Radiology of Asbestos Related Lung Disease

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Glasgow


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Radiology of Asbestos Related Lung Disease

- General term given to a group of fibrous minerals containing silica and a variety of other elements.
- Asbestos: Derived for the Greek word meaning *inextinguishable*.
- Desirable physical properties: sound absorption, resistance to heat, fire, chemical and electrical damage, as well as tensile strength, durability and flexibility.
- Affordable. Therefore with industrialisation in the 20th century, its use rose exponentially.

Radiology of Asbestos Related Lung Disease

- Asbestos production:

  1900 – 500 tons/year

  1981 – 6 million tons/year

  2009 – 2 million tons/year

- Potential for serious harm (and occupational exposure litigation) will remain well into this century:

  Vast tonnages in use.

  Long lag time between exposure and development of adverse effects.


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Radiology of Asbestos Related Lung Disease

- Knowledge of toxicity:

  Pliny the Younger (AD 61-114) “Slaves that worked with mineral asbestos became ill”.

  1924 – first diagnosed case of asbestosis. Published in the BMJ. Nellie Kershaw died from lung fibrosis which was attributed to “occupational exposure to asbestos.”

  (At the inquest, Turner Brothers Asbestos, accepted no liability, paid no compensation and refused to contribute to the funeral expenses as it “would create a precedent and admit liability.”)

  1970s – court documents proved that asbestos industry officials knew about the dangers of asbestos since the 1930s and had concealed them from the public.

Radiology of Asbestos Related Lung Disease

Sources of asbestos exposure:

1) Primary occupations of asbestos mining and its processing in a mill.

2) Numerous secondary occupations, notably shipbuilding in Glasgow.

3) Non occupational or environmental exposure. Post mortem studies have shown great variability in the prevalence of asbestos bodies (~1% in rural Italy, 25-50% in Glasgow).

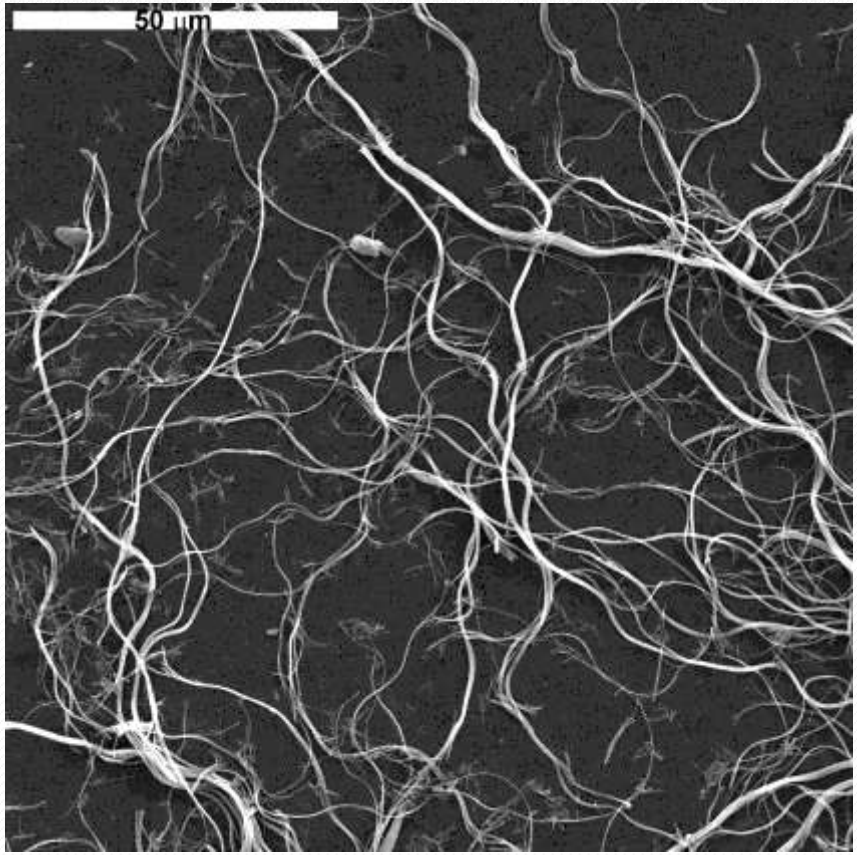
The presence of asbestos bodies in PM studies is not the same as pleuropulmonary disease. But, people with modest exposure can develop pleural plaques and mesothelioma.

Types of Asbestos

- **Serpentines:**

  Chrysotile (white asbestos)

  Curved fibres. More readily fragment and dissolve. Slightly less nasty.



Types of Asbestos

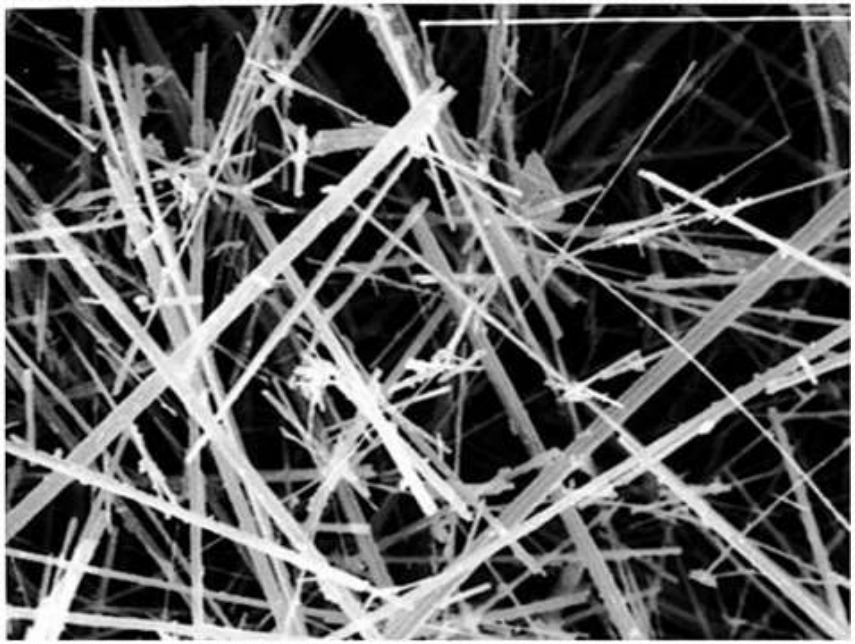
- **Amphiboles:**

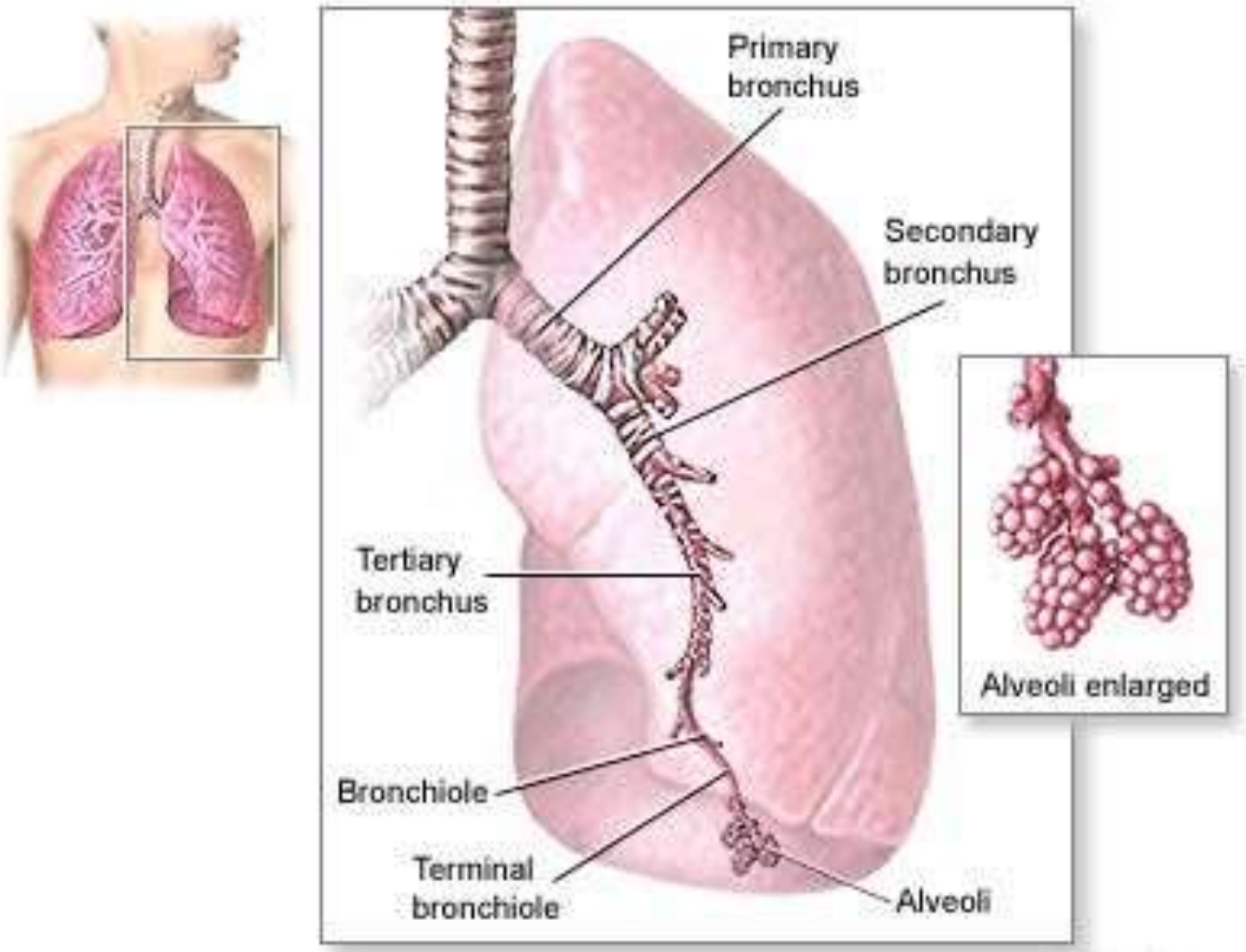
  Amosite (brown asbestos)

  Crocidolite (blue asbestos)

  Tremolite

  Straight, thin fibres. More durable. Nastier. More fibrogenic and carcinogenic.



