Bronchiolitis

ΣΑ Παπίρης
Καθηγητής Ιατρικής
Διευθυντής Β' Πνευμονολογικής Κλινικής
Εθνικό και Καποδιστριακό Πανεπιστήμιο Αθηνών
«Αττικόν» Πανεπιστημιακό Νοσοκομείο

BRONCHIOLITIS

is the inflammation of the bronchiolar wall which is either the prominent histophatologic feature and characterizes a distinct clinical entity (rare),

or participates in the inflammation of the airways and/or surrounding alveolar structures in several already defined clinical entities (common)

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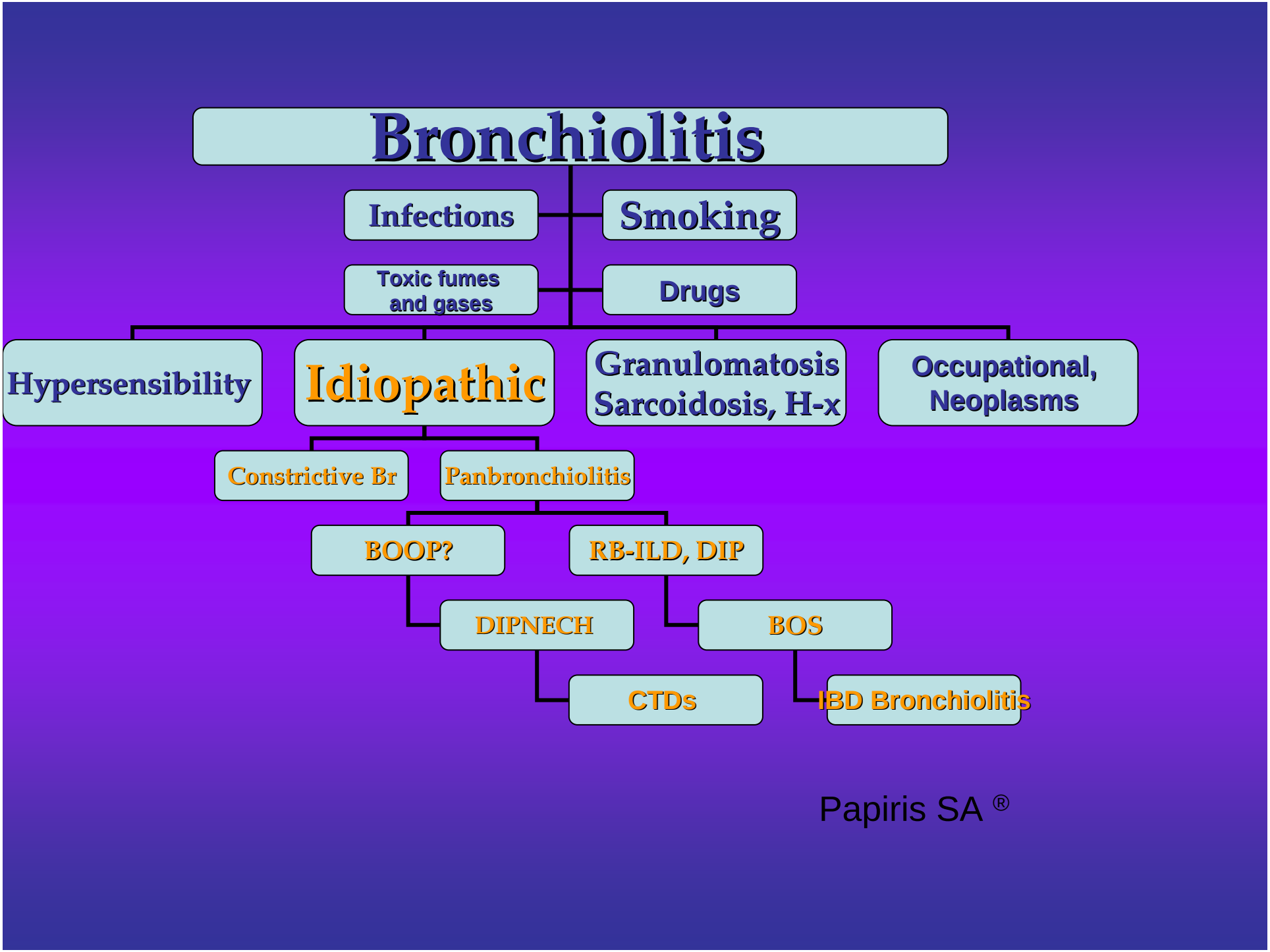
Bronchiolitis
Infections
Smoking
Toxic fumes and gases
Drugs
Hypersensibility
Idiopathic
Granulomatosis Sarcoidosis, H-x
Occupational, Neoplasms
Constrictive Br
Panbronchiolitis
BOOP?
RB-ILD, DIP
DIPNECH
BOS
CTDs
IBD Bronchiolitis

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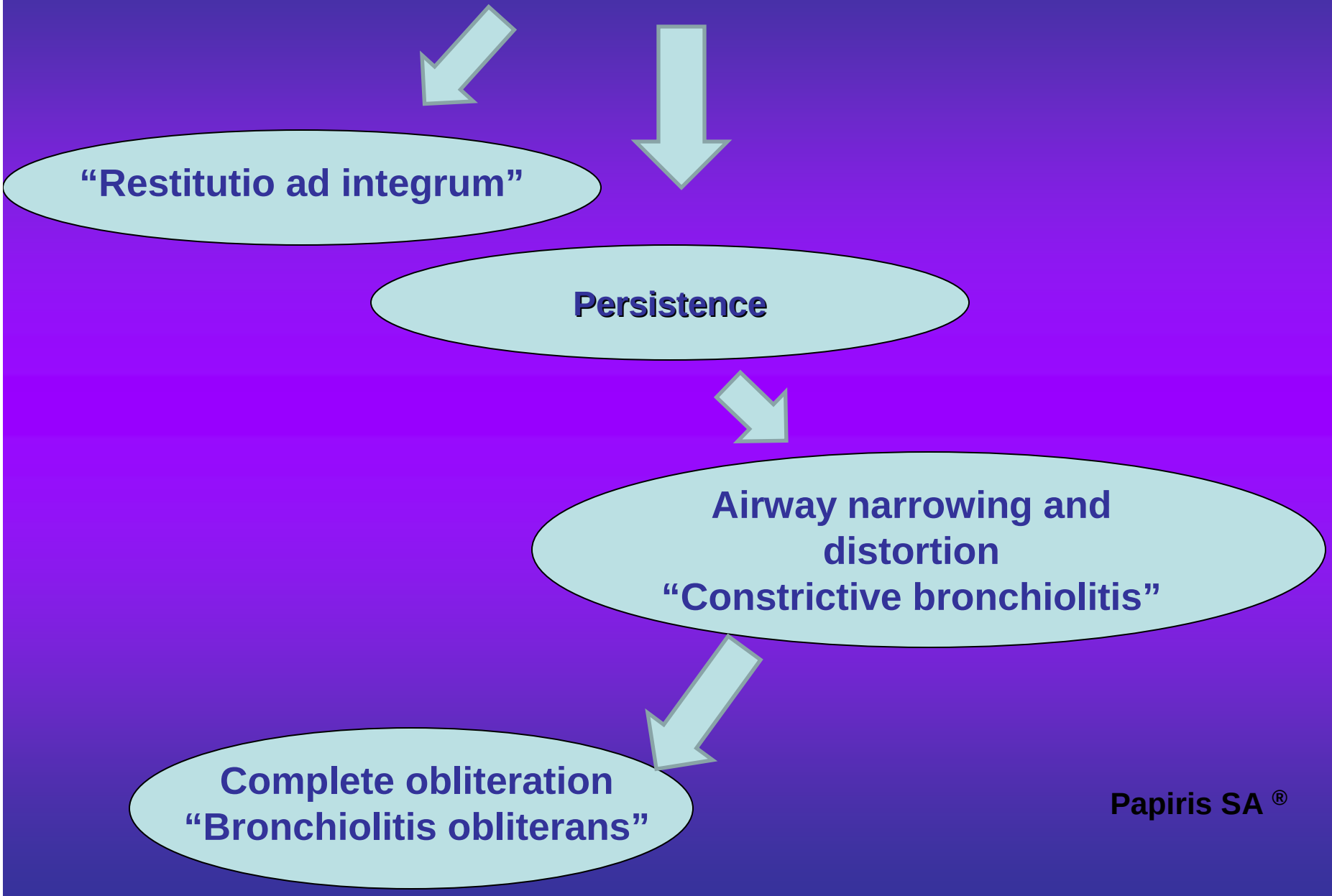
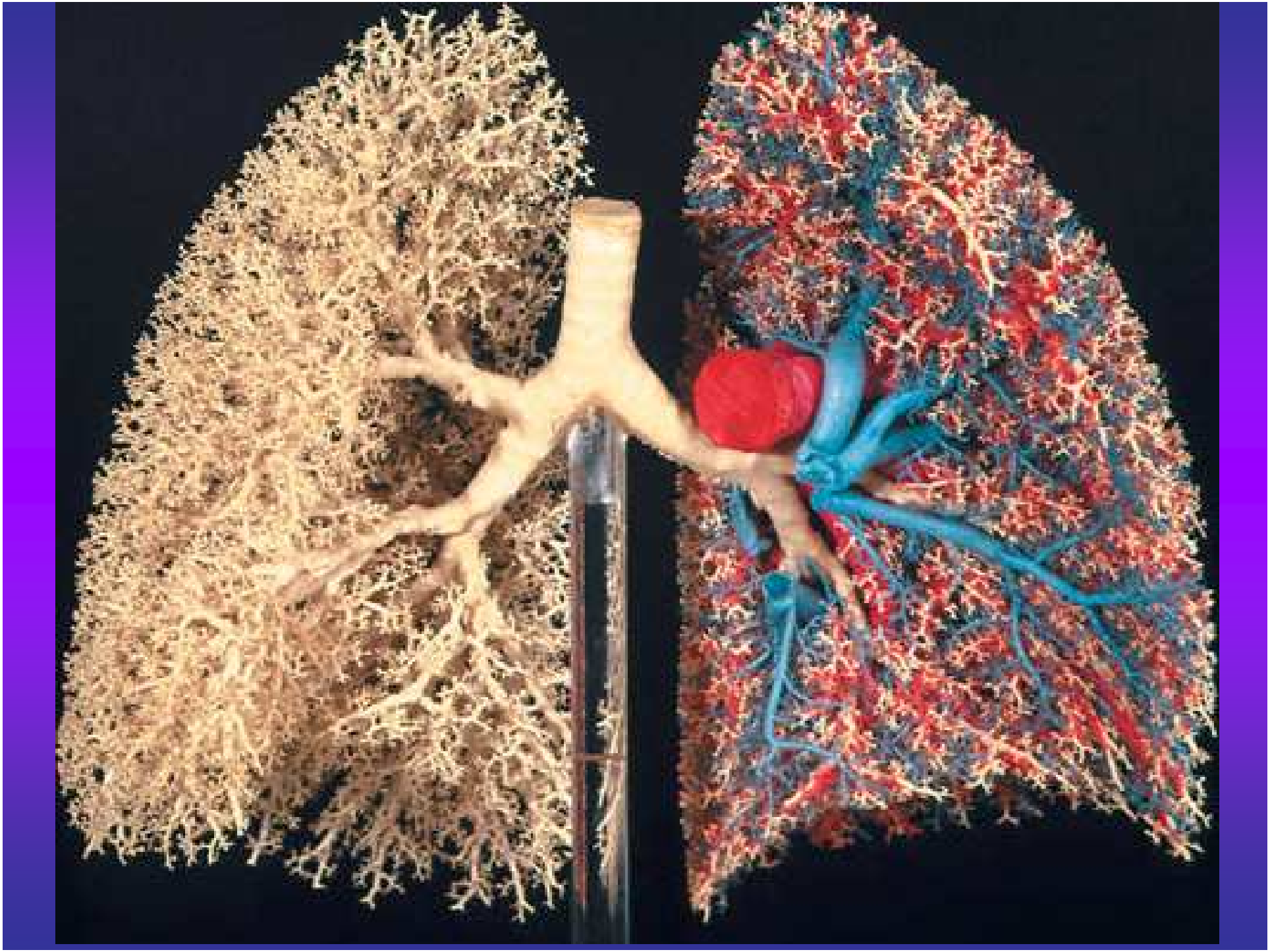
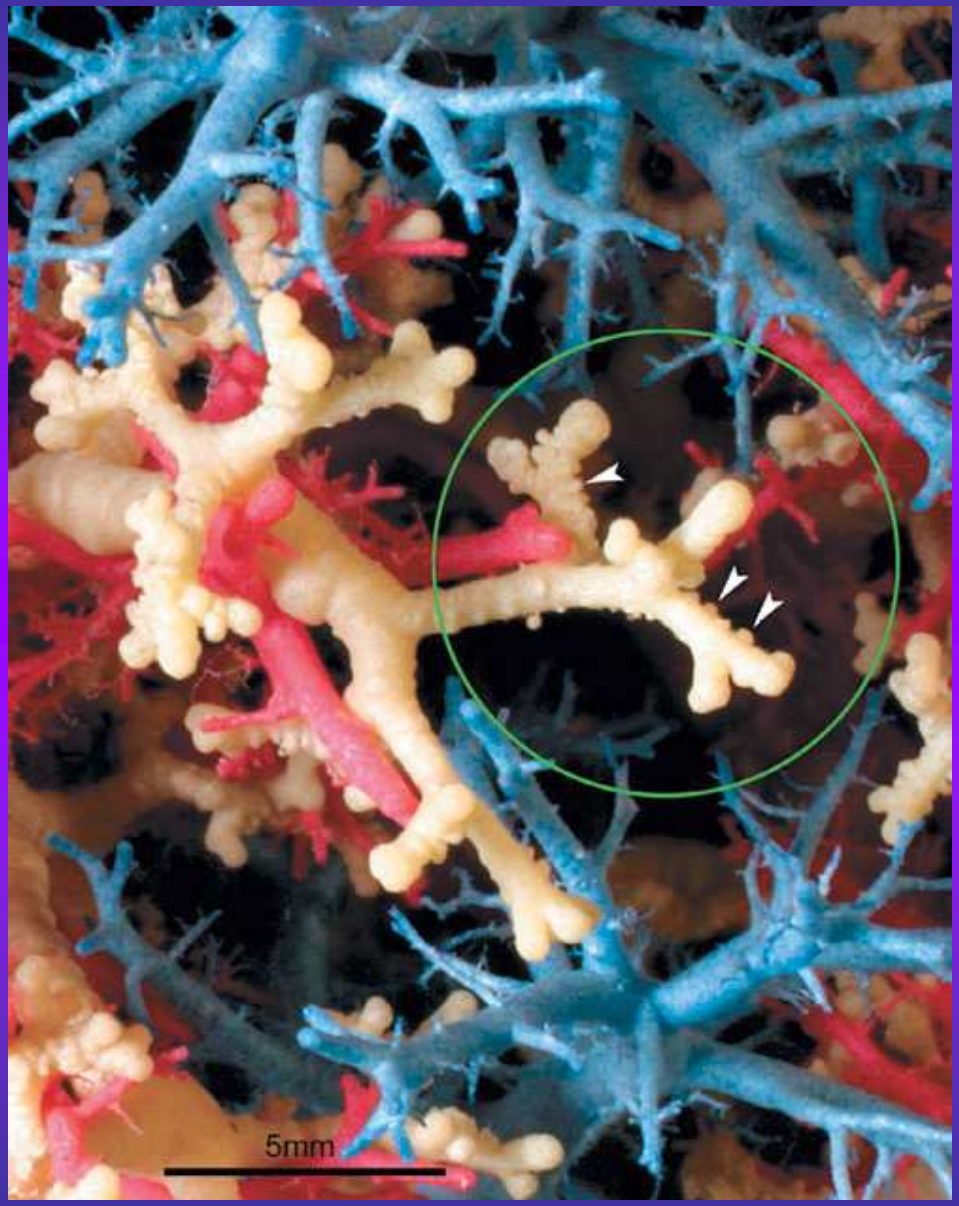
BRONCHIOLITIS



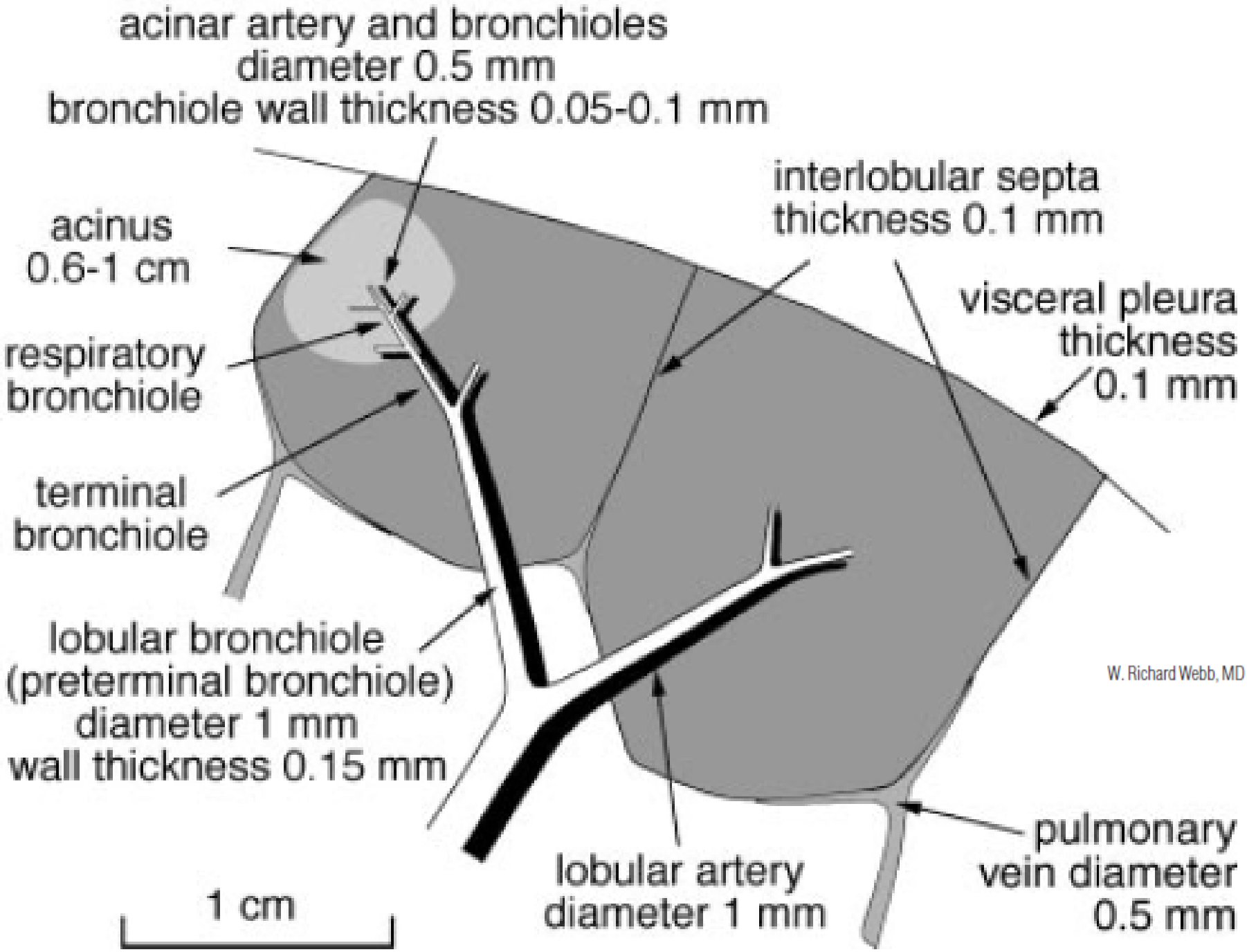
Papiris SA ®

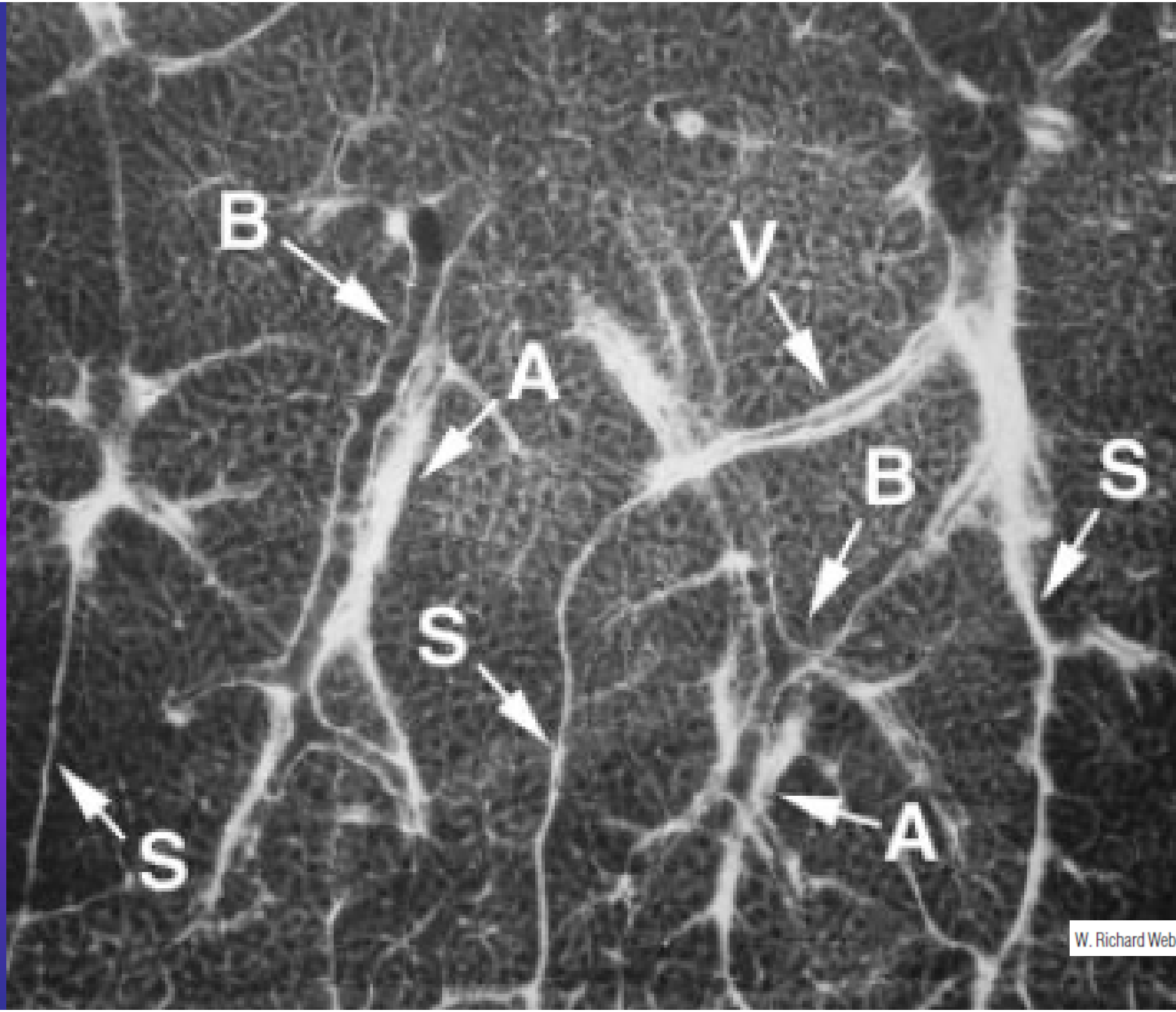



5mm

The secondary pulmonary lobule is a fundamental unit of lung structure, and it reproduces the lung in miniature.

Airways, pulmonary arteries, veins, lymphatics, and the lung interstitium are all represented at the level of the secondary lobule.


acinar artery and bronchioles
diameter 0.5 mm
bronchiole wall thickness 0.05-0.1 mm
interlobular septa
thickness 0.1 mm
acinus
0.6-1 cm
visceral pleura
thickness
0.1 mm
respiratory
bronchiole
terminal
bronchiole
lobular bronchiole
(preterminal bronchiole)
diameter 1 mm
wall thickness 0.15 mm
W. Richard Webb, MD
pulmonary
vein diameter
0.5 mm
lobular artery
diameter 1 mm
1 cm

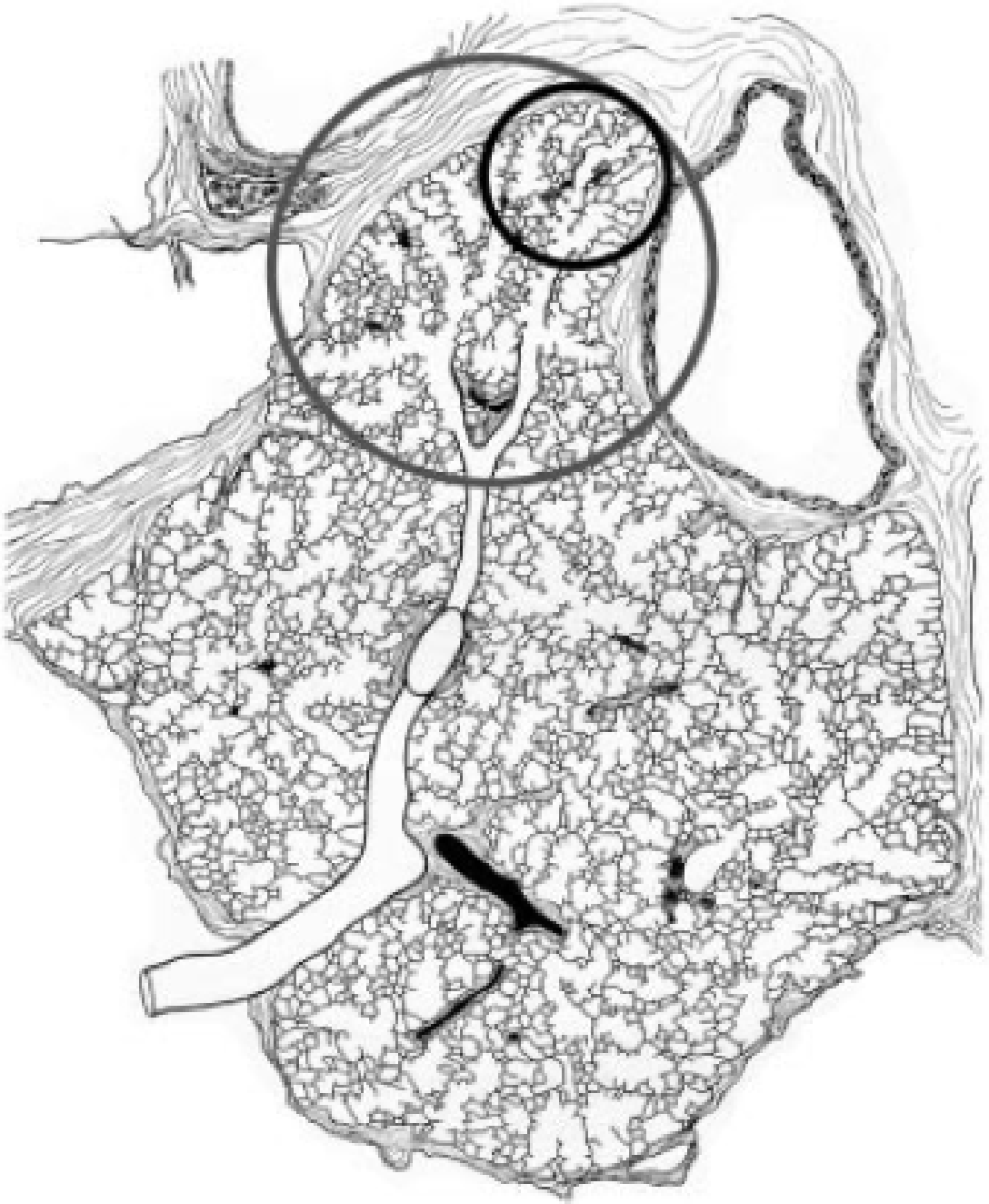


W. Richard Webb, MD

Interlobular septa and septal structures

Centrilobular structures

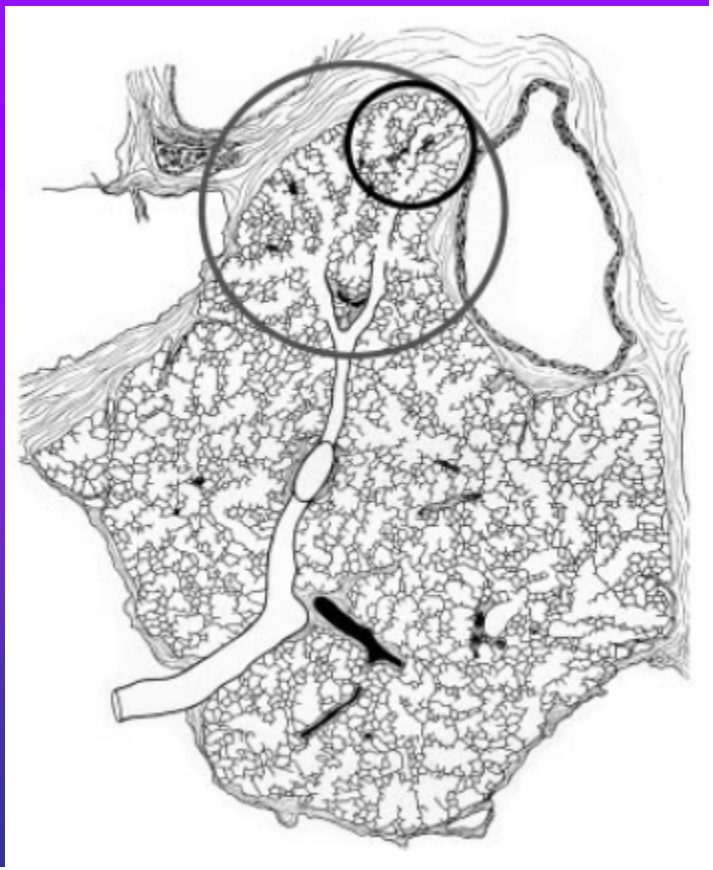
Lobular parenchyma



Thin-Section CT Diagnosis of Lobular Abnormalities

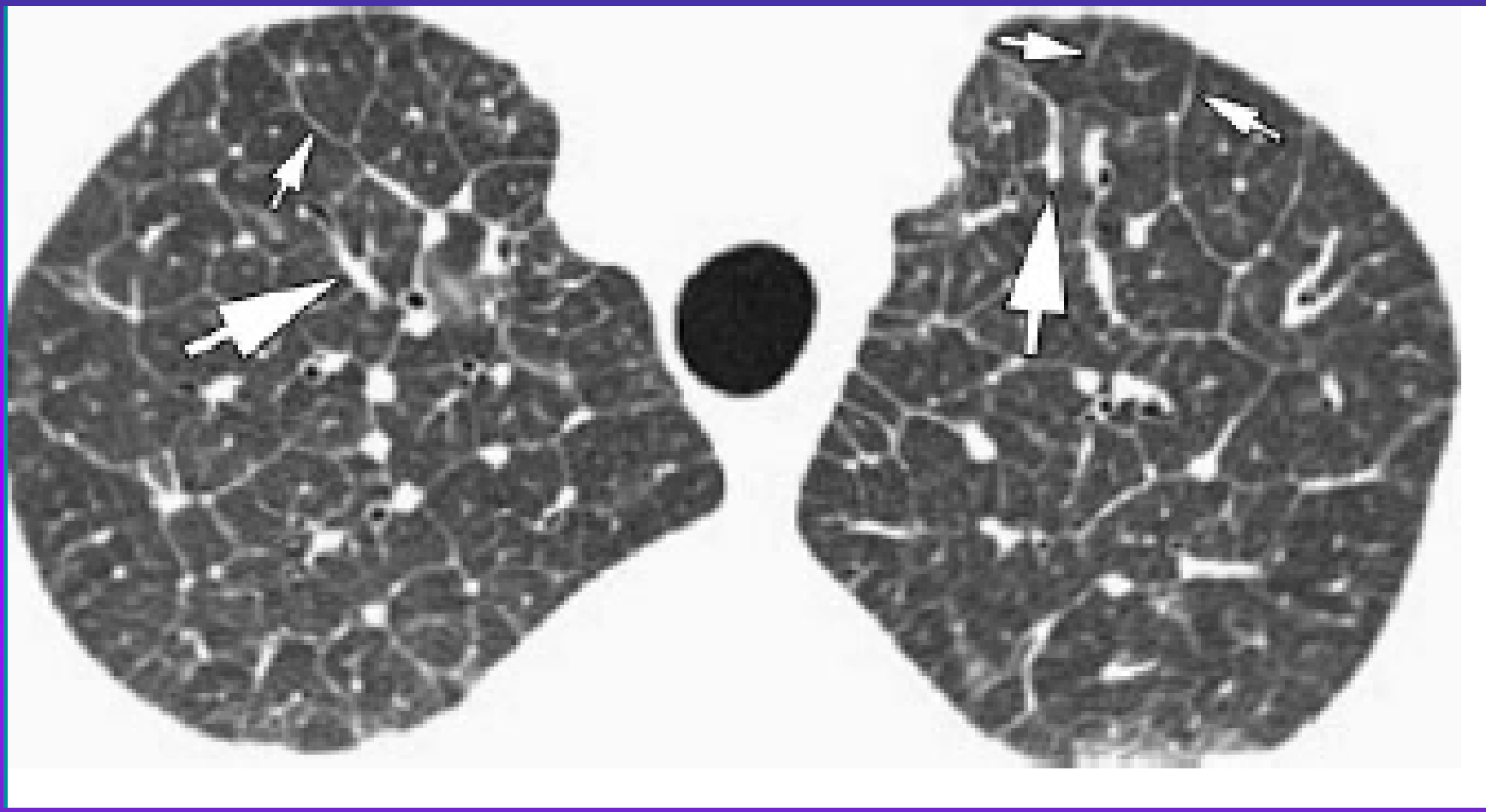
Pathologic alternations in secondary lobular anatomy visible on thin-section CT scans may be described as:

Perilobular (interlobular septal thickening and peripheral lobular diseases),
Centrilobular, and
Panlobular.

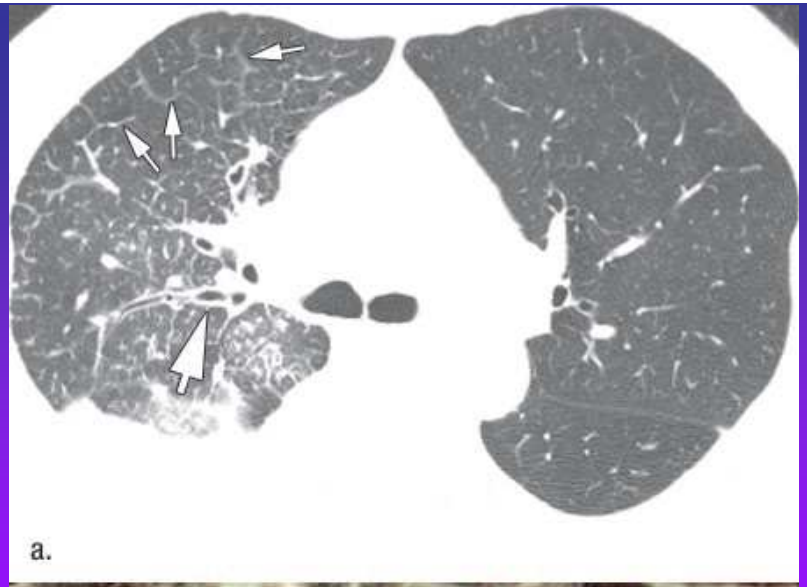


Perilobular Pattern: Differential Diagnosis

CT Finding	Differential Diagnosis
Smooth interlobular septal thickening	Pulmonary edema, hemorrhage, or veno-occlusive disease; lymphangitic spread of neoplasm; lymphangiomatosis; amyloidosis; pneumonia; alveoloar proteinosis
Nodular interlobular septal thickening	Lymphangitic spread of neoplasm, lymphoproliferative disease (eg, lymphocytic interstitial pneumonia), sarcoidosis, silicosis and coal worker's pneumoconiosis, amyloidosis
Irregular interlobular septal thickening	End-stage lung disease, sarcoidosis, usual interstitial pneumonia, asbestosis, hypersensitivity pneumonitis
Peripheral lobular abnormalities	Idiopathic pulmonary fibrosis, usual interstitial pneumonia, organizing pneumonia

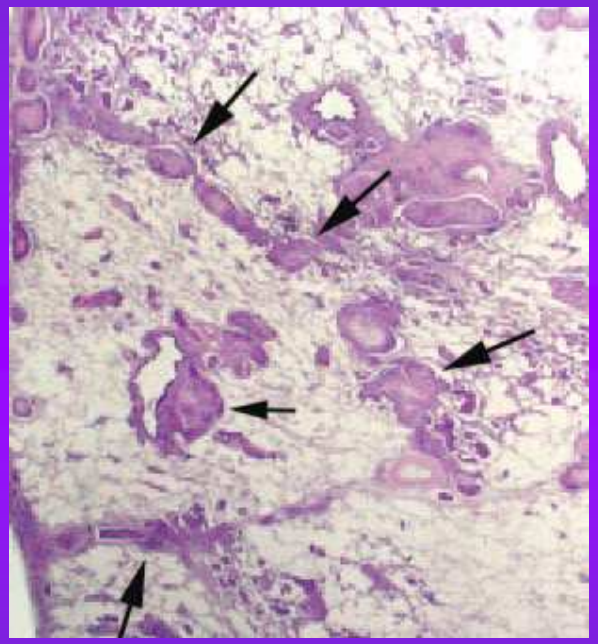


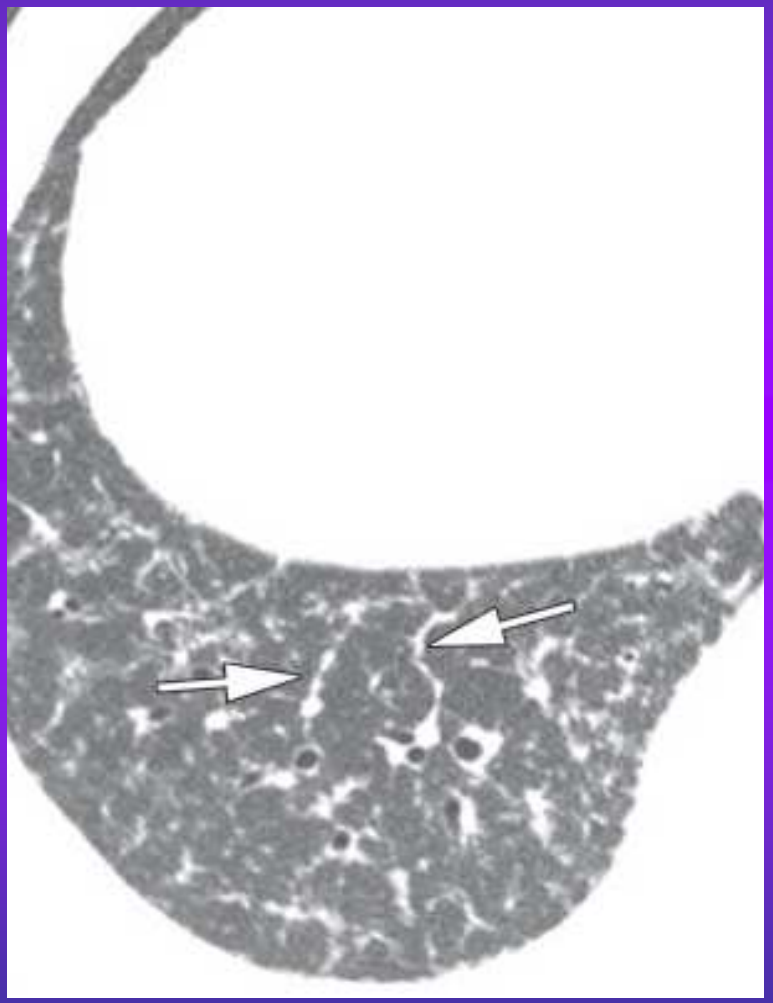
Perilobular



a.

b.



