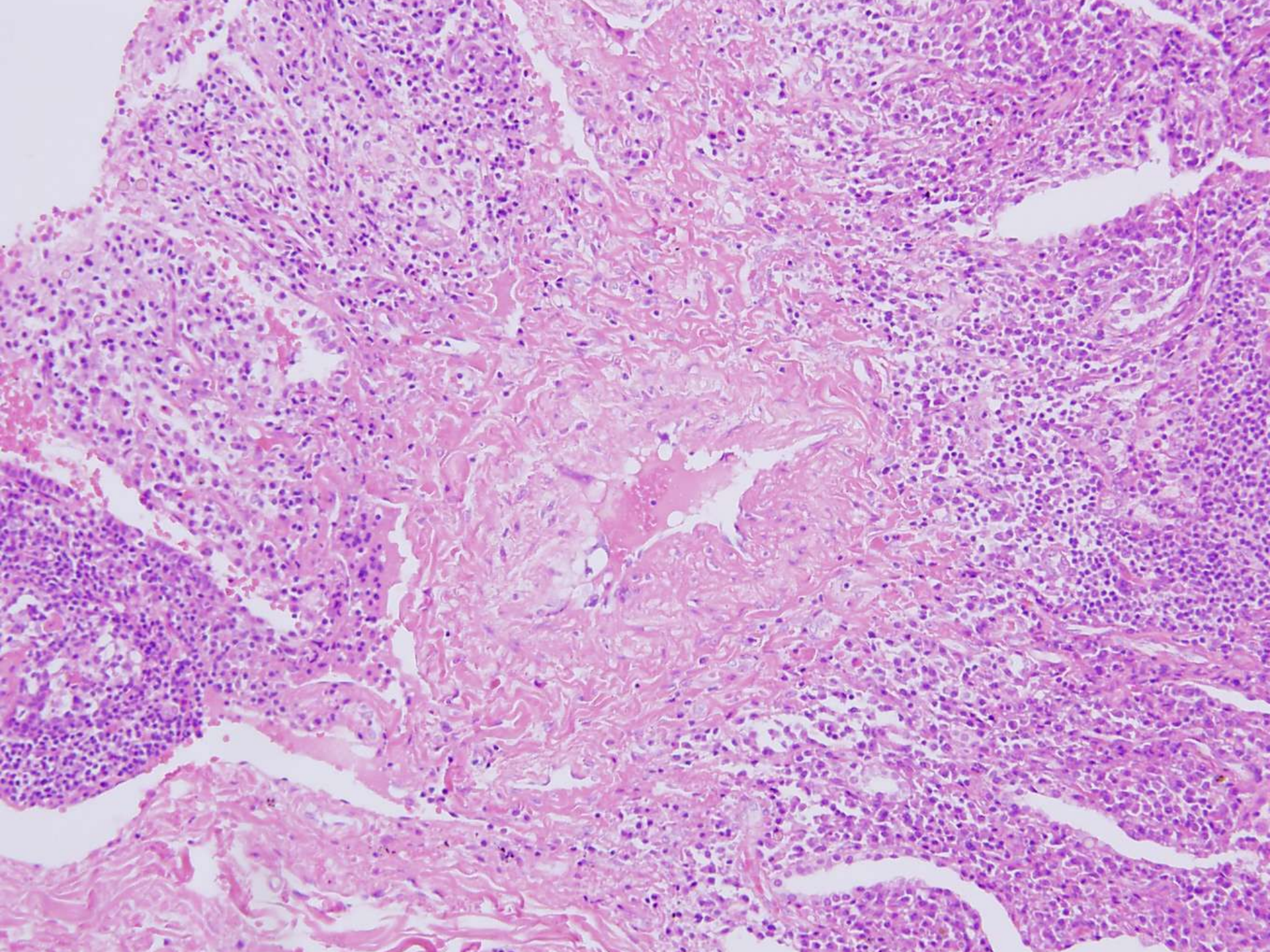
- Primary lung tumor
- Autoimmune disease
- TBC, mycotic infection
- Metastatic spread of tumor (melanoma?)

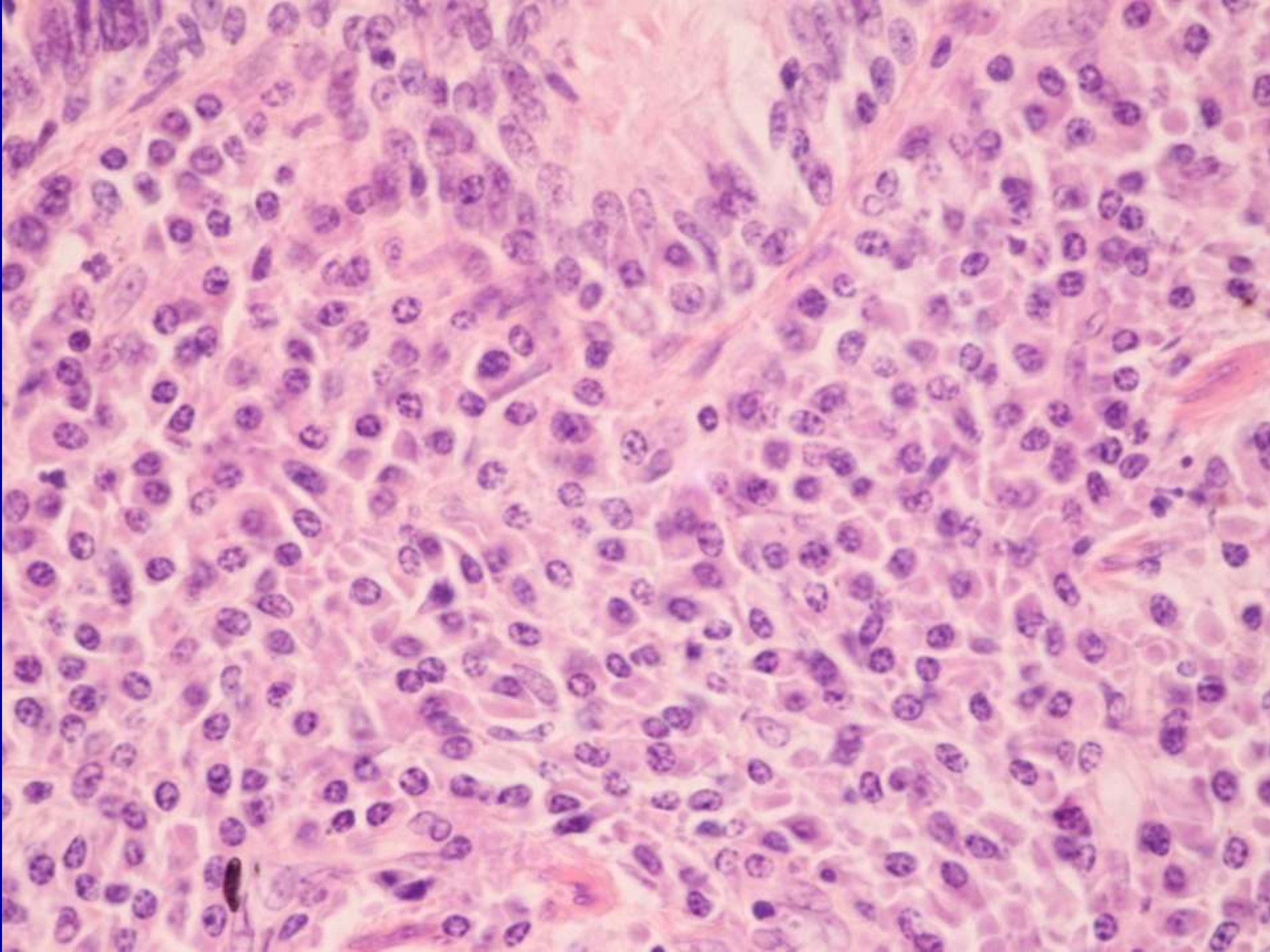
- VATS with surgical biopsy of nodule.