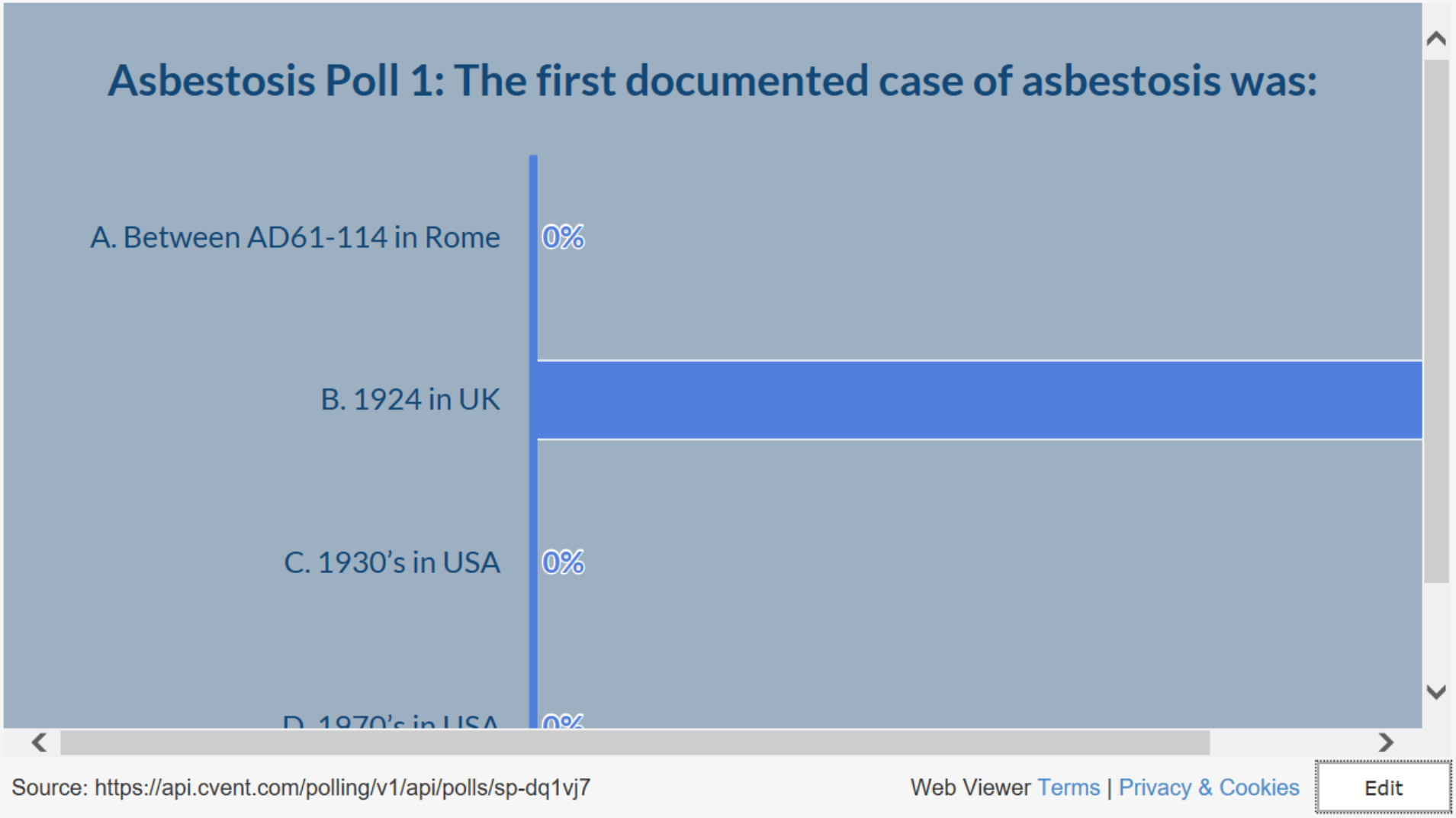

Primary bronchus
Secondary bronchus
Tertiary bronchus
Bronchiole
Terminal bronchiole
Alveoli
Alveoli enlarged

Toxicity and Pathogenesis

- Toxicity is related to fibrous structure (pulverised asbestos is harmless).
- Shape and dimension of fibres (longer, thinner more durable fibres are the most biologically important).
- Exposure dose.
- Host pulmonary clearance.
- Immunological status.
- Presence of other noxious substances, especially cigarette smoke.

Toxicity and Pathogenesis

- Most inhaled fibres are cleared by the tracheobronchial mucociliary escalator.
- Depending on exposure dose and efficiency of fibre clearance, a variable portion enters the interstitium, where, if numerous enough, activate inflammatory processes which lead to fibrosis.
- Host factors, rather than cummulative dose, appear to be more important in determining the total number of fibres reaching the pleura, which may explain why plaques and mesothelioma can be associated low, or intermittent exposure.
- Host factors also appear to play an important role in determining the type of reaction that occurs in the pleura. Animal model: a paucity of macrophages led to an effusion and diffuse pleural thickening, whilst an abundance led to parietal pleural plaques.



Source: https://api.cvent.com/polling/v1/api/polls/sp-dq1vj7

Radiology of Asbestos related Lung Disease

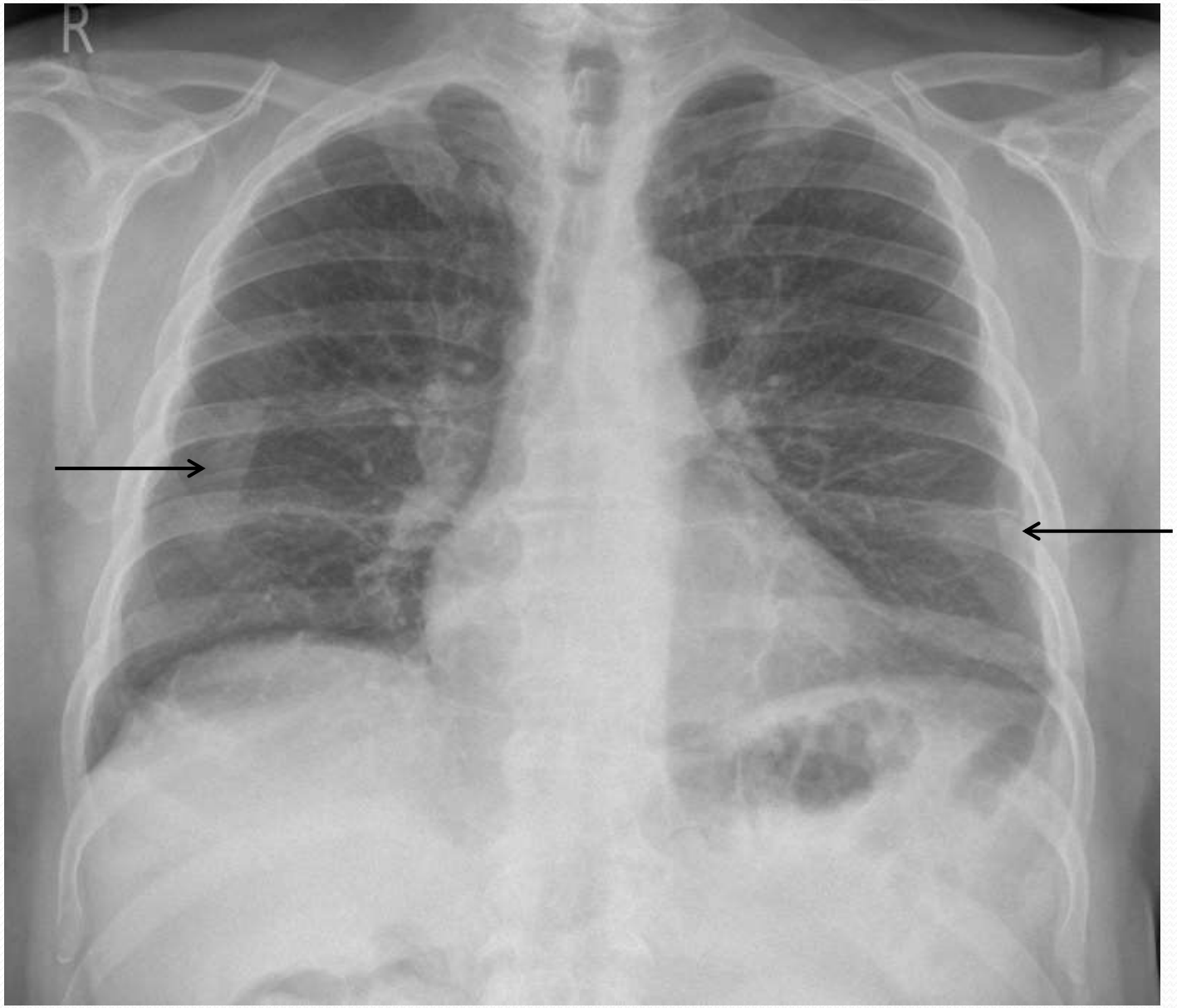
- **Pleural:**
  Plaques
  Benign asbestos pleural effusion
  Diffuse pleural thickening
  Focal visceral pleural fibrosis
- **Lung Parenchymal:**
  Rounded atelectasis
  Asbestosis
- **Neoplastic:**
  Bronchial carcinoma
  Mesothelioma

Pleural Plaques

- Most common radiographic manifestation of asbestos exposure
- Incidence does increase with exposure, but may be seen with minimal exposure.
- Elapsed time from initial exposure is the most important factor. Usually first identified more than 20-30 years after the initial exposure.
- Composed of focal areas of hyalinised fibrous tissue which frequently enlarge and calcify with age. Characteristically located on the parietal pleura, but can occur on the visceral surface (interlobar fissures).

Pleural Plaques

- Usually located adjacent to rigid structures: the tendinous portion of the diaphragms, paravertebral regions and underlying the ribs, *but* with sparing of the apices and costophrenic recesses.
- Usually bilateral
- When unilateral ? Left sided predominance.
- CXR: smooth elevations of pleura, most easily seen in profile. When calcified, they appear linear in profile. En-face, they have a variable appearance but are classically described as “holly leaf”.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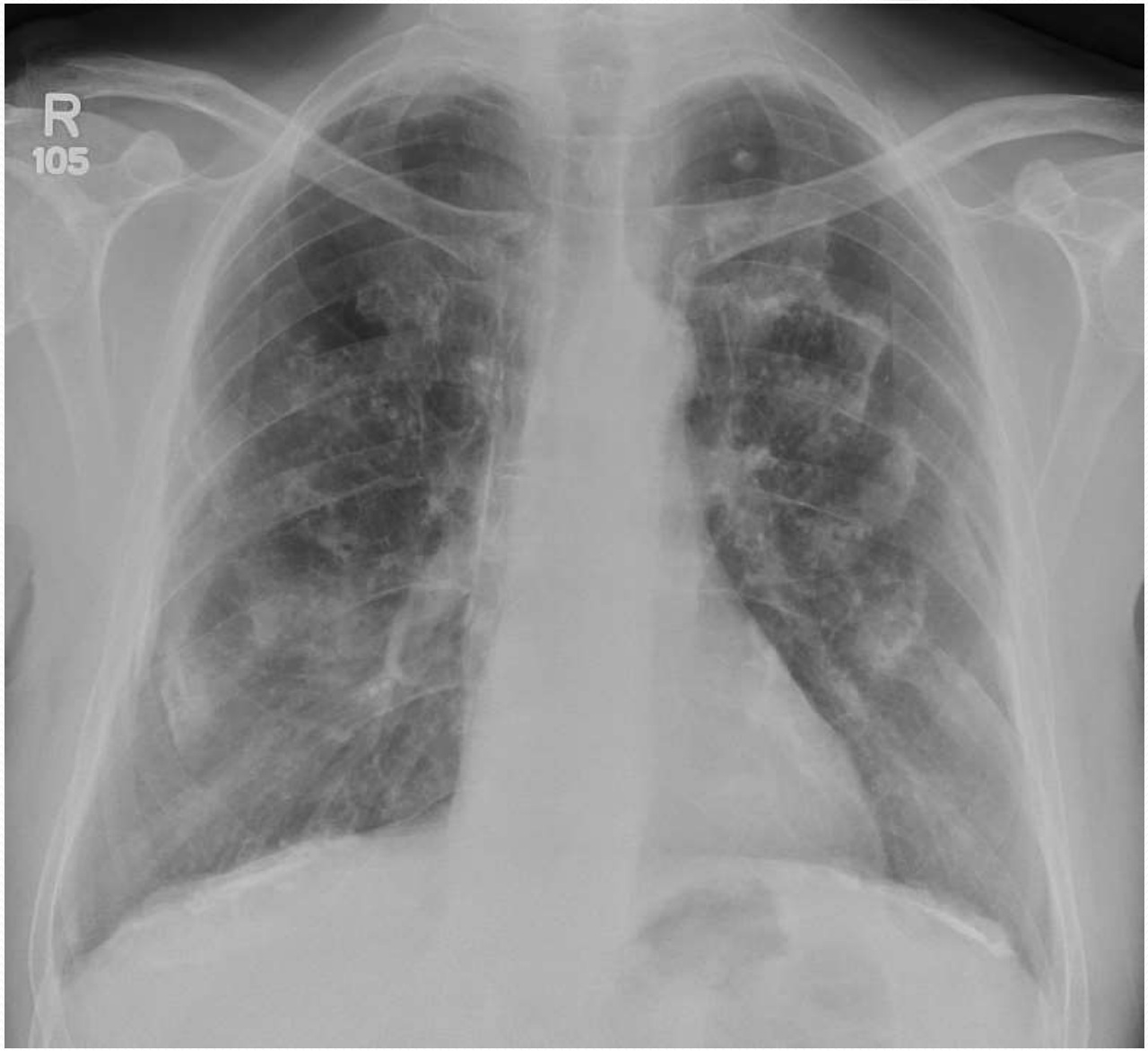
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Pleural Plaques

