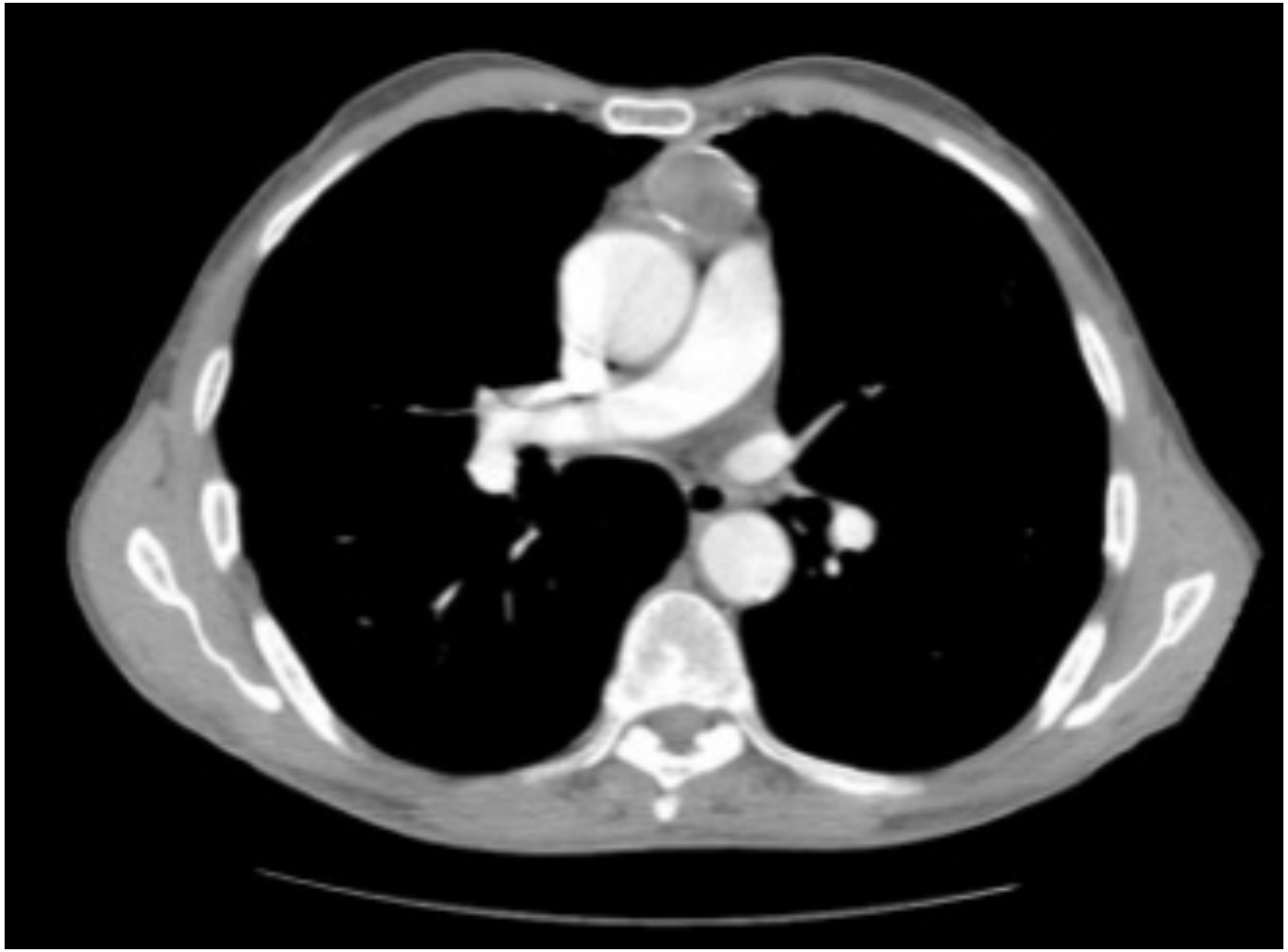
Radiology

Man presents with productive cough, previously well, no pets. Mild esp wheeze and early inspiratory crackles at the bases. CXR normal. CT scan as per.



- What's the diagnosis?
  - A: cystic fibrosis
  - B: hypogammaglobulinaemia
  - C: primary ciliary dyskinesia
  - D: respiratory-muscle weakness
  - E: tuberculosis

There is an anterior mediastinal mass (thymoma) which is associated with hypogammaglobulinaemia and could cause secondary bronchiectasis.

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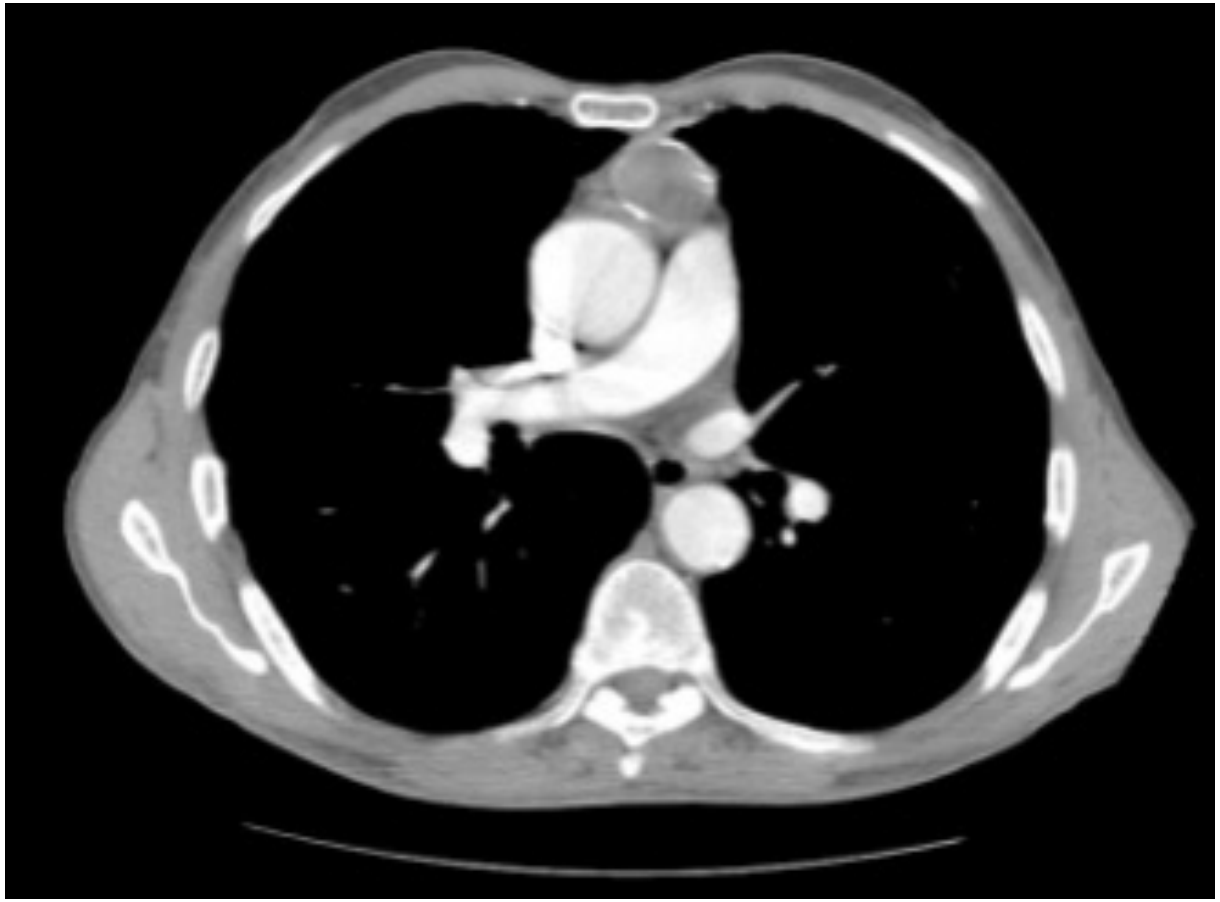
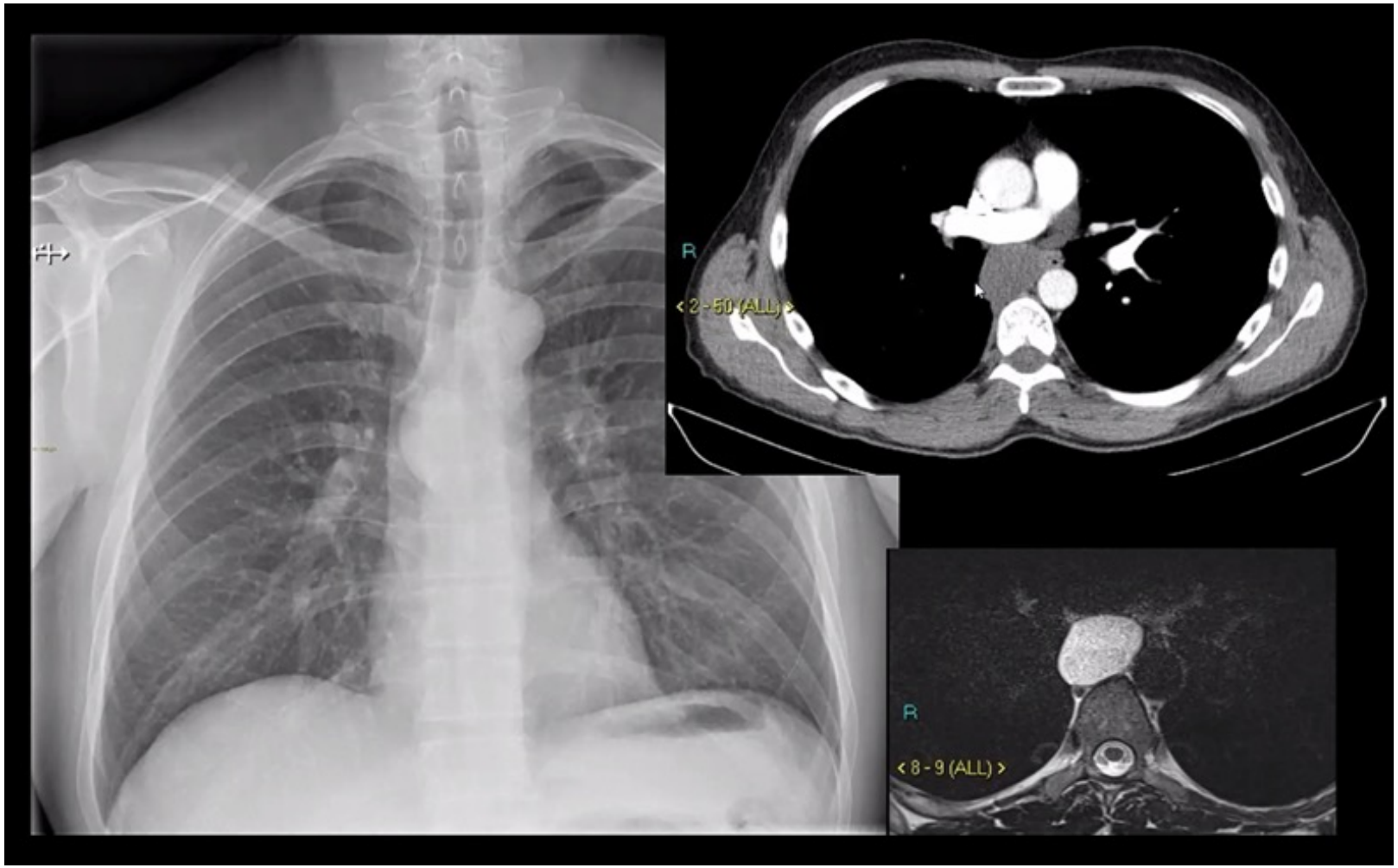


What’s the diagnosis?

A. Bronchogenic cyst
B. Aortic aneurysm
C. Neuroblastoma
D. Germ cell tumour
E. Lymphoma



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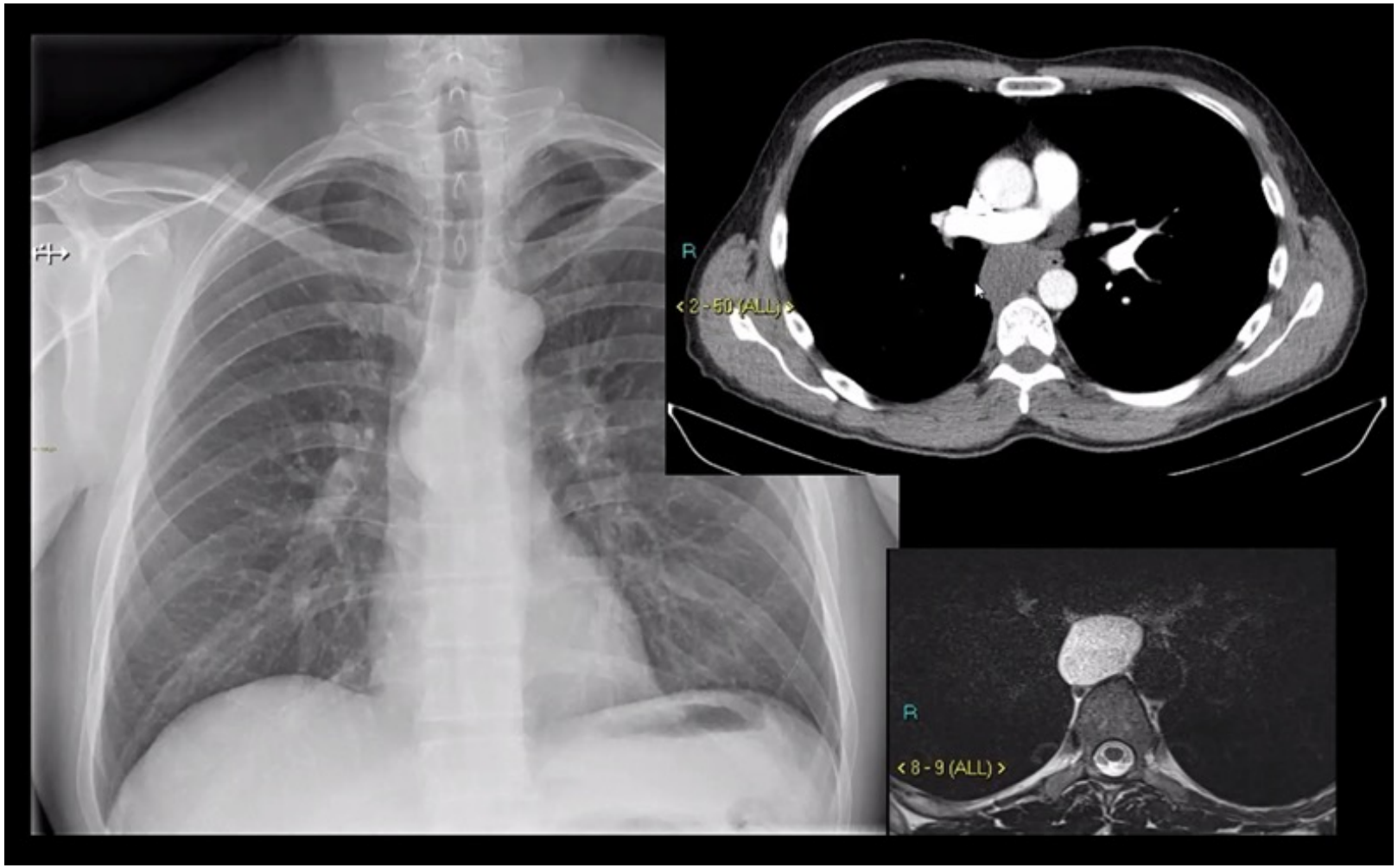

Pul vas disease

SCE Qs

- A 45 year never smoker woman presents herself with severe and progressive dyspnoea. Echocardiography revealed severe pulmonary hypertension with an estimated systolic pulmonary artery pressure of 80 mmHg a dilated right ventricle and a normal function of the left ventricle. Spirometry was normal, but DLCO was reduced (45 % of predicted). HRCT showed paraseptal lines together with mediastinal nodes. Perfusion scintigraphy was normal. Right heart catheterisation confirmed the diagnosis of pulmonary hypertension (mean pulmonary artery pressure of 55 mmHg) together with a normal wedge pressure (12 mmHg). The most likely cause for her pulmonary hypertension is:

Select one:

- Pulmonary veno-occlusive disease
- Vasculitis
- Schistosomiasis
- Undiagnosed left heart disease.

