Lung Diseases They Didn't Teach You About In Medical School

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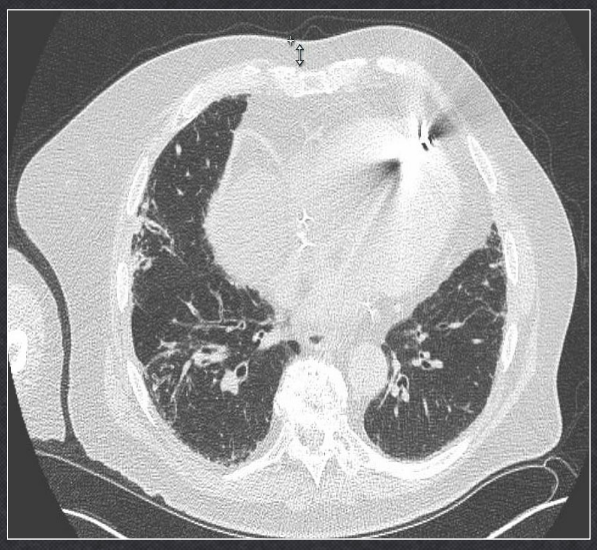
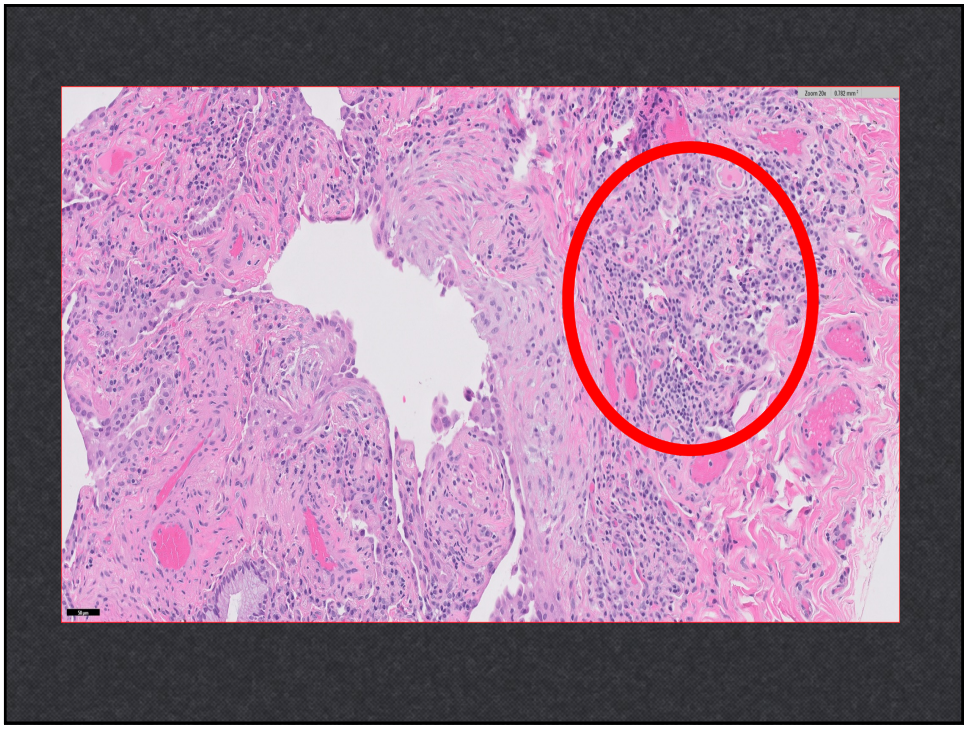
Case #1

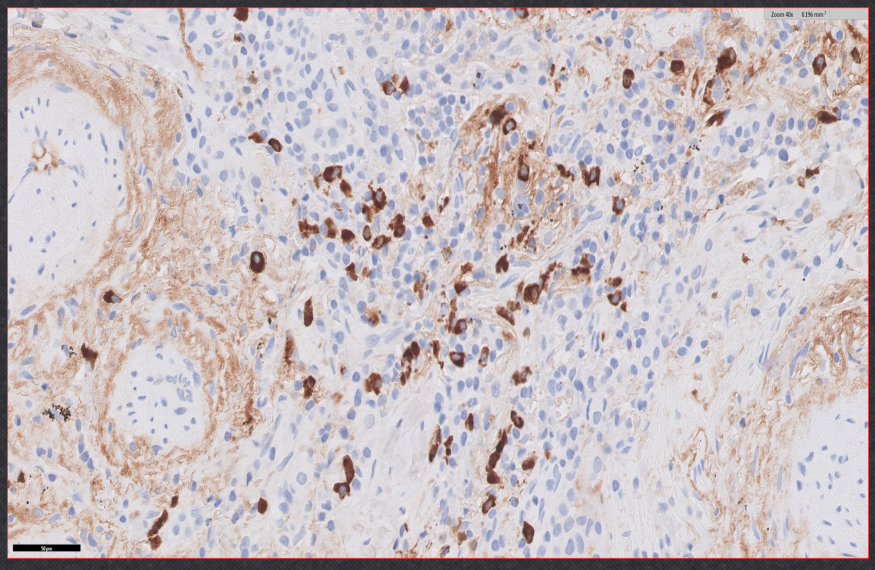
- 76 year old man
- Dyspnea and cough for 5 years
- Past medical history: chronic pancreatitis
- Social history: former smoker
- Exam: bibasilar dry crackles

Case #1 (continued)

- Sed rate > 130
- C-reactive protein elevated at 13.9
- Atypical ANCA positive at 1:80
- SS-A antibody positive
- Eosinophil count elevated at 600
- Serum IgG4 749.0 (121 the upper limit of normal)







IgG4 Stain

Diagnosis: IgG4 Disease

IgG4 Disease: Overview

- Disease did not appear in medical publications until 2003
- Multi-organ immune related condition
- Mimics many malignant, infectious, and inflammatory disorders

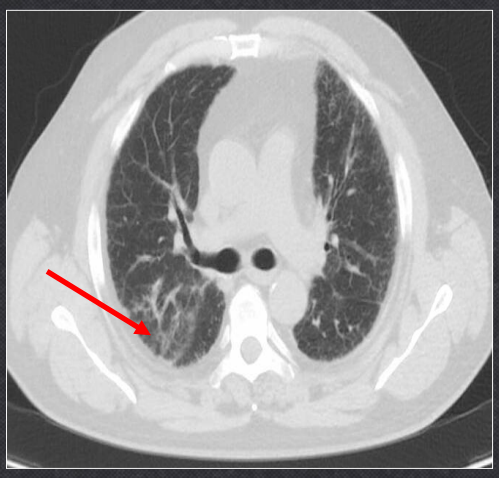
IgG4 Disease: Organs Affected

- Autoimmune pancreatitis
- Salivary/parotid gland enlargement
- Thyroiditis
- Sclerosing cholangitis
- Lymphadenopathy
- Retroperitoneal fibrosis
- Aortitis
- Tubulointerstitial nephritis
- Skin rash
- Interstitial lung disease

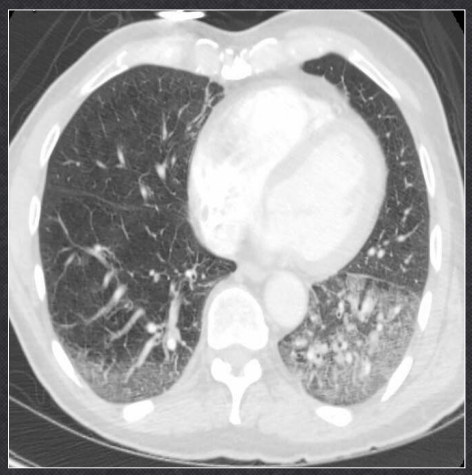
IgG4 Disease: Pulmonary Manifestations

- Parenchymal
  - Nodules or masses
  - Ground glass infiltrates
  - Interstitial infiltrates
- Mediastinal adenopathy
- Bronchostenosis
- Pleural effusions

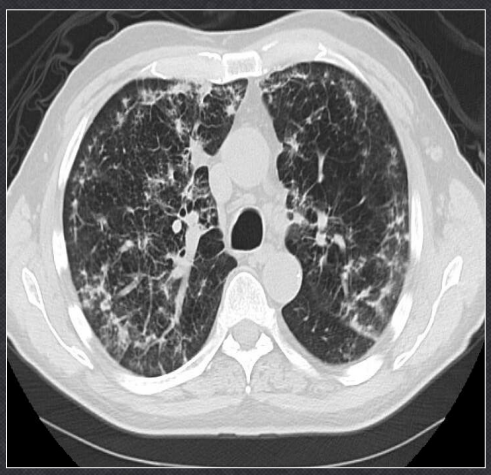
IgG4 Disease Presenting as Interstitial Lung Disease



IgG4 Disease Presenting with Consolidations



IgG4 Disease Presenting as Cryptogenic Organizing Pneumonia



IgG4 Disease: Diagnosis

- Elevated serum IgG4
- Tissue biopsy
  - Dense lymphoplasmacytic infiltrates & storiform fibrosis
  - Increased IgG4-staining plasma cells
  - Increased eosinophils

IgG4 Disease: Treatment

- Corticosteroids
- Mycophenolate
- Azathioprine
- Rituximab

Key Points About IgG4 Disease

- Recently recognized multi-organ immune related condition
- Mimics malignant, infectious, and inflammatory disorders
- Diagnosed by an elevated serum IgG4 and biopsy
- Treatment is immunosuppression

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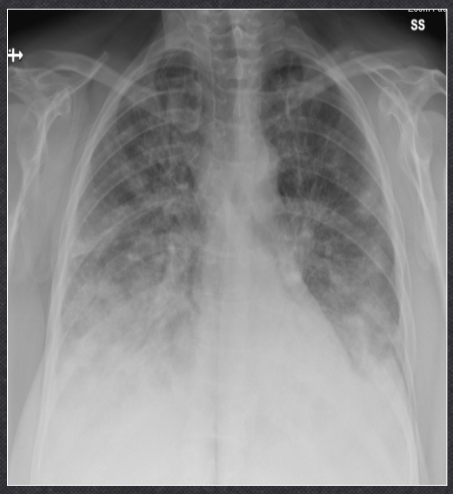
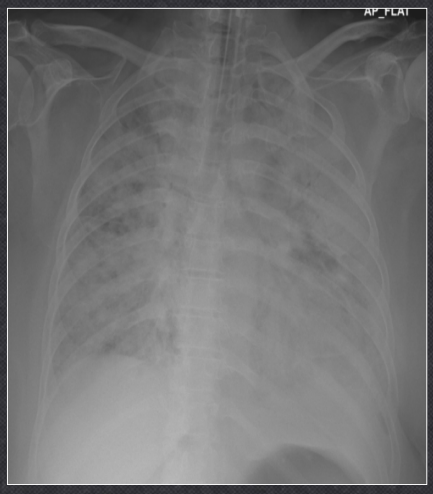
Case #2

- 59-year old woman
- Unremarkable past medical history
- 3-day history of cough and progressive dyspnea
- No improvement with outpatient antibiotic
- Exam: mild bibasilar crackles
- Lab: WBC = 16.3 with 1,200 eosinophils

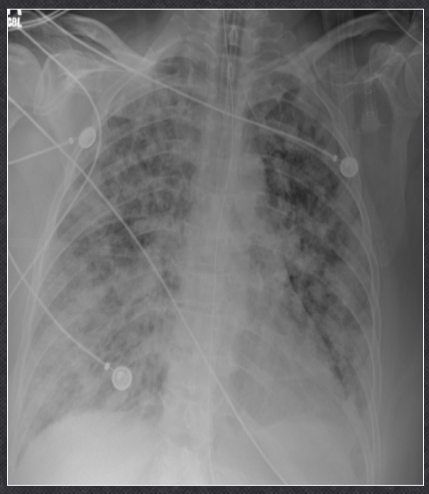
January 21, 2019 – emergency department



January 22, 2019 – intensive care unit



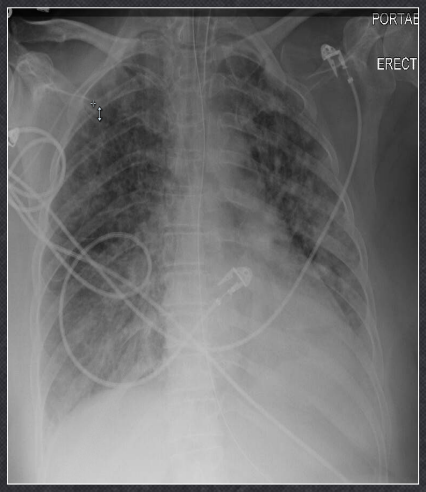
January 23, 2019 – transferred to Ohio State