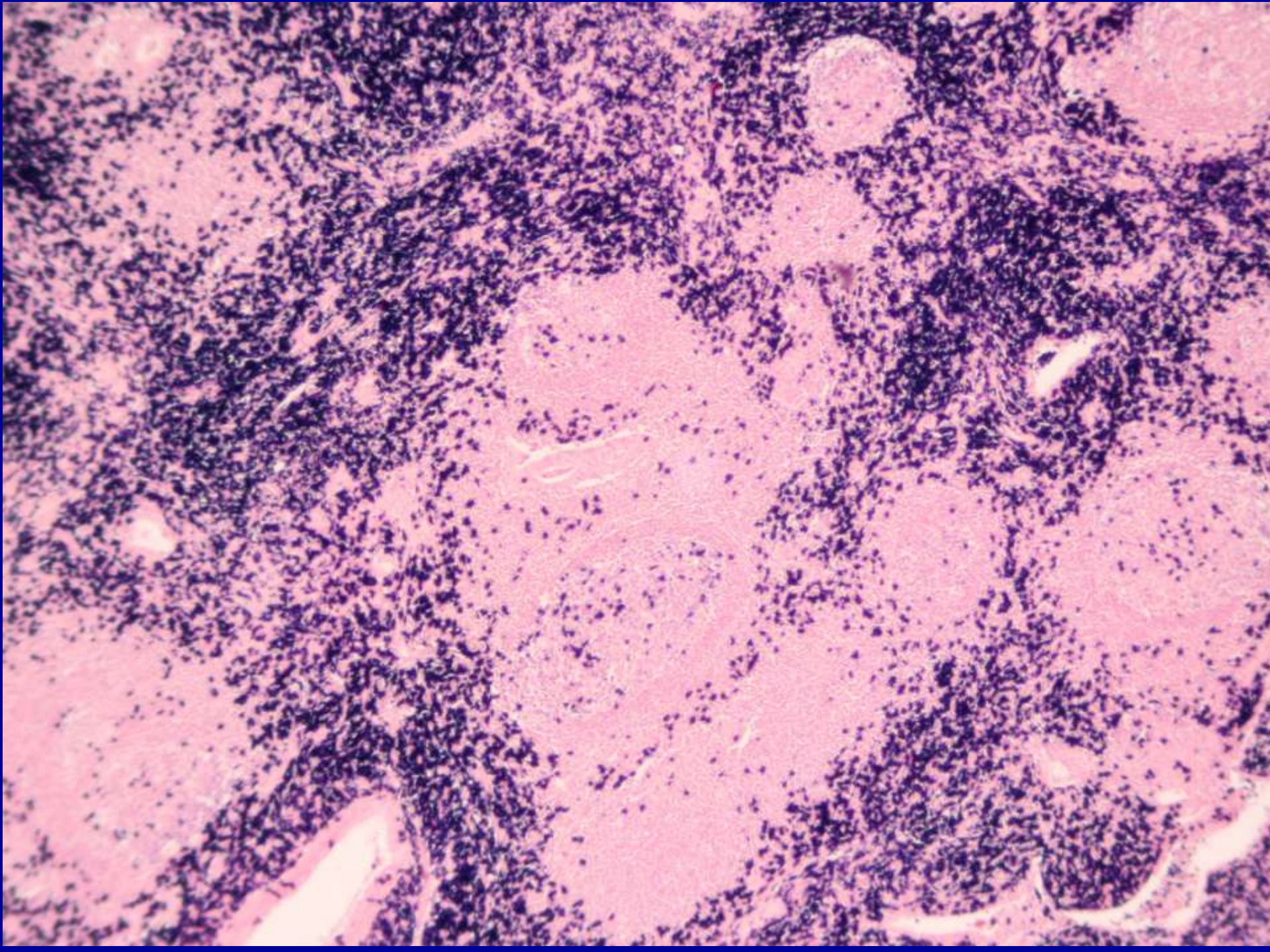


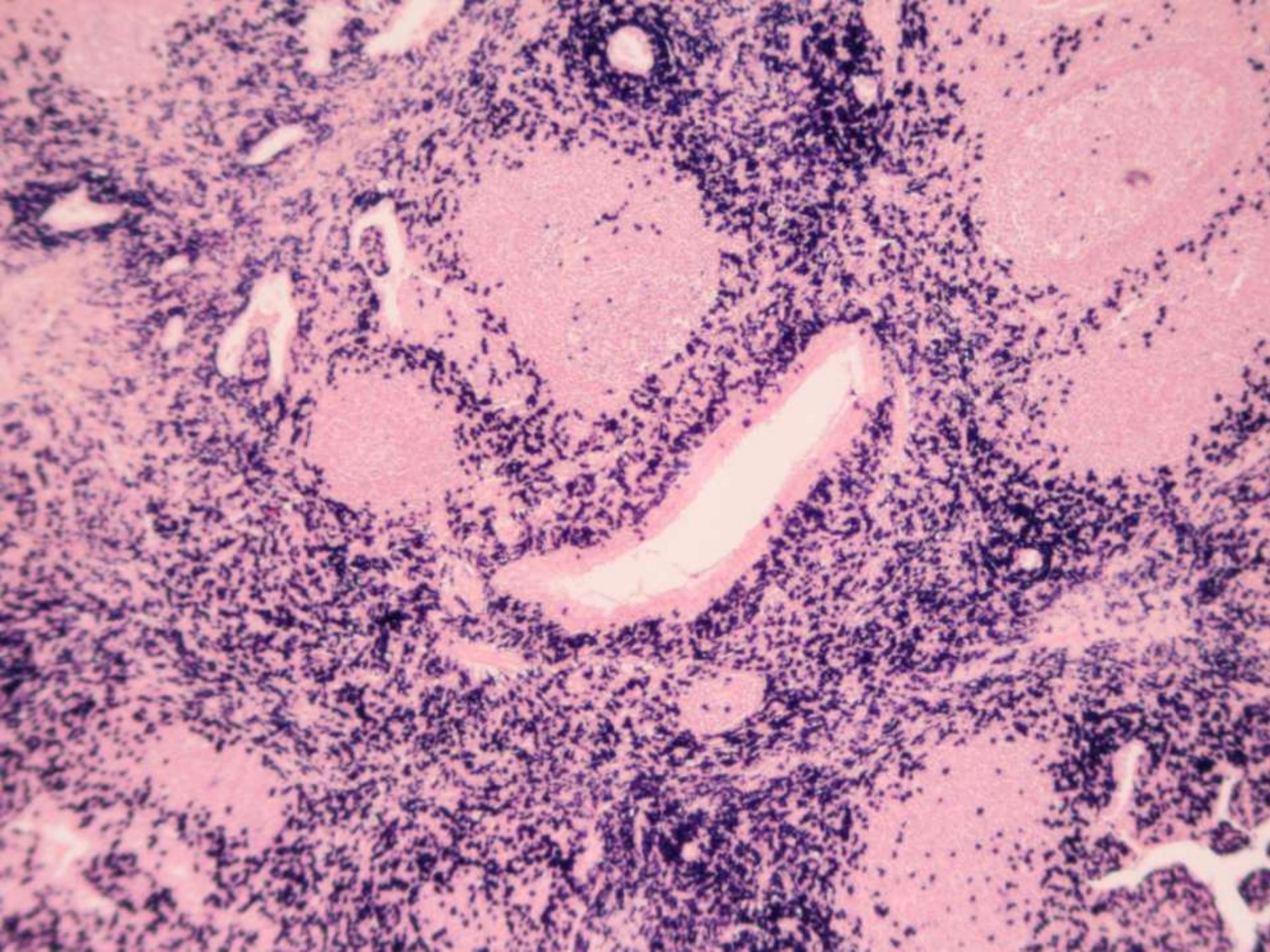


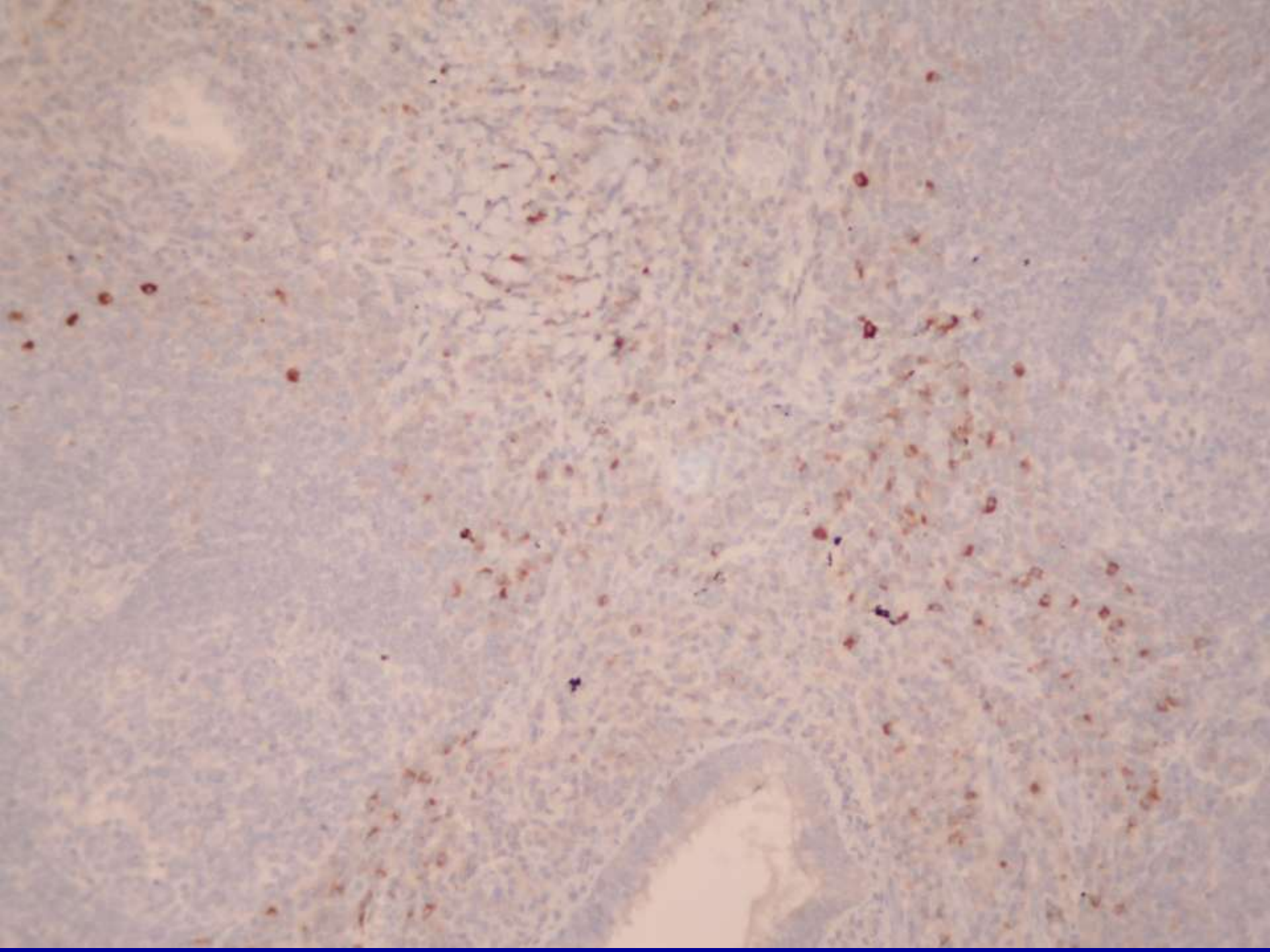












DIAGNOSIS ????

1. HP
2. LIP
3. Generalisation of cancer
4. Plasmacytoma
5. Lymphoma
6. IgG4 related disease

Nodular lymphoid hyperplasia with increasednumber of plasma cells and some hyaline sclerosis. Increased number of IgG4+ plasmocytes raising concern of IgG4 systemic sclerosing disease

Seminars in Diagnostic Pathology (2012) 29, 219-225

ELSEVIER

Seminars in
Diagnostic
Pathology

Pathologic manifestations of Immunoglobulin(Ig)G4-related lung disease

Eunhee S. Yi, MD,[a] Hiroshi Sekiguchi, MD,[b] Tobias Peikert, MD,[b] Jay H. Ryu, MD,[b] Thomas V. Colby, MD[c]

From the [a]Department of Laboratory Medicine and Pathology, Mayo Clinic, Rochester, Minnesota; [b]Division of Pulmonary and Critical Care Medicine, Mayo Clinic, Rochester, Minnesota; and the [c]Department of Laboratory Medicine and Pathology, Mayo Clinic, Scottsdale, Arizona.

Table 1 Other conditions with increased IgG4+ cells (>10/high-power field) in the lung tissue

Case description	IgG/hpf	IgG4/hpf	%IgG4
Idiopathic NSIP	72	19	26
Idiopathic NSIP	35	17	49
Necrotizing granulomatous inflammation	138	69	50
UIP	38	11	29
Rheumatoid arthritis NSIP/ pleuritis	69	20	29
Rheumatoid arthritis UIP	54	23	43
Rheumatoid arthritis nodules	90	13	14
SLE with diffuse alveolar damage	66	47	71
IMT (inflammatory fibrosarcoma)	170	99	58
IMT (plasma cell granuloma)	97	41	42
IMT (plasma cell granuloma)	154	33	21
IMT (plasma cell granuloma)	87	27	31

Modified from Table 6 in ref. 4.

Each row indicates an individual patient.

hpf, high-power field; IMT, inflammatory myofibroblastic tumor; NSIP, nonspecific interstitial pneumonia; SLE, systemic lupus erythematosus; UIP, usual interstitial pneumonia.

MODERN PATHOLOGY (2012) 25, 1181–1192

Consensus statement on the pathology of IgG4-related disease