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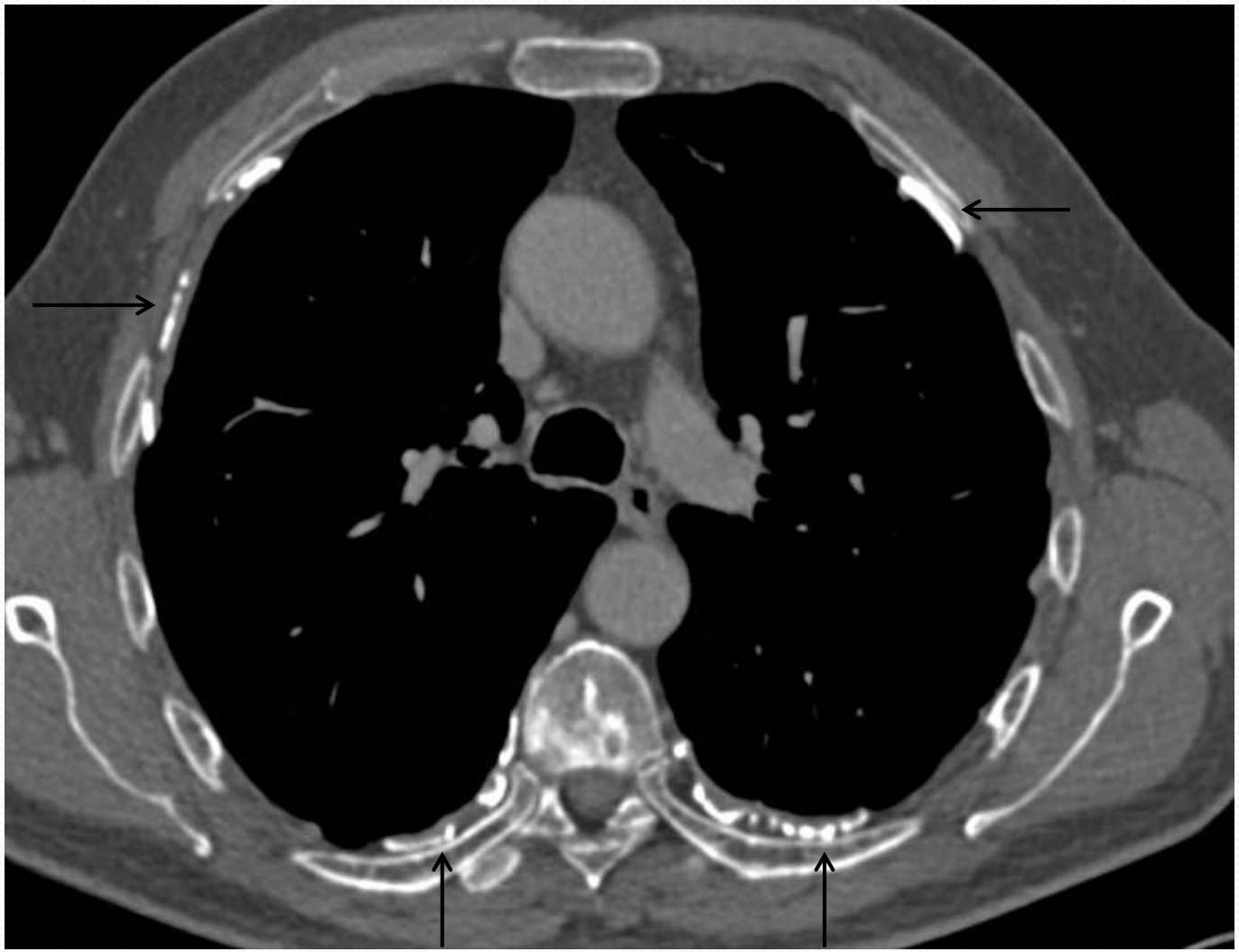
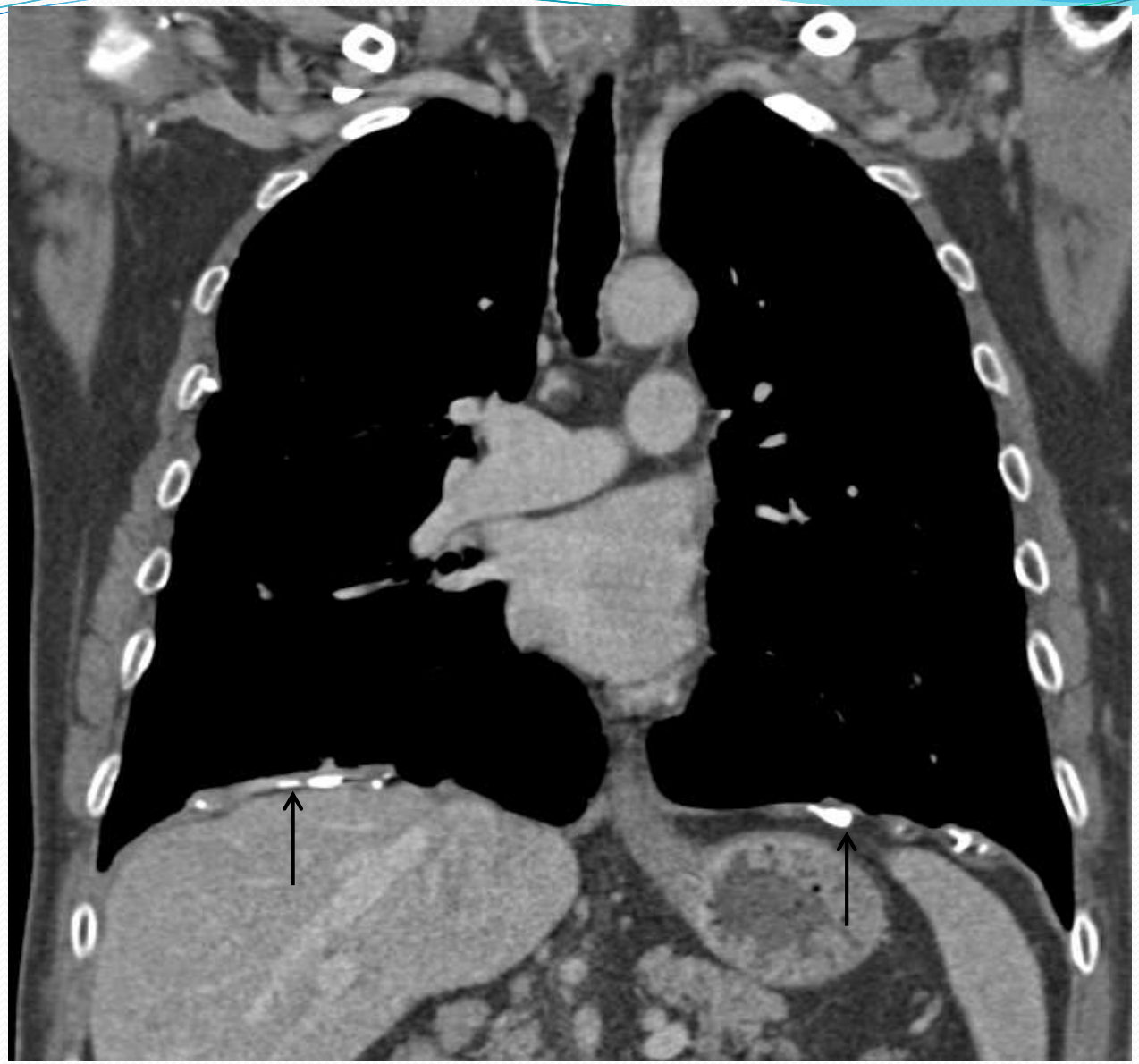
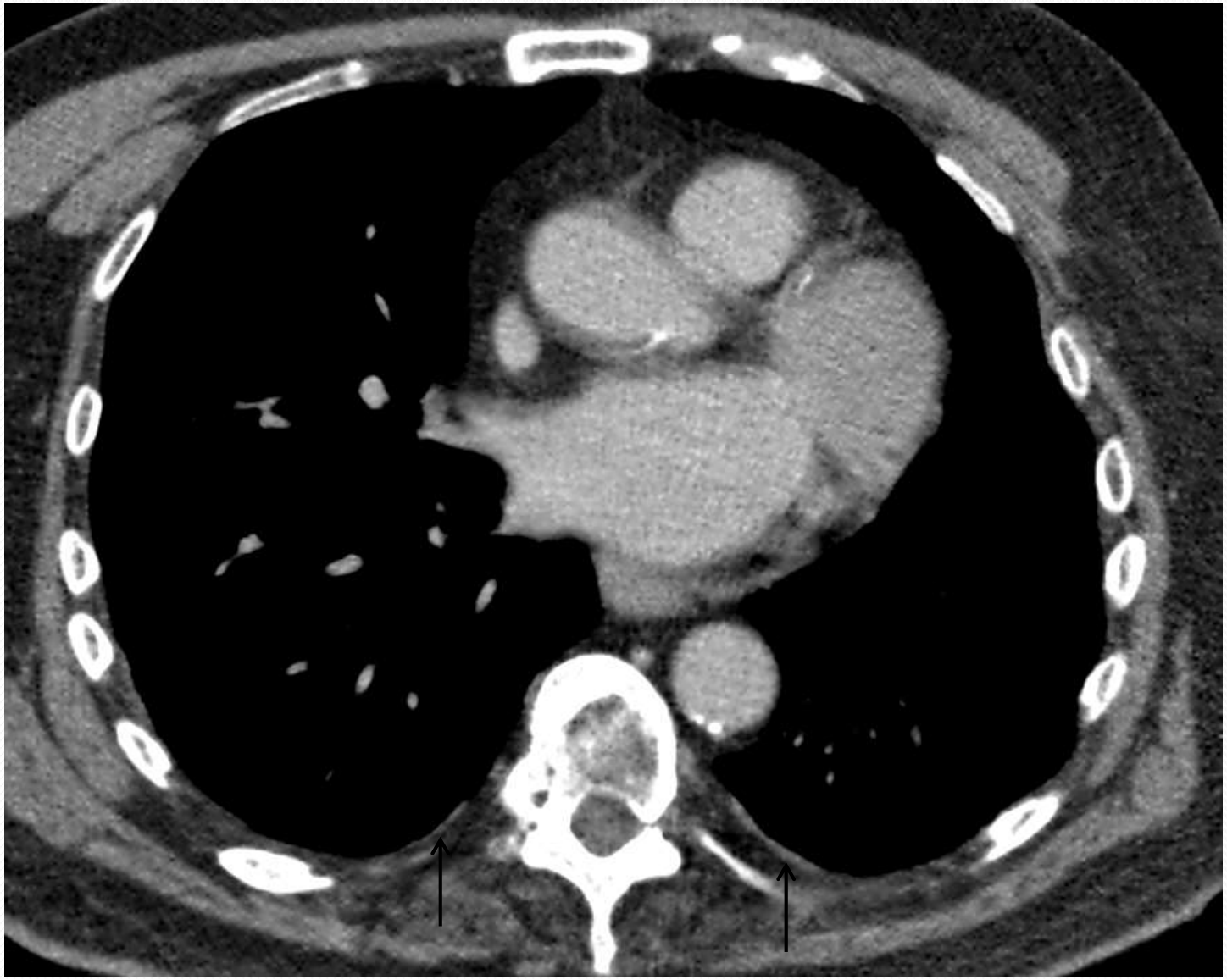
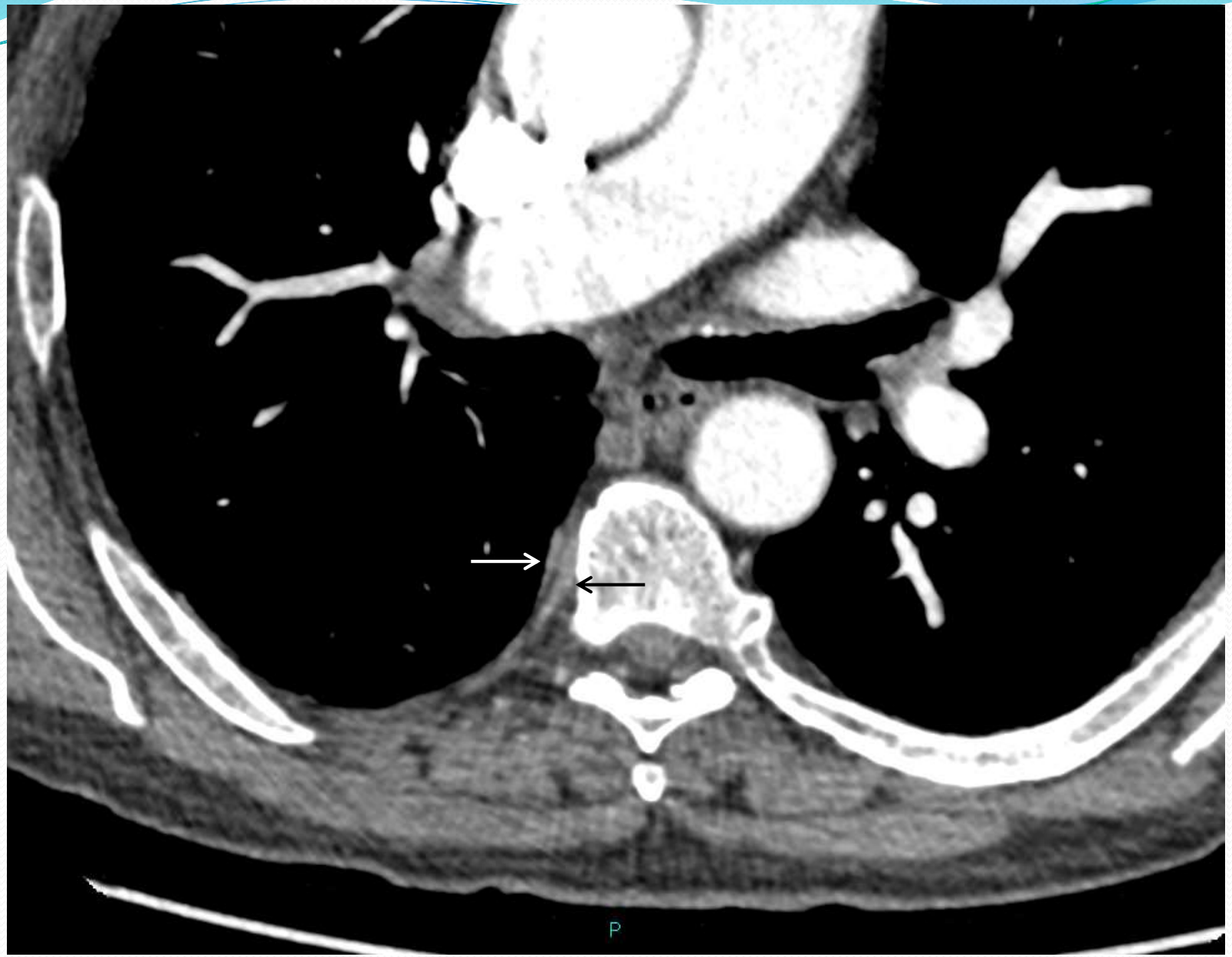
- CXR: smooth elevations of pleura, most easily seen in profile. When calcified, they appear linear in profile. En face, they have a variable appearance but the classic described as “holly leaf”.
- **CT: Much more readily appreciated. Smooth, well demarcated focal areas of pleural thickening +/- calcification. They may project into the lung parenchyma, or appear as subtle smooth pleural thickening, often beneath ribs or separated from ribs by extrapleural fat.**