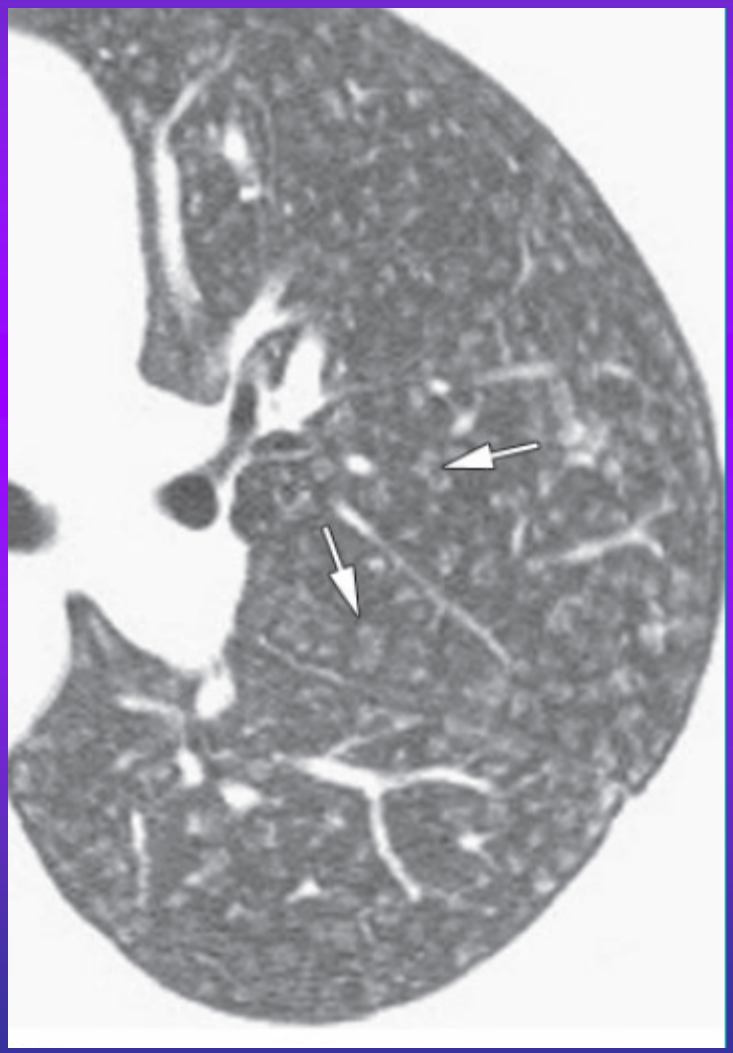


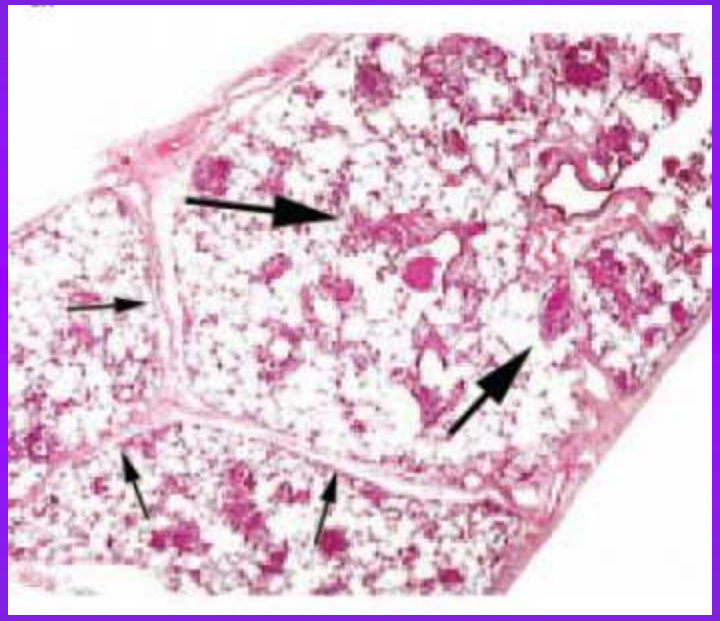
Centrilobular Nodules: Differential Diagnosis

Cause	Differential Diagnosis
Bronchiolar infection	Endobronchial spread of tuberculosis, nontuberculous mycobacteria or other granulomatous infection, bronchopneumonia, infectious bronchiolitis, cystic fibrosis
Bronchiolar inflammation	Hypersensitivity pneumonitis, diffuse panbronchiolitis, asthma, allergic bronchopulmonary aspergillosis, Langerhans cell histiocytosis, organizing pneumonia, bronchiolitis obliterans, respiratory bronchiolitis in smokers, asbestosis, follicular bronchiolitis
Endobronchial spread of tumor	For example, bronchioloalveolar carcinoma
Angiocentric disease	Pulmonary edema, pulmonary vasculitis, talcosis, pulmonary hemorrhage or hemosiderosis, metastatic calcification, pulmonary hypertension
Perilymphatic disease	Sarcoidosis, silicosis and coal worker's pneumoconiosis, lymphangitic spread of neoplasm, lymphocytic interstitial pneumonia

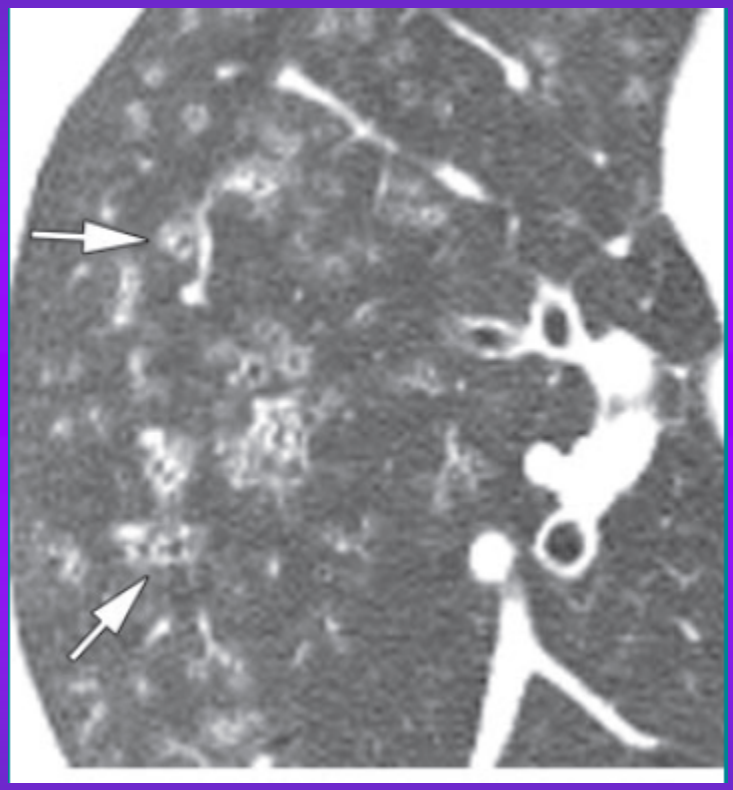
Centrilobular

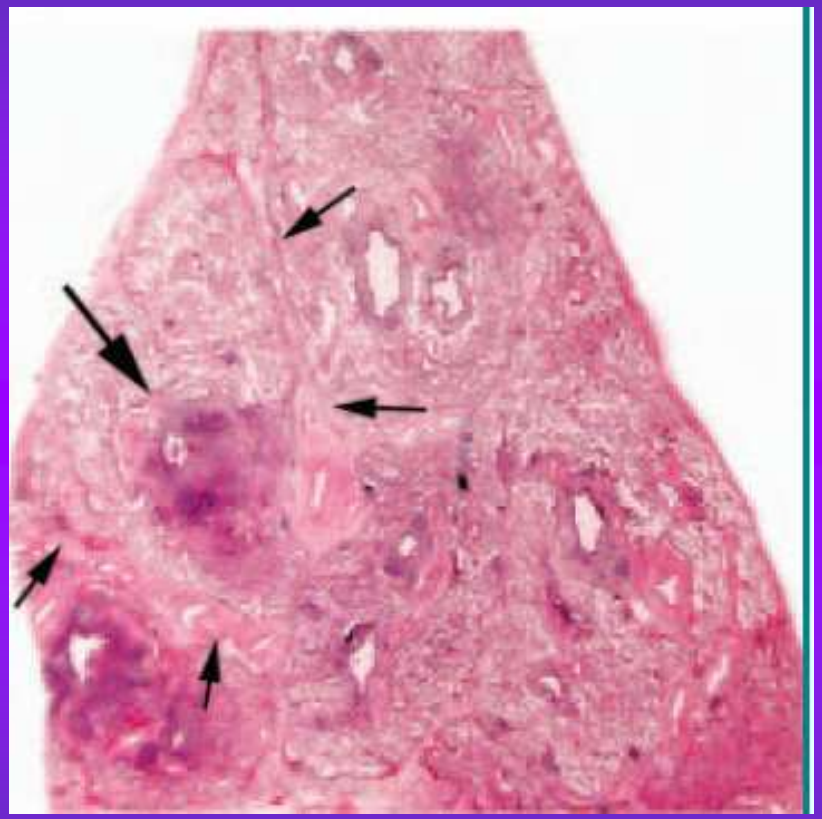
Disease that primarily affects centrilobular bronchioles and results in inflammation, infiltration, or fibrosis of the surrounding interstitium and alveoli



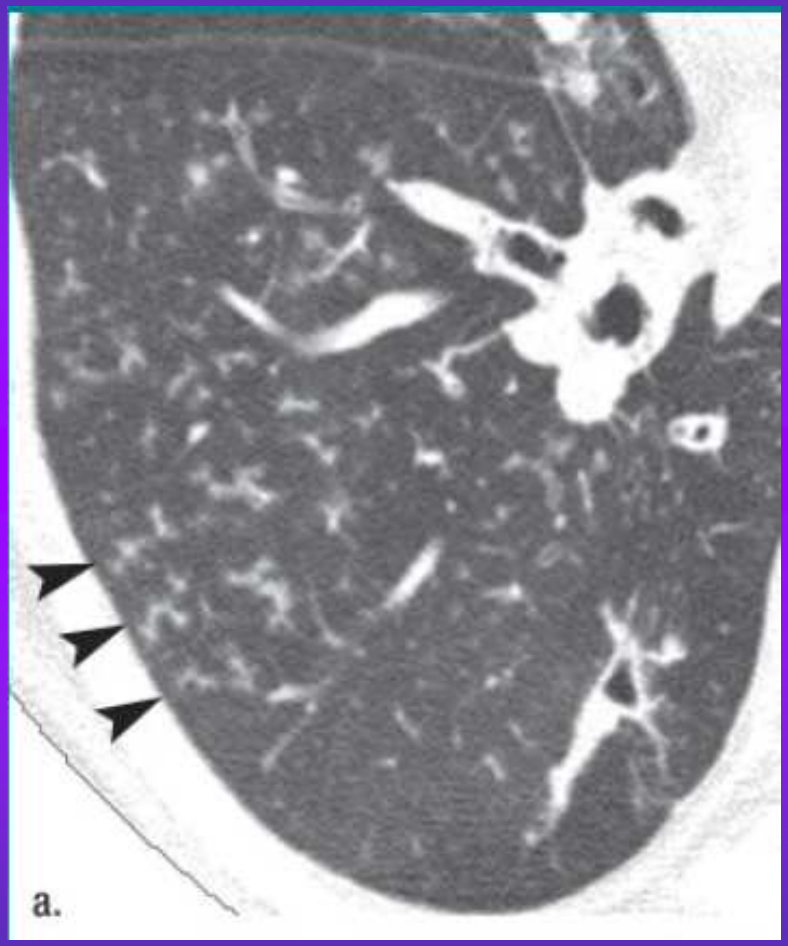


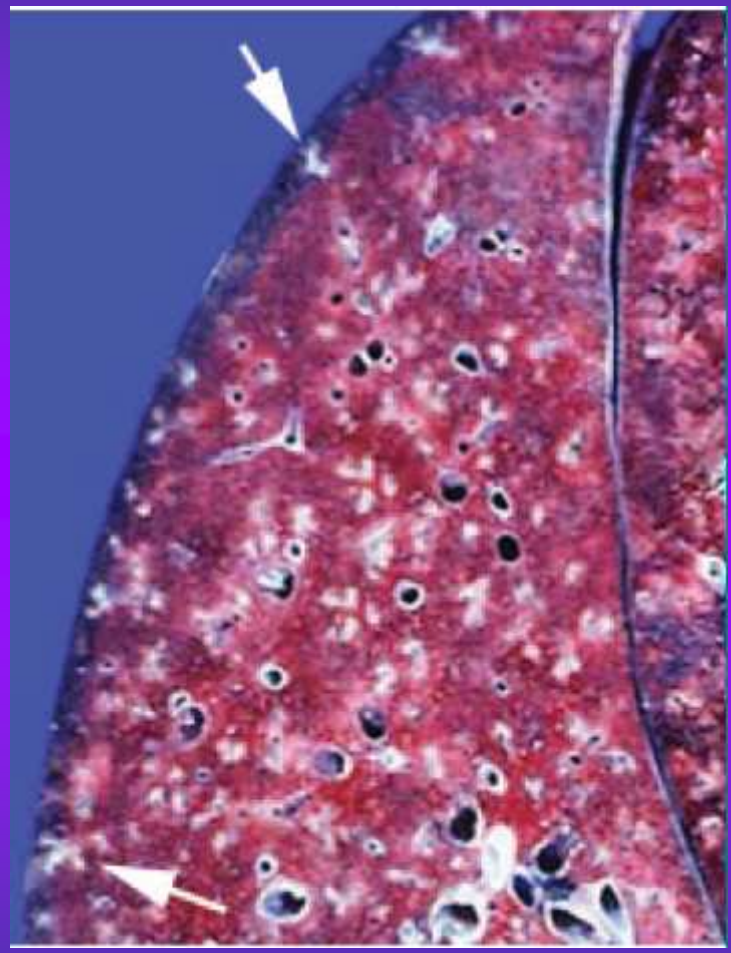
Nodular opacities



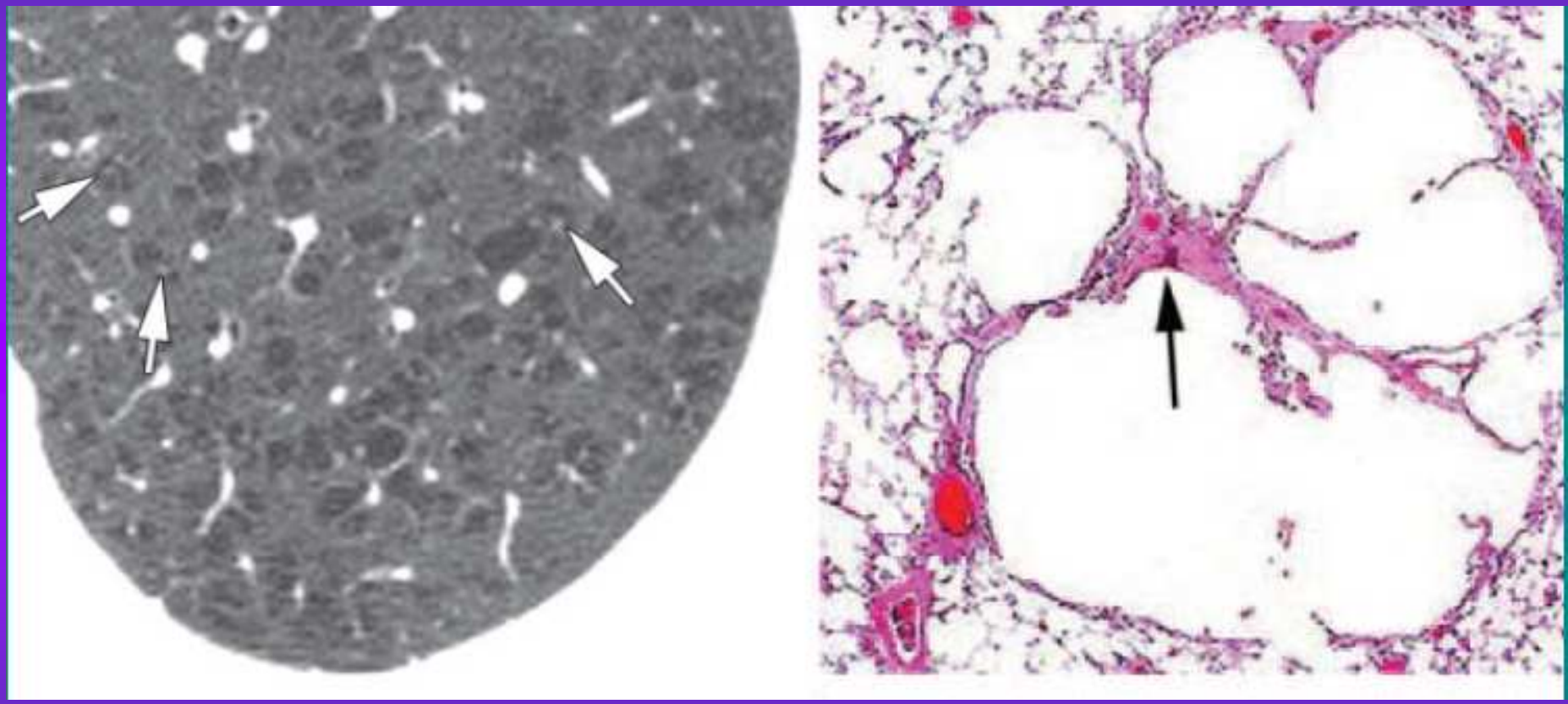


“Tree in bud”





Centrilobular low attenuation

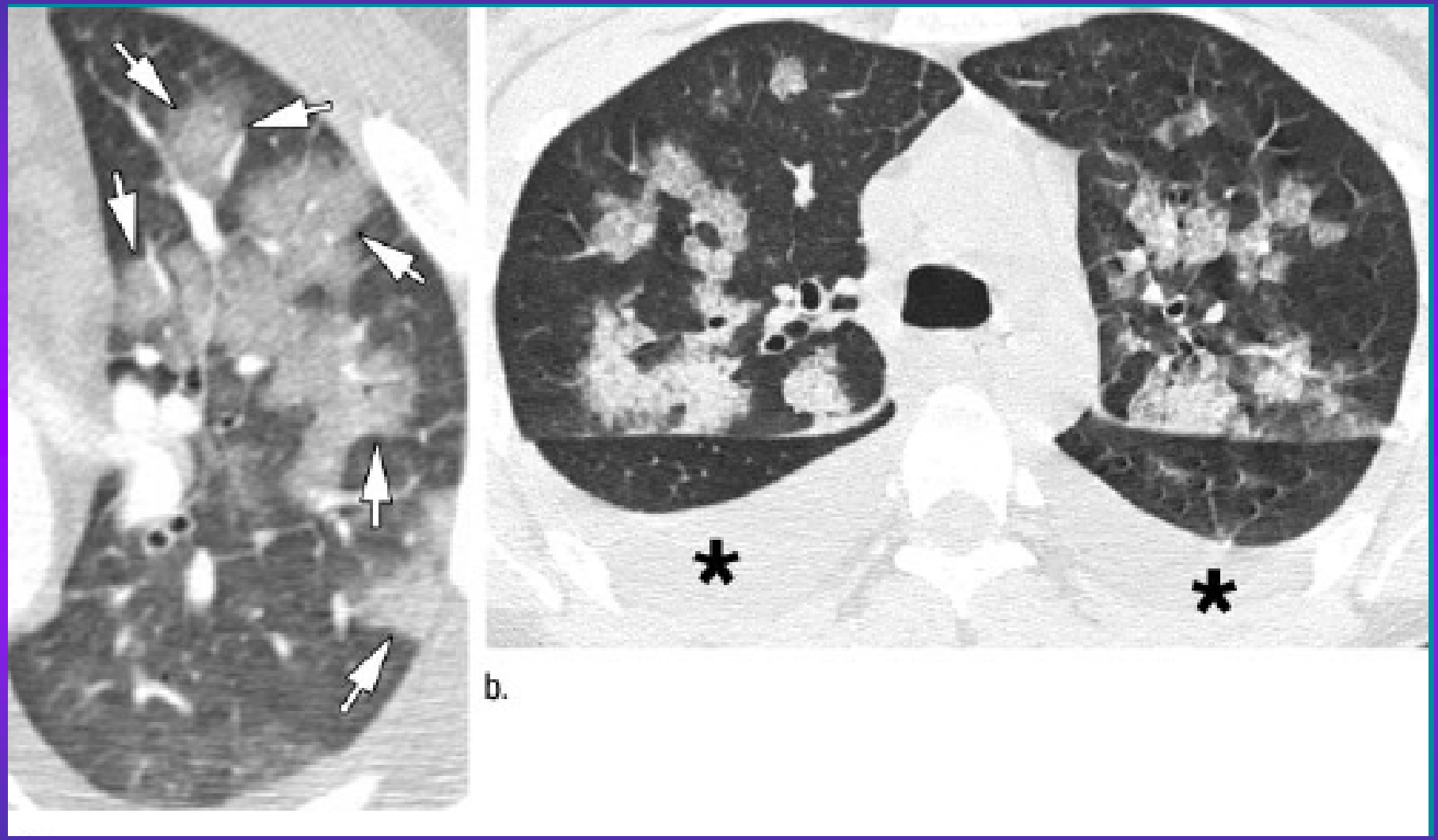


Panlobular

Table 3

Panlobular Nodules: Differential Diagnosis

CT Finding	Differential Diagnosis
Lobular consolidation	Bronchopneumonia, organizing pneumonia, eosinophilic pneumonia, bronchioloalveolar carcinoma
Lobular ground-glass opacity	Bronchopneumonia, viral infection, *Pneumocystis carinii* pneumonia, *Mycoplasma pneumoniae*, pulmonary edema, hypersensitivity pneumonitis, alveolar proteinosis, lipoid pneumonia
Lobular low attenuation	Mosaic perfusion due to airways or vascular disease, emphysema (panlobular or paraseptal)
Headcheese sign	Hypersensitivity pneumonitis, desquamative interstitial pneumonia, respiratory bronchiolitis–interstitial lung disease, sarcoidosis, atypical infections with bronchiolitis (eg, *M pneumoniae*)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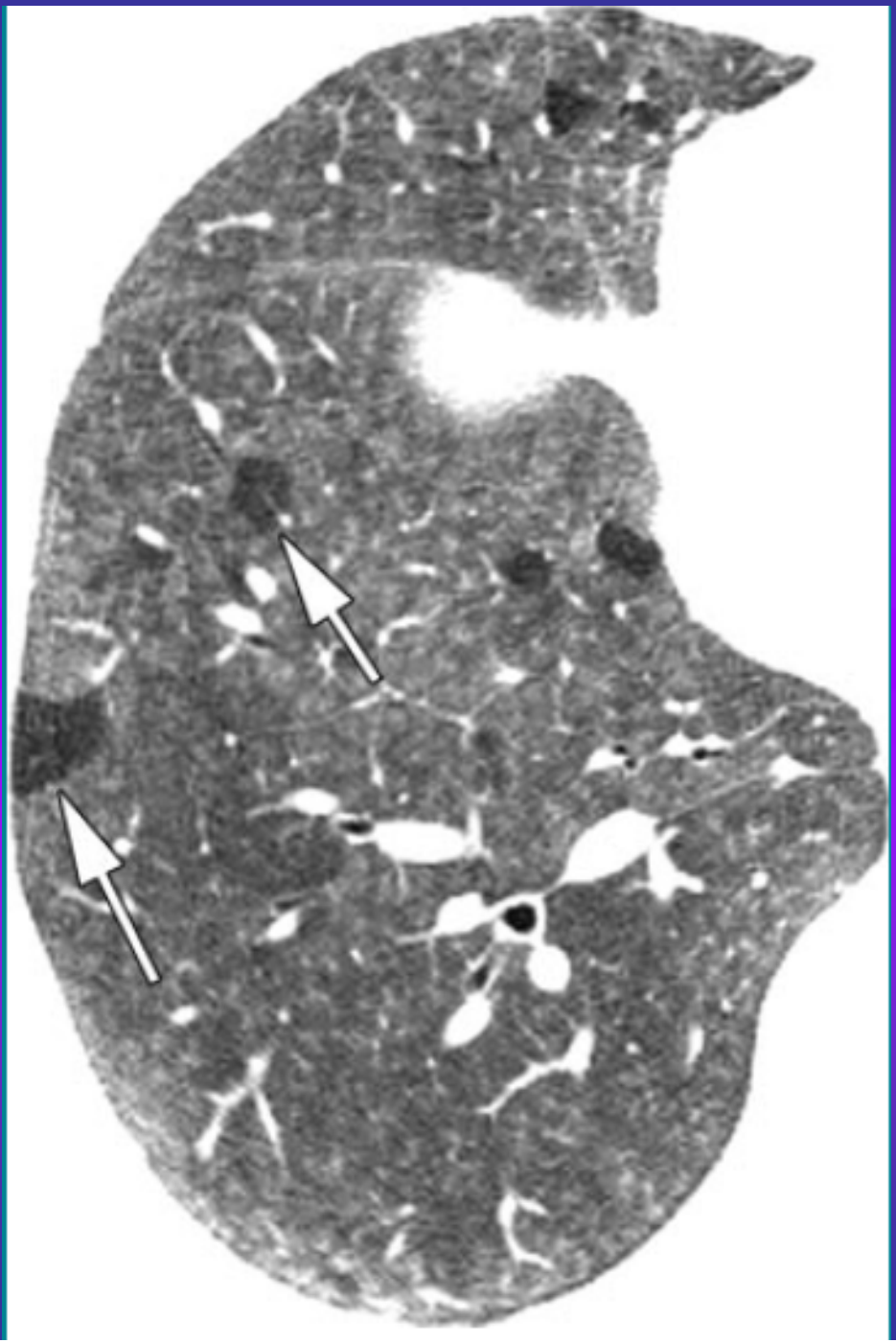
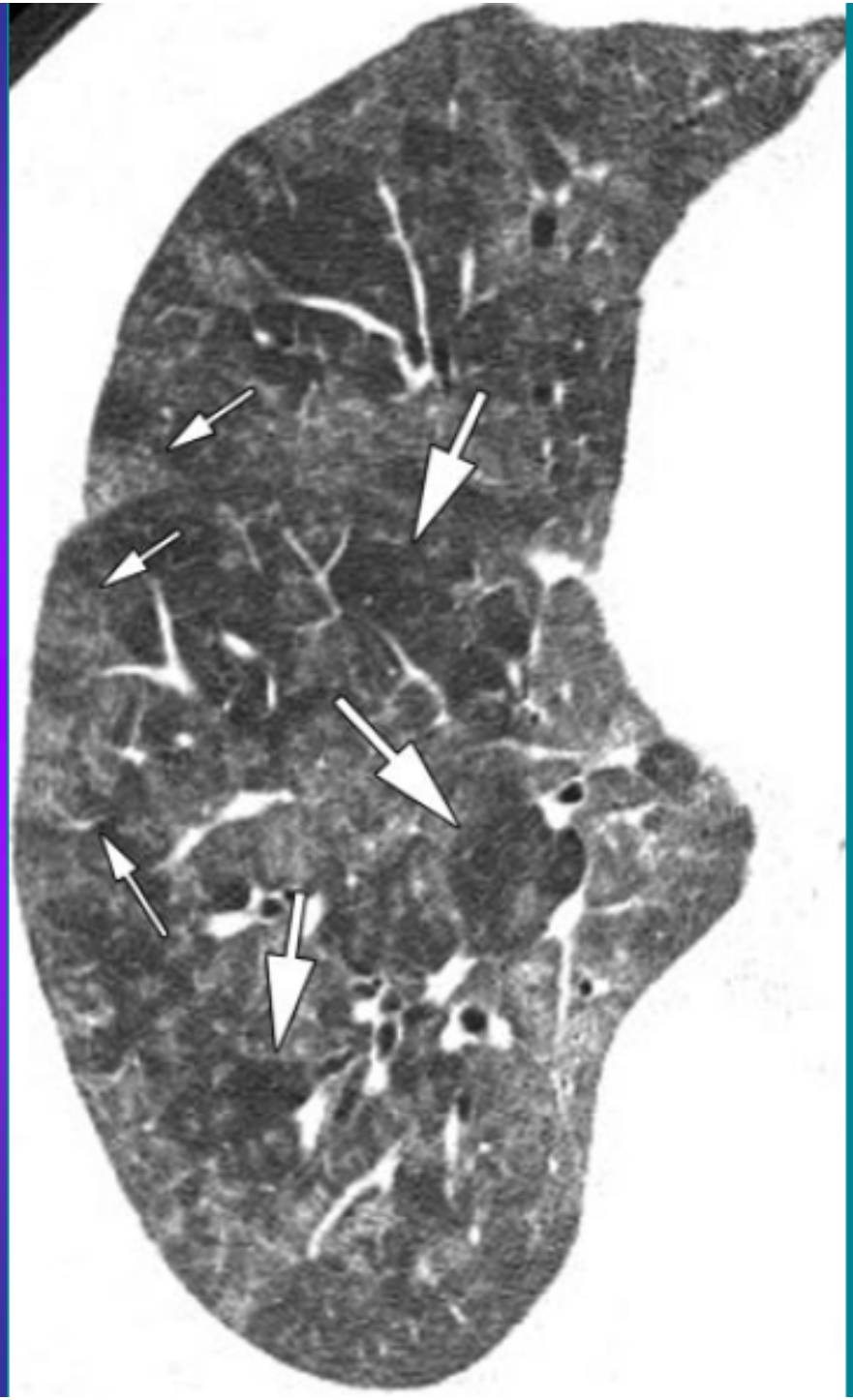
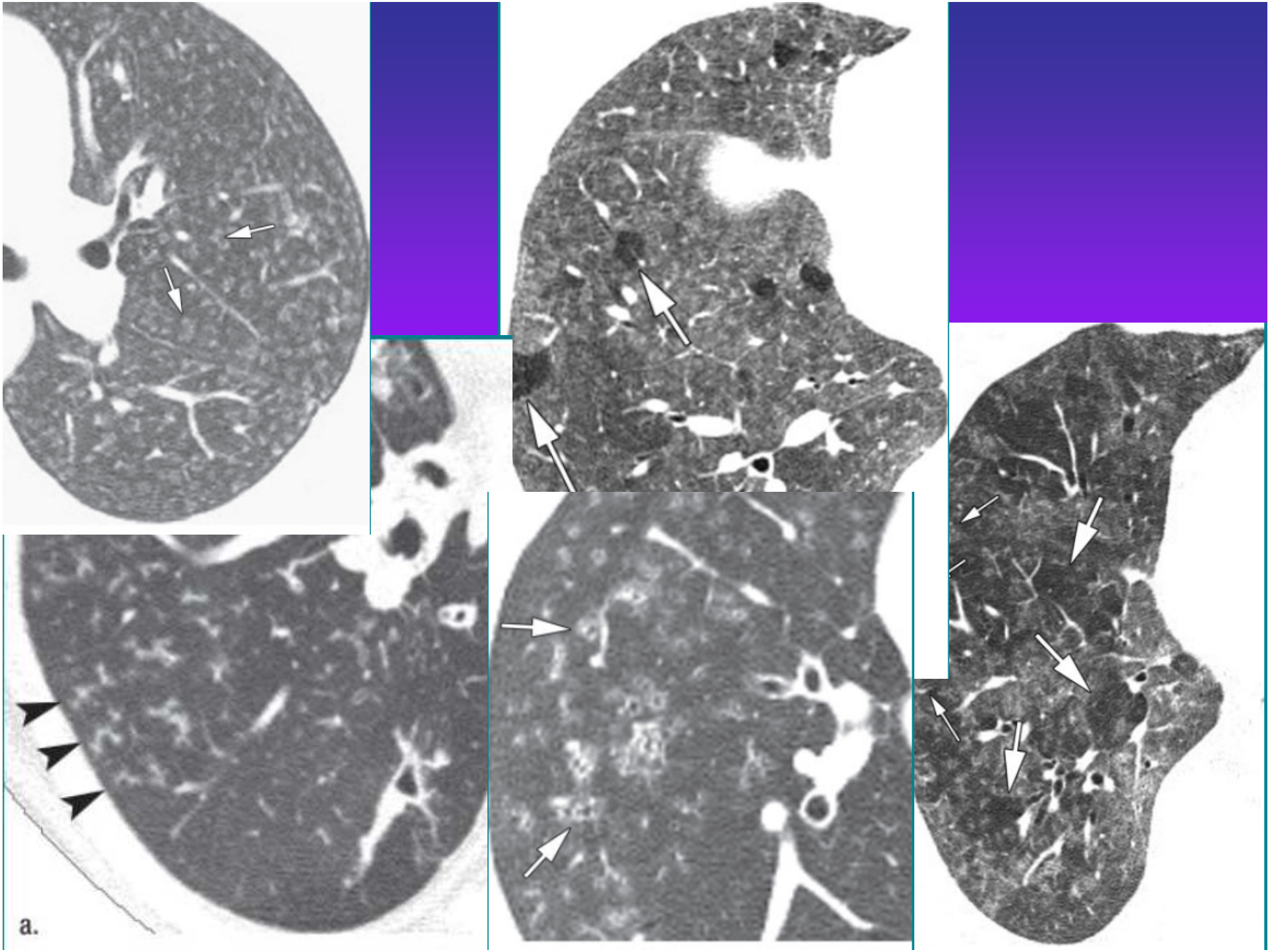
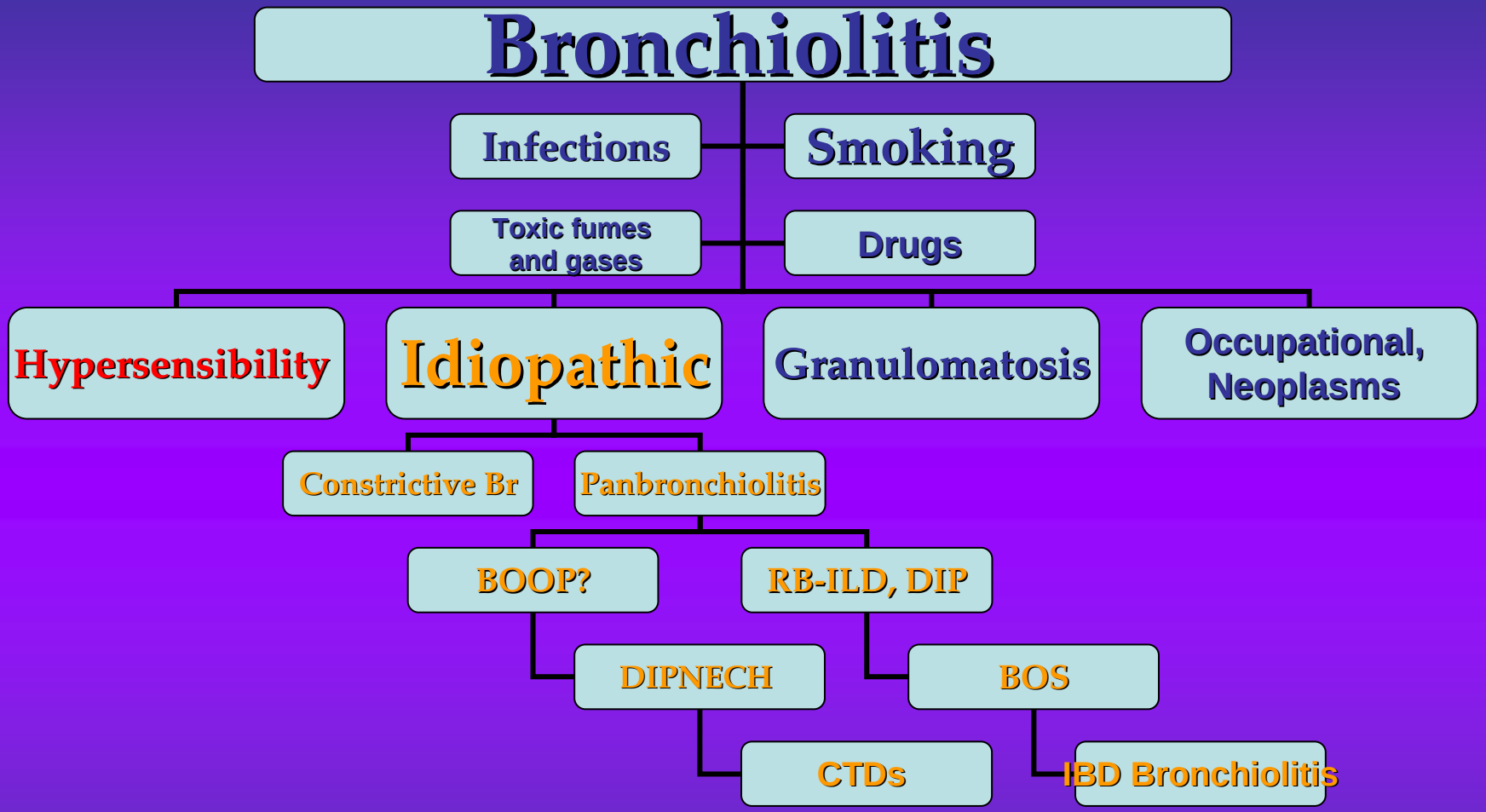





a.


Bronchiolitis
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Panbronchiolitis
BOOP?
RB-ILD, DIP
DIPNECH
BOS
CTDs
IBD Bronchiolitis

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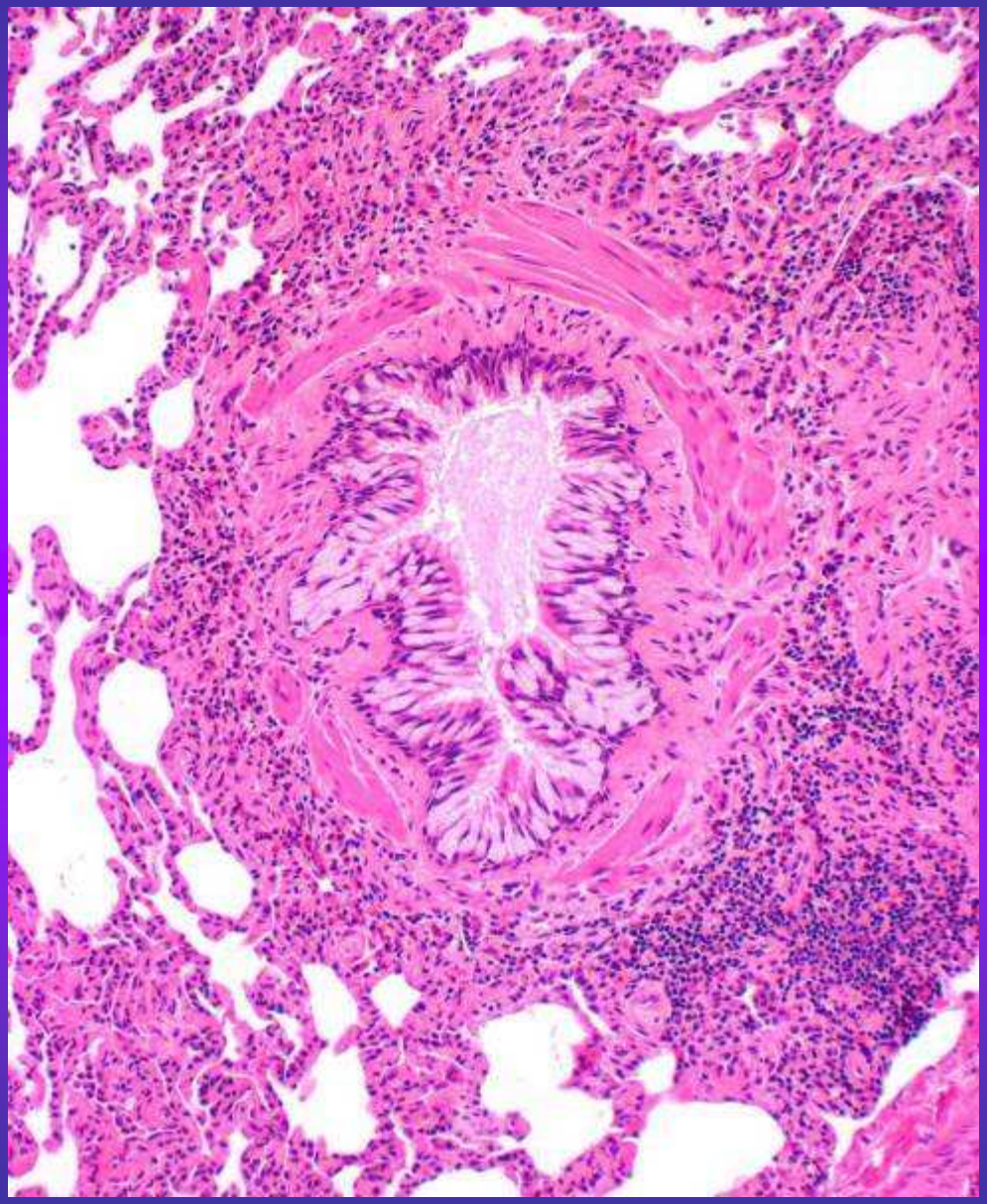
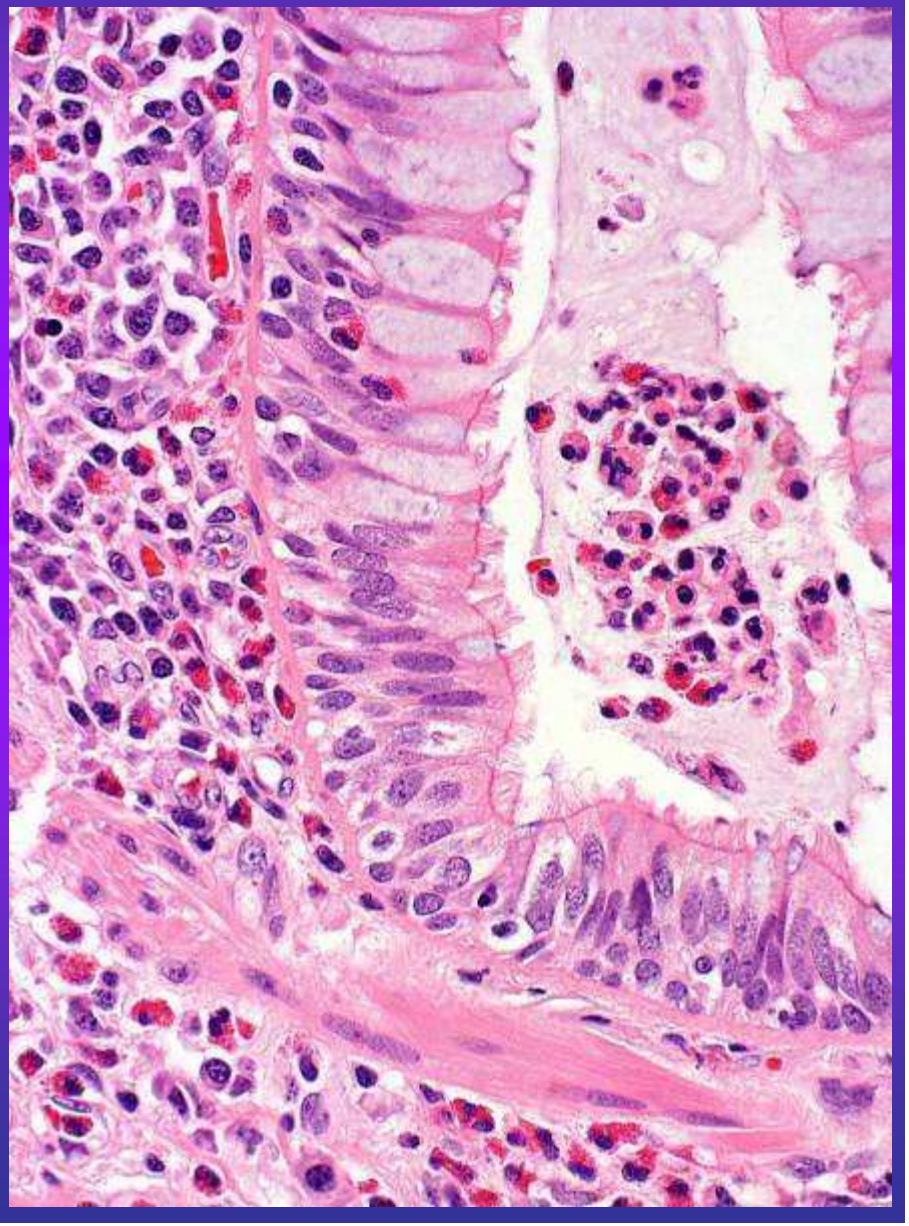
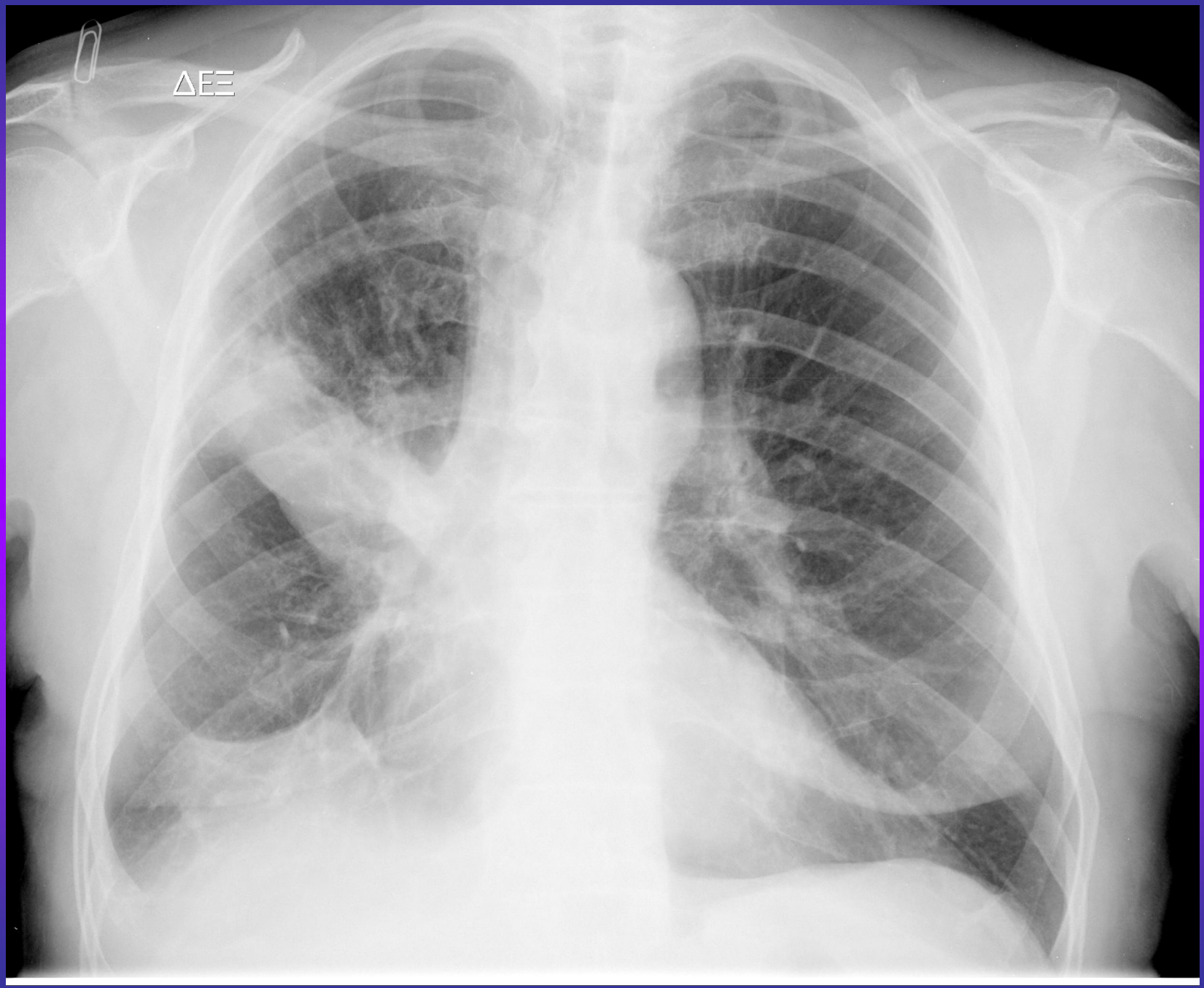
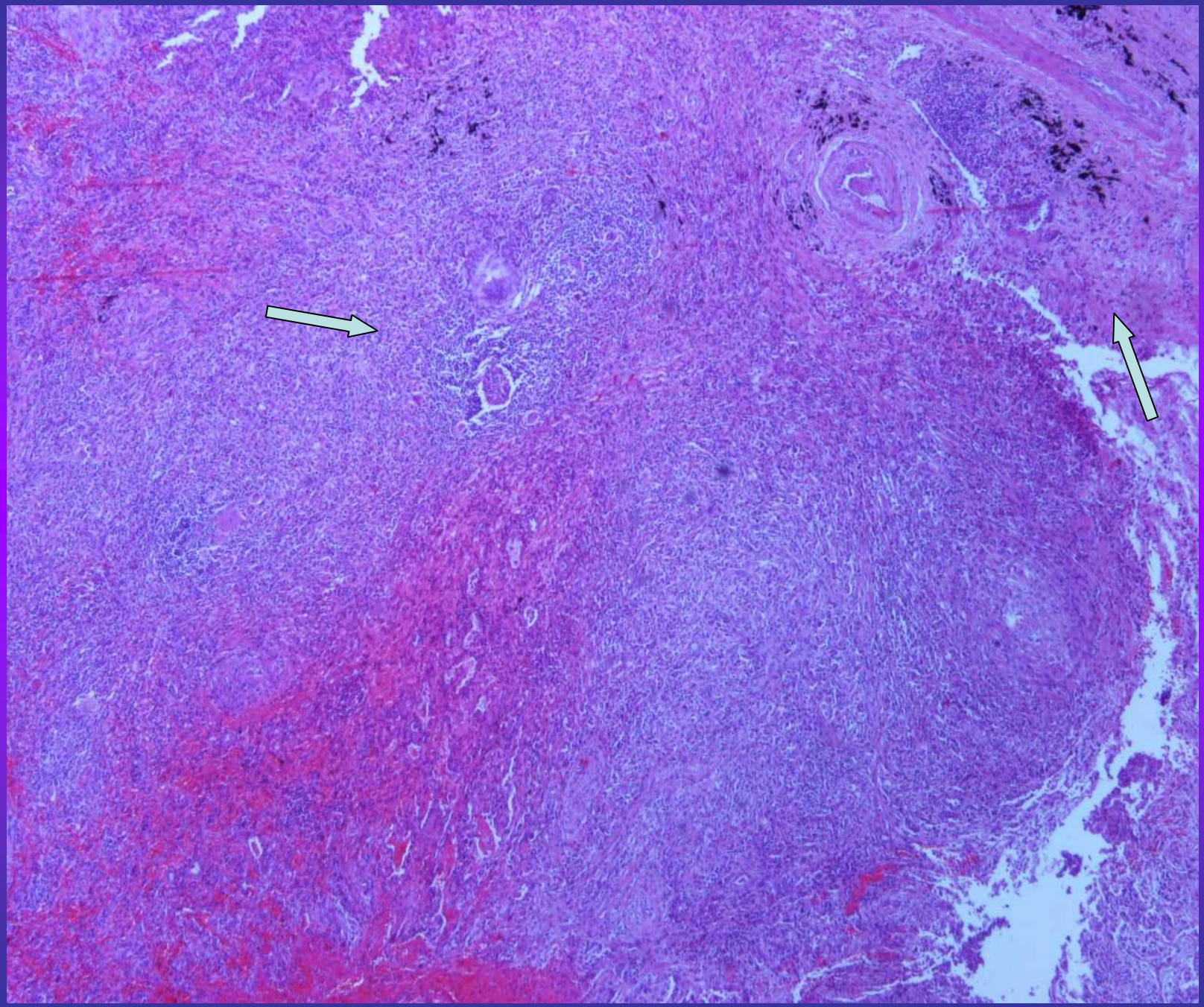
Hypersensitivity Disorders

