SCE practice Qs

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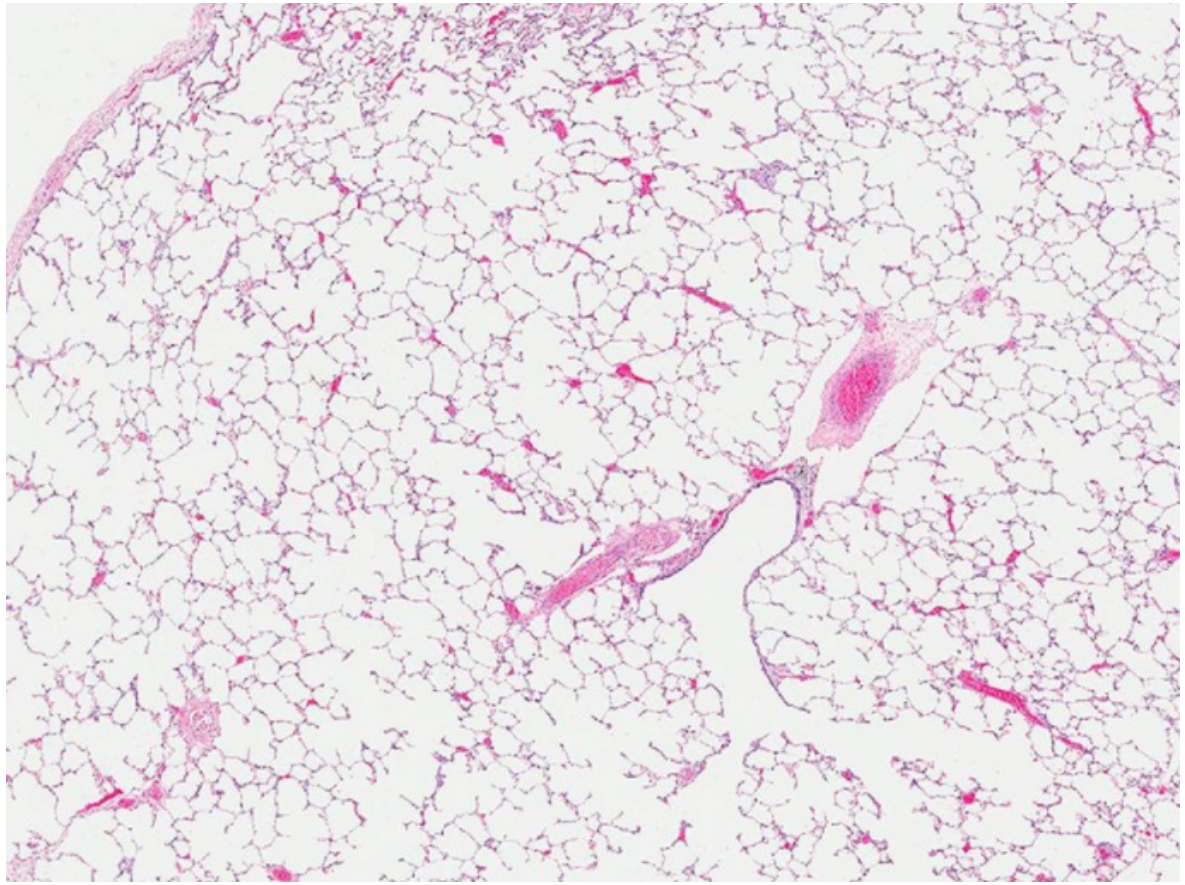
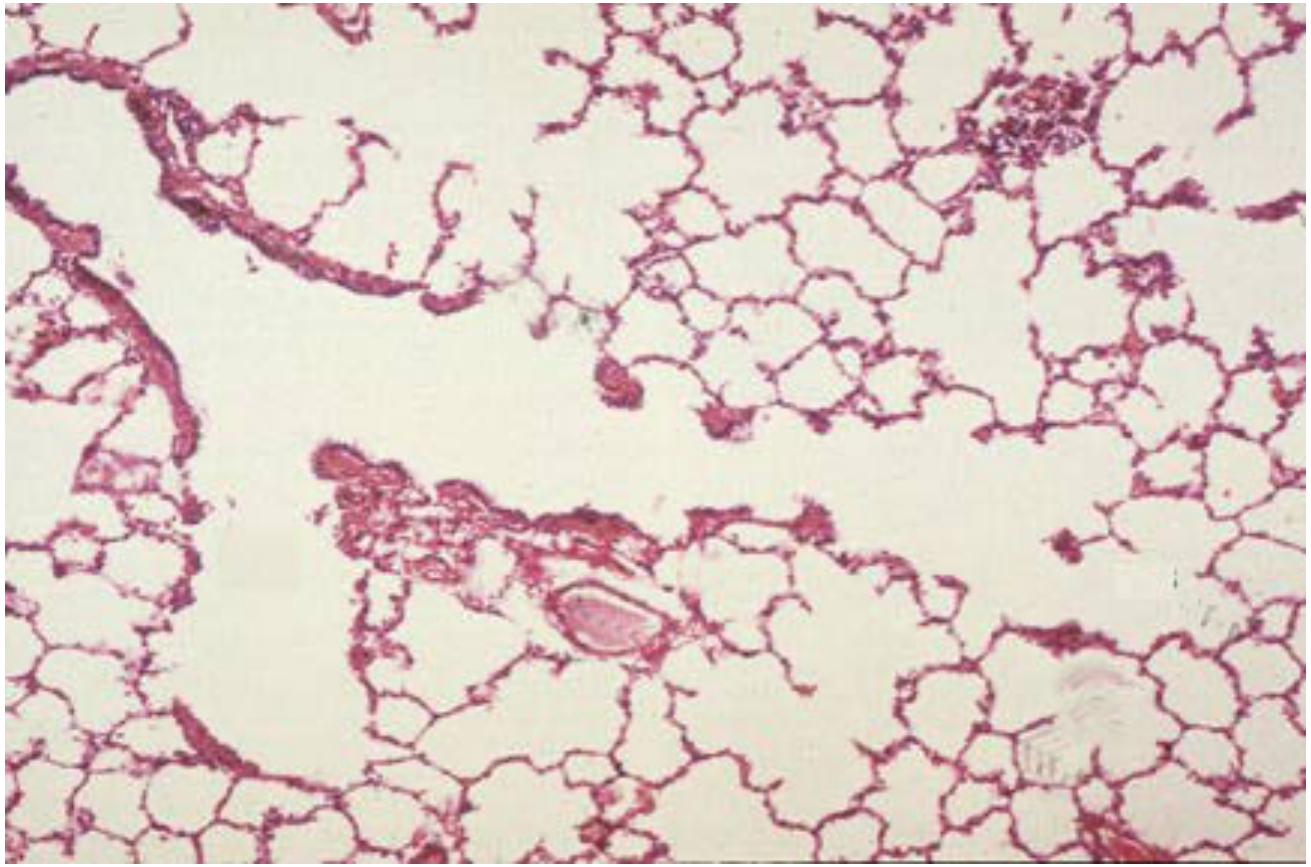
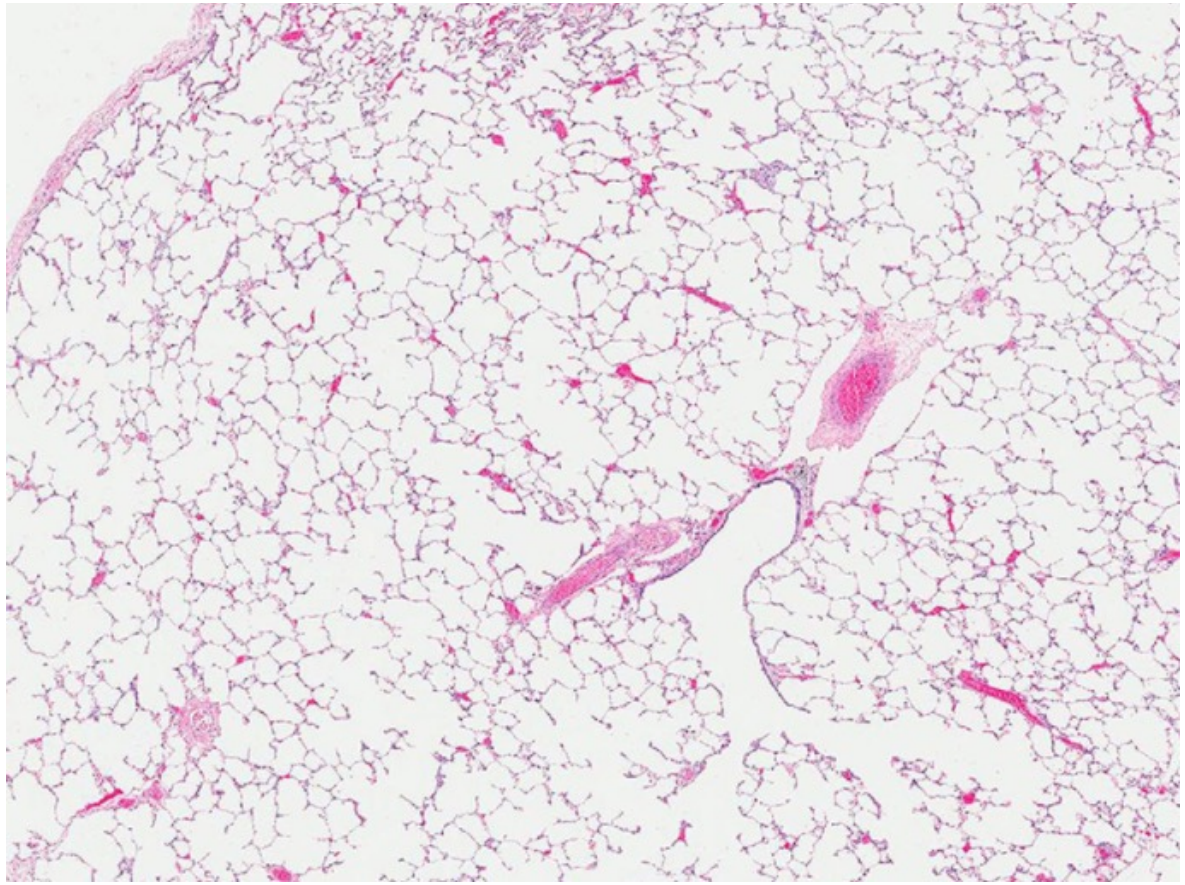
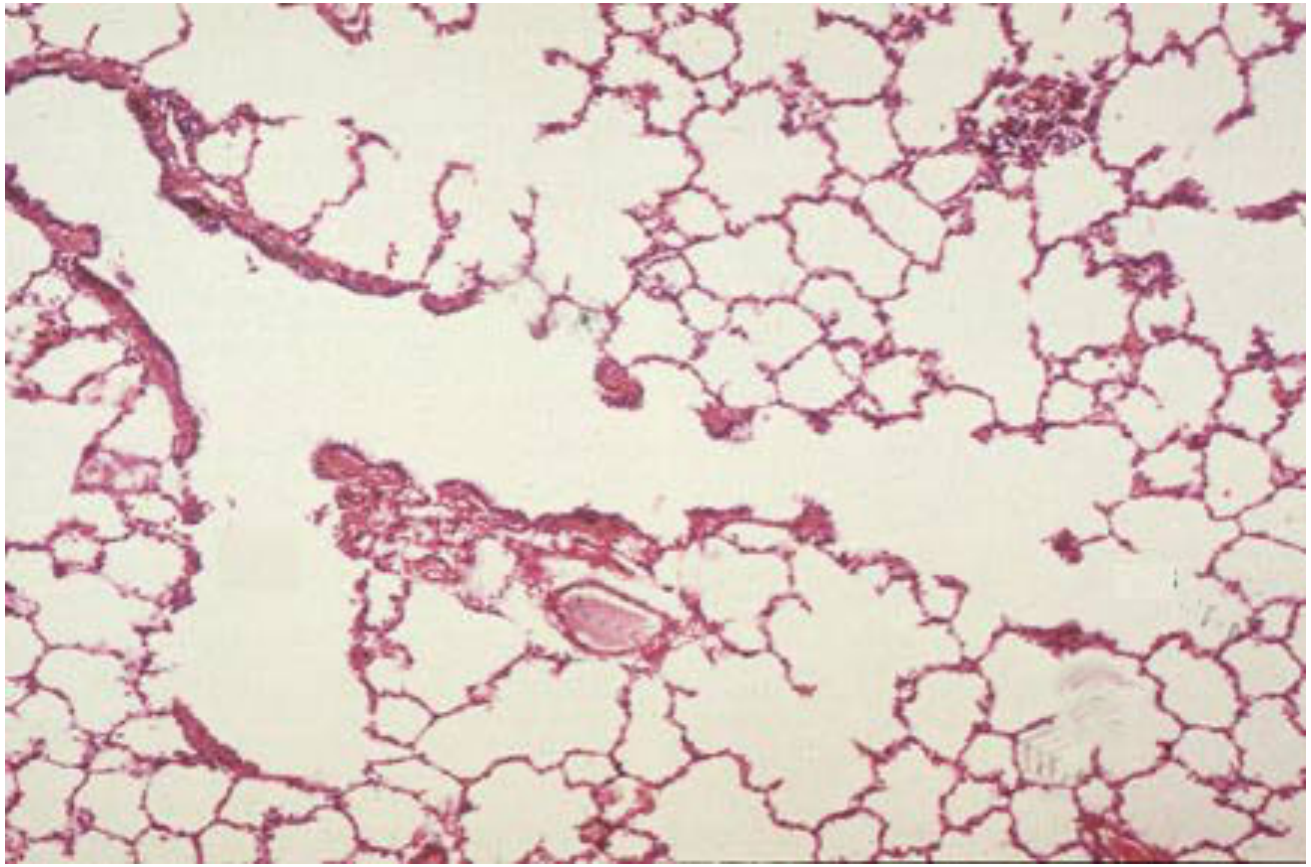
What am I?