SCE Qs

- Pulmonary hypertension and low diffusion:
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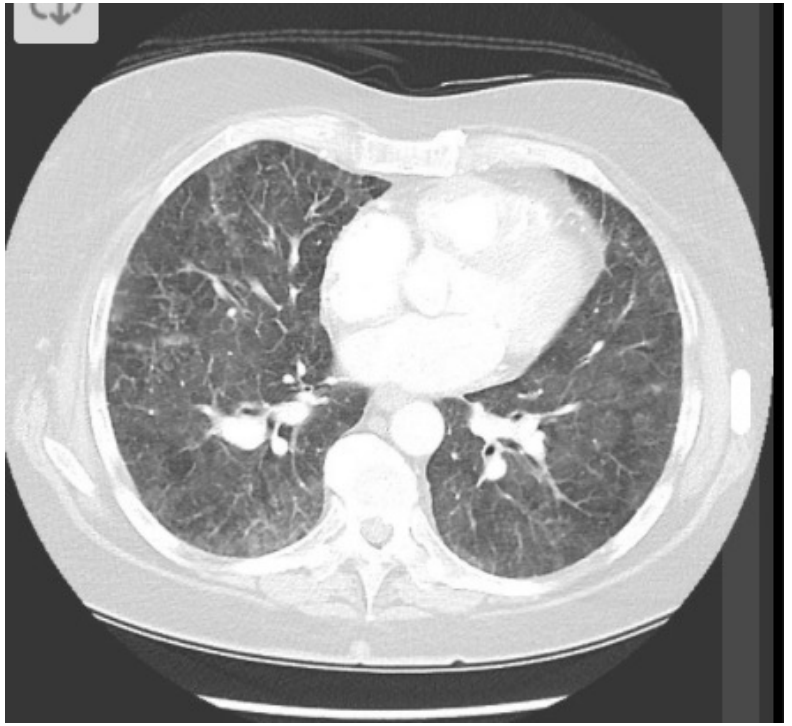

Select one:

- Pulmonary veno-occlusive disease
- Vasculitis
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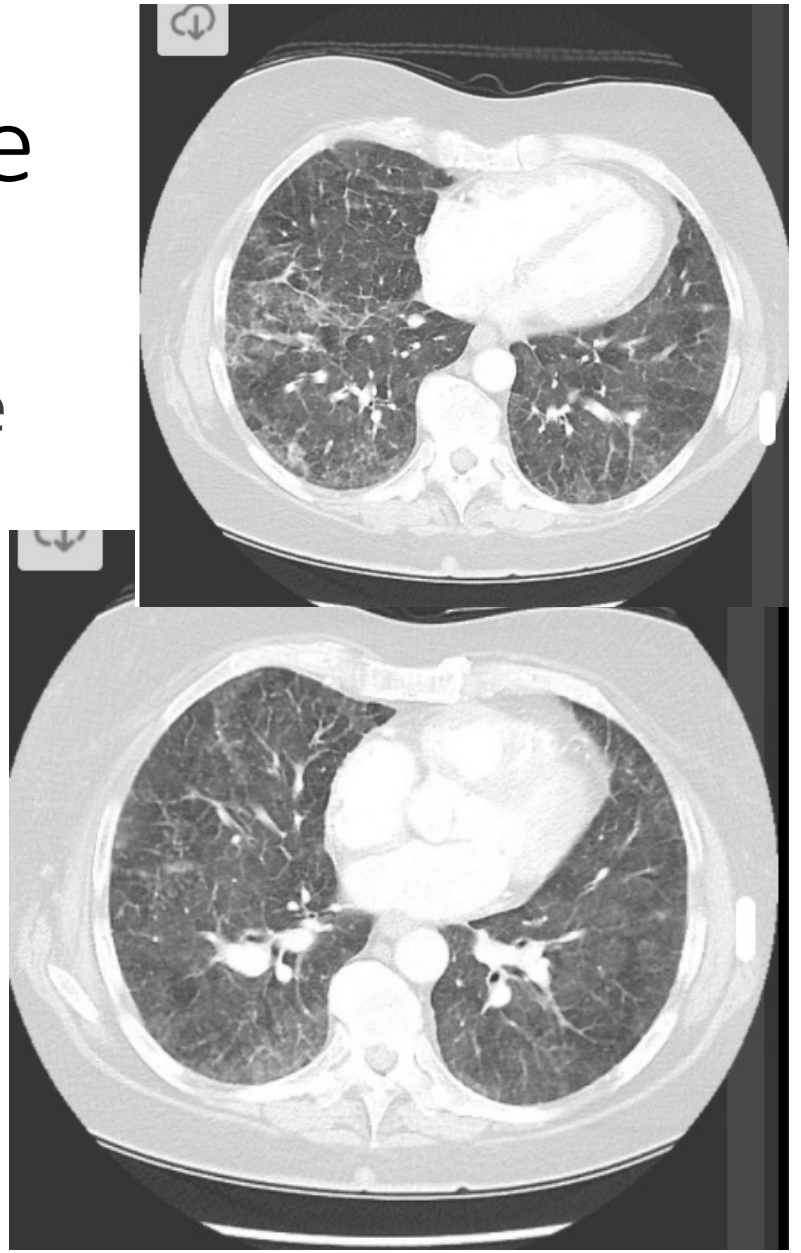
- Undiagnosed left heart disease.

Pulmonary veno occlusive disease

- regions of ground-glass opacities, which can be
  - diffuse multifocal [1,4]
  - perihilar [4]
  - patchy [4]
  - centrilobular [2,4]: most cases [8,9]
- smooth interlobular septal thickening
- mediastinal lymph node enlargement [9]
- pleural effusions
- enlarged central pulmonary arteries
- mosaic pattern of lung attenuation
- normal calibre pulmonary veins



Idiopathic in most part but associated with other conditions often (CTD, HIV, LCH) EIF2AK4 gene as a cause of heritable form

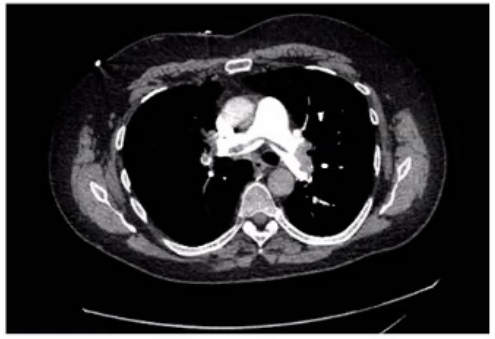
SCE PE Qs

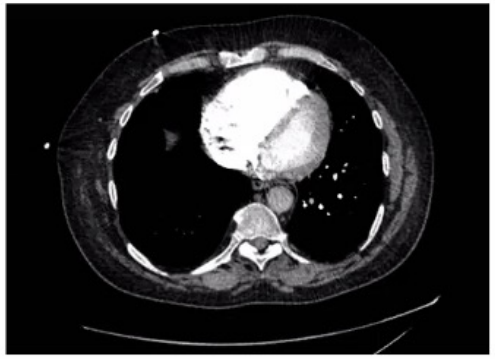
- RV dysfunction and RV
- DVT increases the risk of complication
- 25%

Q 12

A 40 year old man presents acutely with a first PE. On examination he has a pulse 120 bpm, BP 124/86, SaO_2 94% on air and a swollen left leg.
Investigations include an elevated HsTroponin and an elevated lactate. His Full Blood count, and renal function are normal. What is the likelihood that the patient will have a complicated 30 day course.

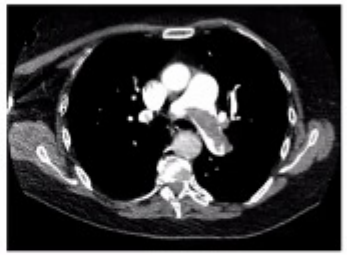
A. < 1 %
B. 2-4 %
C. 5-10 %
D. 10-20 %
E. > 20 %

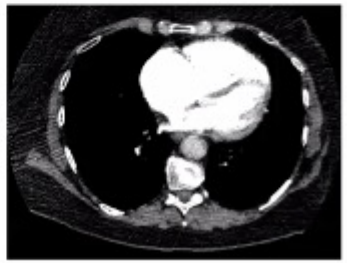




Q11

A 61 year old woman who has recently been on a plane flight from Los Angles presents with sudden onset dyspnoea and chest pain. On examination she has a pulse 123 bpm, BP 80/60 and SaO_2 92% on 15 l/min O_2. Her Full Blood count, and renal function is normal. What treatment would you recommend?





A. Catheter directed therapy
B. Embolectomy
C. Intravenous heparin
D. Low molecular weight heparin
E. Thrombolysis

- E – thrombolysis

Q13

In a patient presenting with an acute pulmonary thromboembolism, chronic thrombo-embolic pulmonary hypertension is thought to occur in:

A.< 0.05 % of patients
B.< 0.5 % of patients
C.≥ 0.5 and < 5% of patients
D.≥ 10 and < 20% of patients
E.≥ 30% of patients

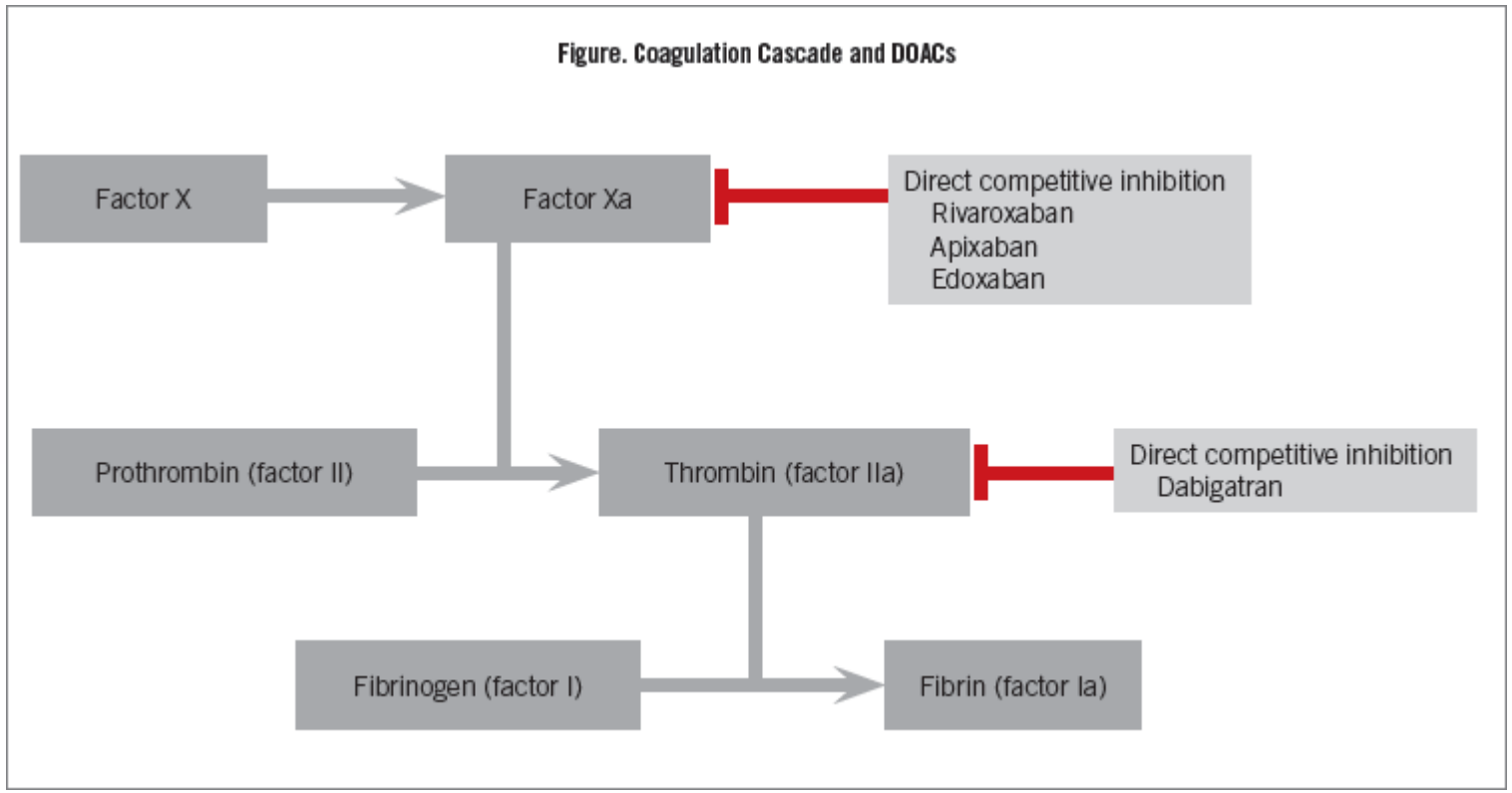
- 0.5-5%
- 3.8% to be exact

Q14

What type of anticoagulant is rivaroxaban?