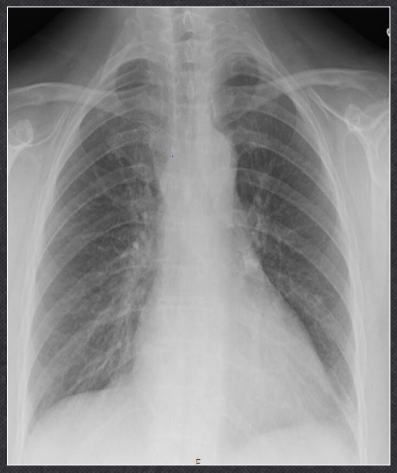
Bronchoalveolar lavage

- 10% Alveolar macrophages
- 2% Neutrophils
- 3% Lymphocytes
- 85% Eosinophils

January 24, 2019



March 1, 2019



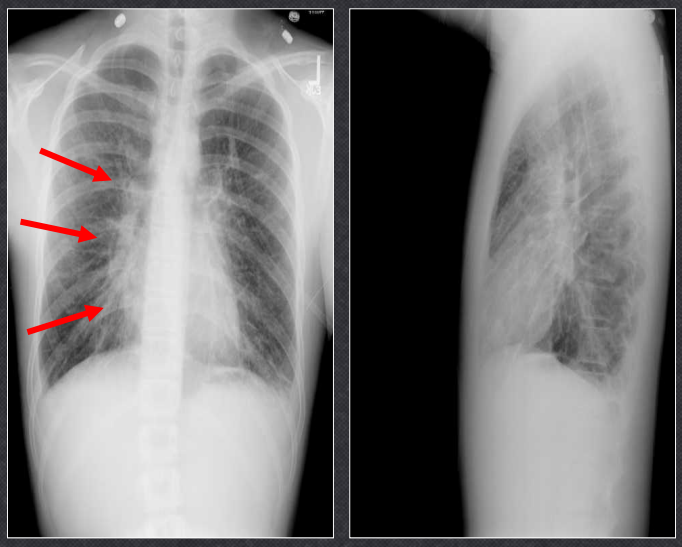
Acute Eosinophilic pneumonia

- Presentation:
  - Average symptoms 4 days
  - Average age 29 yrs
  - Symptoms:
    - Cough 100%
    - Dyspnea 95%
    - Chest pain 73%
    - Myalgias 50%
  - 40% "beginner" smokers
- Exam:
  - Average temperature 101° F
  - Average respiratory rate 32/min
  - Crackles in 80%

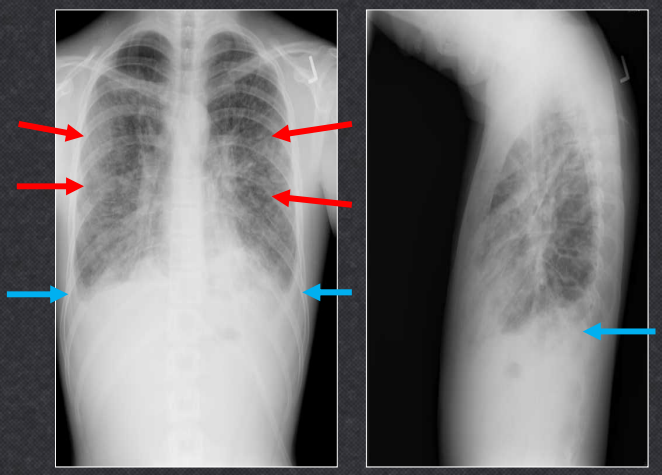
Acute Eosinophilic pneumonia

- Chest X-ray:
  1. Kerley B lines
  2. Interstitial infiltrates
  3. Alveolar infiltrates
  4. Pleural effusions
- Lab:
  - Average WBC 17,000
    - Blood eosinophils may not be elevated
  - Average PO_2 = 57 mm

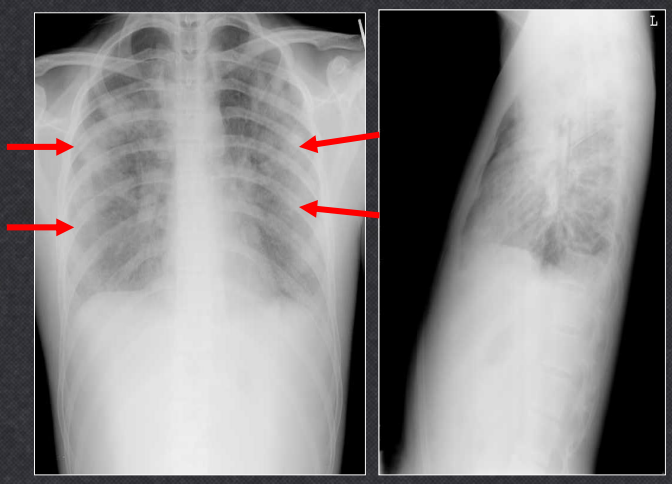
Acute Eosinophilic Pneumonia Day #1



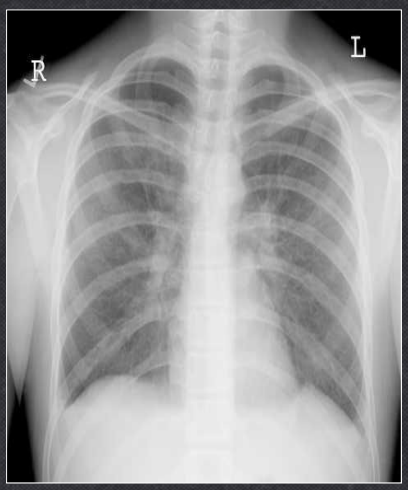
Acute Eosinophilic Pneumonia Day #2



Acute Eosinophilic Pneumonia Day #3

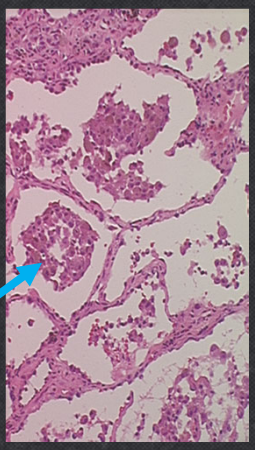


Acute Eosinophilic Pneumonia Day #7



Acute Eosinophilic pneumonia

- Typical BAL:
  - 37% eosinophils
  - 20% lymphocytes
  - 15% neutrophils
  - 28% macrophages
- Lung biopsy:
  - Intra-alveolar eosinophils



Acute Eosinophilic Pneumonia Treatment:

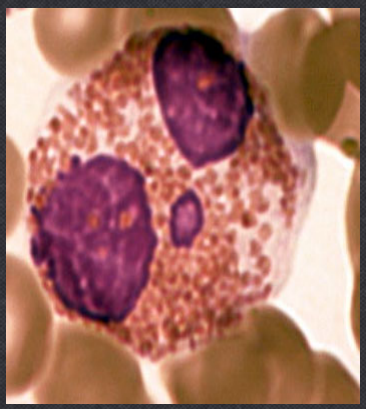
- Initial treatment:
  - With respiratory failure: IV Methylprednisolone 60-125 mg every 6 hours
  - Without respiratory failure: Prednisone 40-60 mg daily
- Subsequent treatment:
  - Prednisone 40-60 mg daily - taper over 2-4 weeks
- Relapses are rare

Acute Eosinophilic Pneumonia: Causes

- Idiopathic
- Cigarette smoking
- Prescription drugs
- Street drugs
- Organic dust inhalation
- Parasites

Key points about acute eosinophilic pneumonia

- It mimics severe pneumonia or ARDS
- Bronchoscopy with BAL is required for diagnosis
- Responds immediately to steroids
- Often associated with beginning cigarette smoking



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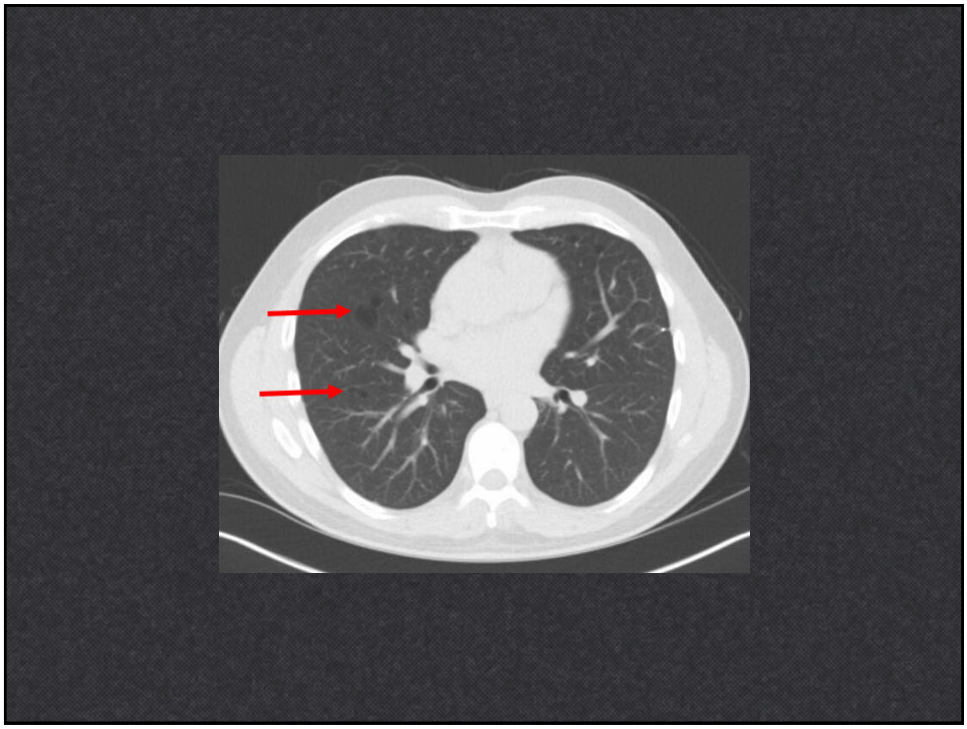
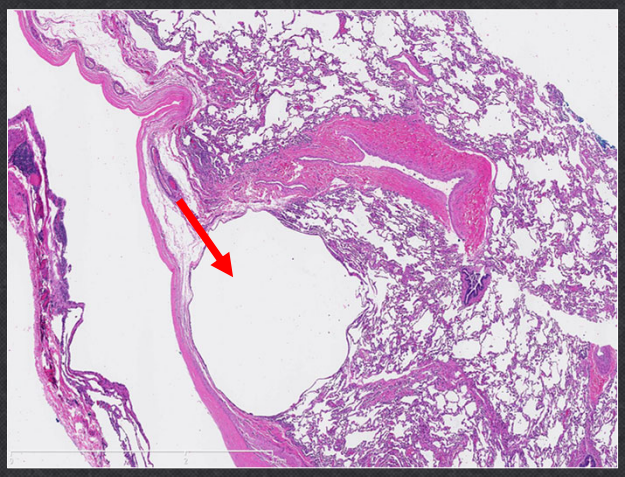
Case #3

- 50 year old man
- Recent spontaneous pneumothorax
- Past medical history: spontaneous pneumothorax 15 years prior
- Family history:
  - Father had spontaneous pneumothorax
  - Brother with kidney cancer
- Exam: normal



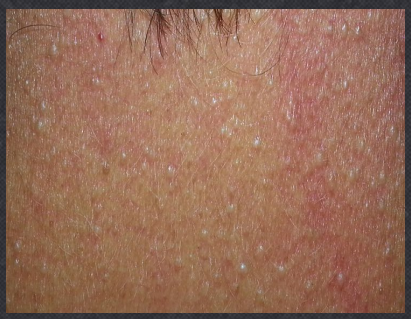
Lung Biopsy at the Time of Surgical Pleurodesis



Diagnosis: Birt-Hogg-Dube Syndrome

Birt-Hogg-Dube Syndrome: Overview

- Autosomal dominant inheritance
- Folliculin (FLCN) gene mutation
- Clinical manifestations
  - Skin fibrofolliculomas
  - Kidney cancer – chromophobe tumors
  - Bilateral pulmonary cysts