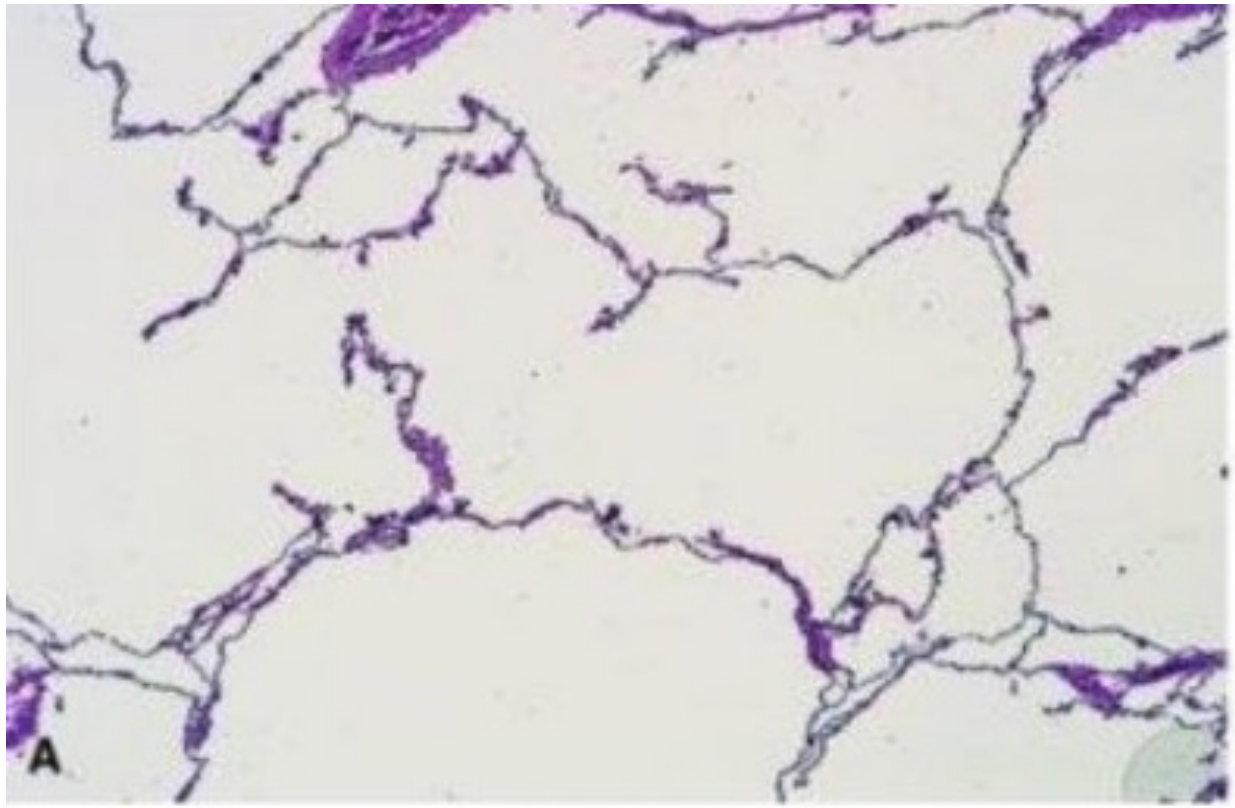




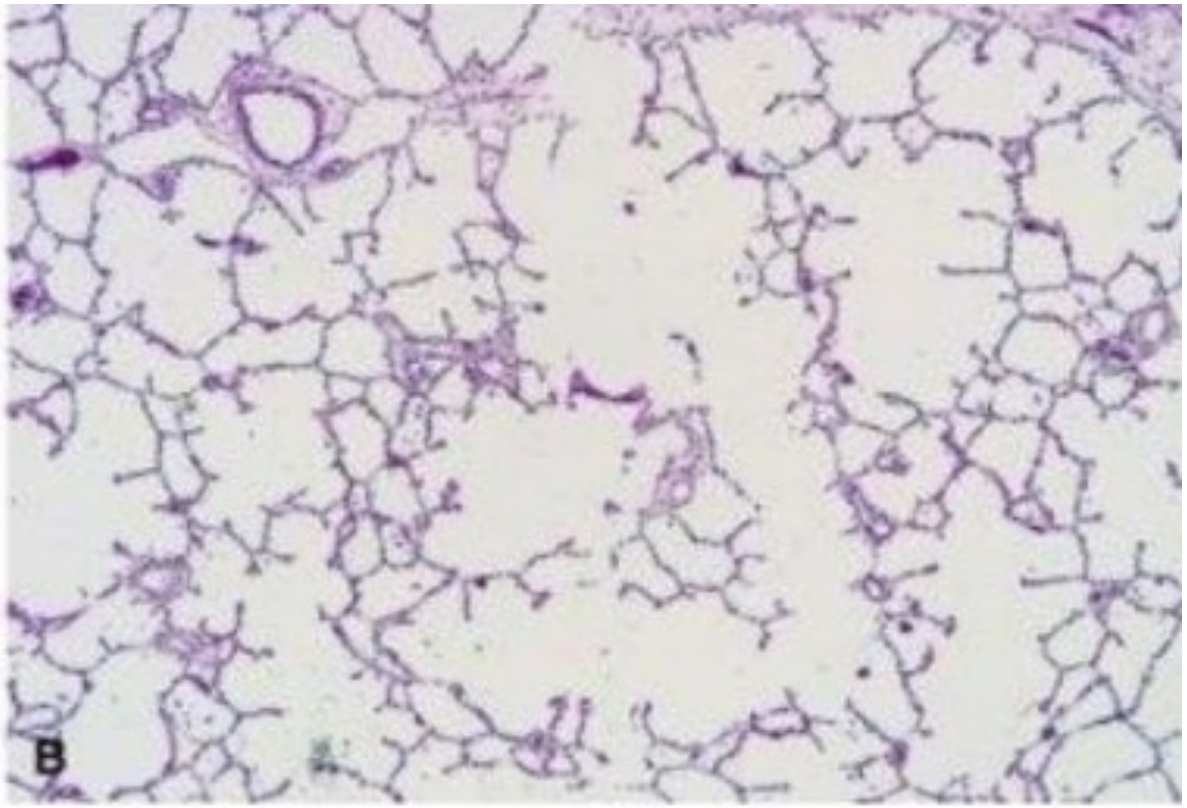
Normal lung



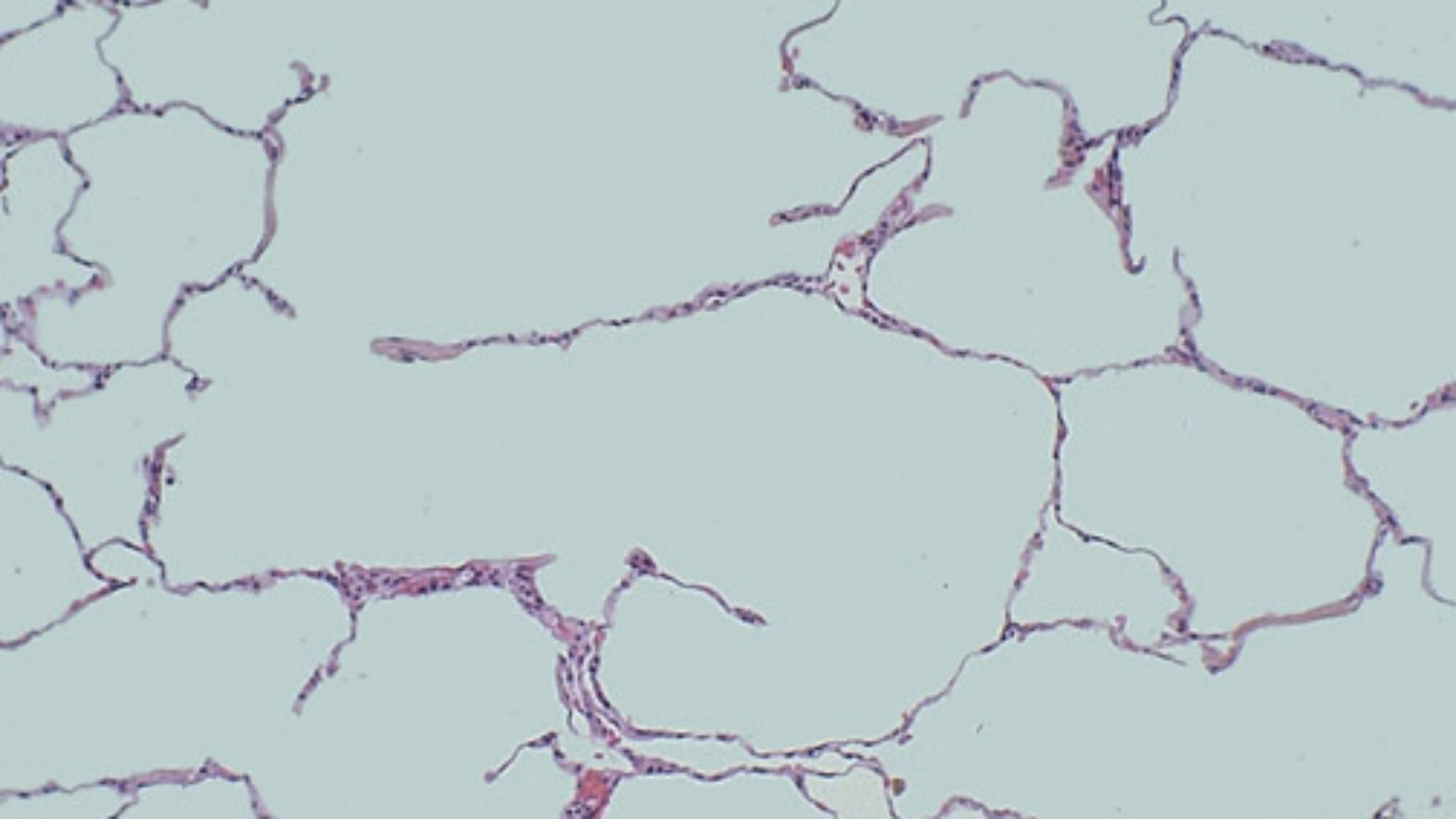




emphysema

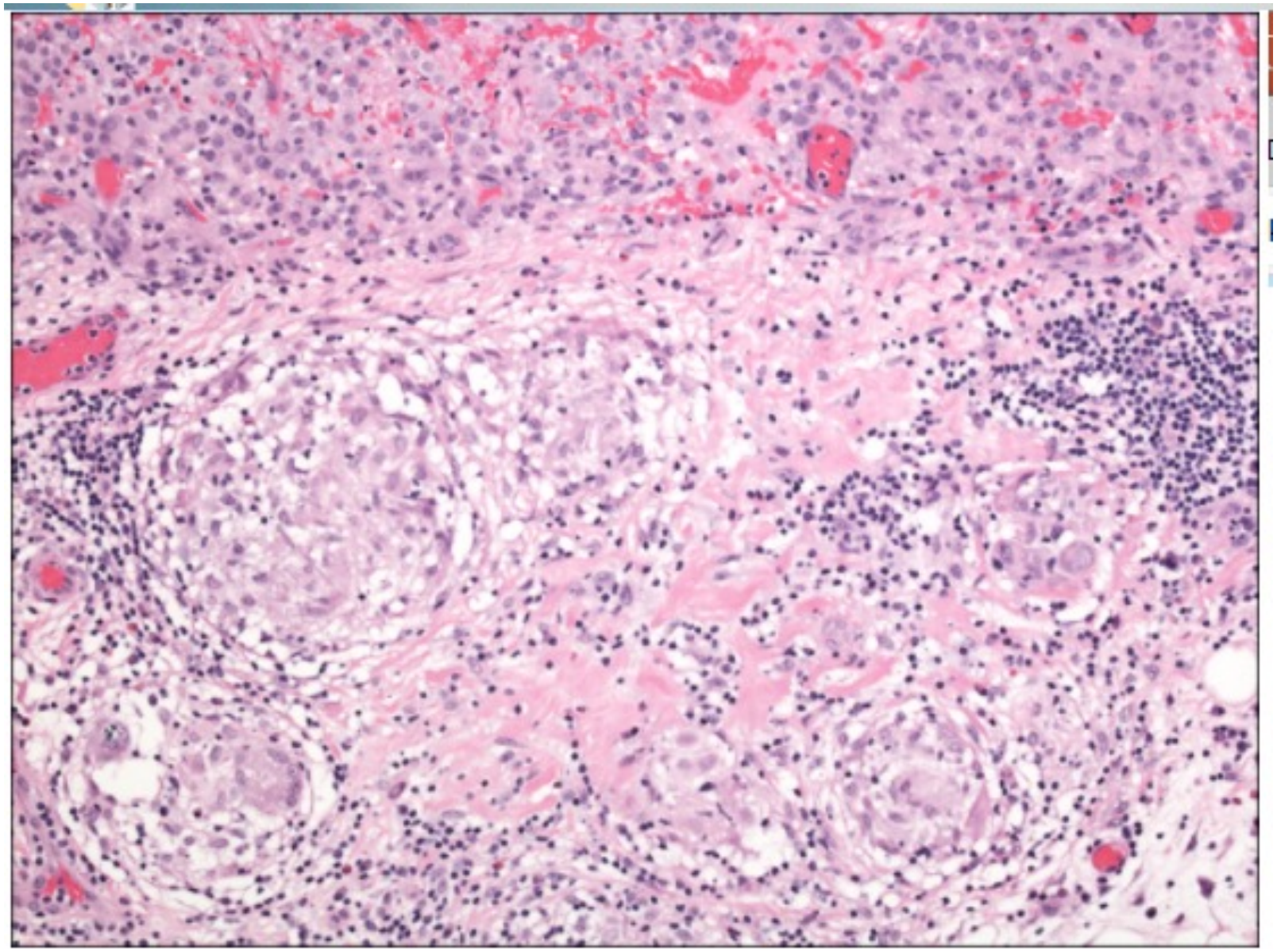


normal lung



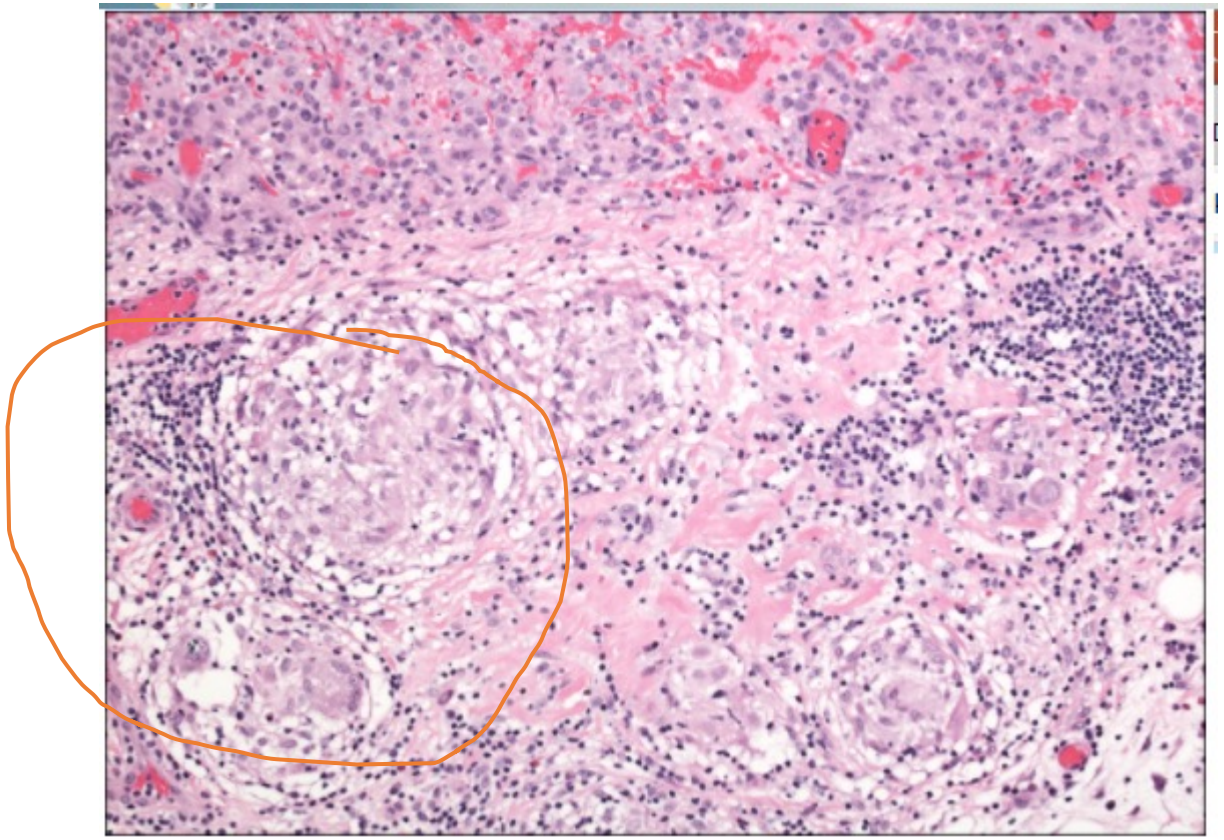
Spot histology

- What's this?



Non-caseating granulomata

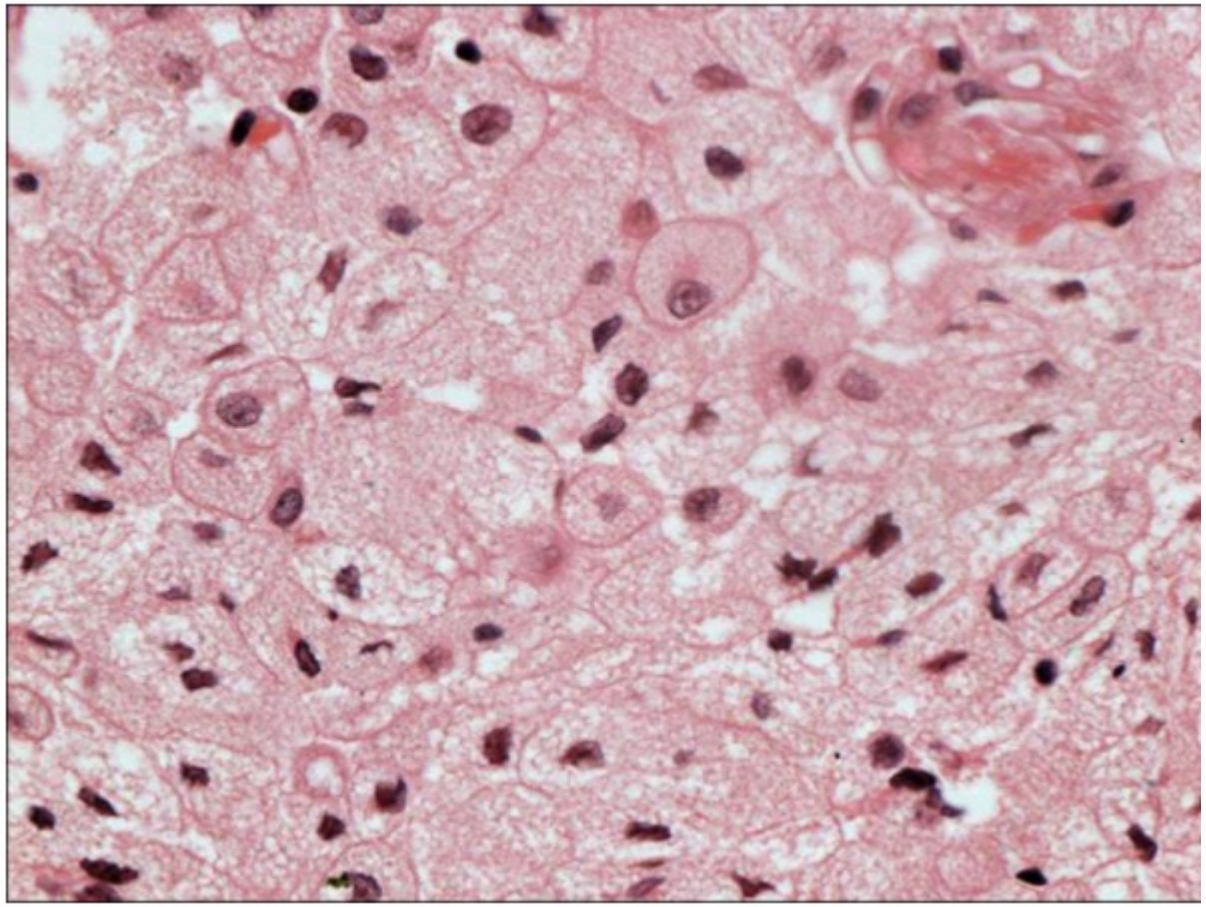
- This is a histology picture showing non-caseating granulomata compatible with sarcoidosis.



What am I? and what's therefore the diagnosis

Hint needed….?

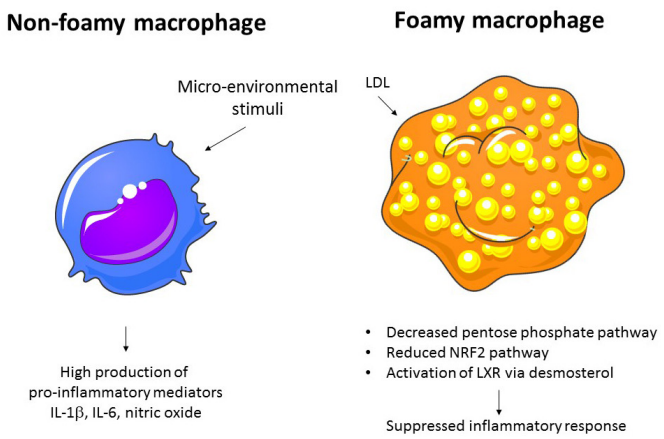
- Hint : Foamy macrophages

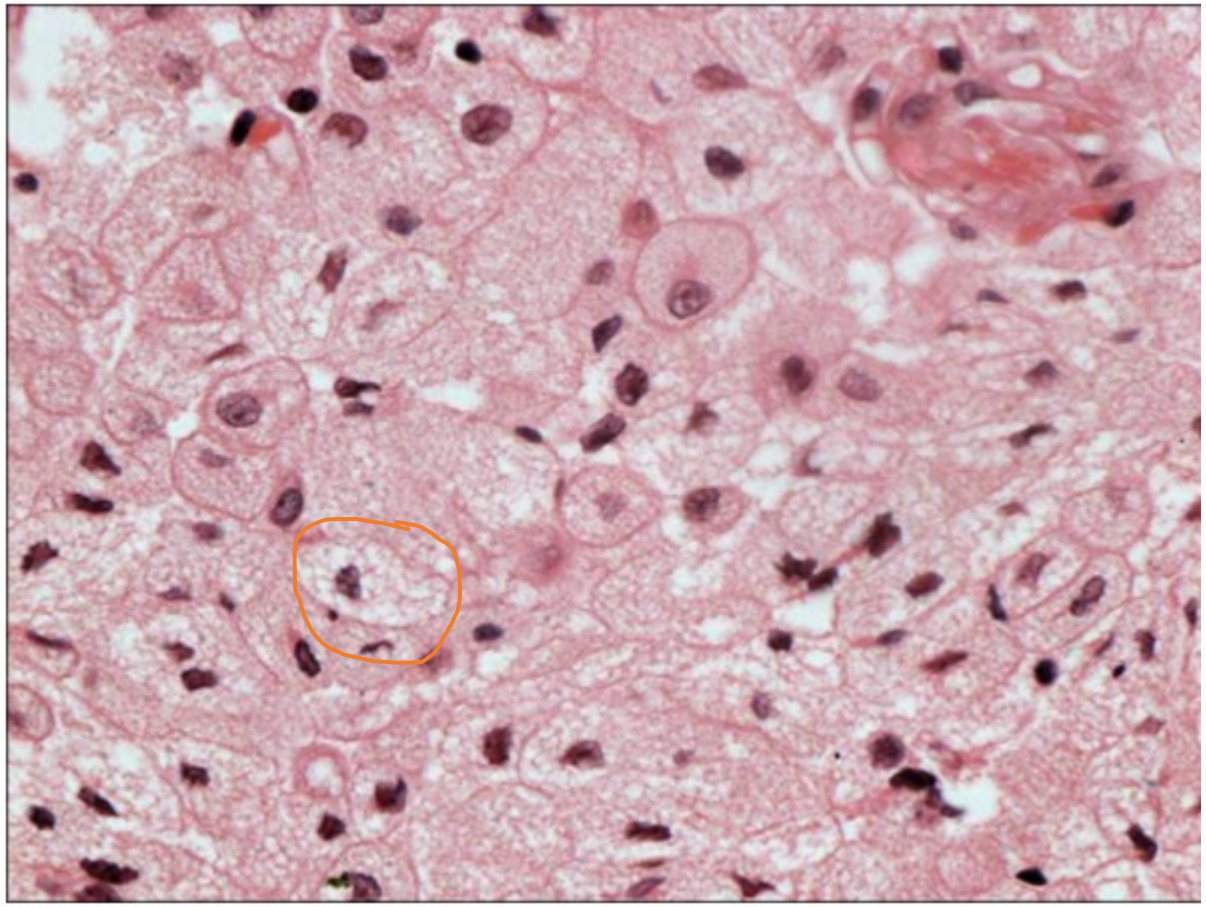


A 68-year-old man presented with progressive breathlessness and a dry cough. He had a 20-year history of well-controlled asthma. Eighteen months previously, he had developed atrial fibrillation, which was refractory to digoxin. He had been treated with amiodarone 400 mg daily for 9 months. His other medication included amlodipine 10 mg daily for hypertension, simvastatin 40 mg at night for hypercholesterolaemia, and warfarin. He had recently completed a course of clarithromycin. On examination, his respiratory rate was 30 breaths/min. There was no finger clubbing or splinter haemorrhages. Auscultation of the chest revealed bilateral crackles in the mid to lower zones. He had bilateral pitting ankle oedema.