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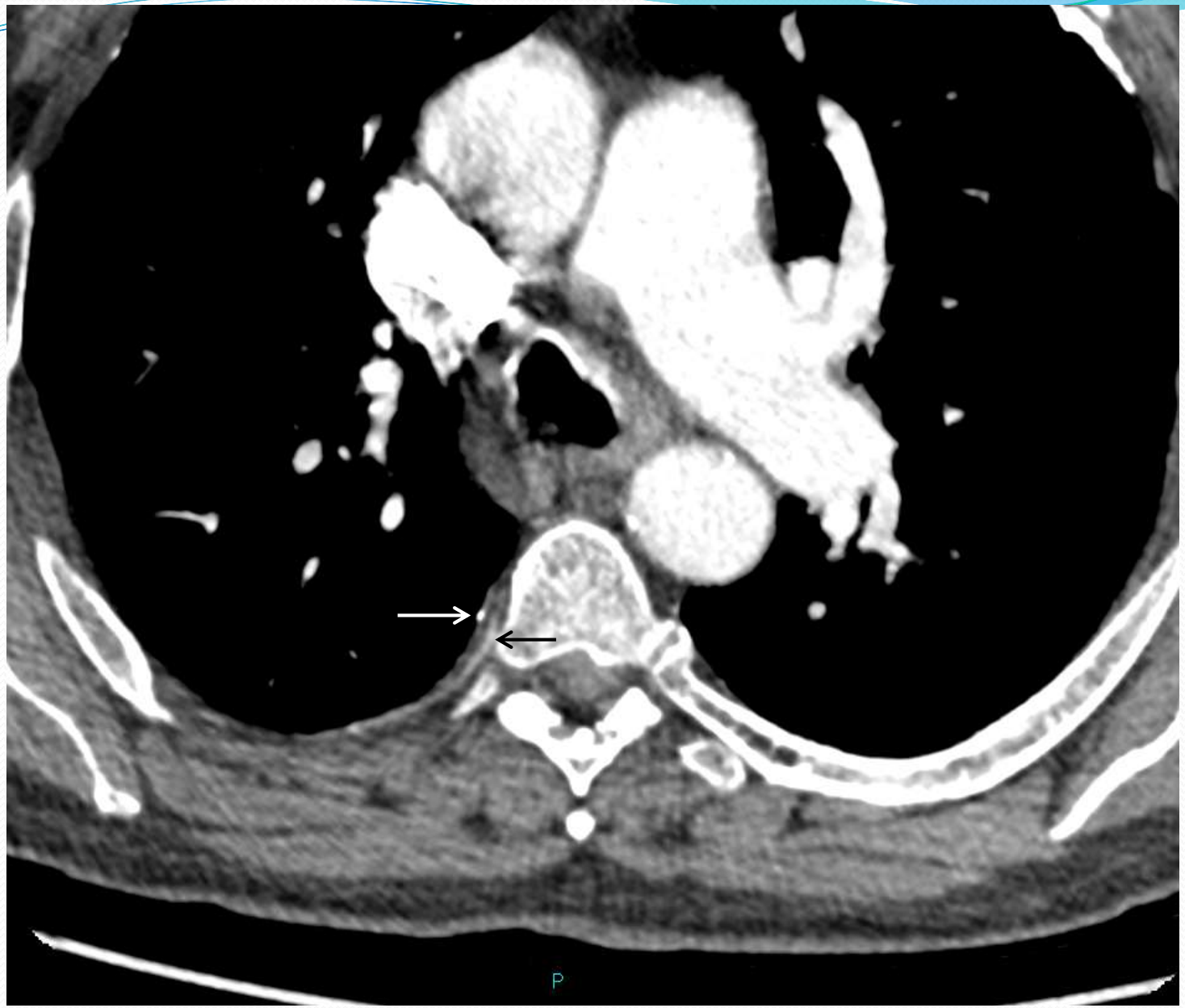
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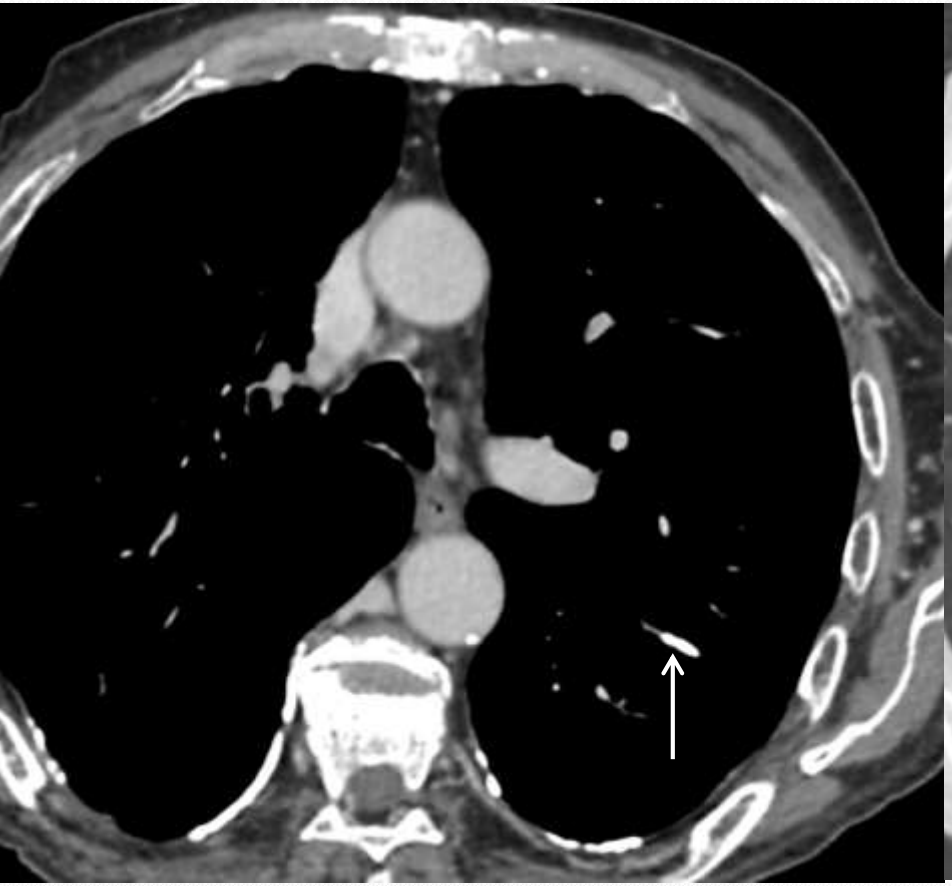
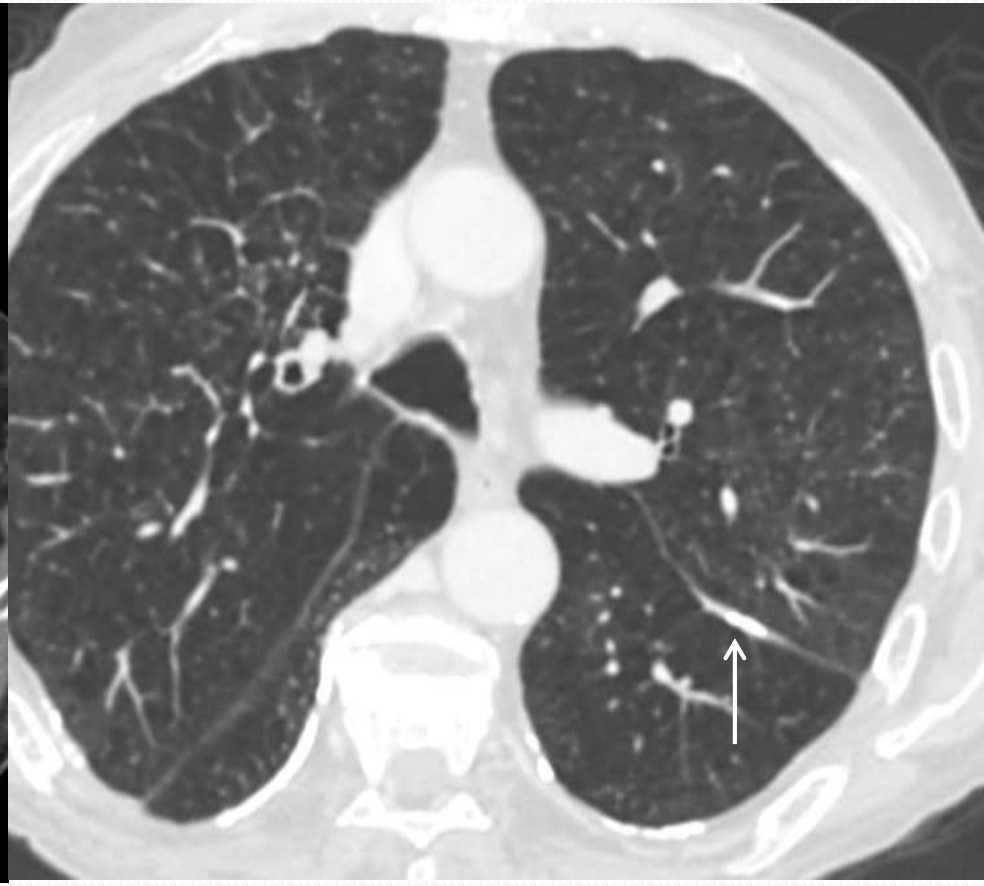
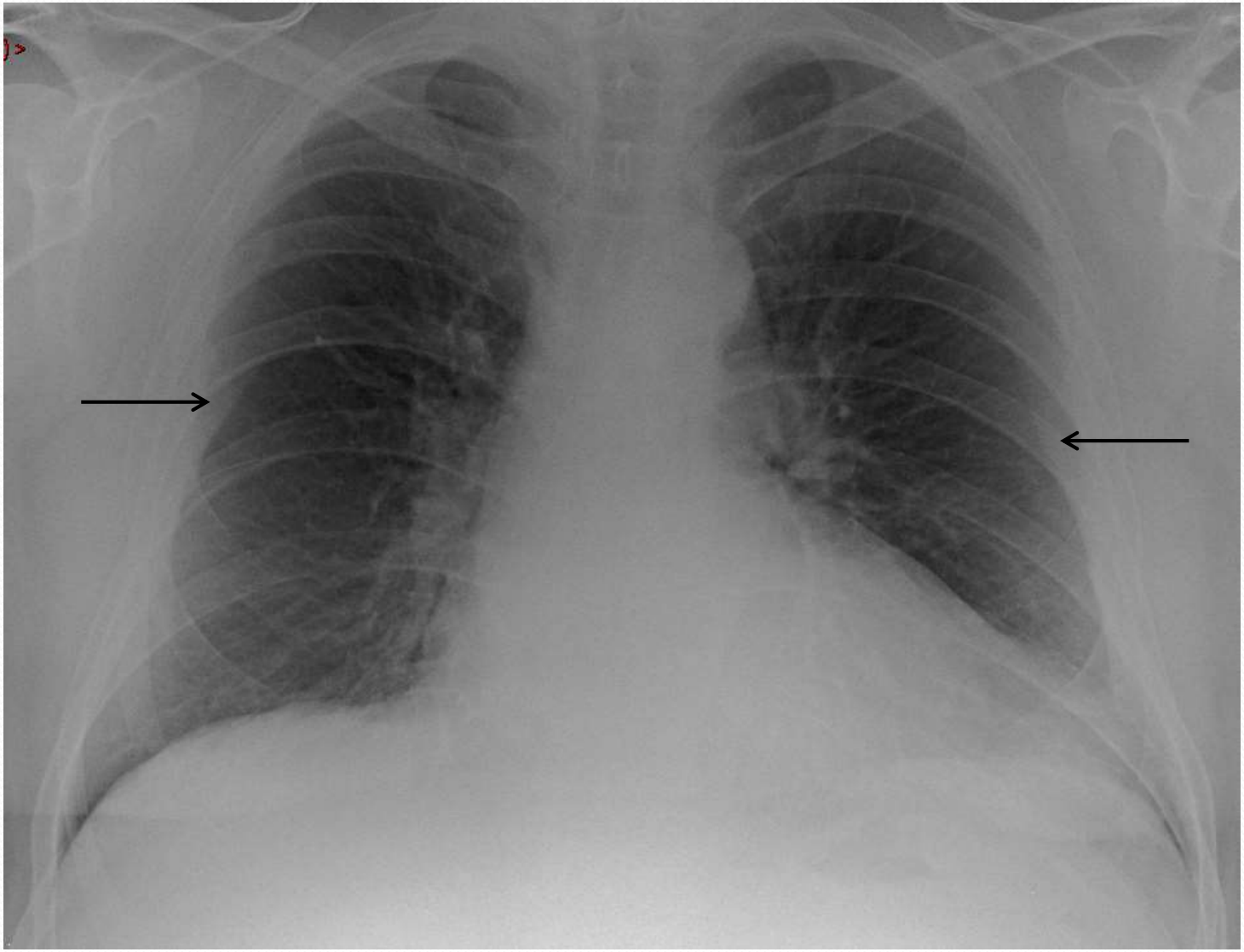
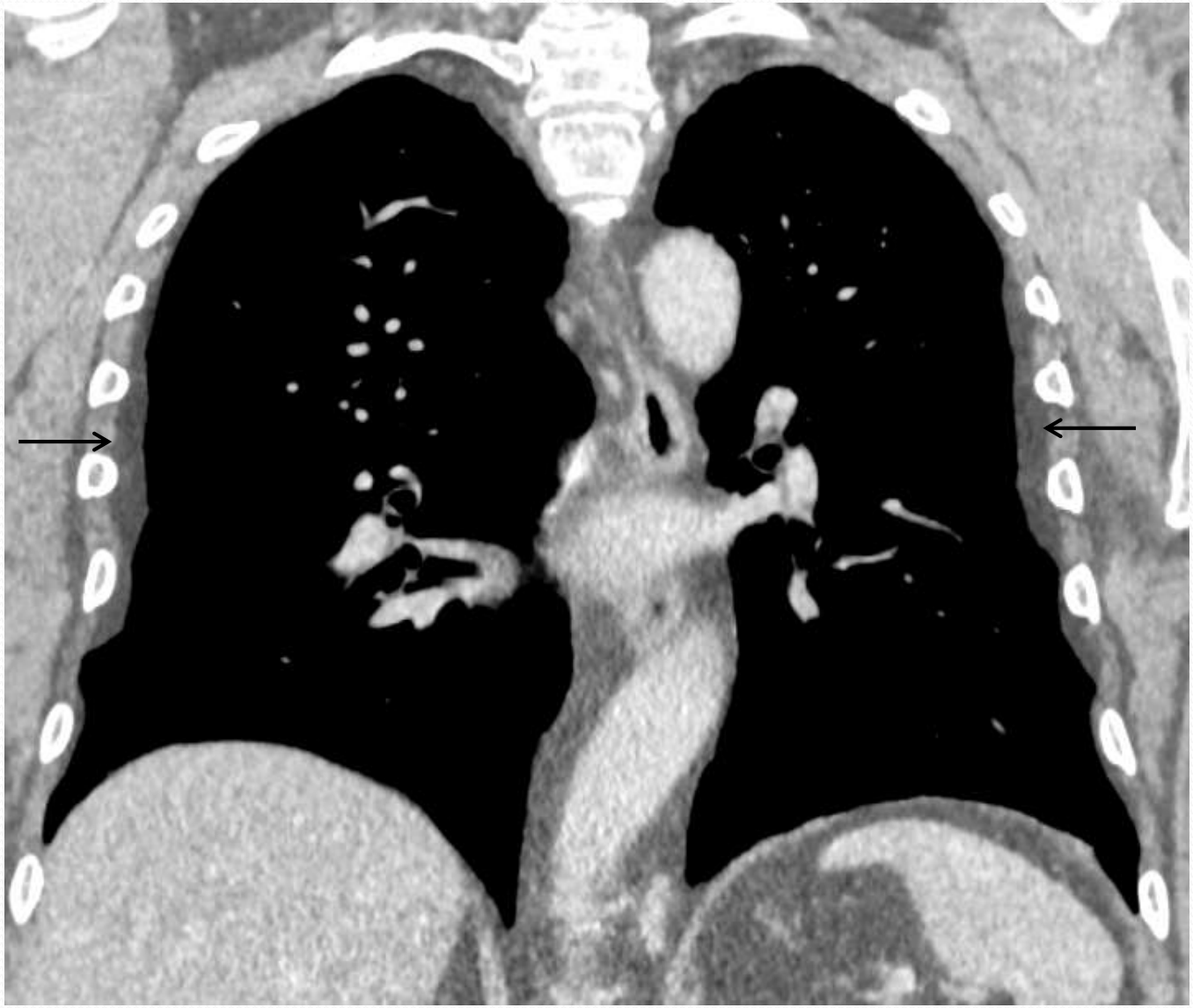