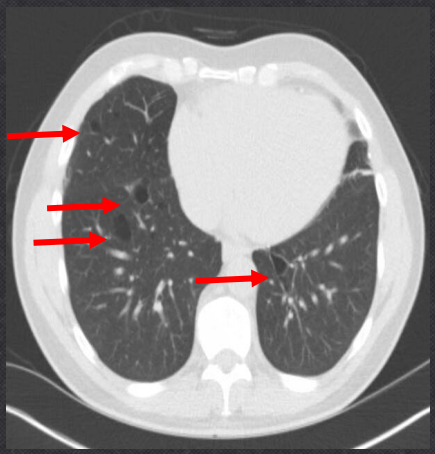


Birt-Hogg Dube Syndrome: Pulmonary Manifestations

- Cystic lung lesions in the 4th to 5th decade of life
- 2 common pulmonary presentations:
  - Incidental pulmonary cystic lesions on chest CT
  - Pneumothorax

Birt-Hogg Dube Syndrome: Radiology

- Basilar predominant pulmonary cystic lesions



Birt-Hogg-Dube Syndrome: Diagnosis

- Pulmonary cysts plus personal or family history of:
  - Pulmonary cysts or pneumothorax
  - Fibrofolliculomas
  - Renal tumors
- Genetic testing for the FLCN gene

Birt-Hogg-Dube Syndrome: Management

- Renal cancer the most threatening manifestation
  - Cancer screening every 1-2 years:
    - Ultrasound
    - CT
    - MRI
- Pulmonary disease
  - Cysts do not usually impair lung function
  - Prevention of pneumothorax recurrence

Key Points About Birt-Hogg-Dube Syndrome

- Syndrome of:
  - Skin fibrofolliculomas
  - Kidney cancer
  - Pulmonary cysts with or without pneumothorax
- Folliculin gene mutation
- Autosomal dominant inheritance

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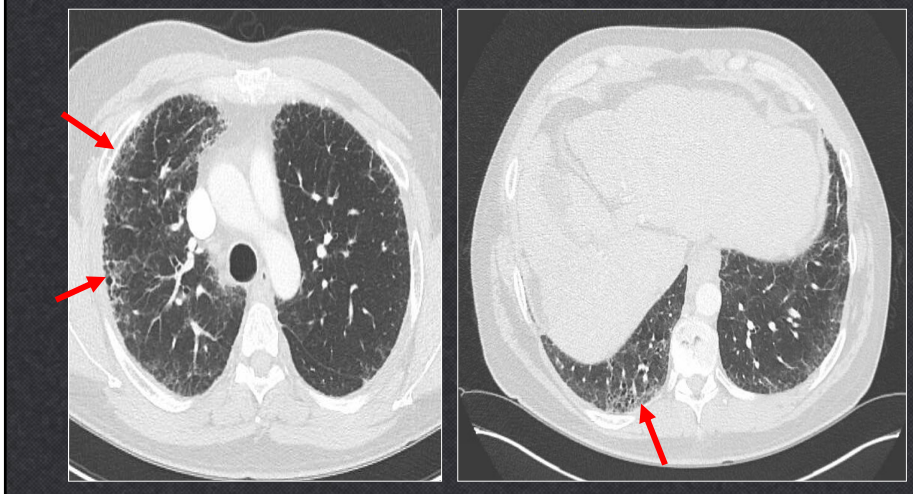
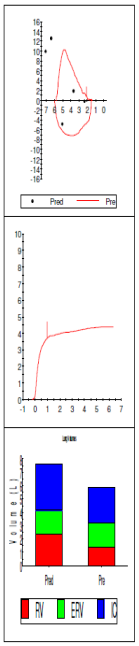
Case #4

- 57 year-old man referred for pulmonary pre-op evaluation
- History of cirrhosis due to NASH
  - Undergoing liver transplant evaluation
- Progressive dyspnea for 6 months
- Hair turned gray at age 16
- Father died of “asbestosis” and cirrhosis
- Exam: bibasilar dry crackles



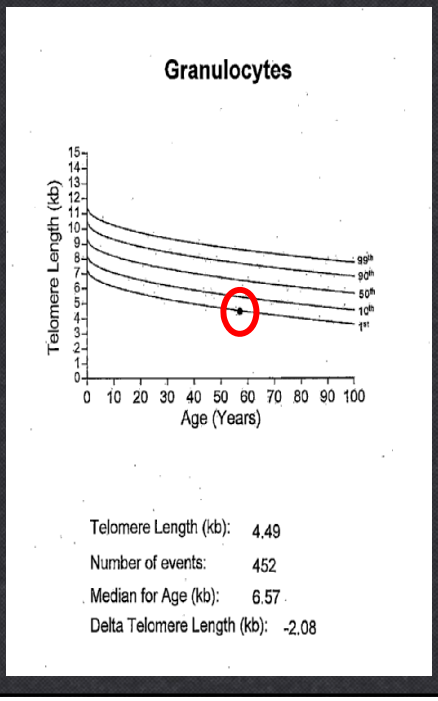
	Pre-Bronch					Post-Bronch		
	Pred	LLN	ULN	Actual	%Pred	Actual	%Pred	%Chng
---- SPIROMETRY ----								
FVC (L)	5.40	4.51	6.29	*4.38	*81			
FEV1 (L)	4.11	3.43	4.79	3.71	90			
FEV1/FVC (%)	76	63	89	85	111			
FEV1/SVC (%)	76	64	89	83	108			
FEF 25-75% (L/sec)	3.41	2.85	3.97	*4.19	*122			
FEF 25% (L/sec)	12.73	10.63	14.83	*10.17	*79			
FEF 50% (L/sec)				5.54				
FEF 75% (L/sec)	1.96	1.64	2.28	*1.42	*72			
FEF Max (L/sec)	10.14	8.47	11.81	10.38	102			
FIF 50% (L/sec)	4.78	3.99	5.57	*7.18	*150			
FEF50%/FIF50% (%)	90-100	79	119	77				
---- LUNG VOLUMES								
TLC (Pleth) (L)	7.64	6.11	9.17	*5.90	*77			
SVC (L)	5.40	4.51	6.29	*4.48	*83			
IC (L)	3.49	2.91	4.07	*2.66	*76			
FRC (Pleth) (L)	4.01	3.21	4.81	*2.99	*74			
ERV (L)	1.74	1.45	2.03	1.57	90			
RV (Pleth) (L)	2.37	1.90	2.84	*1.41	*59			
RV/TLC (Pleth) (%)	32	26	38	*24	*74			
Vpant (L)								
---- DIFFUSION ----								
DLCOunc (ml/min/mm	30.34	24.27	36.41	*18.16	*59			
DLCOcor (ml/min/mm	30.34	24.27	36.41	*19.08	*62			
DL/VA (ml/min/mmHg/	4.13	3.30	4.96	*3.11	*75			
VA (L)	7.45	6.22	8.68	*6.14	*82			
IVC (L)				4.60				
BHT (sec)								



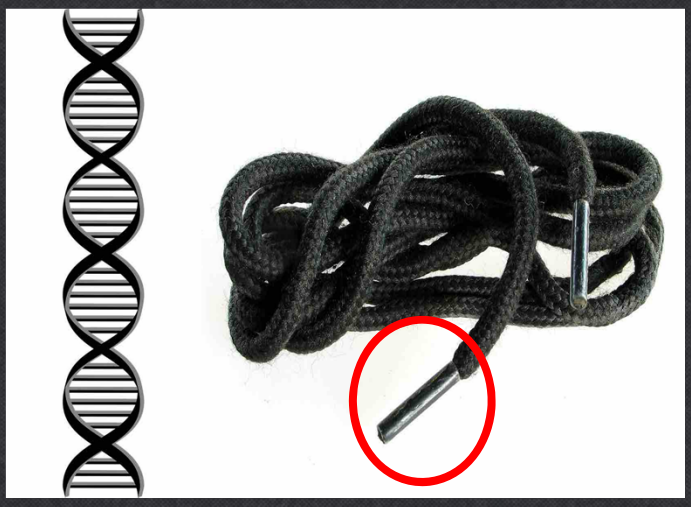
Idiopathic pulmonary fibrosis (IPF)

- 85% are sporadic
- 15% are familial/inherited
  - Family members may have been diagnosed with “asbestosis”, “black lung”, or “lung scarring”
  - Many genes are involved
    - MUC5B gene
    - Telomerase genes
- Sporadic and familial idiopathic pulmonary fibrosis are treated the same

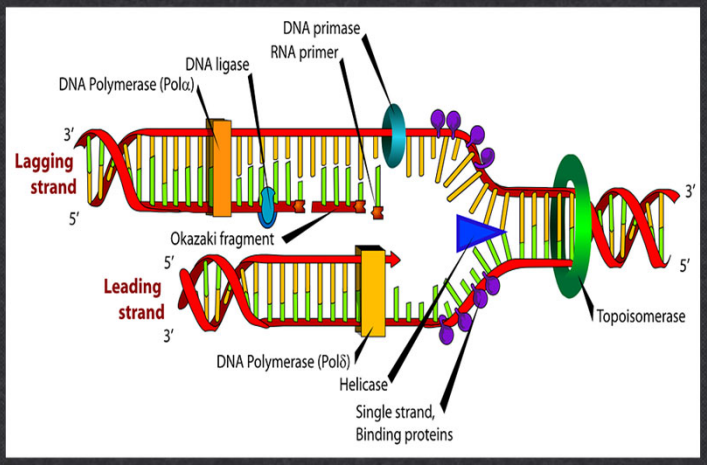
Telomere Length Testing



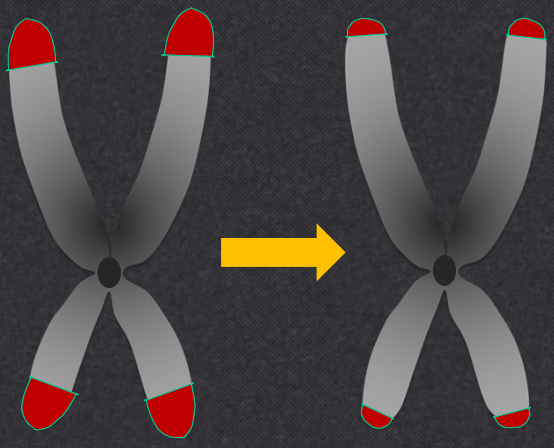
Telomeres



50 - 100 base pairs of telomere DNA is lost at the end of the lagging strand with every replication



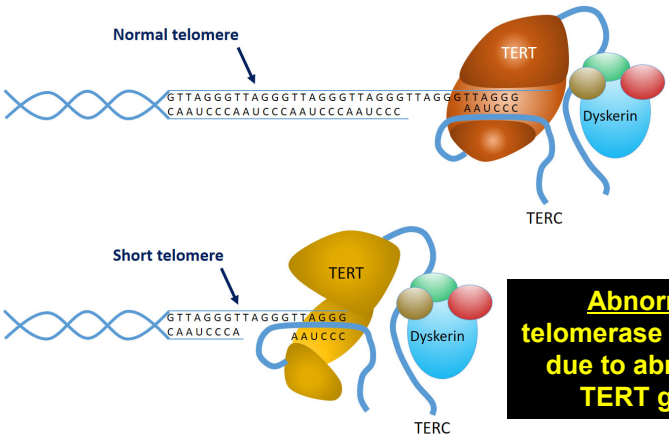
Telomeres
Repeating sequences of TTAGGG



11 Kilobases At Birth 4 Kilobases At Age 80

Telomeres are the body's chromosomal clocks

Telomerase replenish telomeres at the end of DNA



Normal telomerase complex

<u>Abnormal</u> telomerase complex due to abnormal TERT gene

Short telomere syndromes

- Familial idiopathic pulmonary fibrosis
- Premature graying of the hair (before age 30)
- Cryptogenic cirrhosis
- Aplastic anemia
- Myelodysplasia

Short Telomere Syndrome Key Points

- Presentations:
  - Idiopathic pulmonary fibrosis
  - Cirrhosis
  - Myelodysplasia
- A good family history is essential
- Ask the patient when their hair turned gray
- Telomere length testing is supportive

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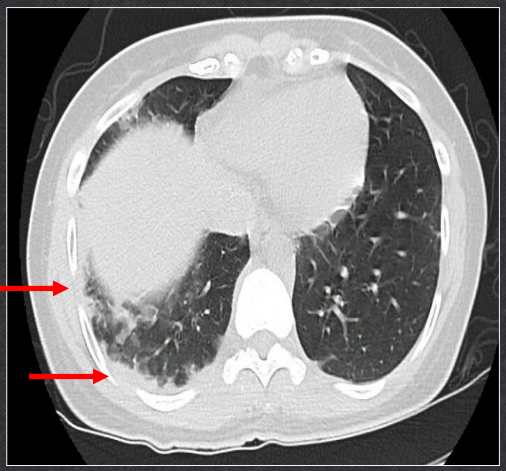
Case #5