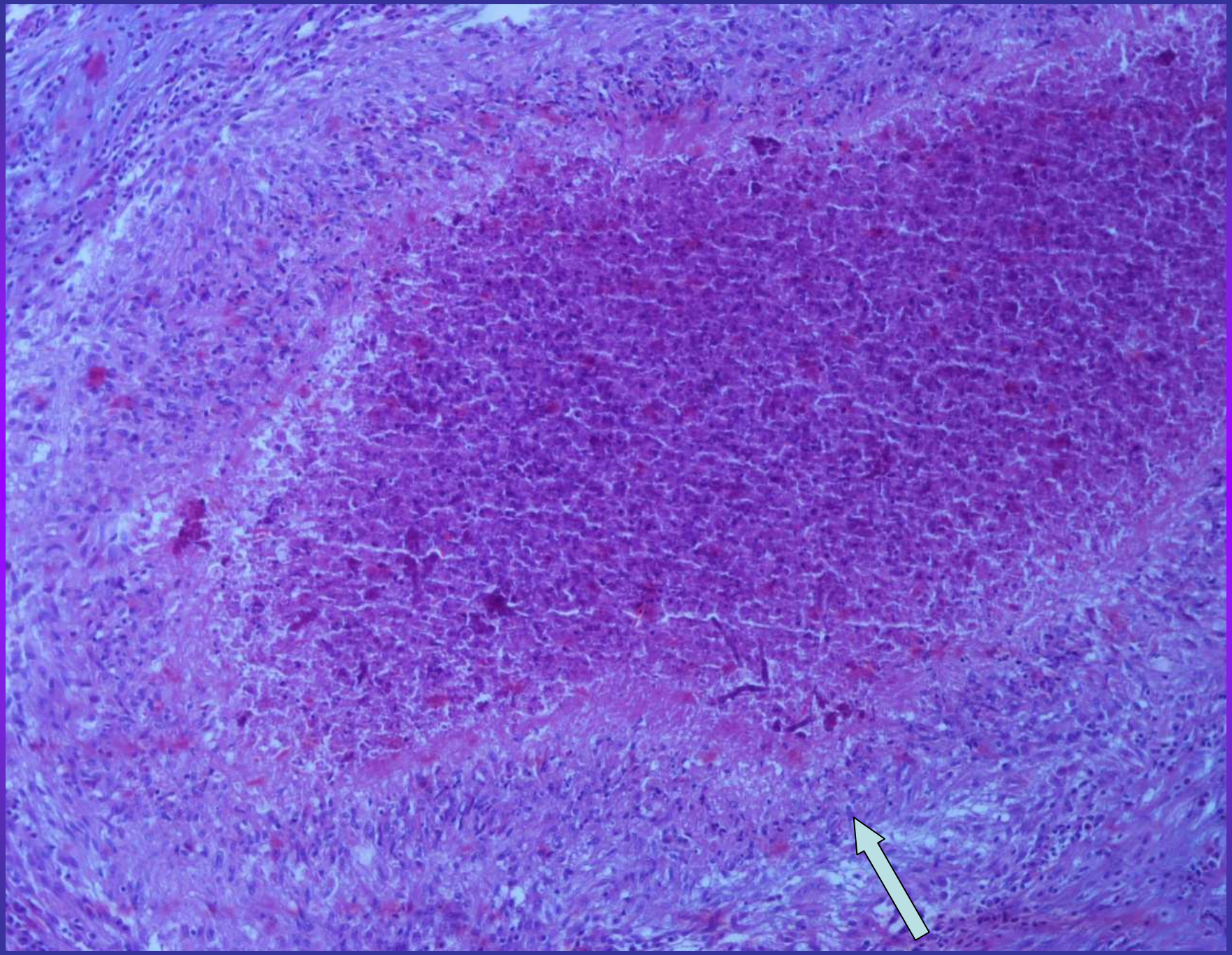
- Bronchial Asthma
- ABPA (ABPM)
- Bronchocentric granulomatosis
- Hypersensitivity Pneumonitis
- Chronic eosinophilic pneumonia
- Churg-Strauss vasculitis

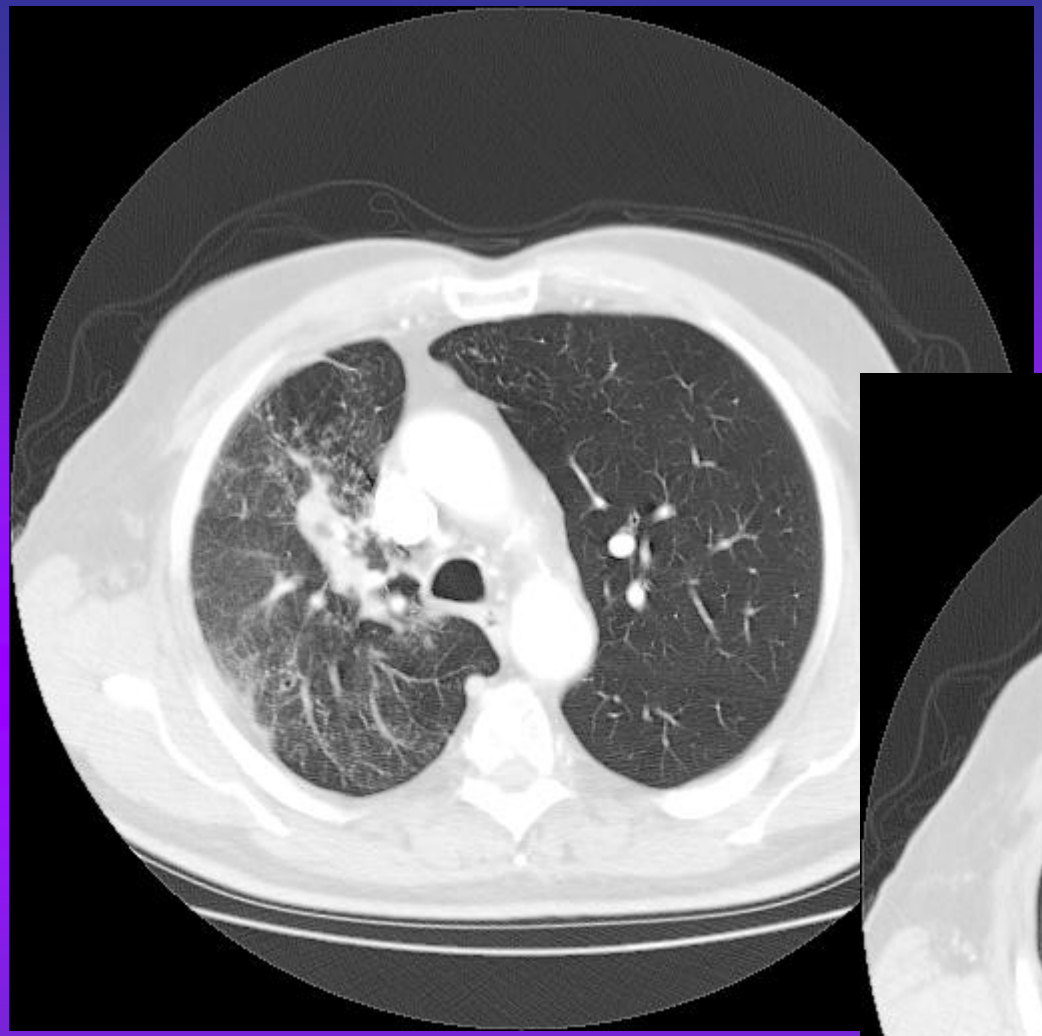




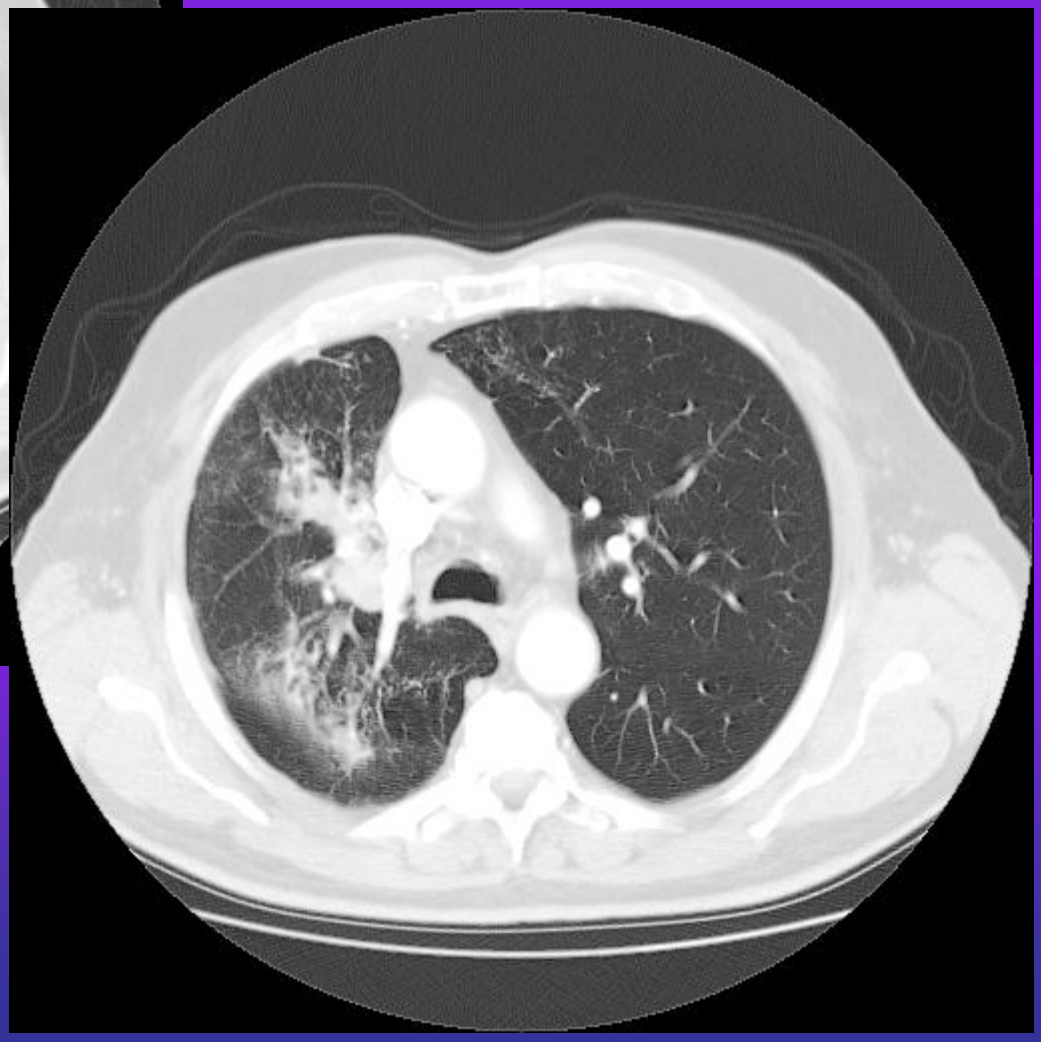

ΔΕΞ





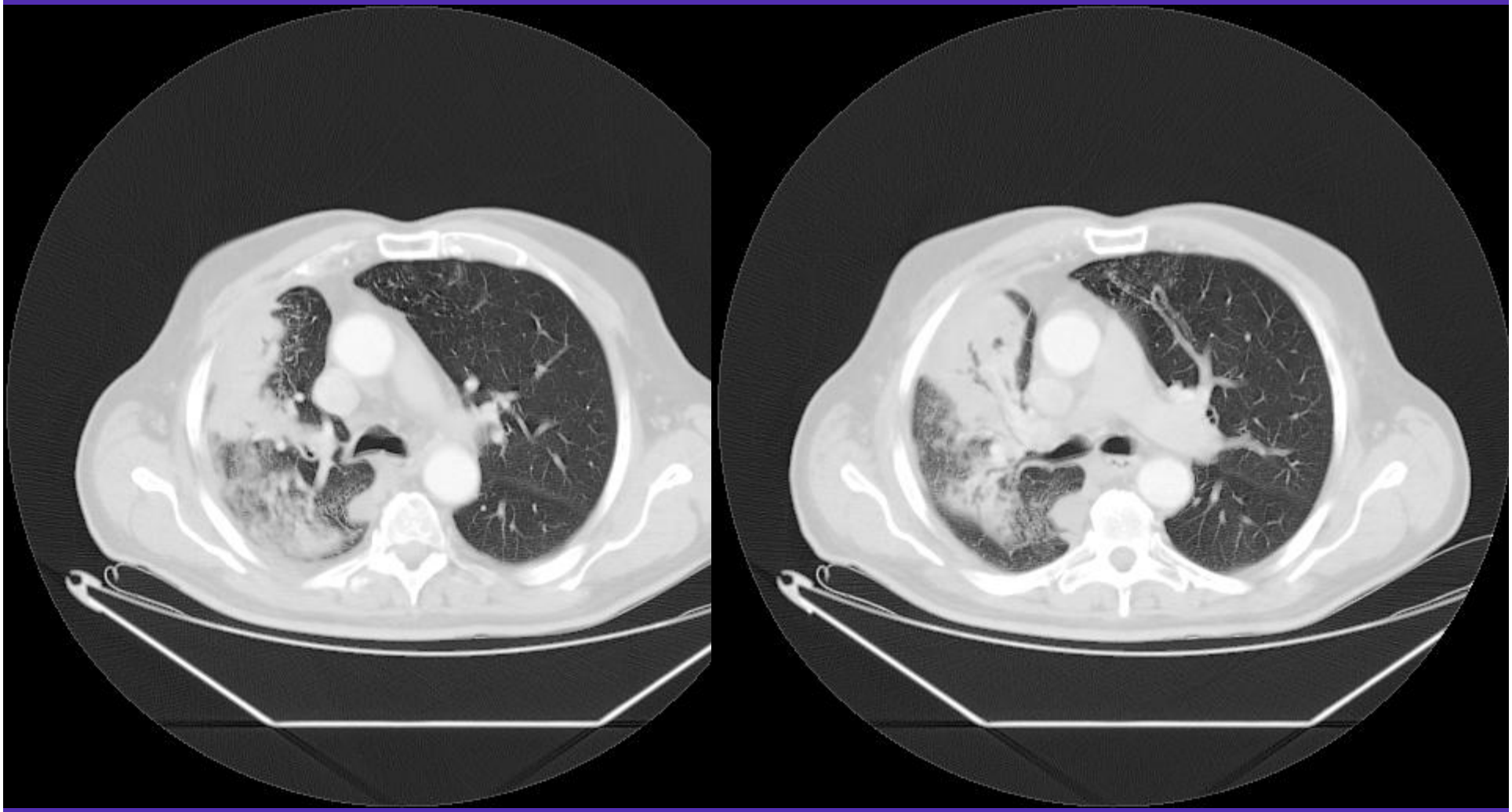


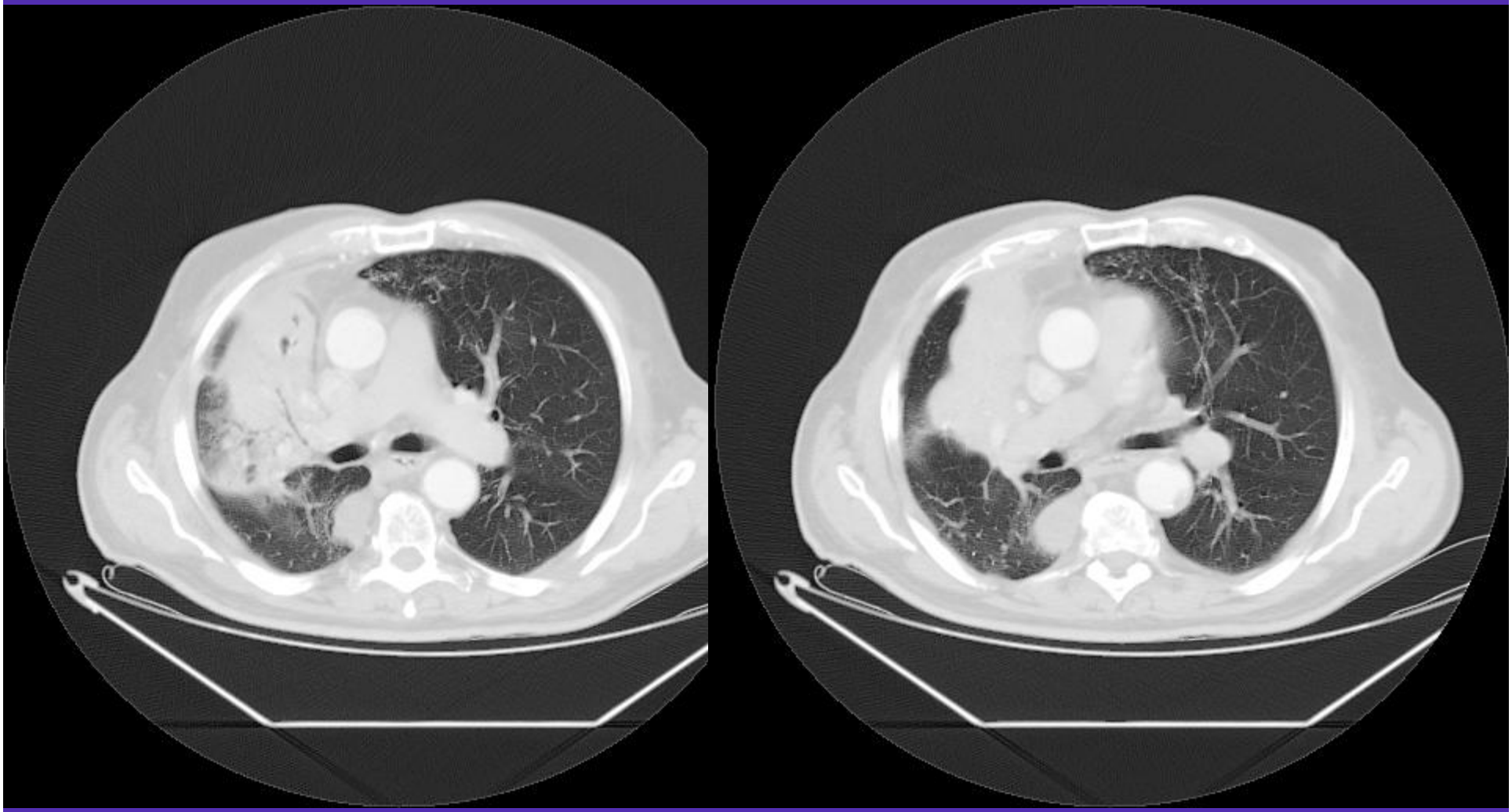
The patient was advised to be treated with corticosteroids per os but was not compliant to his treatment

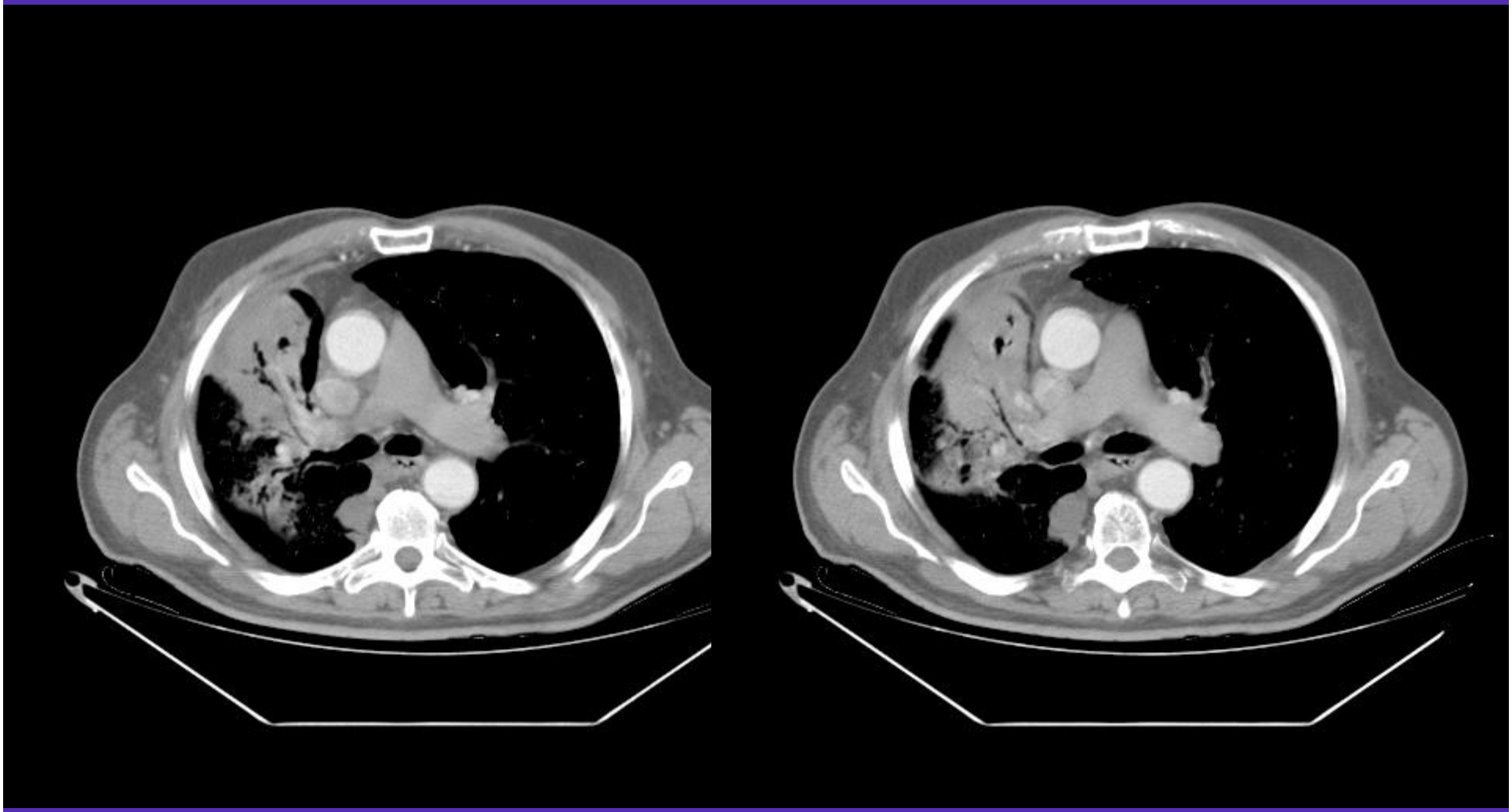


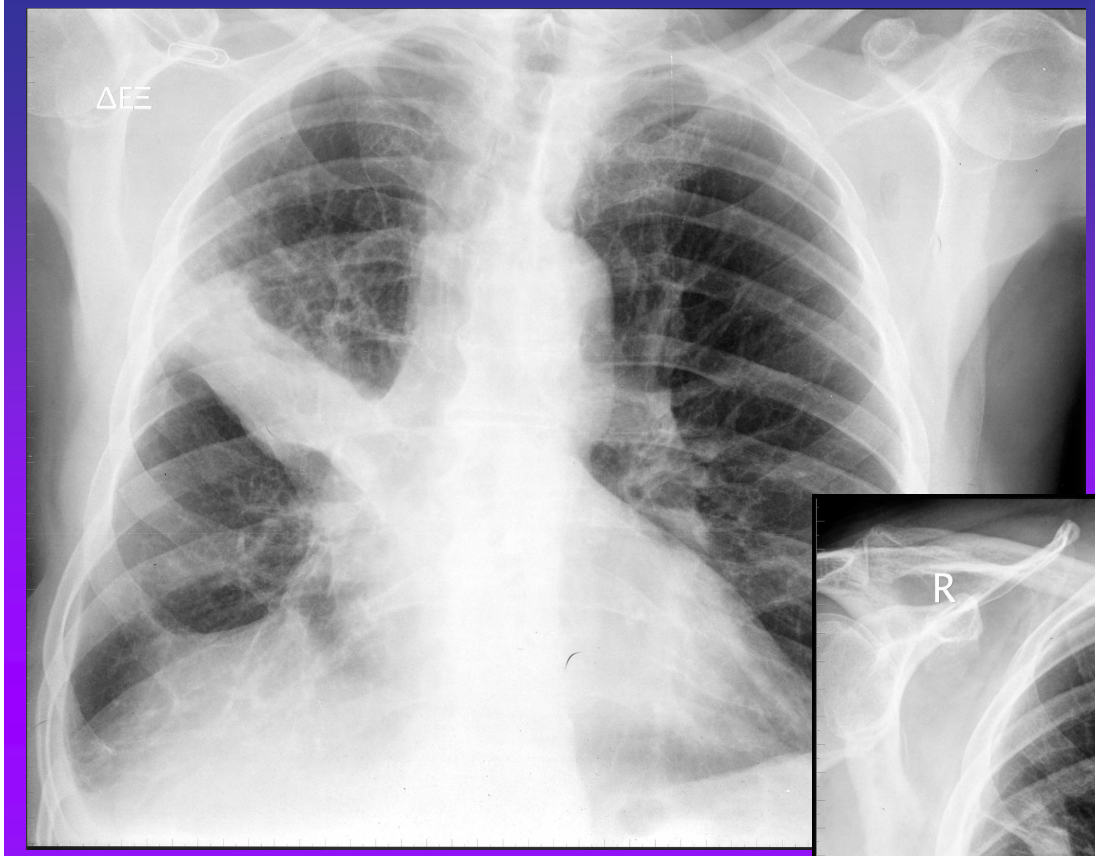
Post surgery

On admission



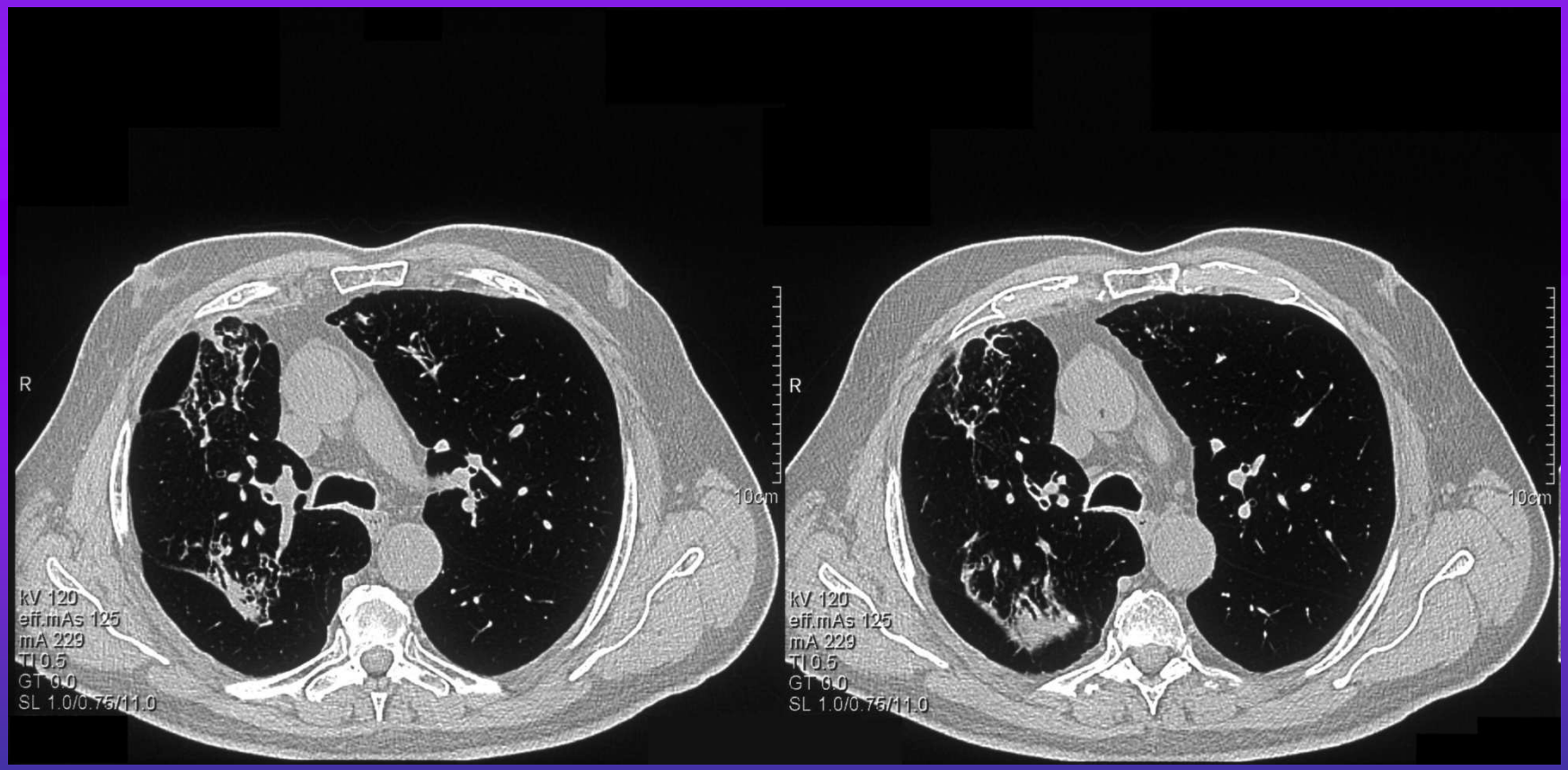


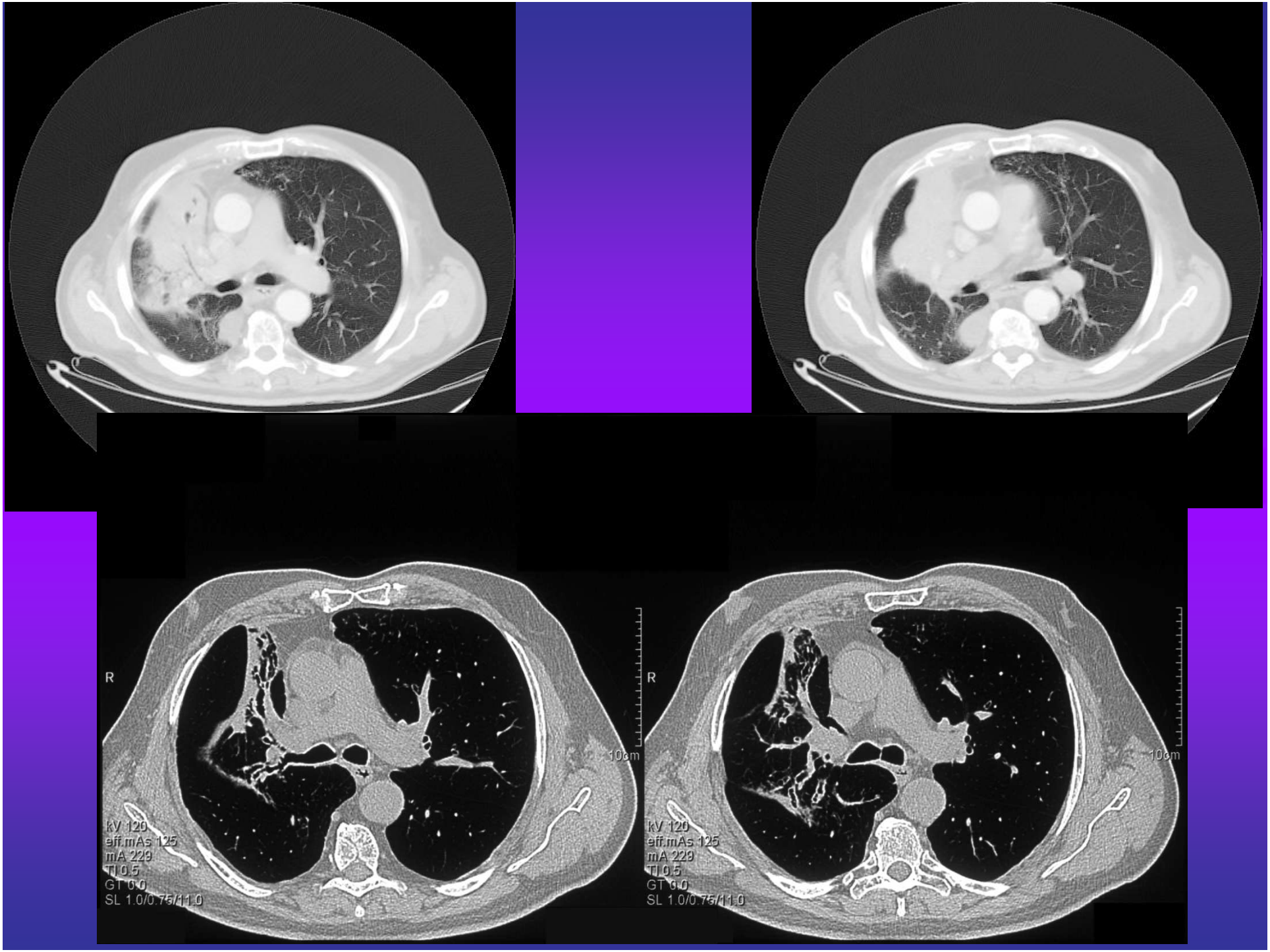


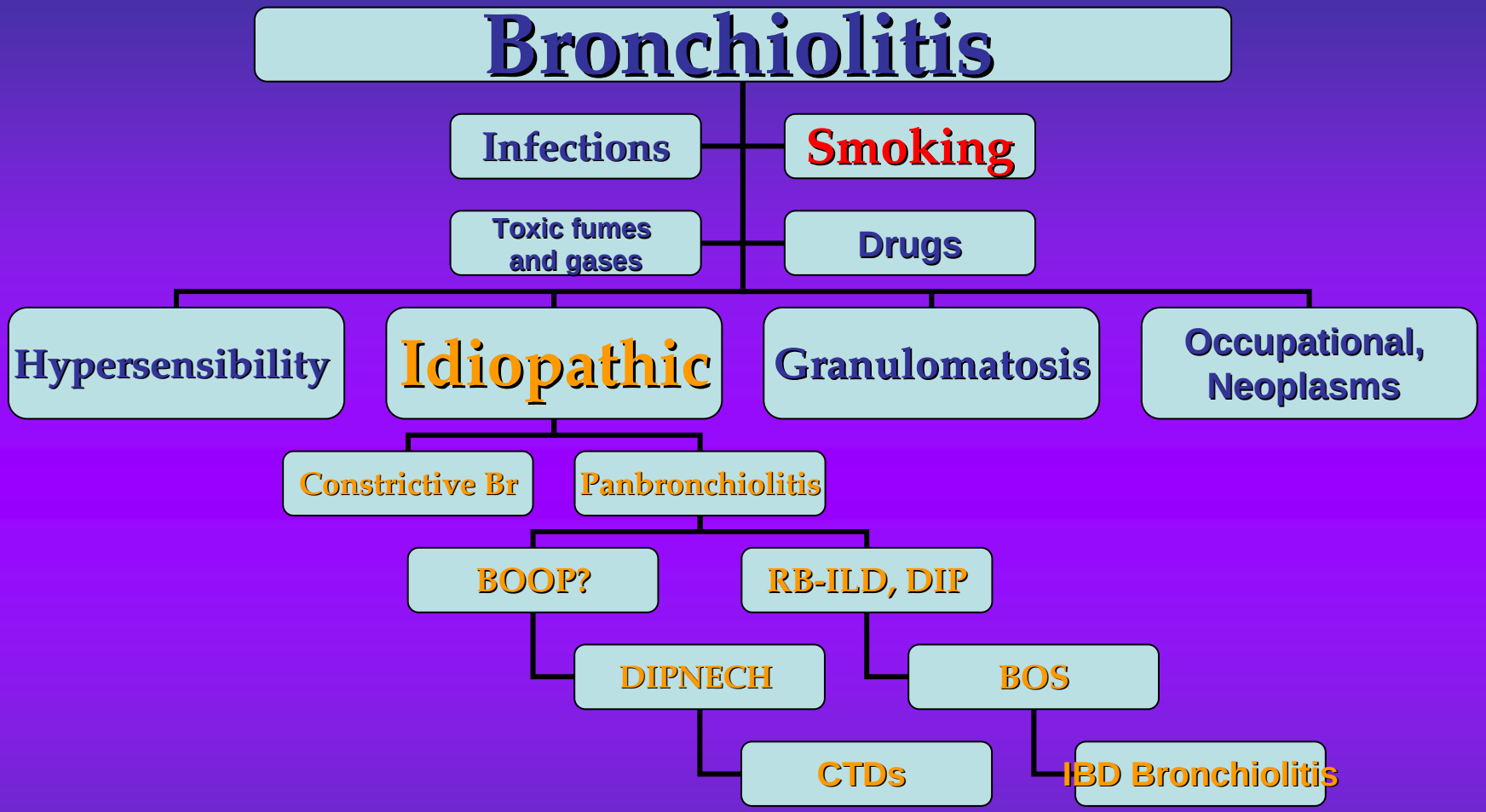

ΔΕΞ


R
DIGITAL X-RAY

The patient was treated with mycophenolate mofetil and low dose of prednisone with significant response to tx




R
10cm
kV 120
eff.mAs 125
mA 229
TI 0.5
GT 0.0
SL 1.0/0.75/11.0
R
10cm
kV 120
eff.mAs 125
mA 229
TI 0.5
GT 0.0
SL 1.0/0.75/11.0


Bronchiolitis
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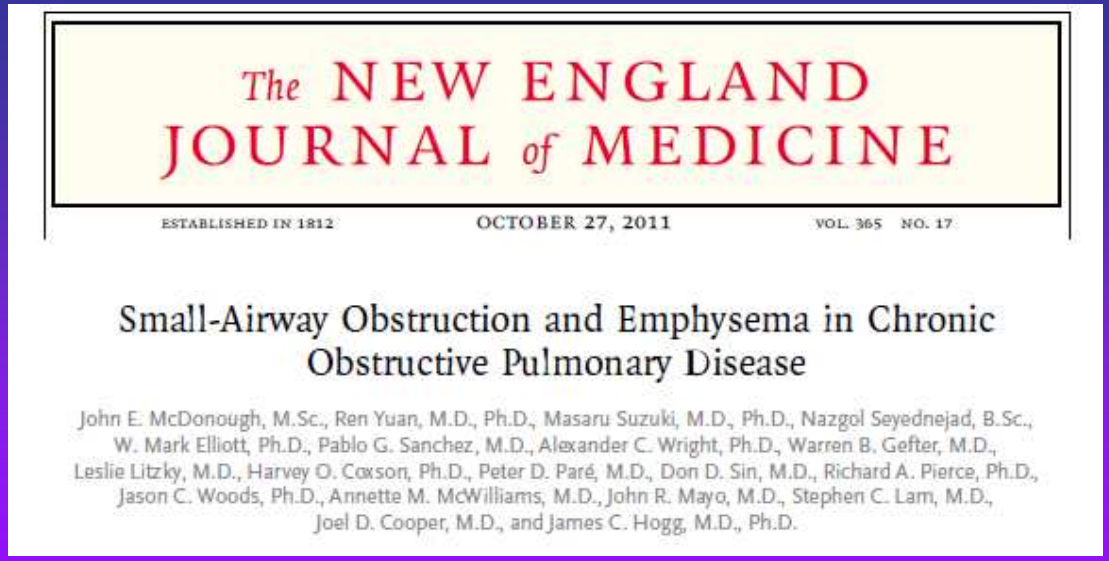
SMOKING

- Chronic Bronchitis Bronchiolitis
- Centrilobular empysema
- Panlobular empysema
- RB-ILD
- Langerhan's cells granulomatosis H-x



The NEW ENGLAND JOURNAL of MEDICINE

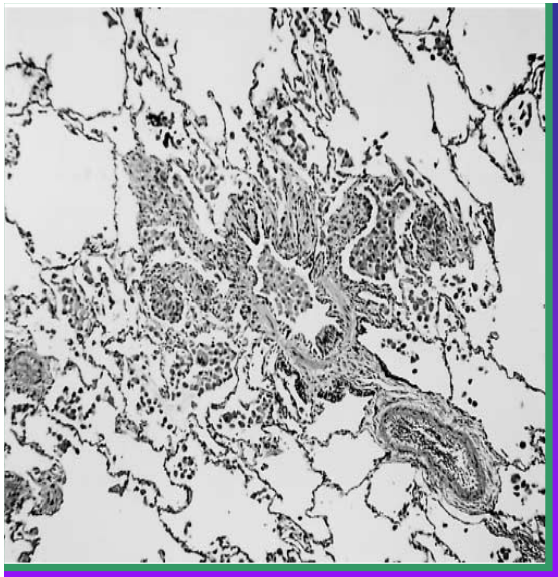
ESTABLISHED IN 1812 OCTOBER 27, 2011 VOL. 365 NO. 17

Small-Airway Obstruction and Emphysema in Chronic Obstructive Pulmonary Disease

John E. McDonough, M.Sc., Ren Yuan, M.D., Ph.D., Masaru Suzuki, M.D., Ph.D., Nazgol Seyednejad, B.Sc., W. Mark Elliott, Ph.D., Pablo G. Sanchez, M.D., Alexander C. Wright, Ph.D., Warren B. Gefter, M.D., Leslie Litzky, M.D., Harvey O. Coxson, Ph.D., Peter D. Paré, M.D., Don D. Sin, M.D., Richard A. Pierce, Ph.D., Jason C. Woods, Ph.D., Annette M. McWilliams, M.D., John R. Mayo, M.D., Stephen C. Lam, M.D., Joel D. Cooper, M.D., and James C. Hogg, M.D., Ph.D.