Pleural Plaques: Pitfalls

- Extrapleural fat.



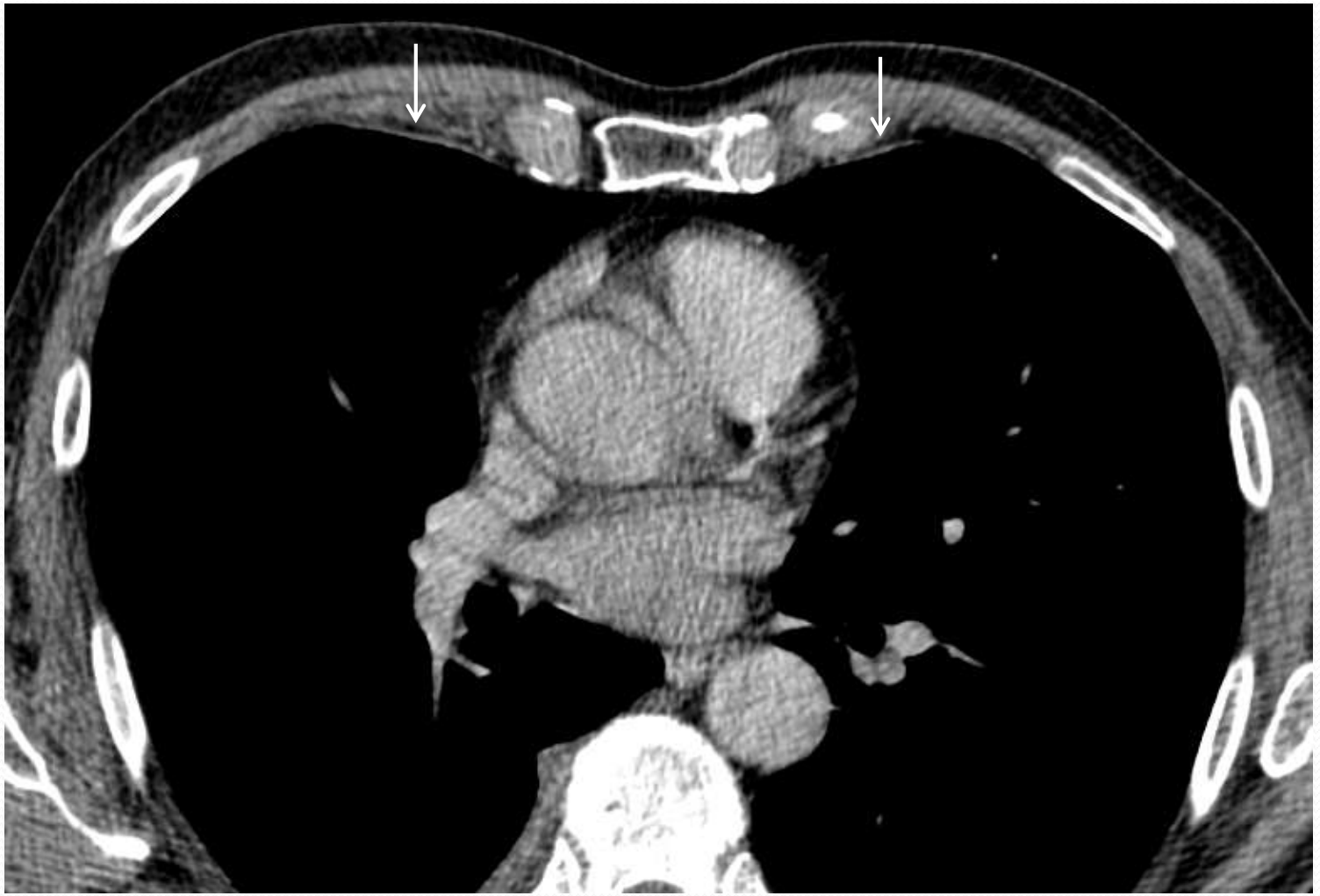


Pleural Plaques: Pitfalls

- Extrapleural fat.
- **Transversus thoracis and subcostalis muscles:**

  1-2 mm thick smooth line internal to anterior end of ribs or costal cartilages at level of heart (transversus thoracis), or internal to one or more posterolateral ribs at the same level (subcostalis).