- Previously healthy 29 year old woman
- 2 months of progressive dyspnea
- No improvement despite multiple antibiotics for pneumonia
- Social history: never smoker
- Review of systems notable for Raynaud's phenomenon

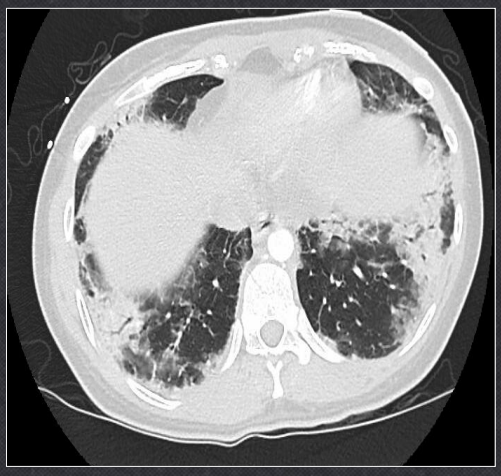
Case #5 (continued)

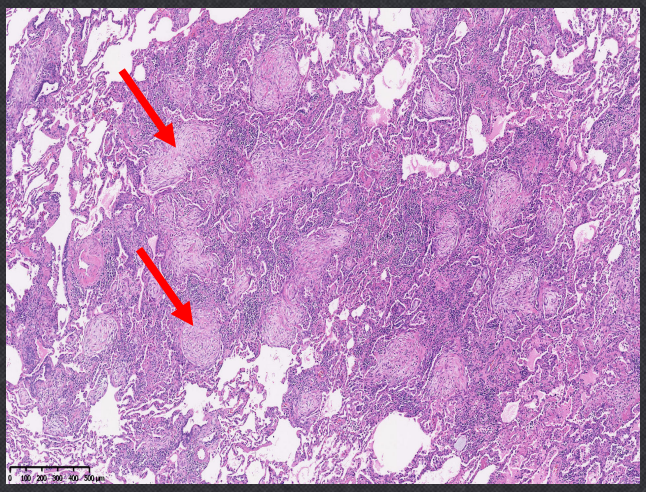
- Exam:
  - Bilateral diffuse crackles
  - Mild mechanic's hands
- Labs:
  - Elevated aldolase 15.1 (upper limit 8.1)
  - SS-A & CCP antibody positive
  - Anti-Jo1 antibody positive

Chest CT



Chest CT 6 Weeks Later

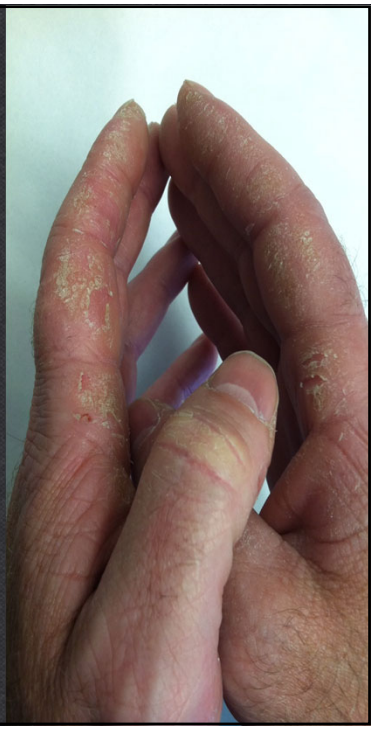




Diagnosis: Organizing Pneumonia due to an Idiopathic Inflammatory Myopathy

Idiopathic Inflammatory Myopathy: Overview

- Polymyositis, dermatomyositis, and inclusion body myositis
- Muscular signs and symptoms
- Extramuscular signs and symptoms
  - Constitutional
  - Dermatological signs
  - Raynaud's
  - Arthralgias
  - Pulmonary



Idiopathic Inflammatory Myopathy: Pulmonary Manifestations

- Prevalence may approach 65%
- Most significant extramuscular contributor to morbidity
- Interstitial lung disease is the hallmark
  - May precede muscular signs in up to 20% of cases

Rapidly Progressive Interstitial Lung Disease

- Acute interstitial pneumonia progressing over several weeks or months
  - Strongly suggestive of an idiopathic inflammatory myositis
- Associated with anti-MDA5 (melanoma differentiation associated protein 5) antibody

Idiopathic Inflammatory Myopathy: Radiology & Pathology

- Radiology
  - Common: linear opacities, ground-glass opacities, reticulation
    - Organizing pneumonia and non-specific interstitial pneumonia patterns (NSIP)
  - Less frequent: honeycombing
    - Usual interstitial pneumonia
- Pathology
  - Organizing pneumonia
  - NSIP

Idiopathic Inflammatory Myopathy: Diagnosis

- Clinical history & exam
- Serological testing
  - Creatine kinase, aldolase, myositis-specific antibodies (i.e. anti-Jo1, anti-MDA5, etc.)
- Exclusion of infection
- Compatible chest CT
- +/- lung biopsy

Idiopathic Inflammatory Myopathy: Management

- Corticosteroids
- Mycophenolate
- Azathioprine

Key Points About Idiopathic Inflammatory Myopathy

- Muscular and extramuscular signs and symptoms
  - Pulmonary disease the primary extramuscular contributor to morbidity
- Diagnosed through clinical history, serology, radiology, +/- pathology
  - Organizing pneumonia the most common radiology and pathology
- Rapidly progressive pulmonary form associated with anti-MDA5 antibody
- Treatment is immunosuppression

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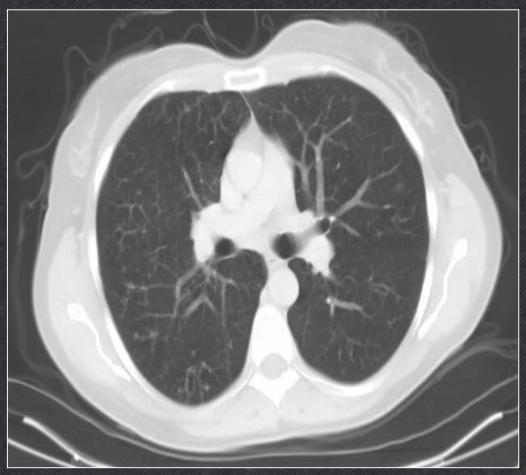
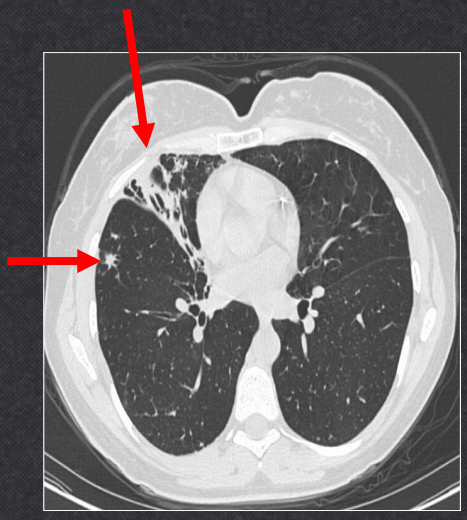
Case #6

- 42 year-old woman with 14 year history of recurrent pneumonia
- Normal sweat chloride, IgG, IgE, alpha-1-antitrypsin
- 2004 BAL = negative AFB culture
- 2006 BAL = Mycobacterium avium complex & Mycobacterium abscessus
- 2008: completed 18 months of antibiotics
- 2015: recurrent sputum production and pneumonias

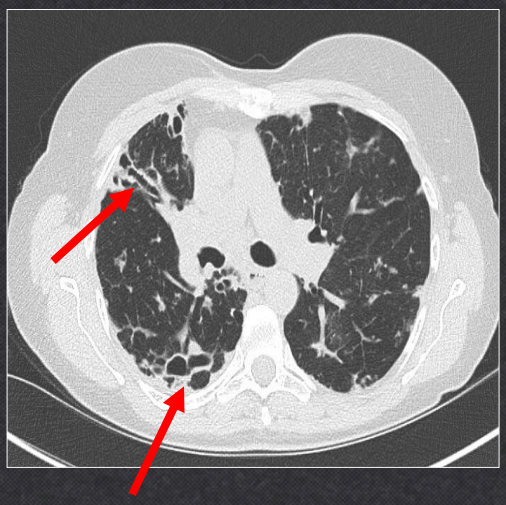
2003



2007

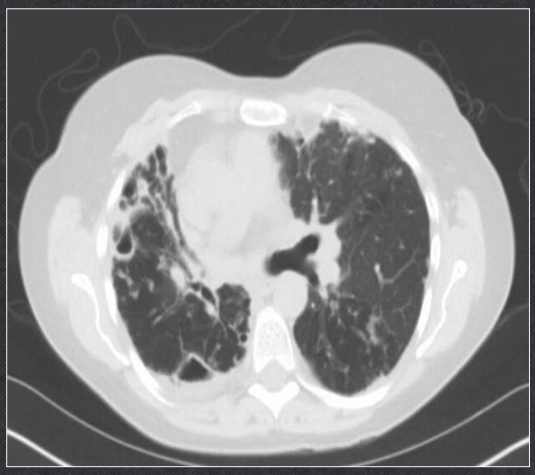


2015



Repeat BAL: Scedosporium

2018

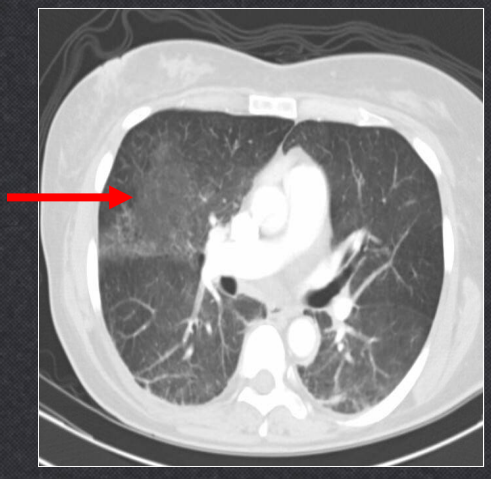


Typical clinical presentations

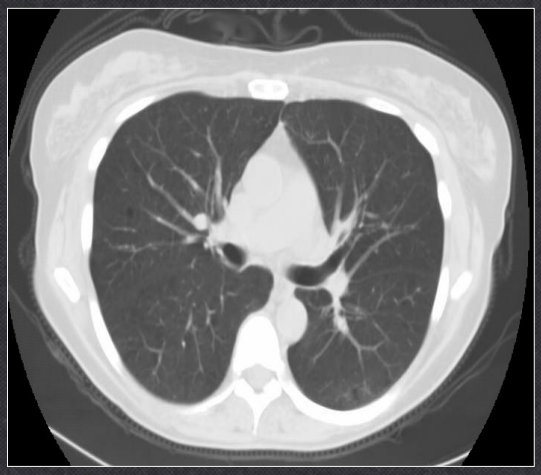
- Ophthalmologic
- Brain abscess
- Skin and soft tissues
- Pulmonary infections

- Patients are often immunosuppressed
- Chronic pneumonia in immunocompetent patients can occur
- Diagnosis is by fungal culture
- Treatment:
  - Voriconazole
  - Surgical debridement