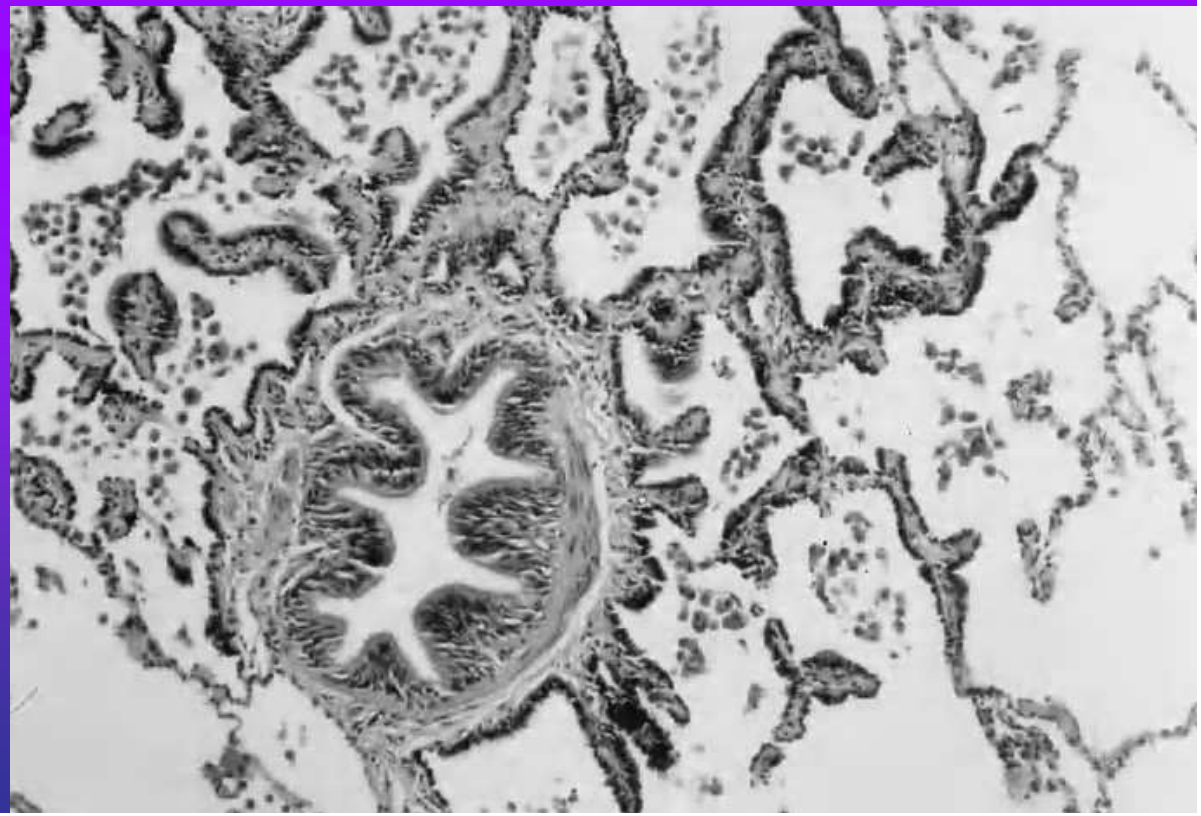
The micro CT results extend earlier reports by showing that there is both widespread narrowing and loss of smaller conducting airways before the onset of emphysematous destruction in both centrilobular and panlobular emphysema phenotypes of COPD.

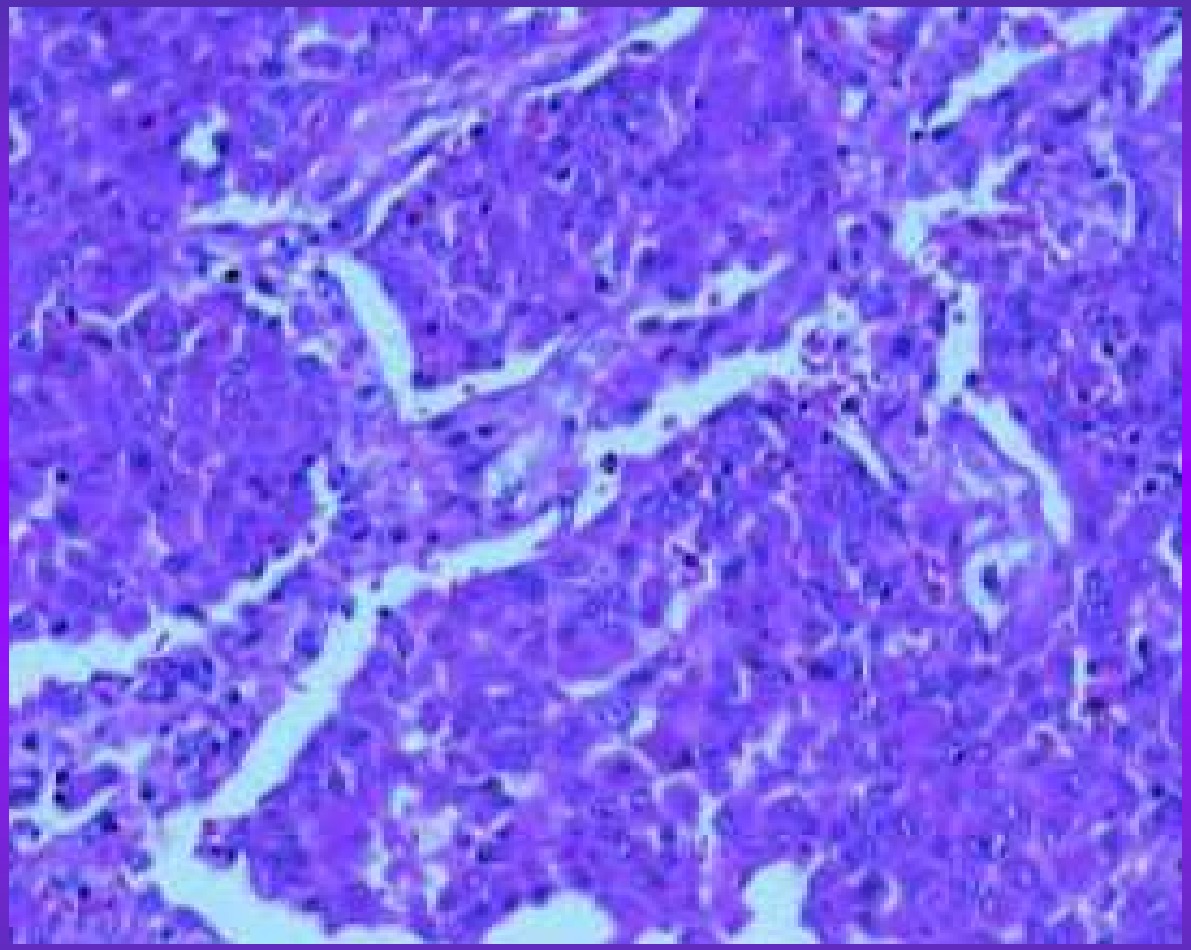
This process readily explains the observed increase by a factor of 4 to 40 in small-airway resistance in patients with COPD



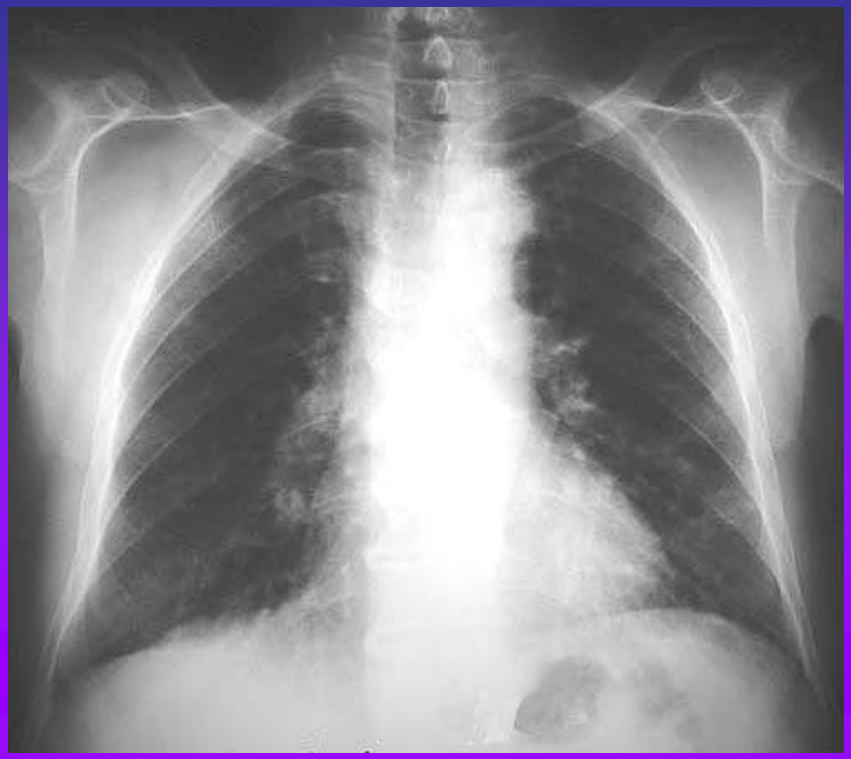
RB-ILD / DIP

RB occurs as an incidental finding in smokers, so it is impossible to know whether it is the main problem or there is some other lesion not been sampled





A surgical lung biopsy is required to make a confident diagnosis of RB-ILD and associated DIP.

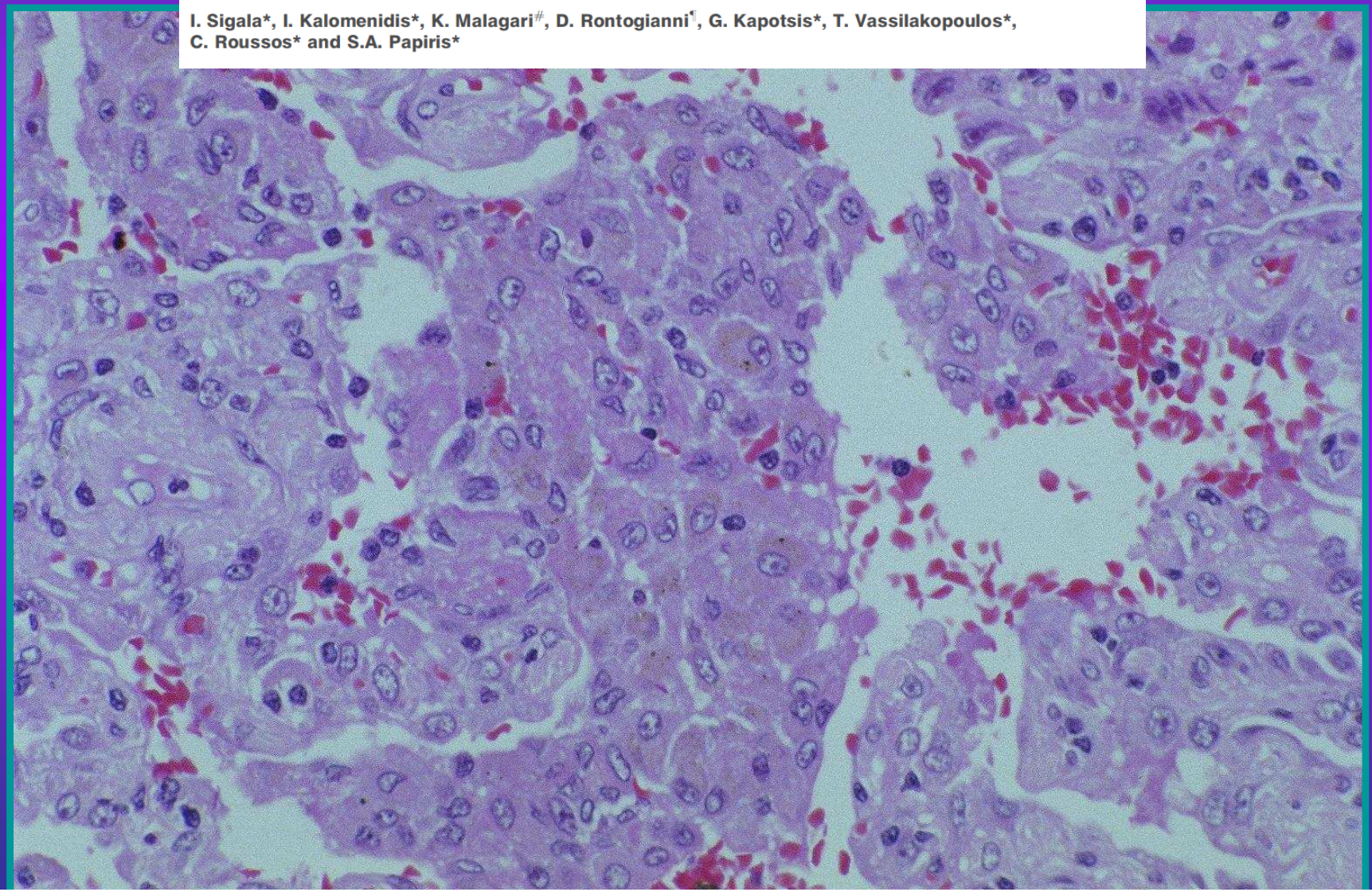


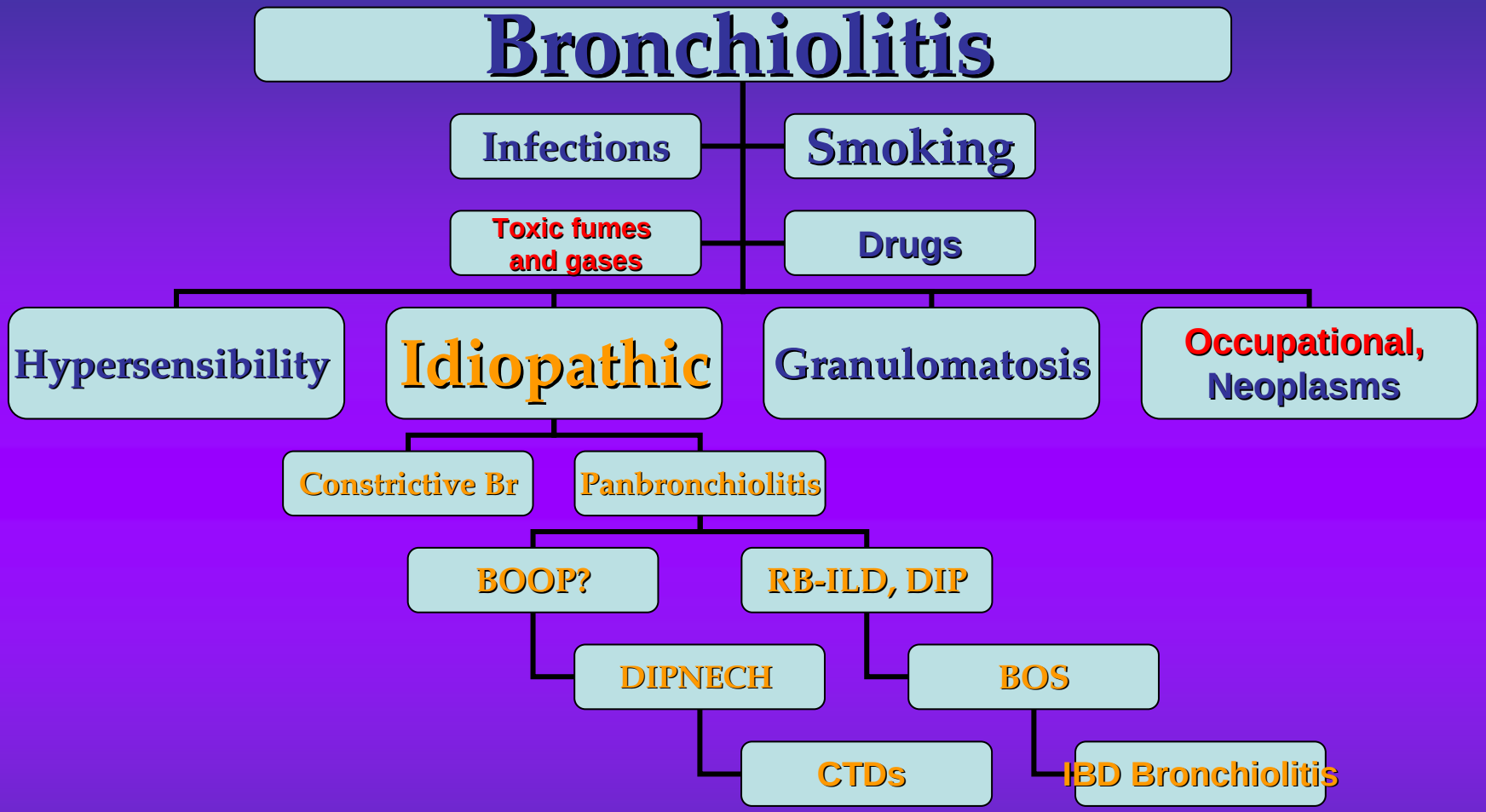
Eur Respir J 2005; 25: 1122–1125
DOI: 10.1183/09031936.05.00088304

CASE FOR DIAGNOSIS

Dry cough and dyspnoea rapidly increasing to respiratory failure in a male smoker

I. Sigala*, I. Kalomenidis*, K. Malagari#, D. Rontogianni¶, G. Kapotsis*, T. Vassilakopoulos*, C. Roussos* and S.A. Papiris*

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IBD Bronchiolitis

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Toxic Exposures Associated with Bronchiolitis, with or without Obliterans

- Nitrogen dioxide ("nitrous fume")*
  - Spillage of nitric acid (component of jet and missile fuels)
  - Metal pickling
  - Silo gas
  - Chemical manufacturing (explosives, dyes, lacquers, celluloid)
  - Detonation of explosives
  - Electric arc or acetylene gas welding
  - Contamination of anesthetic gases (nitrous oxide gas cylinder)
  - Nitrocellulose combustion
  - Tobacco smoke
  - Fire smoke (firemen, astronauts, others exposed to burning materials)*,†
- Sulfur dioxide†
  - Burning of sulfur-containing fossil fuels
  - Bleaching of wool, straw, wood pulp
  - Sugar refining, fruit preserving
  - Fungicides
  - Refrigerants
  - Ore smelting
  - Acid production
- Ammonia†
  - Fertilizer and explosives, production, refrigeration

Chlorine[+]
Bleaching, disinfectant and plastic making
Phosgene*
Chemical industry, dye and insecticide manufacturing
Chloropicrin
Trichlorethylene
Ozone
Arc welding and air, sewage, and water treatment
Cadmium oxide
Ore smelting, alloying, and welding
Methyl sulfate
Hydrogen sulfide
Natural gas retrieval, paper pulp, sewage treatment, tannery work
Hydrogen fluoride
Etching, petroleum industry, silk working
Talcum powder (hydrous magnesium silicate)
Stearate of zinc powder
Oxygen toxicity
Asbestos (chrysotile and amphibole)
Iron oxide[§]
Aluminum oxide[§]
Silica[§]
Sheet silicates (talc, mica, etc.)[§]

Coal[§]
Activated charcoal
Talc
Free-base cocaine*
Chemical weapons (mustard gas and the nerve gases, sarin, VX, and tabun)[†]

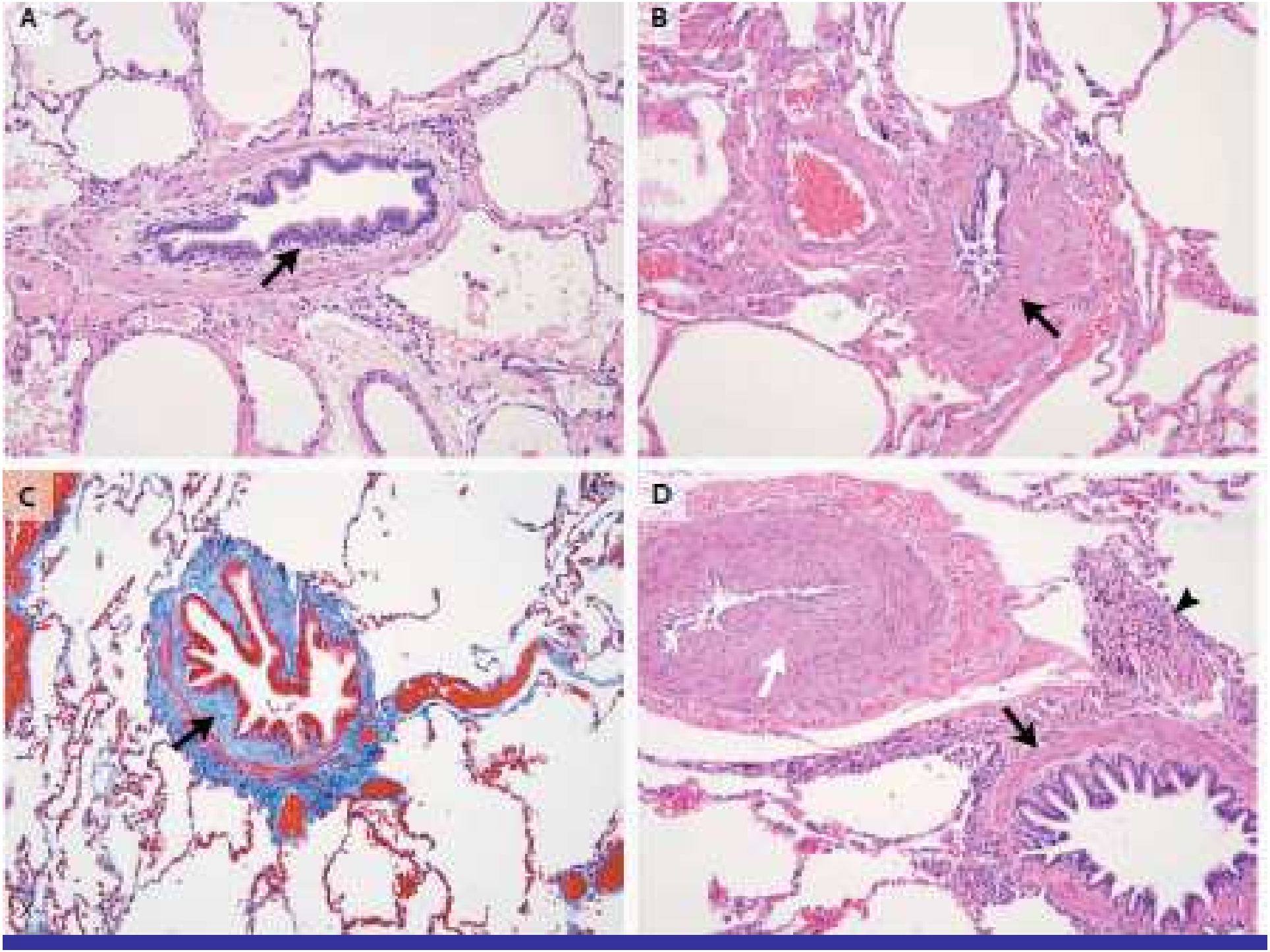
ORIGINAL ARTICLE

Constrictive Bronchiolitis in Soldiers Returning from Iraq and Afghanistan

Matthew S. King, M.D., Rosana Eisenberg, M.D., John H. Newman, M.D., James J. Tolle, M.D., Frank E. Harrell, Jr., Ph.D., Hui Nian, Ph.D., Mathew Ninan, M.D., Eric S. Lambright, M.D., James R. Sheller, M.D., Joyce E. Johnson, M.D., and Robert F. Miller, M.D.

Table 3. Pathological Features of Biopsy Samples Obtained from 38 Soldiers with Constrictive Bronchiolitis.

Variable	No. of Patients
Bronchiolar luminal constriction*	38
Predominant constrictive stroma	
Smooth muscle	7
Fibrous tissue	3
Mixed	28
Pigment deposition	37
Polarizable material within pigment	36
Peribronchiolar inflammation	34
Hypertensive-type arterial change	28
Respiratory bronchiolitis	27
Prominent bronchial-associated lymphoid tissue	19
Mucus plugging	13
Eosinophils in bronchiolar wall	7
Luminal granulation	2
Obliteration of bronchioles	0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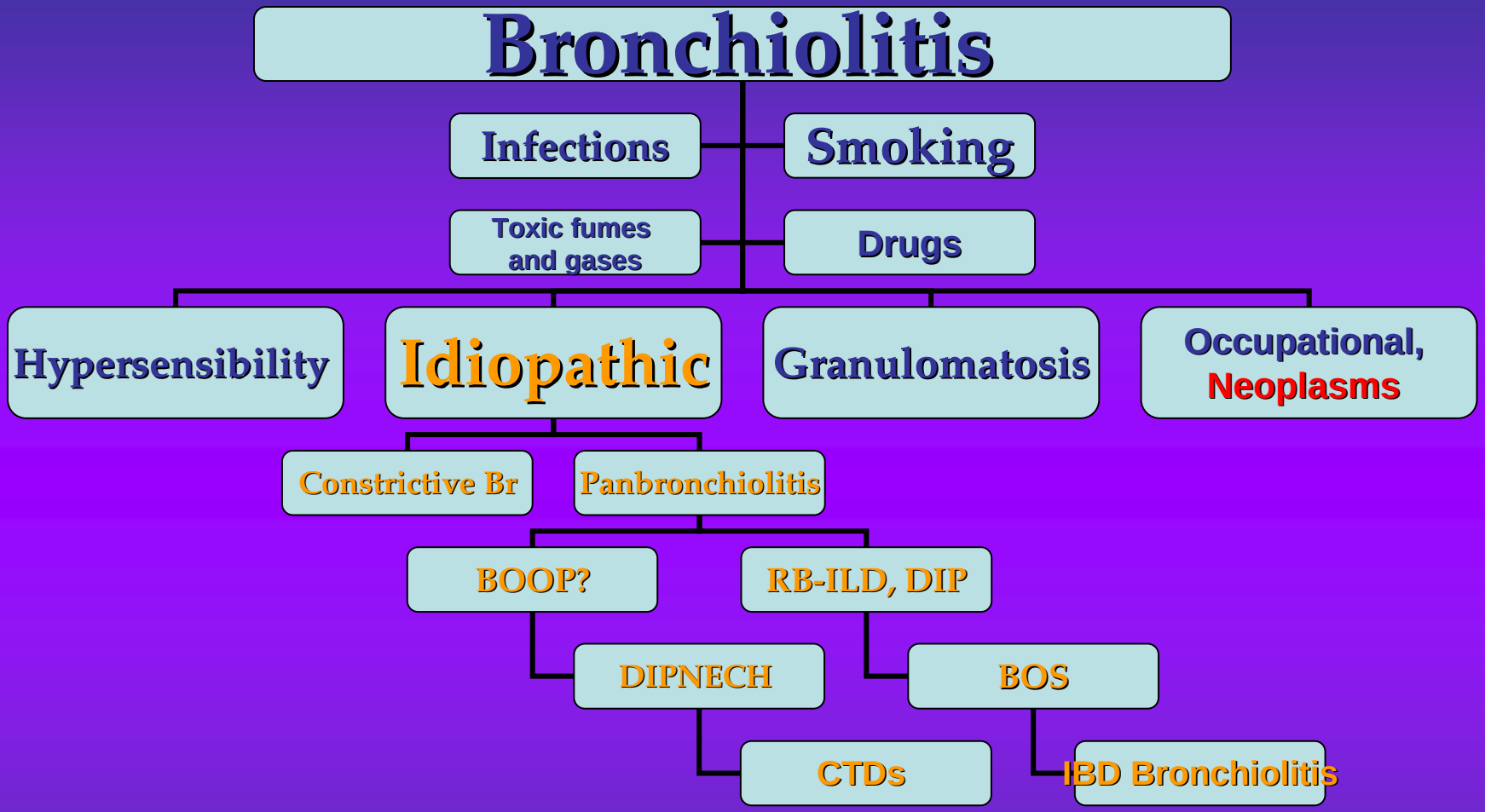
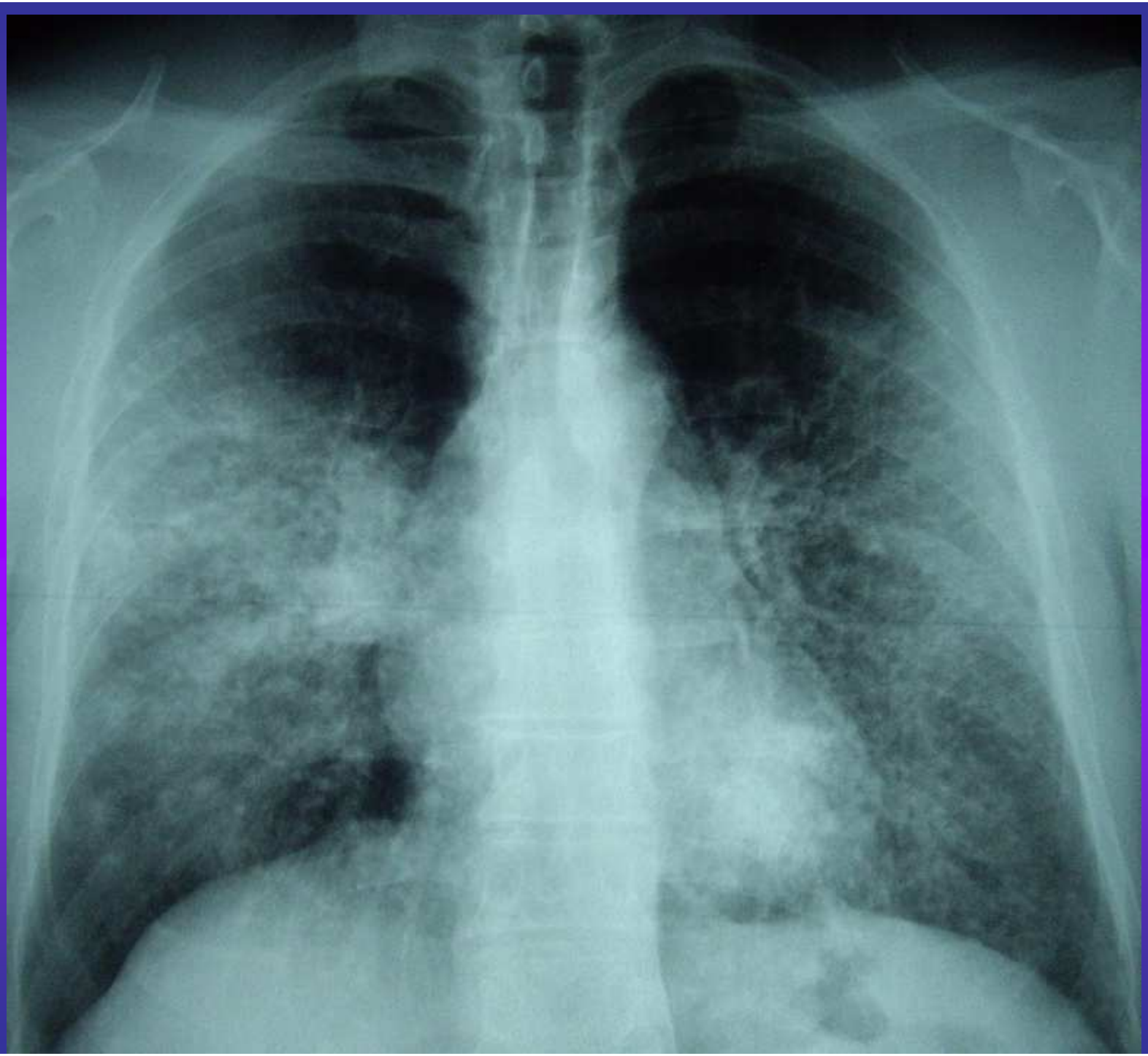

Bronchiolitis
Infections
Smoking
Toxic fumes and gases
Drugs
Hypersensibility
Idiopathic
Granulomatosis
Occupational, Neoplasms
Constrictive Br
Panbronchiolitis
BOOP?
RB-ILD, DIP
DIPNECH
BOS
CTDs
IBD Bronchiolitis

Papiris SA ®

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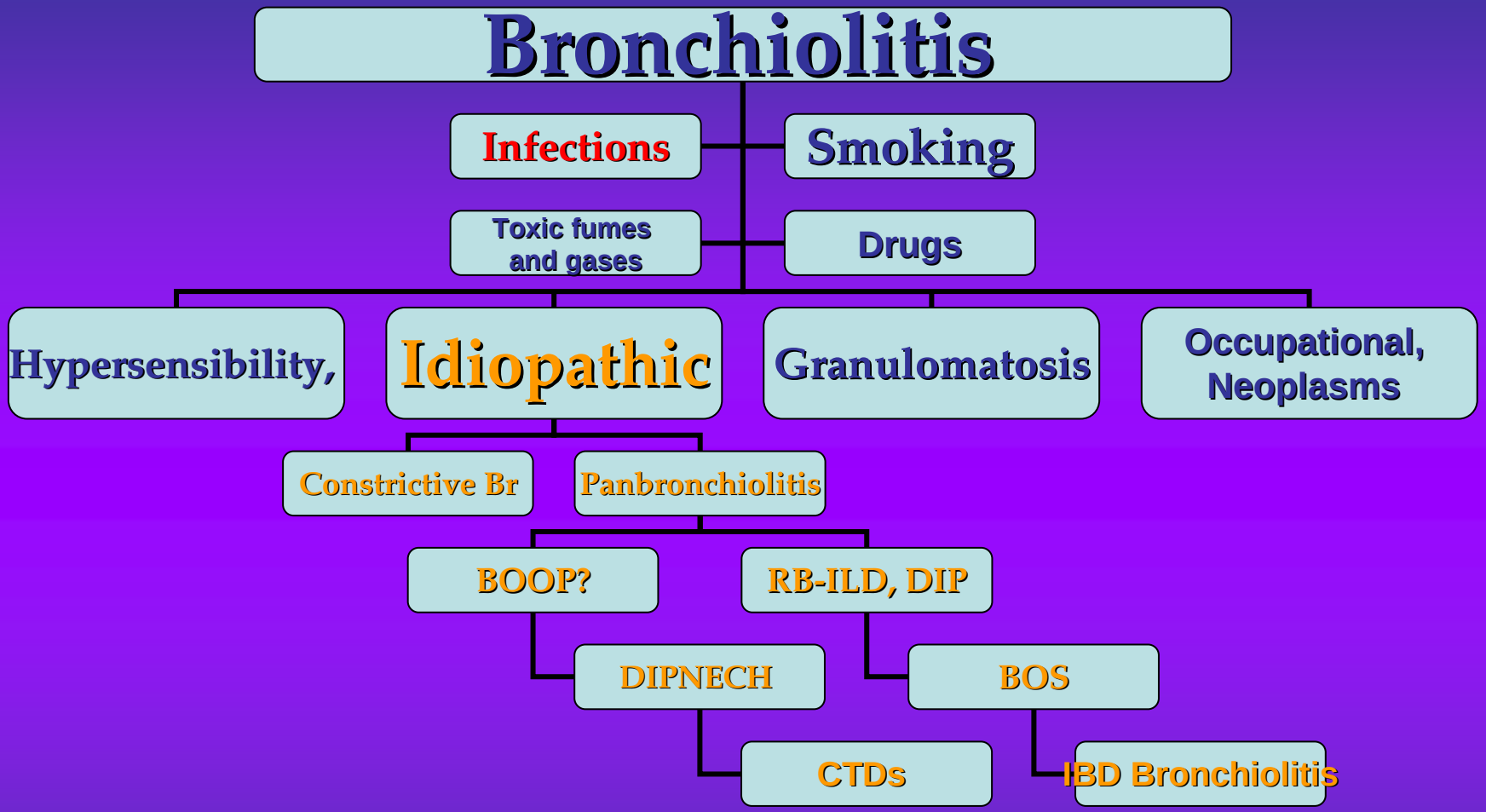
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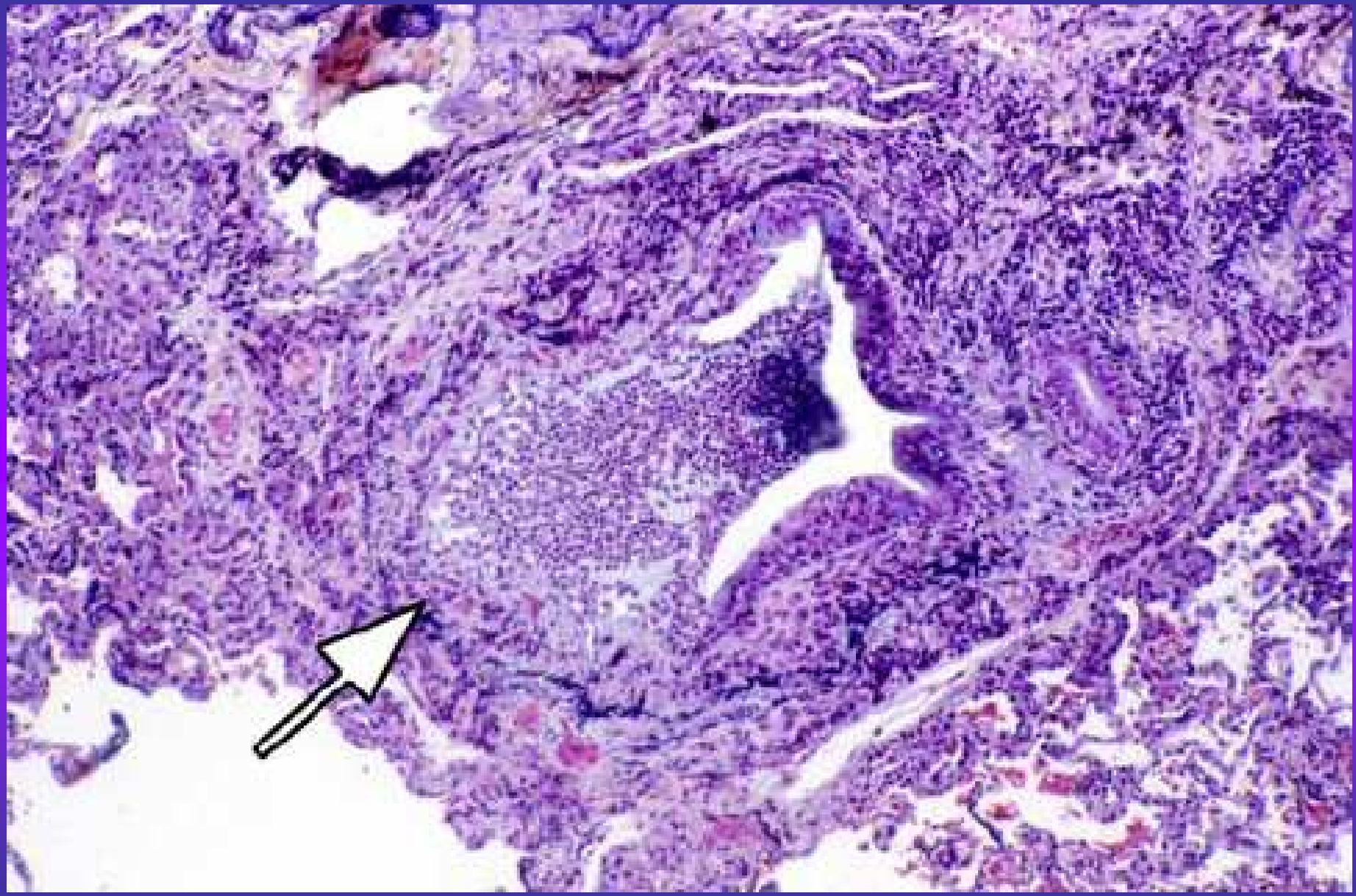