A. Vitamin K antagonist
B. Factor Xa inhibitor
C. Direct thrombin inhibitor
D. Unfractionated heparin
E. None of the above

- B



A 65-year-old woman with type 2 diabetes mellitus and systemic hypertension presented with a 12-month history of progressive breathlessness. Her medical history included deep venous thrombosis and heart block treated by pacemaker insertion.

On examination, her JVP was elevated. Her heart sounds were normal, and her lung fields were clear.

Investigations:

chest X-ray	cardiomegaly
CT scan of thorax with contrast	both atria dilated; no evidence of pulmonary embolism; normal lung parenchyma
right heart catheter:	
mean arterial pressure	95 mmHg (90)
mean right atrial pressure	11 mmHg (3)
mean pulmonary arterial pressure	38 mmHg (15)
mean pulmonary arterial wedge pressure	20 mmHg (9)
cardiac index	4.2 L/min/m^2 (2.8–4.2)

What is the most likely diagnosis?

Answers

A: chronic thromboembolic pulmonary hypertension
B: pulmonary arterial hypertension
C: pulmonary capillary haemangiomatosis
D: pulmonary hypertension associated with left heart disease
E: pulmonary hypertension associated with respiratory disease

A 65-year-old woman with type 2 diabetes mellitus and systemic hypertension presented with a 12-month history of progressive breathlessness. Her medical history also included deep venous thrombosis and heart block treated by pacemaker insertion.

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Answers

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B: pulmonary arterial hypertension
C: pulmonary capillary haemangiomatosis
D: pulmonary hypertension associated with left heart disease
E: pulmonary hypertension associated with respiratory disease

Correct answer: D

Explanation

This is pulmonary hypertension, since the pressure is more than 25 mmHg, and the raised pulmonary arterial wedge pressure indicates it is due to left heart disease.

Airways

1. Which of the following tests a direct airway challenge? more than 1 may apply

A. Exercise
B. Hypertonic saline challenge
C. Mannitol challenge
D. Histamine challenge
E. Metacholine

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Indirect felt to be more ‘useful’ in asthma

2. What are the cut offs to define a positive mannitol and positive methacholine challenge for AHR

- Methacholine PC20, Mannitol PD15
- Methacholine PD15, Mannitol PD20
- Methacholine PC15, Mannitol PD20
- Methacholine PC10, Mannitol PD15
- Methacholine PC20, Mannitol PD 10

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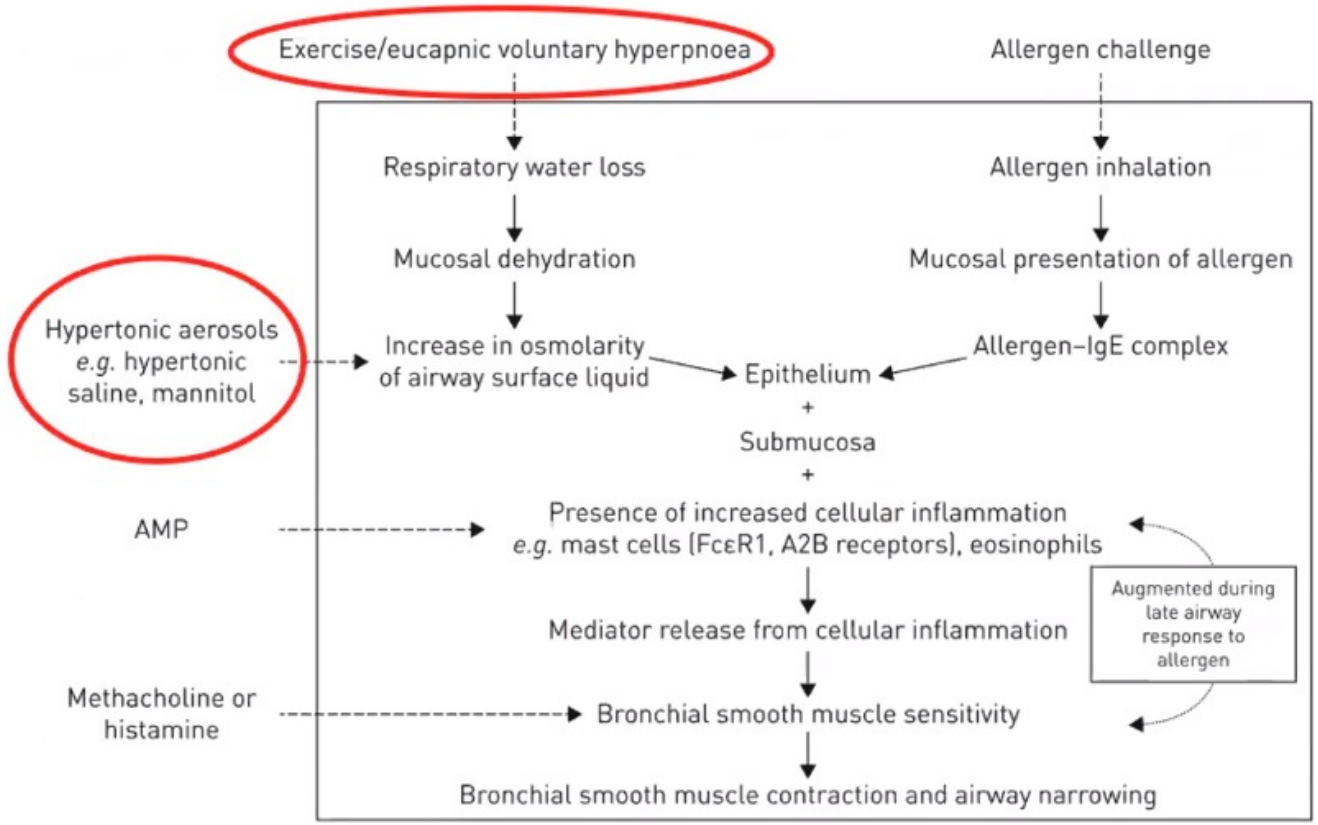
Challenge tests – can be direct or indirect

Challenge test (either direct or indirect) aim to cause dehydration of bronchial epithelium which causes an inflammatory response

Indirect

- Indirect challenges can be with exercise/hypertonic saline/mannitol
- Good negative PPV, negative PPV isnt as good

Mechanisms of action of indirect challenge tests used in the clinical and research setting.



Teal S. Hallstrand et al. Eur Respir J 2018;52:1801033

Challenge Testing

DIRECT challenge test

- Methacholine or histamin considered negative, PC20 >16mg/ml
  - Just trying to
  - 20% change in FEV1 at a PC20 of <1 mg/ml-16mg/ml is regarded as positive
  - Trying to make them drop their FEV1
  - Methacholine or histamine

Referral for challenge tests should be considered in adults with no evidence of airflow obstruction on initial assessment in whom other objective tests are inconclusive but asthma remains a possibility

Methacholine

- PC20 (FEV1 drop 20% = stop)
- Thresholds of drug
  - >16mg = normal

CATEGORIZATION OF BRONCHIAL RESPONSIVENESS

PC_{20} (*mg/ml*)	Interpretation*
> 16	Normal bronchial responsiveness
4.0–16	Borderline BHR
1.0–4.0	Mild BHR (positive test)
< 1.0	Moderate to severe BHR

* Before applying this interpretation scheme, the following must be true: (*1*) baseline airway obstruction is absent; (*2*) spirometry quality is good; (*3*) there is substantial postchallenge FEV_1 recovery.

This is the test for airway hyperresponsiveness

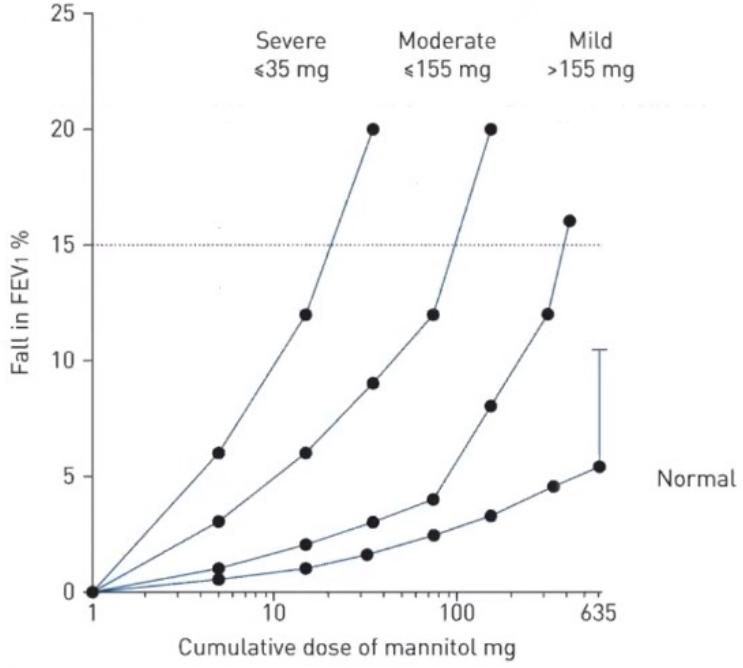
Interpreting test

- Measure FEv1 at the start and then after each dose
- Stop when FEV1 drops by defined % or you reach max dose
- Dose u stop at tells u how easily provoked their airway is
- PD = provocative dose (mannitol 15% FEV1 drop = u stop test)
- PC = provocative concentration (methacholine 20% FEV1 drop = u stop)
- Smalls numbers after = threshold FEV1 drop
- PD15 = 15% drop in FEV1
- PC20 means connc that cause 20% drop in FEV1

Mannitol (indirect)

- PD 15 (15% FEV1 drop u stop)
- Drug thresholds
  - Max drug dose 635mg mannitol
  - <35mg to provoke 15% FEV1 drop = severe
  - <155mg to provoke 15% FEV1 drop = moderate
  - >155mg to provoke 15% FEV1 drop = mild

Classification of the severity of airway hyperresponsiveness according to the response to dry powdered mannitol challenge.



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3. Which of the following lower you FeNO – 2 to find

- Allergic sensitisation and exposure
- Smoking
- Rhinovirus
- Rhinitis and nasal polyposis
- COPD
- Children/young adult
- Beetroot salad

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- Allergic sensitisation and exposure
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- Beetroot salad (nitrate intake – dark choc)

FeNO thresholds

- Both ATS and NiOX suggest the following thresholds in **adults**:

FeNO (ppb)*	LOW	INTERMEDIATE	HIGH
Adults	<25	25-50	>50

- NICE uses a cut off of **40** as high (in the context of diagnosis) and 25-39 as intermediate.
- In those 16 or under, NICE recommends cut off of **35**ppb

4. Which biologic will drop your FeNO most?

A. Omalizumab
B. Dupilumab
C. Mepolizumab
D. Benralizumab

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FeNO

- FeNO is an INDIRECT measure of Th2 inflammation and eosinophilia
- Activated via IL-4/IL-13

DUPIXENT® (**dupilumab**) inhibits signalling of both IL-4 and IL-13

This is relevant when ur thinking about biologics

- Biologicals are very specific targets so
- Anti-IL5s wont neccearily drop your FeNO
- But if u give Dupilumab then FeNO will drop

5. A patient comes to clinic with a diagnosis of asthma. She takes Fostair 200/6 BD, good inhaler technique, but complains of ongoing breathlessness and nocturnal cough. She takes her SABA 3 x week currently. What would u do?

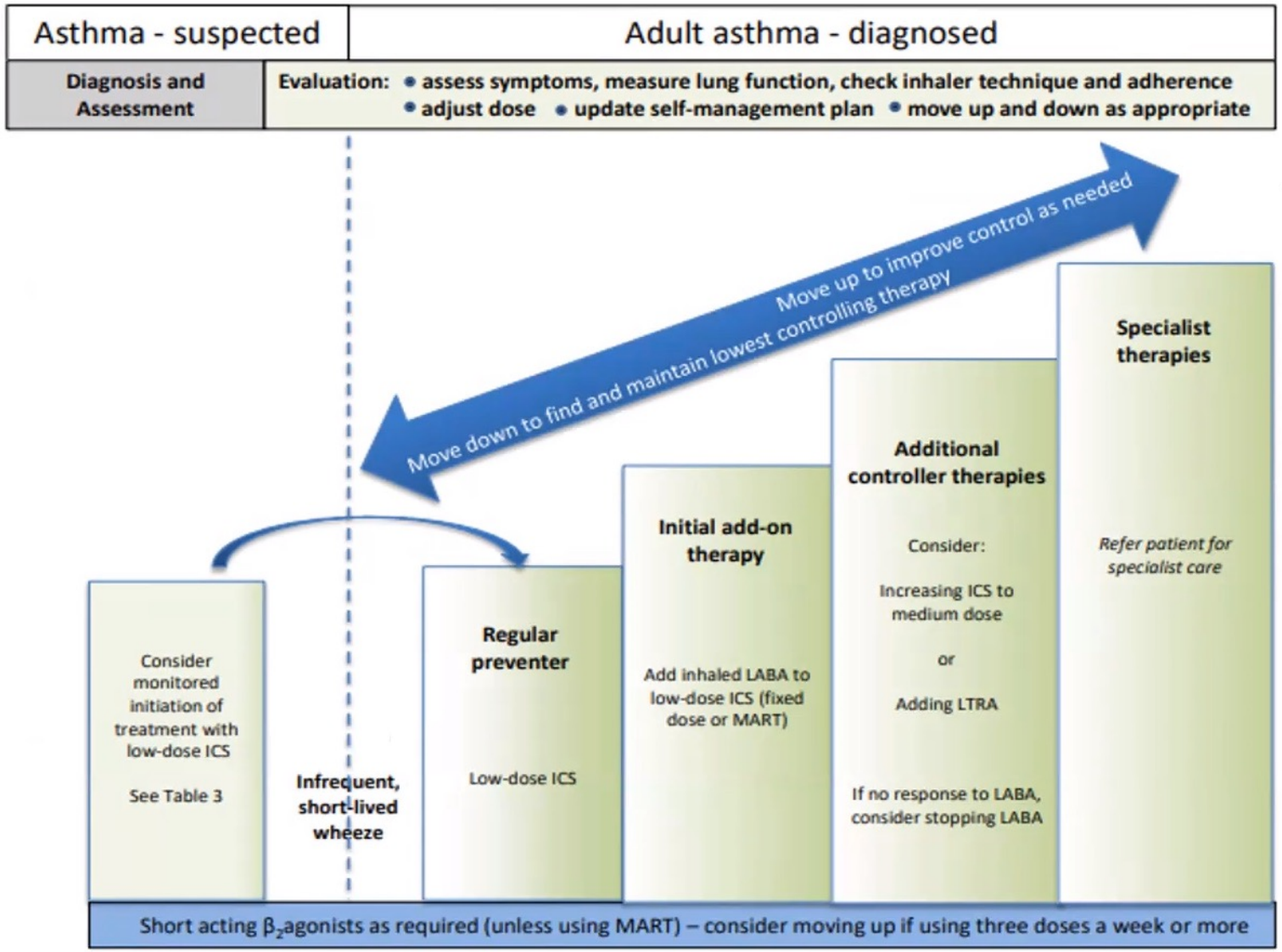
A. Add in oral theophiline
B. Add in LTRA
C. Add in LAMA
D. Ref to biologics
E. Add in low dose oral prednisolone

SIGN 158

British guideline on the management of asthma

A national clinical guideline

First published 2003
Revised edition published July 2019



What are the key differences between the SIGN/BTS$_{2019}$ versus NICE guidelines$_{(2021)}$ for asthma?