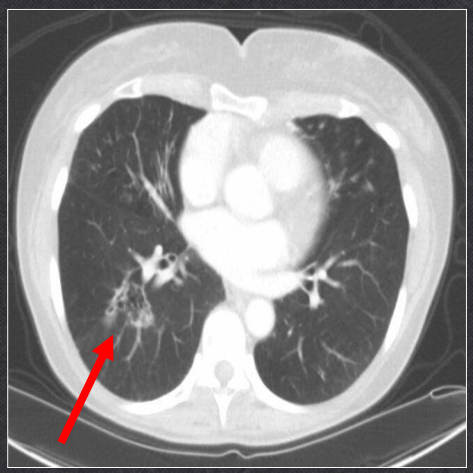
January 5, 2007



January 31, 2007

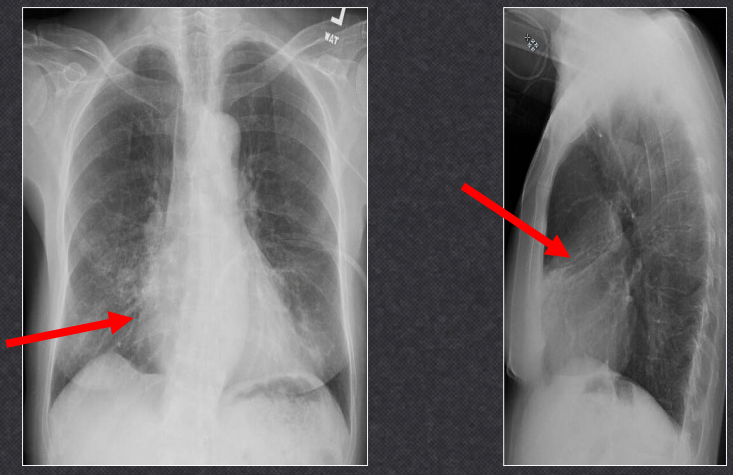


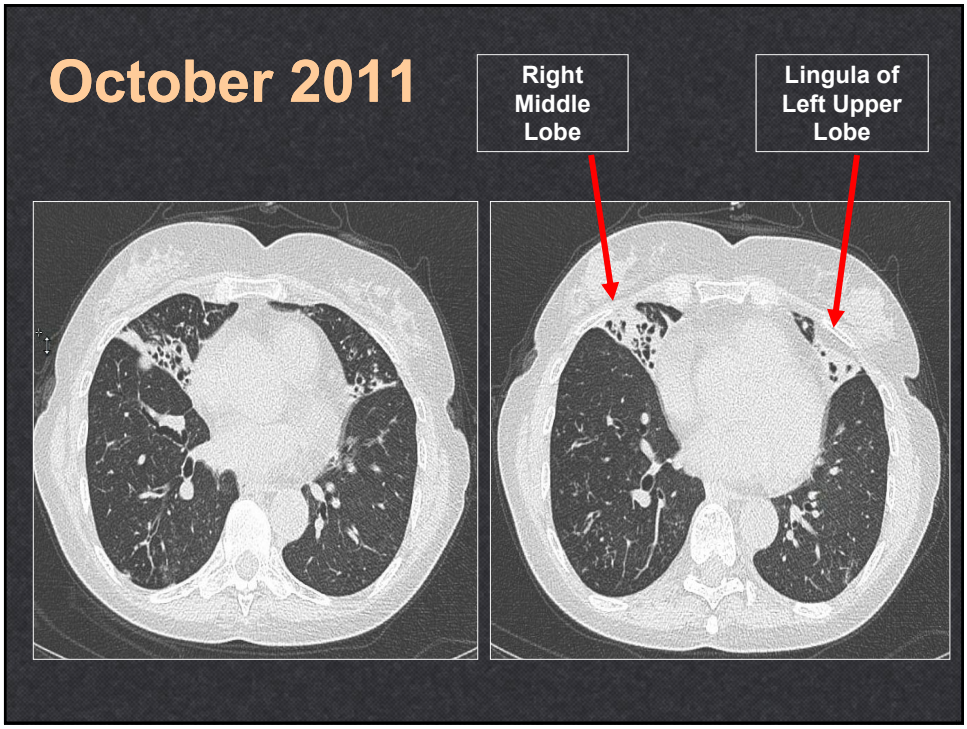
2019

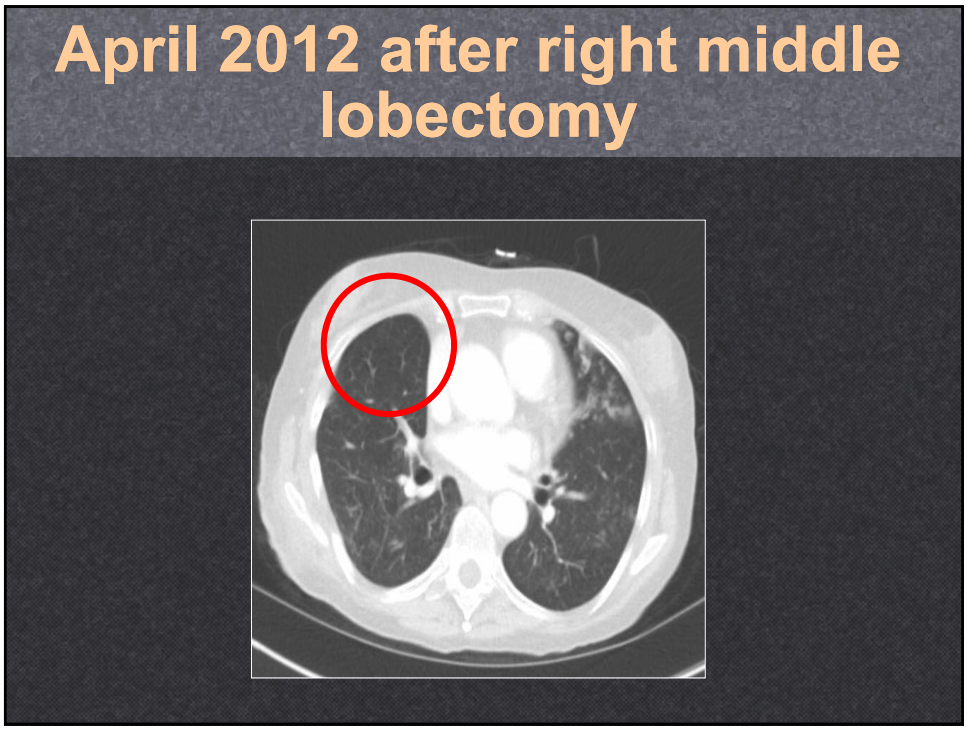


Sometimes surgery is the only effective cure

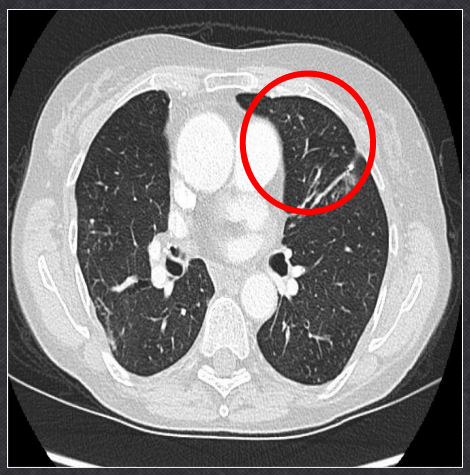
October 2011




October 2011
Right Middle Lobe
Lingula of Left Upper Lobe


April 2012 after right middle lobectomy

2018 after lingula segmentectomy



Key Points About Scedosporium

- Patients often are immunosuppressed
- But… chronic pulmonary infection can occur in normal patients and those with underlying bronchiectasis
- Anti-fungal antibiotics are often ineffective
- Surgery is often necessary

Lung Diseases They Didn't Teach You About In Medical School

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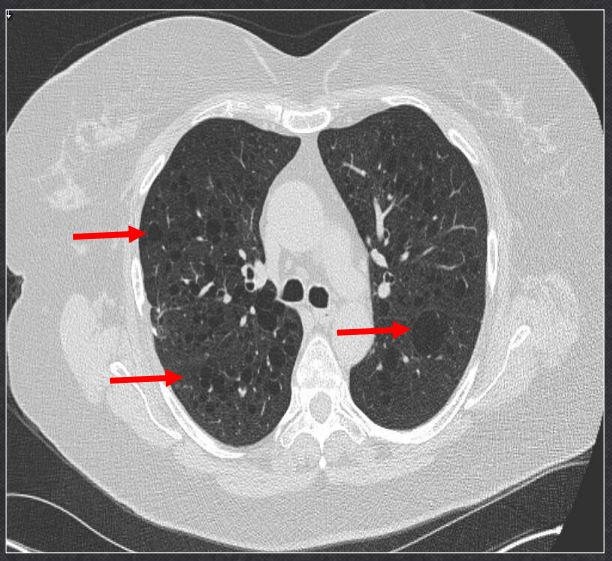
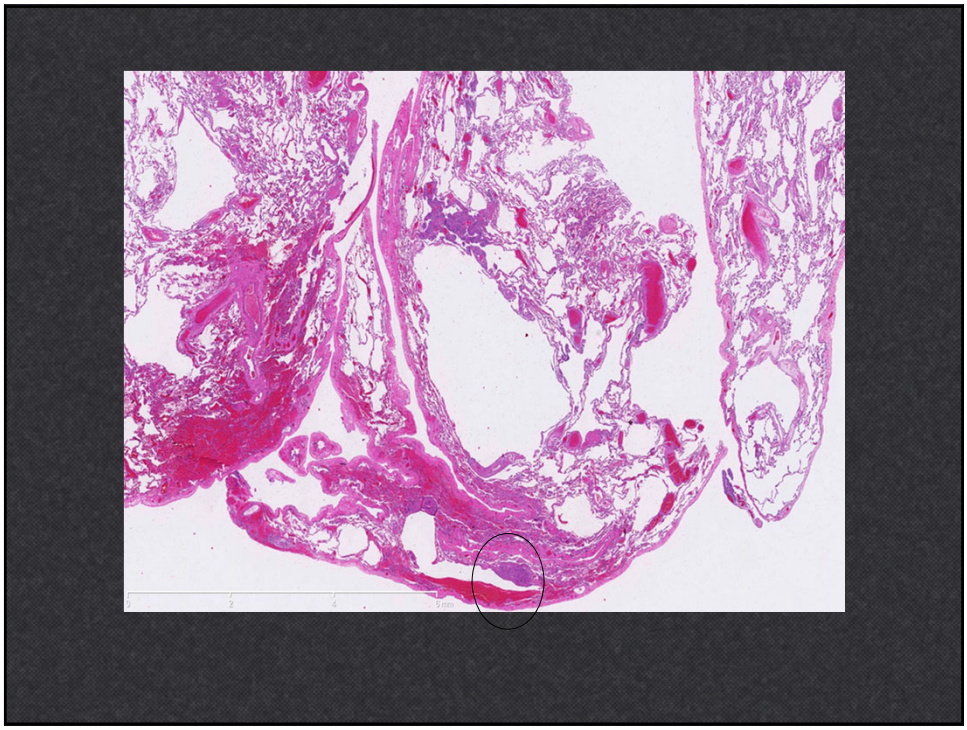
Case #7

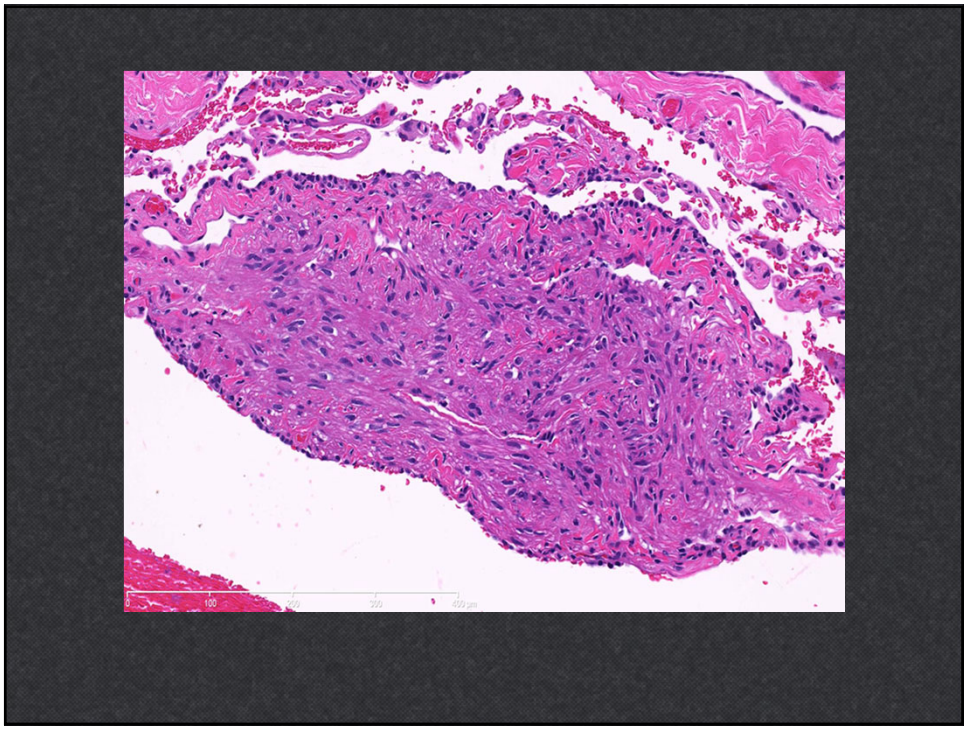
- Previously healthy 39 year old woman
- Progressive shortness for several months
- Social history: never smoker
- Exam: normal