Acute Bronchiolitis

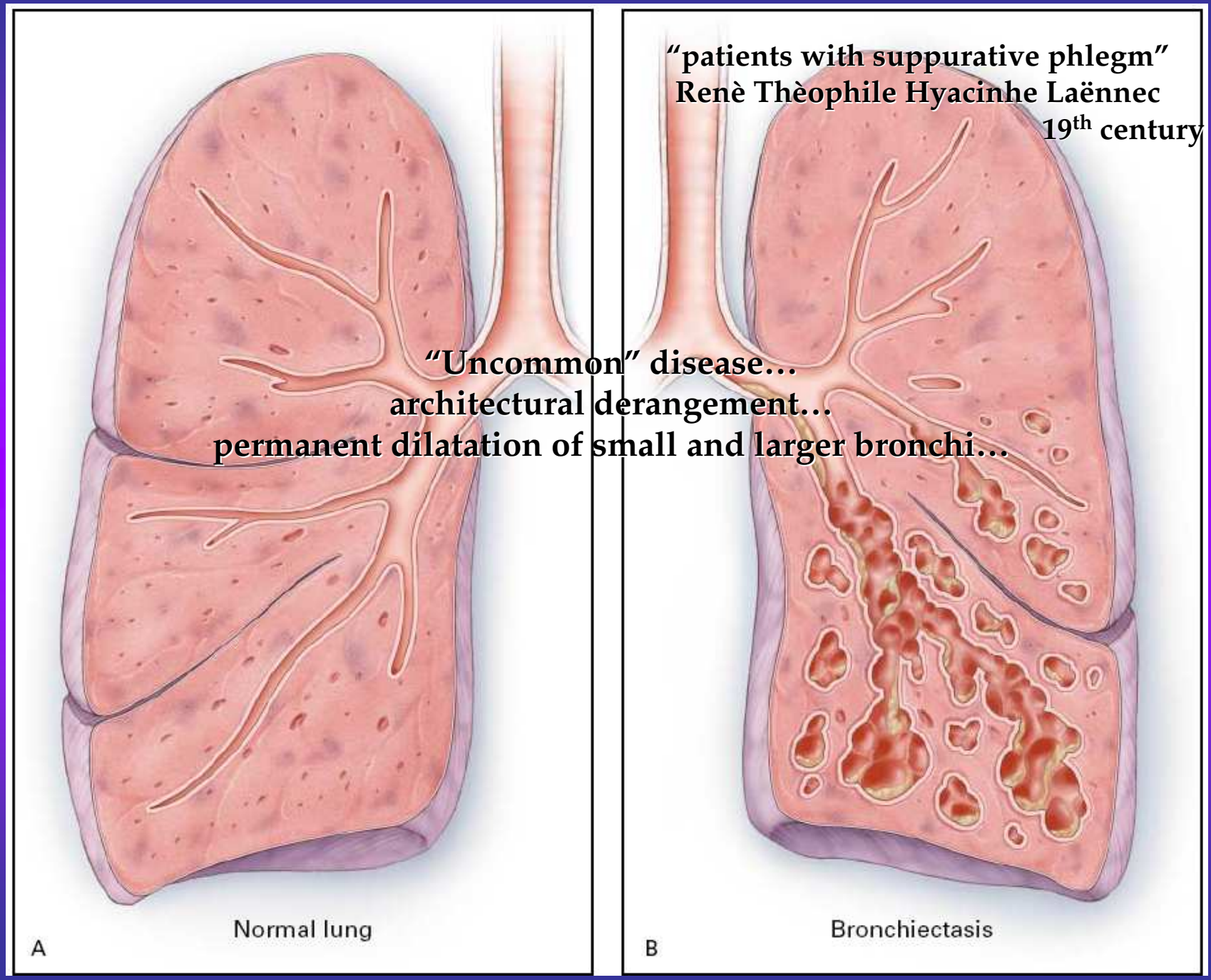
- **Acute bronchiolitis is a term most often used to describe an illness in infants and children characterized by acute wheezing with concomitant signs of respiratory viral infection**

- **Acute bronchiolitis is the most common disease of the respiratory tract during the first year of life and occurs in annual epidemics during winter**

TABLE 1. Most Common Human Respiratory Viruses and Associated Syndromes

	Serotypes	Envelope	Nucleic Acid	Illness
Adenovirus	1–49	No	dsDNA	Pneumonia, acute respiratory distress
Coronavirus	229E, OC43	Yes	(+) ssRNA	Common cold
Influenza	A, B, C	Yes	(−) ssRNA	Pneumonia
Parainfluenza	1–4	Yes	(−) ssRNA	Croup/bronchitis bronchiolitis (PIV-3)
RSV	A, B	Yes	(−) ssRNA	Bronchiolitis, pneumonia
Rhinovirus	100+	No	(+) ssRNA	Common cold

dsRNA, double stranded RNA; ssRNA, single stranded RNA; PIV, parainfluenza virus.

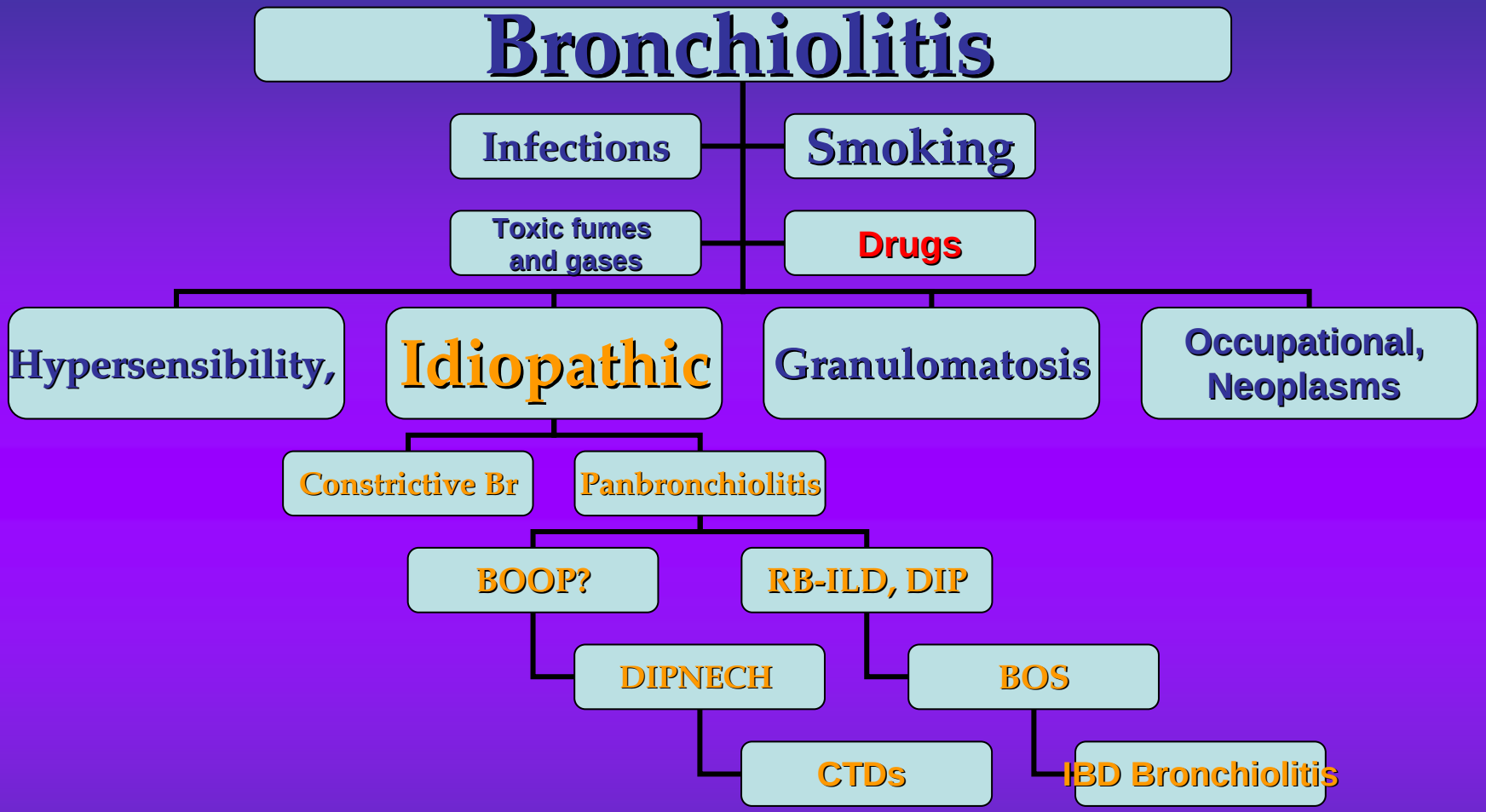


"patients with suppurative phlegm"
Renè Thèophile Hyacinhe Laënnec
19th century

"Uncommon" disease…
architectural derangement…
permanent dilatation of small and larger bronchi…

TABLE 1. CONDITIONS ASSOCIATED WITH BRONCHIECTASIS.

- Postinfectious conditions
  - Bacteria (pseudomonas, haemophilus)
  - Mycobacterium tuberculosis
  - Aspergillus species
  - Virus (adenovirus, measles virus, influenzavirus, human immunodeficiency virus)
- Congenital conditions
  - Primary ciliary dyskinesia
  - $Alpha_1$-antitrypsin deficiency
  - Cystic fibrosis
  - Tracheobronchomegaly (Mounier–Kuhn syndrome)
  - Cartilage deficiency (Williams–Campbell syndrome)
  - Pulmonary sequestration
  - Marfan's syndrome
- Immunodeficiency
  - Primary
    - Hypogammaglobulinemia
  - Secondary
    - Caused by cancer (chronic lymphatic leukemia), chemotherapy, or immune modulation (after transplantation)
- Sequelae of toxic inhalation or aspiration
  - Chlorine
  - Overdose (heroin)
  - Foreign body
- Rheumatic conditions
  - Rheumatoid arthritis
  - Systemic lupus erythematosus
  - Sjögren's syndrome
  - Relapsing polychondritis
- Other
  - Inflammatory bowel disease (chronic ulcerative colitis or Crohn's disease)
  - Young's syndrome (secondary ciliary dyskinesia)
  - Yellow nail syndrome (yellow nails and lymphedema)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DRUGS

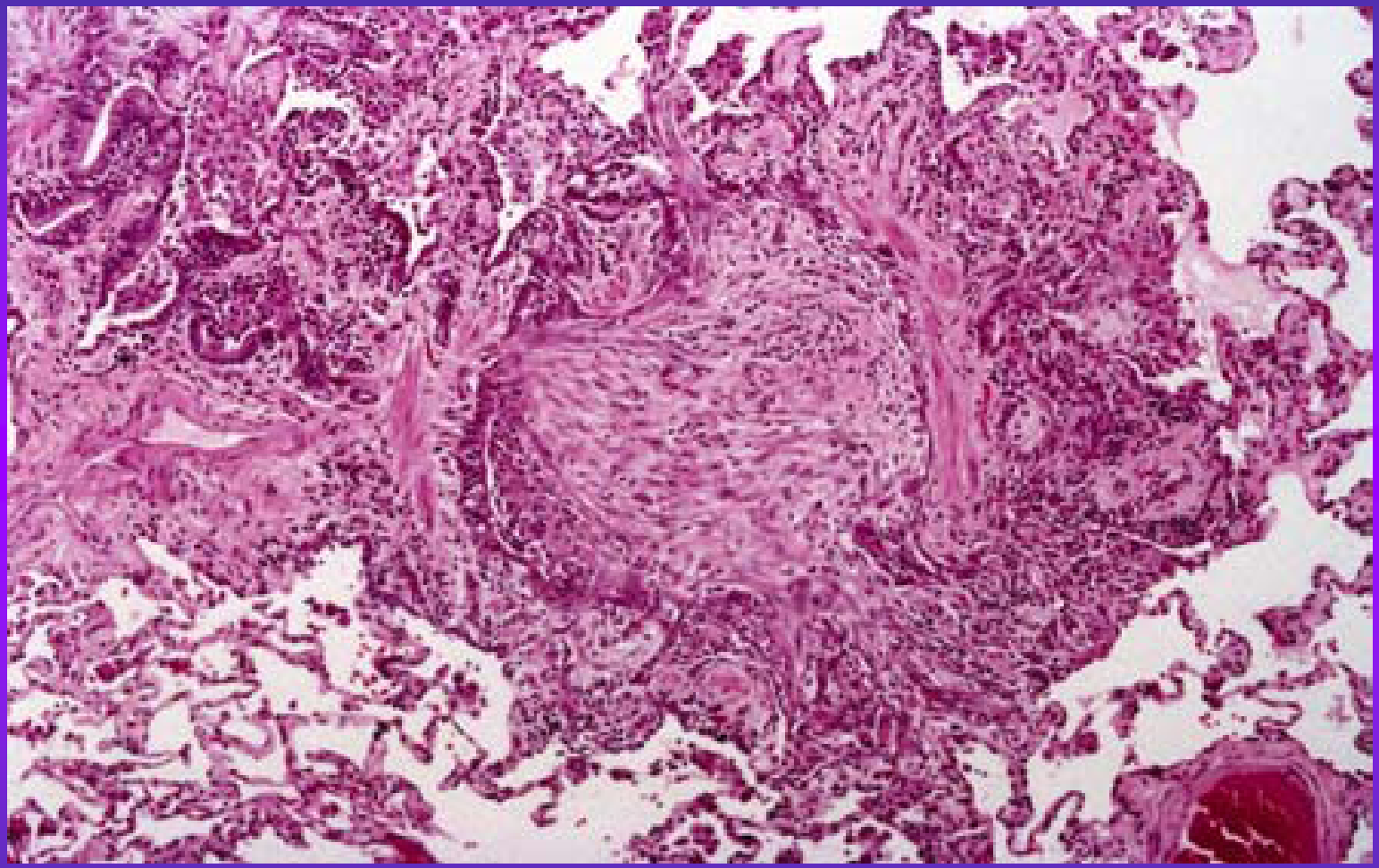
- **Penicillamine**
- **Hexamethonium**
- **l-Tryptophan**
- **Busulfan**
- **Gold**
- **Cephalosporin**
- **Sulfasalazine**
- **Amiodarone**
- **Acebutolol**
- **Sulindac**
- **Paraquat poisoning**
- ***Sauropus Androgynus***

Bronchiolitis Obliterans
Sauropus androgynus

- Leafy vegetable in India, Indonesia, China
- Juice mixture with pineapples or guavas
- Consumed for body weight reduction and blood pressure control

- Severe airflow obstruction, FEV_1 < 1.0 liter
- Insp/exp HR chest CT correlates with airflow obstruction

Chang AJSPath;21:35-42, 1997&Yang AJR 168:1045-50,1997



Eur Respir J, 1996, 9, 1317–1319
DOI: 10.1183/09031936.96.09061317

European Respiratory Journal
ISSN 0903 - 1936

CASE STUDY

Bronchiolitis obliterans in a patient with localized scleroderma treated with D-penicillamine

A. Boehler*, P. Vogt**, R. Speich*, W. Weder+, E.W. Russi*

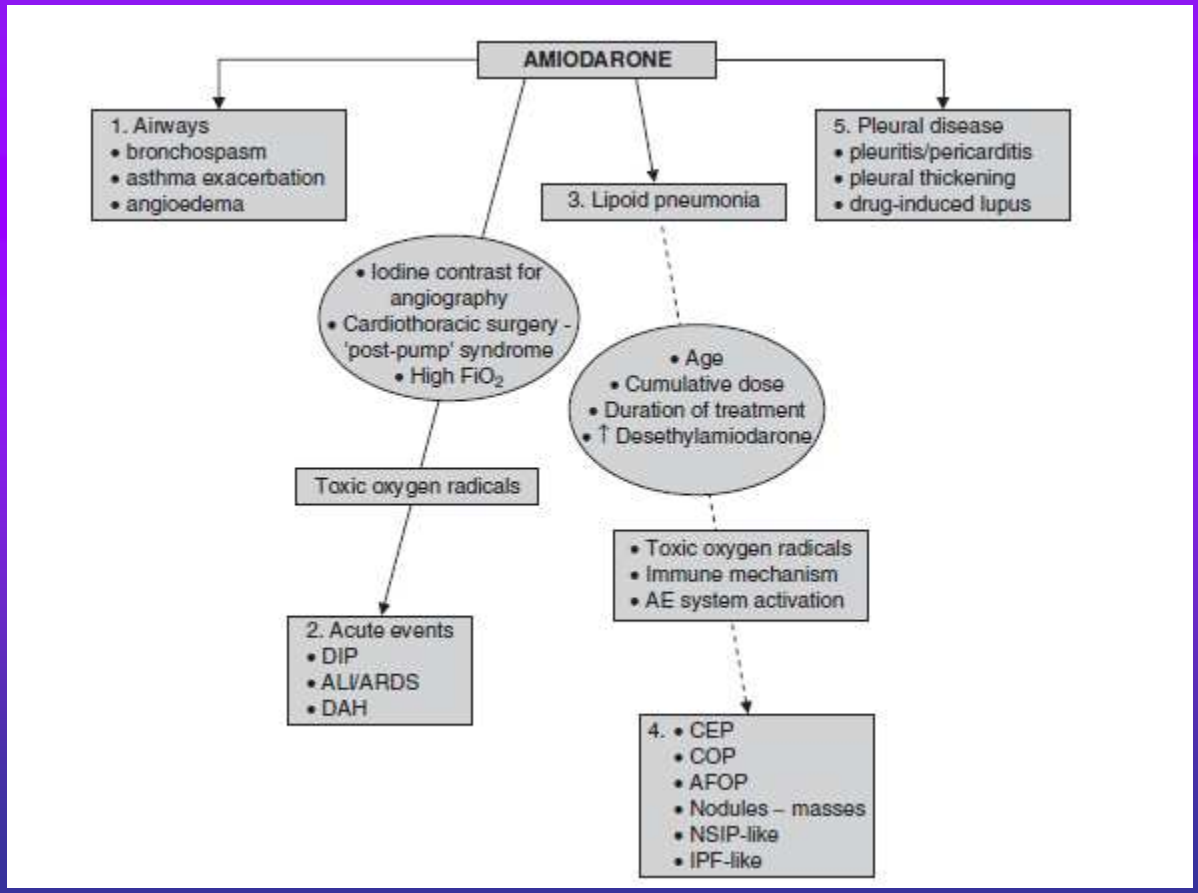
REVIEW ARTICLE

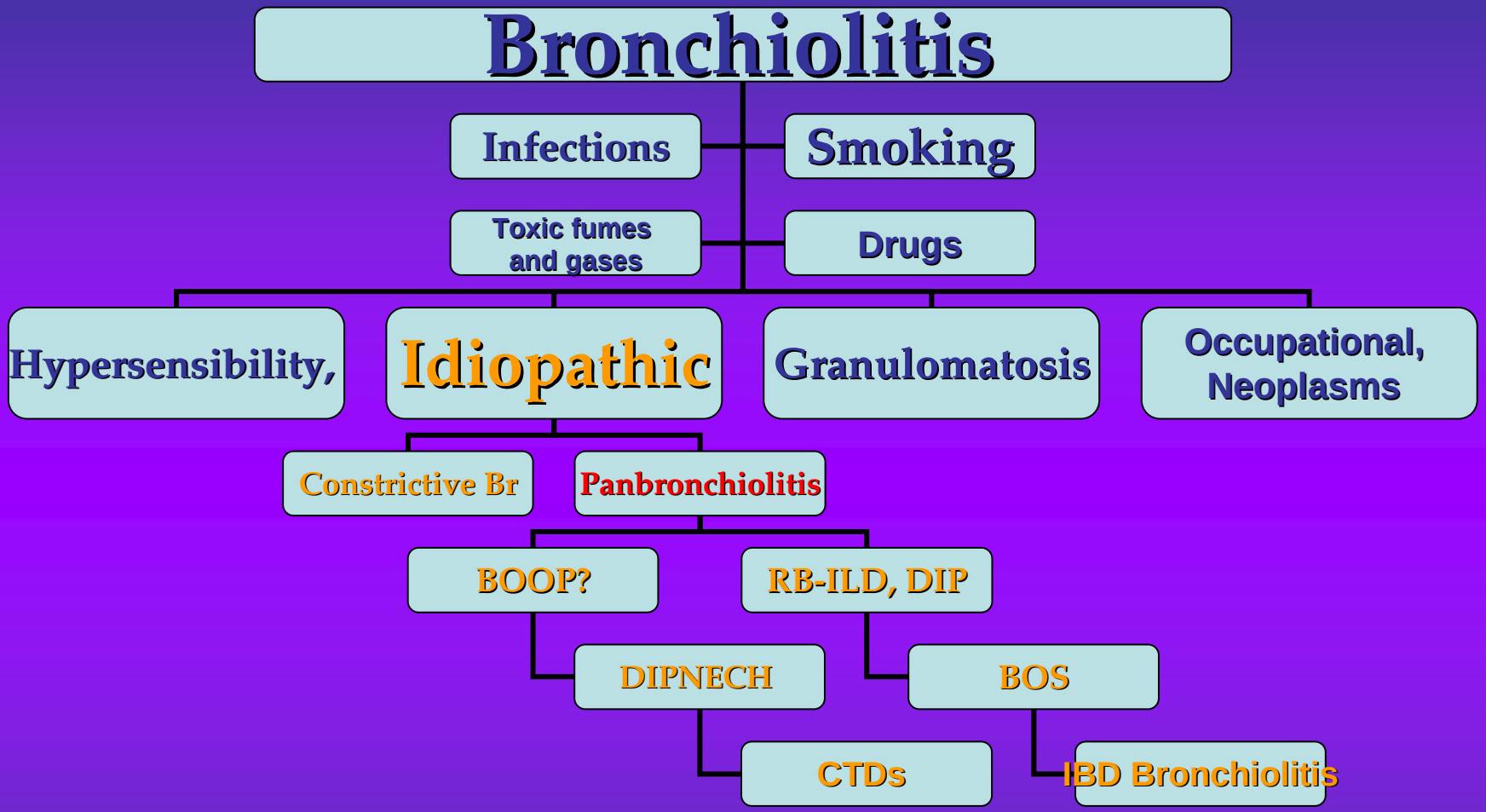
Drug Saf 2010; 33 (7): 539-558
0114-5916/10/0007-0539/$49.95/0

Amiodarone

Review of Pulmonary Effects and Toxicity

Spyros A. Papiris, Christina Triantafillidou, Likurgos Kolilekas, Despoina Markoulaki and *Effrosyni D. Manali*

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Eur Respir J 2006; 28: 862–871
DOI: 10.1183/09031936.06.00131805

SERIES "RARE INTERSTITIAL LUNG DISEASES"
Edited by C. Vogelmeier and U. Costabel
Number 4 in this Series

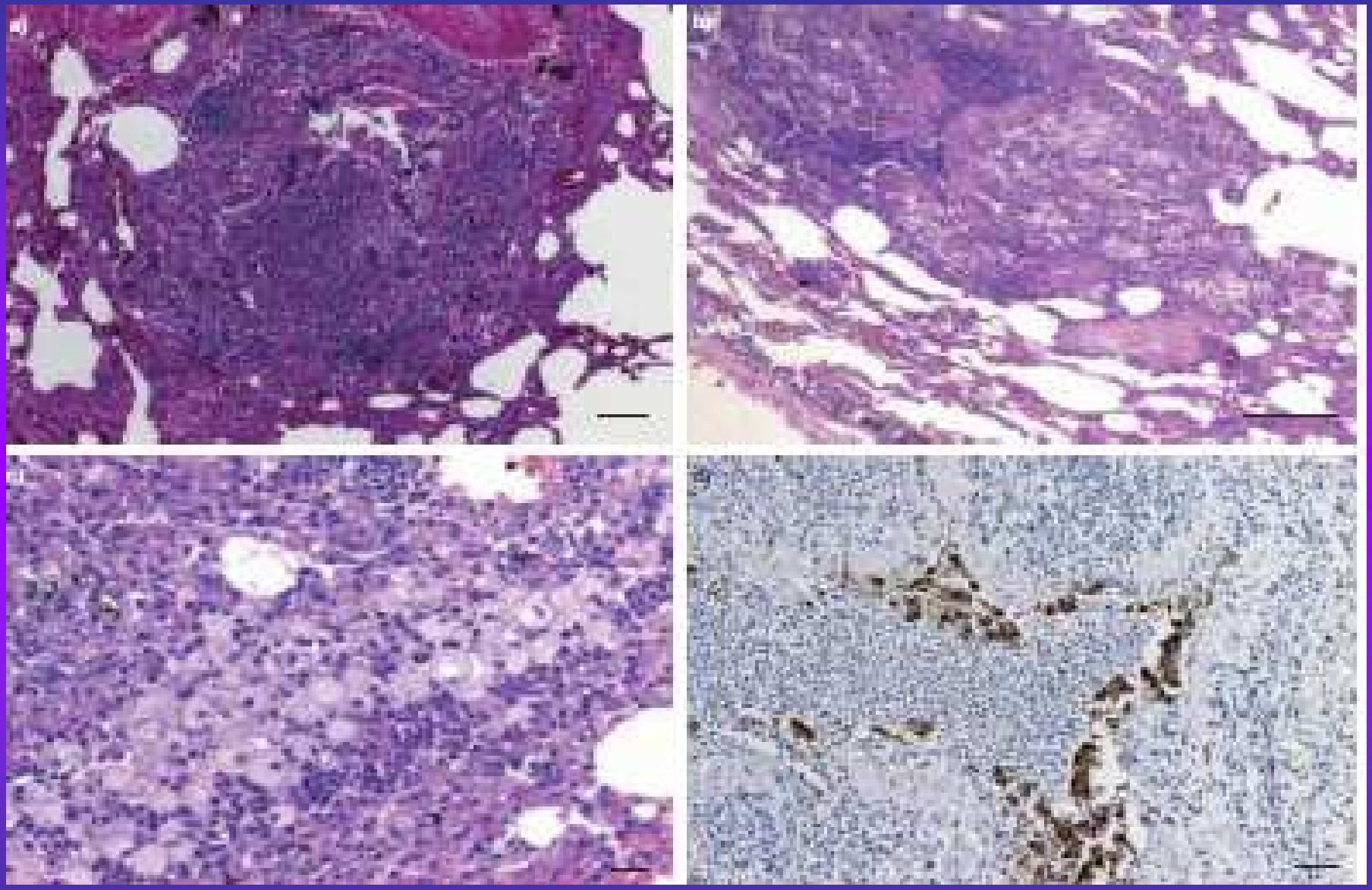
Diffuse panbronchiolitis

V. Poletti*,#, G. Casoni*, M. Chilosi¶ and M. Zompatori+

Diffuse panbronchiolitis is an idiopathic inflammatory disease, well recognised in Japan and principally affecting the respiratory bronchioles, causing a progressive suppurative and severe obstructive respiratory disorder. If left untreated, DPB progresses to bronchiectasis, respiratory failure and death.

Diffuse Panbronchiolitis

- "Diffuse" refers to the distribution of the lesions throughout both lungs, and "pan-" refers to the involvement of inflammation in all layers of the respiratory bronchioles
- rare form of bronchiolitis and chronic sinusitis
- Few cases occurring in non-Asian patients have been described .
- HLA Bw54



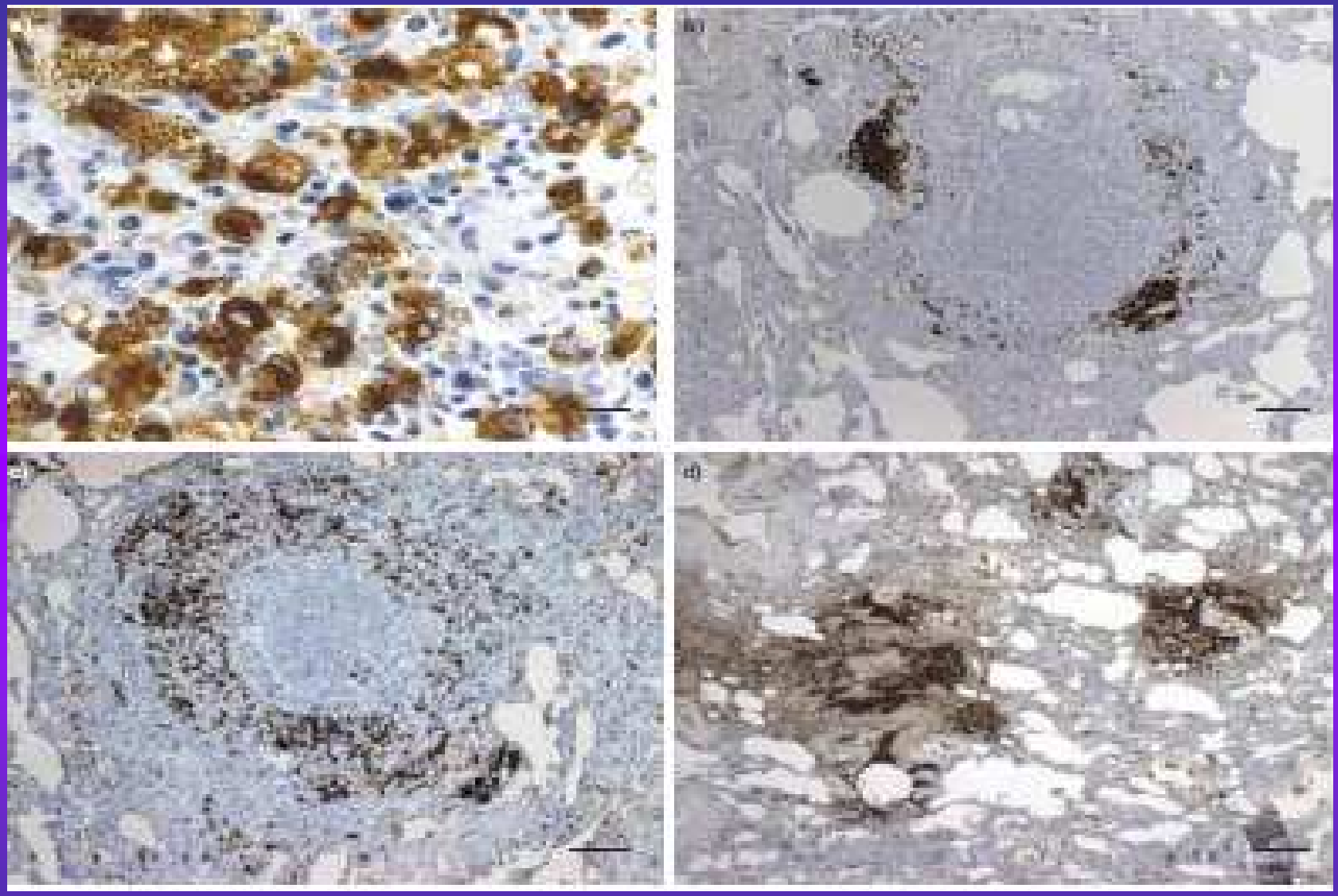


TABLE 2 Diagnostic criteria for diffuse panbronchiolitis

1. Persistent cough, sputum and exertional dyspnoea
2. History of chronic paranasal sinusitis
3. Bilateral diffuse small nodular shadows on a plain chest radiography film or centrilobular micronodules on chest computed tomography images
4. Coarse crackles
5. FEV1/FVC <70% and $P_{a}O_{2}$ <80 mmHg
6. Titre of cold haemagglutinin ≥64

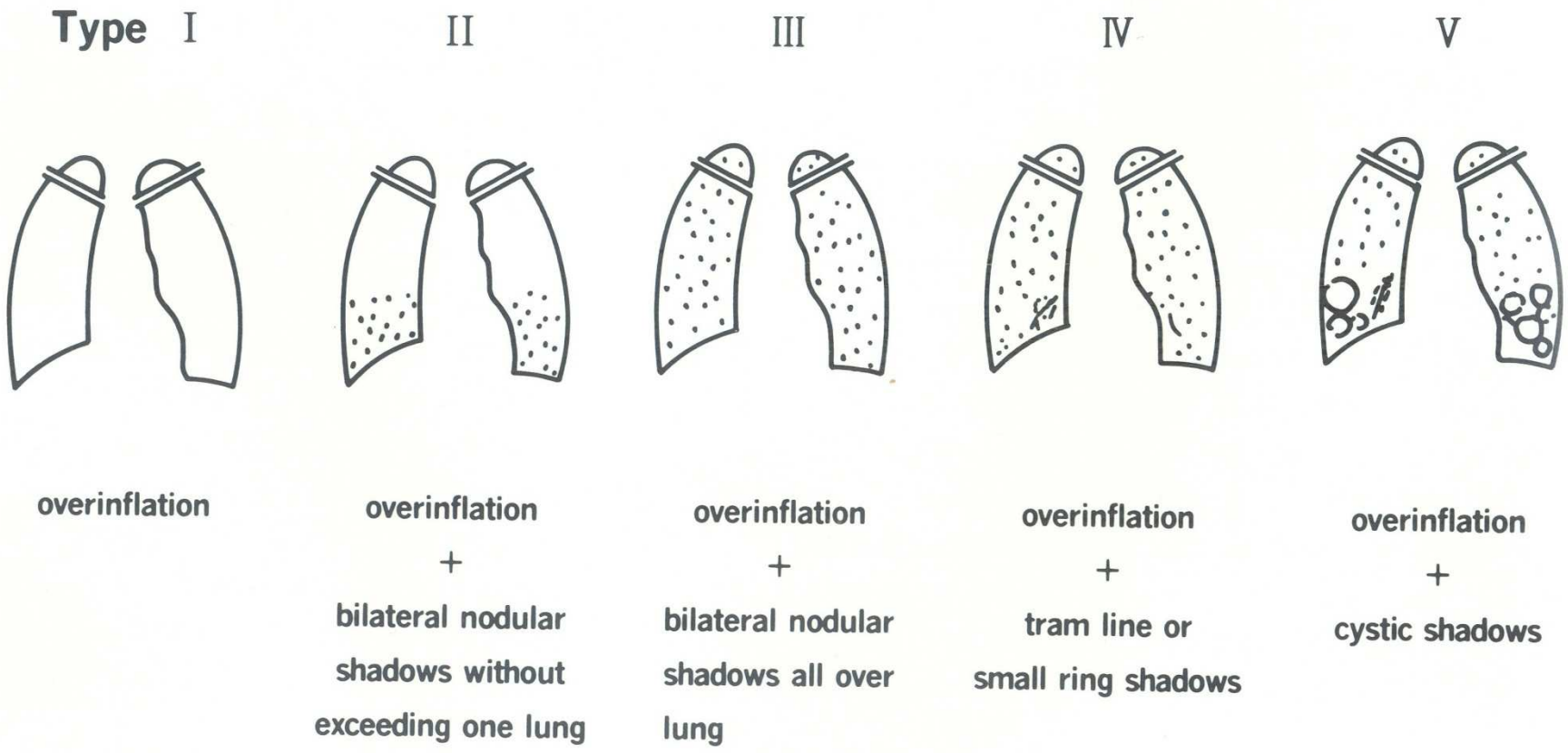
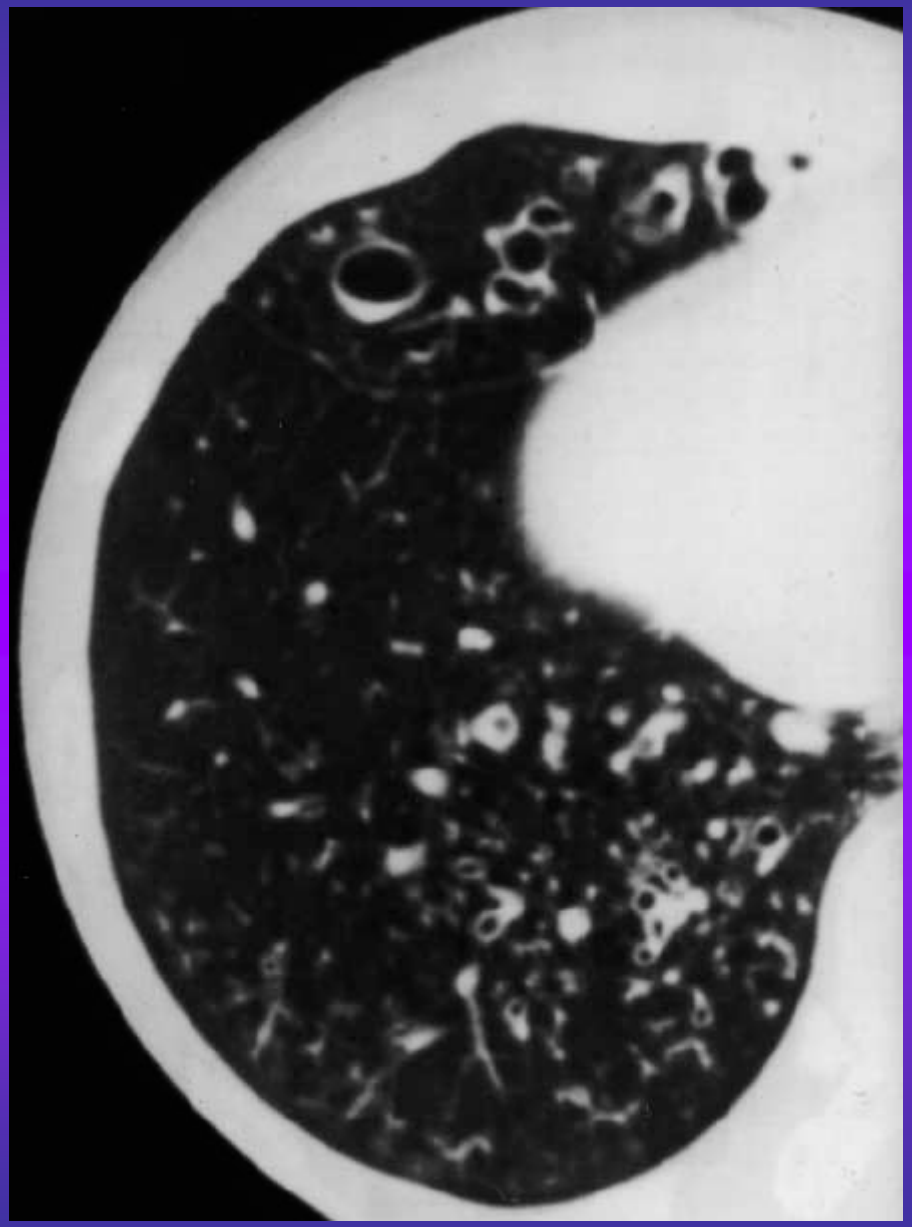
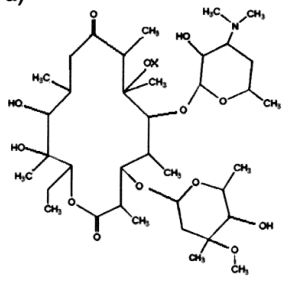


FIG. 2. Radiographic classification of diffuse panbronchiolitis.



a)



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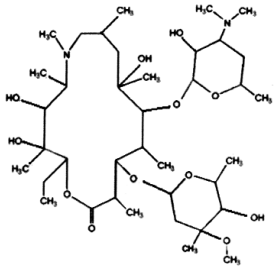
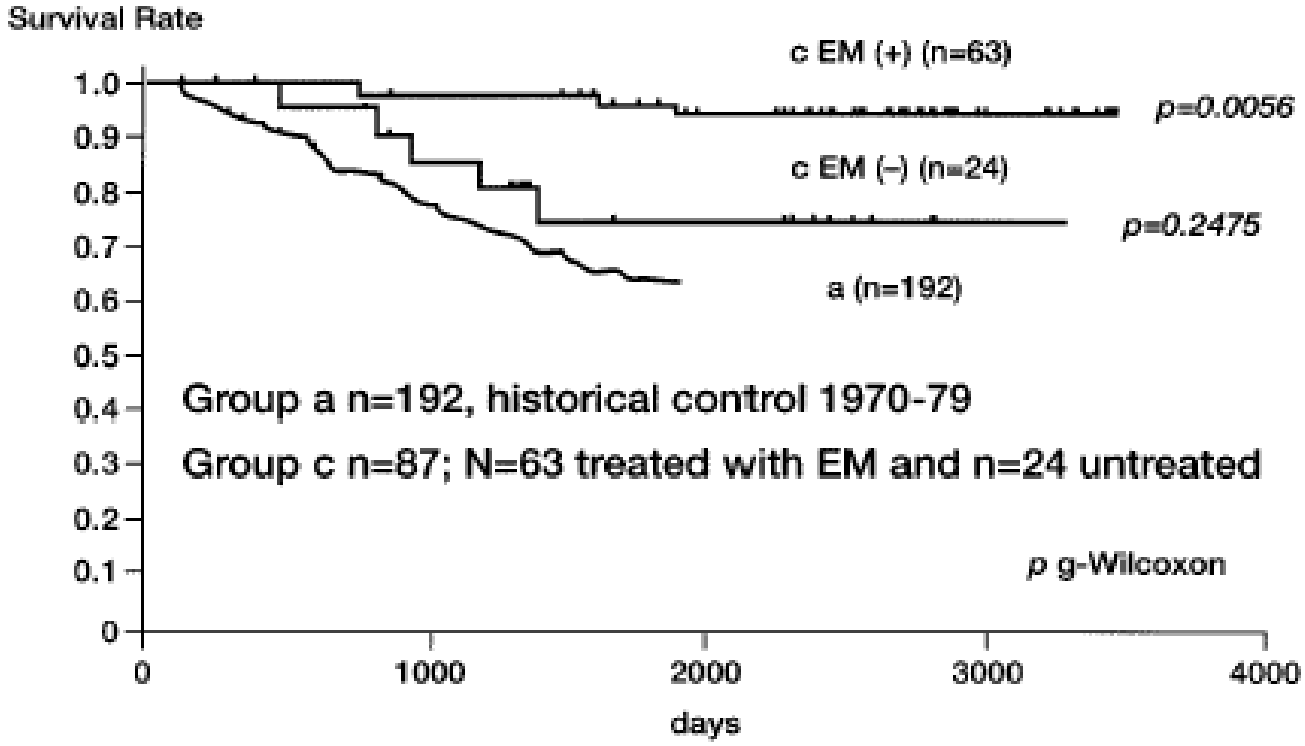


TABLE 1—Structural Differences in Macrolides

Structure of ring	Name
14-membered	Erythromycin, roxithromycin, clarithromycin, and troleandomycin
15-membered	Azithromycin
16-membered	Spiramycin, josamycin, and midecamycin

Diffuse panbronchiolitis – effects of macrolides on survival



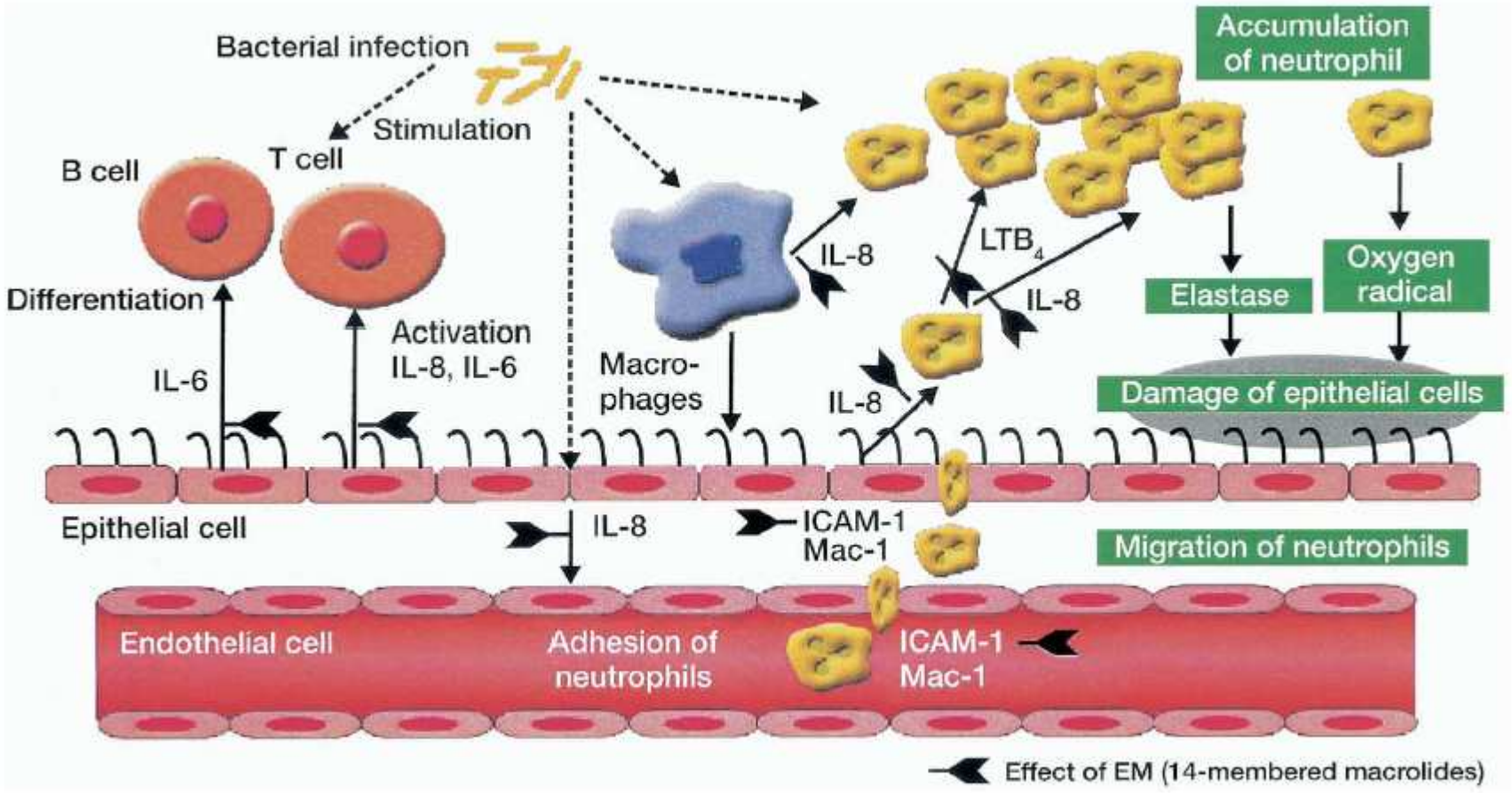


Figure 1. Role of neutrophil accumulation in chronic inflammation of airway disease. Chronic inflammation of the airways is characterized by airway epithelial damage, goblet cell hyperplasia, and mucociliary dysfunction. The increased airway mucus layer may cause mucociliary dysfunction and recurrent airway infection. Chronic inflammation is mediated by cytokines, chemokines, oxygen radicals, and chemical mediators, all of which are targets of macrolide therapy. EM = erythromycin; ICAM-1 = intercellular cell adhesion molecule–1; IL = interleukin; LTB_4 = leukotriene B_4; Mac-1 = neutrophil adhesion molecule Mac-1.