A. NICE advocates use of SABA alone as a 1st step in ALL patients
B. NICE favours use of LTRA before ICS/LABA
C. SIGN/BTS favours the use of addition of LAMA when patient still uncontrolled

6. Which interleukin activates neutrophils in T2 low asthma?

A. IL-4 activates neutrophils
B. IL-5 activates neutrophils
C. IL-9 activates neutrophils
D. IL-13 activates neutrophils
E. IL-17 activates neutrophils

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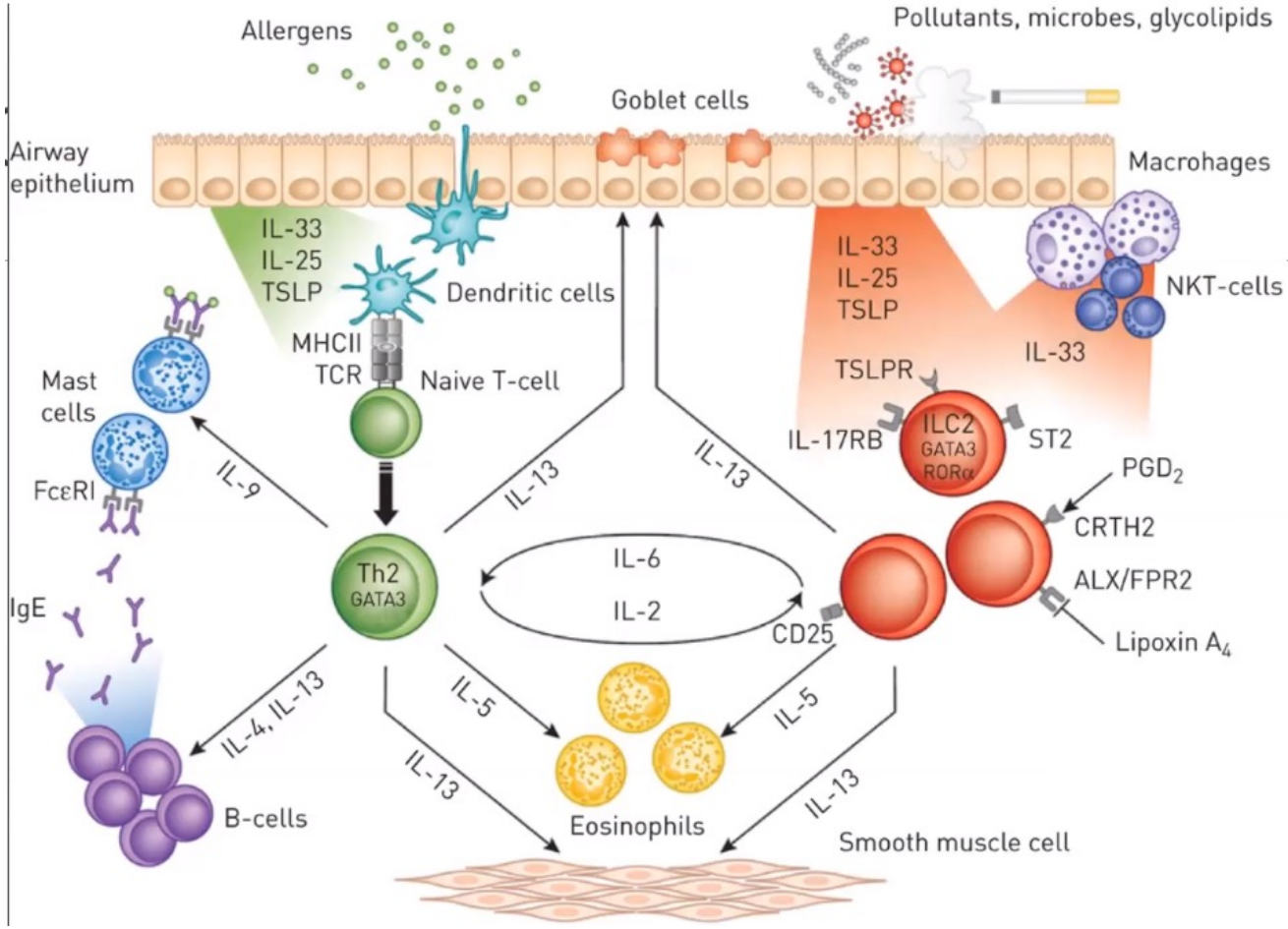

A 30-year-old gentleman is referred to the Severe asthma clinic with recurrent exacerbations. He has had difficult to manage asthma for about 6 years and is currently on high dose inhaled corticosteroid, an inhaled long acting beta-2 agonist, a leukotriene receptor antagonist and oral prednisolone 7.5mg OD. He is an ex-smoker with a 10-pack year history. He has had 4 exacerbations in the past year requiring oral antibiotics and steroids. Recent blood tests show the following: WCC 5.7 Eosinophil 0.2 Plt 255 Hb 135 Total IgE 465, RAST positive to house dust mite, cat dander and tree pollen. Recent spirometry shows a FEV_1 55% predicted, FVC 90% predicted. Which management option is most likely to improve his exacerbation frequency?

a) Benralizumab

b) Increase oral steroids to 10mg OD

c) Mepolizumab

d) Omalizumab

e) Resilizumab

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Eosinophils not high enough for IL5

Which of the following is **not** common side effect of mepolizumab?

a) Headache

b) Fatigue

c) Flu-like symptoms

d) Pruritus

e) Systemic rash

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9. Which Biologic is delivered intravenously?

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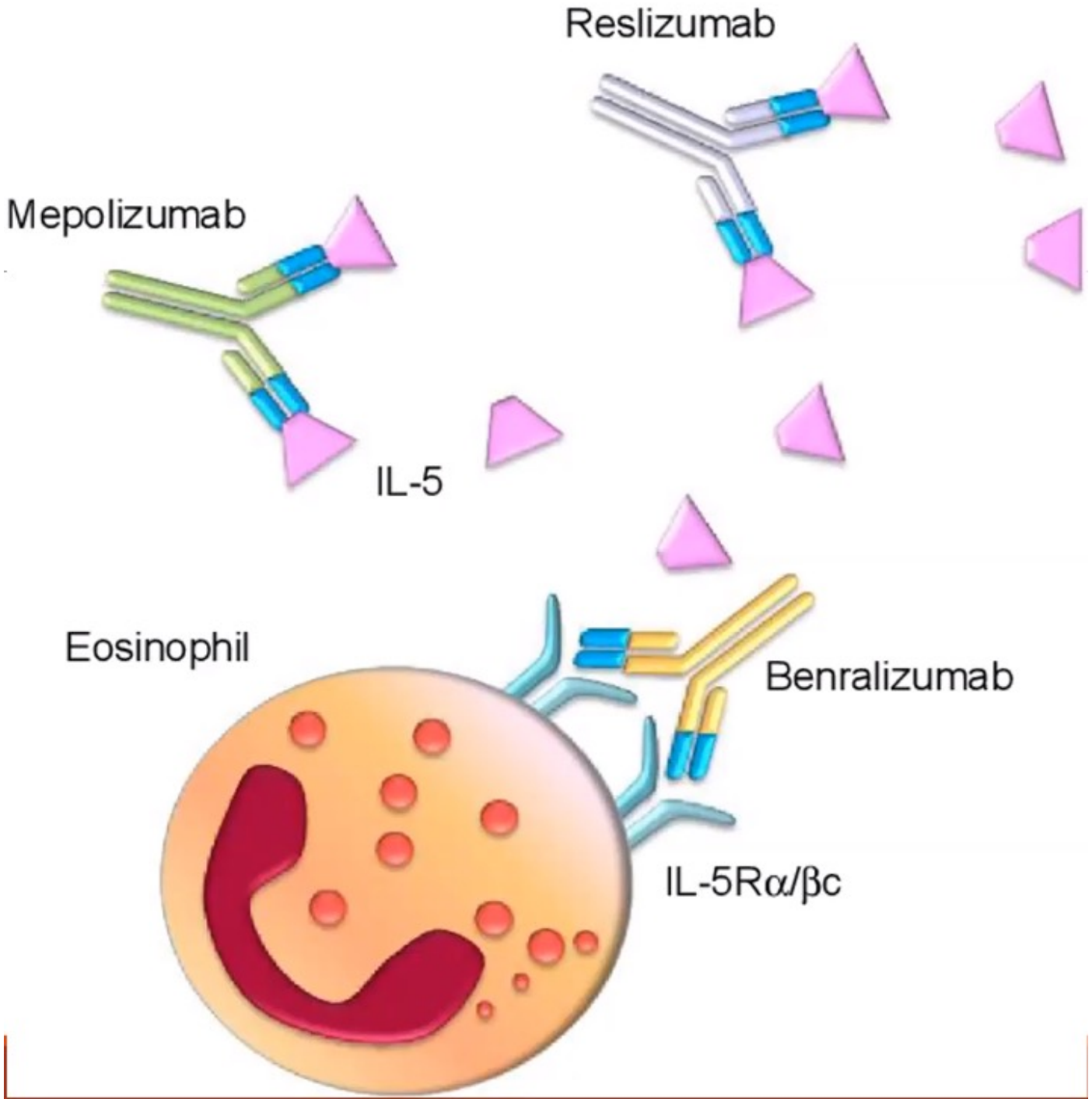
Summary of biologics - NICE criteria

1. Omalizumab: Anti IgE
   a) Severe allergic asthma
   b) Allergic to perianal air allergen (cat dog or pet, house dust mike)
   c) IgE of 30-1200 can be given
   d) But IgE >700 (unlikely to be doseable)
   e) Body weight dose
   f) 4 course of steroids or on maintenance
   g) 16 week trial
2. Mepolizumab: Il-5
   a) Severe eosinophilic asthma
   b) 4 exacerbation and 300 eosinophils
   c) 3 exacerbation and 400 or more eosinophils
   d) 6 month trial (12 m top)
3. Benralizumab Il-5
   a) Eosinophil depleting/eosinophilic apotosis
   b) Dosed every 8 weeks
   c) Severe eosinophilic asthma
   d) 4 exacerbation and 300 eosinophils
   e) 3 exacerbation and 400 or more eosinophils
4. Reslizumab Il-5
   a) Severe eosinophilic asthma
   b) 3 exacerbations
   c) Blood eosinp 400
   d) IV prep on IV prep
5. Dupilumab IL13/Il4
   a) not NICE approval yet!!


Reslizumab
Mepolizumab
IL-5
Eosinophil
Benralizumab
IL-5Rα/βc

Which of the following would cause a raised KCO (more than 1 may apply)?

A. Neuromuscular disease
B. Polycythaemia
C. Emphysema
D. Severe CHF
E. Pulmonary AVMs
F. Anaemia
G. Obesity
H. Inadequate inspiration/poor technique
I. Alveolar haemorrhage

Which of the following would cause a raised KCO (more than 1 may apply)?

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B. Polycythaemia
C. Emphysema (lowers KCO)
D. Severe CHF (lowers KCO)
E. Pulmonary AVMs (lowers KCO pulmonary capillary dilation)
F. Anaemia (lowers KCO)
G. Obesity
H. Inadequate inspiration/poor technique
I. Alveolar haemorrhage

What are the key differences between the SIGN/BTS versus NICE guidelines for asthma?

A. NICE advocates use of SABA alone as a 1st step in ALL patients
   - For adults (aged 17 and over) with asthma who have infrequent, short-lived wheeze and normal lung function, consider treatment with SABA reliever therapy alone

B. NICE favours use of LTRA before ICS/LABA

C. SIGN/BTS favours the use of addition of LAMA when patient still uncontrolled

There are some important differences between the current British Thoracic Society and NICE asthma guidelines. NICE places emphasis on the cost-effectiveness of treatments, and recommend adding a leukotriene receptor antagonist to low-dose ICS as the next step in asthma treatment, in contrast to other guidelines which advocate adding a long-acting beta-agonist as the next step.

What type of hypersensitivity reaction is seen in ABPA?

A. Type I
B. Type II
C. Type III
D. Type IV

What type of hypersensitivity reaction is seen in ABPA?