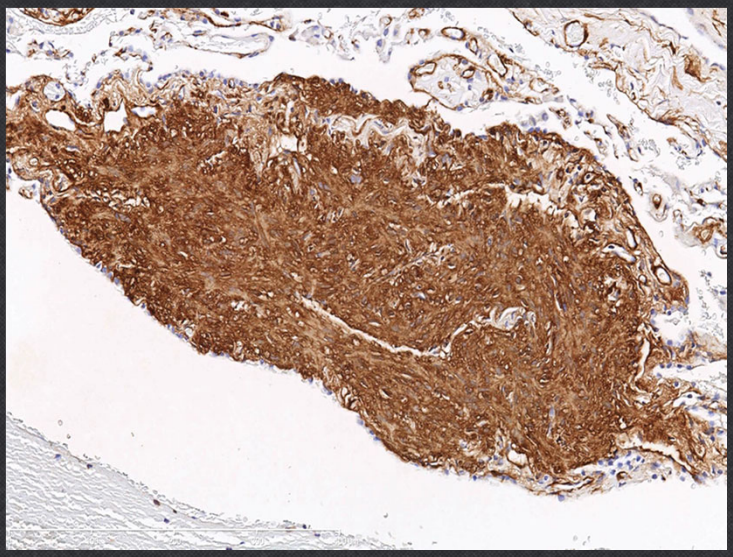


Case #7

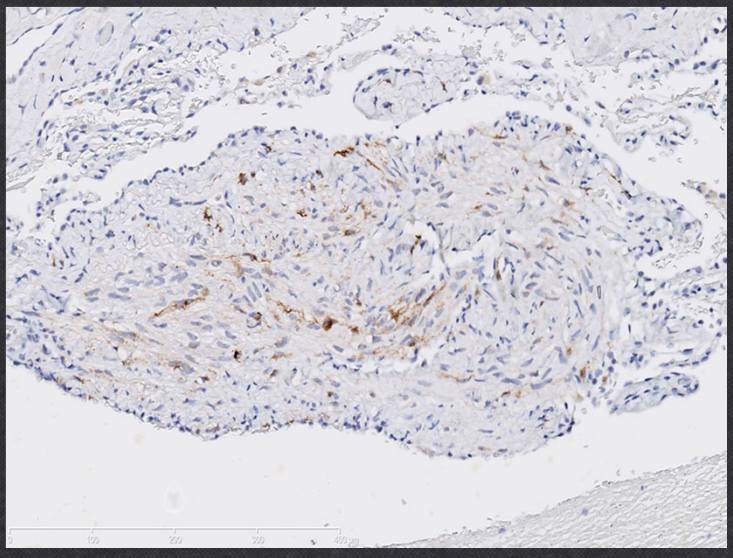
- Labs:
  - ANCA negative
  - Alpha-1-antitrypsin negative
  - ANA negative
  - Serum vascular endothelial growth factor (VEGF): 802 pg/ml (upper limit of normal 310)







SMOOTH MUSCLE ACTIN



HMB45

Diagnosis: Lymphangioleiomyomatosis (LAM)

LAM: Overview

- Cystic lung disease caused by infiltration of the lung with smooth muscle
- 2 forms
  - Patients with tuberous sclerosis (TSC)
  - Sporadic form
- Caused by genetic mutations in either of 2 TSC genes
- Associated with pulmonary manifestations, angiomyolipomas, and lymphangioleiomyomas

LAM: Epidemiology

- Average age at diagnosis: 35 years
- Almost entirely restricted to women

LAM: Pulmonary Manifestations

- Pulmonary cysts
- Pneumothorax
- Chylous pleural effusion
- Obstructive lung disease

LAM: Pathology

- Smooth muscle infiltration of parenchyma, airways, lymphatics, blood vessels
- Thin-walled cystic changes

LAM: Diagnosis

- Index of suspicion when:
  - Young female with a pneumothorax
  - Incidental discovery of pulmonary cysts, angiomyolipoma, or lymphangiomyoma
  - Unexplained chylous pleural effusion or ascites
- Compatible chest CT plus any 1 of the following:
  - Angiomyolipoma, lymphangiomyoma, chylous pleural effusion
  - VEGF greater than 800 pg/ml
  - Lung biopsy

LAM: Management

- **Sirolimus** for patients with a FEV1 less than 70%

Key Points About LAM

- Cystic lung disease in females
  - Consider in any young female with pulmonary cysts +/- pneumothorax
- Diagnosed via radiology, serum VEGF level, +/- biopsy
- **Sirolimus** is the treatment for patients with impaired pulmonary function

Lung Diseases They Didn't Teach You About In Medical School

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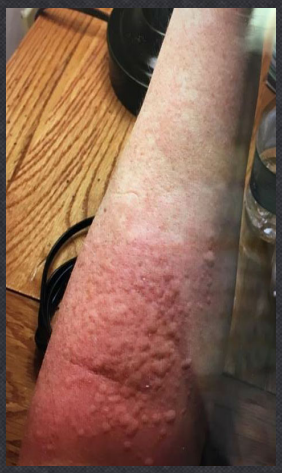
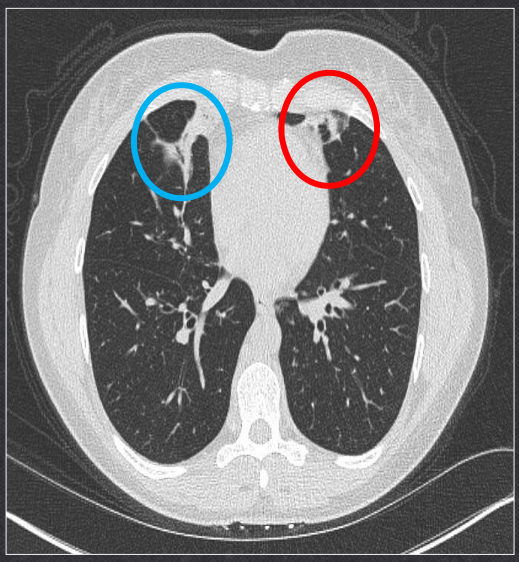
Division of Pulmonary and Critical Care Medicine
The Ohio State University Wexner Medical Center

Case #8

- 44 year-old woman with recurrent pneumonia for 5 months
- Intermittent fevers to 103° – resolve with prednisone and antibiotics
- Episodic urticarial skin rash
- Testing:
  - Spirometry = moderate obstruction
  - IgE elevated at 6,204
  - Eosinophil count elevated at 780

Left forearm





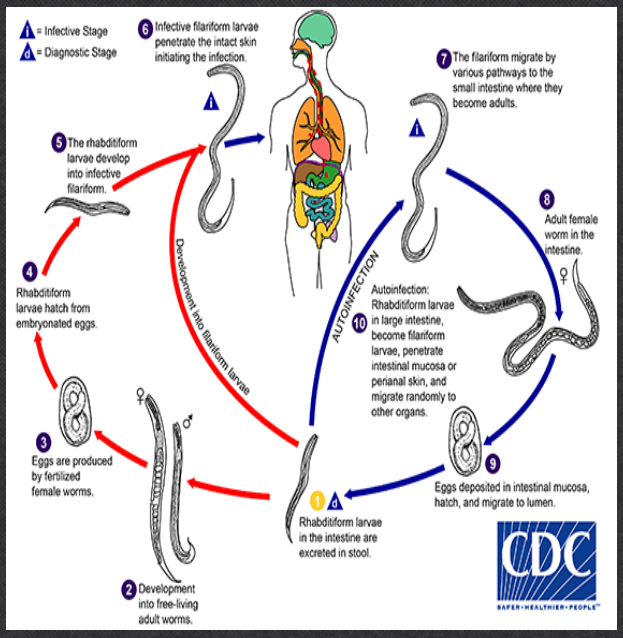
Strongyloides Symptoms

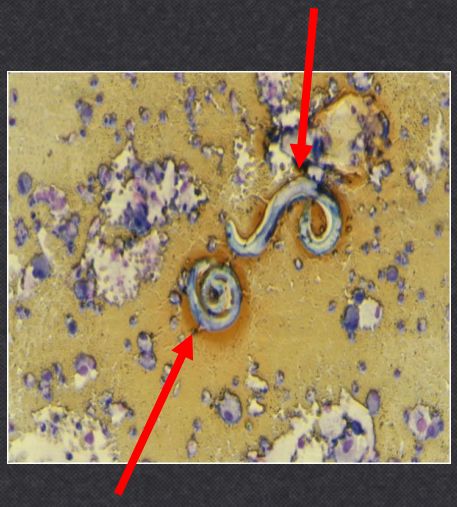
Systemic:

- Skin Rash
- Abdominal Pain
- Diarrhea
- Increased blood eosinophil count

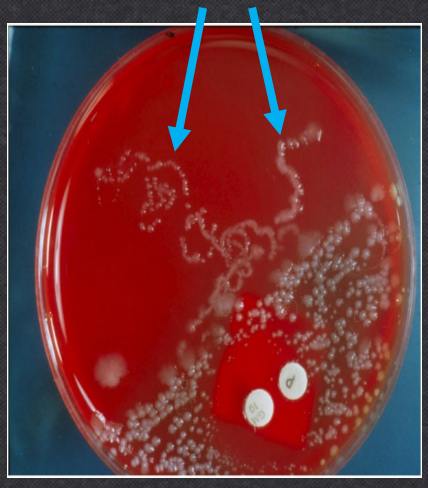
In the lung:

- Wheezing
- Recurrent pneumonias (especially gram negative)
- Migratory pulmonary infiltrates
- Cough
- Hemoptysis





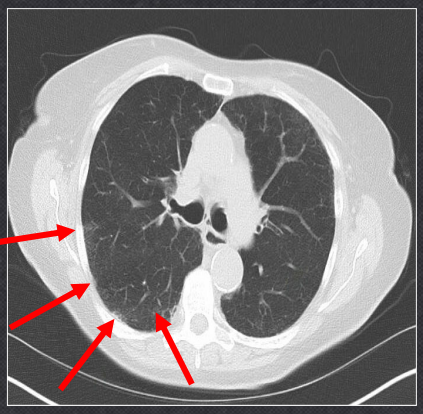
Bronchoalvoelar Lavage:
Low Power Photomicrograph



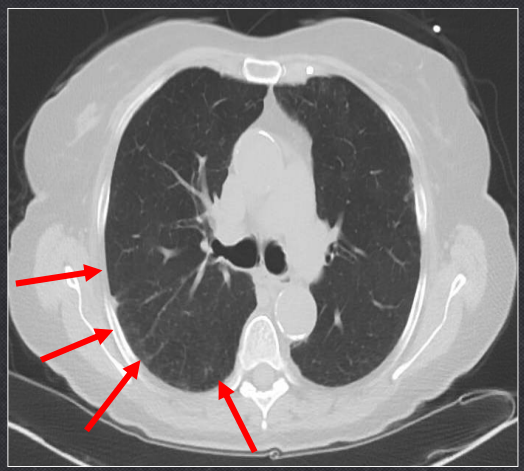
Agar Culture Plate:
Larvae Leaving Bacterial Trails

77 year-old woman with Takayasu's arteritis

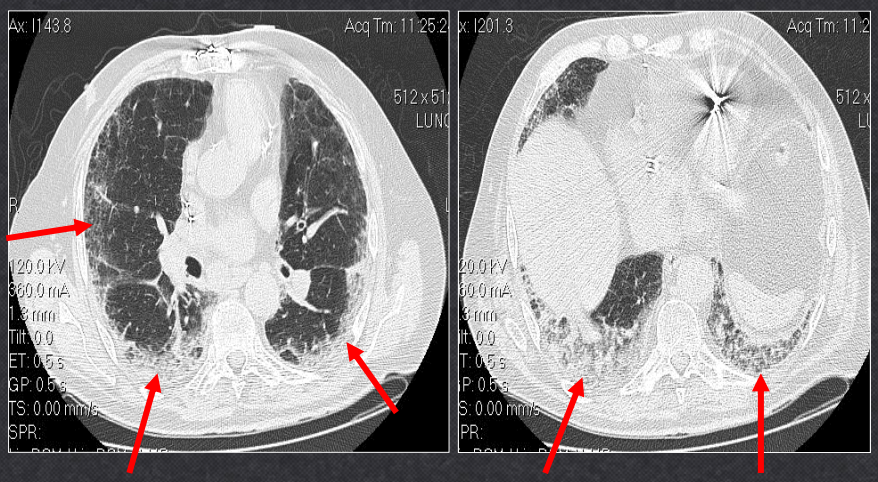
- Previously treated with methotrexate
- Now with cough and dyspnea for 3 years
- No improvement with inhalers or prednisone
- Testing:
  - PFTs = severe obstruction
  - Eosinophil count 1,200