  Always smooth, uniform thickness and symmetrical bilaterally.



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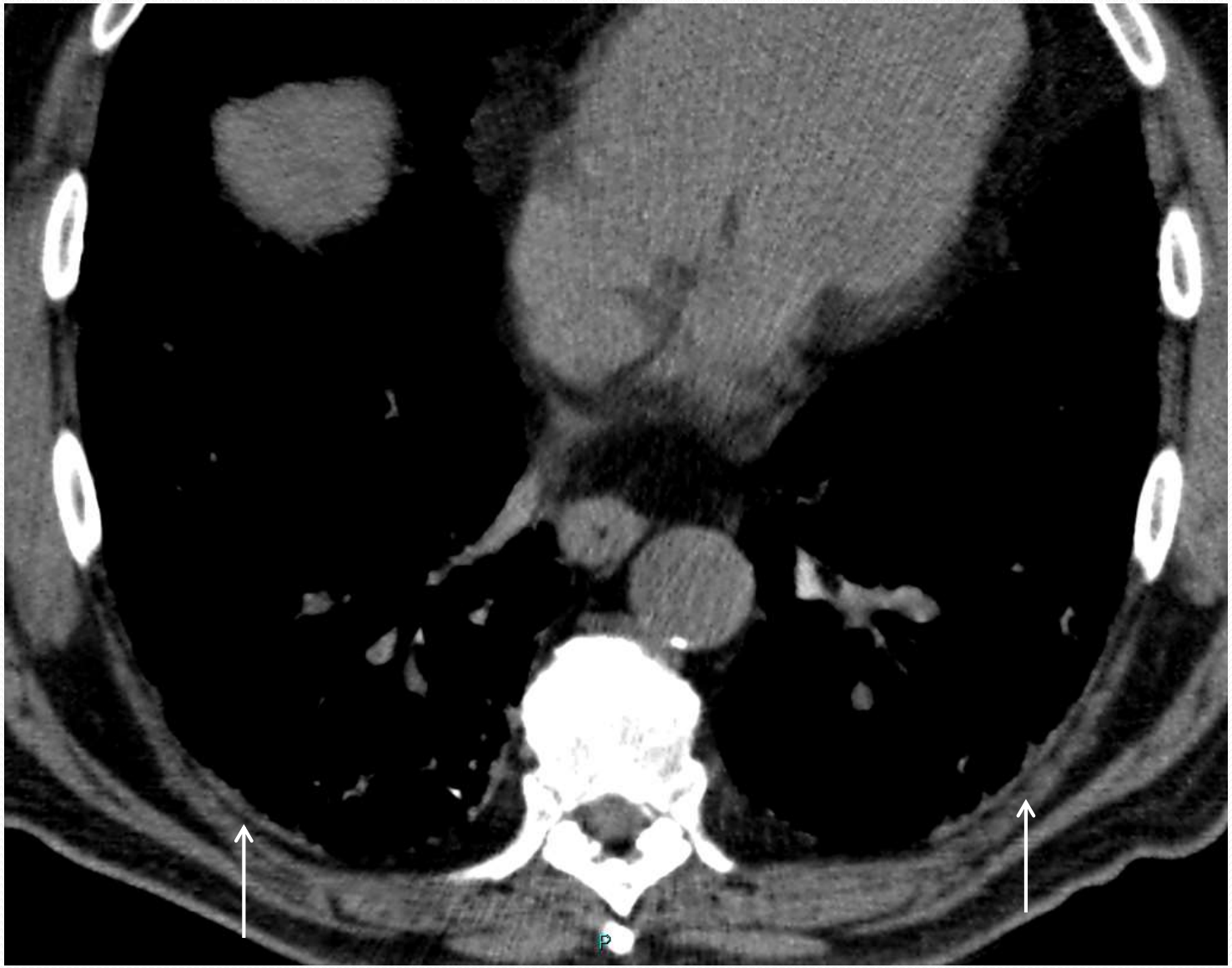
- **Thickening of the subpleural interstitium due to lung parenchymal disease.**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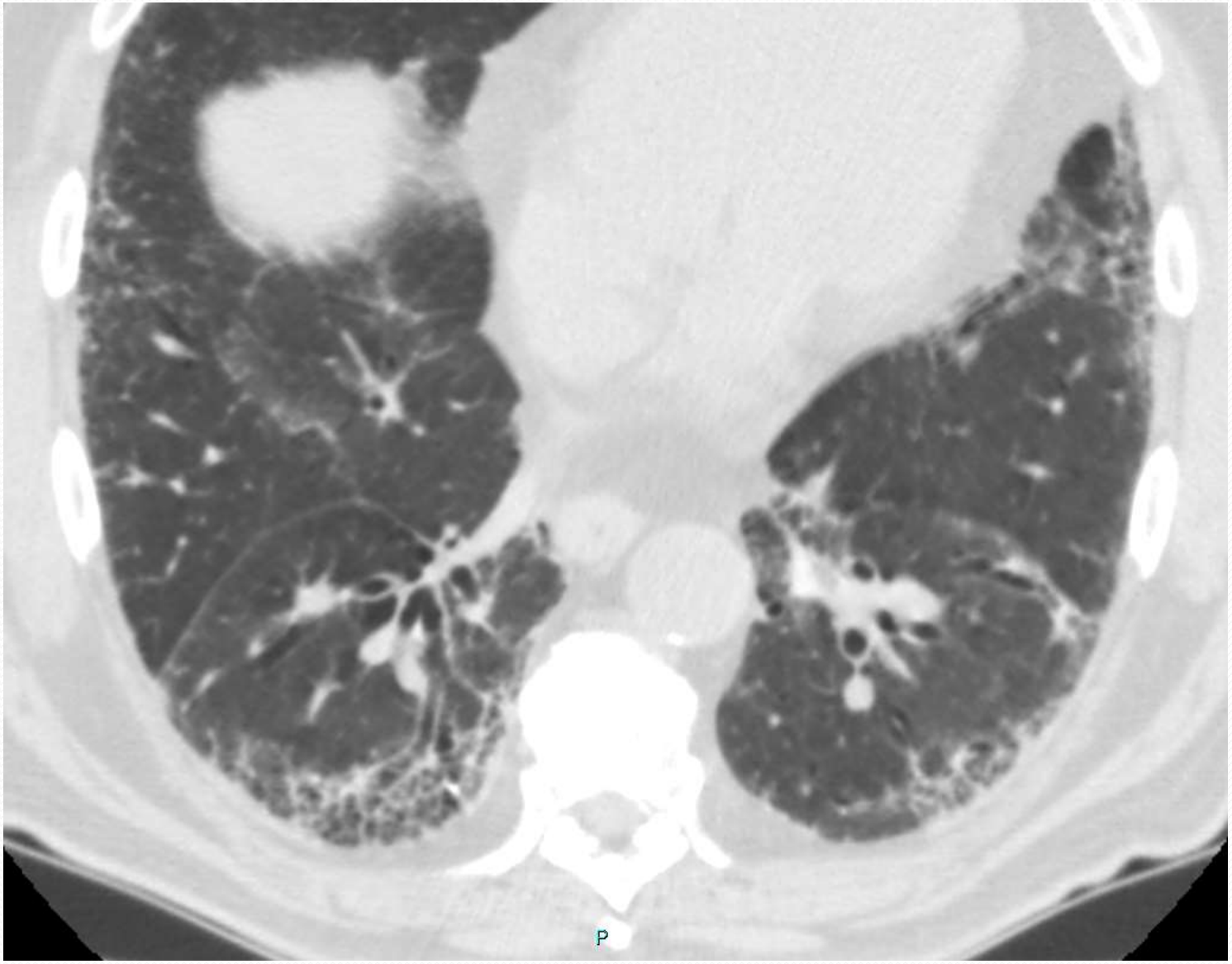
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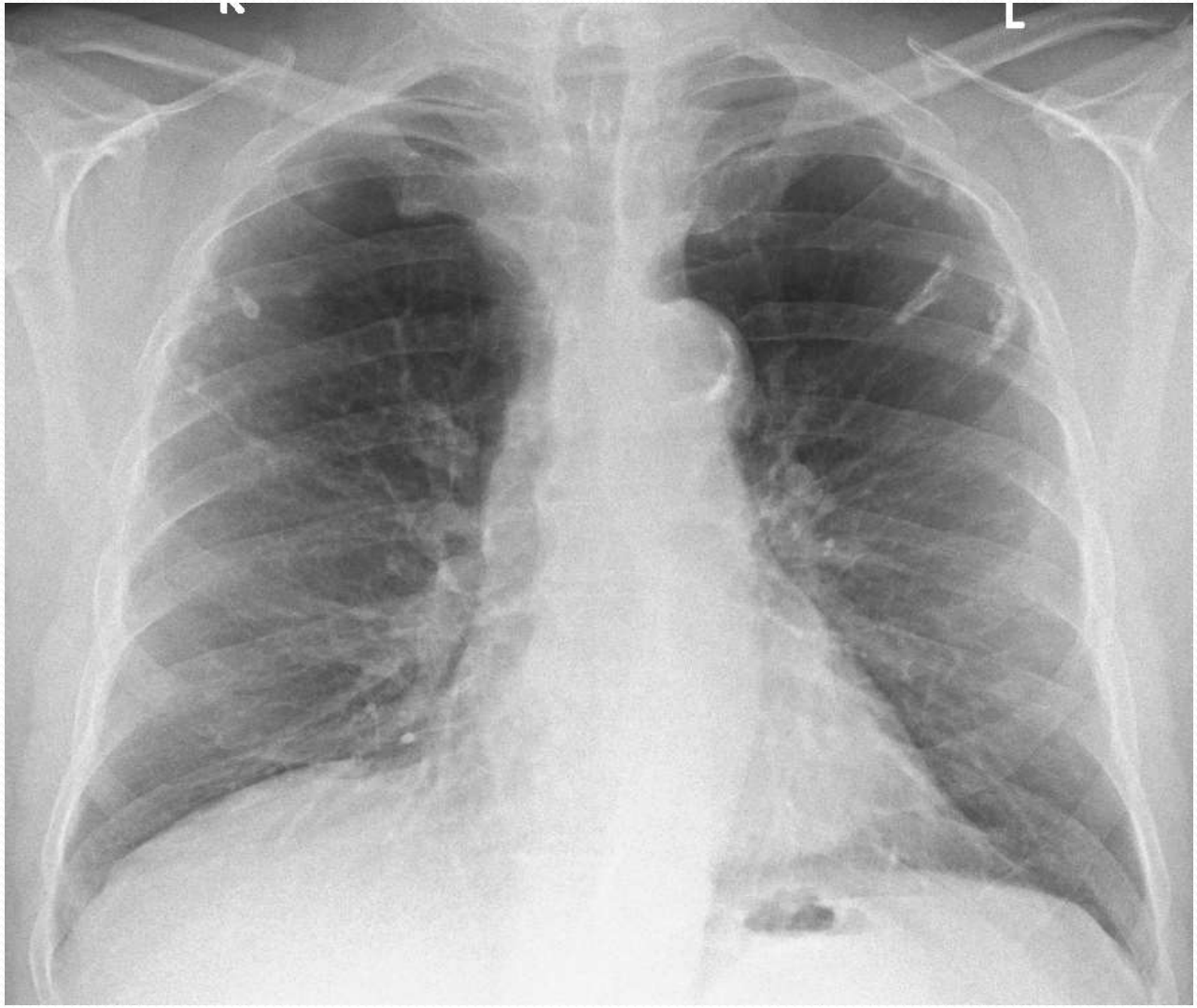
- Thickening of the subpleural interstitium due to lung parenchymal disease.
- **Pseudoplauqes (silicosis, pneumoconiosis, sarcoidosis)**