Vikram Deshpande[1,31], Yoh Zen[2,31], John KC Chan[3], Eunhee E Yi[4], Yasuharu Sato[5], Tadashi Yoshino[5], Günter Klöppel[6], J Godfrey Heathcote[7], Arezou Khosroshahi[8], Judith A Ferry[1], Rob C Aalberse[9], Donald B Bloch[8], William R Brugge[10], Adrian C Bateman[11], Mollie N Carruthers[8], Suresh T Chari[12], Wah Cheuk[3], Lynn D Cornell[13], Carlos Fernandez-Del Castillo[14], David G Forcione[10], Daniel L Hamilos[15], Terumi Kamisawa[16], Satomi Kasashima[17], Shigeyuki Kawa[18], Mitsuhiro Kawano[19], Gregory Y Lauwers[1], Yasufumi Masaki[20], Yasuni Nakanuma[21], Kenji Notohara[22], Kazuichi Okazaki[23], Ji Kon Ryu[24], Takako Saeki[25], Dushyant V Sahani[26], Thomas C Smyrk[13], James R Stone[1], Masayuki Takahira[27], George J Webster[28], Motohisa Yamamoto[29], Giuseppe Zamboni[30], Hisanori Umehara[20] and John H Stone[8]

Table 1 Histopathology of IgG4-related disease: variability of findings in certain organs

	Inflammation	*Fibrosis*	*Phlebitis*	*Others*
Lacrimal gland	No unique features	Typical storiform fibrosis is relatively uncommon. More often collagenous fibrosis	Sometimes lacks obliterative phlebitis	
Salivary gland	Often associated with conspicuous lymphoid follicle formation	Storiform fibrosis is rare in parotid and minor salivary glands	Sometimes lacks obliterative phlebitis	
Lymph node	No unique features	Fibrosis is only seen in inflammatory pseudotumor-like lesions	Most often lacks obliterative phlebitis	Five histological patterns are recognized: (1) multicentric Castleman's disease-like, (2) follicular hyperplasia, (3) interfollicular expansion, (4) progressive transformation of germinal center, and (5) nodal inflammatory pseudotumor-like. The specificity of these histologic changes in the absence of other evidence of IgG4-RD remains controversial
Lung	Small aggregates of neutrophils may be present in alveolar spaces or within the inflammatory infiltrates	Sometimes lacks storiform fibrosis, particularly in non-solid lesions (eg, interstitial pneumonia)	No unique features	Obliterative arteritis is often seen in pulmonary manifestations, particularly solid lesions
Kidney	No unique features	No unique features	Obliterative phlebitis is less common particularly in needle biopsies	