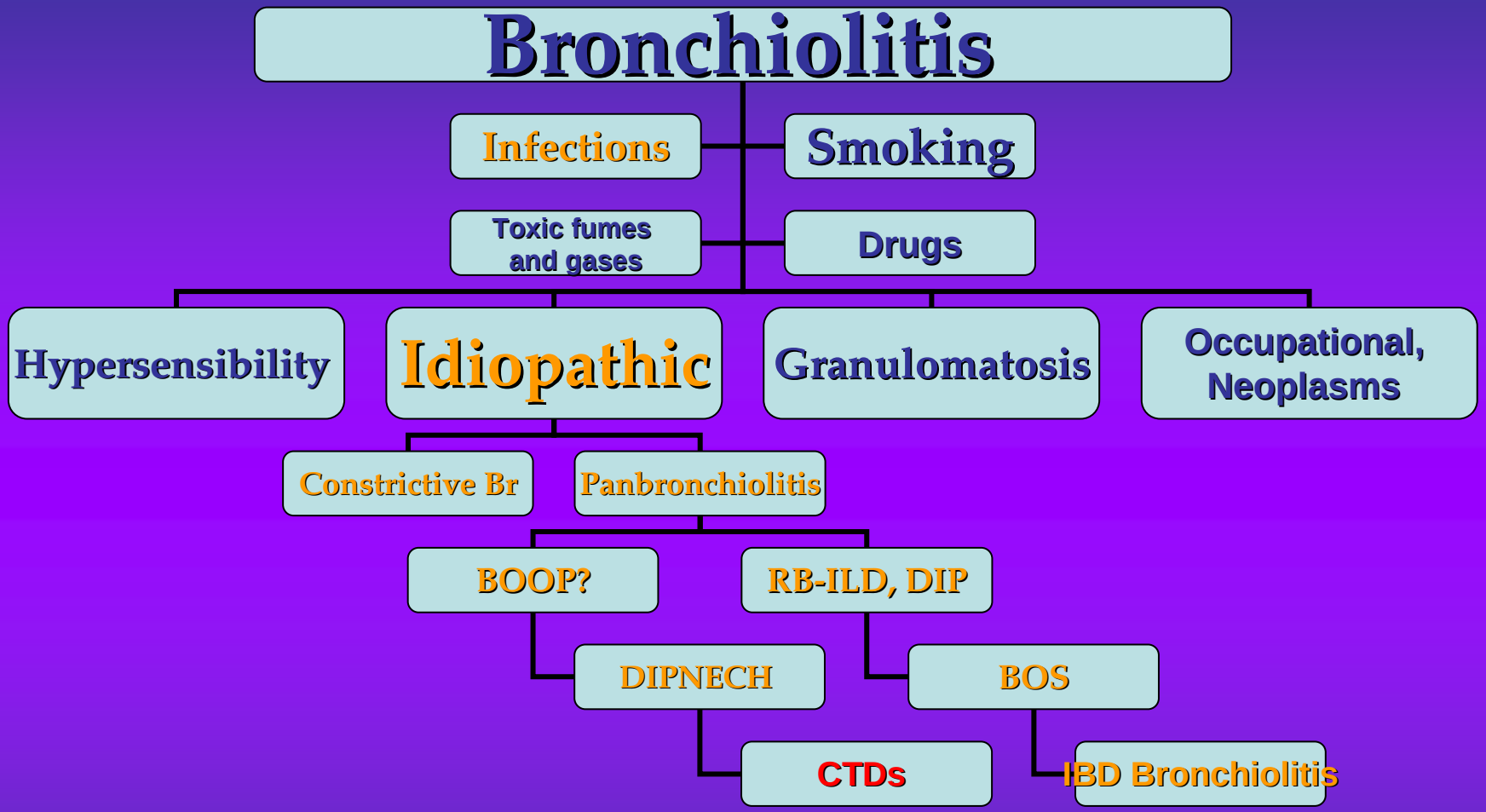
TABLE 3 Clinical guidelines for diffuse panbronchiolitis therapy[#]

Macrolides should be applied soon after the diagnosis is made, as there is a better clinical response in the earlier stage

- First choice: erythromycin 400 or 600 mg orally
- When it is no longer effective or if administration should be stopped because of adverse effects or drug interactions
- Second choice: clarithromycin 200 or 400 mg orally or roxithromycin 150 or 300 mg orally[¶]

Assessment of response and duration of treatment

- Although clinical response can usually be noted within 2–3 months, the treatment should be continued for ≥6 months, after which time the overall response should be assessed
- Treatment should be completed after a period of 2 yrs when clinical manifestations, radiological findings and pulmonary function measurements have improved and stabilised and without any significant impairment of daily activity
- Treatment should be resumed if symptoms reappear after the cessation
- In cases of an effective cure in advanced cases with extensive bronchiectasis or respiratory failure, treatment should be continued for >2 yrs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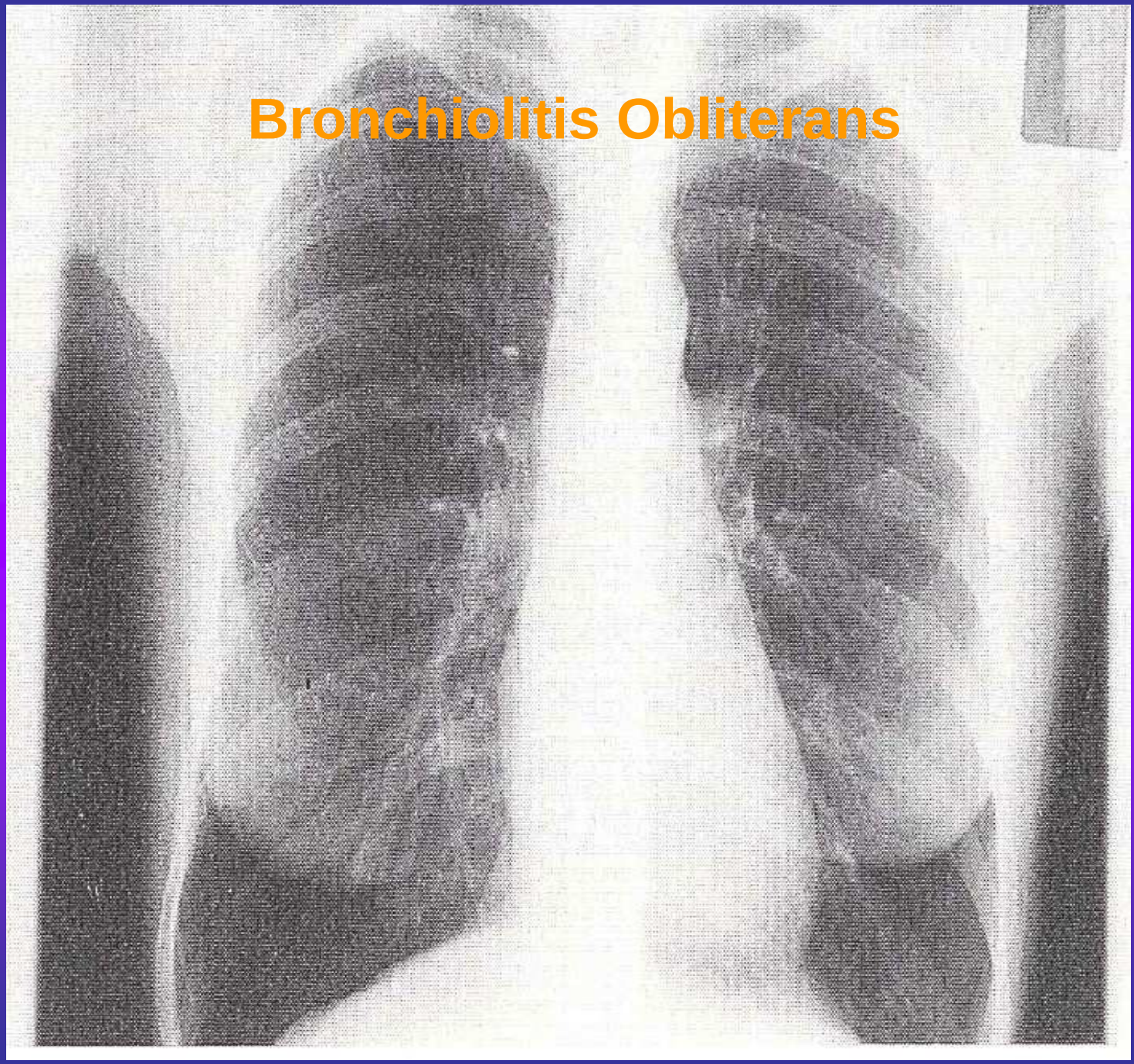
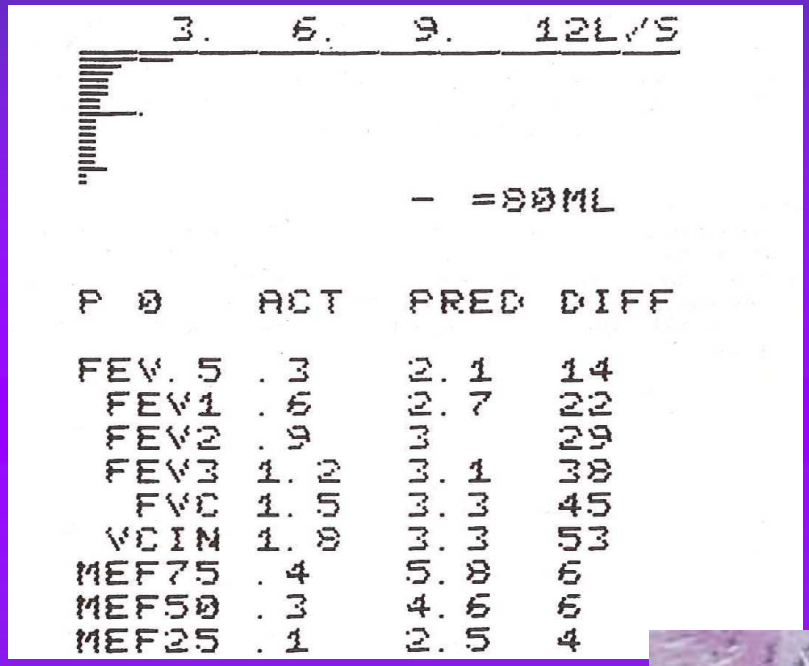
Autoimmune Rheumatic Disorders

- Rheumatoid Arthritis
- Sjögren’s syndrome
- SLE
- Polymyositis Dermatomyositis

Bronchiolitis Obliterans



Bronchiolitis Obliterans



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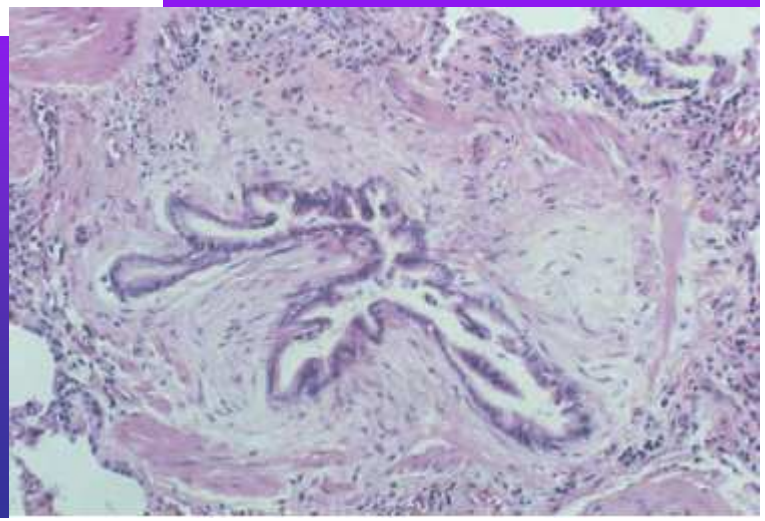
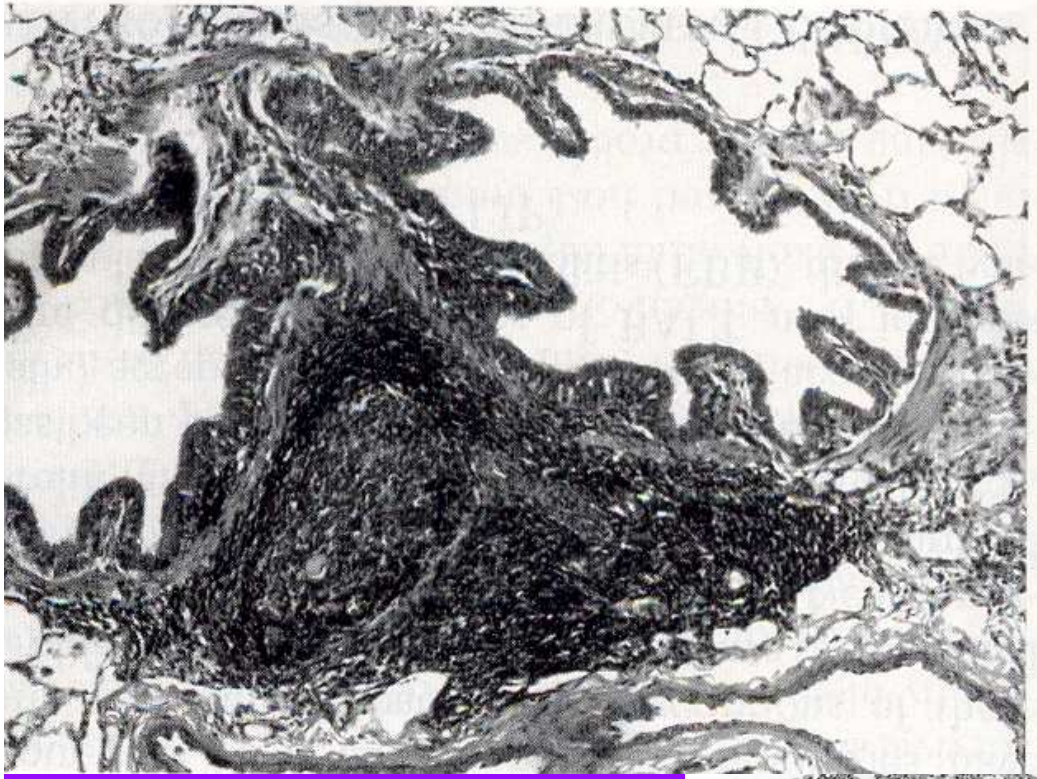
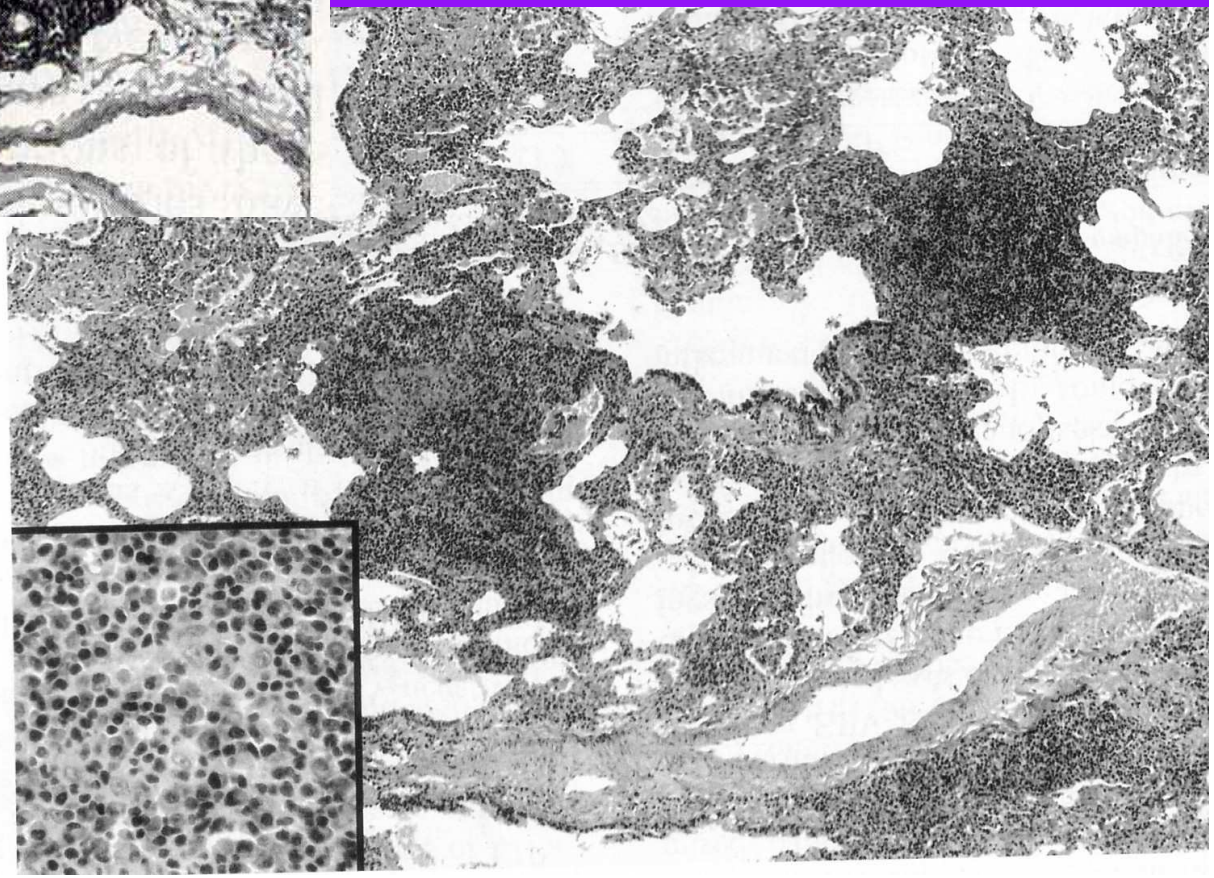
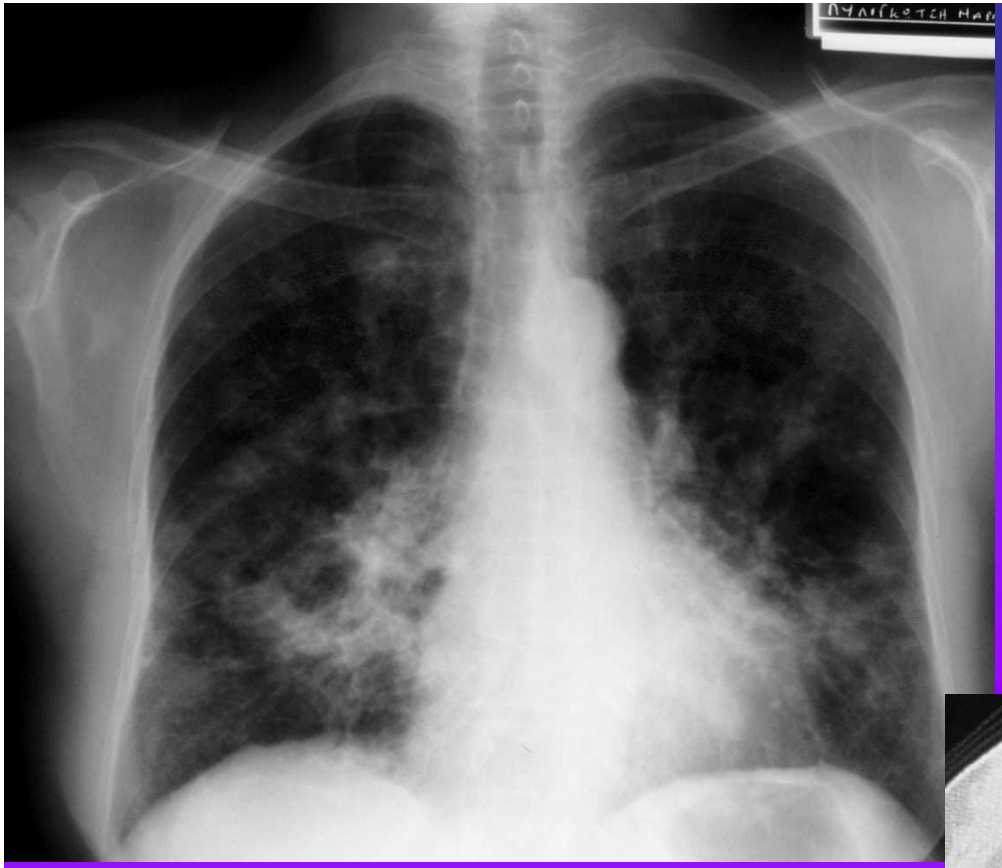
FEV1	.6	2.7	22
FEV2	.9	3	29
FEV3	1.2	3.1	38
FVC	1.5	3.3	45
VCIN	1.8	3.3	53
MEF75	.4	5.8	6
MEF50	.3	4.6	6
MEF25	.1	2.5	4

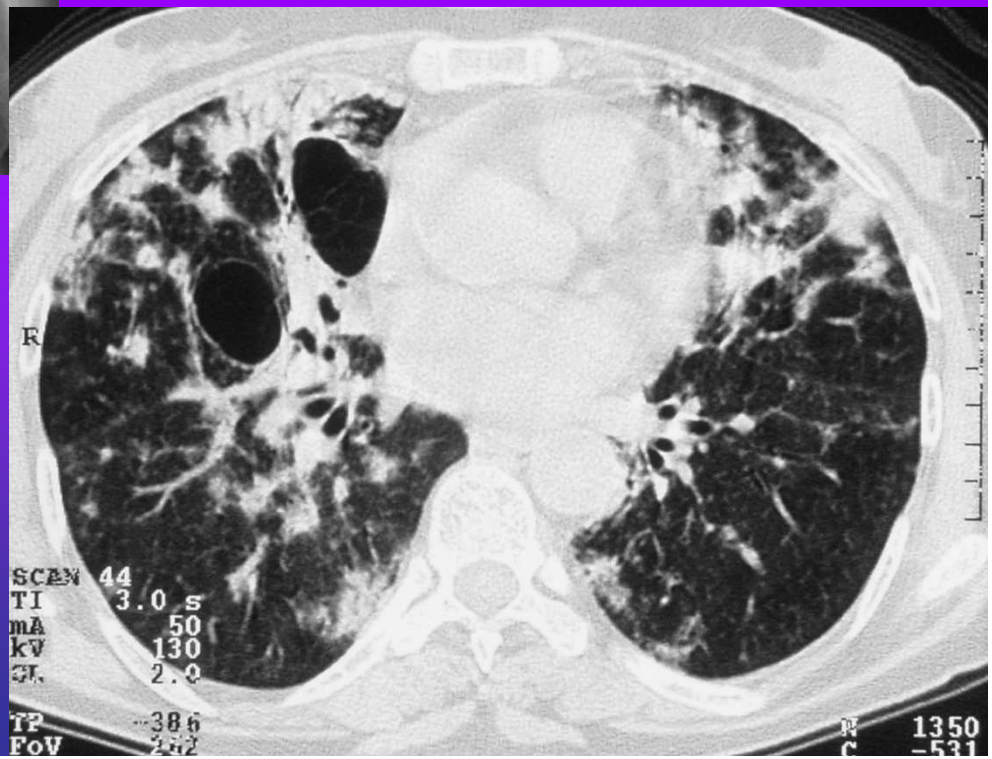


BALT →→LIP





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Respiratory Medicine (2007) 101, 84–92

ELSEVIER

respiratoryMEDICINE

BALT lymphoma

Extranodal marginal zone B-cell lymphoma of the lung in Sjögren's syndrome patients: Reappraisal of clinical, radiological, and pathology findings

Spyros A. Papiris[a,*], Ioannis Kalomenidis[b], Katerina Malagari[c], George E. Kapotsis[b], Nikolaos Harhalakis[d], Effrosyni D. Manali[e], Dimitra Rontogianni[f], Charis Roussos[b], Haralampos M. Moutsopoulos[g]

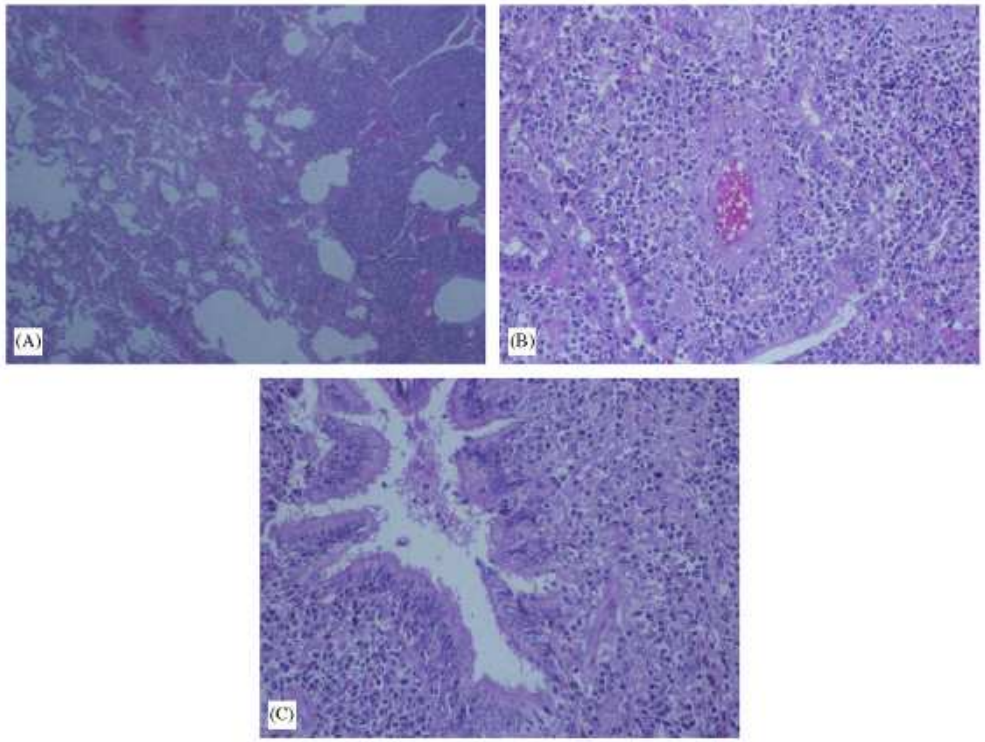

Figure 4 Histopathology of MALT-type pulmonary lymphoma. (A) A dense lymphoid infiltrate affects the pulmonary parenchyma and focally invades the alveolar septa. Both, nodular confluent and interstitial infiltration patterns of development (H&E, × 100). (B) Angiocentric (perivascular) and diffuse alveolar infiltration patterns of development of the lymphomatoid cells (H&E, × 200). (C) Bronchocentric (peribronchiolar) infiltration pattern of development of the lymphomatoid cells (H&E, × 200).


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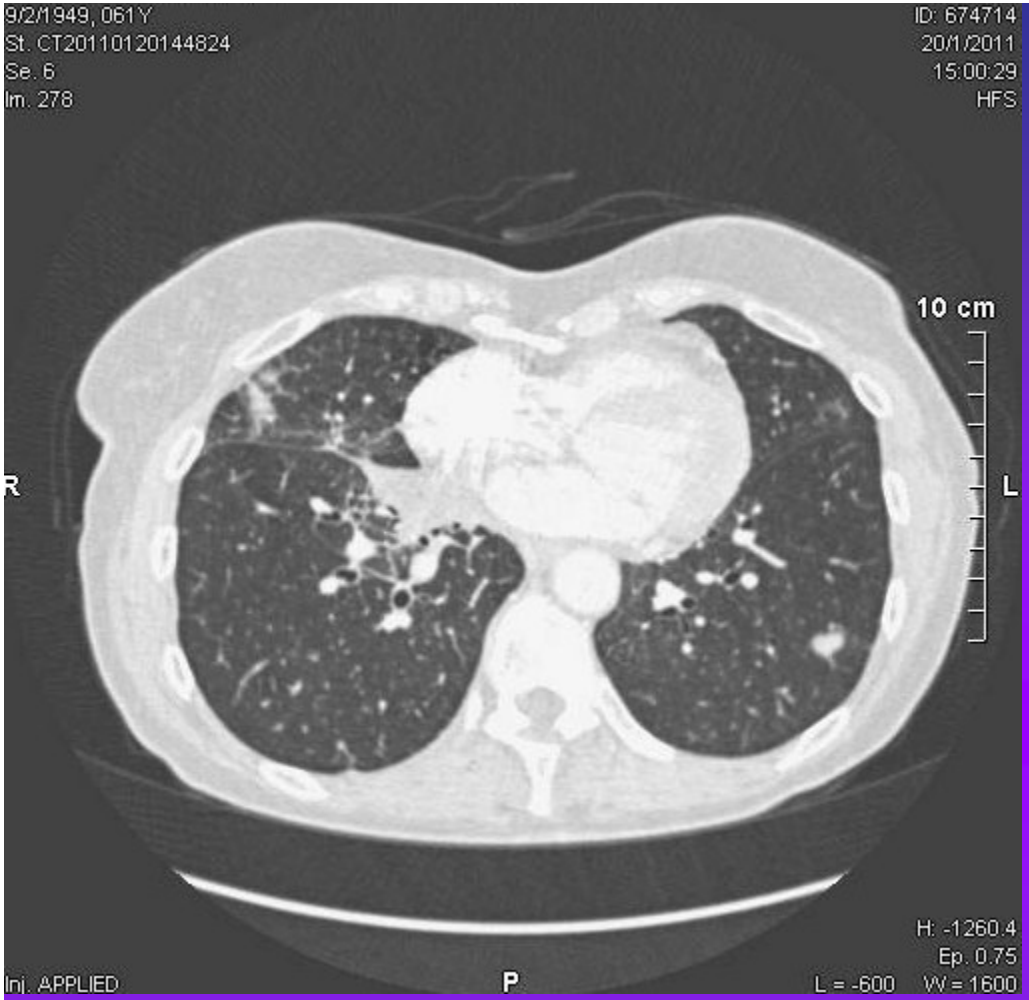
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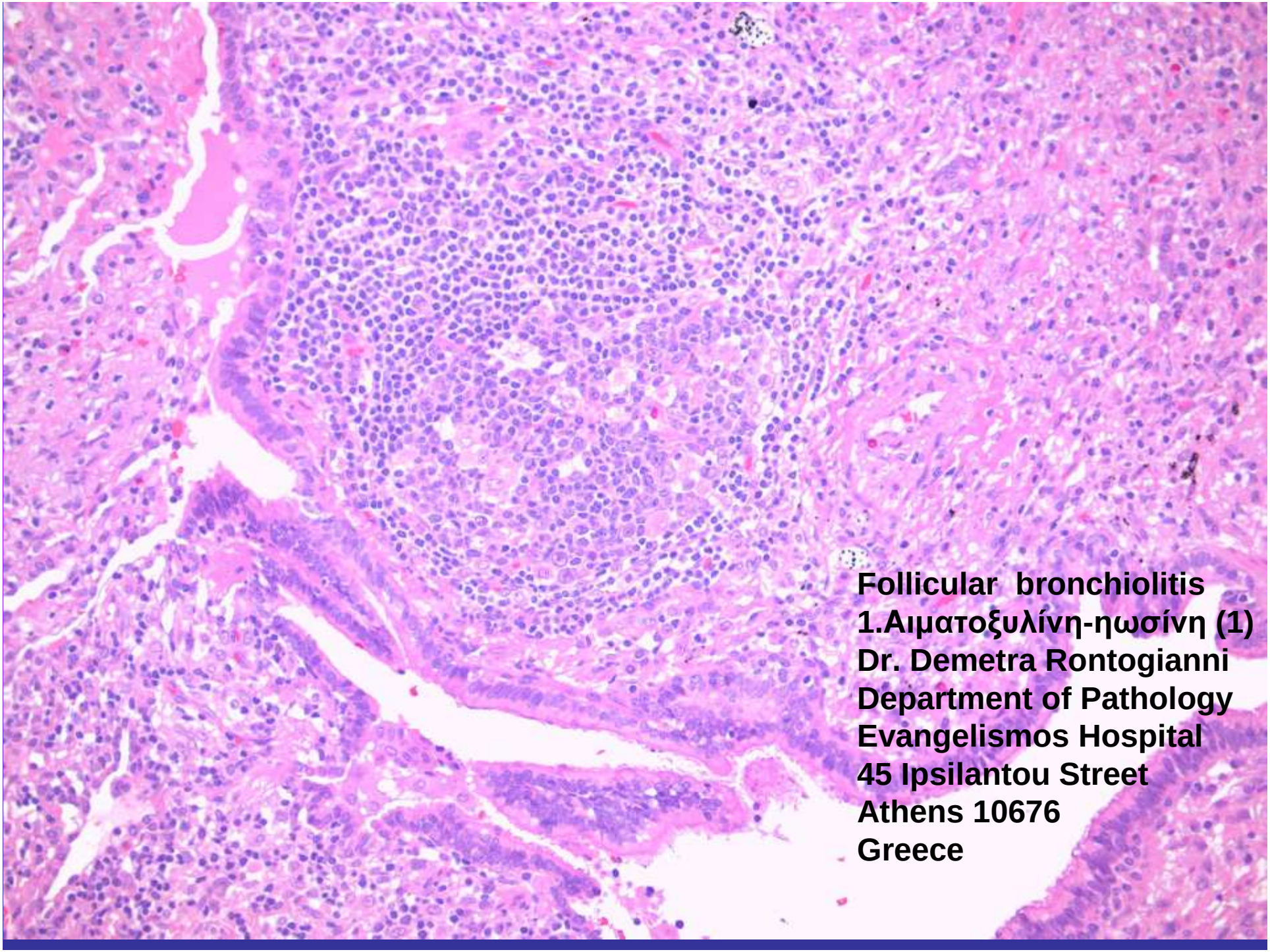
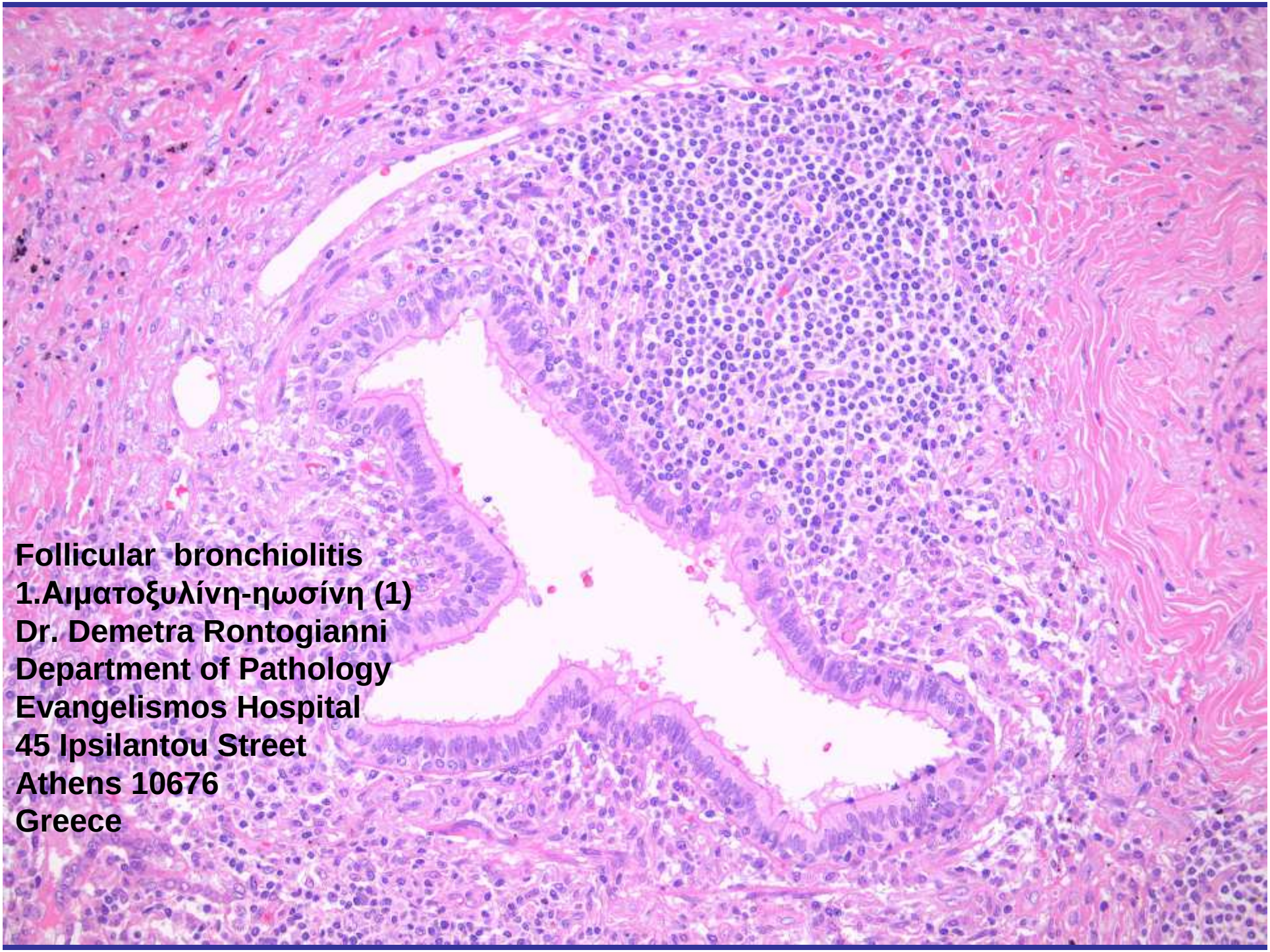
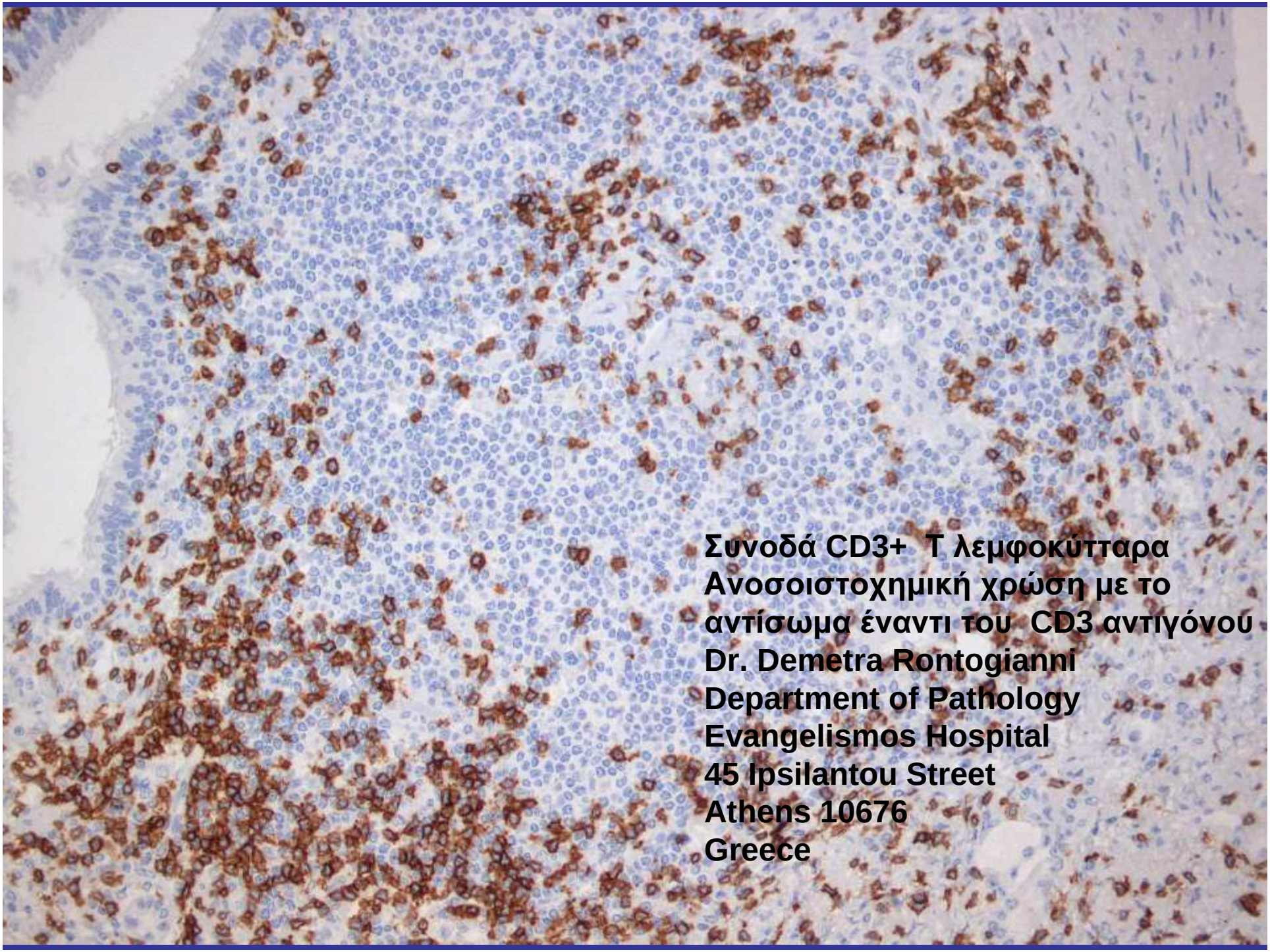