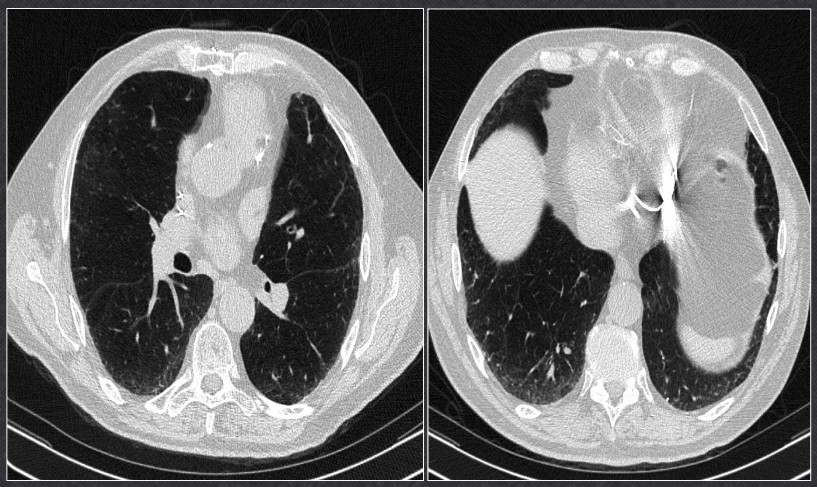
After treatment with ivermectin



79-year old man with recurrent pneumonias and peripheral blood eosinophilia

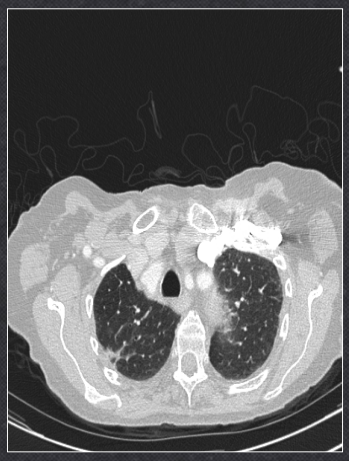


After treatment with ivermectin



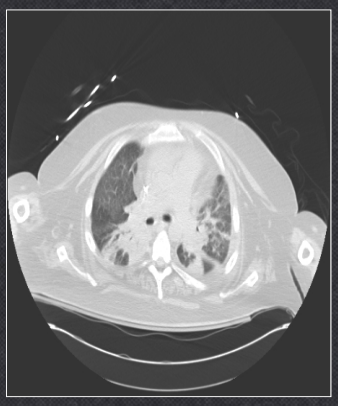
Strongyloides is sneaky

- 87 year old mother of OSU physician
- Recurrent “colitis”, eosinopilia, pulmonary infiltrates and cough for 20 years
- Positive anti-strongyloides antibody
- Symptoms resolved with ivermectin



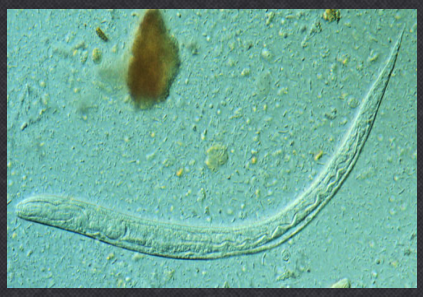
You’ll miss it if you only order the regular stool O&P exam

- 35-year old man
- Multiple ICU admissions
- Recurrent fevers & pseudomonas pneumonia
- Persistent fevers and blood eosinophilia (up to 2,700)
- Stool O&P negative (antigenic)
- Strongyloides antibody positive



Key Points About Strongyloides:

- Worms are everywhere
- Non-resolving pneumonia + peripheral eosinophilia = order a Strongyloides antibody test



Lung Diseases They Didn't Teach You About In Medical School

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Division of Pulmonary, Allergy, Critical Care and Sleep Medicine
The Ohio State University Wexner Medical Center

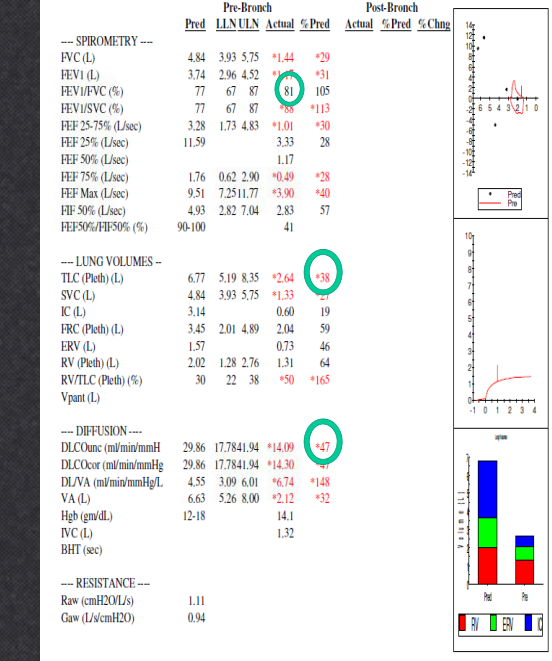
Case #9

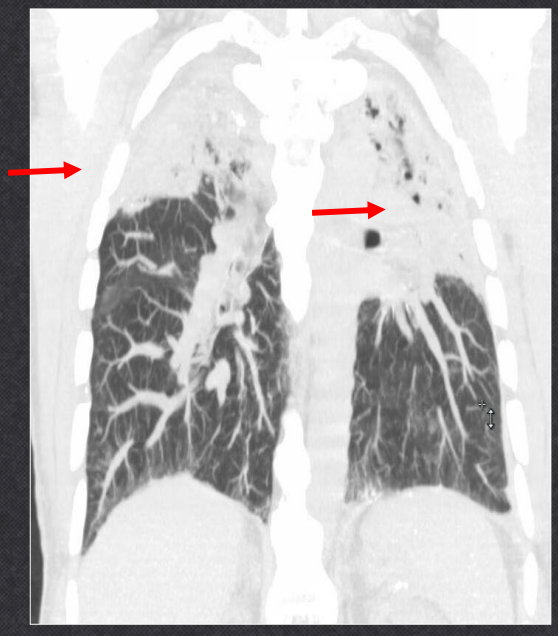
- 63 year old man
- Dyspnea and nonproductive cough for 8 months
- Past medical history:
  - Psoriatic arthritis
  - Lupus, recently diagnosed
- Medications:
  - Methotrexate for 3 years; stopped 5 years prior
  - Prednisone, started 2 months prior

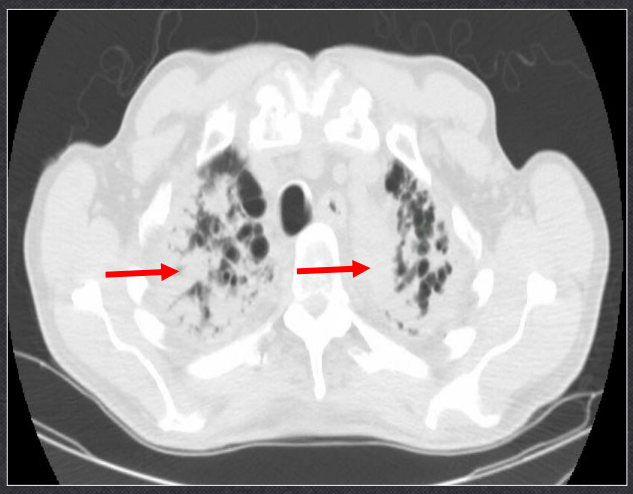
Case #9 (continued)

- Social history: never smoker
- Exam: biapical dry crackles
- Labs:
  - ANA positive
  - All other serological studies negative

	Pred	LLN	ULN	Pre-Bronch Actual	Pre-Bronch %Pred	Post-Bronch Actual	Post-Bronch %Pred	Post-Bronch %Chng
--- SPIROMETRY ---								
FVC (L)	4.84	3.93	5.75	*1.44	*29			
FEV1 (L)	3.74	2.96	4.52	*1.16	*31			
FEV1/FVC (%)	77	67	87	81	105			
FEV1/SVC (%)	77	67	87	*88	*113			
FEF 25-75% (L/sec)	3.28	1.73	4.83	*1.01	*30			
FEF 25% (L/sec)	11.59			3.33	28			
FEF 50% (L/sec)				1.17				
FEF 75% (L/sec)	1.76	0.62	2.90	*0.49	*28			
FEF Max (L/sec)	9.51	7.25	11.77	*3.90	*40			
FIF 50% (L/sec)	4.93	2.82	7.04	2.83	57			
FEF50%/FIF50% (%)	90-100			41				
--- LUNG VOLUMES --								
TLC (Pleth) (L)	6.77	5.19	8.35	*2.64	*38			
SVC (L)	4.84	3.93	5.75	*1.33	*27			
IC (L)	3.14			0.60	19			
FRC (Pleth) (L)	3.45	2.01	4.89	2.04	59			
ERV (L)	1.57			0.73	46			
RV (Pleth) (L)	2.02	1.28	2.76	1.31	64			
RV/TLC (Pleth) (%)	30	22	38	*50	*165			
Vpant (L)								
--- DIFFUSION ---								
DLCOunc (ml/min/mmH	29.86	17.78	41.94	*14.09	*47			
DLCOcor (ml/min/mmHg	29.86	17.78	41.94	*14.30	*48			
DL/VA (ml/min/mmHg/L	4.55	3.09	6.01	*6.74	*148			
VA (L)	6.63	5.26	8.00	*2.12	*32			
Hgb (gm/dL)	12-18			14.1				
IVC (L)				1.32				
BHT (sec)								
--- RESISTANCE ---								
Raw (cmH2O/L/s)	1.11							
Gaw (L/s/cmH2O)	0.94							







Pathology Report

- Pleuritis
- Pleural blebs associated with subpleural fibrobullous disease/scarring in upper and middle lobes.
- The etiology of these changes is not apparent
- UIP/IPF was considered in the differential; however, the predominance of changes in upper lobe and radiologically apparent sparing of lower lobe argue against that consideration.

Diagnosis: Pleuropulmonary Fibroeslastosis (PPFE)

PPFE: Overview

- Upper-lobe-dominant slowly progressive pulmonary fibrosis
- 1st description in 1992
- The name, PPFE, was coined in 2004 and is descriptive
- Unknown etiology
  - Idiopathic form
  - Form associated with lung and bone marrow transplants, chemotherapy, infections, autoimmune diseases

PPFE: Epidemiology

- Median age at diagnosis: 53 years
- No sex predilection
- Smoking not a risk factor

PPFE: Pulmonary Manifestations

- Restrictive ventilatory impairment
- Interstitial lung disease
- Pneumothorax

PPFE: Radiology & Pathology

- Radiology
  - Early bilateral and irregular apical pleural thickening
  - Later reticular and fibrotic parenchymal changes
  - Biapical blebs
- Pathology
  - Fibro:
    - Fibrous thickening of the visceral pleura
    - Dense intra-alveolar fibrosis
  - Septal elastosis
- Abrupt transition from normal to abnormal tissue

PPFE: Diagnosis

- No agreed upon consensus statement
- Clinical signs and symptoms, radiology, +/- pathological exam

PPFE: Management

- Prognosis is highly variable
- No treatment has yet been demonstrated to alter disease progression
- Supportive care
- Lung transplant

Key Points About Pleuroparenchymal Fibroelastosis

- Relatively recently (1992) recognized entity
- The diagnostic term is descriptive for radiological and pathological features
- No consensus diagnostic criteria
- Clinical course variable
- No disease modifying treatment

Lung Diseases They Didn't Teach You About In Medical School

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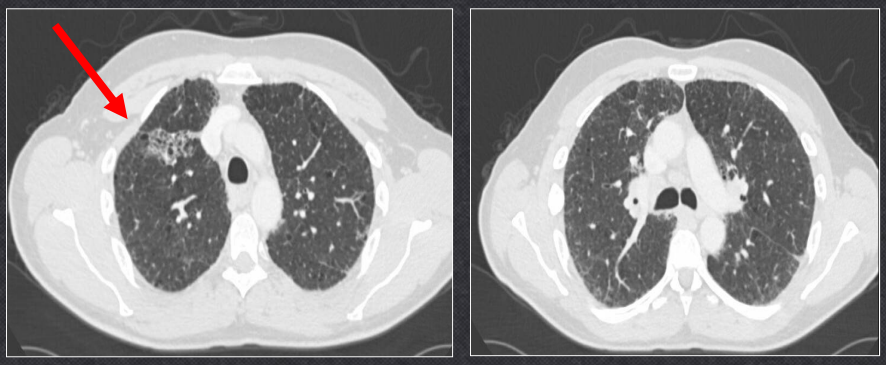
The Ohio State University Wexner Medical Center