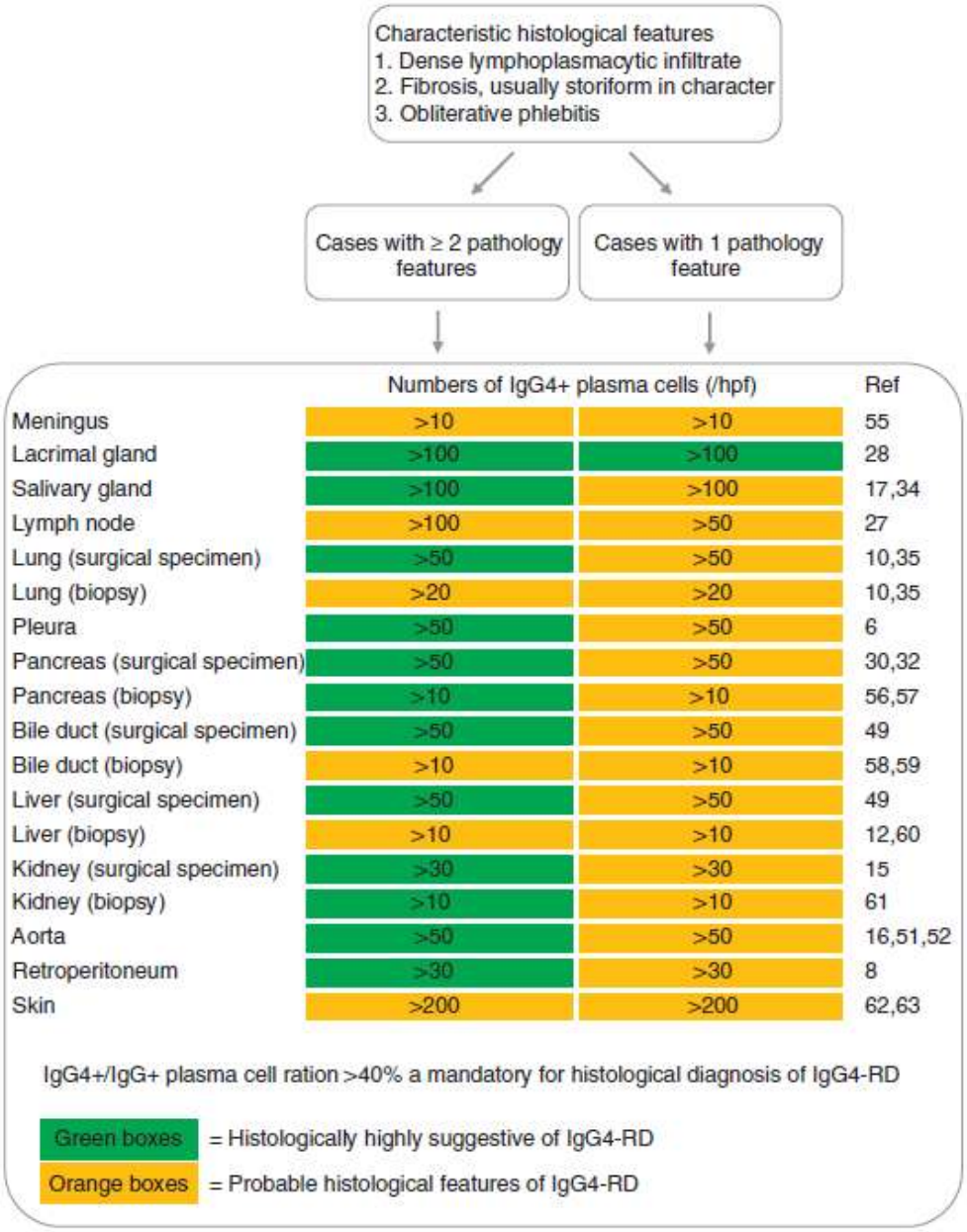


Figure 3 Histological diagnostic scheme of IgG4-related disease.[6,8,10,12,15–17,27,28,30,32,34,35,49,51,52,55–63]

INTERNATIONAL SYMPOSIUM
IgG4-RD & Associated Conditions

Symposium dates: February 16-19, 2014

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International Symposium 2014

The 2nd International Symposium on IgG4-Related Diseases & Associated Conditions will be held at the **Sheraton Waikiki Hotel** in Honolulu, Hawaii from **February 16-19, 2014**. This event will be hosted by a collaboration of American and Japanese investigators within this disease area.

Since the first International Symposium held in Boston in 2011, the field of IgG4-RD research and clinical care has expanded substantially. New publications about IgG4-RD and its associated conditions are now appearing on a daily basis.

The Second International Symposium will focus on advances in understanding of the pathophysiology of IgG4-RD and the impact of this new understanding on clinical care. We are expecting worldwide attendance from most sub-specialties including investigators and experts in the area of IgG4 and related disciplines.

REGISTER

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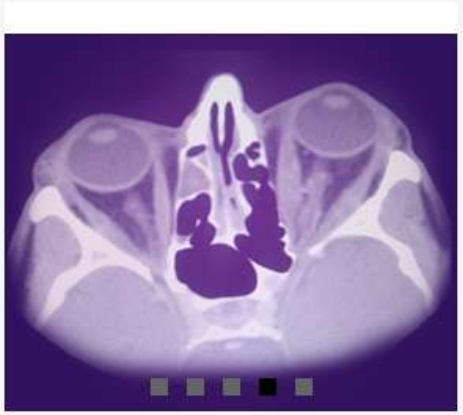
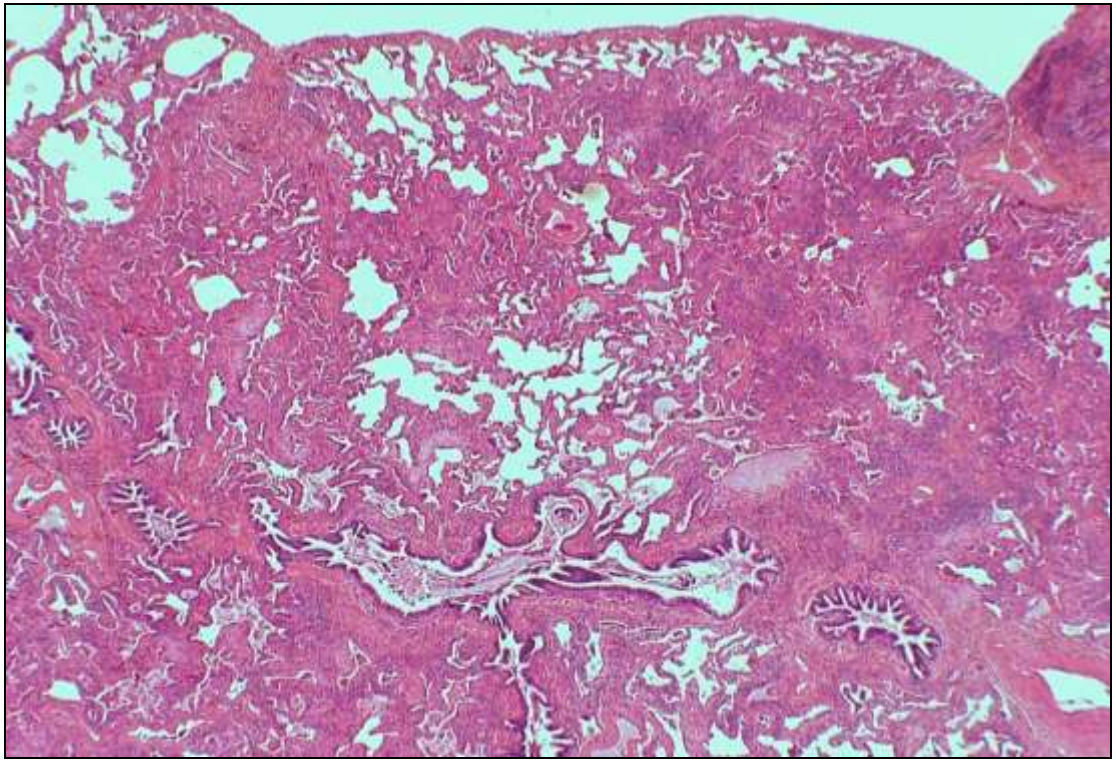
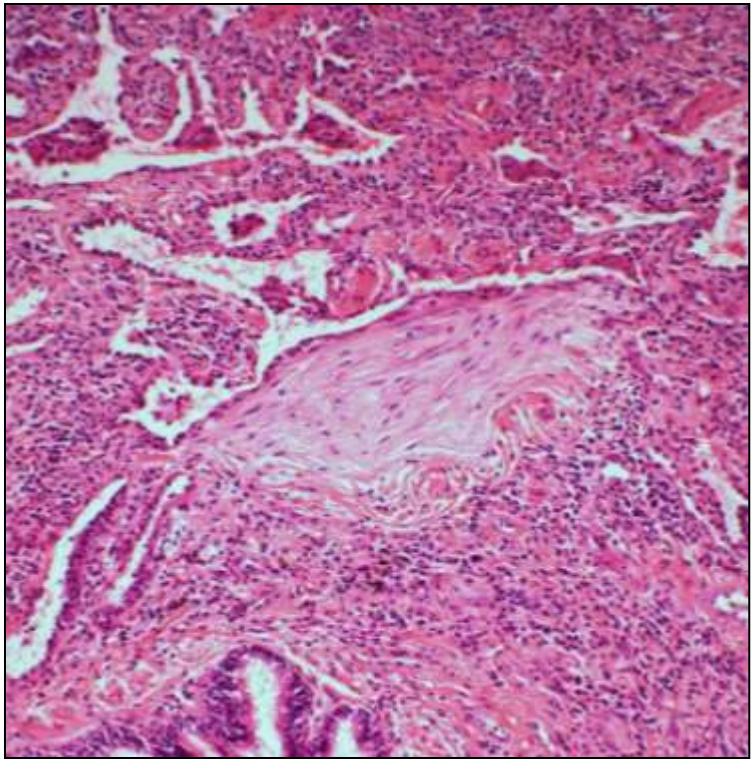