A. Type I
B. Type II
C. Type III
D. Type IV

	Type I	Type II	Type III	Type IV
Immune reactant	IgE	IgG	IgG	T cells
Antigen	Soluble molecule	Cell associated molecule	Soluble molecule	Soluble or cell associated molecule
Graphic Icon				
Mechanism	IgE induced mast cell activation	Complement mediated phagocytosis	Tissue damage induced by immune complexes	T cell mediated inflammation or cytotoxicity
Examples	Allergic rhinitis, allergic asthma	Chronic urticaria (auto antibodies)	Serum sickness, arthus reaction	Multiple sclerosis, Contact dermatitis, Crohn's disease, Rheumatoid arthritis

Allergy ABPA

HP

但部份并非由 IgE 抗体引起的

Lung Cancer

Surgery for lung cancer

- **A 72 year old smoker (45PY) presents with cough and haemoptysis. Imaging shows a 4cm cavitating lung mass with obstruction of the main bronchus. What is the most likely diagnosis?**

  A. Squamous cell carcinoma

  B. Adenocarcinoma

  C. Large cell neuroendocrine carcinoma

  D. Small cell lung cancer

  E. Carcinoid tumour

Surgery for lung cancer

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A. Squamous cell carcinoma — Large central tumours with cavitation

B. Adenocarcinoma — Adeno – peripheral, small tumours

C. Large cell neuroendocrine carcinoma

D. Small cell lung cancer

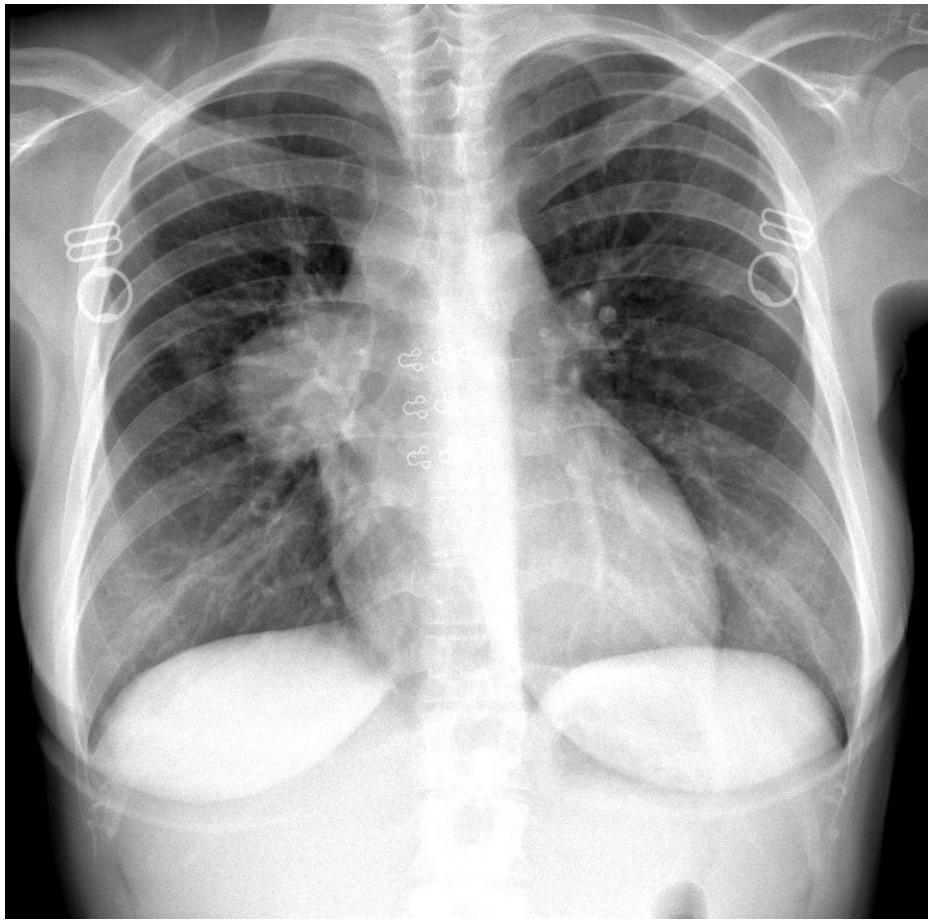
E. Carcinoid tumour

A

Q

What is the most likely cause of this CXR and extra point what the radiological sign?

A. Hodgkin's lymphoma
B. Bronchogenic carcinoma
C. Sarcoidosis
D. Thoracic aortic aneurysm



Ans

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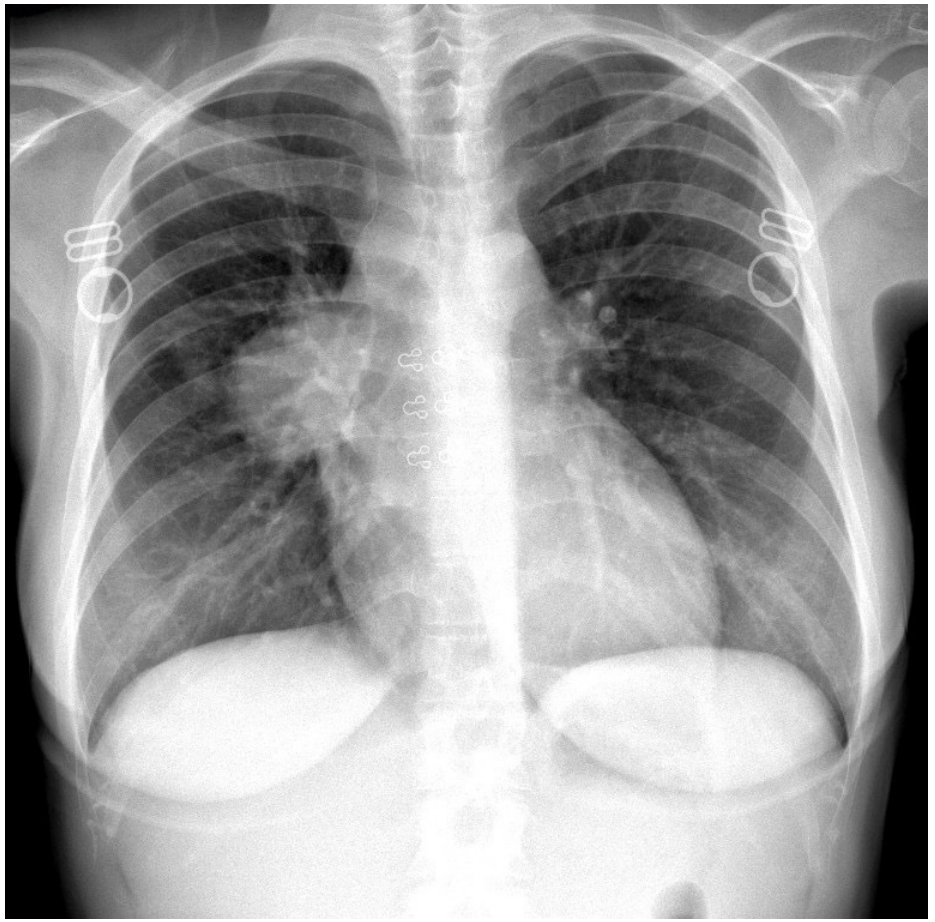


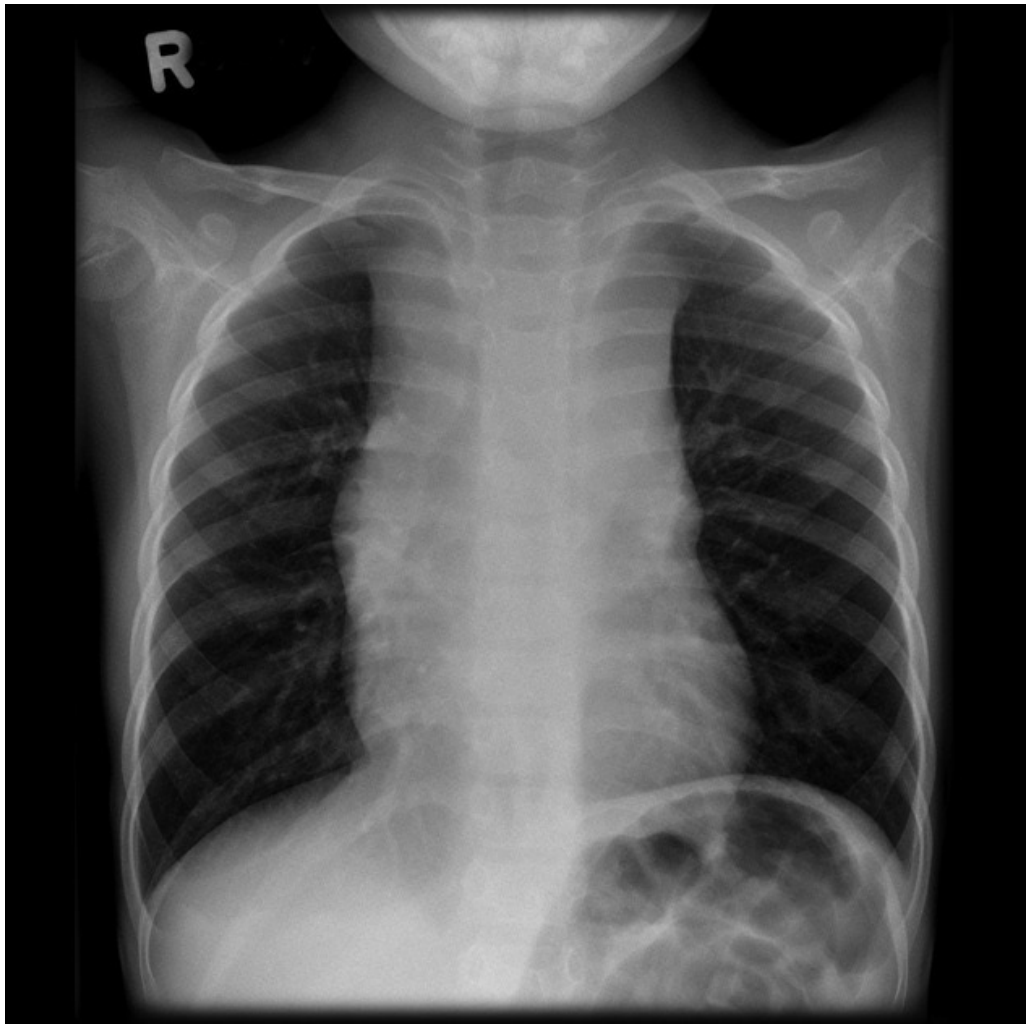
Hilar overlay sign

- The **hilum overlay sign** CXR outline of the hilum can be seen at the level of a mass or collection in the mid chest.
- It implies that the mass is not in the middle mediastinum, and is either from anterior or posterior mediastinum(most of the masses arise from the anterior mediastinum
- The sign refers to preserved visualisation of the hilar vessels, excluding abnormalities that localise to the middle mediastinum

Q

A 50 year old male presents with night sweats, fever, weight loss and has the following CXR. What is the MOST likely cause of this CXR

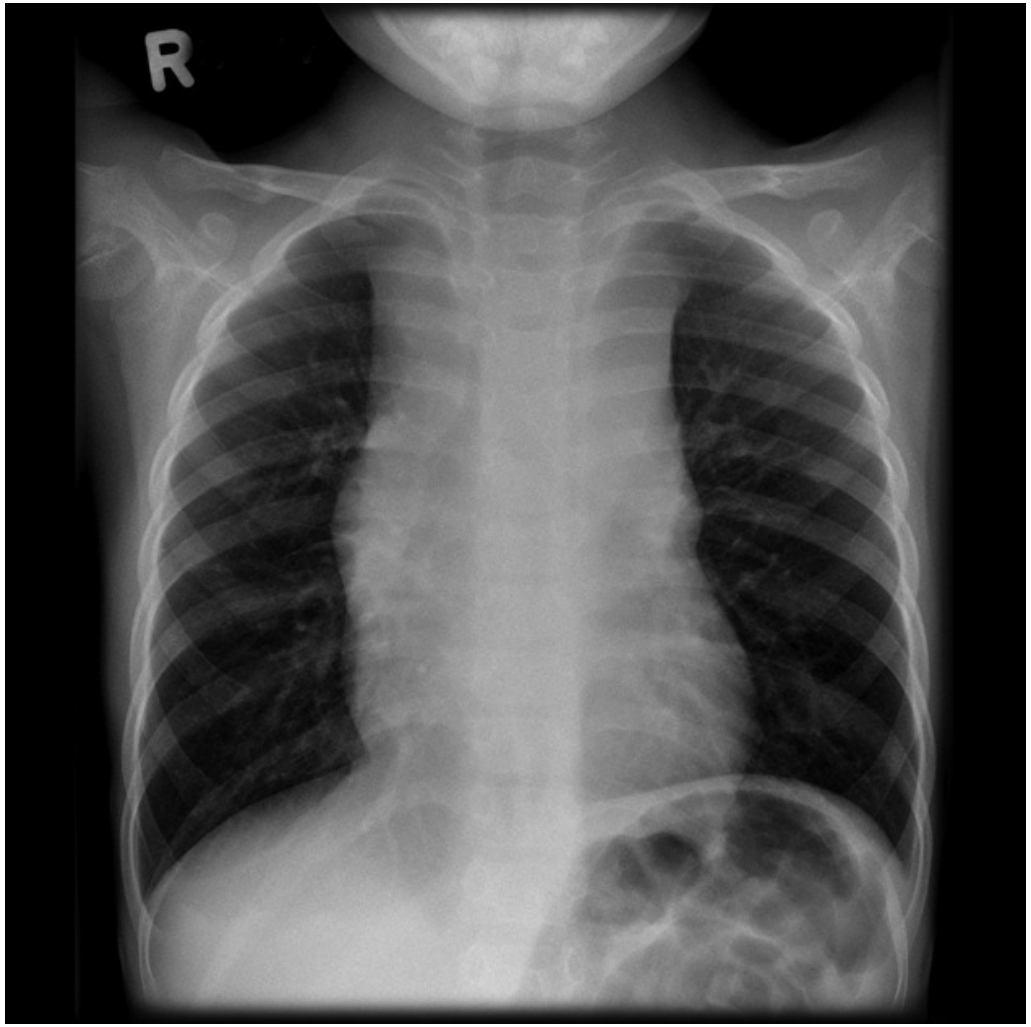
A. Hodgkin's lymphoma
B. Non-hodgkins lymphoma
C. TB
D. Sarcoidsosis
E. Thymoma



Ans

A 55 year old male presents with night sweats, fever, weight loss and has the following CXR. What is the MOST likely cause of this CXR

A. Hodgkin's lymphoma
B. Non-hodgkins lymphoma
C. TB
D. Sarcoidsosis
E. Thymoma



The **majority of non-Hodgkin patients are over the age of 55 when first diagnosed**, whereas the median age for diagnosis of Hodgkin lymphoma is 39. non-Hodgekins 60% Hodgekins 40%

Q

43 year old female presents, history of myasthenia. Patient presents with SVCO with cough for 3 months. Following CT and CXR. What's the diagnosis?