Amiodarone pulmonary toxicity

- A characteristic finding is accumulation of amiodarone-phospholipid complexes, lipid-laden **'foamy' macrophages** in alveolar spaces Rx: steroids
- Amiodarone >400mg daily

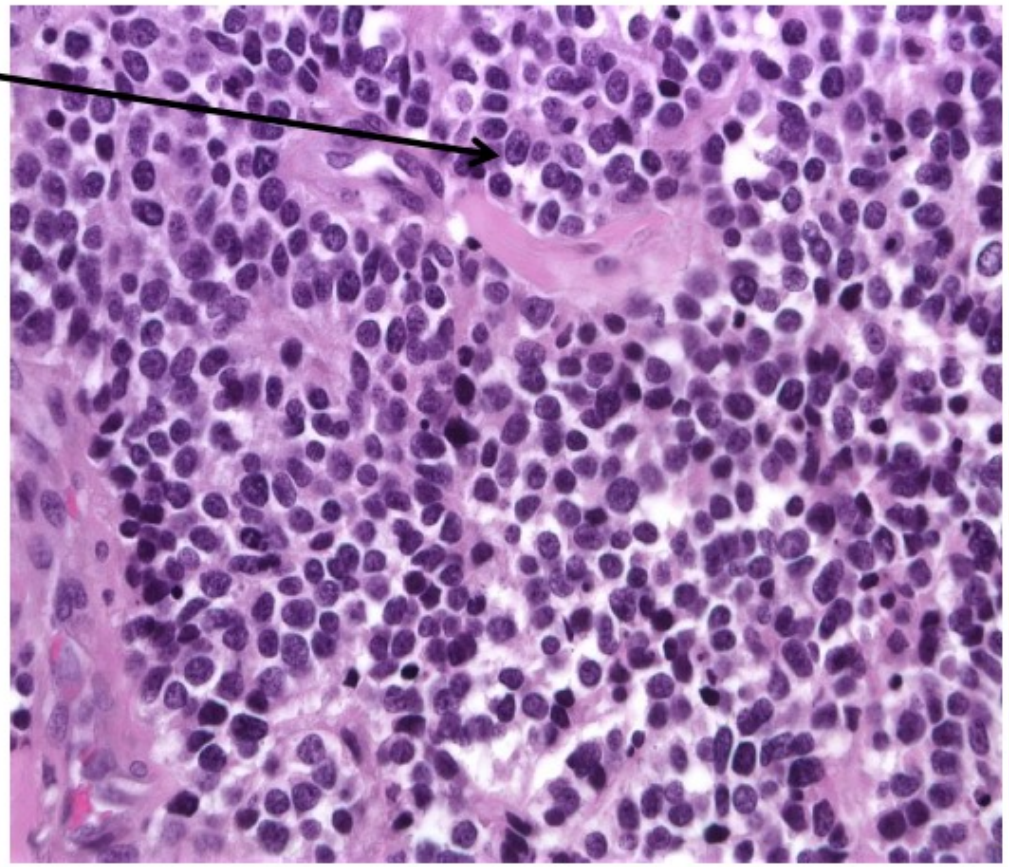


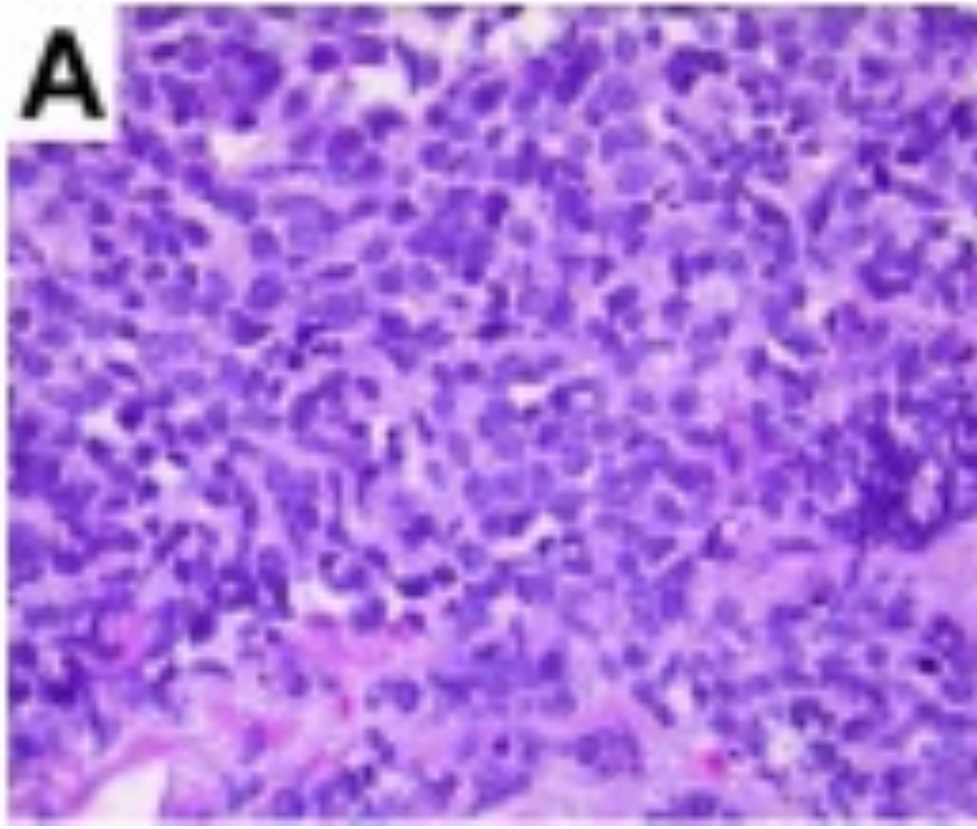


Q

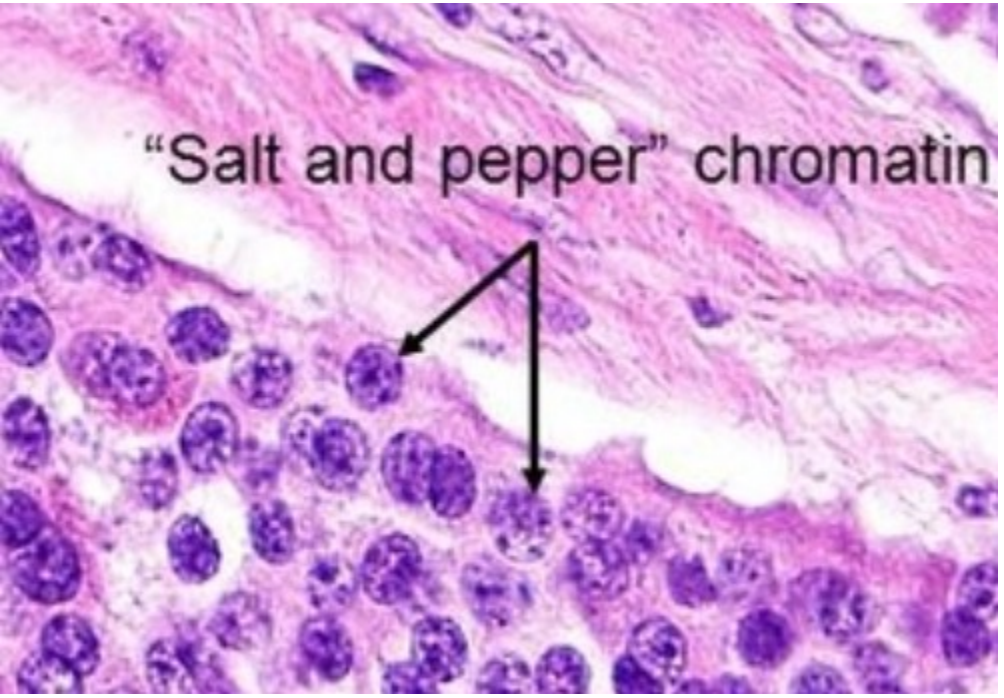
- Histology shows a mitotically active nuclei with salt and pepper chromatin – what am I?

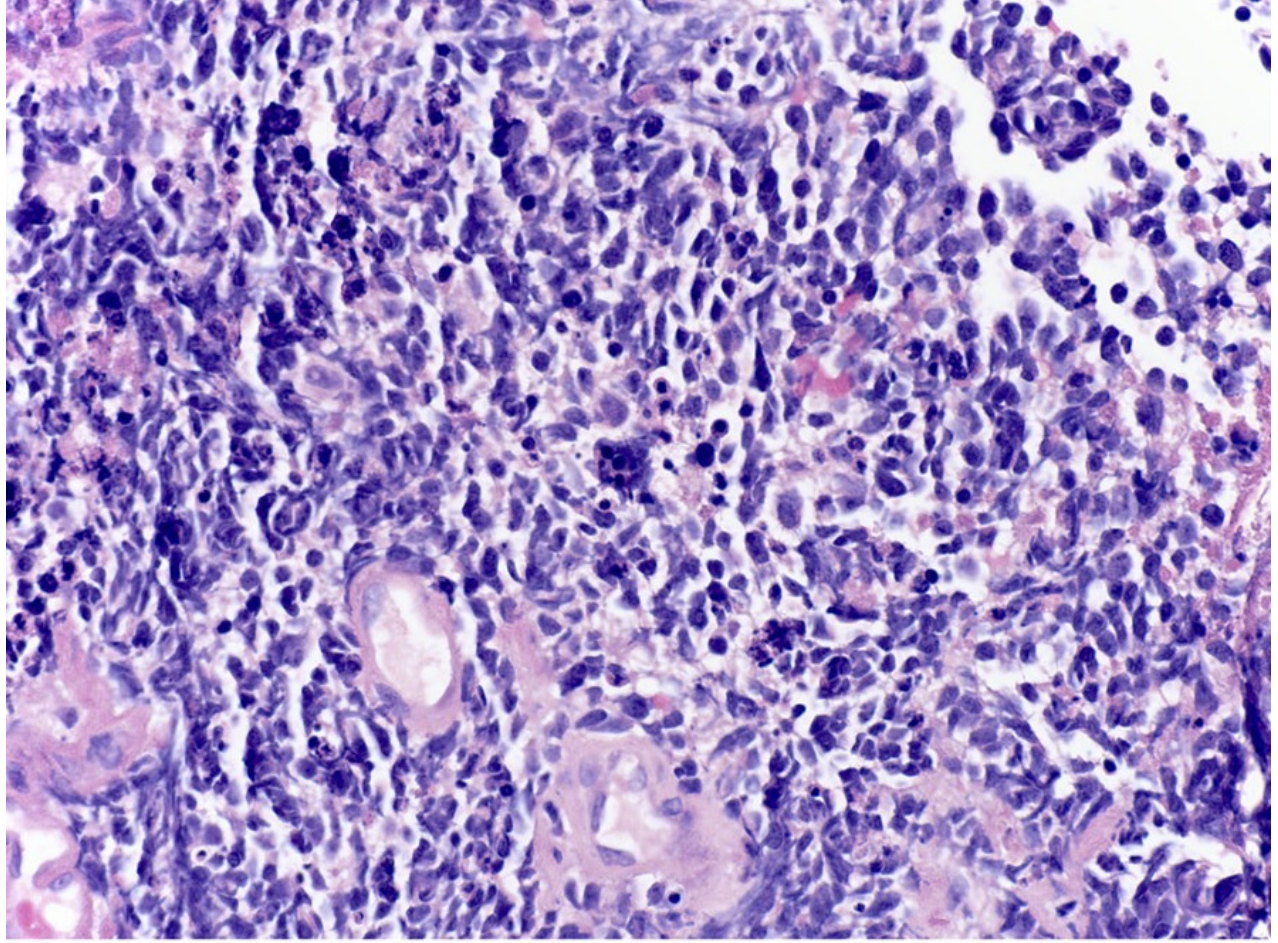
- Small cells, 2-3 times size of lymphocyte
- High nuclear to cytoplasm ratio
- Nuclei with stippled (salt & pepper) chromatin





Small cell

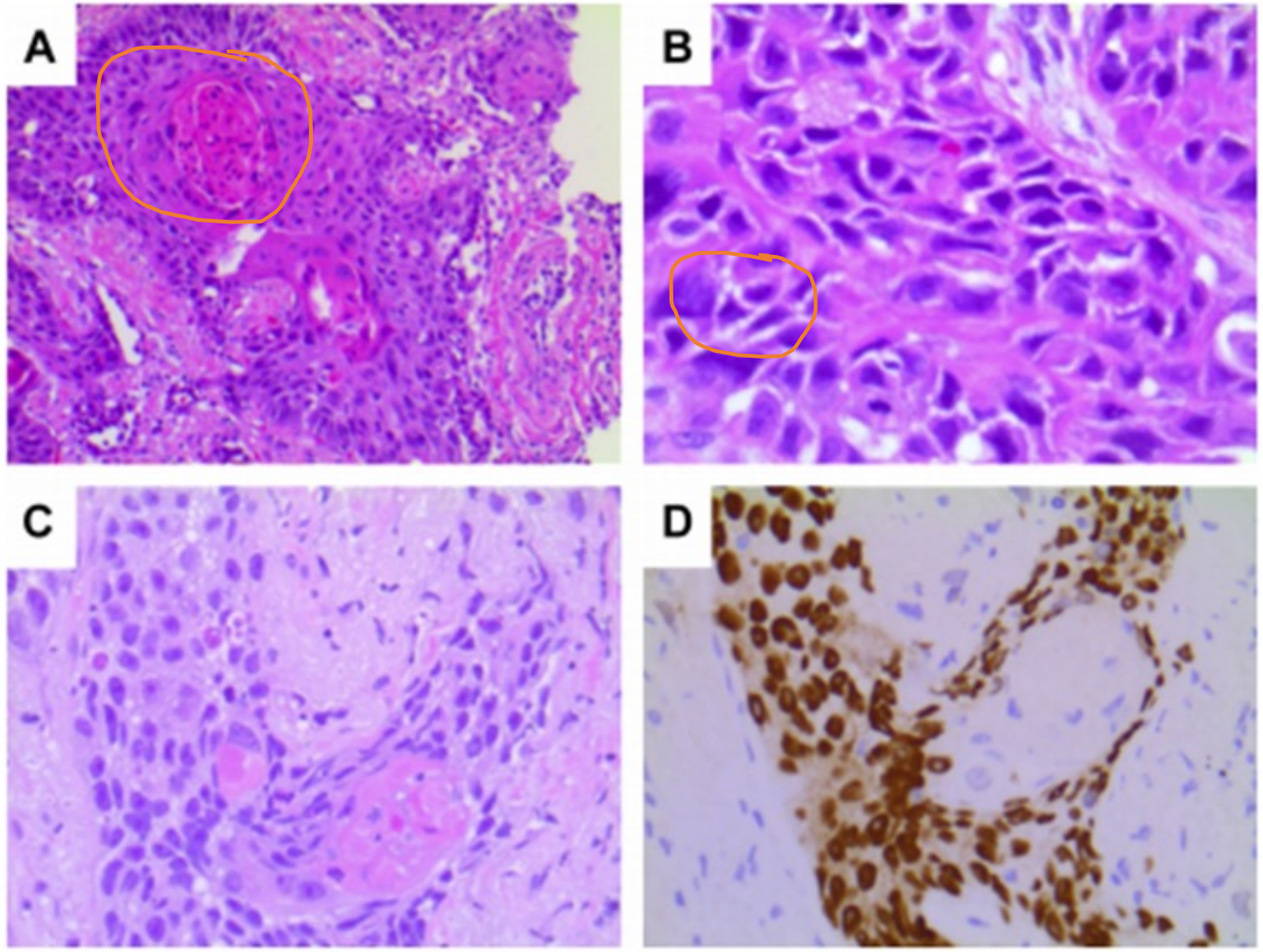


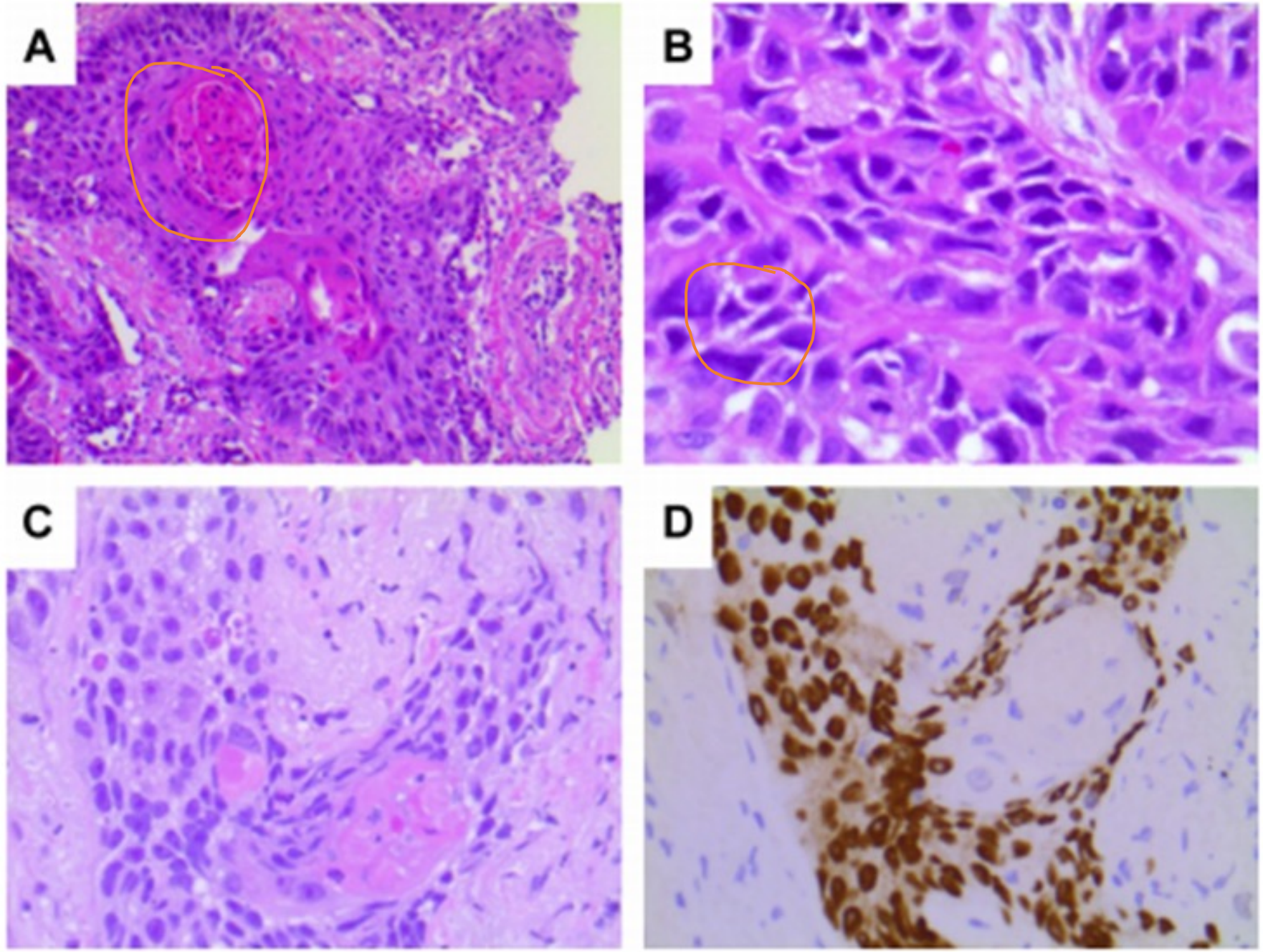


High power view of small cell lung carcinoma demonstrates many of the nuclear features characteristic of the disease; multiple mitotic figures are identified as we as scattered apoptotic tumor cells; the finely dispersed or salt and pepper chromatin with no distinct nucleoli is apparent in many of the cells, although the crush artifact caused by the biopsy process can make this feature more difficult to distinguish; the small cells lie amongst a background of delicate stroma that is sparse compared to the dense sheet of tumor cells

Q

- Histology shows keratin pearl formation and intracellular bridges, what am I ?




A
B
C
D

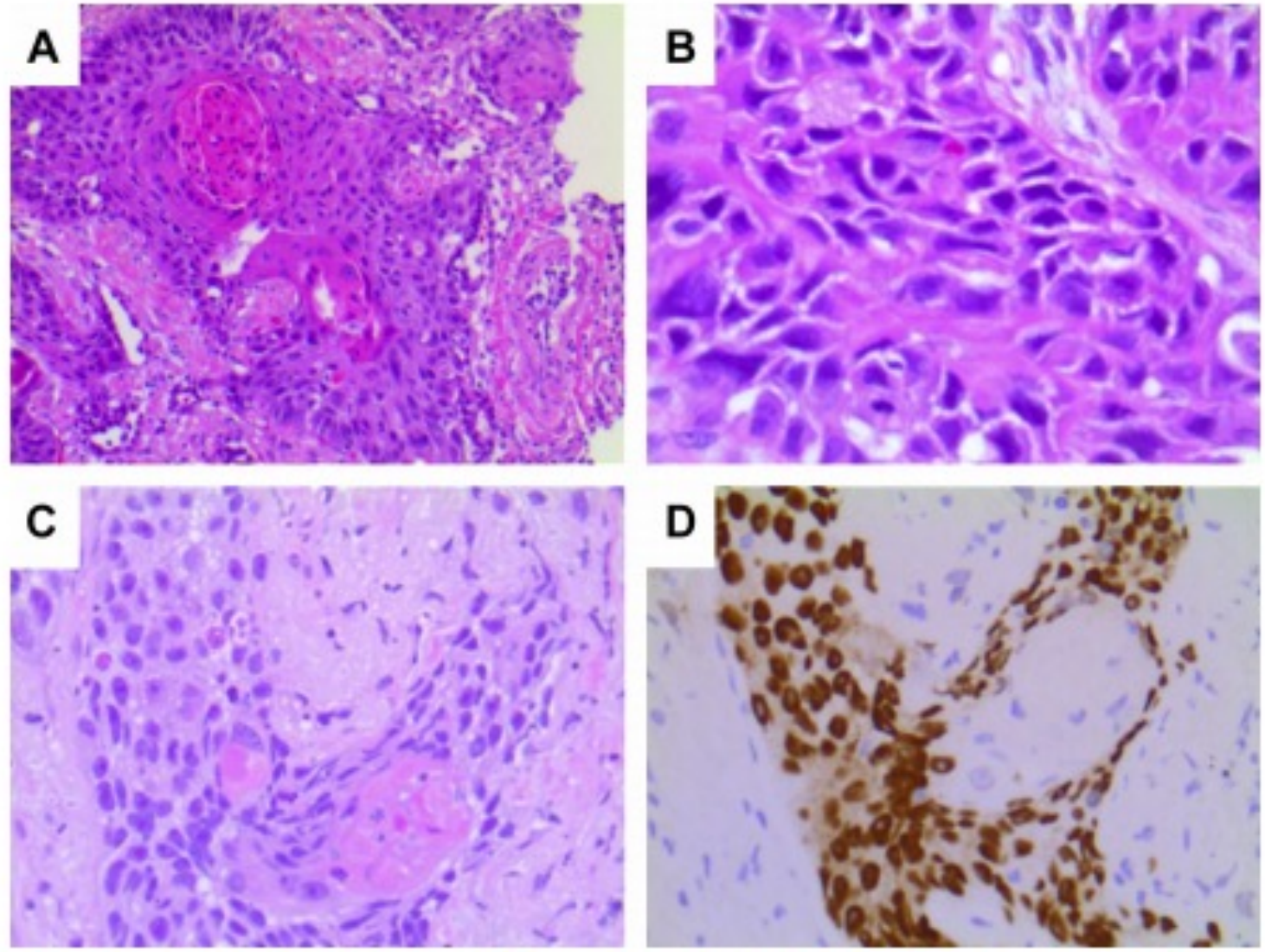


Fig. 2. SCC. (*A*) Keratinizing SCC with keratin pearl formation (H&E, original magnification ×200). (*B*) Intercellular bridge formation (H&E, original magnification ×400). (*C*) Nonkeratinizing SCC without apparent keratinization or discernible intercellular bridges (H&E, original magnification ×400). (*D*) Tumor cells stain positive for p40 (nuclear stain) (p40 stain, original magnification ×400).