Case 7 (TV13-216)

TV13-216

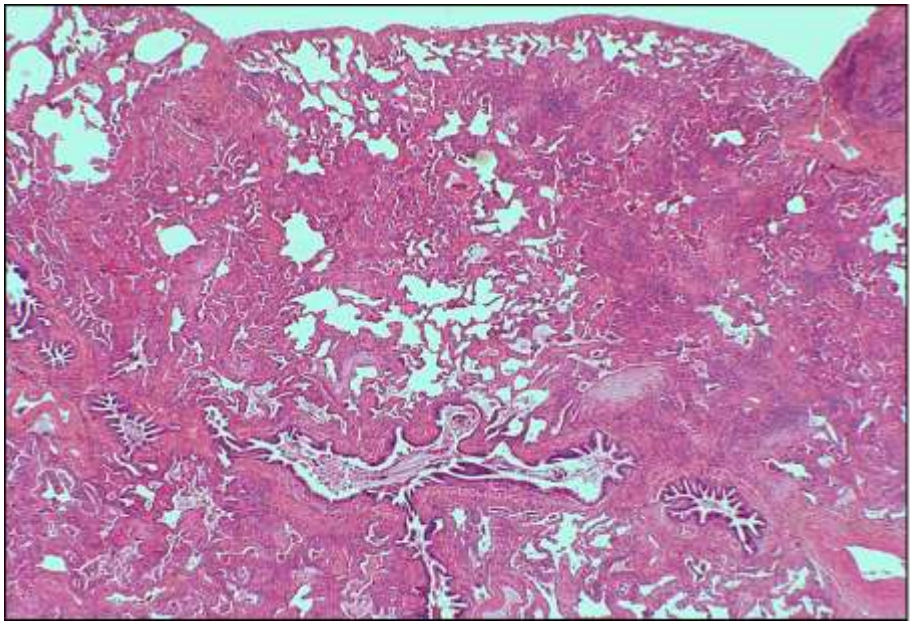
- Surgical lung biopsy for interstitial lung disease

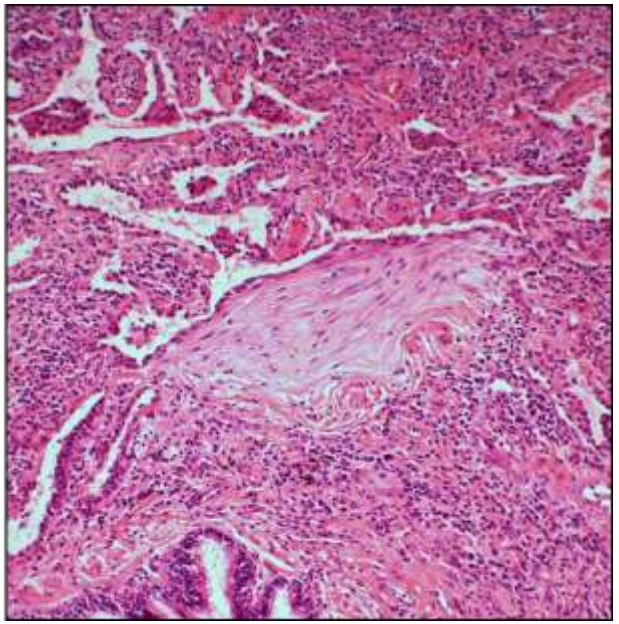




TV13-216 QUESTION 1

- Which of the following is true?
  1. It is patchy and shows subpleural predominance
  2. It is patchy and shows centrilobular distribution
  3. It is patchy and includes fibroblast foci
  4. Most of the changes are acute
  5. I have no idea!





Here is some history TV13-216

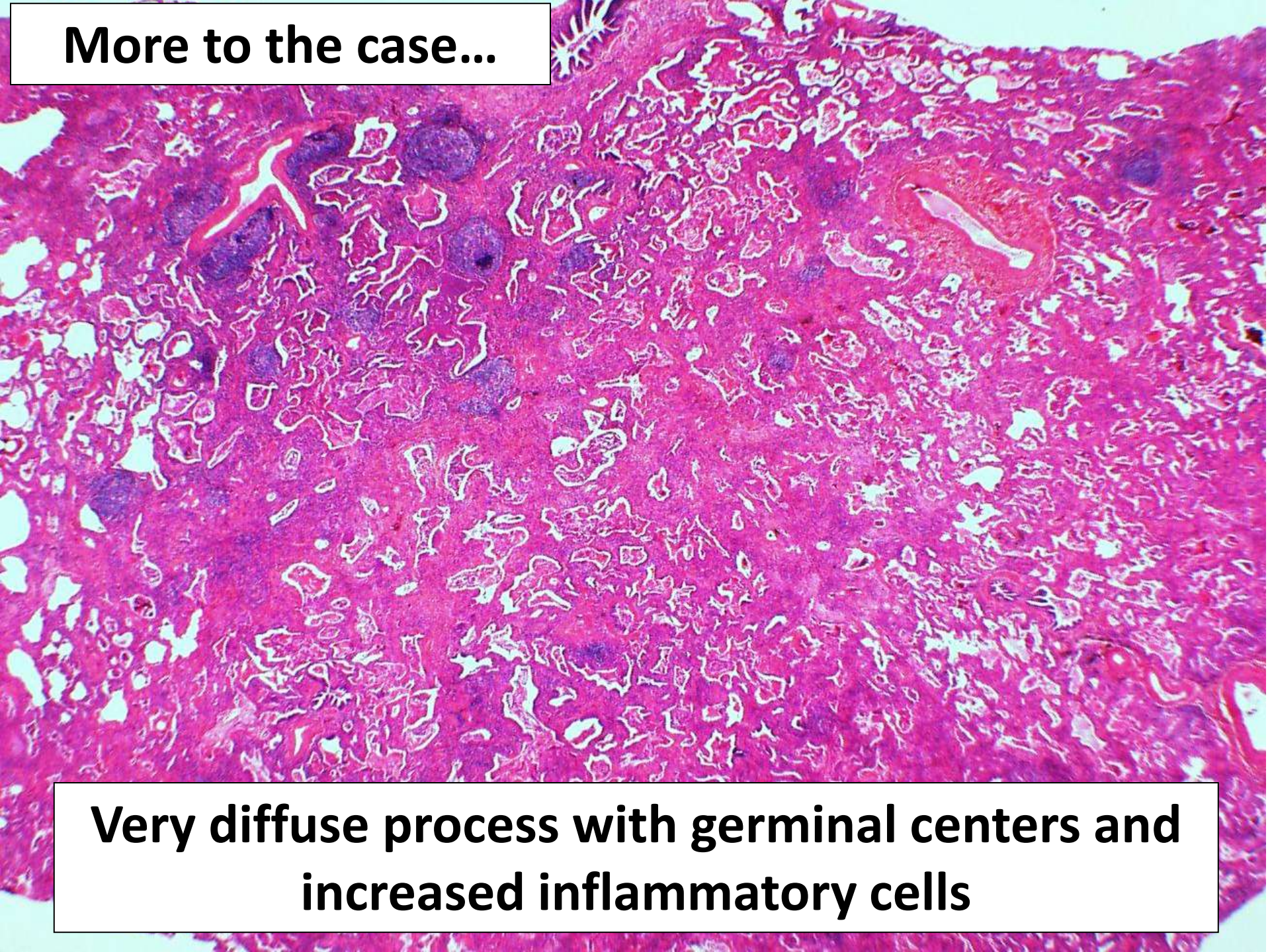
- 45 yr. old physician
- 3 month history of: cough, dyspnea, and fatigue