A. Thyroid goitre
B. Thymic hyperplasia
C. Lymphoma
D. Mediastinal germ cell tumour
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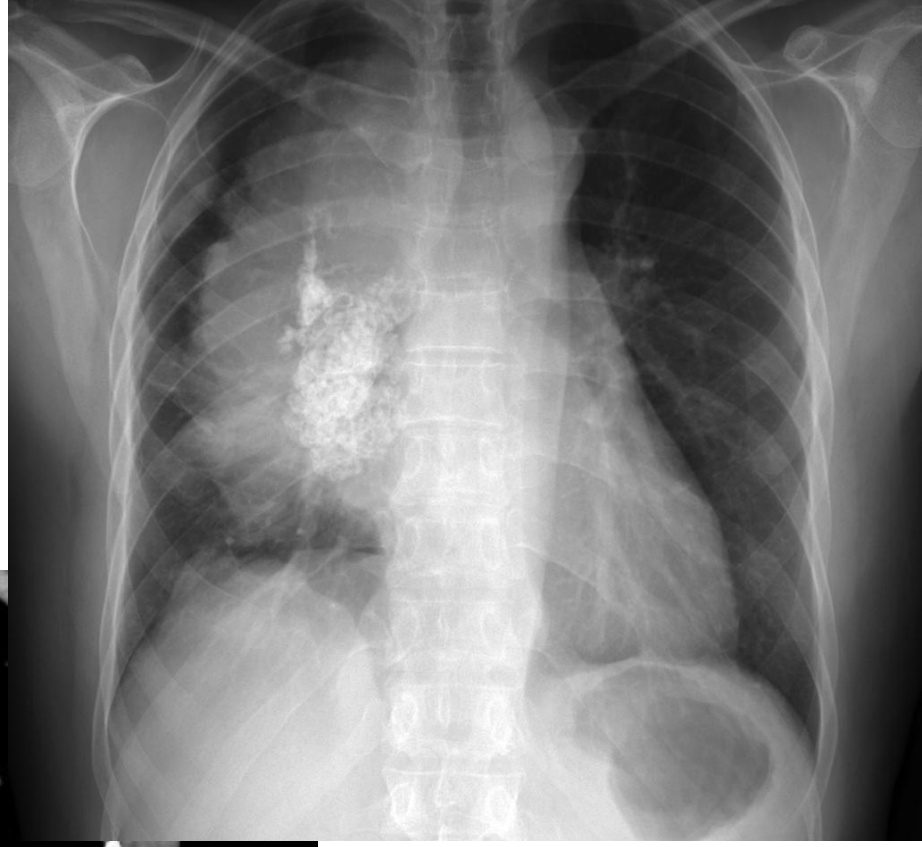

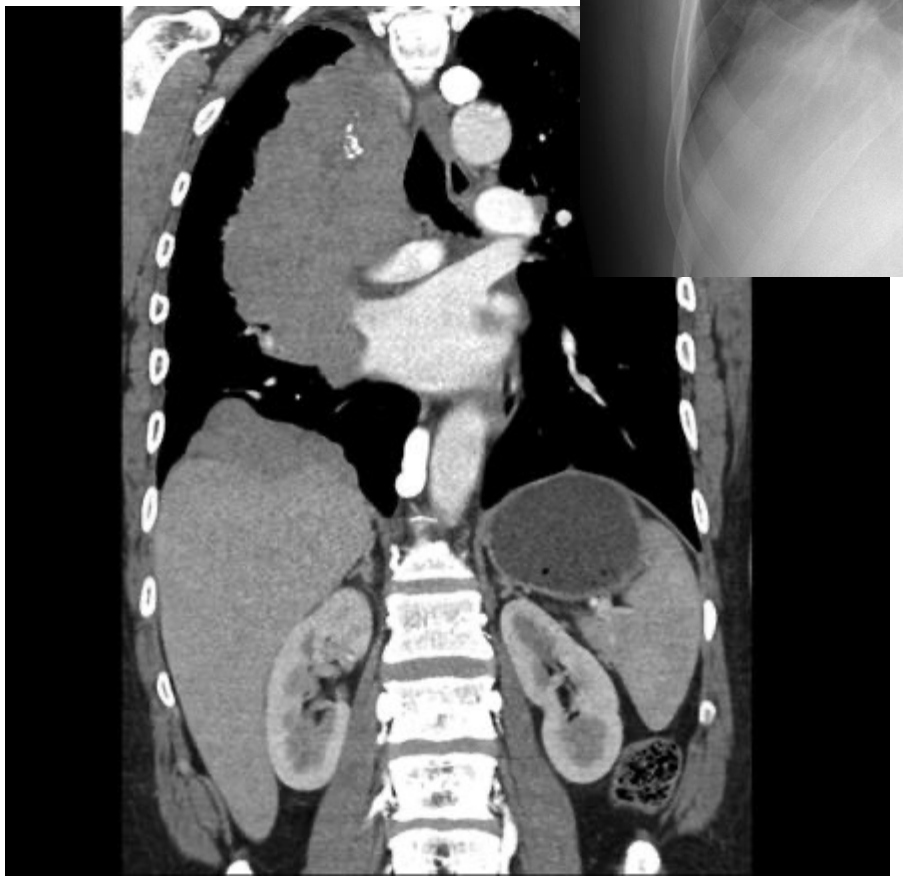

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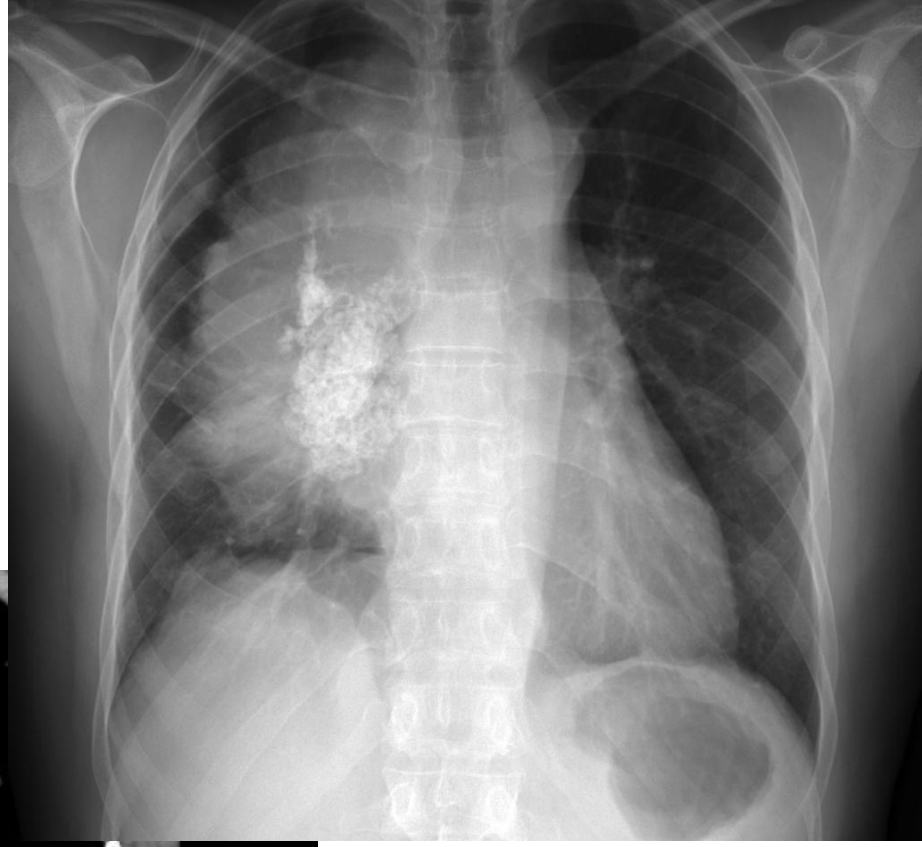
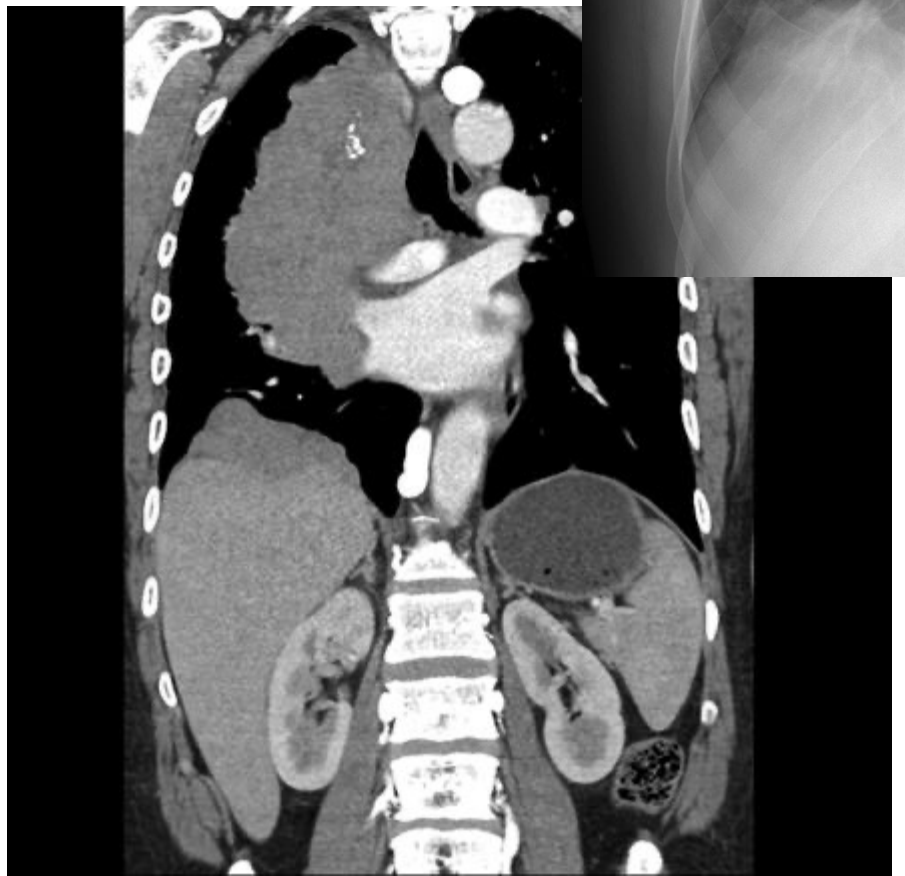




Thymic Epithelial Tumours.

- type A: tumours (medullary histology thymomas), are typically rounded, smooth or somewhat lobulated masses of soft tissue attenuation
- type B: tumours more frequently demonstrate calcification, although calcification is also frequently seen in thymic carcinoma [6]
- type C:
  - tumours (thymic carcinoma) usually demonstrate an invasion of mediastinal fat or mediastinal structures and are usually much larger than type A or B tumours
  - mediastinal lymph node enlargement may be present although the reported frequency of this finding varies widely (13-44%) [6]

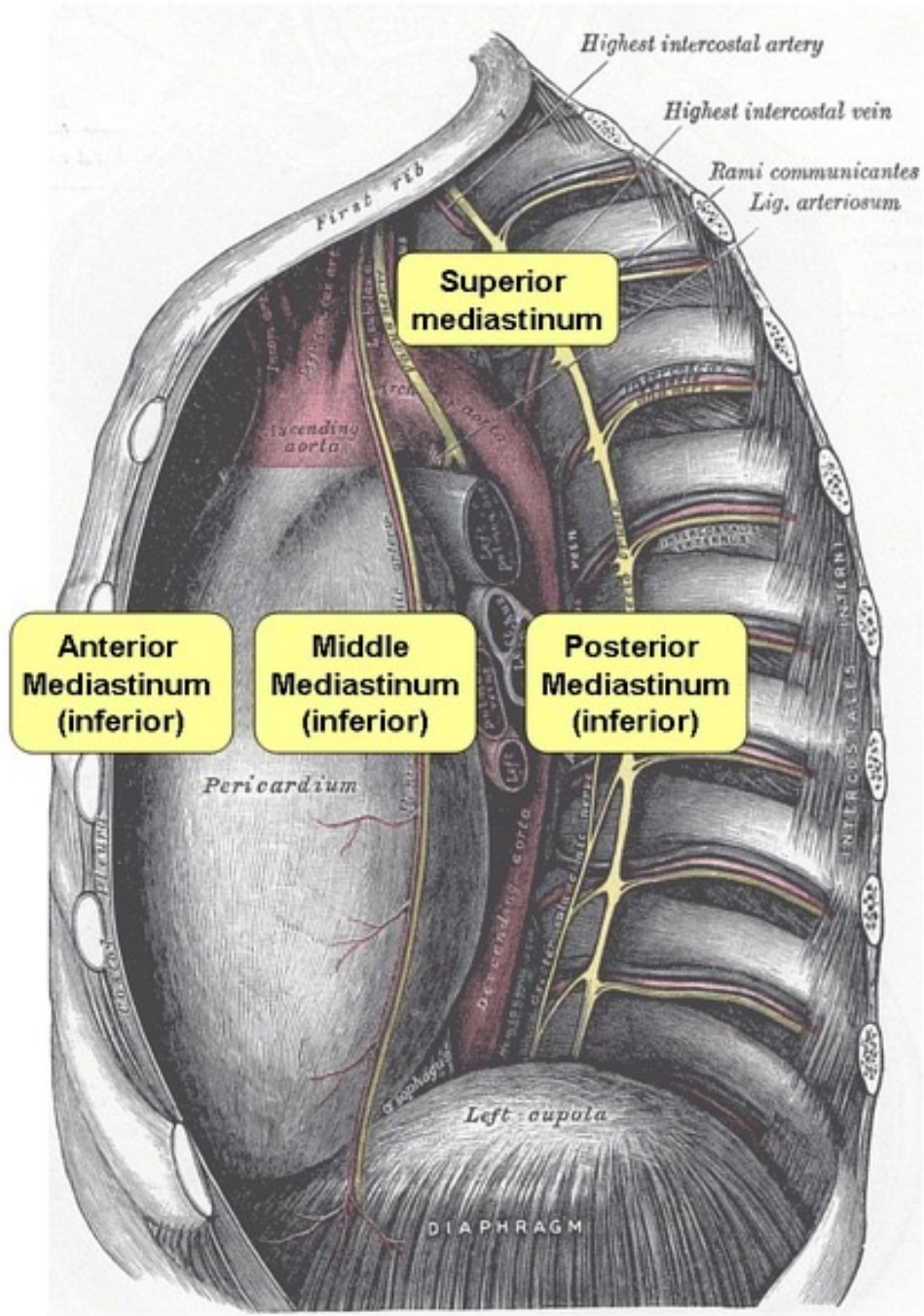
Pleural seeding is seen in invasive thymoma or thymic carcinoma.