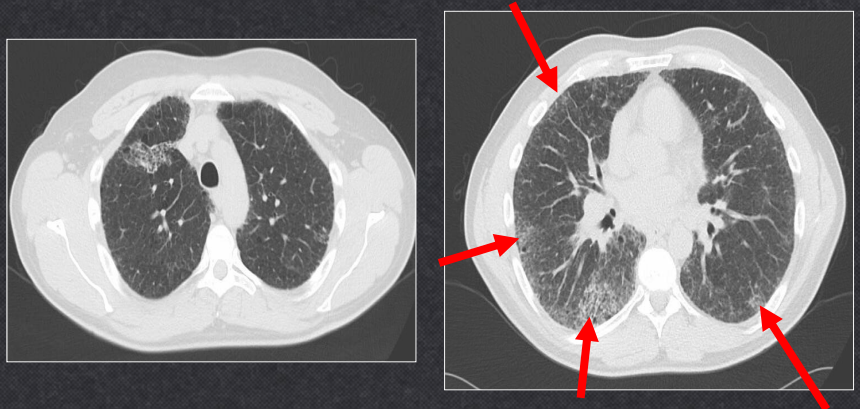
Case #10

- 48 year-old man with cough onset January 2016
- Referred for interstitial lung disease evaluation July 2017
- Past medical history = HIV (well-controlled)
- Family history: Mother & maternal aunt had idiopathic pulmonary fibrosis
- Exam: bibasilar dry crackles
- BAL: 89% neutrophils (normal < 2%), no cancer

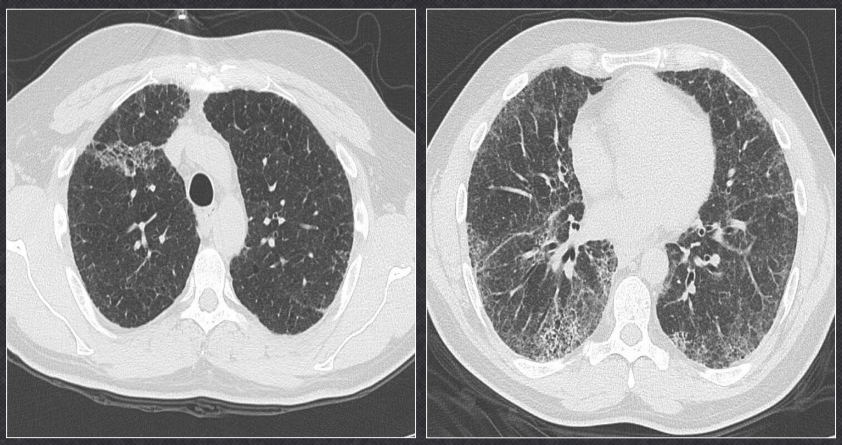
May 2016



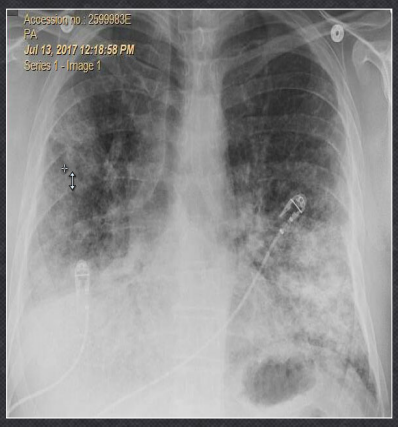
August 2016

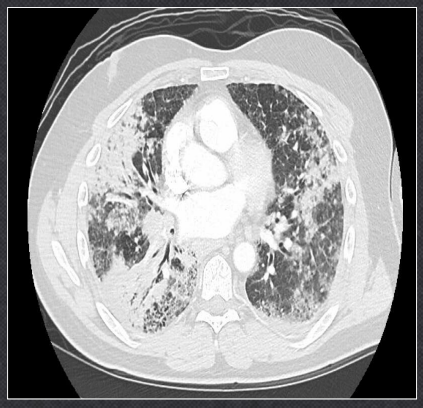


November 2016

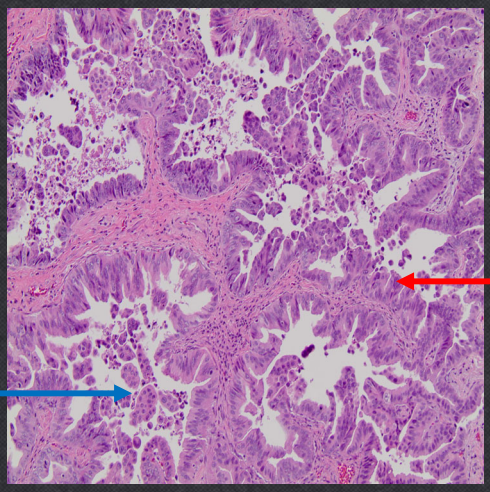


June 2017

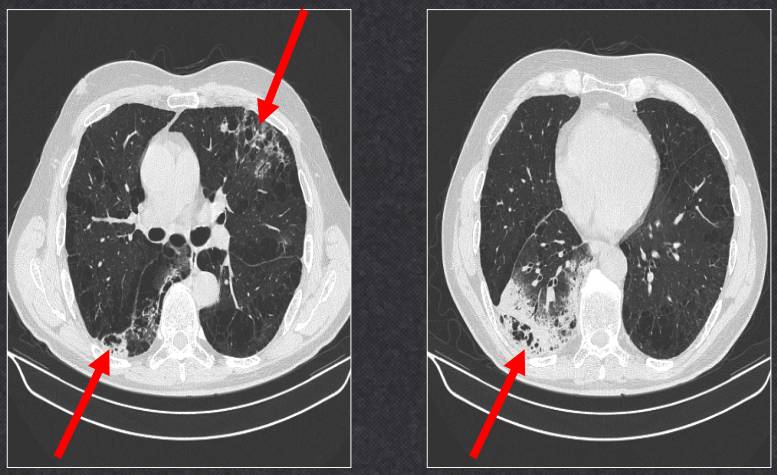




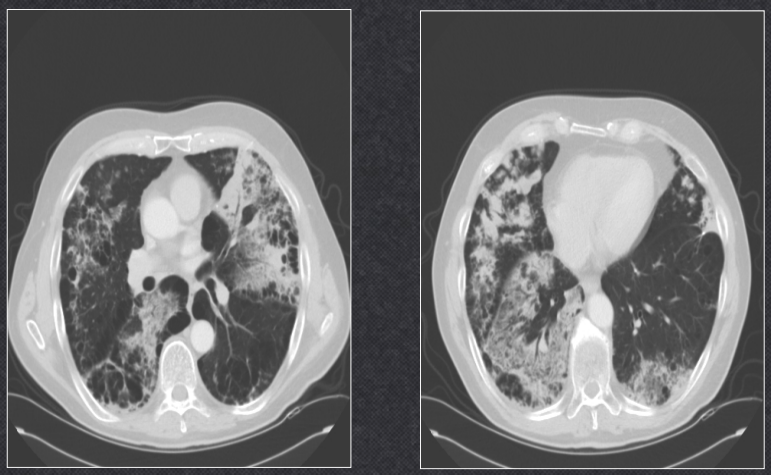
Adenocarcinoma ("bronchoalveolar carcinoma")



Adenocarcinomas can be missed on biopsy

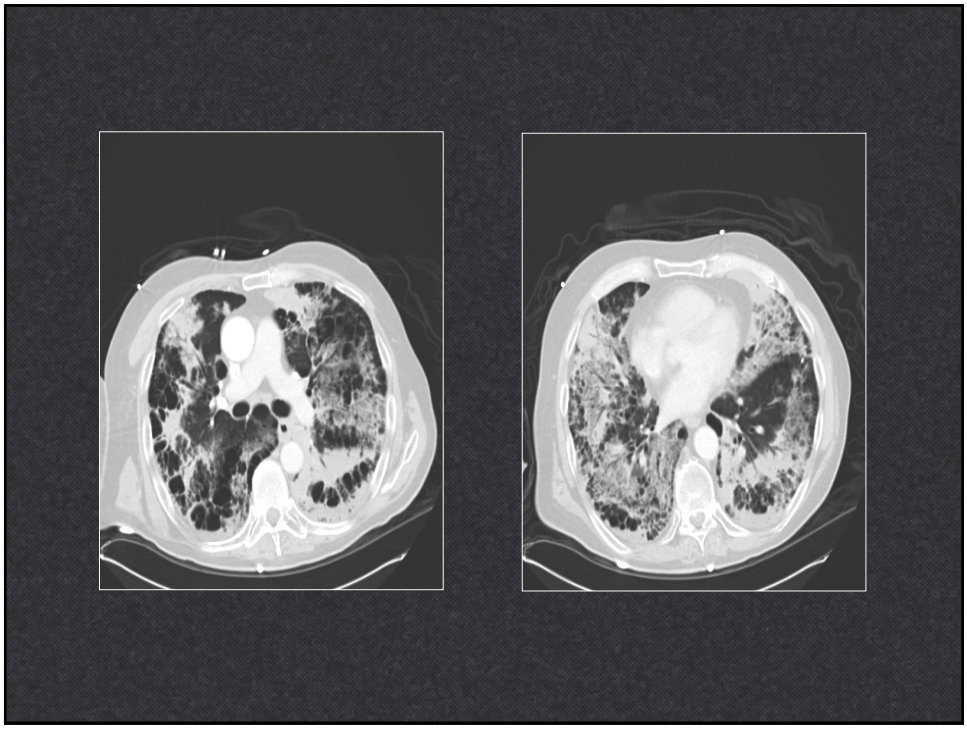


Adenocarcinomas can be missed on biopsy

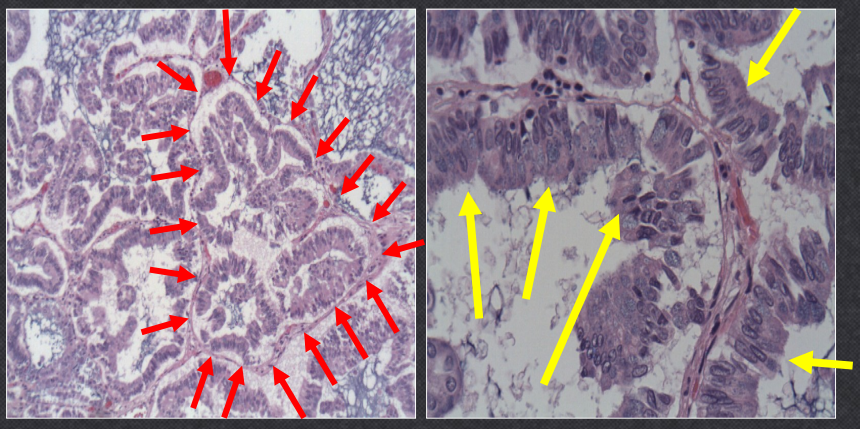


Adenocarcinomas can be missed on biopsy

- Bronchoscopy #1
  - BAL – 52% macrophages, 27% neutrophils, 15% lymphocytes, 6% eosinophils; negative AFB, fungal cultures; cytology negative
  - Brushings – negative cytology
  - Transbronchial biopsy – calcified granuloma with histoplasmosis organisms seen; no cancer
- Bronchoscopy #2
  - Negative cytology
- Surgical lung biopsy
  - Organizing pneumonia (pathologist #1)
  - Hypersensitivity (pathologist #2)
  - Adenomatous hyperplasia (pathologist #3)
  - Organizing pneumonia versus hypersensitivity pneumonitis but no cancer (pathologist #4)
- Bronchoscopy #3
  - Transbronchial biopsy = no cancer



Autopsy: Adenocarcinoma



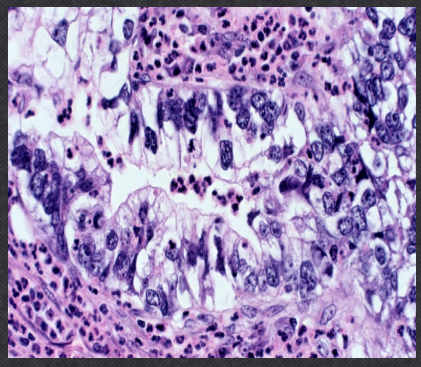
Adenocarcinoma

~~Bronchoalveolar Carcinoma~~

- Current classification of adenocarcinoma of the lung:
  - Lepidic
  - Acinar
  - Papillary
  - Solid
  - Invasive mucinous
  - Colloid
  - Minimally invasive
  - Adenocarcinoma in situ

Key Points About Adenocarcinoma

- The term "bronchoalveolar carcinoma" is no longer used
- Well-differentiated adenocarcinomas can be difficult to diagnose
- Cytology and even surgical lung biopsies can be false-negative
- Adenocarcinoma can mimic many interstitial lung diseases
- Driver mutations determine treatment choices



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