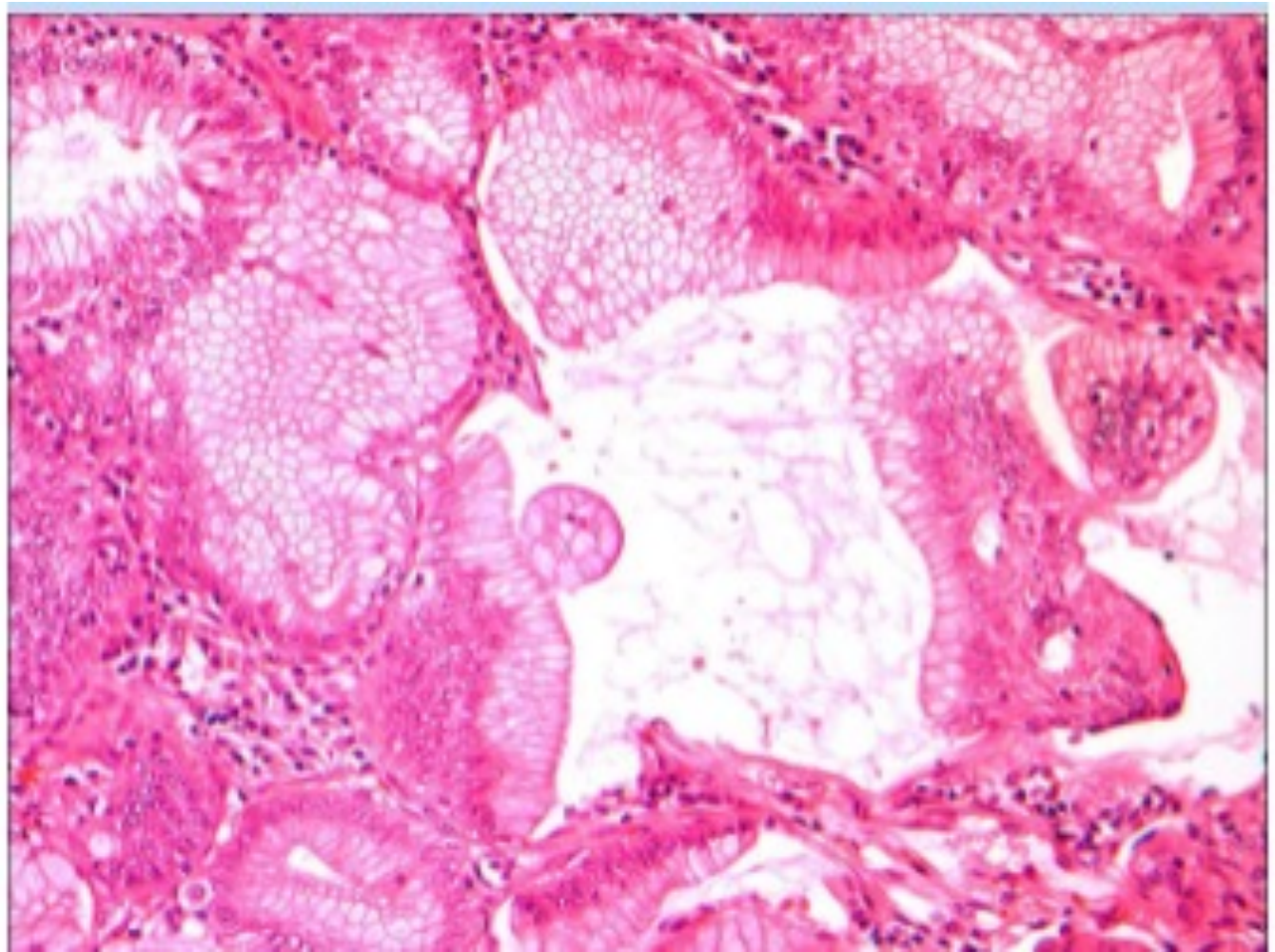
Spot diagnosis

columnar morphology with more pleomorphism, nuclear moulding and cellular crowding

glandular differentiation with or without mucin production

This shows typical histology for what lung condition?

A 59-year-old woman attended the outpatient clinic with a 6-month history of cough. She had no previous illnesses of note. She had a 10 pack-year smoking history, and had given up 25 years previously. Her husband was a heavy smoker.



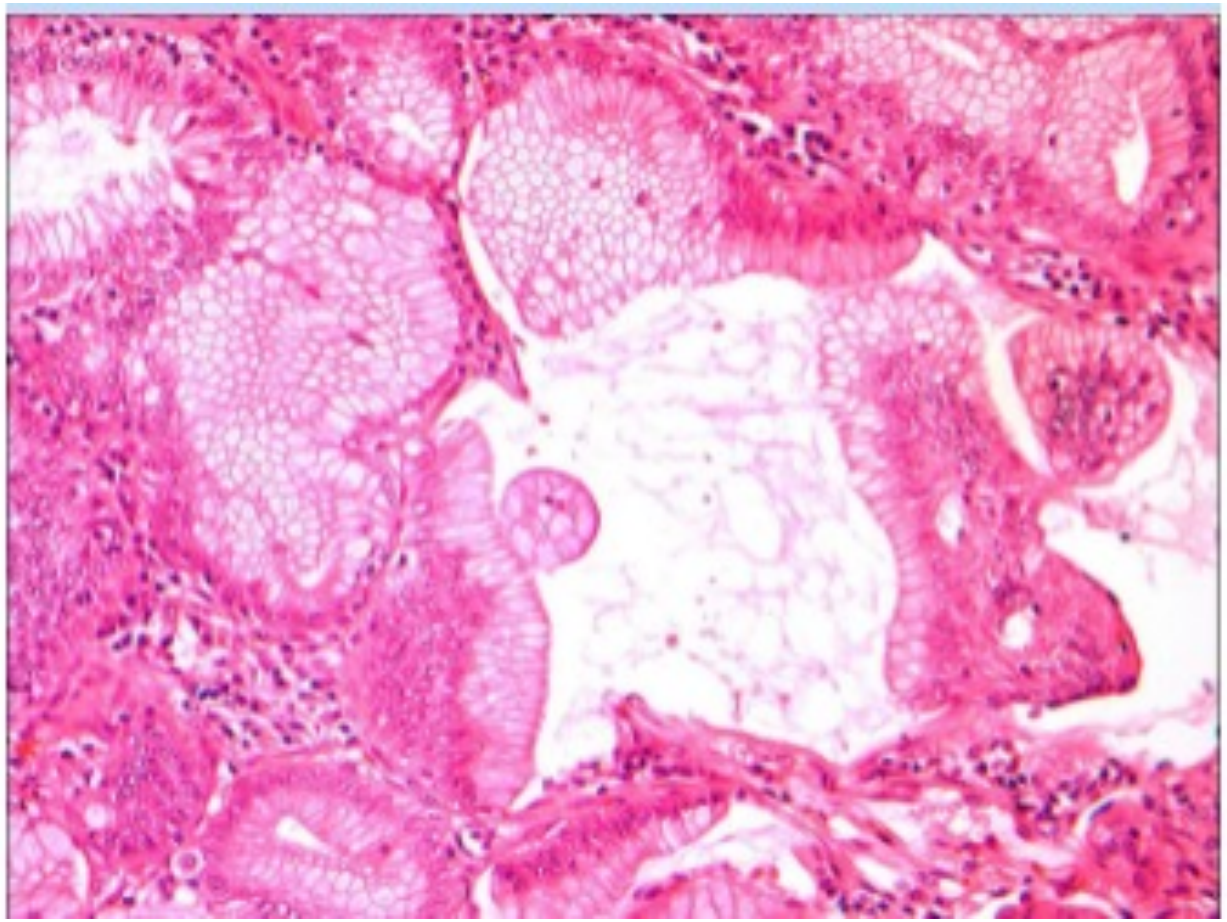
Q – hint

It’s a peripheral lung tumour which demonstrated glandular differentiation with or without mucin production….

columnar morphology with more pleomorphism, nuclear moulding and cellular crowding

Adenocarcinoma

- This is a typical pathology slide consistent with adenocarcinoma with glandular formation with/without mucin production.



Pathology – Adeno

- Mucin, if mucinous type – otherwise hard to diff from meso
- Adeno tends to have columnar morphology with more pleomorphism, nuclear moulding and cellular crowding

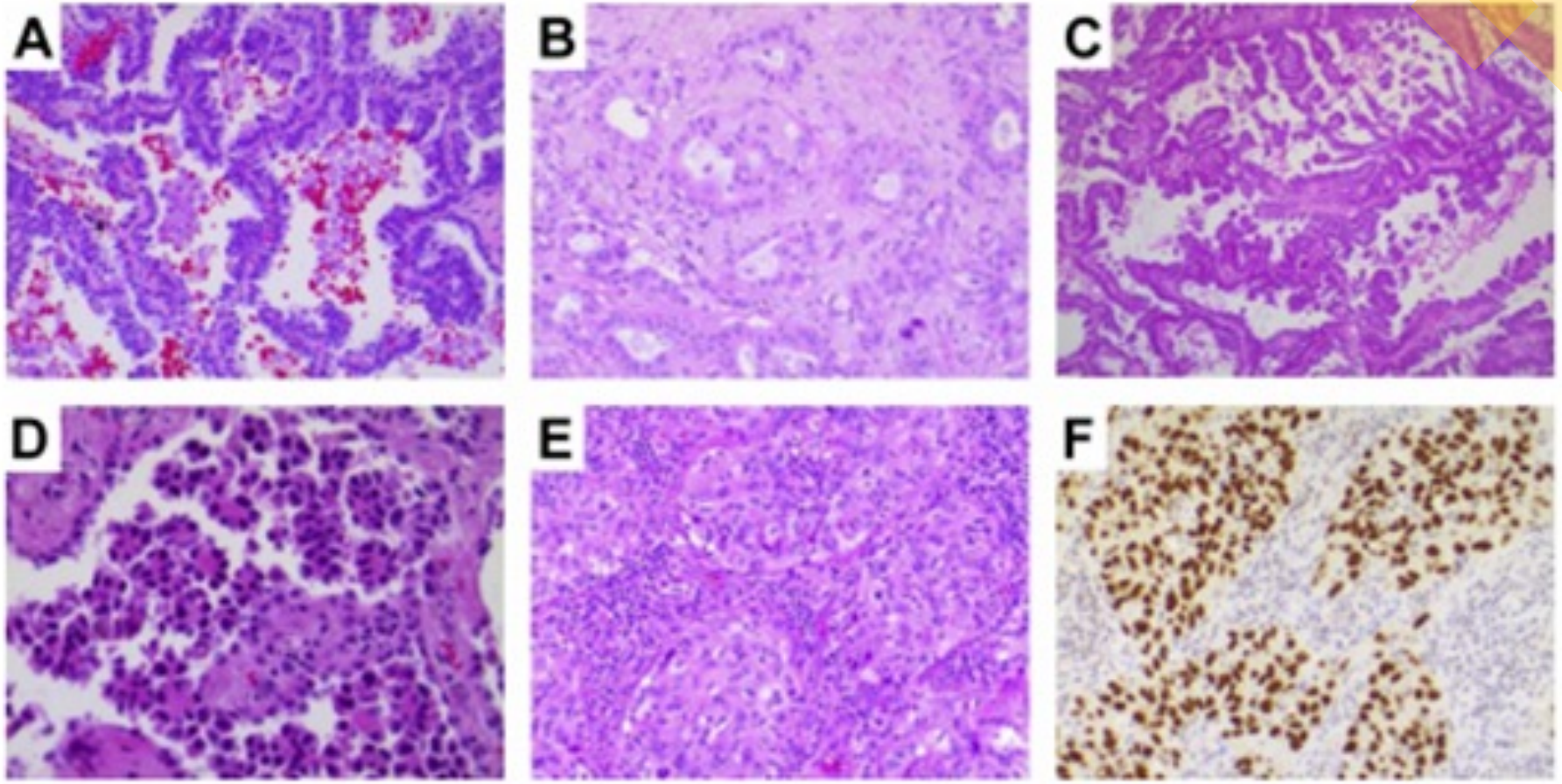


Fig. 1. Adenocarcinomas. (*A*) Lepidic adenocarcinoma (H&E, original magnification ×200). (*B*) Acinar adenocarcinoma (H&E, original magnification ×200). (*C*) Papillary adenocarcinoma (H&E, original magnification ×100). (*D*) Micropapillary adenocarcinoma (H&E, original magnification ×200). (*E*) Solid adenocarcinoma (H&E, original magnification ×100). (*F*) Solid adenocarcinoma (TTF-1 stain, original magnification ×100).

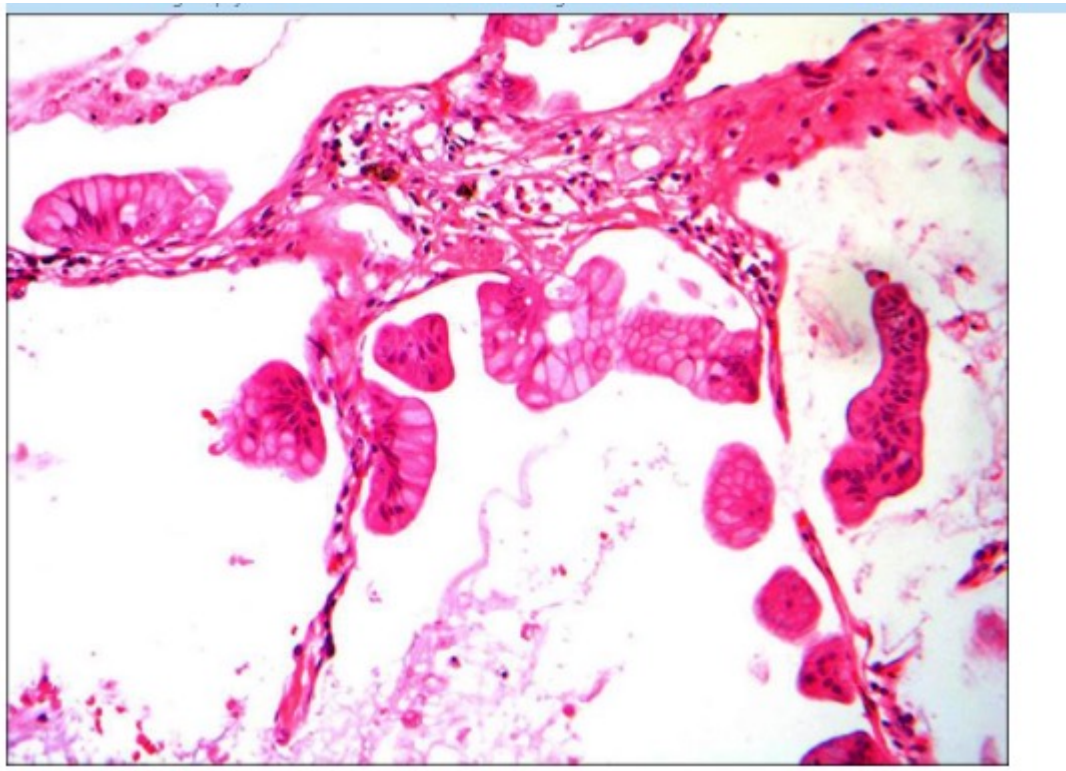
A 59-year-old woman presented to the outpatient clinic with a 6-month history of cough, and weight loss of 2 kg. Her father had had pulmonary tuberculosis when she was young, but she could not remember any further details. She had a 20 pack-year smoking history, and had stopped smoking 4 years previously.

On examination, she looked unwell. There were scattered crackles throughout her chest.

Investigations:

chest X-ray	several areas of patchy shadowing involving both lung fields
transbronchial lung biopsy	see image

What does this show?

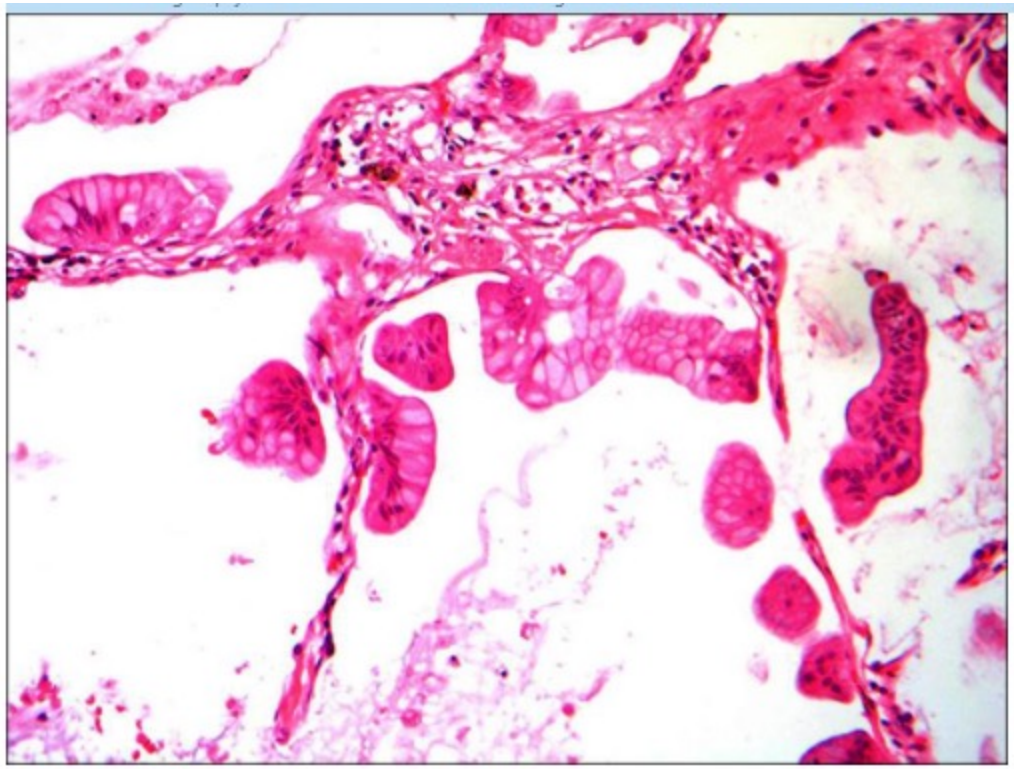


Answers

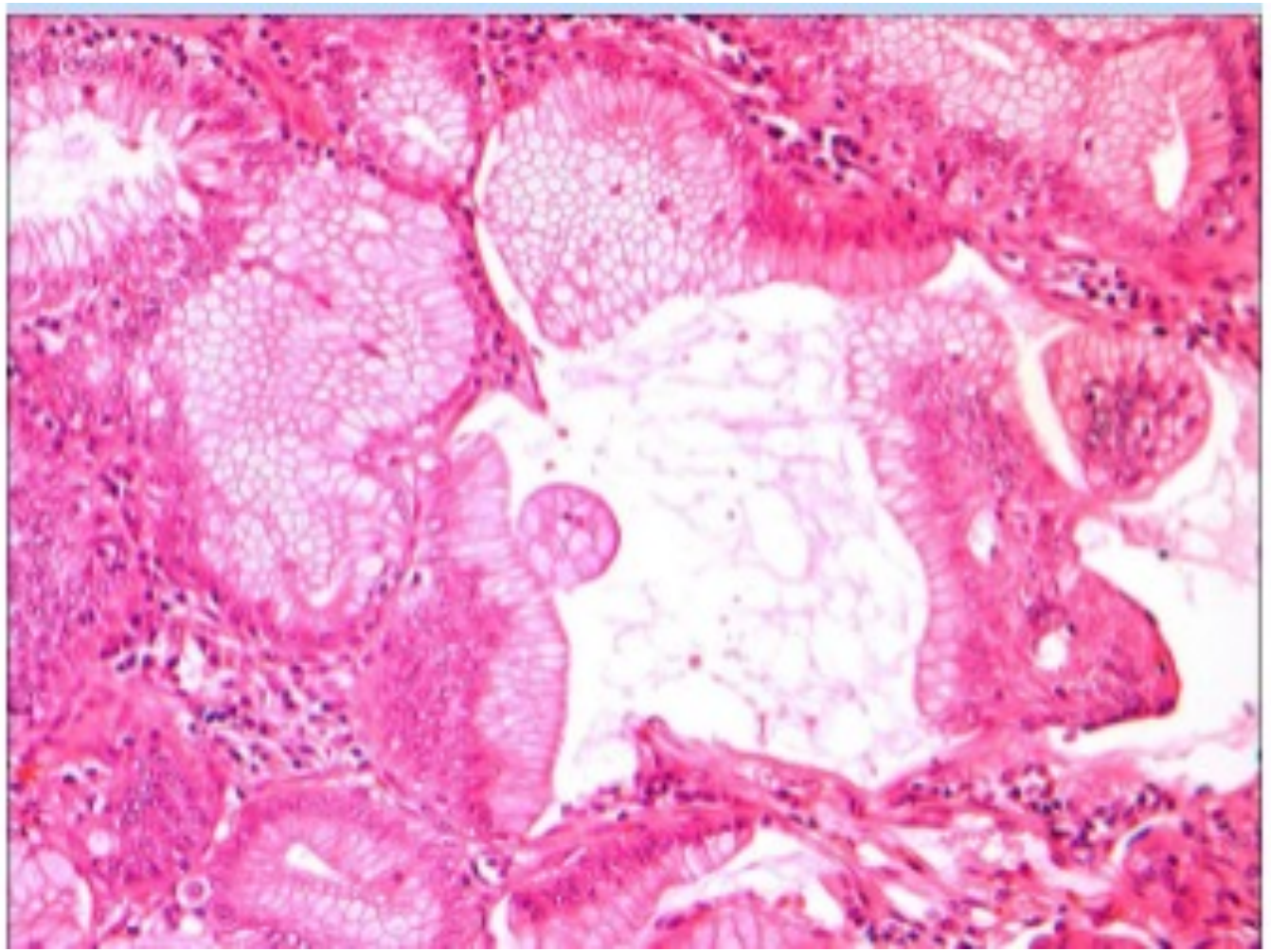
- A: COPD
- B: cryptogenic organising pneumonia
- C: lepidic adenocarcinoma (bronchioloalveolar carcinoma)
- D: pulmonary tuberculosis
- E: sarcoidosis