Anterior mediastinal masses

- **5 Ts**

Mnemonic

- **T:** thymus
- **T:** thyroid
- **T:** thoracic aorta
- **T:** terrible lymphoma
- **T:** teratoma and germ cell tumours - see mediastinal germ cell tumours


Highest intercostal artery
Highest intercostal vein
Rami communicantes
Lig. arteriosum
First rib
Superior mediastinum
Anterior Mediastinum (inferior)
Middle Mediastinum (inferior)
Posterior Mediastinum (inferior)
Pericardium
Left cupola
DIAPHRAGM

Q

- A 55year old is brought to A&E following a RTA . He undergoes trauma CT scan which picks up a 5mm nodule sub solid nodule in the right upper lobe. He has 5 pyh of smoking. How will you manage him?

- 1) Follow up scan in 3 months
- 2) Follow up scan in 12 months
- 3) PET CT
- 4) Brocks score
- 5) Discharge

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Q

- A 40 year old has a CT scan of abdomen for ?? Pancreatitis. This scan picks up left lower lobe subpleural triangular opacity measuring of 7mm. Next step
- 1) Brocks score
- 2) CT scan in 12 months
- 3) CT biopsy
- 4) Discharge
- 5) CT scan in 3 months

Q

- A 40 year old has a CT scan of abdomen for ?? Pancreatitis. This scan picks up left lower lobe subpleural triangular opacity measuring of 7mm. Next step
- 1) Brocks score
- 2) CT scan in 12 months
- 3) CT biopsy
- 4) Discharge
- 5) CT scan in 3 months

Q

- What is the mode of action of Nivolimumab?
- 1) PDL1
- 2) PD1
- 3) EGFR INHIBITOR
- 4) ALK inhibitor
- 5) ROS 1 Inhibitor

Ans

- PDL1: Atezolizumab
- PD1 : Pembrolizuman, nivolimumab

- Tumor markers:
- Squamous cell carcinoma : P63, P42,
- Adeno carcinoma: TTF1, Napsin, BPR 4,
- Neuroendocrine : Synaptophysin, chromogranin A
- Mesothelioma: Wilms tumor,CK5/6, Calretinin

Q

You see a 55 year old in RALC with new right upper lobe mass measuring 3cm and enlarged right hilar and subcarinal node along with atelectasis and right sided pleural effusion. Stage?

- 1) T2N1M0
- 2) T2N2M1A
- 3) T3N2M0
- 4) T1N1M1A
- 5) T4N2M1A

Ans

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- 4) T1N1M1A
- 5) T4N2M1A

Q

What size of tumour should make u consider post operative adjuvant chemotherapy?

1. 2cm
2. 3cm
3. 4cm
4. 5cm
5. 6cm

Ans

What size of tumour should make u consider post operative adjuvant chemotherapy?

1. 2cm
2. 3cm
3. 4cm
4. 5cm
5. 6cm

- Consider postoperative chemotherapy for people with good performance status (WHO 0 or 1) and T2b–4 N1-2, M0 NSCLC or with tumours greater than 4 cm in diameter.SO either if nodal disease present or a large tumour

Q

A 65-year-old lady with squamous cell carcinoma is due to undergo a curative right lower lobe lobectomy.

She has a preoperative FEV1 of 1.9 L.

What is her predicted postoperative FEV1?

A. 1.0 L
B. 1.2 L
C. 1.4 L
D. 1.5 L
E. 1.6 L

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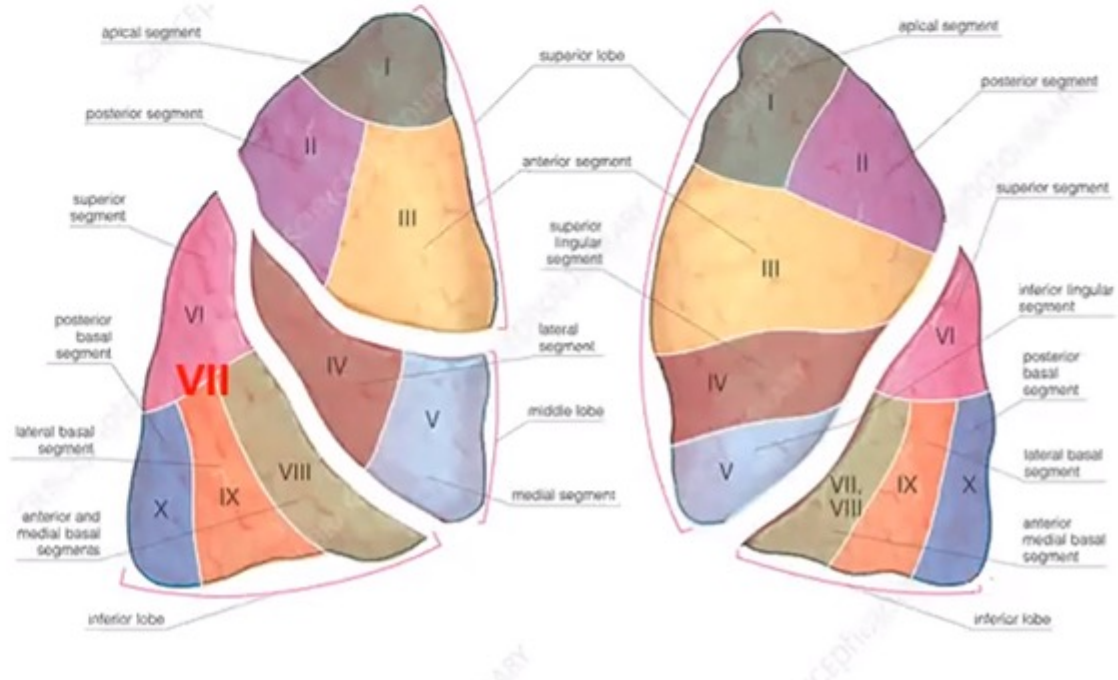
E. 1.6 L

Predicted Post-Operative Lung Function

ppoValue = Pre-Op Value/**T**otal X **R**esidual

- Pre-Op FEV1 and/or DLCO
- **T**otal Segments = 19 – "Obstructed"
  - i.e. Don't include non-functioning segments
- **R** = Residual Segments left behind post-op
  - i.e. T- Functioning segments to be resected

- The number of segments to be resected is:
  - RUL 3
  - RML 2
  - RLL 5
  - LUL 5 (3 upper division, 2 lingula)
  - LLL 4



Lung cancer Qs

Q1

- 56 year old male presented with confusion and constipation to the acute medical take. He has an established but recent diagnosis of T3N2M1b lung cancer. His bloods show a raised CRP 56, Hb 110, WBC 5.4, Corrected Ca is 3.40mmol/L, PTH-related protein (PTHrH) is raised. What cell type is the most likely cause of his presentation?

1. Small cell carcinoma with adrenal gland mets
2. Adenocarcinoma cell carcinoma with bony mets
3. Adenocarcinoma
4. Small cell carcinoma with bone metastasis
5. Squamous cell carcinoma

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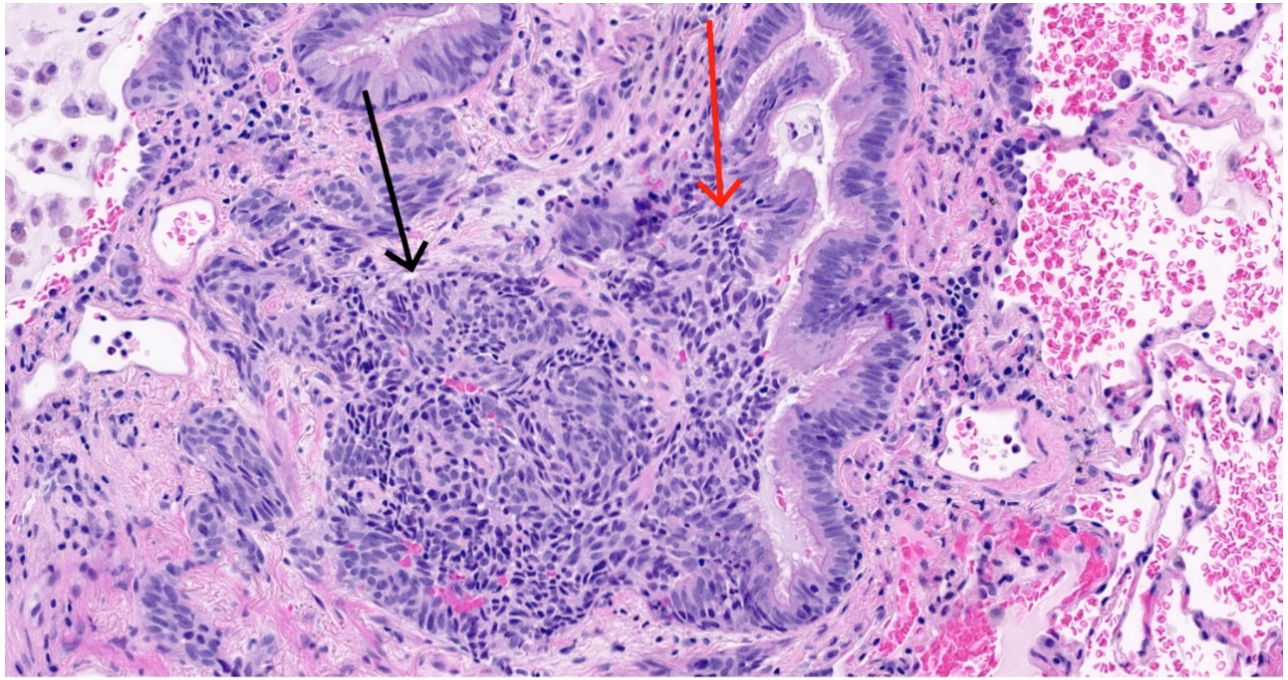
Hypercalcaemic and Lung ca

- Hypercalcaemia and elevated parathyroid hormone related peptide levels are most commonly associated with squamous cell carcinoma.
- Small cell carcinoma and cancers with bone metastases may also be associated with hypercalcaemia but not with increased PTHrH levels.
- Sr Ca over 2.75 is abnormal, a serum level over 3.25mmol/L is rare outside malignancy (exception of sarcoidosis)
- In malignancy raised Ca is due to increased osteoclast activity which can be from bony mets or production of PTH-related prtoein

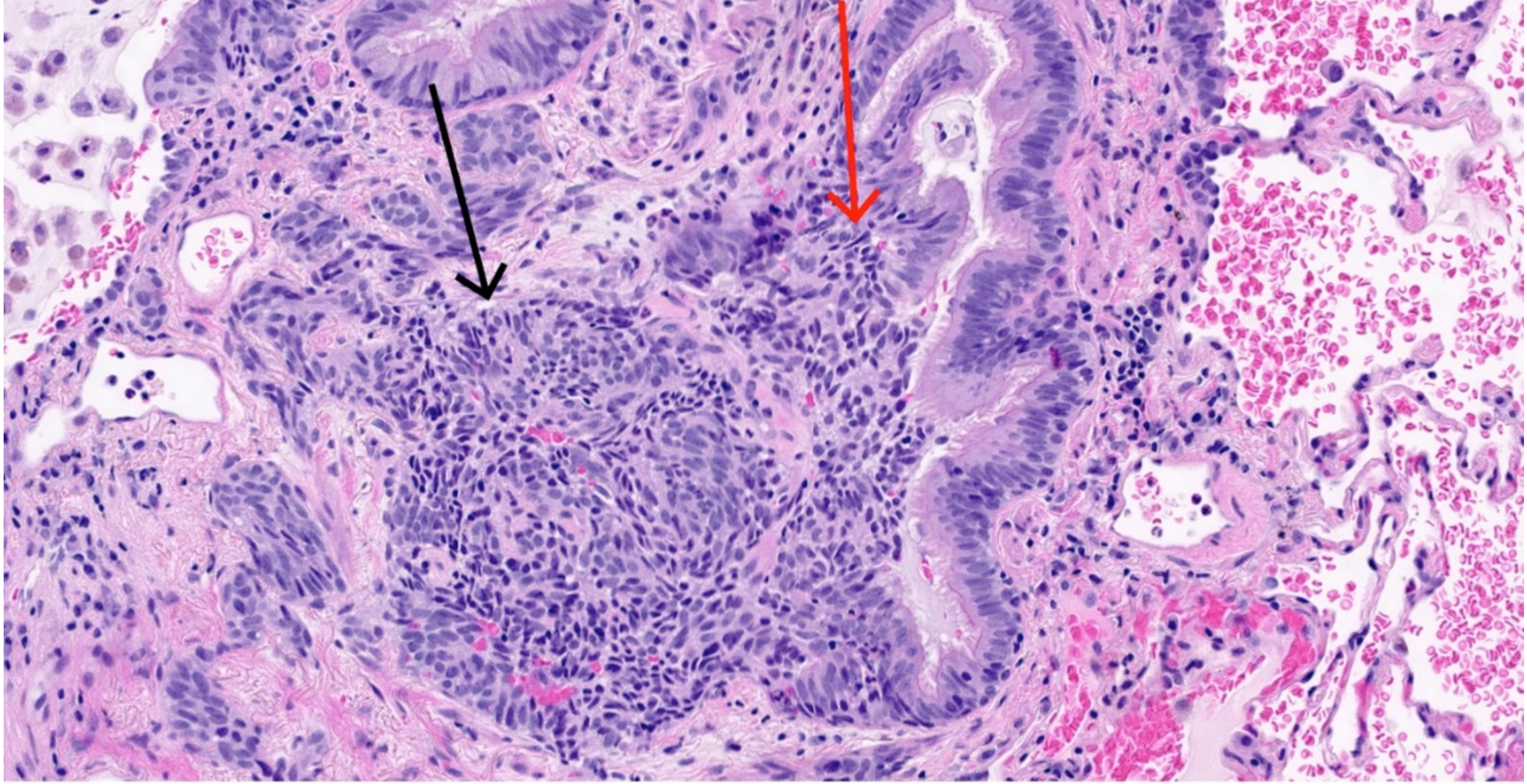
Q2

A 44 year old women has recently been diagnosed with a typical carcinoid tumour following a excision and histology which demonstrated salt and pepper chromatin. She comes to see u in a clinic and asked for information about survival. What is her 5 year survival based on the information above?