  Confluent subpleural nodules +/- calcification.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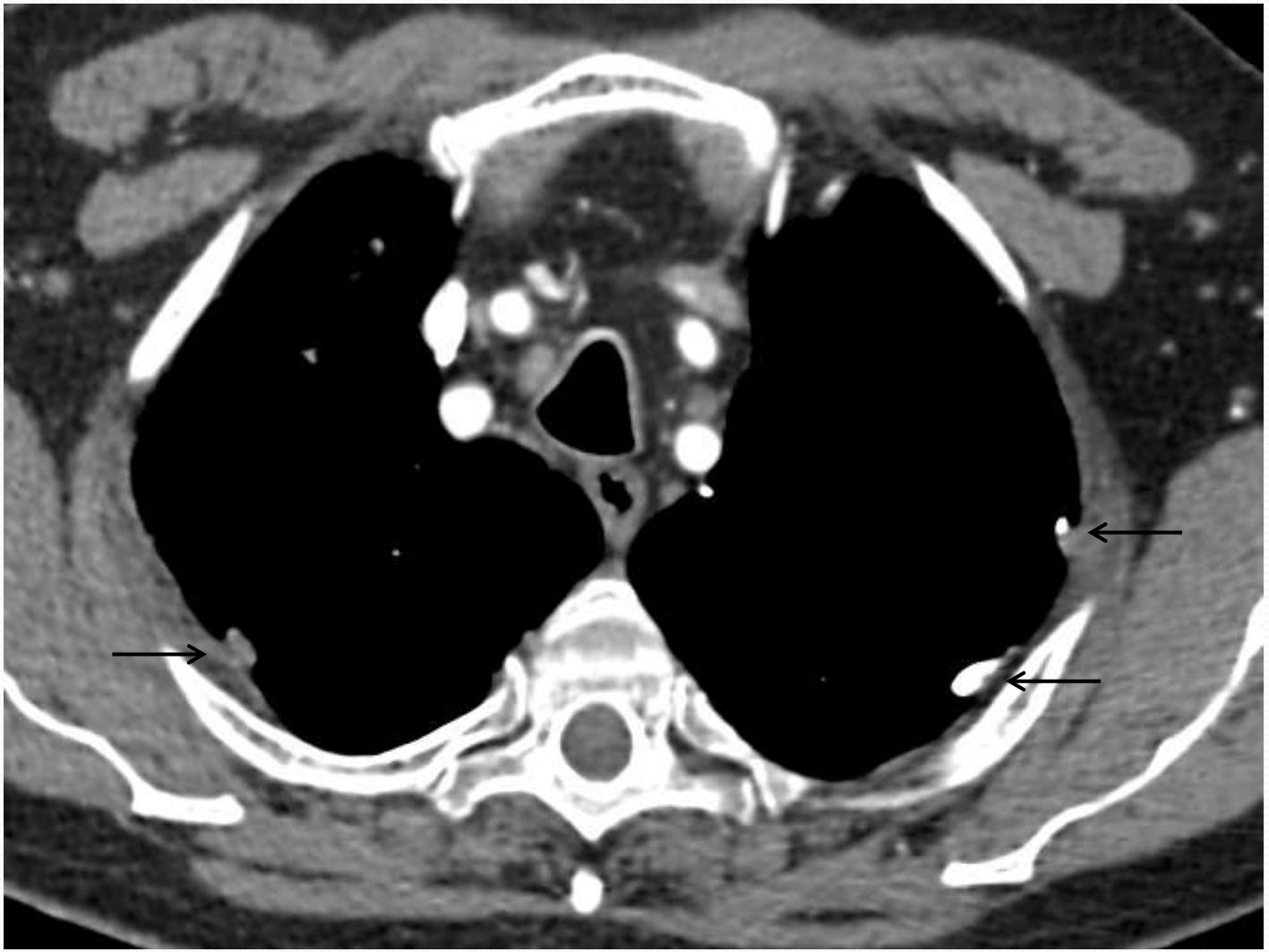

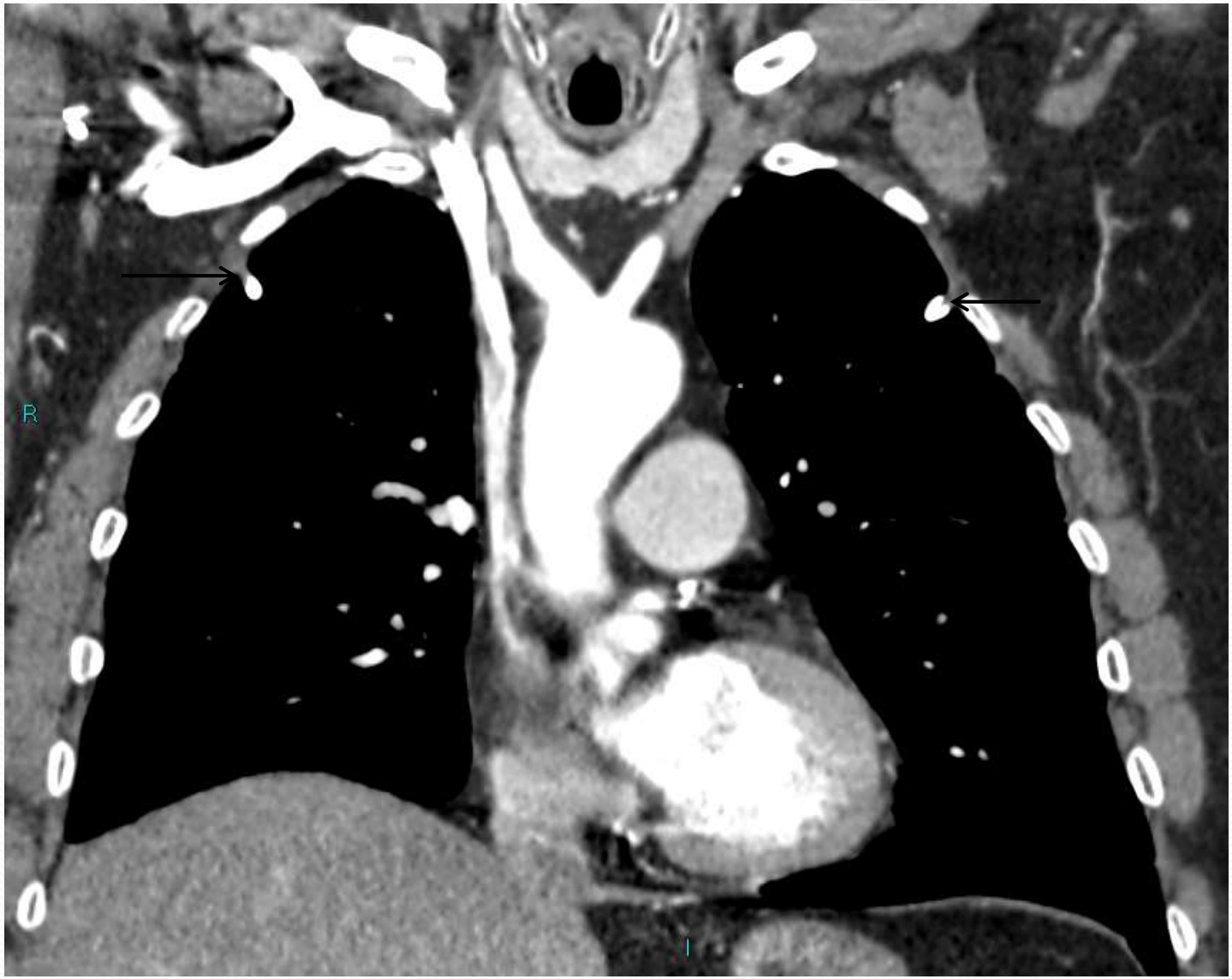
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Pleural Plaques

Pleural plaques usually enlarge as they age, and may coalesce to form large areas of confluent pleural thickening. (Up to 10 cm has been described).

However, although large, the plaques still move independently, so, unlike diffuse pleural thickening, pulmonary constriction doesn't occur.

Diffuse Pleural Thickening

- Diffuse pleural thickening is thought to most often represent the sequelae, or organisation of a previous benign asbestos induced pleural effusion.
- Identical imaging findings are seen following the organisation of exudative effusions due to a number of different aetiologies (empyema, haemothorax, connective tissue disease, uraemia, drug reaction), when the confluent pleural opacity is often referred to as a fibrothorax.
- When extensive, there may be significant volume loss and ventilatory impairment, which obviously has clinical and medical legal implications.

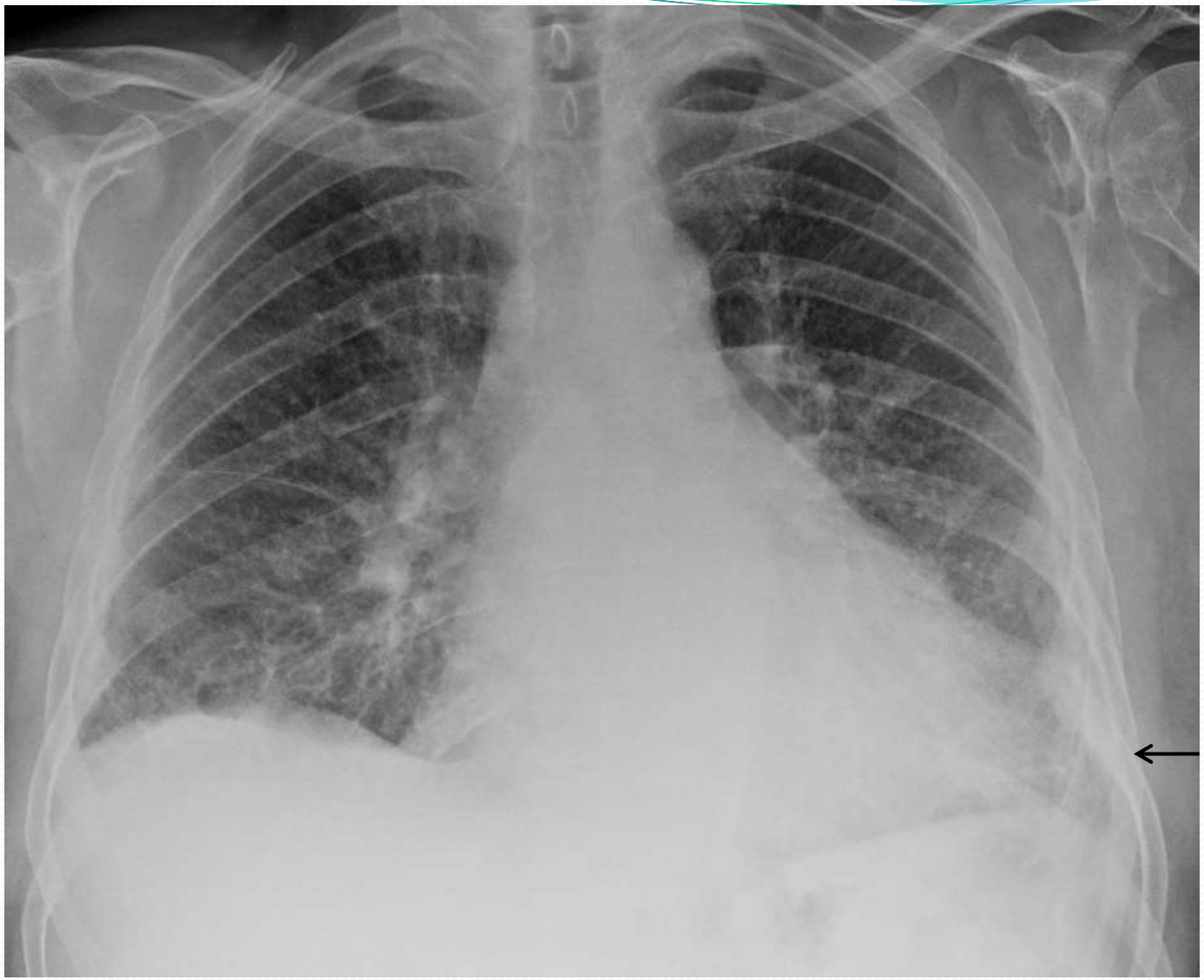
Diffuse Pleural Thickening (DPT)