Hint: tumour with a tendency to spread locally using the lung structure as a stroma (lepidic growth) with preservation of the underlying architecture

Lepidic adenocarinoma



Invasive adenocarinoma



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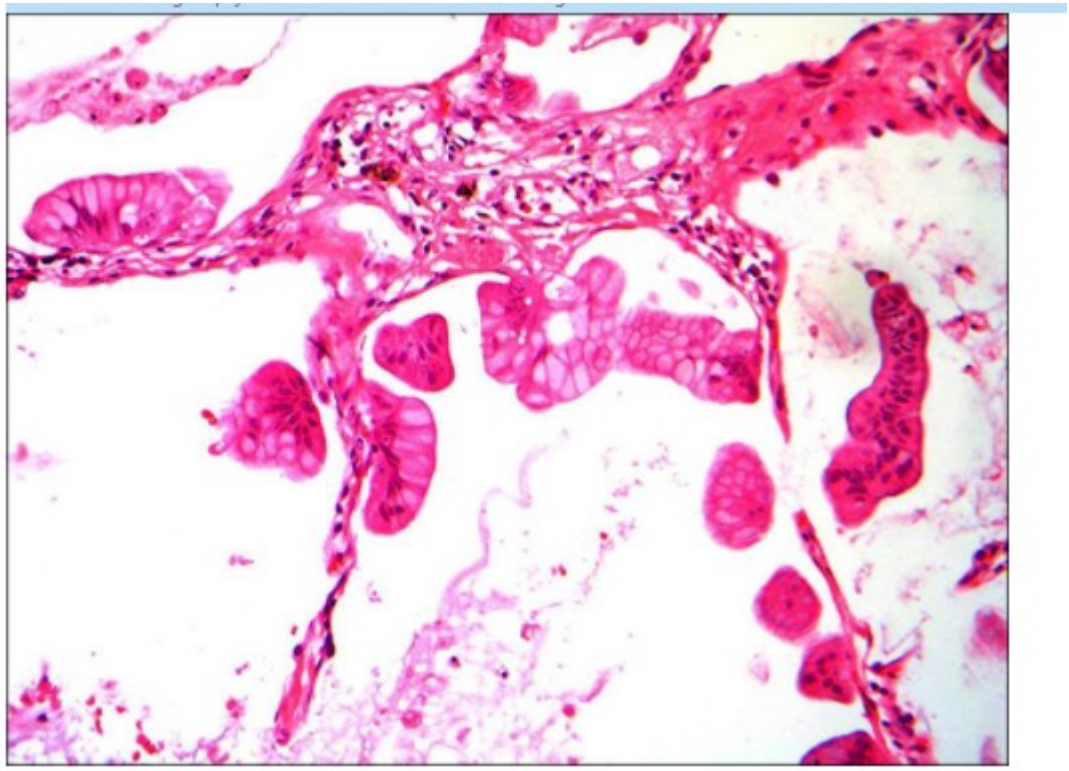
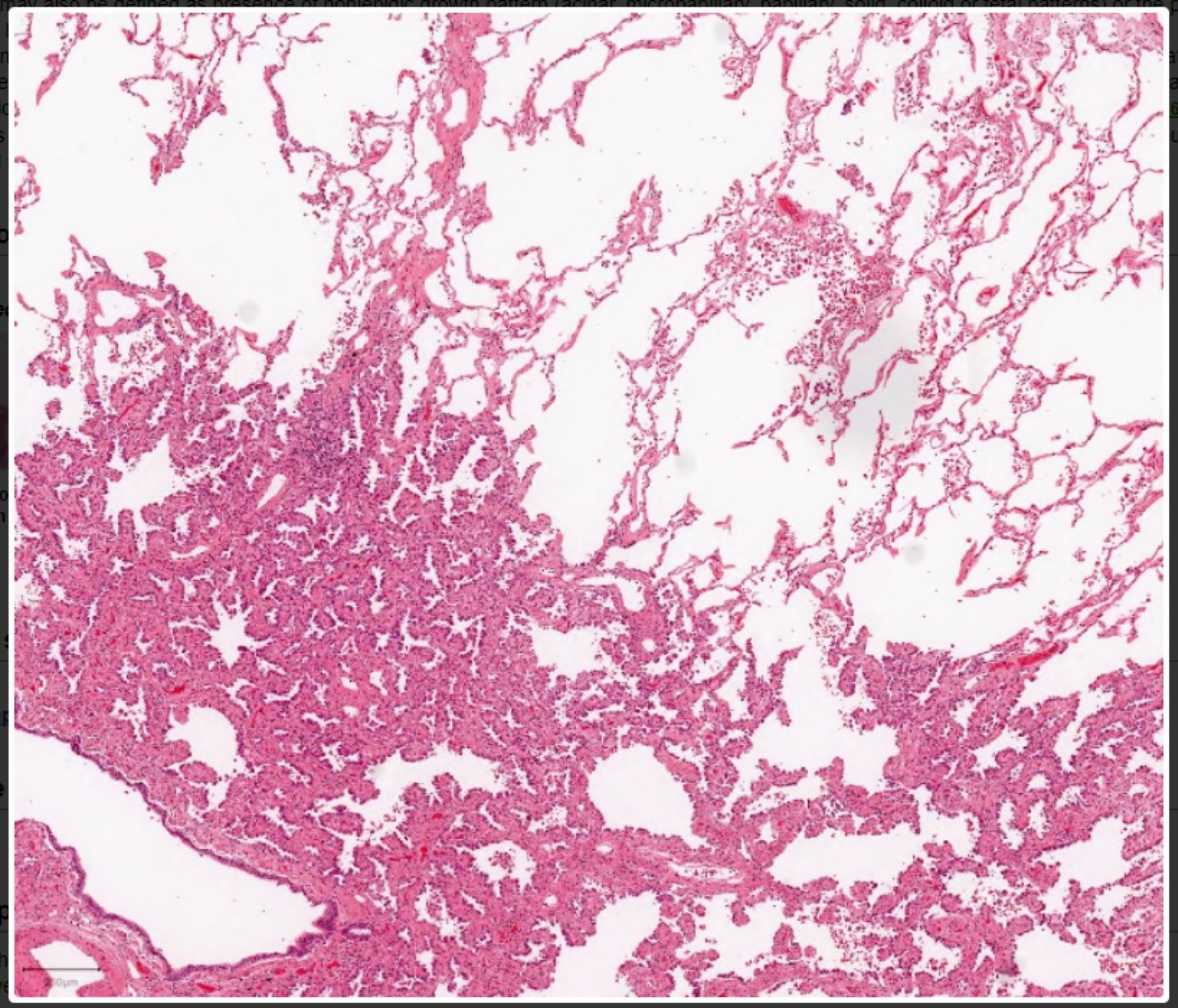
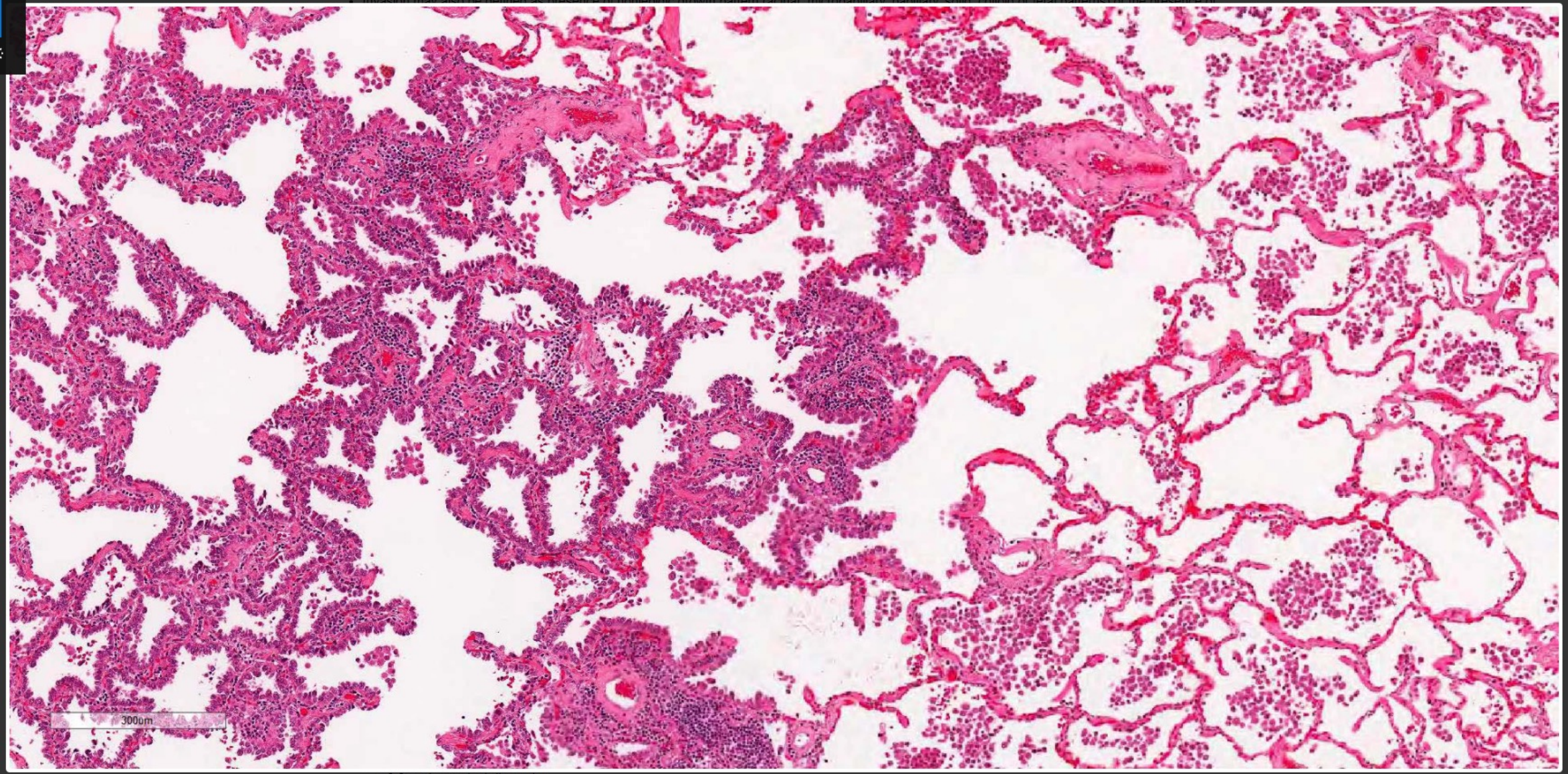


Lepidic adenocarinoma

The slide shows tumour with a tendency to spread locally using the lung structure as a stroma (lepidic growth) with preservation of the underlying architecture.



Medium power photomicrograph of lepidic adenocarcinoma (bottom) juxtaposed with adjacent uninvolved lung parenchyma (top). The lepidic adenocarcinoma is characterized by an abrupt transition to thickened alveolar septa lined by atypical overlapping cuboidal cells.
Contributed by Jonathan Keow, M.D., Ph.D.



Noninvasive lepidic adenocarcinoma (left) juxtaposed with adjacent uninvolved lung parenchyma (right). The lepidic adenocarcinoma is characterized by thickened alveolar septa lined by atypical overlapping cuboidal cells.
Contributed by Jonathan Keow, M.D., Ph.D.

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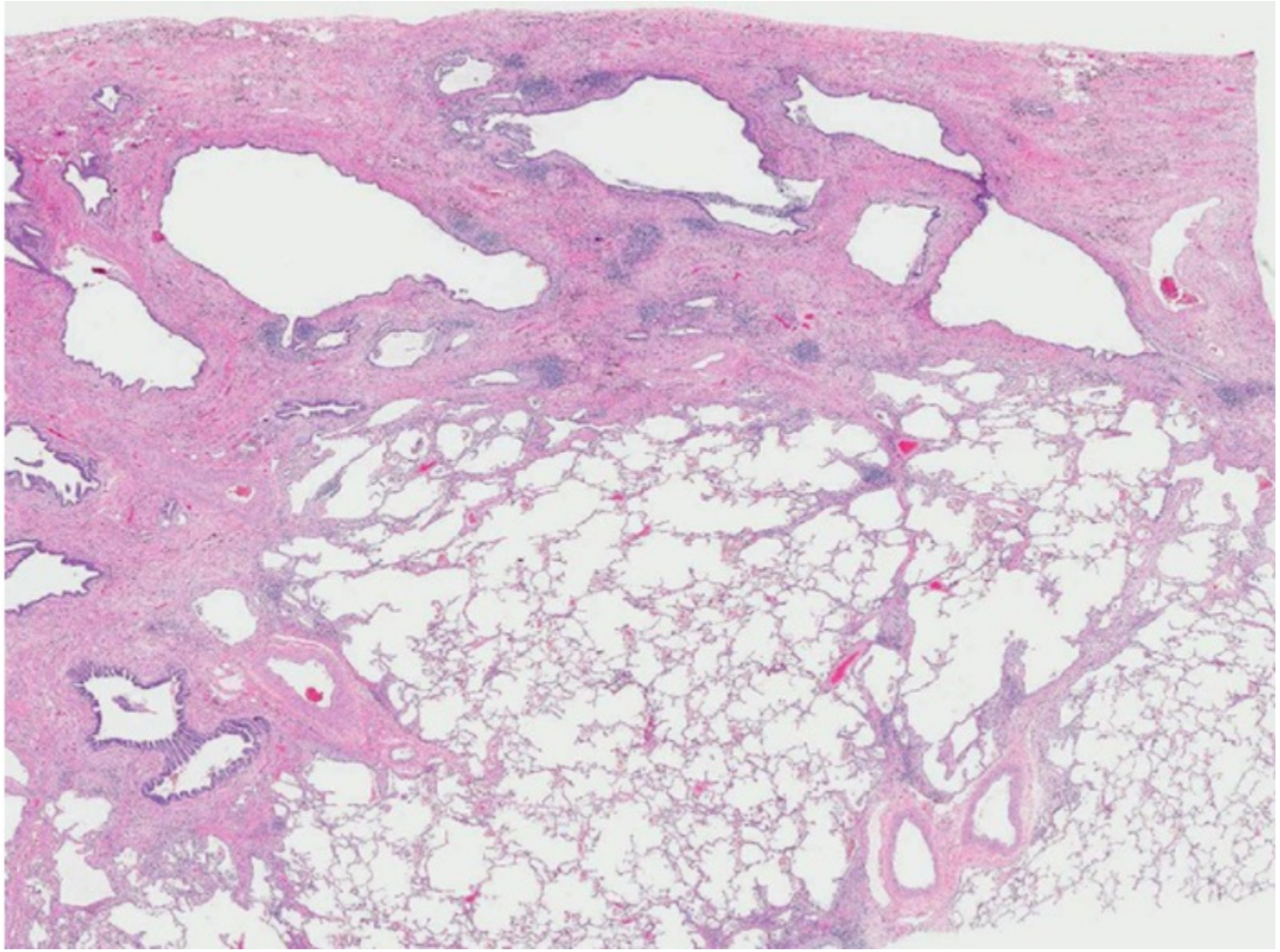
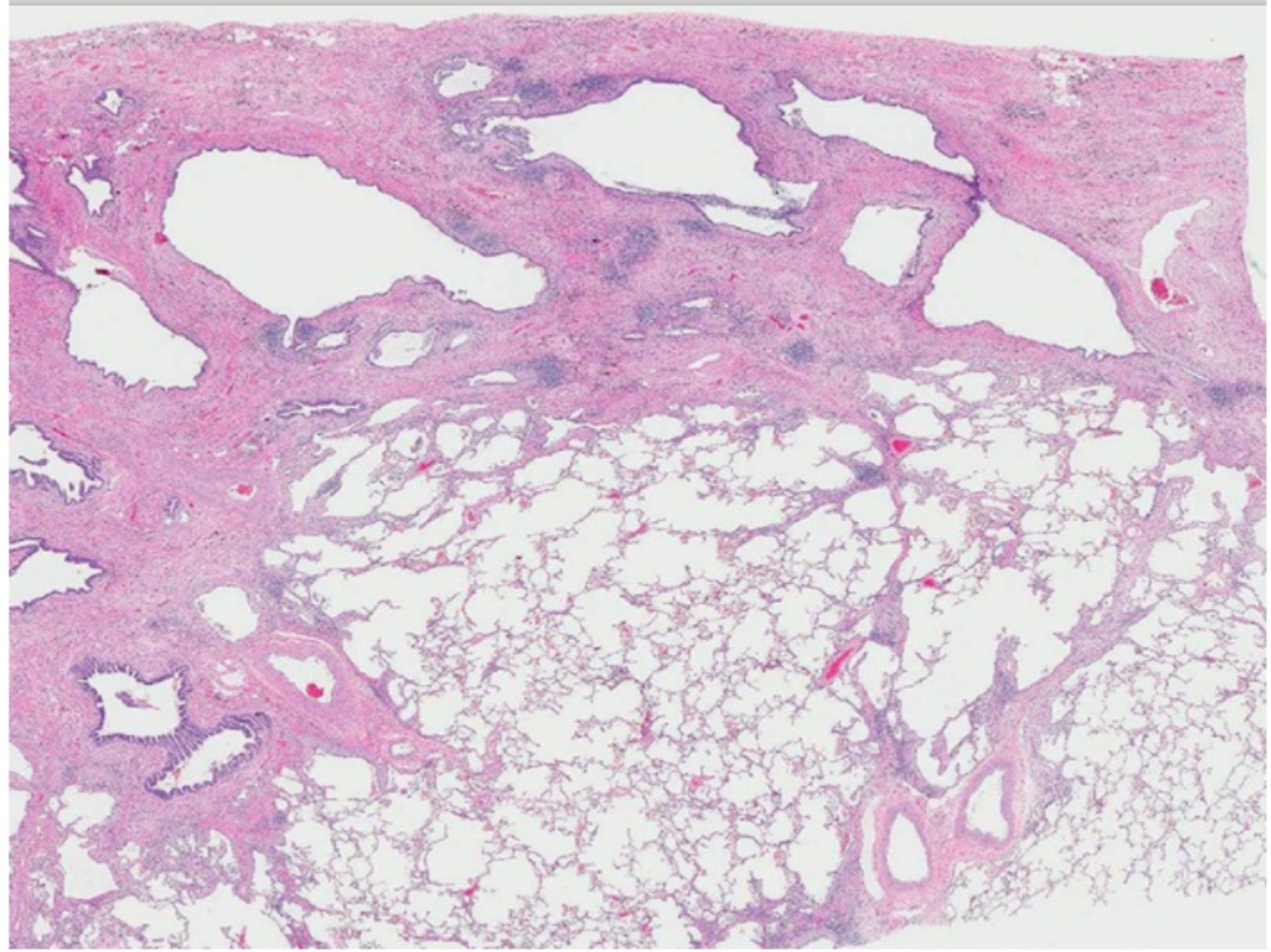
More histology

What’s this?



UIP



Low magnification photomicrograph showing the heterogeneous patchwork distribution of abnormalities classically seen with usual interstitial pneumonia (UIP). UIP appears as areas of fibrotic scarring with honeycomb change primarily affecting the subpleural and paraseptal parenchyma alternating with regions of normal lung tissue.