  ILD on CXR; No CT at that time
- BAL with 19% lymphs

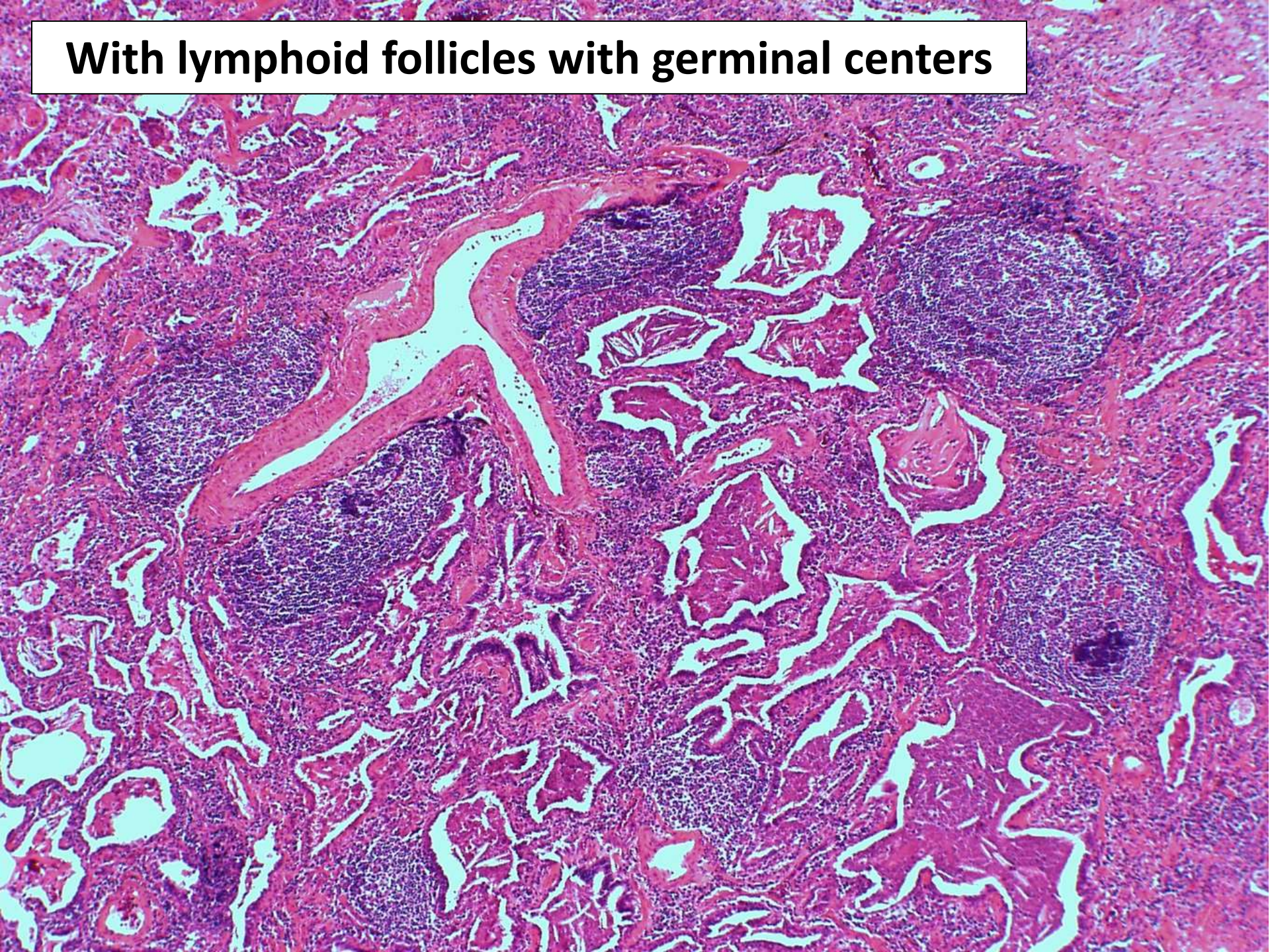
  Surgical lung biopsy performed: "Drs -- and Colby feel that this represents "diffuse interstitial pneumonitis with moderate fibrosis, so-called 'usual type" = UIP