1. 10%
2. 20%
3. 60%
4. 70%
5. 90%



Proliferating neuroendocrine cells in the bronchial mucosa (red arrow) that cross beyond the mucosal basal lamina to form a tumorlet (black arrow) (H&E; 200X)



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Carcinoid/Neuroendocrine Tumour

Carcinoid type	Typical carcinoid	Atypical Carcinoid	Large cell NET	Small cell SCLE
5 year survival	90%	60%	20%	10%
Features		Neuroendocrine tumour with 2-10 mitoses/2mm2 or presence of necrosis	>10 mioses/ 2mm2, extensive and geographical necrosis. Large cells.	Round/oval blue bells with minimal cytoplasma, usually small-medicum sixes

- Lymph node metastasis:
  - 9% of typical carcinoids
  - 36% of atypical carcinoids

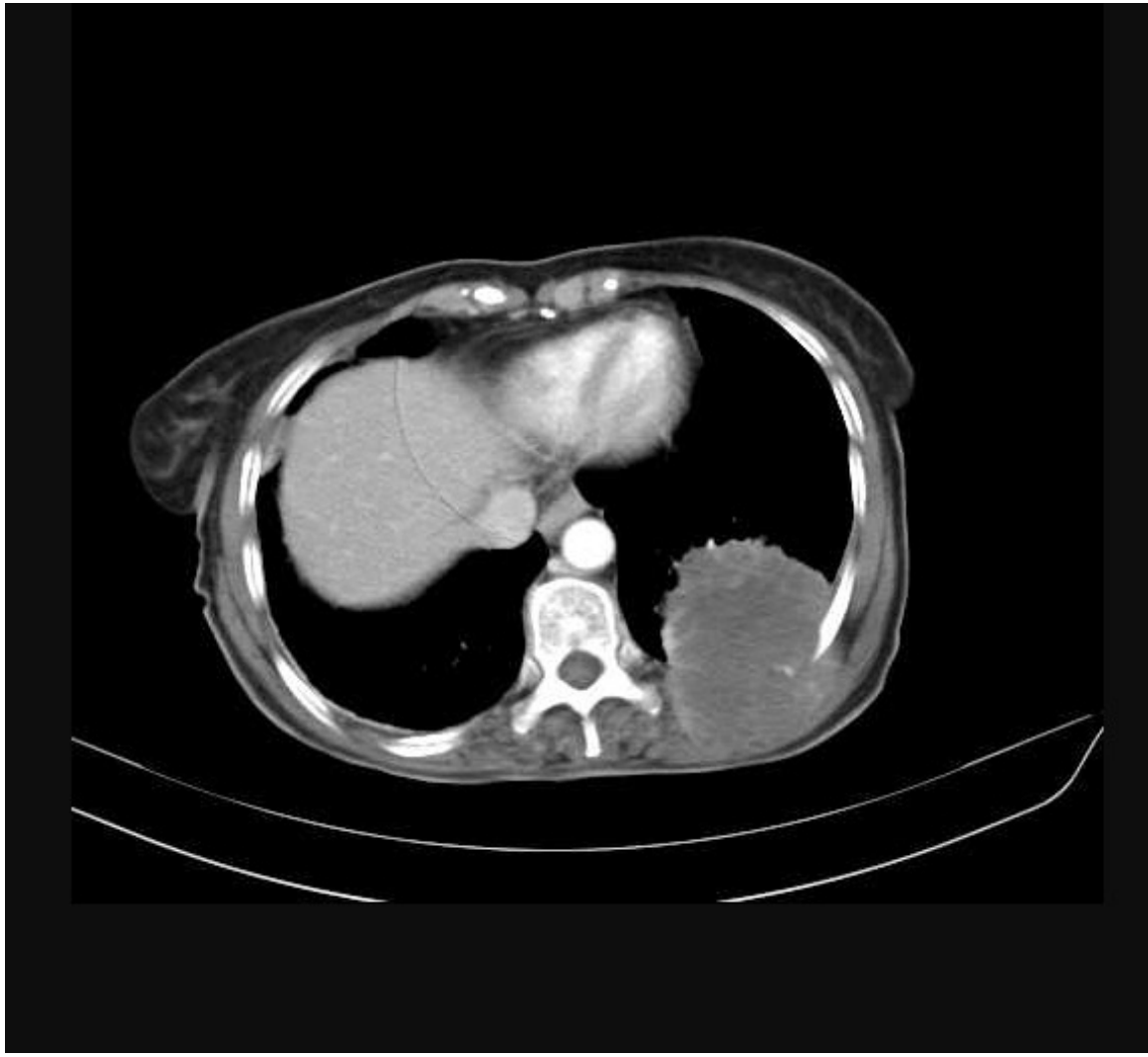
- Metastatic disease at time of diagnosis: 13%
  Large cell NET 40%
- Salt & pepper chromatin

5 year survival (Arch Pathol Lab Med 2010;134:1628)

Q3

A 76 year old PS 1 male, with a smoking history of 50 PYs presents with pleuritic chest pain and the following CT scan is completed showing a 6.5cm lesion. EBUS demonstrated hilar and intrapulmonary lymph nodes only to be positive. No distant metastasis are seen. What would the most appropriate staging be?

1. **T3N2M0**
2. **T4N2M0**
3. **T3N1M0**
4. **T4N1M0**
5. **T2N2M1a**



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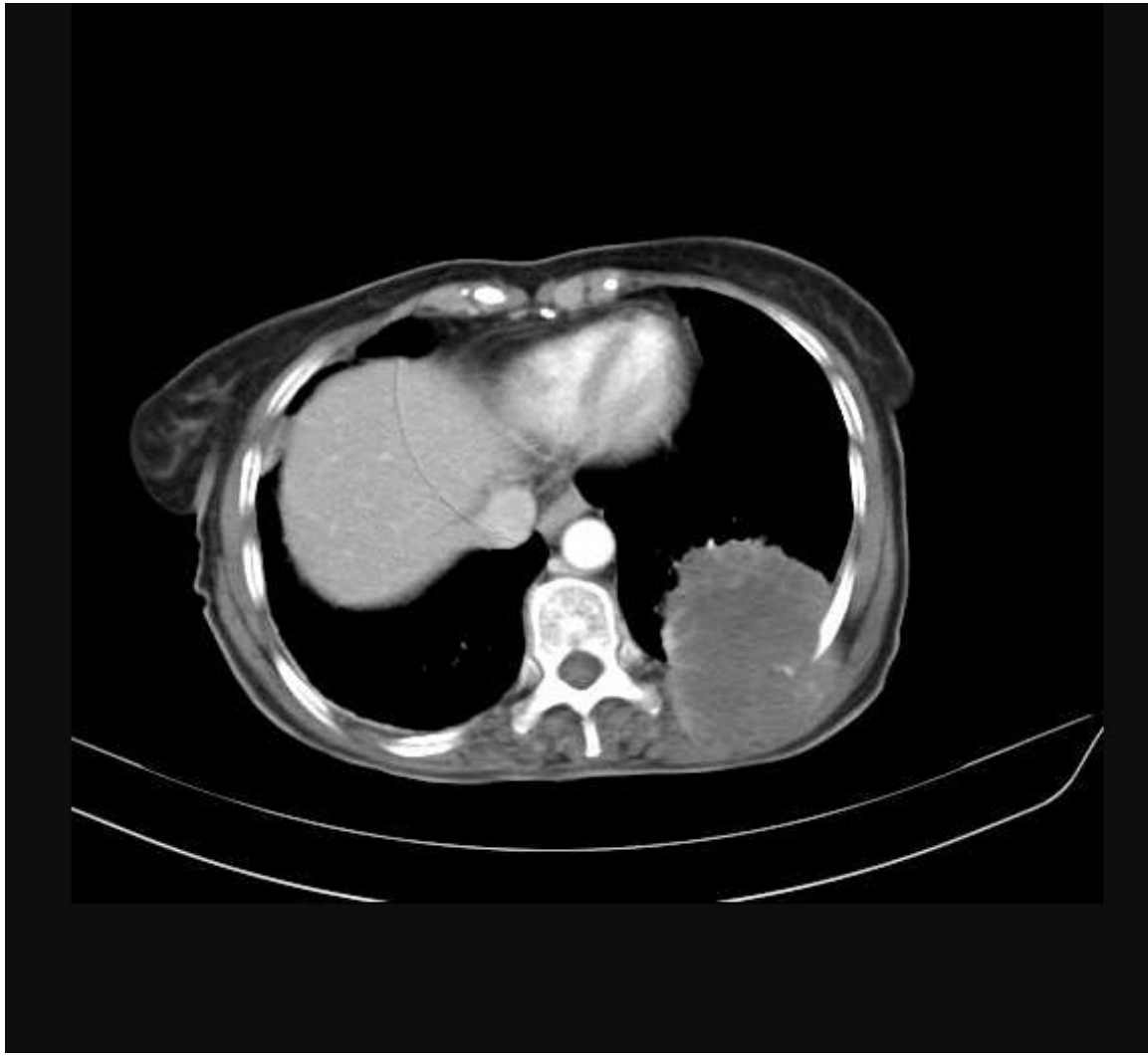
3. **T3N1M0**
4. **T4N1M0**
5. **T2N2M1a**



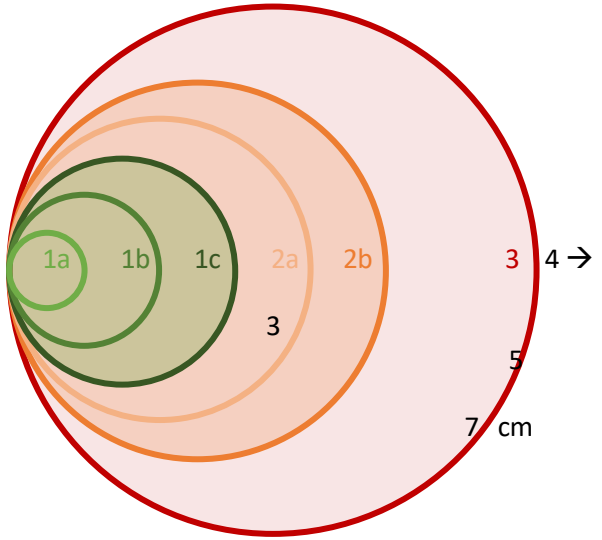
Hilar = N1
Mediastinal LN = N2

TNM 8th - Primary tumor characteristics

T_x	Tumor in sputum/bronchial washings but not be assessed in imaging or bronchoscopy
T_0	No evidence of tumor
T_{is}	Carcinoma in situ
T_1	≤ 3 cm surrounded by lung/visceral pleura, not involving main bronchus
$T_{1a(mi)}$	Minimally invasive carcinoma
T_{1a}	≤ 1 cm
T_{1b}	> 1 to ≤ 2 cm
T_{1c}	> 2 to ≤ 3 cm
T_2	> 3 to ≤ 5 cm *or* involvement of main bronchus without carina, regardless of distance from carina or invasion visceral pleural *or* atelectasis or post obstructive pneumonitis extending to hilum
T_{2a}	>3 to ≤4cm
T_{2b}	>4 to ≤5cm
T_3	>5 to ≤7cm in greatest dimension *or* tumor of any size that involves chest wall, pericardium, phrenic nerve *or* satellite nodules in the same lobe
T_4	> 7cm in greatest dimension *or* any tumor with invasion of mediastinum, diaphragm, heart, great vessels, recurrent laryngeal nerve, carina, trachea, oesophagus, spine *or* separate tumor in different lobe of ipsilateral lung
N_1	Ipsilateral peribronchial and/or hilar nodes and intrapulmonary nodes
2	Ipsilateral mediastinal and/or subcarinal nodes
3	Contralateral mediastinal or hilar; ipsilateral/contralateral scalene/ supraclavicular
M_1	Distant metastasis
M_{1a}	Tumor in contralateral lung or pleural/pericardial nodule/malignant effusion
M_{1b}	Single extrathoracic metastasis, including single non-regional lymphnode
M_{1c}	Multiple extrathoracic metastases in one or more organs

Prefix	
c	Clinical
p	Pathologic
y	Restaging
r	Recurrence
a	Autopsy
Symbol	Post-op
R0	No residual
R1	Microscopic residual
R2	Gross residual

Chest
2017 http://www.ncbi.nlm.nih.gov/pubmed/27780786



Q4

A 54 year old female of South Korean decent who has never smoked is diagnosed with a T1N0M0 lung cancer. Tumours markers are sent. Which is most like to be true for this patient?

1. EGFR mutations are more common in lung cancers affecting females, never smokers, and those of Asian-Pacific descent

2. This cancer is most likely a small cell tumour.

2. EGFR mutations are more common in lung cancers affecting male, smokers, and those of African descent

3. This lung cancer is most likely a carcinoid tumour

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2. This cancer is most likely a small cell tumour.

2. EGFR mutations are more common in lung cancers affecting male, smokers, and those of African descent

3. This lung cancer is most likely a carcinoid tumour

Tumour markers,

- EGFR mutations are more common in lung cancers affecting females, never smokers, and those of Asian-Pacific descent.
- They are predominantly present in non-small cell cancers with adenocarcinoma differentiation

Q5

- What is the first line systemic anti-cancer therapy SACT for *non squamous* advanced NSCLC with >50% PDL1 expression and no gene mutation or fusion protein?

1. Crizotinib
2. Osimertinib
3. Pemetrexed/Carboplatin
4. Pembrolizumab
5. Pembrolizumab and Pemetrexed

Q5

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4. Pembrolizumab
5. Pembrolizumab and Pemetrexed

EGFR-TK mutation	1. **Osimertinib** / Gefitinib / Erlotinib / Afatinib 2. Progression with EGFR T790M mutation: Osimertinib 3. Progression → CHEMO: Pemetrexed/Carboplatin 4. Progression: atezolizumab, nivolumab, pembrolizumab and nintedanib with docetaxel or docetaxel monotherapy
ALK gene rearrangement	ALK = anaplastic lymphoma kinase 1. **Crizotinib**, Ceritinib, Alectinib 2. Progression → CHEMO: Pemetrexed/Carboplatin 4. as above
ROS1 positive	1. **Crizotinib** 2. Pemetrexed/Carboplatin 3. Progression: atezolizumab, nivolumab, pembrolizumab and nintedanib with docetaxel or docetaxel monotherapy
PDL1 ≥50% and no gene mutation or fusion protein	1. **Pembrolizumab** +/- combo 2. Pemetrexed/Carboplatin 3. Nintedanib/Docetaxel
PDL1 <50% and no mutation/fusion protein/biomarker	1. Pembrolizumab + Pemetrexed/Cis- or carboplatin 2. Progression: atezolizumab, nivolumab, pembrolizumab and nintedanib with docetaxel or docetaxel monotherapy