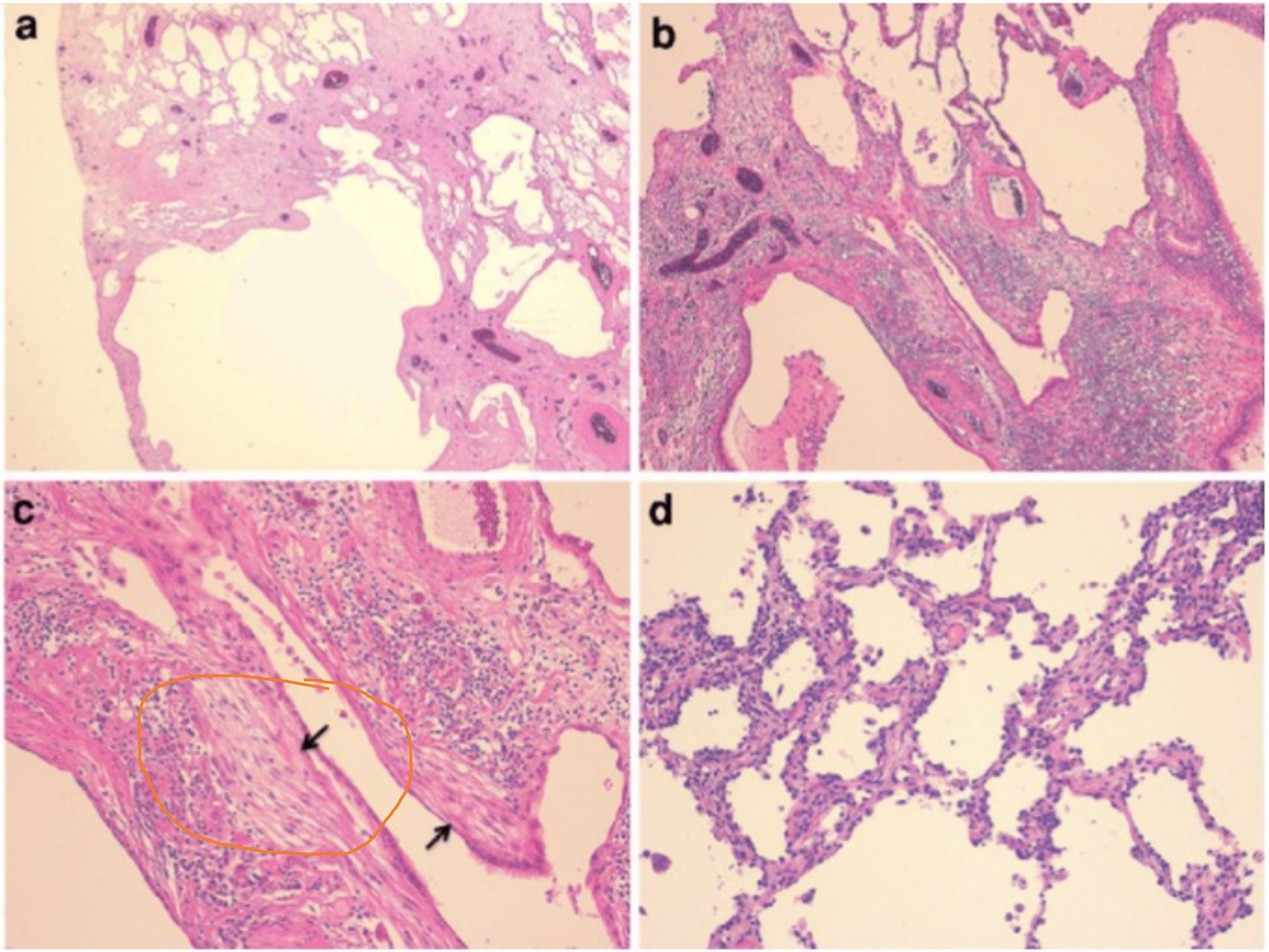
What am I?

- Histology says “Numerous fibroblasts”

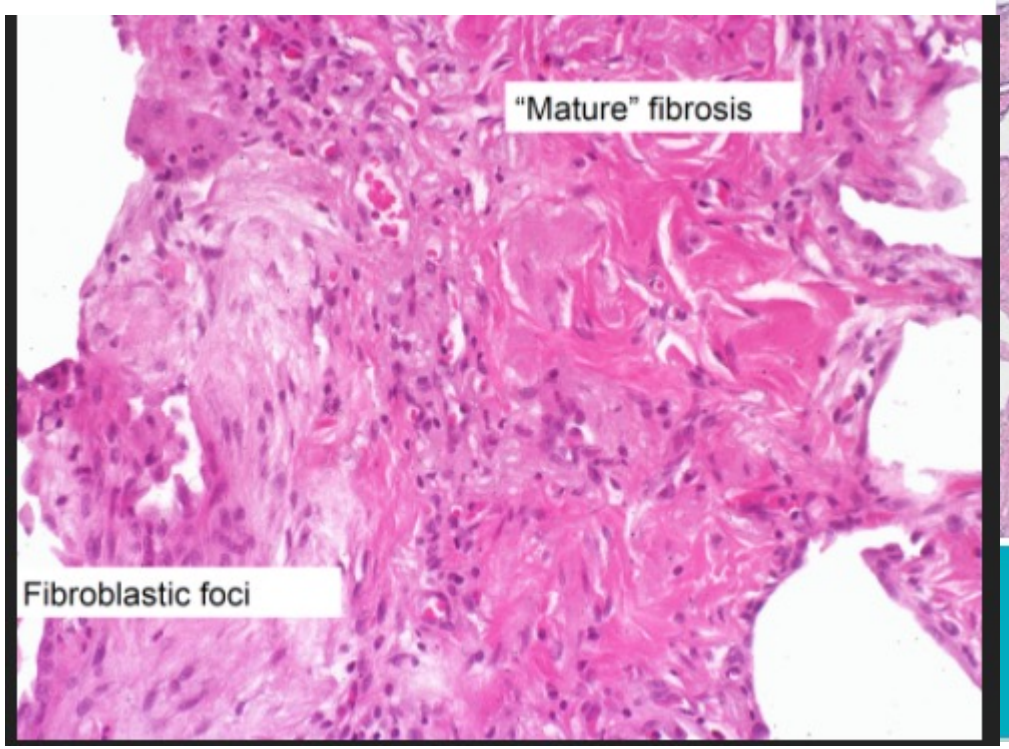
- Want another hint…???

What am I?

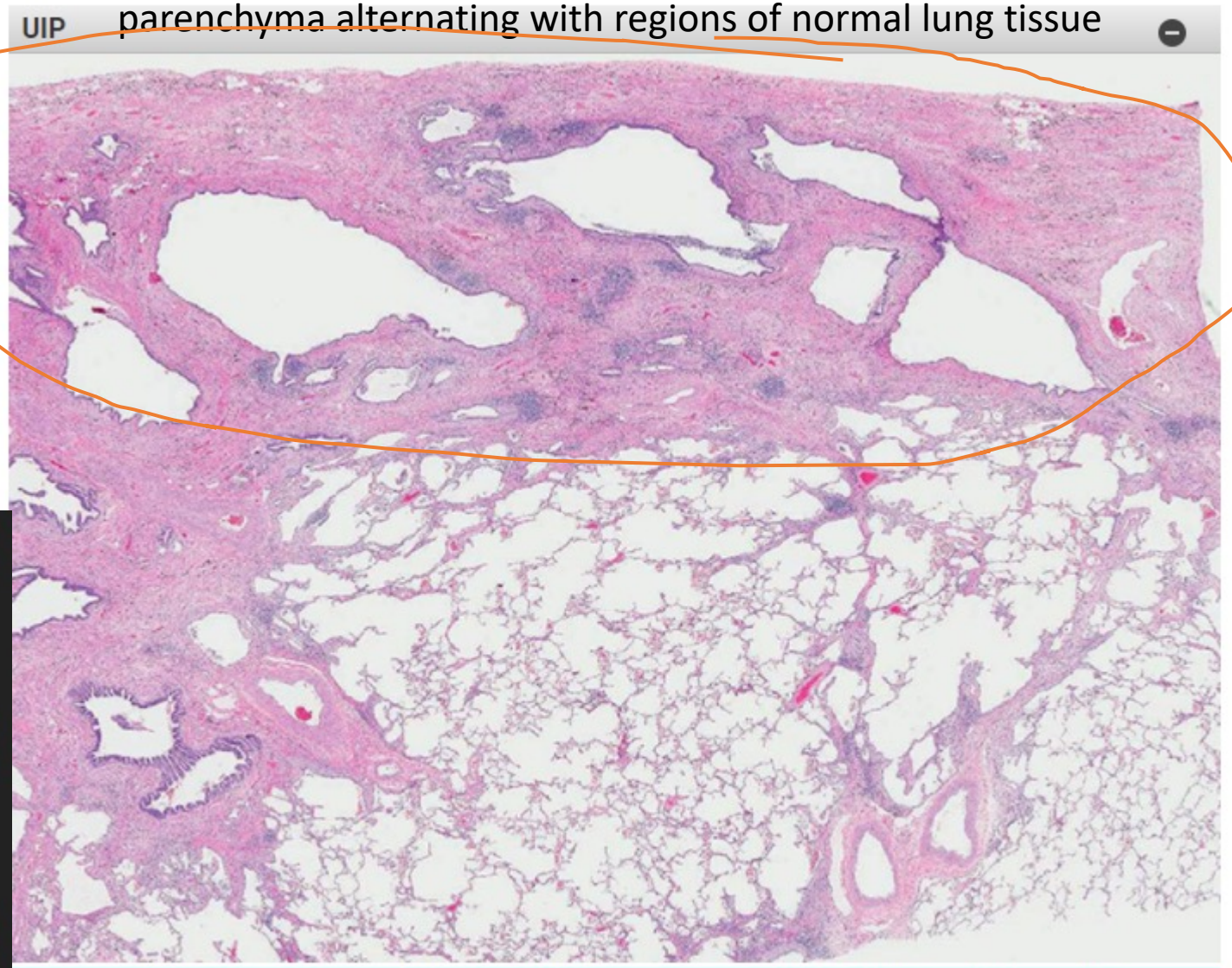
- Numerous fibroblasts
- Areas of fibrotic scarring in subpleural and paraseptal parenchyma alternating with regions of normal lung tissue


a
b
c
d

Numerous fibroblastic foci are characteristic of UIP

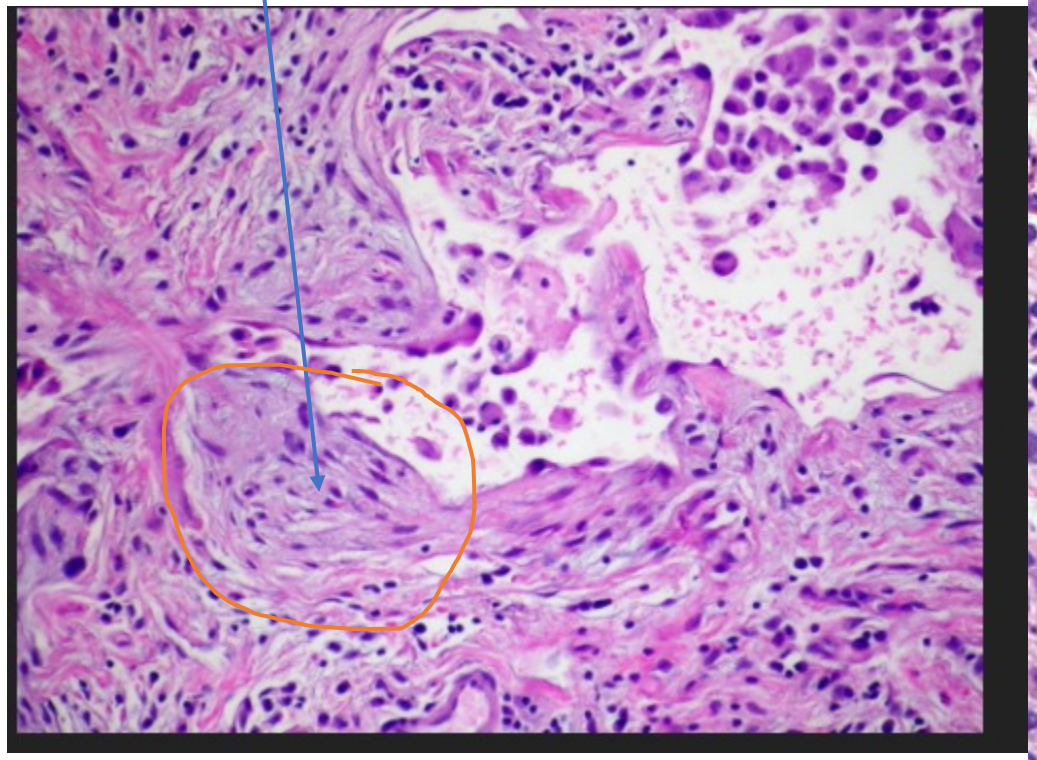


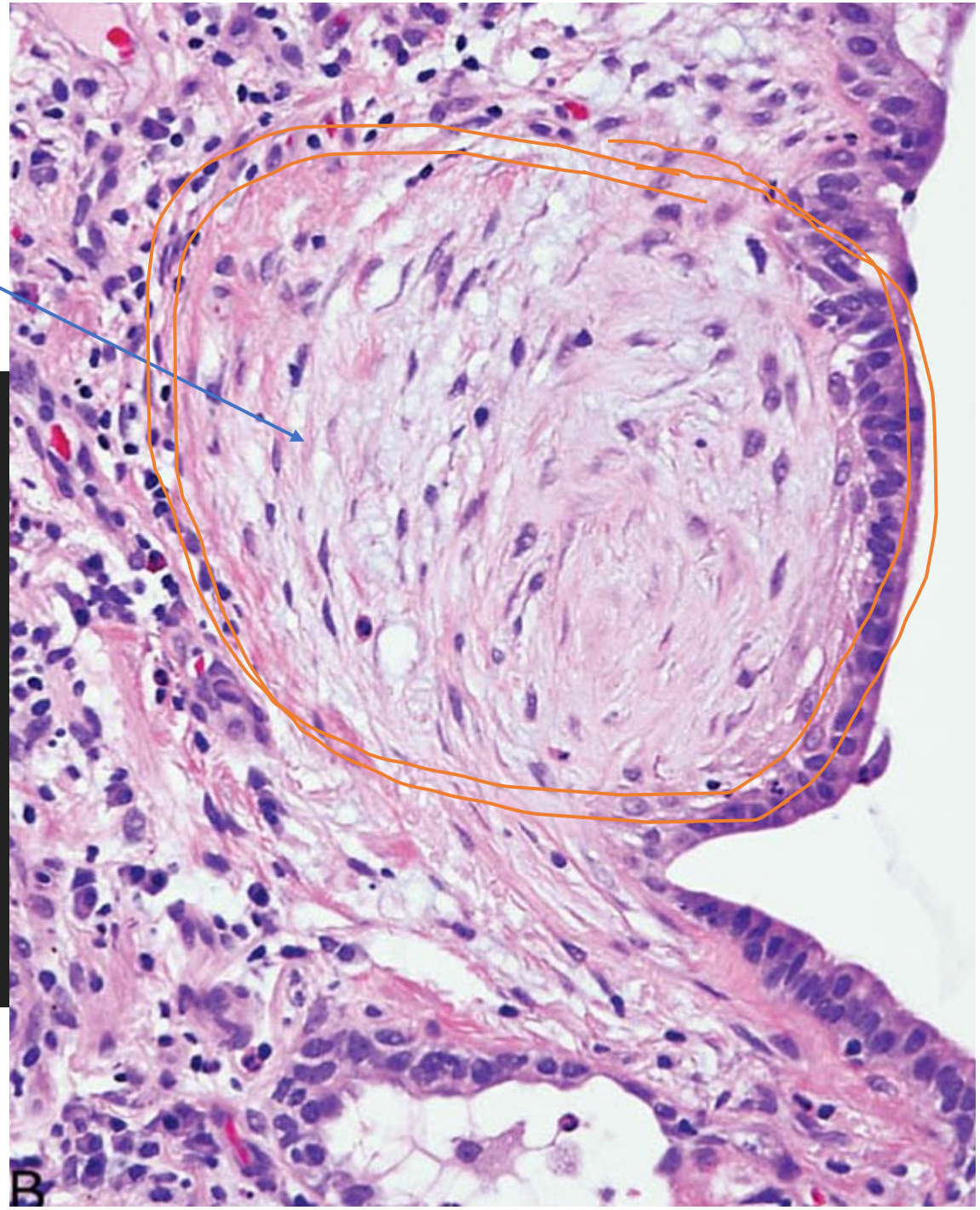
Areas of fibrotic scarring in subpleural and parasetpal parenchyma alternating with regions of normal lung tissue



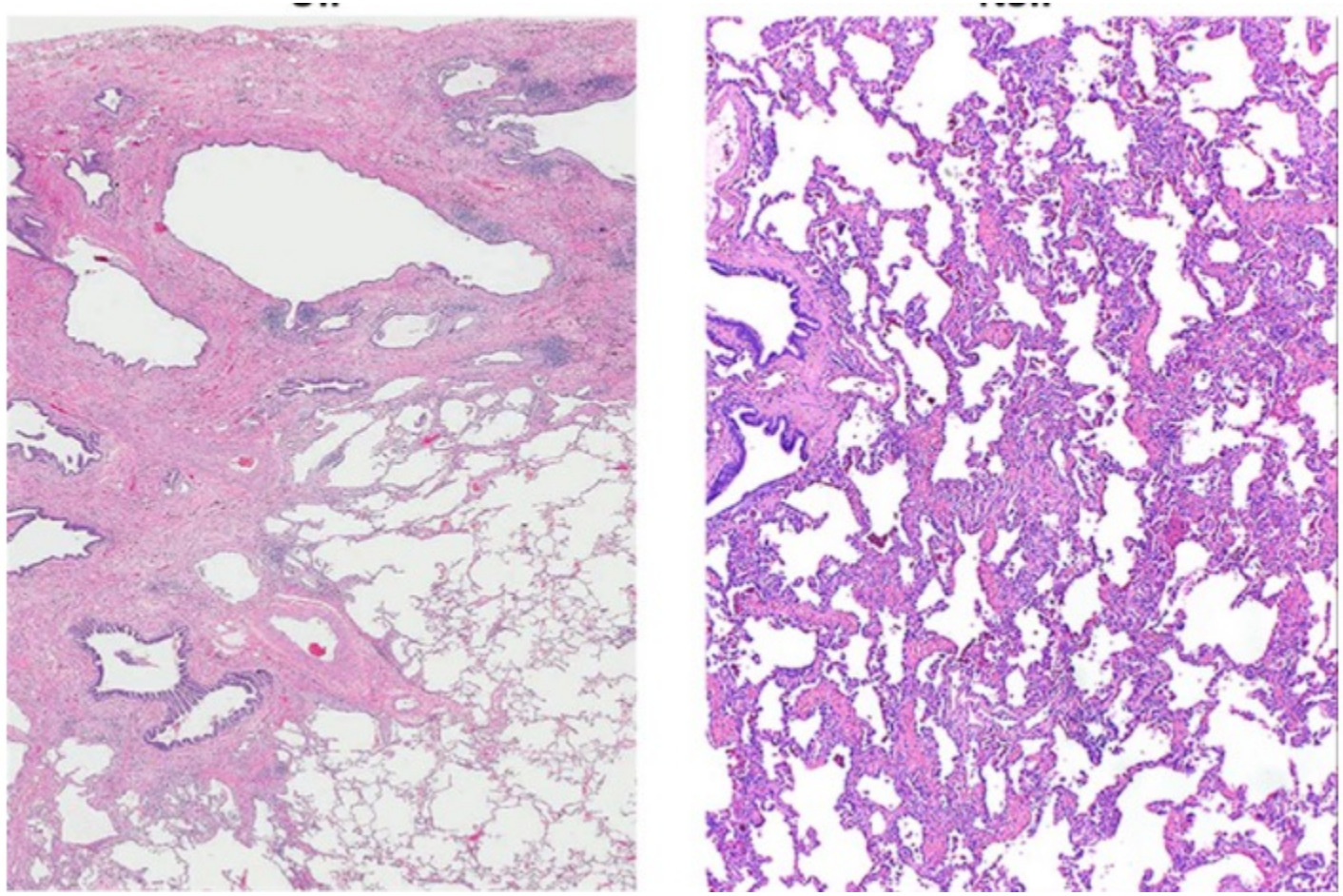
Low magnification photomicrograph showing the heterogeneous patchwork distribution of abnormalities classically seen with usual interstitial pneumonia (UIP). UIP appears as areas of fibrotic scarring with honeycomb change primarily affecting the subpleural and paraseptal parenchyma alternating with regions of normal lung tissue.

Numerous fibroblastic foci are characteristic of usual interstitial pneumonia.