Follicular bronchiolitis
1.Αιματοξυλίνη-ηωσίνη (1)
Dr. Demetra Rontogianni
Department of Pathology
Evangelismos Hospital
45 Ipsilantou Street
Athens 10676
Greece



Follicular bronchiolitis
1.Αιματοξυλίνη-ηωσίνη (1)
Dr. Demetra Rontogianni
Department of Pathology
Evangelismos Hospital
45 Ipsilantou Street
Athens 10676
Greece

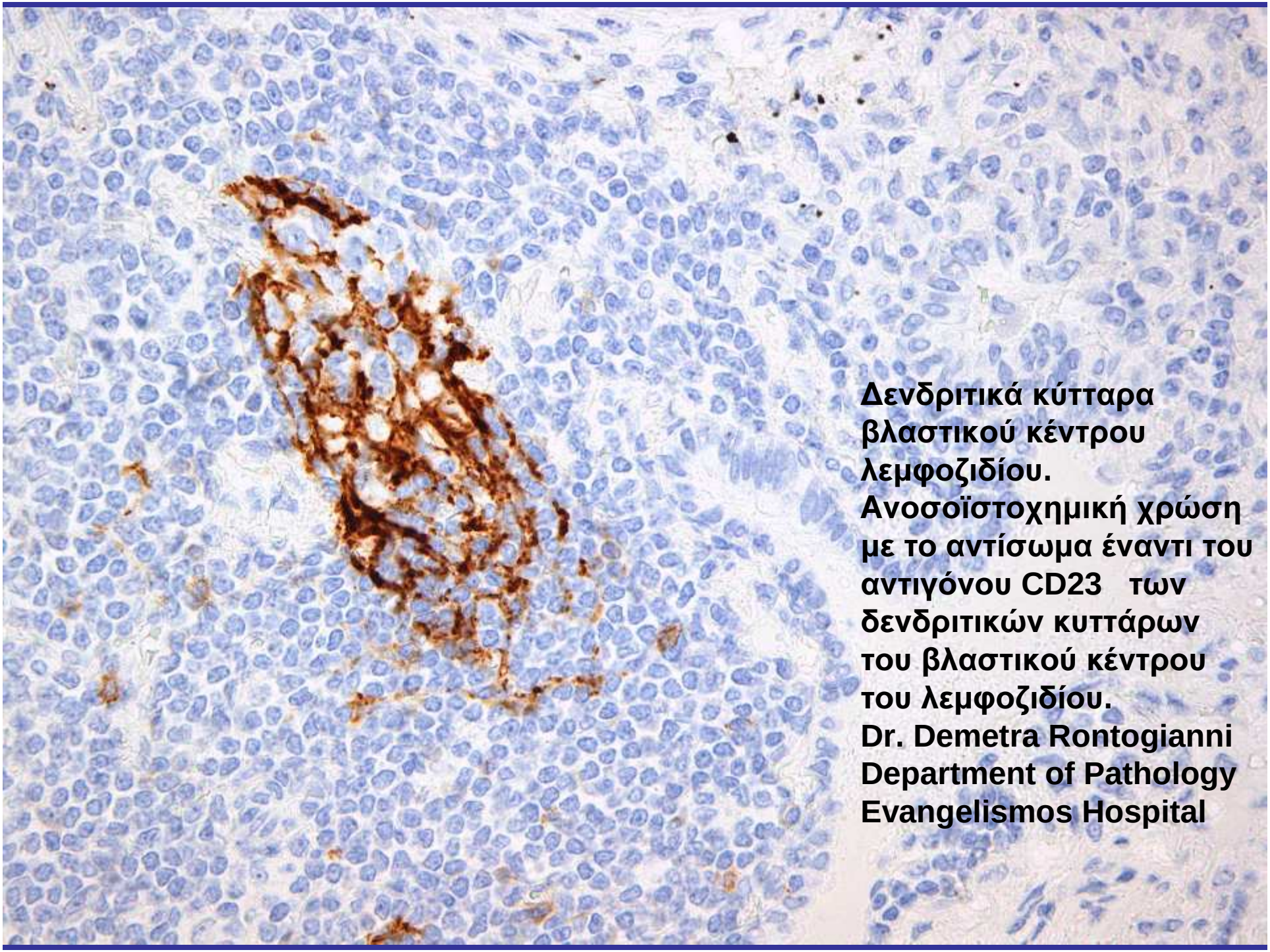
Ανοσοιστοχημική χρώση με το αντίσωμα έναντι του αντιγόνου CD20(Β λεμφοκύτταρα με βλαστικό κέντρο)

Dr. Demetra Rontogianni
Department of Pathology
Evangelismos Hospital
45 Ipsilantou Street
Athens 10676
Greece



Συνοδά CD3+ Τ λεμφοκύτταρα
Ανοσοιστοχημική χρώση με το αντίσωμα έναντι του CD3 αντιγόνου

Dr. Demetra Rontogianni
Department of Pathology
Evangelismos Hospital
45 Ipsilantou Street
Athens 10676
Greece



Δενδριτικά κύτταρα βλαστικού κέντρου λεμφοζιδίου.
Ανοσοϊστοχημική χρώση με το αντίσωμα έναντι του αντιγόνου CD23 των δενδριτικών κυττάρων του βλαστικού κέντρου του λεμφοζιδίου.
Dr. Demetra Rontogianni
Department of Pathology
Evangelismos Hospital

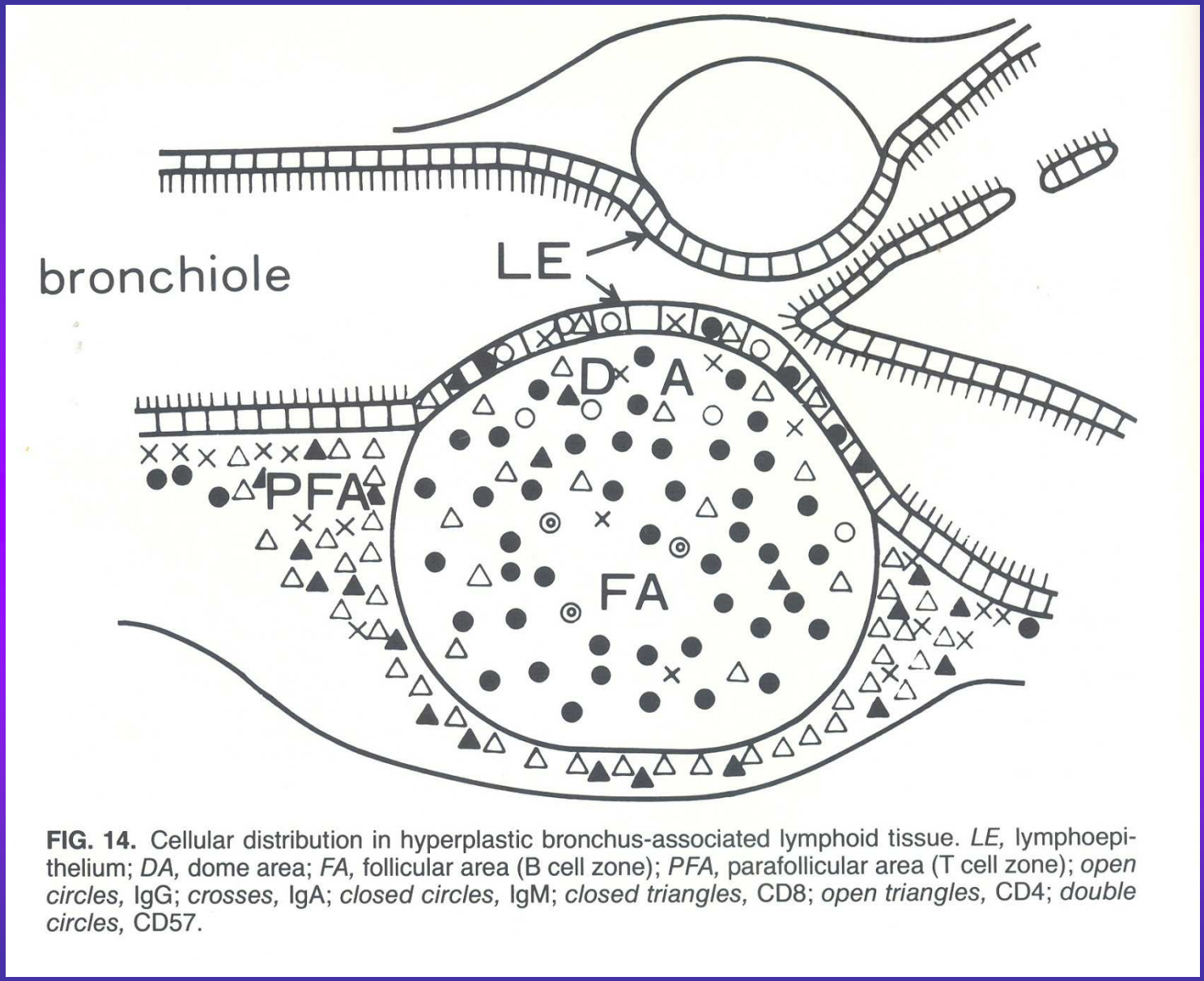
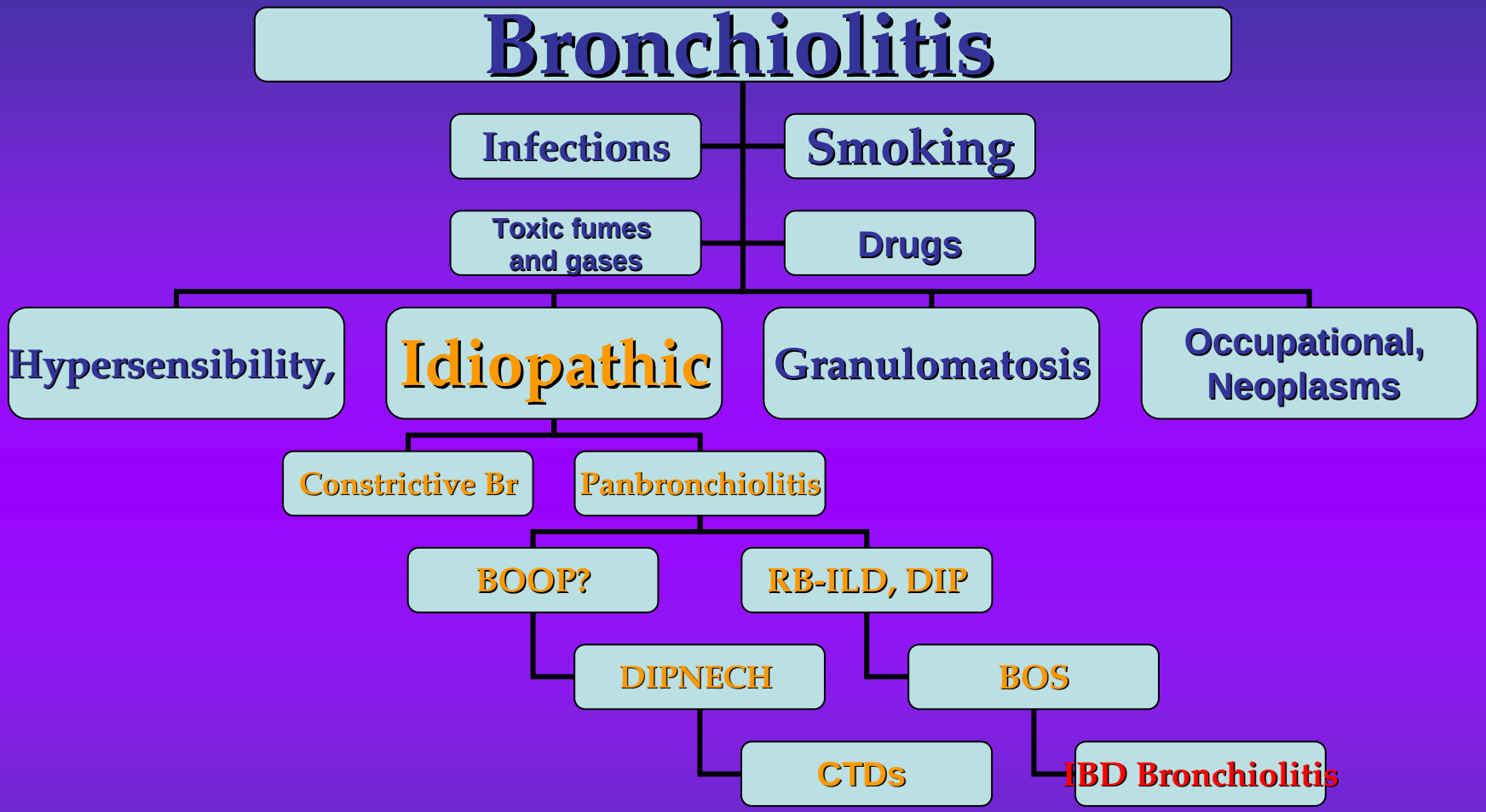


FIG. 14. Cellular distribution in hyperplastic bronchus-associated lymphoid tissue. *LE,* lymphoepithelium; *DA,* dome area; *FA,* follicular area (B cell zone); *PFA,* parafollicular area (T cell zone); *open circles,* IgG; *crosses,* IgA; *closed circles,* IgM; *closed triangles,* CD8; *open triangles,* CD4; *double circles,* CD57.


Bronchiolitis
Infections
Smoking
Toxic fumes and gases
Drugs
Hypersensibility,
Idiopathic
Granulomatosis
Occupational, Neoplasms
Constrictive Br
Panbronchiolitis
BOOP?
RB-ILD, DIP
DIPNECH
BOS
CTDs
IBD Bronchiolitis

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Official publication of the American College of Chest Physicians

Thoracic Manifestations of Inflammatory Bowel Disease

Hugh Black, Mark Mendoza and Susan Murin

Chest 2007;131;524-532
DOI 10.1378/chest.06-1074

A focused literature review of respiratory abnormalities in 155 patients with known IBD.

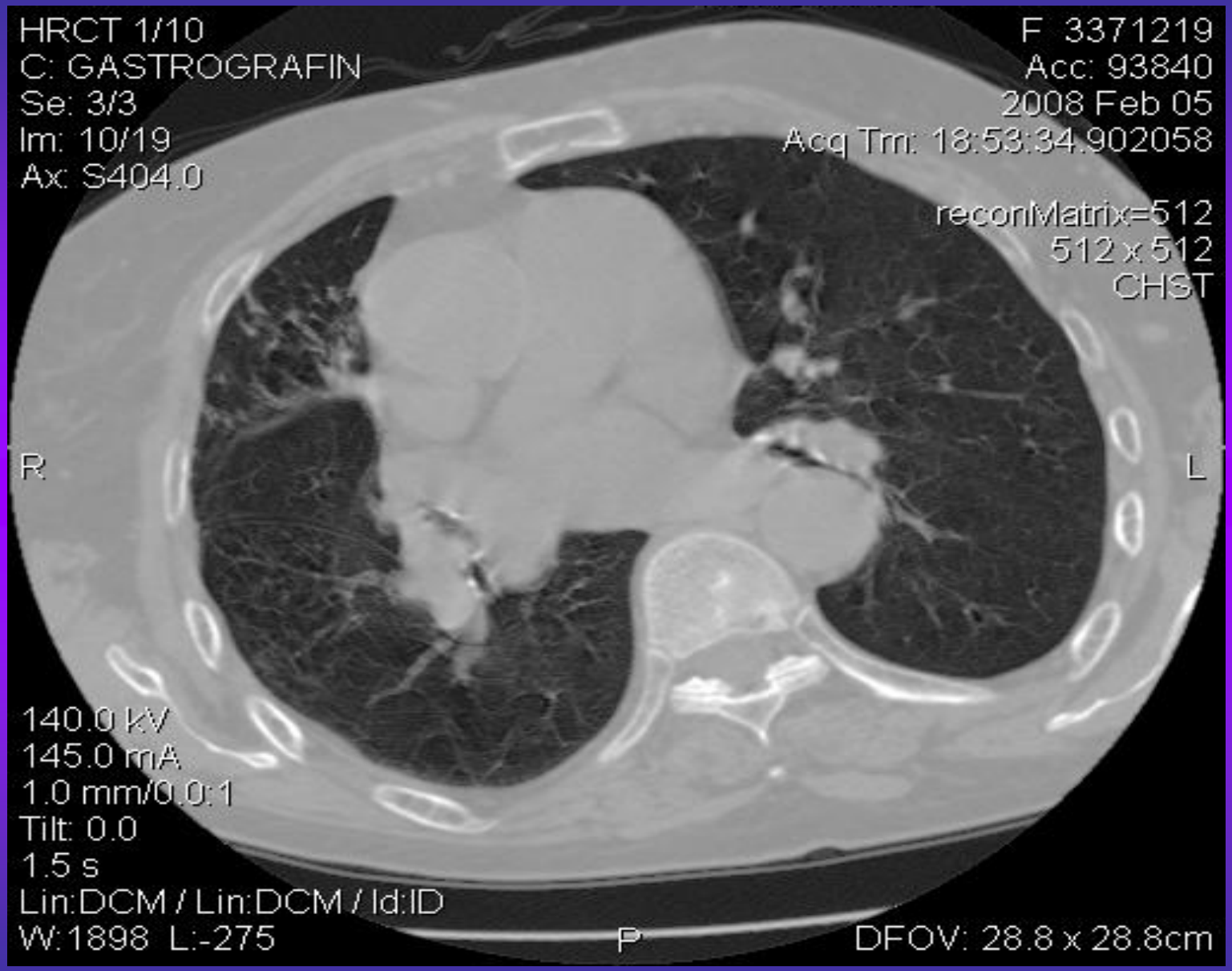
Respiratory symptoms and diagnosed respiratory system disorders are more common among patients with IBD than generally appreciated. The spectrum of respiratory disorders occurring among patients with IBD is very broad.

Bronchiectasis is the most frequently reported form of IBD-associated lung disease.

BOOP and serositis follow in frequency

IBD patients also have a threefold risk of venous thromboembolism, and recent investigations have also revealed possible ties between IBD and other diseases involving the respiratory system, including sarcoidosis, asthma, and 1-antitrypsin deficiency.

Site of Involvement	Cases, No.
Upper airway[29–39]	15
Trachea	15
Larynx/glottis	2
Large airways[29–31,40–53]	67
Bronchiectasis	44
Chronic bronchitis	13
Suppurative airway disease	5
Acute bronchitis	2
Small airways[32,46,50,54–61]	17
Bronchiolitis	10
Bronchiolitis obliterans	6
Diffuse panbronchiolitis	1
Parenchyma[30,45,46,53,56,61–76]	40
BOOP	21
Nodules	6
Interstitial lung disease not otherwise specified	6
Pulmonary interstitial emphysema	3
Desquamative interstitial pneumonia	1
Nonspecific interstitial pneumonia	1
Fibrosing alveolitis	1
Eosinophilic pneumonitis	1
Sarcoidosis	NS
α1-Antitrypsen deficiency	NS
Pulmonary vasculature[30,60,73–75,77–81]	10
Wegener granulomatosis	3
Churg-Strauss syndrome	1
Microscopic polyangiitis	2
Pulmonary vasculitis not otherwise specified	4
Serosa[30,59,65–67,73–75,82–87]	22
Pleural disease	12
Pericardial disease	15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Acc: 93840
2008 Feb 05
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145.0 mA
1.0 mm/0.0:1
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1.5 s
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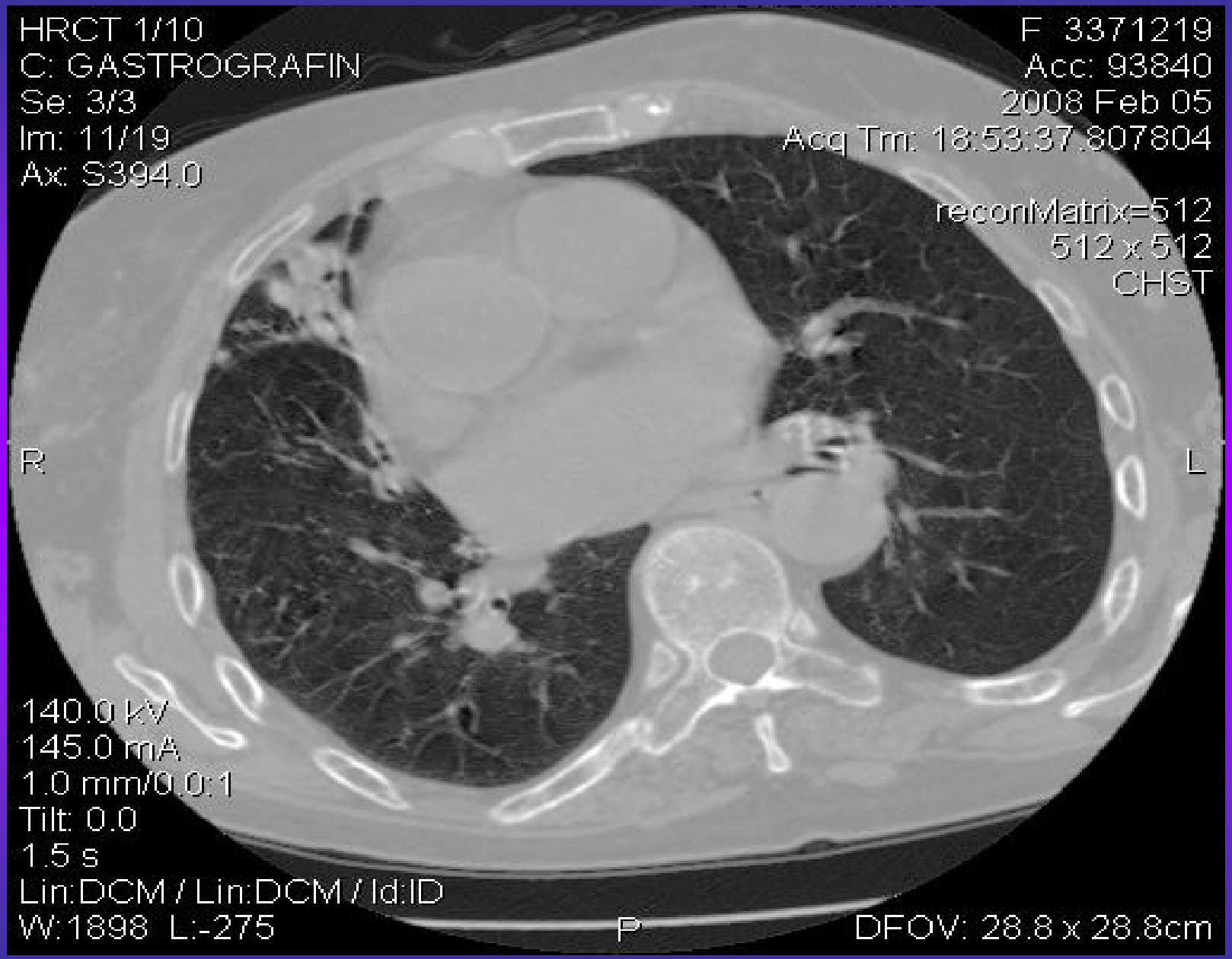
DFOV: 28.8 x 28.8cm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W:1898 L:-275
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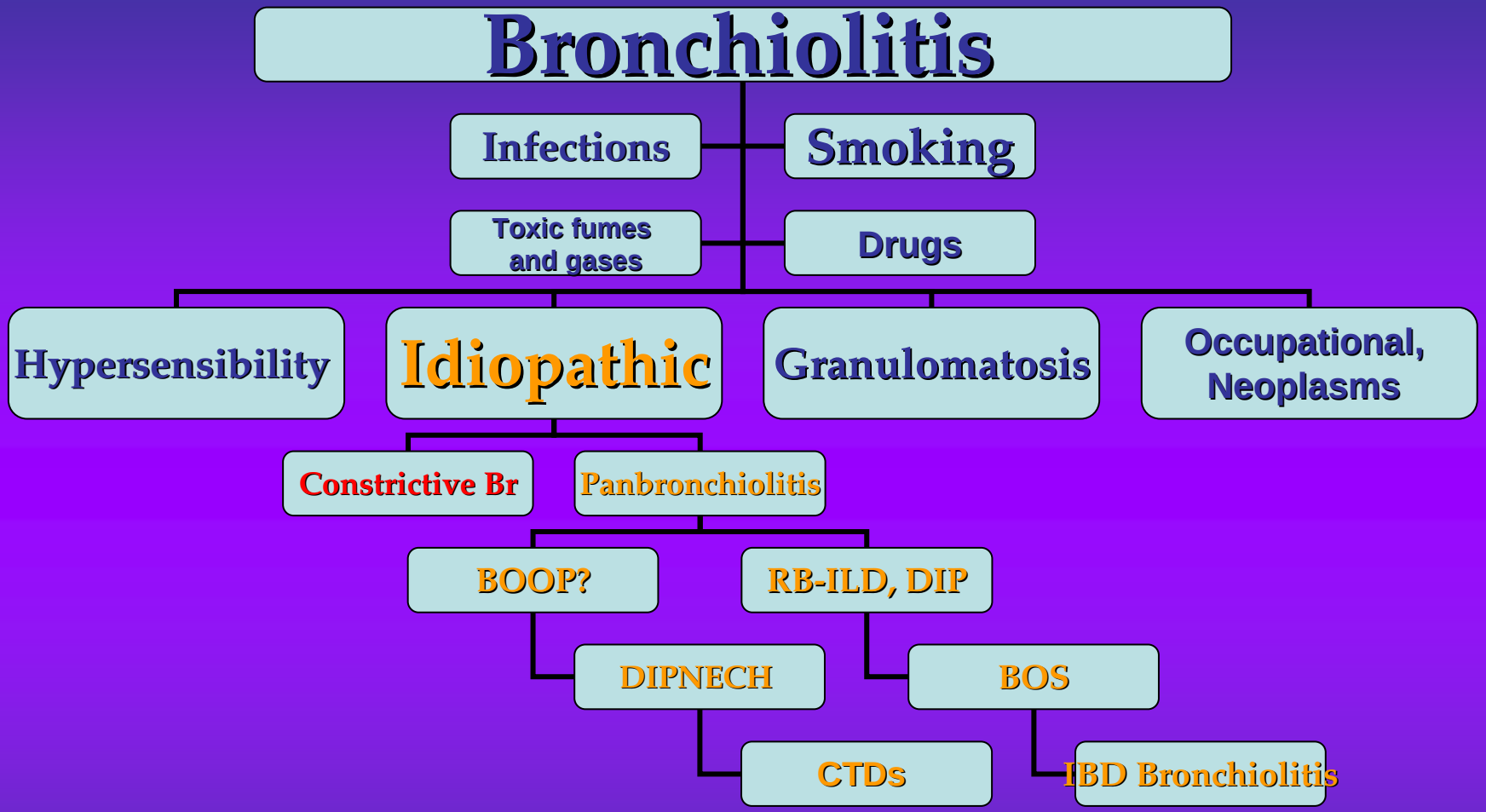
DFOV: 28.8 x 28.8cm


Bronchiolitis
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BOOP?
RB-ILD, DIP
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BOS
CTDs
IBD Bronchiolitis

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State of the Art

Am J Respir Crit Care Med Vol 168. pp 1277–1292, 2003

Bronchiolar Disorder

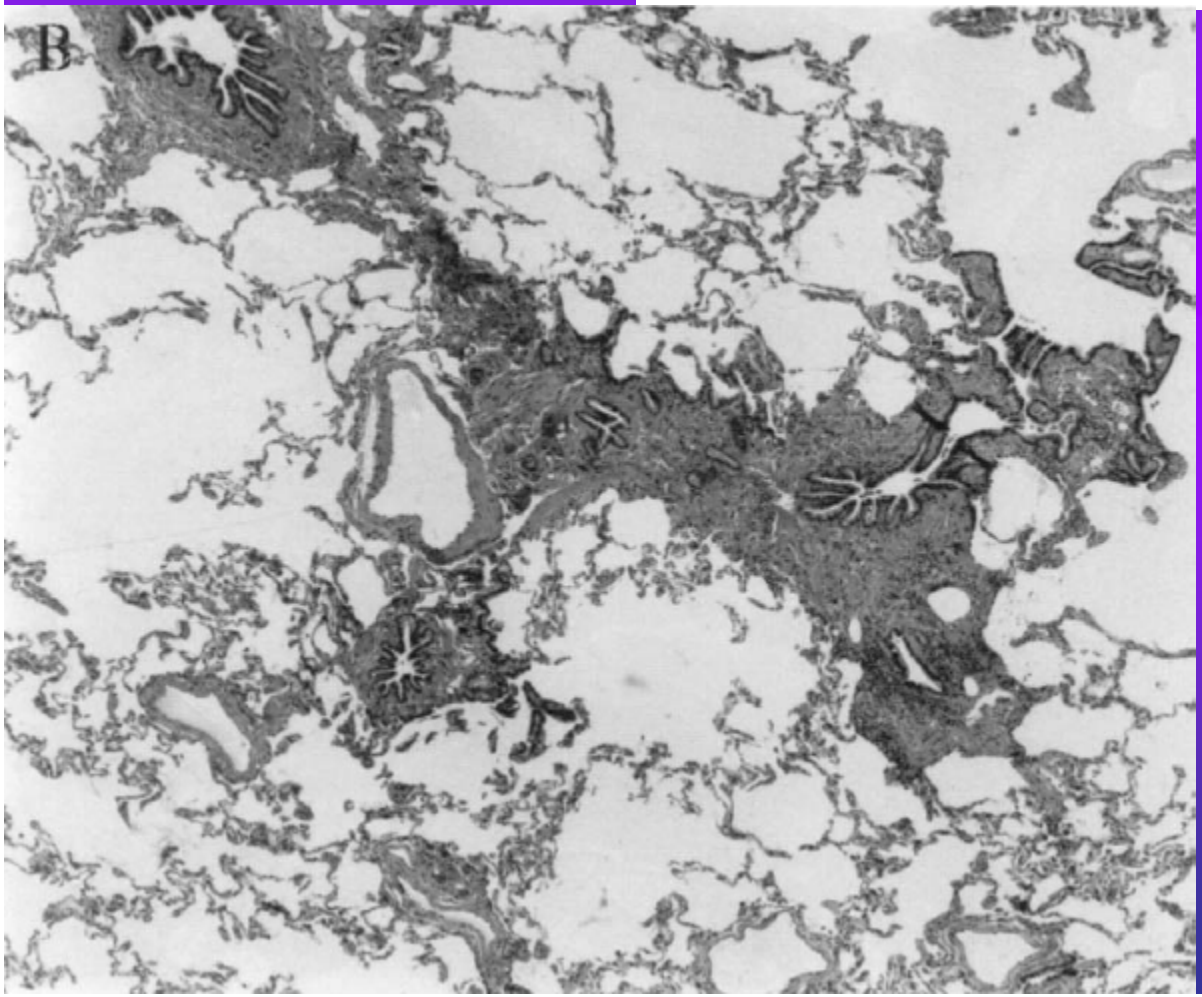
Jay H. Ryu, Jeffrey L. Myers, and Stephen

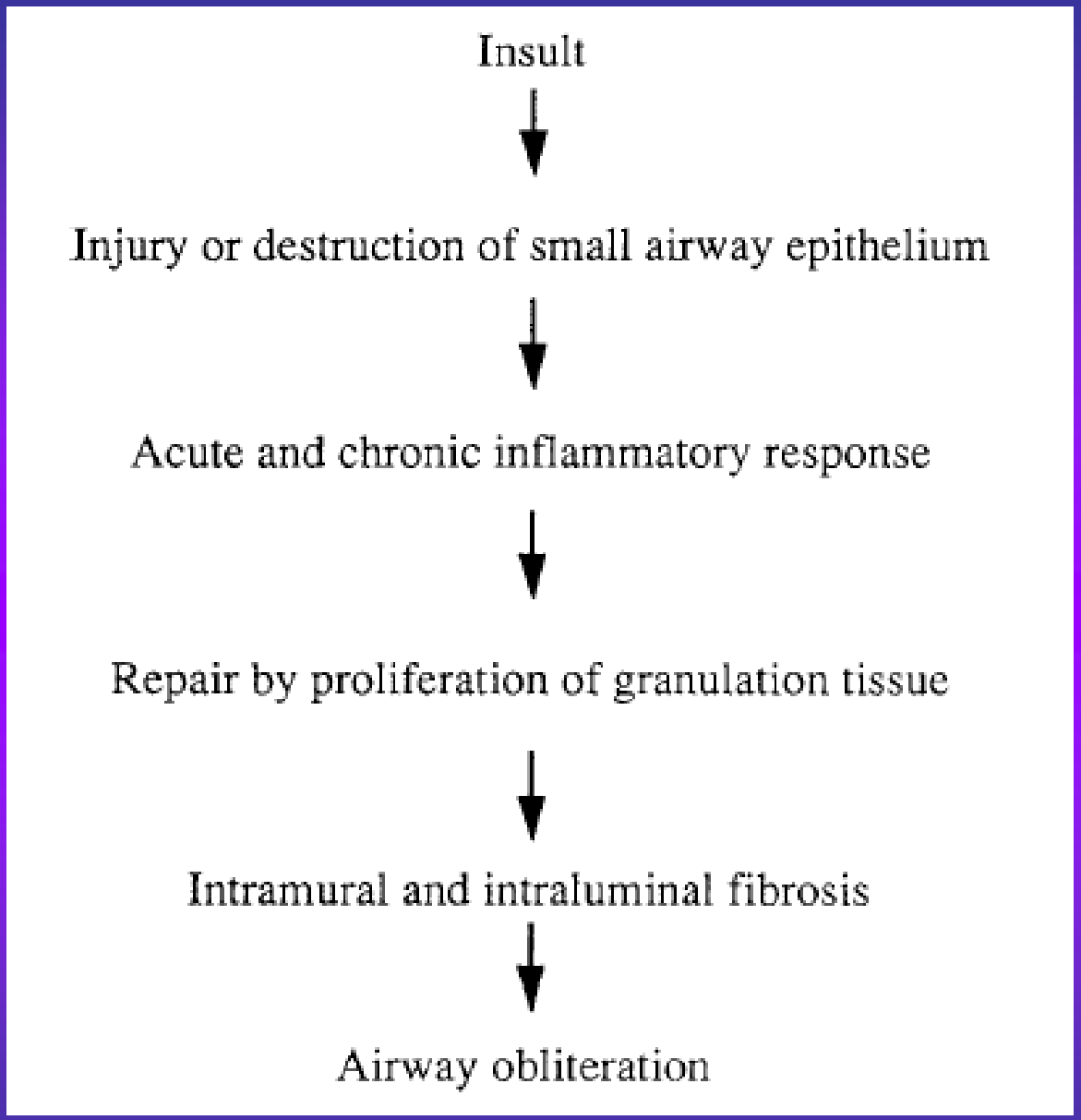
Cryptogenic Constrictive Bronchiolitis

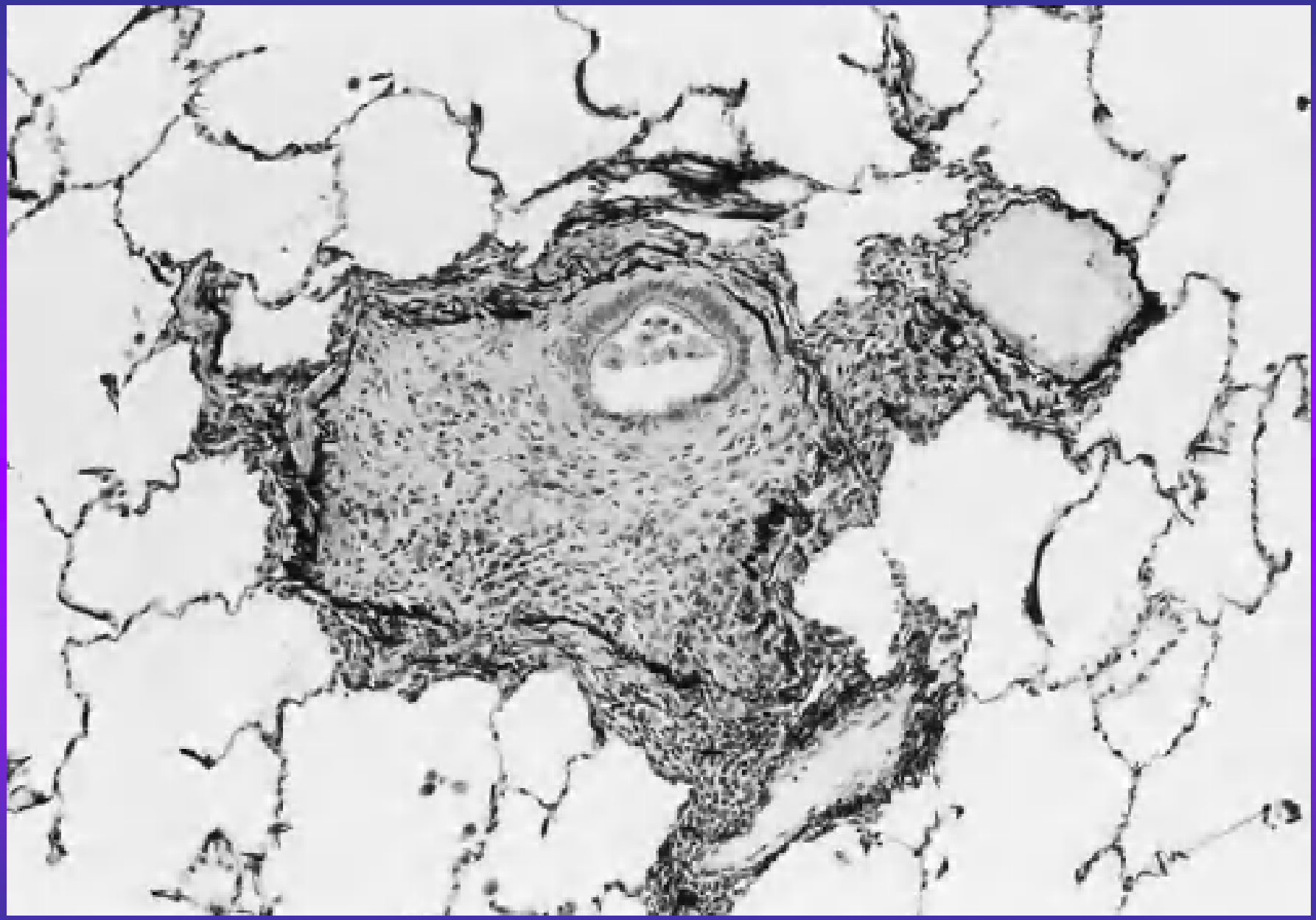
A Clinicopathologic Study

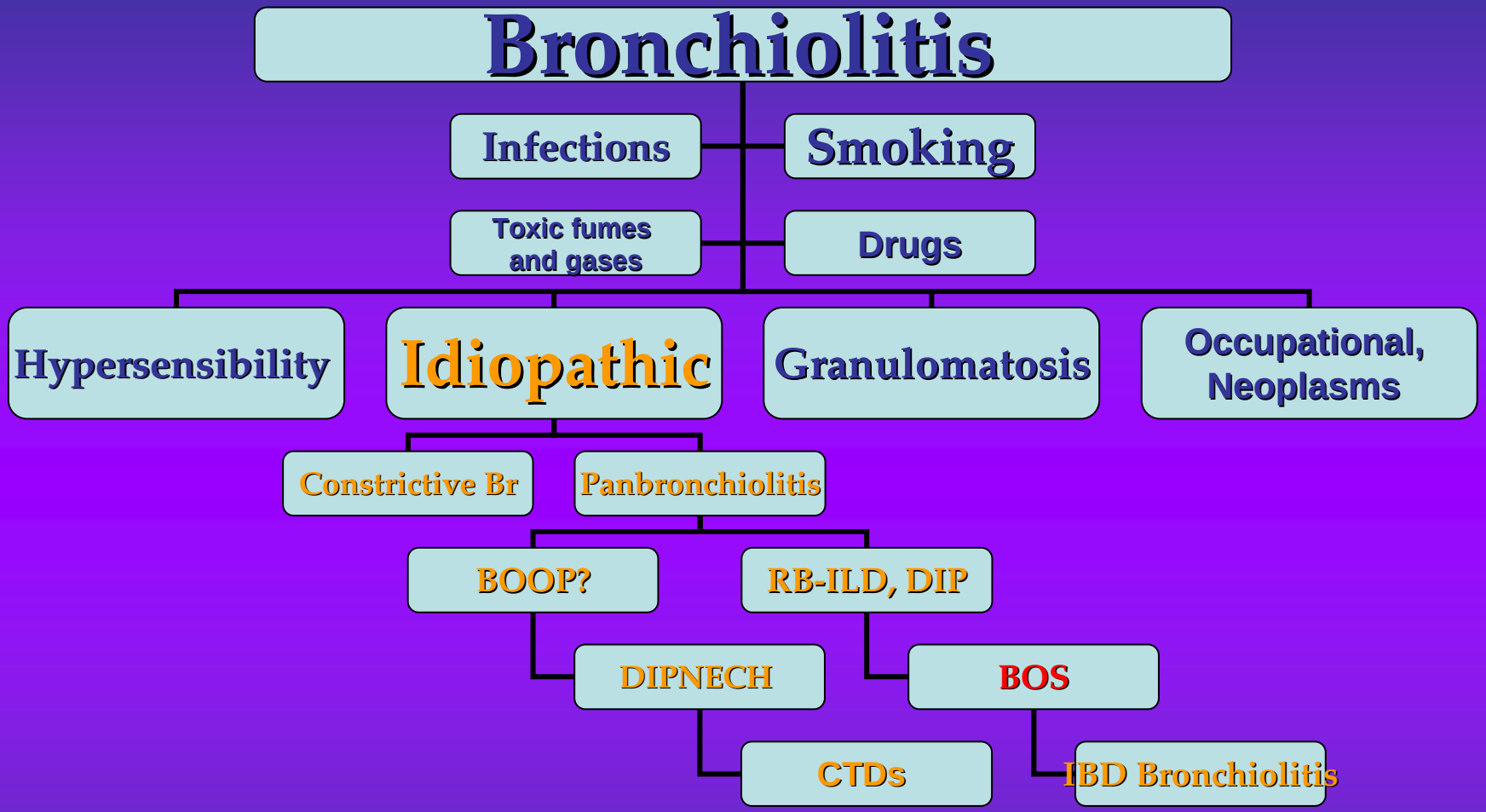
MONICA KRAFT, REBECCA L. MORTENSON, THOMAS V. COLBY, LEE NEWMAN, JAMES A. WALDRON, JR., and TALMADGE E. KING, JR.