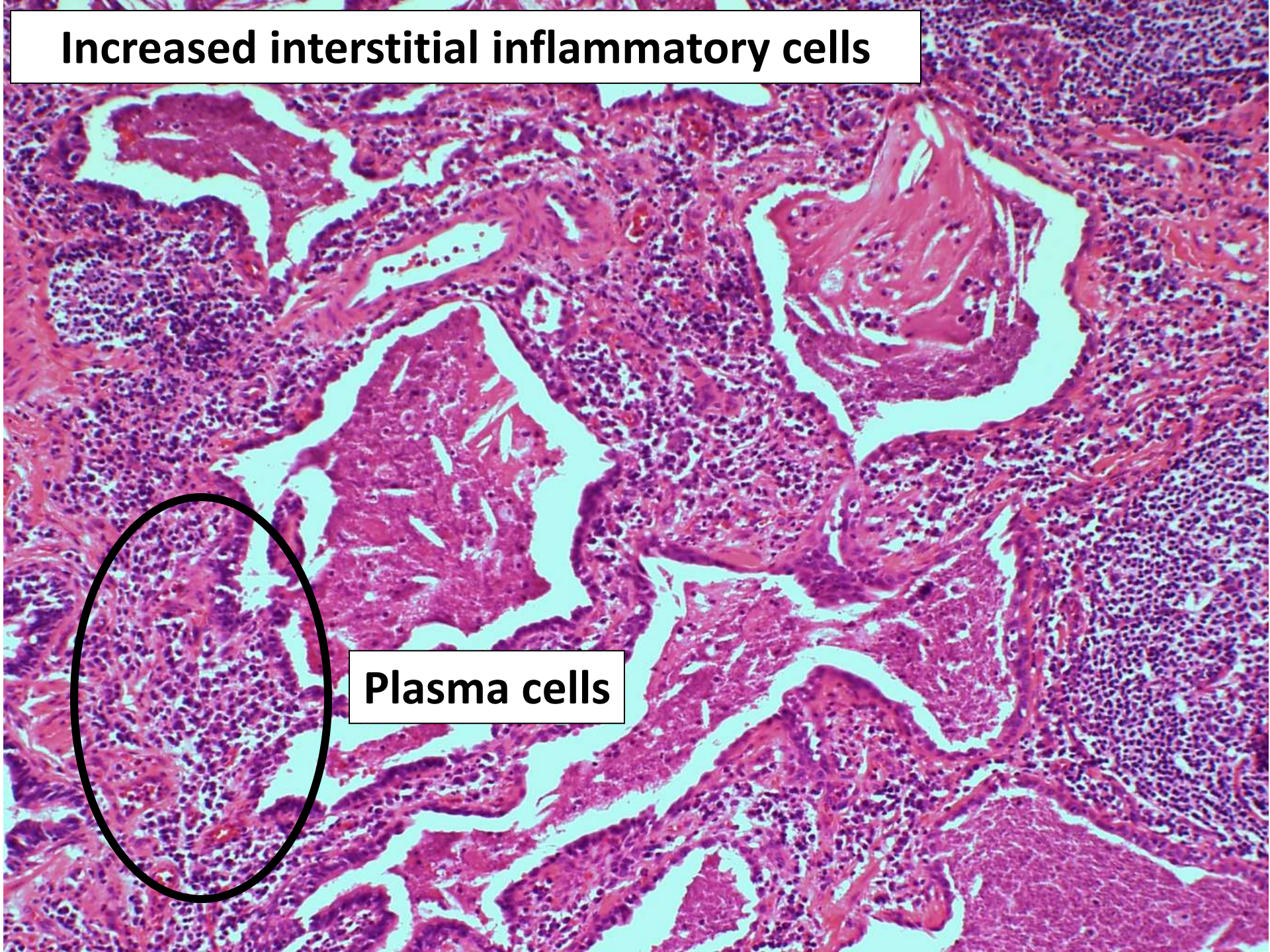
- **Atypical for UIP in IPF**

More to the case…

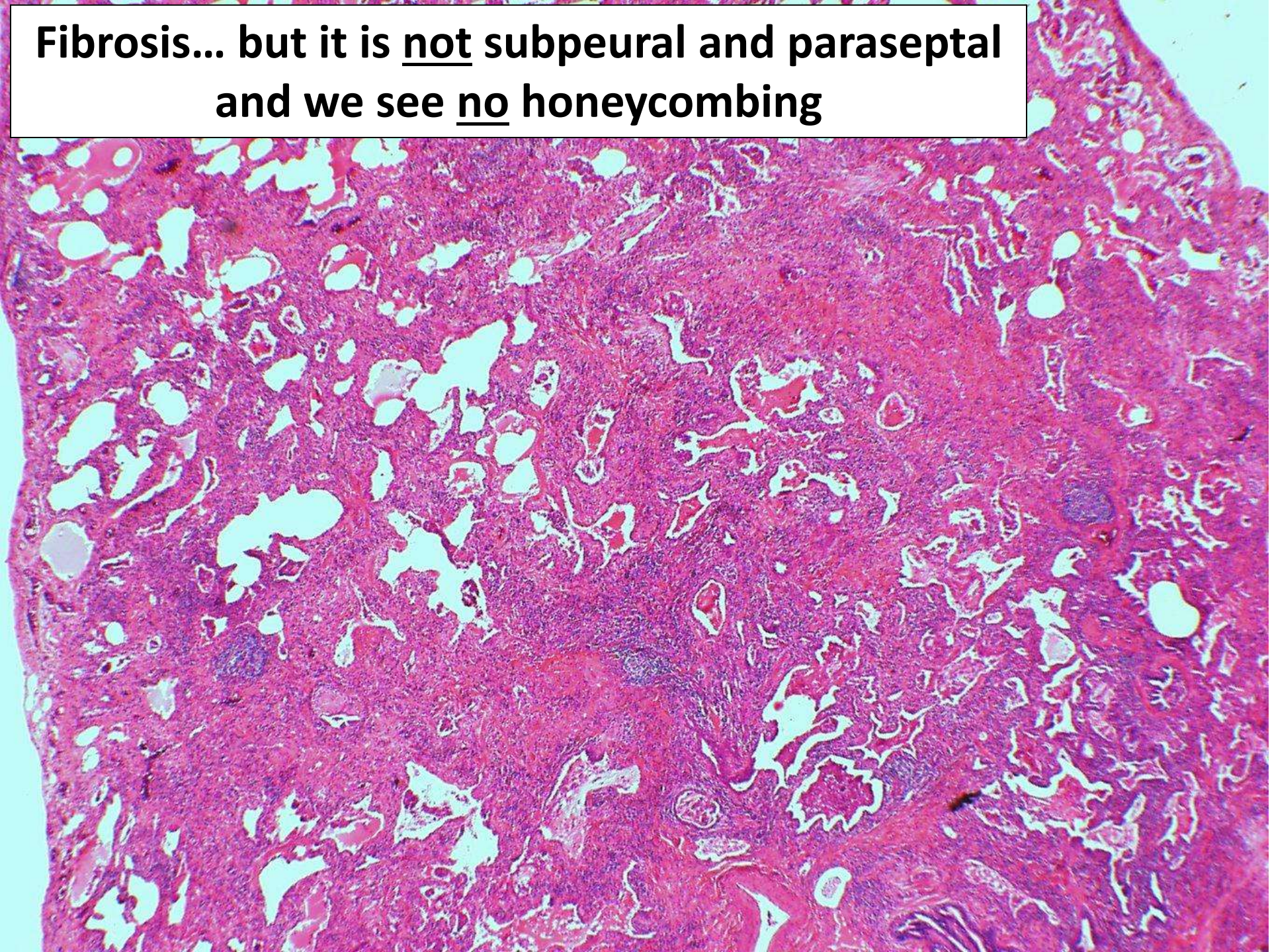


Very diffuse process with germinal centers and increased inflammatory cells

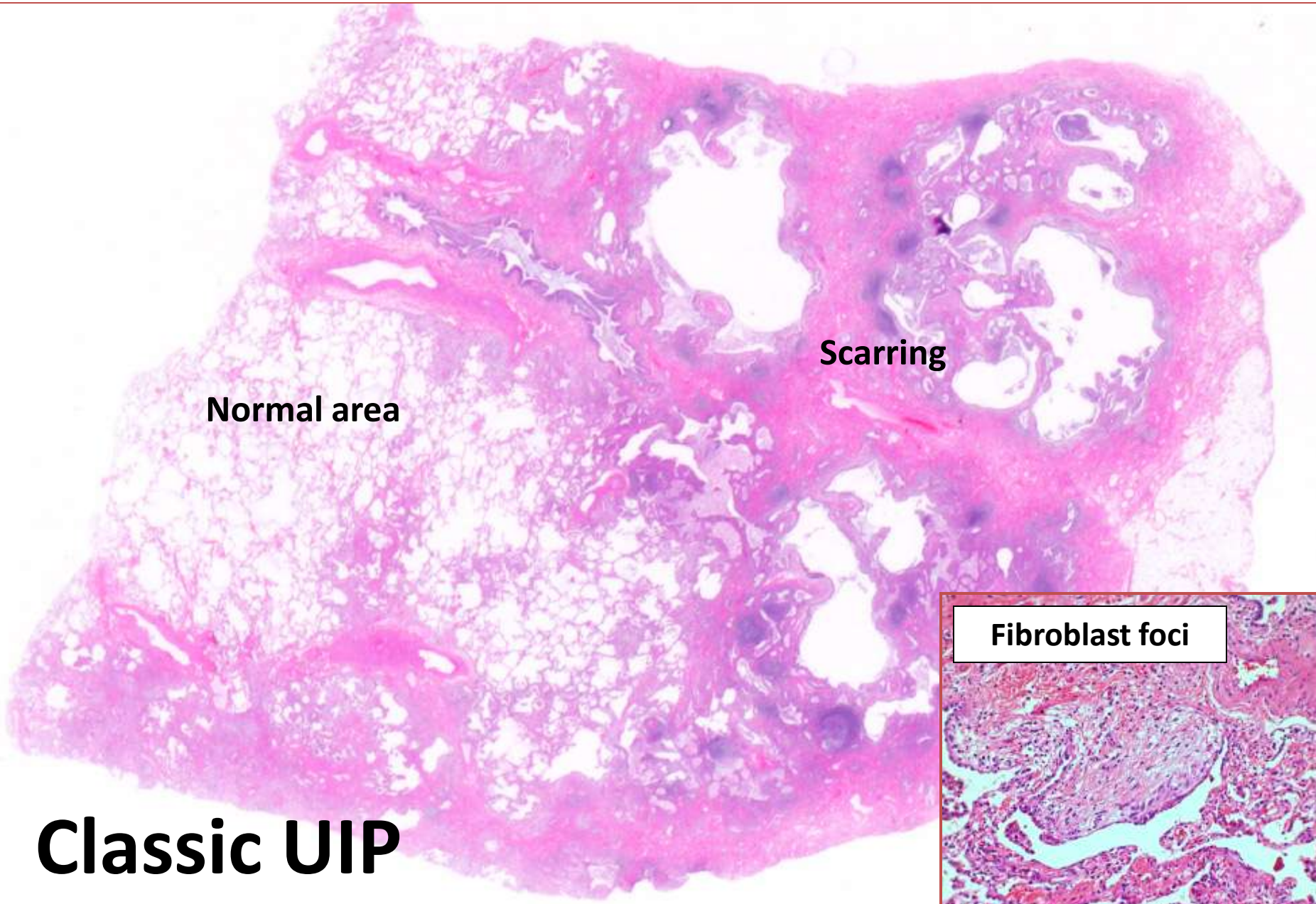

With lymphoid follicles with germinal centers


Increased interstitial inflammatory cells
Plasma cells

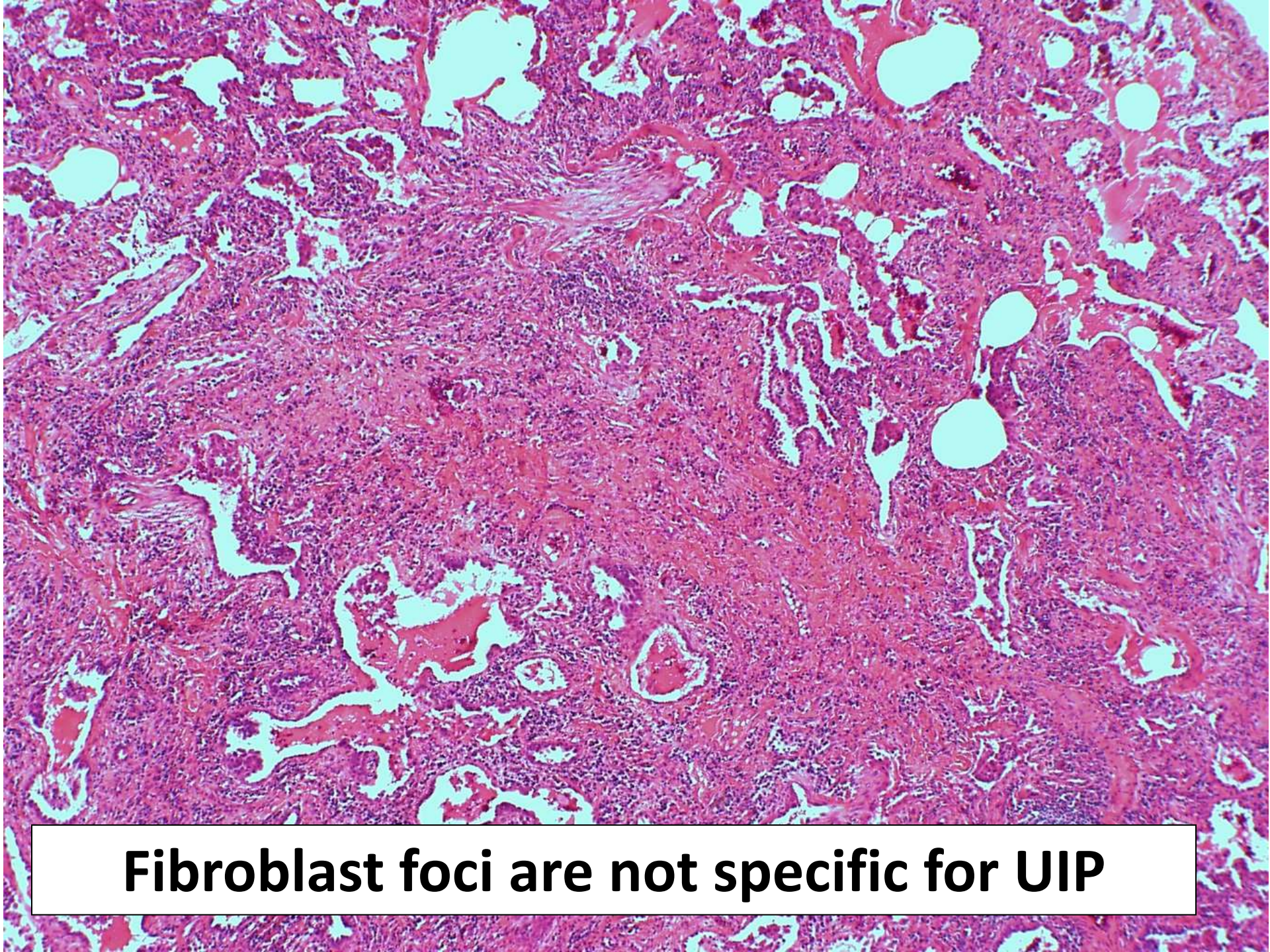
Fibrosis… but it is not subpeural and paraseptal and we see no honeycombing

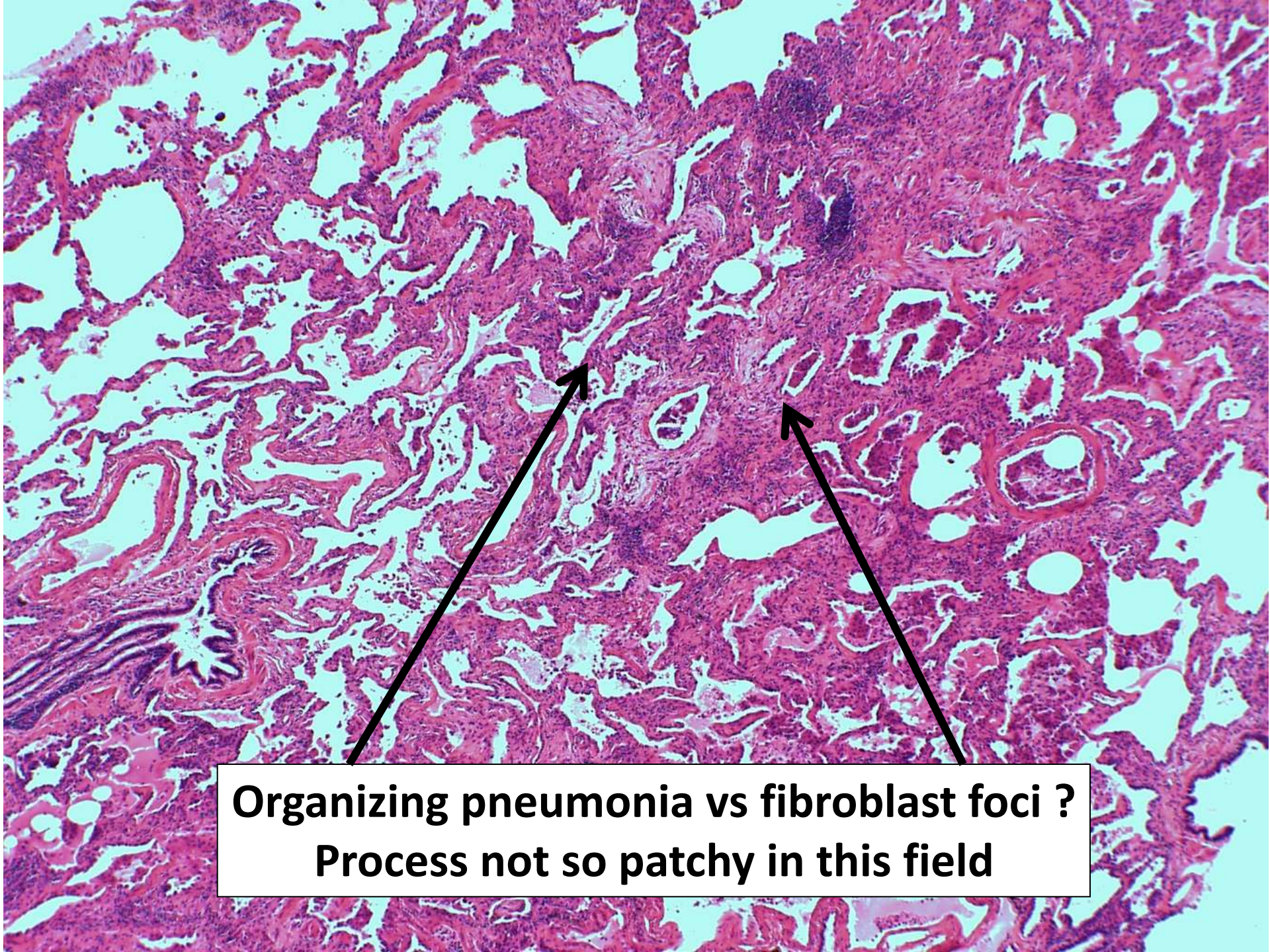


For comparison …



Classic UIP


Fibroblast foci are not specific for UIP


Organizing pneumonia vs fibroblast foci ?
Process not so patchy in this field

TV13-216 Follow-up

- Placed on steroids, 60 mg. per day
- 1 year later stable on 30 mg prednisone per day with PFTs “about 65% of normal”
- Alive and stable 22 yrs after biopsy; no details on functional or radiologic findings
- Retrospective diagnosis: Fibrotic NSIP, ? In association with CTD.

TV13-216 QUESTION 2

- Which diagnosis do you prefer ??
  (There is no correct answer)
  1. Fibrotic NSIP
  2. Fibrotic NSIP with features suggesting CTD
  3. Unclassifiable chronic interstitial pneumonia
  4. Fibrosing interstitial pneumonia with some features suggesting CTD
  5. Atypical UIP