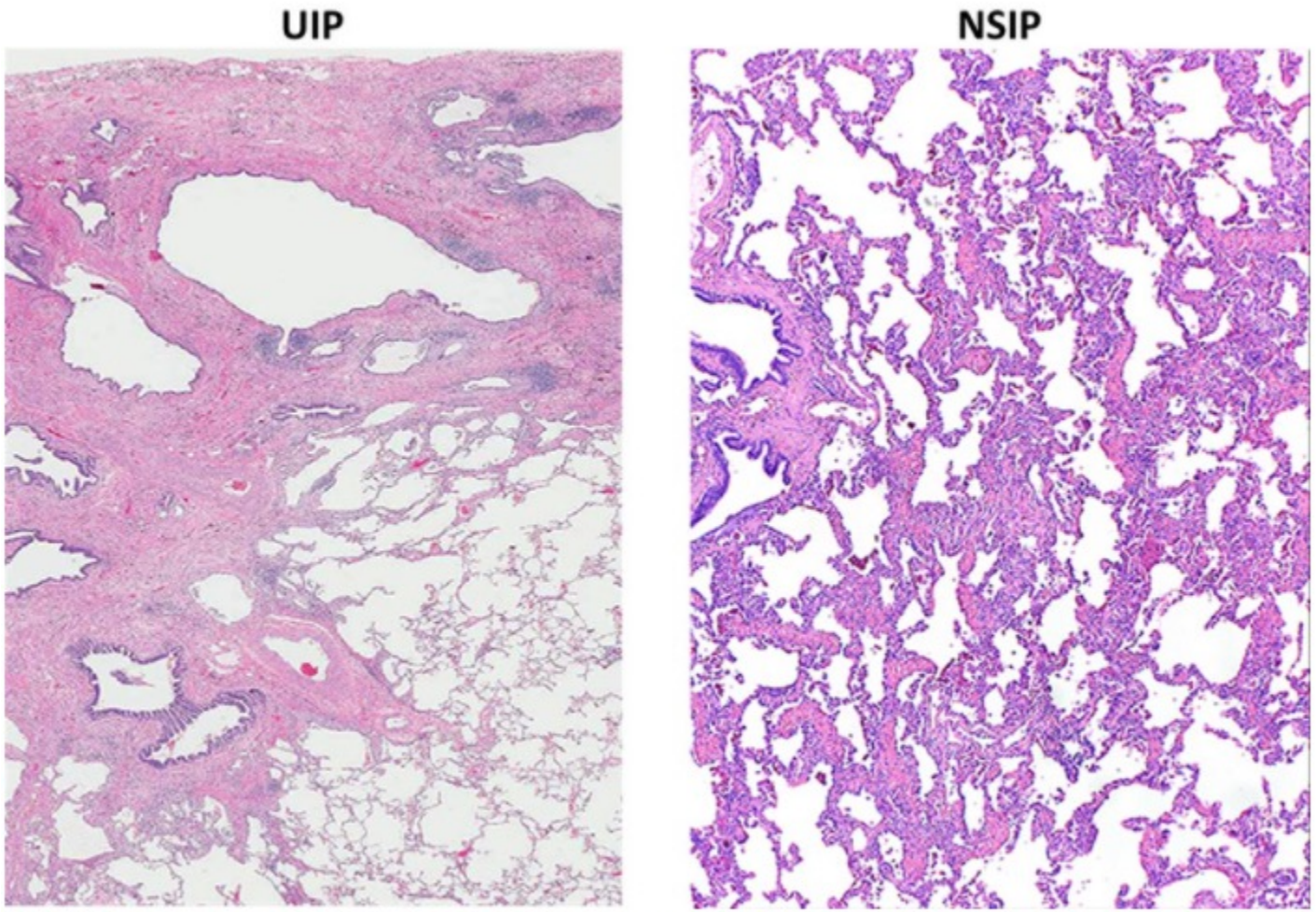




What are these two (different)



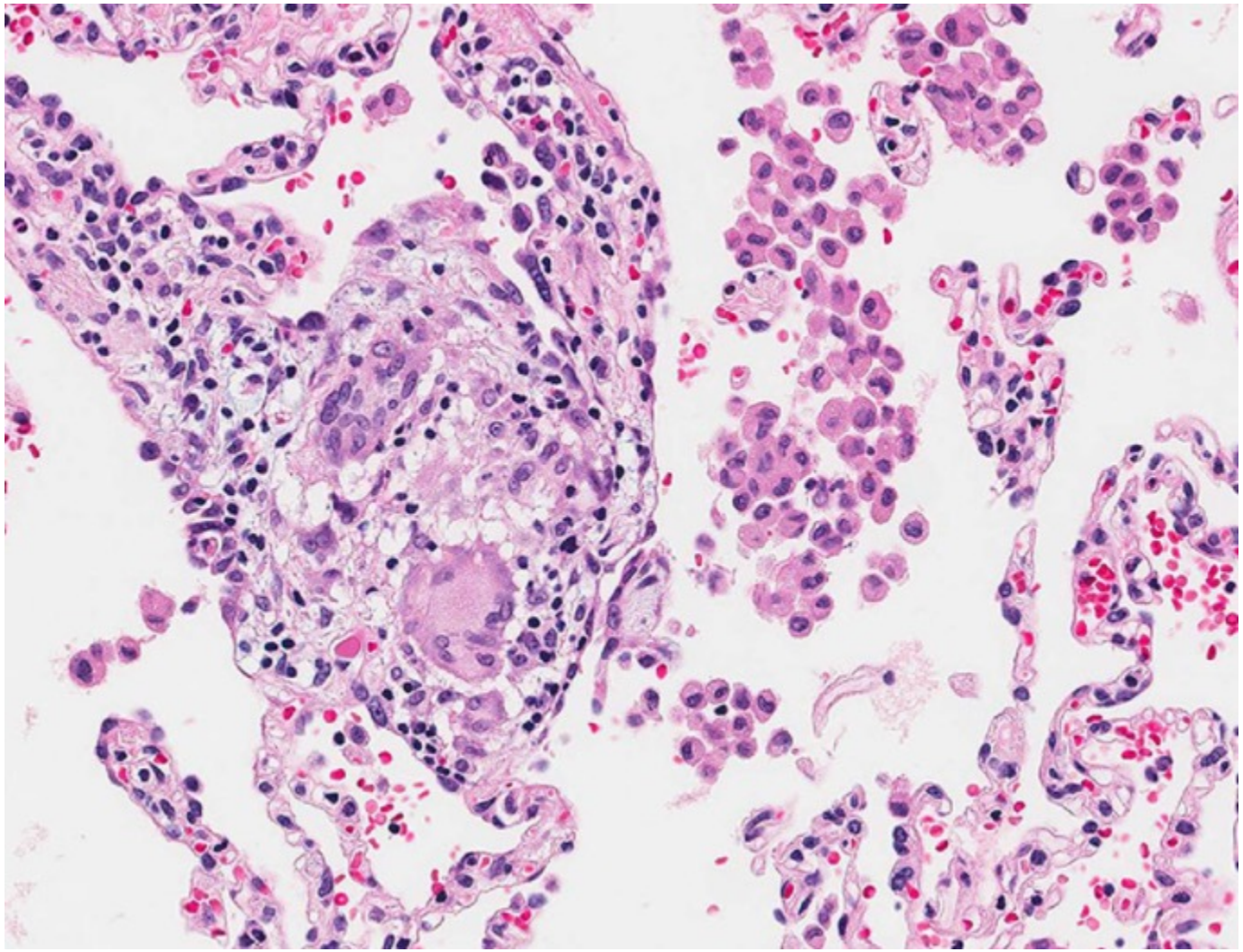
Histology: UIP vs NSIP



Side-by-side comparison of photomicrographs from patients with usual interstitial pneumonia (UIP, left) and nonspecific interstitial pneumonia (NSIP, right)). Note the similarity in the abnormality but the difference in distribution. UIP is a heterogeneous process, interspersed with normal lung, whereas NSIP is more homogeneous, affecting the entire lung.

What's this?

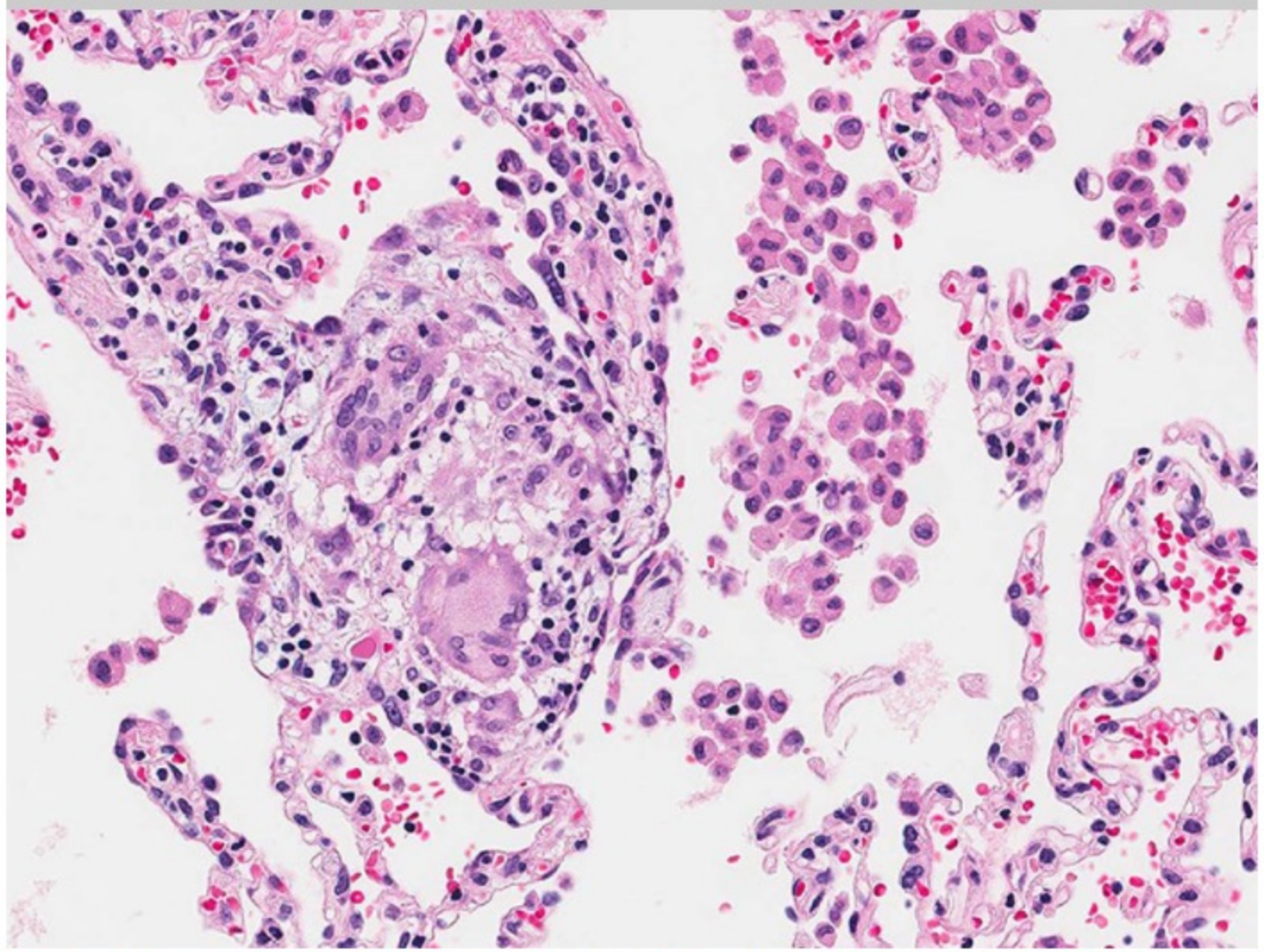
Hint small poorly formed interstitial granuloma



HP

Low-resolution photomicrograph illustrating the peribronchiolar fibrosis typical of chronic hypersensitivity pneumonitis.

Image courtesy of and used with permission from Kirk Jones, MD.



High resolution photomicrograph illustrating small interstitial granulomas typical of hypersensitivity pneumonitis.

Image courtesy of and used with permission from Kirk Jones, MD.

Q

A 52-year-old woman presented with a 6-month history of breathlessness on exertion.

On examination, bilateral inspiratory squeaks were heard on auscultation of the lungs.

Investigations:

high-resolution CT scan of chest	patchy ground-glass change and air trapping
video-assisted thorascopic surgical lung biopsy	hypersensitivity pneumonitis (extrinsic allergic alveolitis)

What best describes the pathogenesis of the changes in the lungs?

Answers

A: circulating autoantibodies cause cell cytotoxicity
B: IgG-containing immune complexes are formed
C: infiltrating eosinophils damage tissue by releasing enzymes and oxidants
D: inhaled antigen leads to IgE-mediated inflammation
E: neutrophil oxidative burst causes tissue damage

Ans

- **Correct answer: B**
- **Explanation**
- The pathogenesis of hypersensitivity pneumonitis is classically type 3 hypersensitivity mediated by IgG immune complexes.

Diagnosis: Histologic Findings in Subacute HP

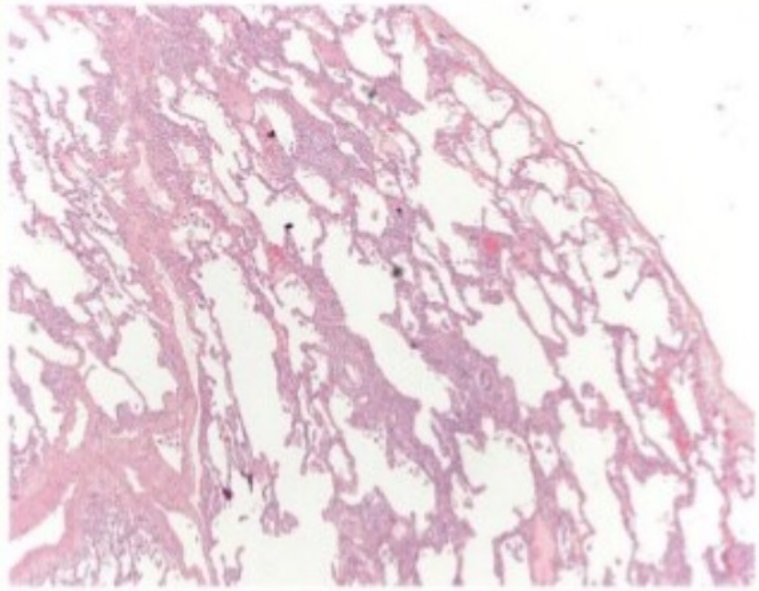


Figure 5. A classic example of subacute hypersensitivity pneumonitis characterized by a cellular infiltrate with centrilobular accentuation. A few small granulomas can be seen even at low magnification (hematoxylin-eosin, original magnification x40).

The classic histologic triad of subacute HP includes

1. Interstitial infiltrate
2. Cellular bronchiolitis
3. Poorly formed granulomas

This triad is present in up to 75% of cases

Spagnolo P, J Investig Allergol Clin Immunol 2015; Vol. 25(4): 237-250

HP Triad histology

Histological diagnosis of hypersensitivity pneumonia is predicated on recognition of a classical triad:

1)bronchiolocentric cellular chronic interstitial pneumonia,

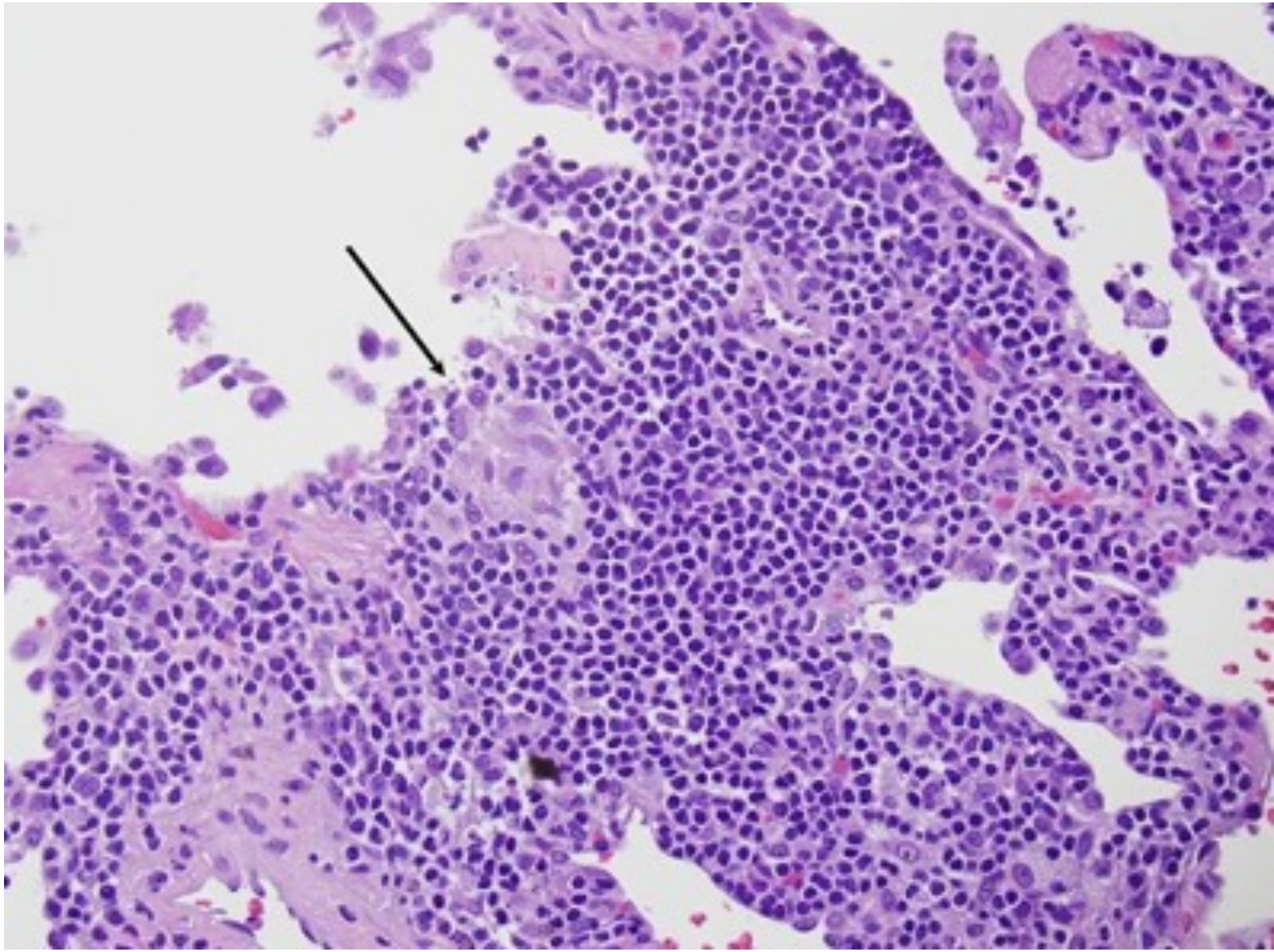
2)chronic bronchiolitis, and

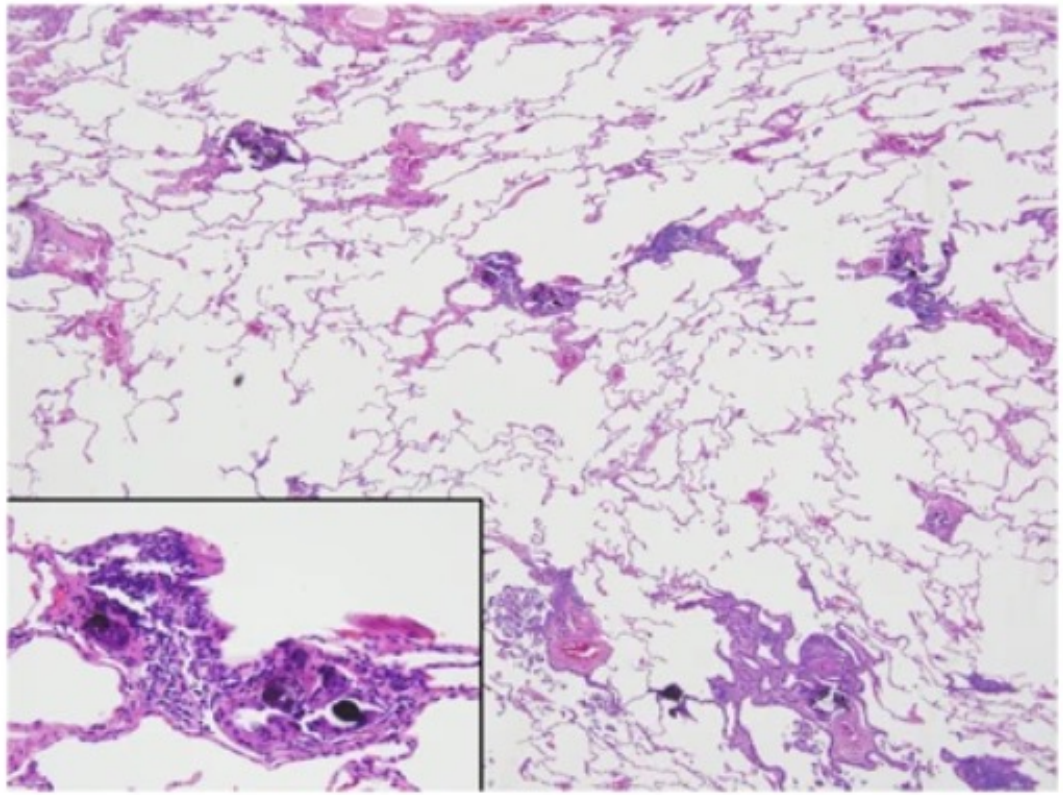
3)non-necrotizing granulomatous inflammation affecting the peribronchiolar interstitium