Am Rev Respir Dis 1993; 148:1093–101.




Insult
Injury or destruction of small airway epithelium
Acute and chronic inflammatory response
Repair by proliferation of granulation tissue
Intramural and intraluminal fibrosis
Airway obliteration

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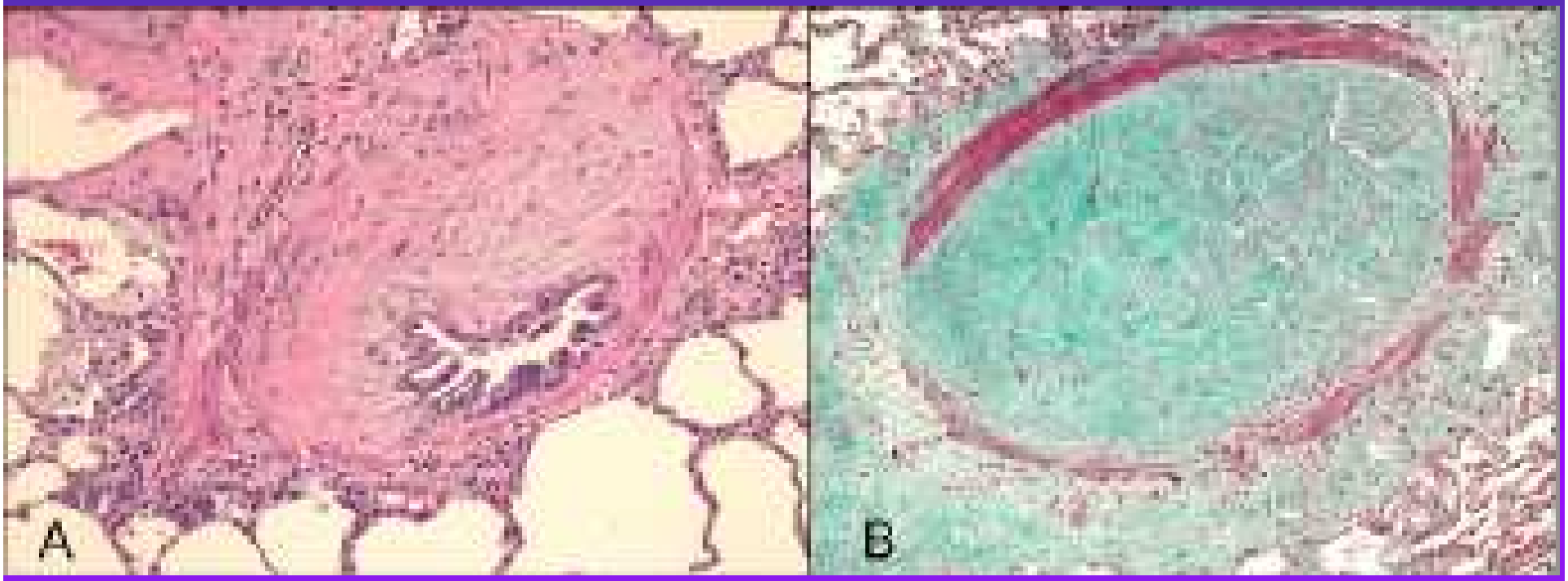
CHEST®

Official publication of the American College of Chest Physicians

Bronchiolitis Obliterans Syndrome : The Final Frontier for Lung Transplantation

Jamie L. Todd and Scott M Palmer

Chest 2011;140;502-508
DOI 10.1378/chest.10-2838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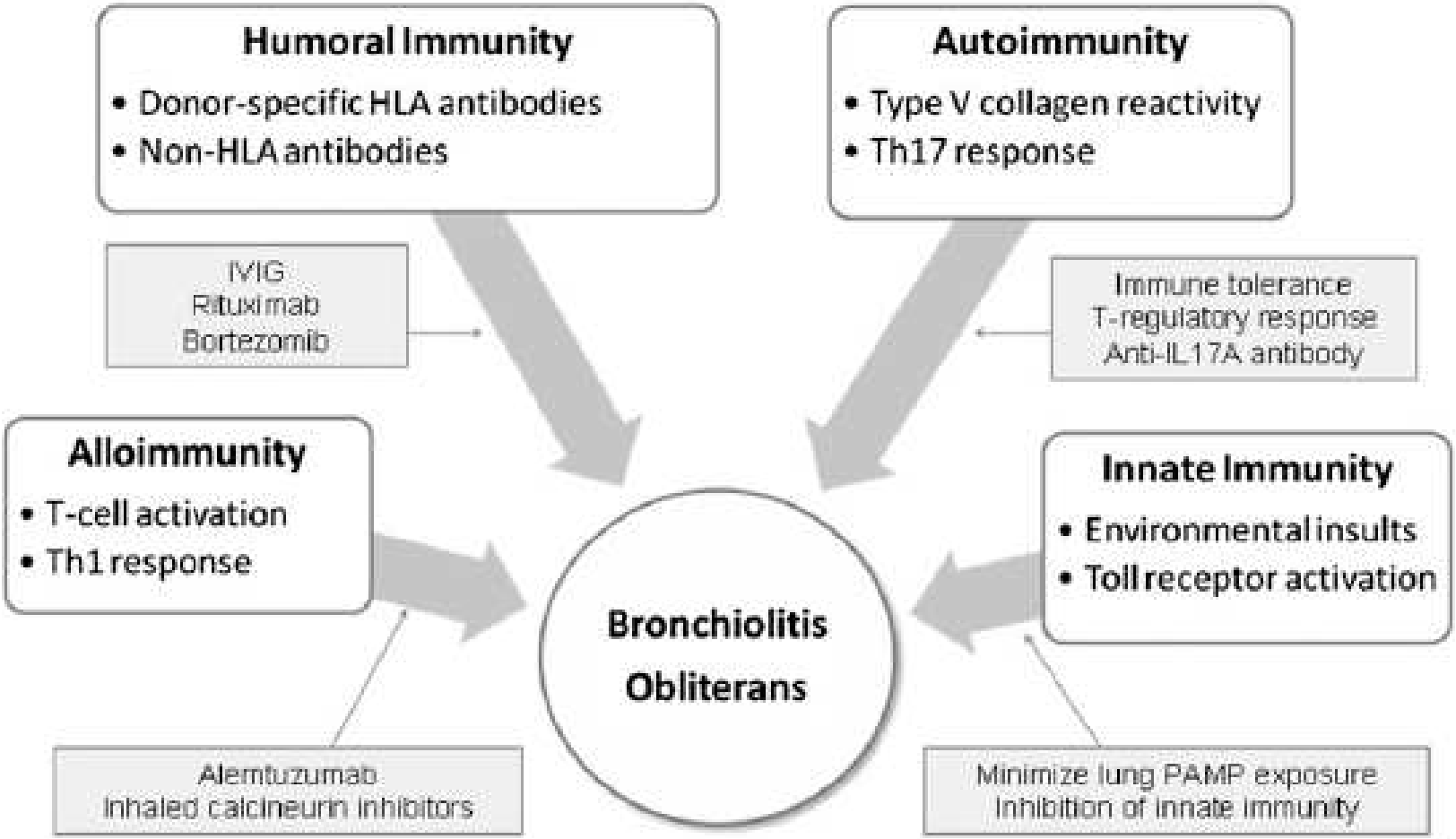
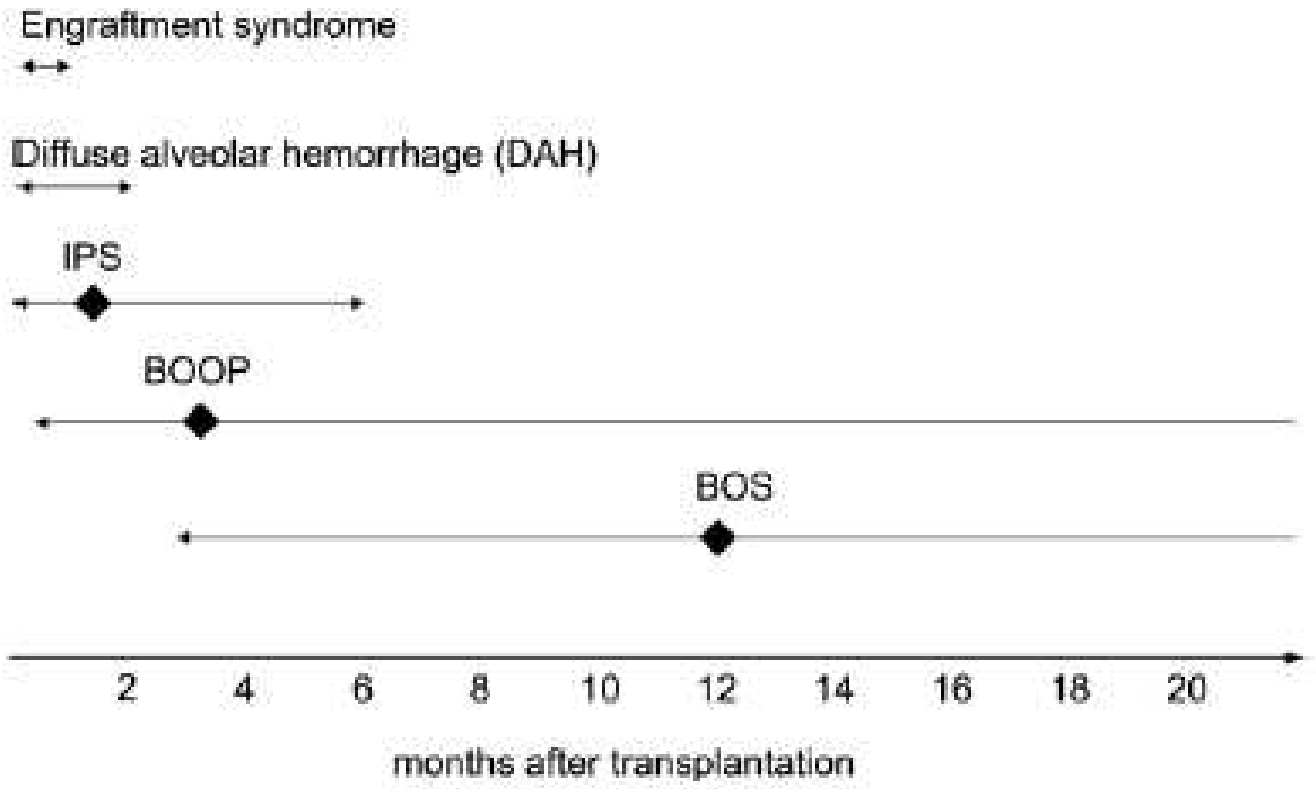
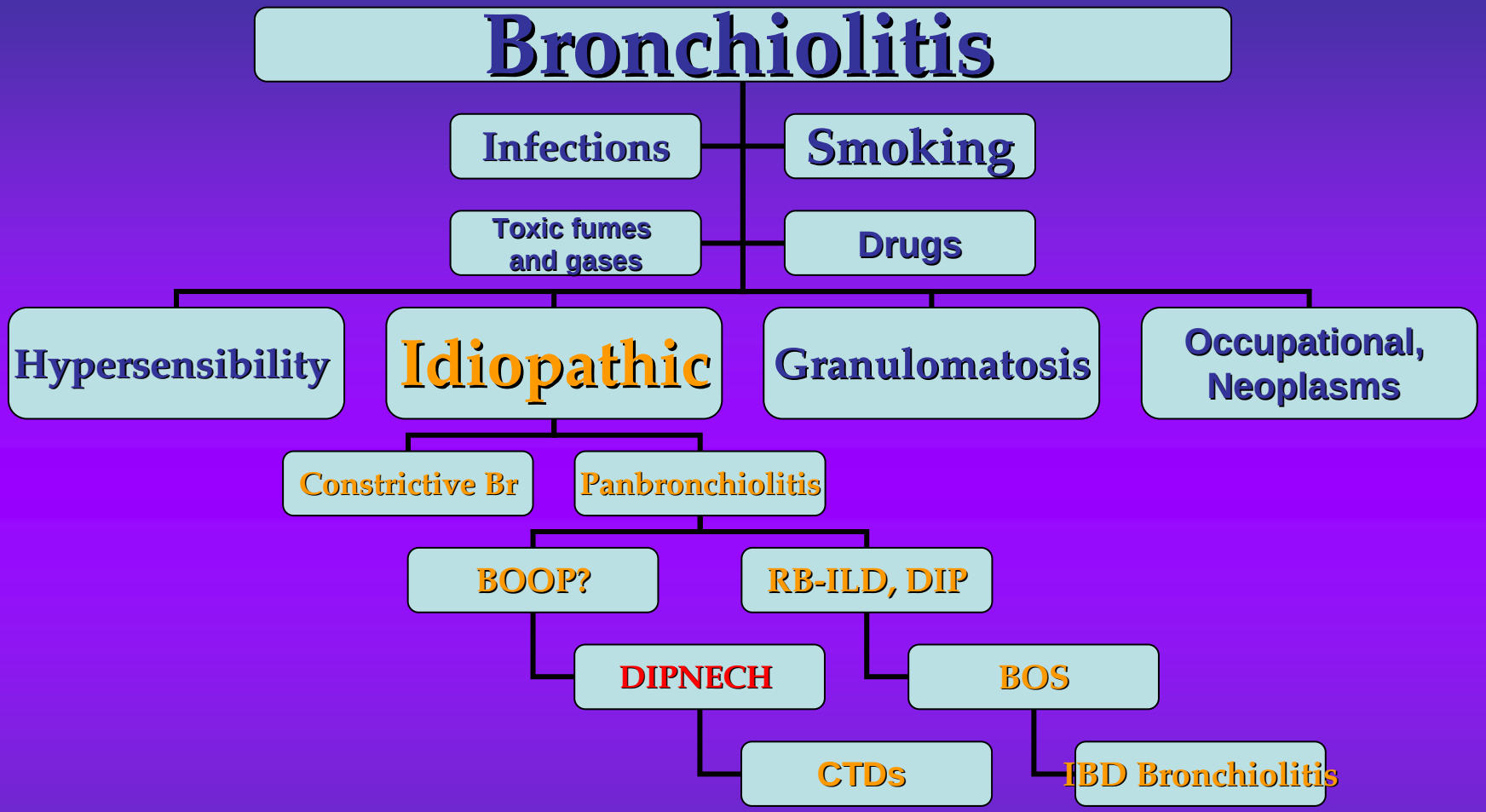

Humoral Immunity
• Donor-specific HLA antibodies
• Non-HLA antibodies
Autoimmunity
• Type V collagen reactivity
• Th17 response
IVIG
Rituximab
Bortezomib
Immune tolerance
T-regulatory response
Anti-IL17A antibody
Alloimmunity
• T-cell activation
• Th1 response
Innate Immunity
• Environmental insults
• Toll receptor activation
Bronchiolitis Obliterans
Alemtuzumab
Inhaled calcineurin inhibitors
Minimize lung PAMP exposure
Inhibition of innate immunity

ASBMT
American Society for Blood and Marrow Transplantation

Bronchiolitis Obliterans Syndrome After Allogeneic Hematopoietic Stem Cell Transplantation—An Increasingly Recognized Manifestation of Chronic Graft-versus-Host Disease

Jason W. Chien,[1] Steven Duncan,[2] Kirsten M. Williams,[3] Steven Z. Pavletic[3]

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IBD Bronchiolitis

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BRIEF REPORT: IDIOPATHIC DIFFUSE HYPERPLASIA OF PULMONARY NEUROENDOCRINE CELLS AND AIRWAYS DISEASE

SAMUEL M. AGUAYO, M.D., YORK E. MILLER, M.D.,
JAMES A. WALDRON, JR., M.D., PH.D.,
ROBERT M. BOGIN, M.D.,
MARY E. SUNDAY, M.D., PH.D.,
GERALD W. STATON, JR., M.D.,
WILLIAM R. BEAM, M.D.,
AND TALMADGE E. KING, JR., M.D.

THE NEW ENGLAND JOURNAL OF MEDICINE Oct. 29, 1992

CANCER

Diffuse idiopathic pulmonary neuroendocrine cell hyperplasia: an under-recognised spectrum of disease

Susan J Davies, John R Gosney, David M Hansell, Athol U Wells, Roland M du Bois, Margaret M Burke, Mary N Sheppard, Andrew G Nicholson

Thorax 2007;**62**:248–252. doi: 10.1136/thx.2006.063065

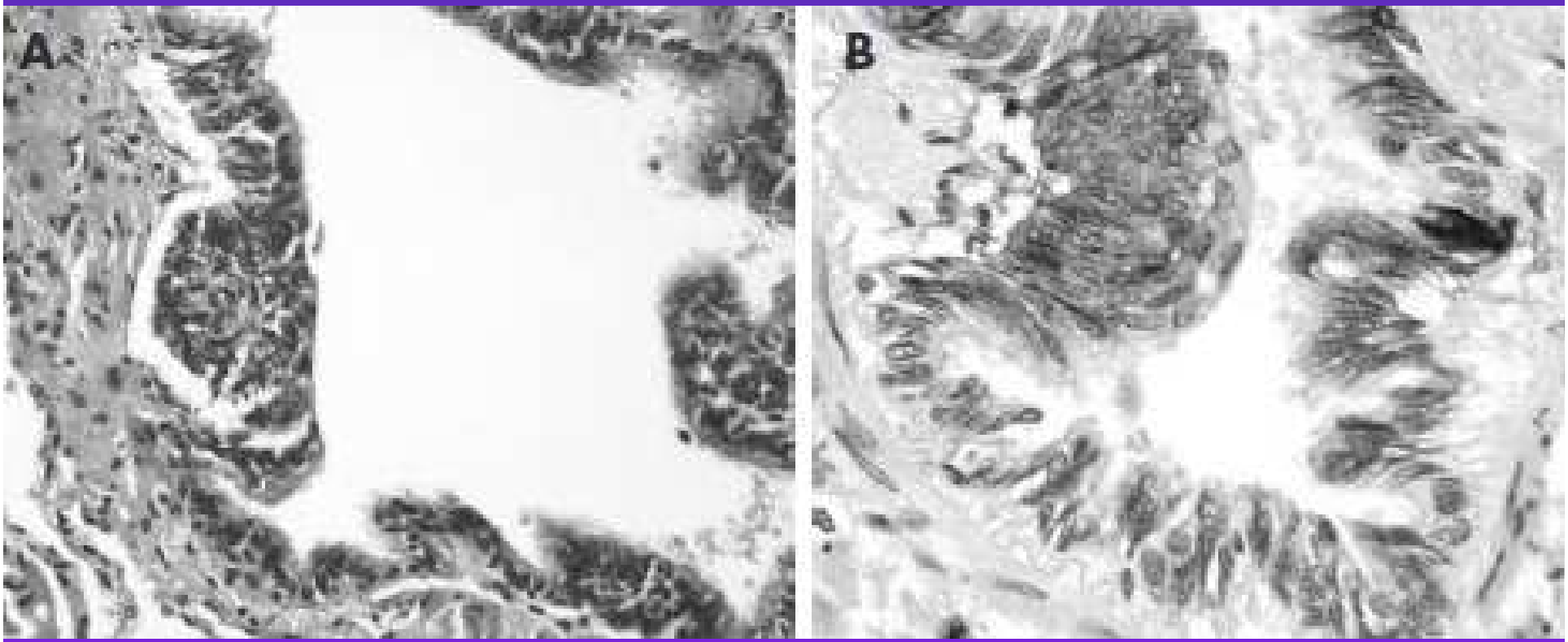
Pulmonary Perspective

Diffuse Idiopathic Pulmonary Neuroendocrine Cell Hyperplasia

A Systematic Overview

Adrienne A. Nassar[1], Dawn E. Jaroszewski[2], Richard A. Helmers[3], Thomas V. Colby[4], Bhavesh M. Patel[5], and Farouk Mookadam[6]

[1]Division of Internal Medicine, Department of Medicine; [2]Division of Cardiothoracic Surgery, Department of Surgery; [3]Division of Pulmonary Medicine, Department of Medicine; [4]Department of Laboratory Medicine and Pathology; [5]Department of Critical Care; and [6]Division of Cardiovascular Diseases, Department of Medicine, Mayo Clinic, Scottsdale, Arizona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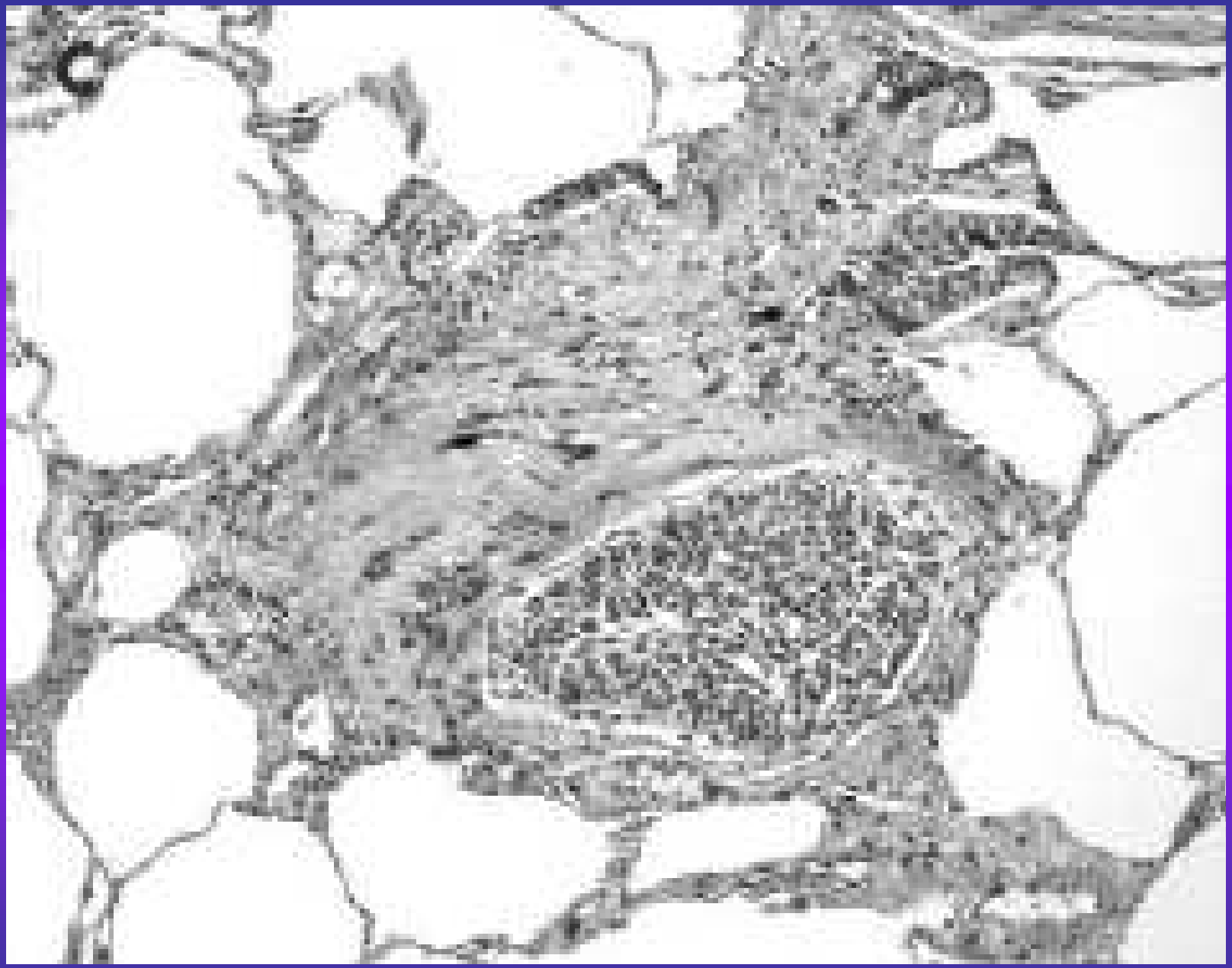

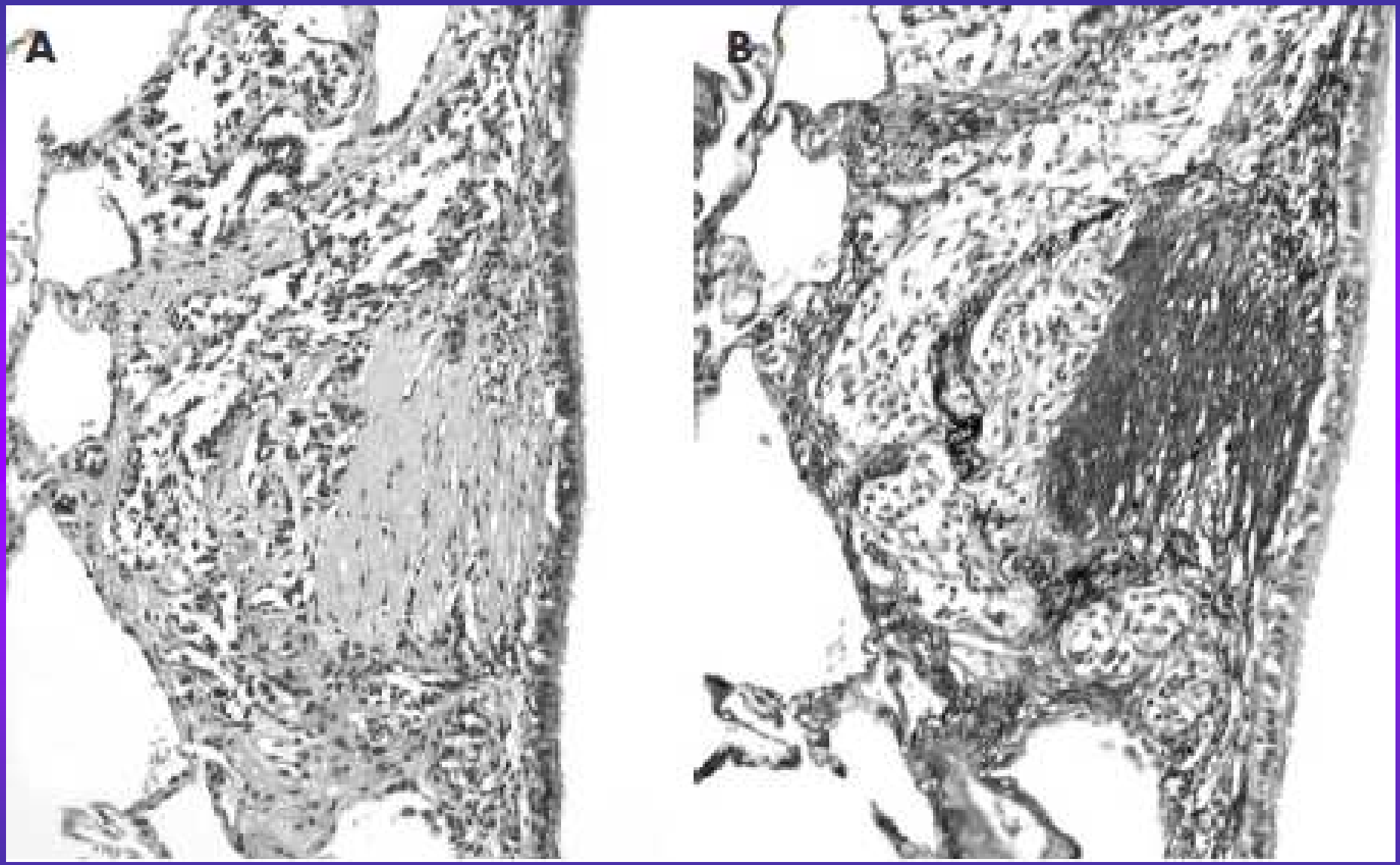
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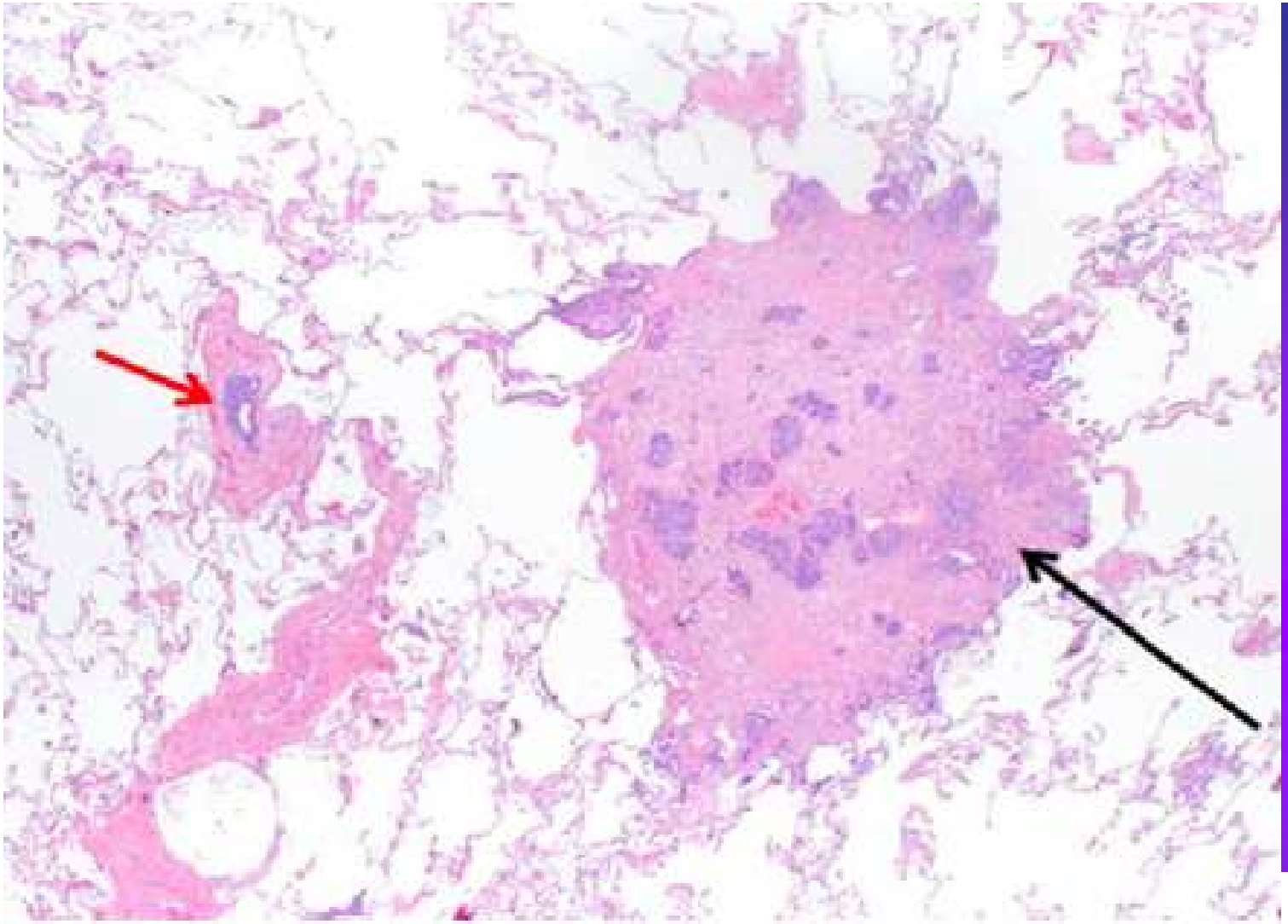

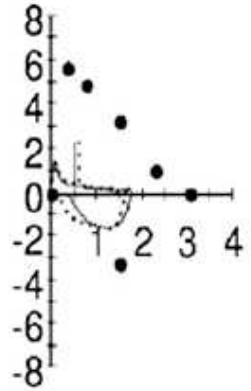
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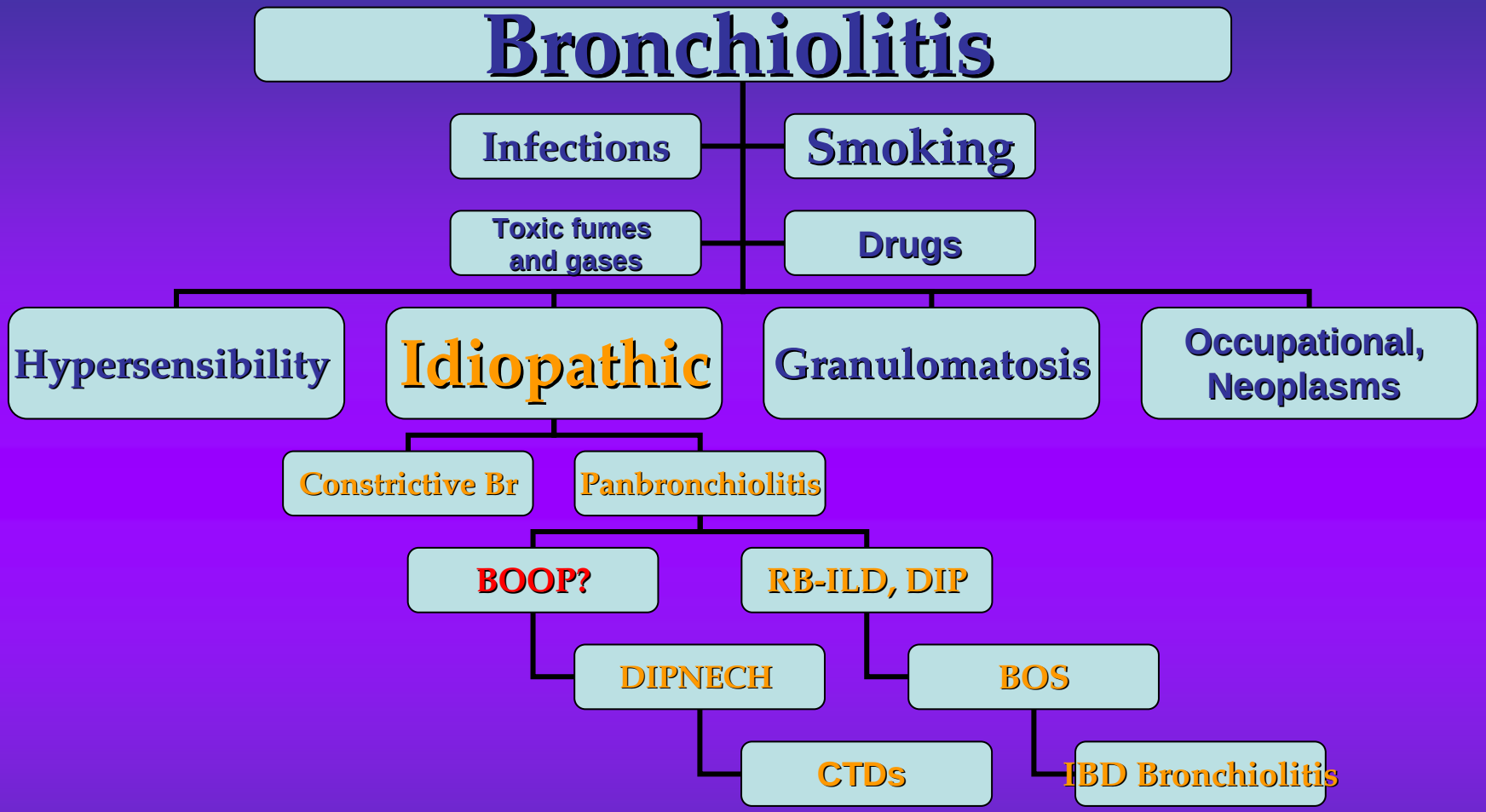
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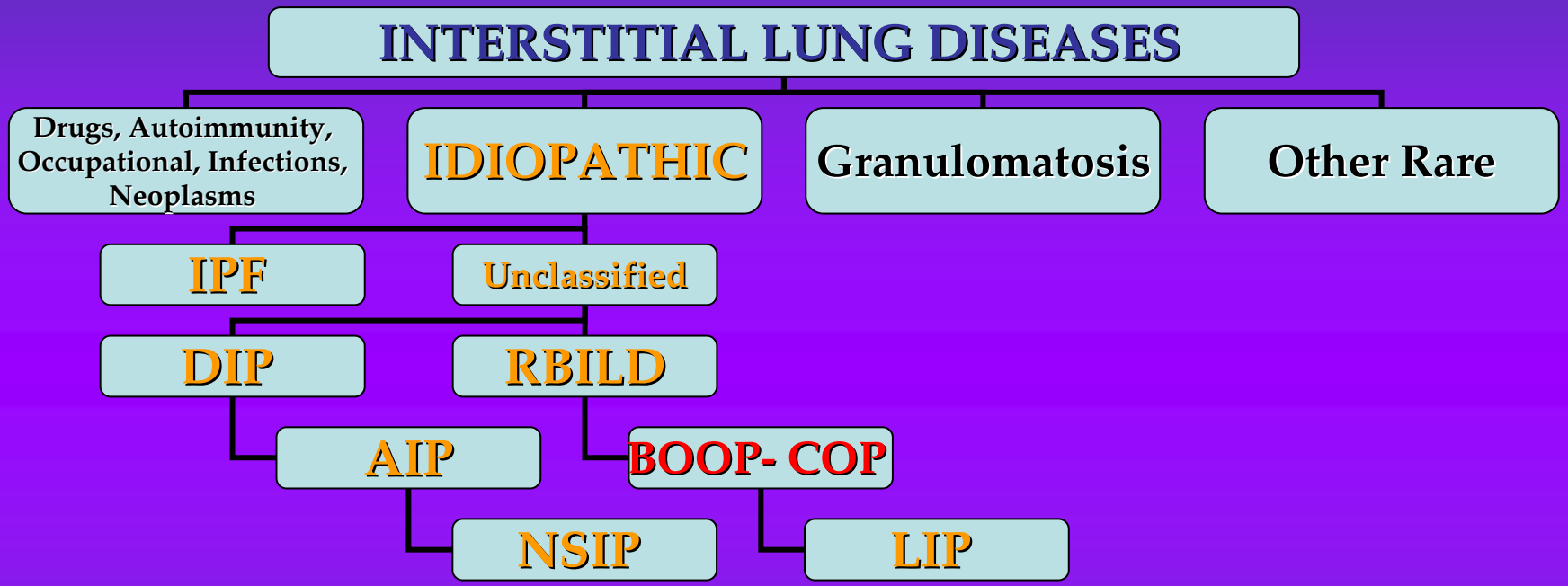

American Thoracic Society/European Respiratory Society International Multidisciplinary Consensus Classification of the Idiopathic Interstitial Pneumonias

THIS JOINT STATEMENT OF THE AMERICAN THORACIC SOCIETY (ATS), AND THE EUROPEAN RESPIRATORY SOCIETY (ERS) WAS ADOPTED BY THE ATS BOARD OF DIRECTORS, JUNE 2001 AND BY THE ERS EXECUTIVE COMMITTEE, JUNE 2001