HP

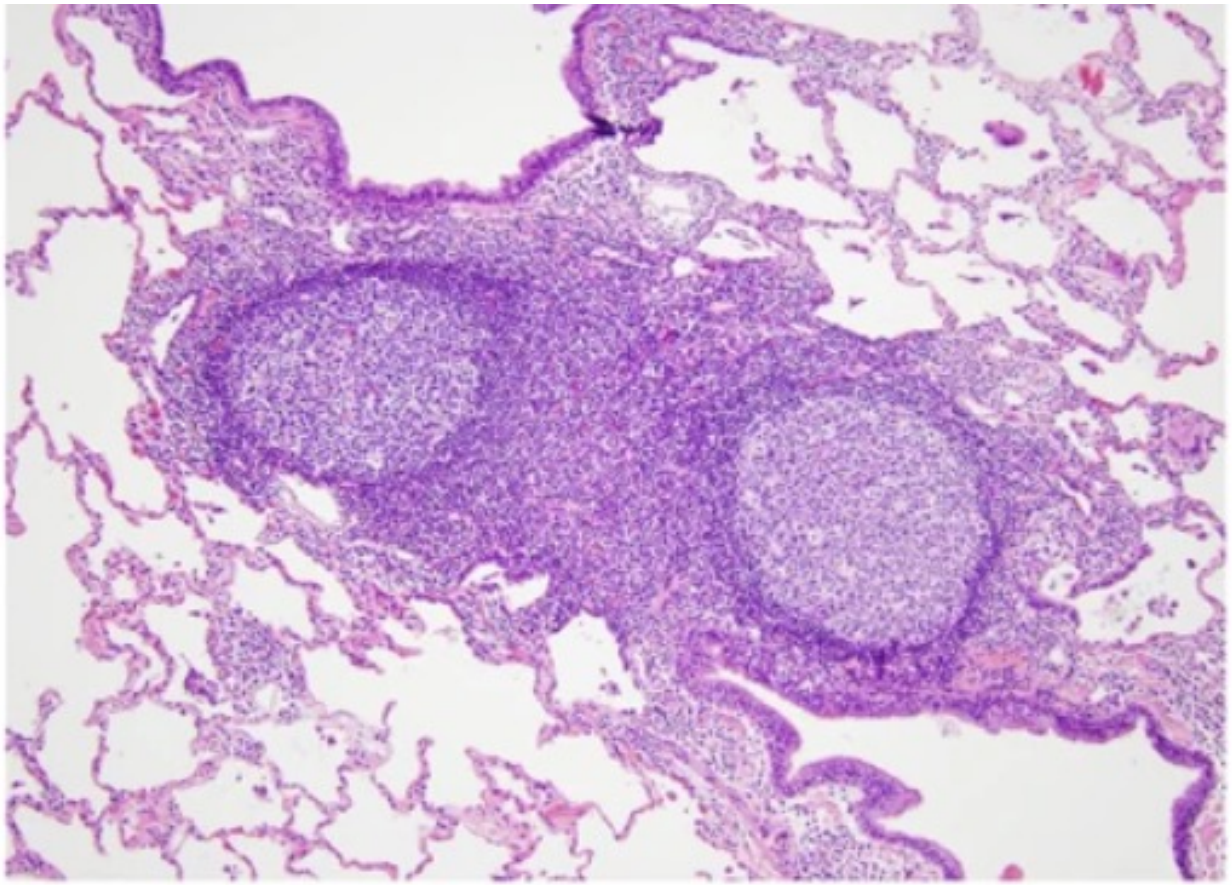
Showing a lymphocyte-rich interstitial infiltrate.

A single cluster of loosely organized epithelioid histiocytes (arrow) is present, resulting in a vaguely granulomatous appearance.



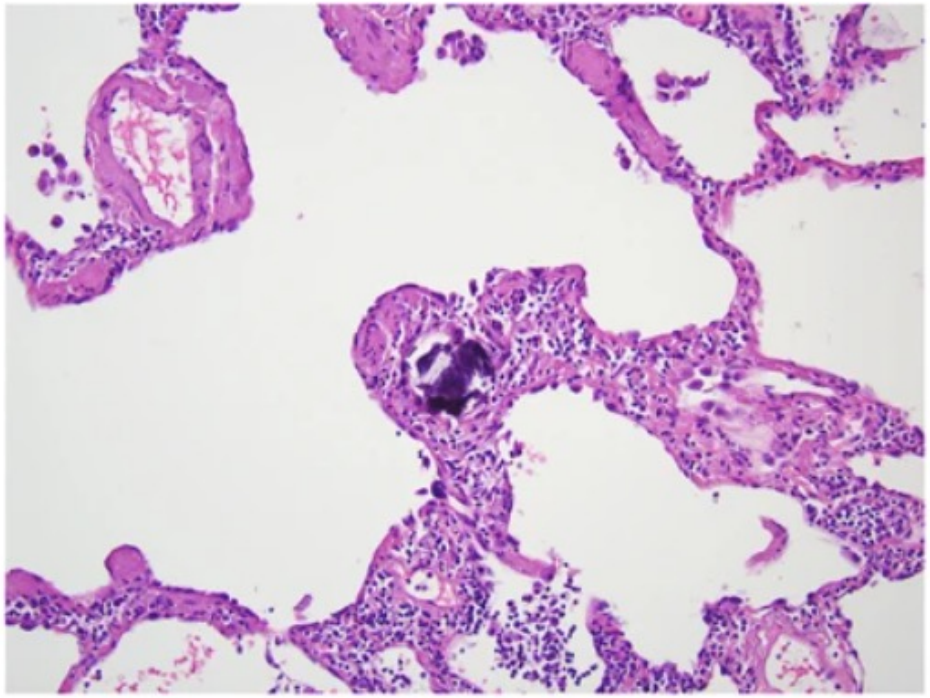


Low-magnification photomicrograph (hematoxylin and eosin; × 40) of surgical lung biopsy from patient with hypersensitivity pneumonia resulting from exposure to a pet bird. The biopsy shows only chronic bronchiolitis in which there is an exquisitely patchy, airway-centered interstitial infiltrate of lymphocytes, with associated multinucleated giants cells. Many of the giant cells contain calcified Schaumann bodies (inset; × 200), a common but nonspecific finding that serves as a helpful diagnostic clue.



Intermediate-magnification photomicrograph (hematoxylin and eosin; × 100) demonstrating the peribronchiolar lymphoid aggregates with germinal centers in surgical lung biopsy from patient with hypersensitivity pneumonia.

Separating chronic HP from UIP



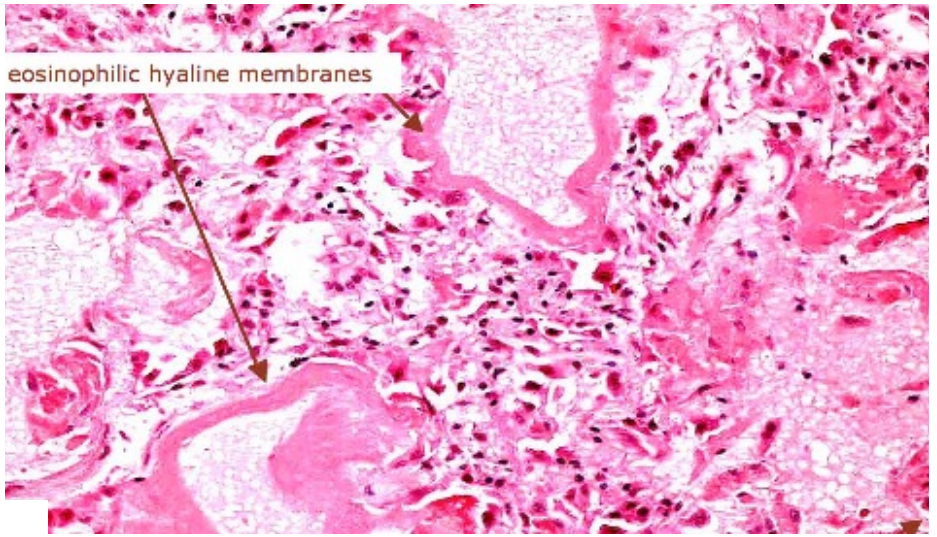
Lymphocytic bronchiolitis with granulomatous features is key to distinguishing the two

Higher-magnification photomicrograph (hematoxylin and eosin; × 200) from central area of field illustrated in Figure 10, showing peribronchiolar lymphocytic infiltrate ('bronchiolitis') with a single multinucleated giant cell largely obscured by cytoplasmic calcifications. The presence of a lymphocytic bronchiolitis with granulomatous features typical of hypersensitivity pneumonia is the key in separating usual interstitial pneumonia/idiopathic pulmonary fibrosis (UIP/IPF) from late, fibrotic-stage hypersensitivity pneumonia.

Q

What am I?.

Shows diffuse alveolar damage, a key feature of which is hyaline membranes in the alveoli.

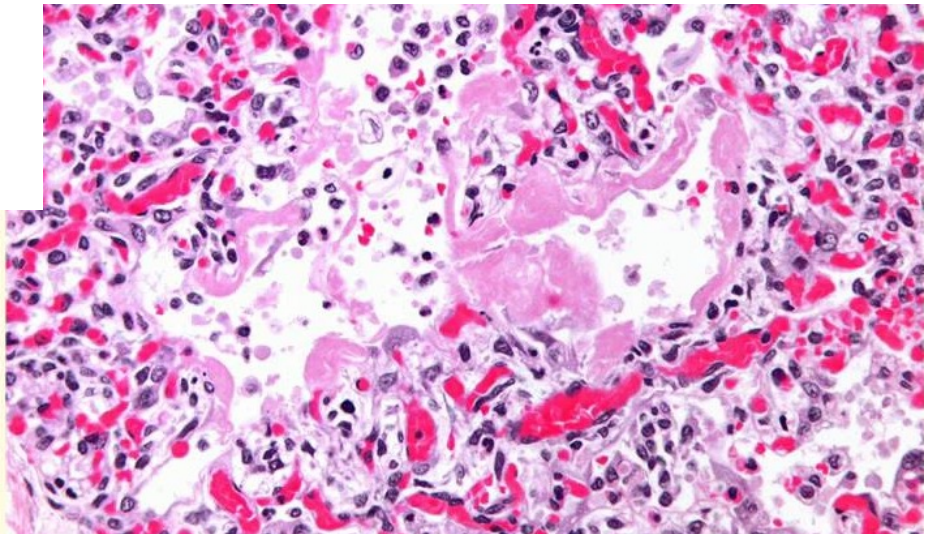


A previously fit 40-year-old plumber presented with a 7-day history of flu-like symptoms and worsening dyspnoea. A chest X-ray showed bilateral infiltrates, but all cultures and serological investigations were negative or normal. He rapidly developed type I respiratory failure, requiring mechanical ventilation, but died of multiple organ failure 10 days later despite being treated with antibiotics, antivirals and high-dose corticosteroids.

What histological abnormality is most likely to be found in the lung at autopsy?

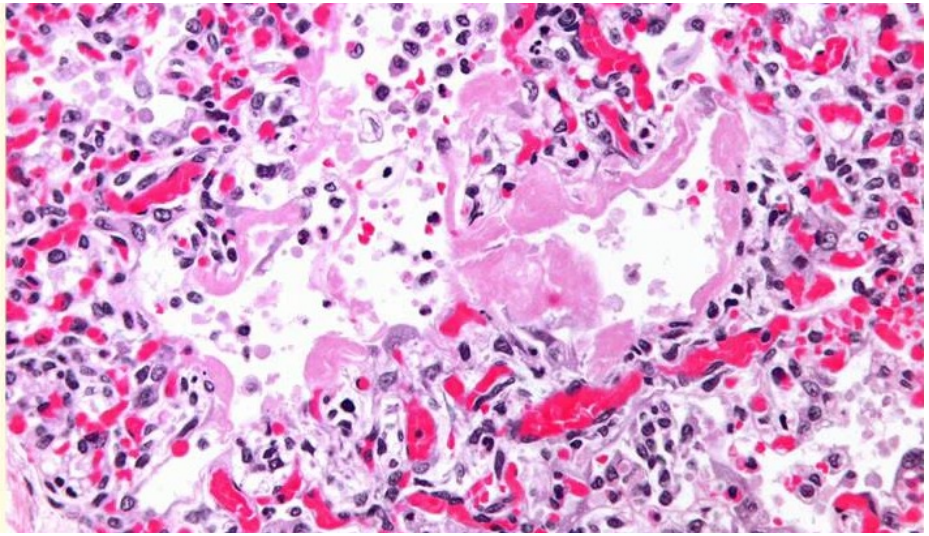
Answers

A: desquamative interstitial pneumonia
B: giant cell pneumonia
C: hyaline membranes
D: neutrophilic alveolitis
E: obliterative bronchiolitis



This is acute interstitial pneumonia. The pathology is diffuse alveolar damage, a key feature of which is hyaline membranes in the alveoli

acute interstitial pneumonia

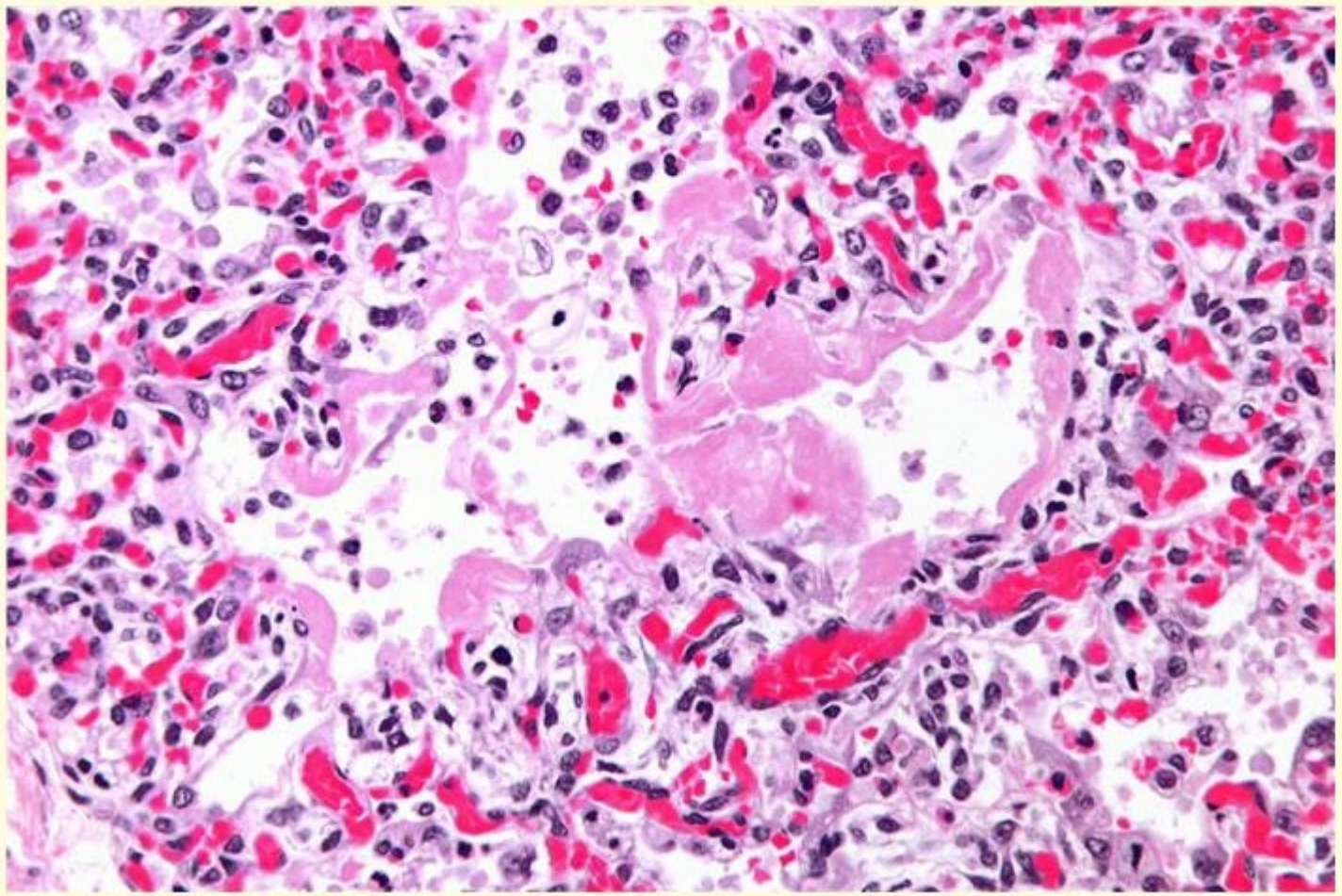


- cause for AIP is not known.
- Idiopathic form of ADRS
- Viral-type illness followed by rapid onset OSB, widespread crackles. CXR bilarteral shadowing,
- Treatment is primarily supportive. Management in an intensive care unit is required and the need for mechanical ventilation is common. Therapy with corticosteroids is generally attempted, though their usefulness has not been established. The only treatment that has met with success to date is a lung transplant
- 60%of people with acute interstitial pneumonitis will die in the first six months of illness

Histology

- Shows diffuse alveolar damage, a key feature of which is hyaline membranes in the alveoli
- oedema, interstitial inflammation, alveolar septal thickening.
- Can progress to cyst and honeycombing

Acute interstitial pneumonitis



BAL

- Bronchoalveolar lavage (BAL) typically demonstrates a lymphocytosis >50% with a predominance of CD8+ T cells CD4:CD8 ratio is low in which condition?

HP

HP

Diagnosis: Bronchoalveolar Lavage

The most sensitive tool to detect alveolitis in patients suspected of having HP

The total cell yield is usually very high, more than 20 million from a BAL of 100 mL total instillation

The most typical pattern is a marked lymphocyte-rich alveolitis (>20% and often >50% of the total cells recovered)

Lymphocyte count is usually higher than 50% in subacute HP and accompanied by an increase of CD8+ T cells

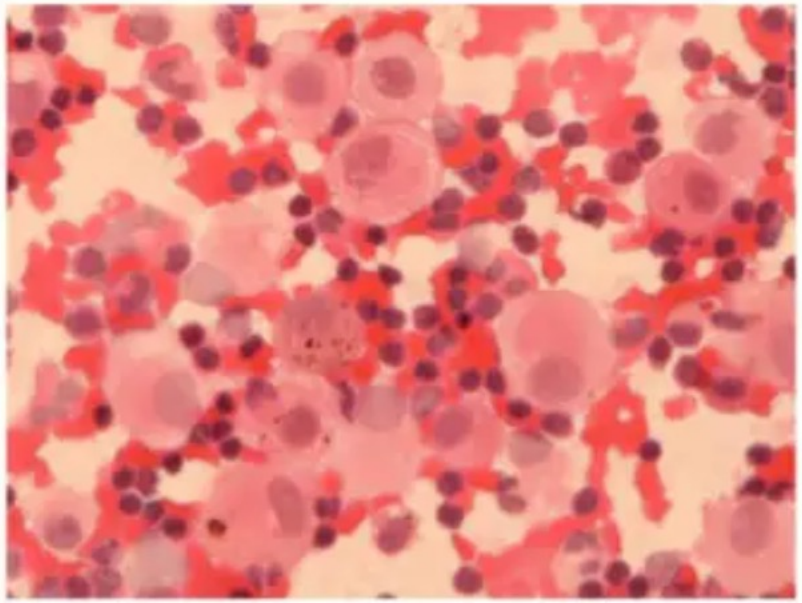


Figure 8. Bronchoalveolar lavage fluid showing some foamy macrophages and lymphocytic alveolitis (hematoxylin-eosin, original magnification x200).

Spagnolo P, J Investig Allergol Clin Immunol 2015; Vol. 25(4): 237-250

Diagnosis: Bronchoalveolar Lavage

- The presence of mast cells, plasma cells, and foamy macrophages in the BAL are additional features in support of a diagnosis of HP
- A cutoff level of 30% for lymphocytes confidently differentiates chronic HP from IPF

Spagnolo P, J Investig Allergol Clin Immunol 2015; Vol. 25(4): 237-250

Histology found to have pigmented macrophages with particular focus around the bronchi.

- RB-ILD
- Sarcoid
- DIP
- Smoking related ILD
- LCH

Histology found to have pigmented macrophages with particular focus around the bronchi.

- RB-ILD
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Stellyte cells are typical histo of LCH