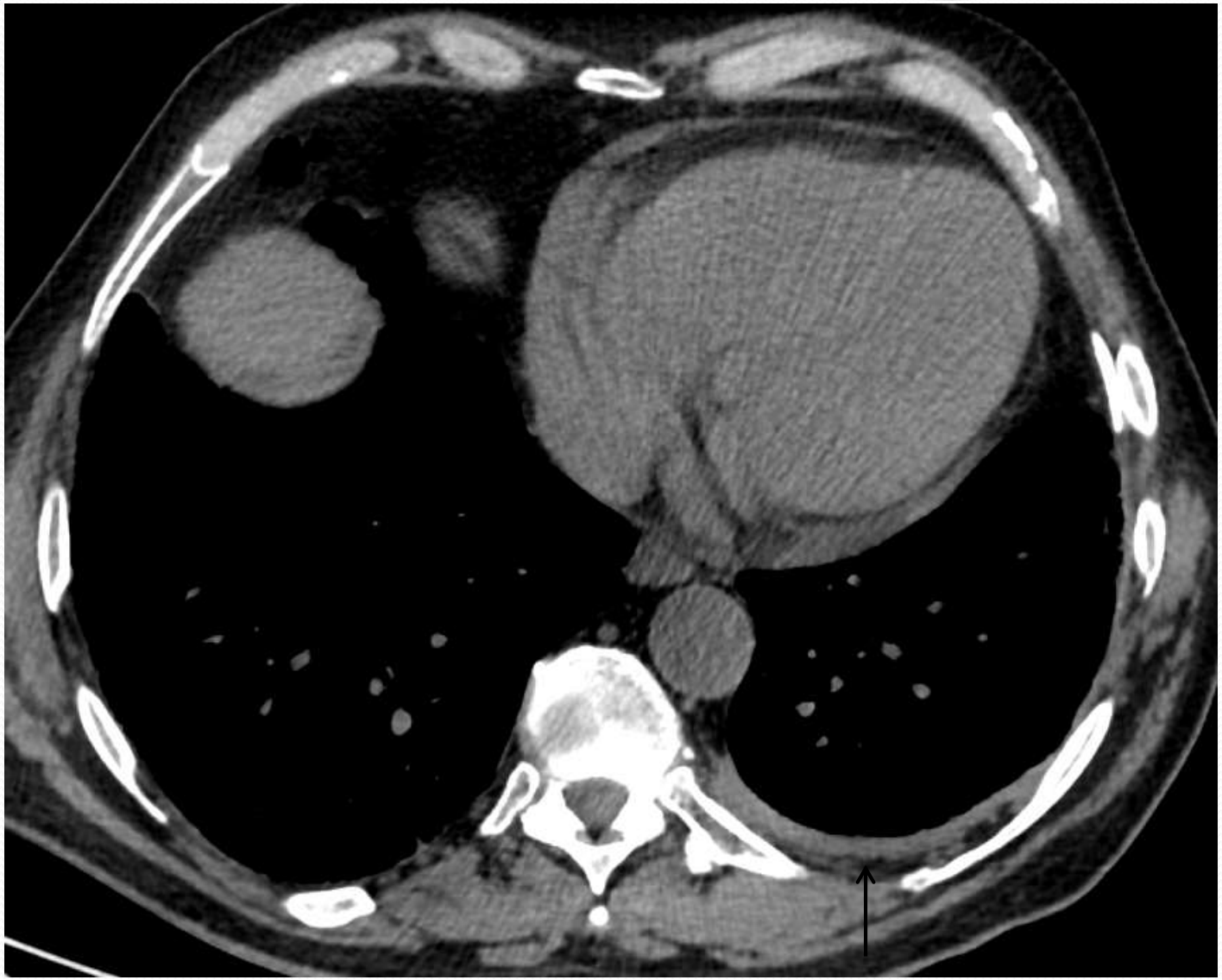
- ILO 2011 Definition:

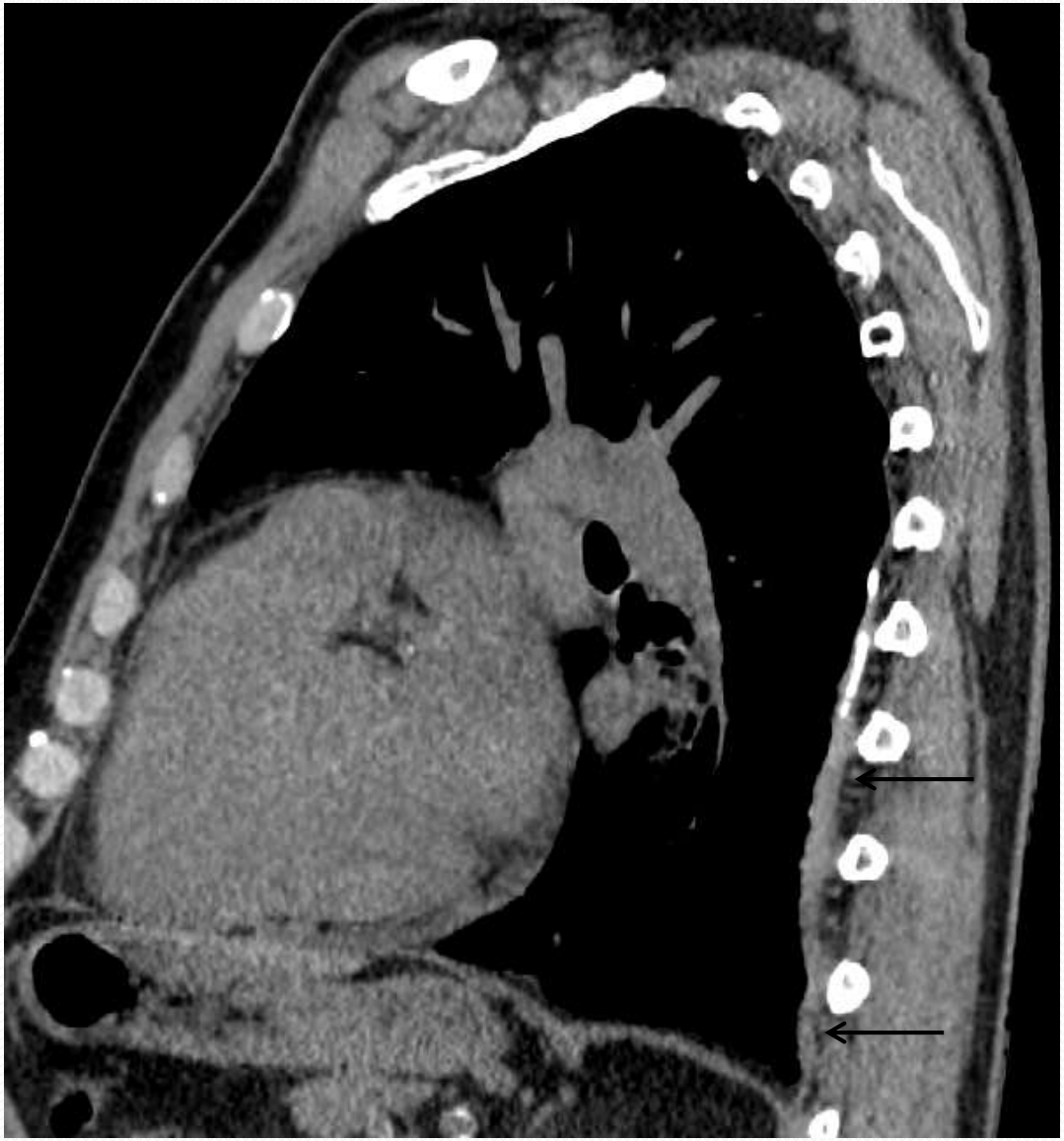
  "DPT is present radiographically when there is uninterrupted smooth pleural density extending over at least a quarter of the chest wall with associated obliteration of the costophrenic angle".

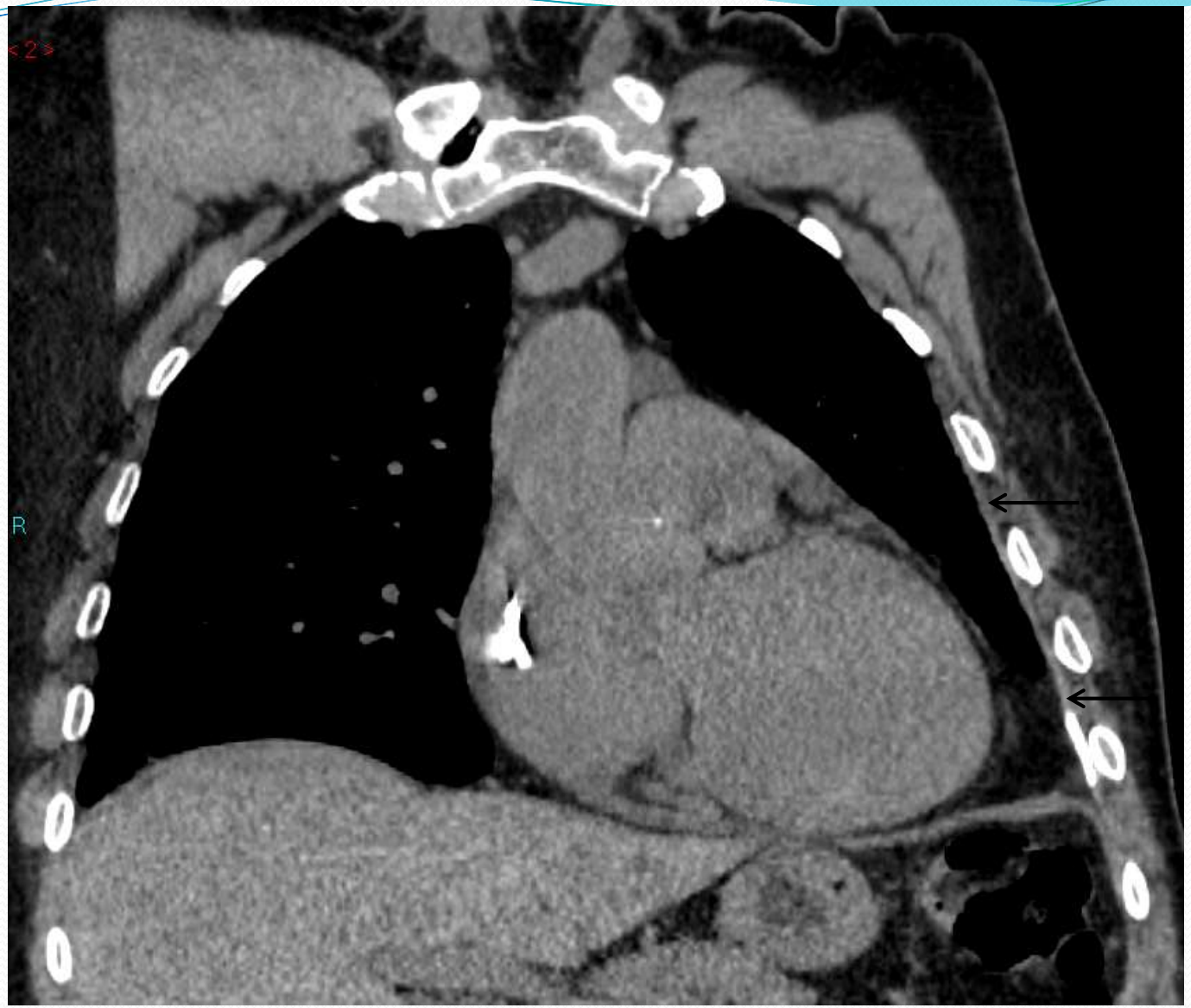
- DPT on CT:

  Continuous smooth pleural thickening that measures greater than 8 cm in cranio caudal direction, 5 cm around the hemithorax and 3 mm in thickness. Importantly, it seldom involves the mediastinal pleura and frequently is extends into, and obliterates the costophrenic recess.





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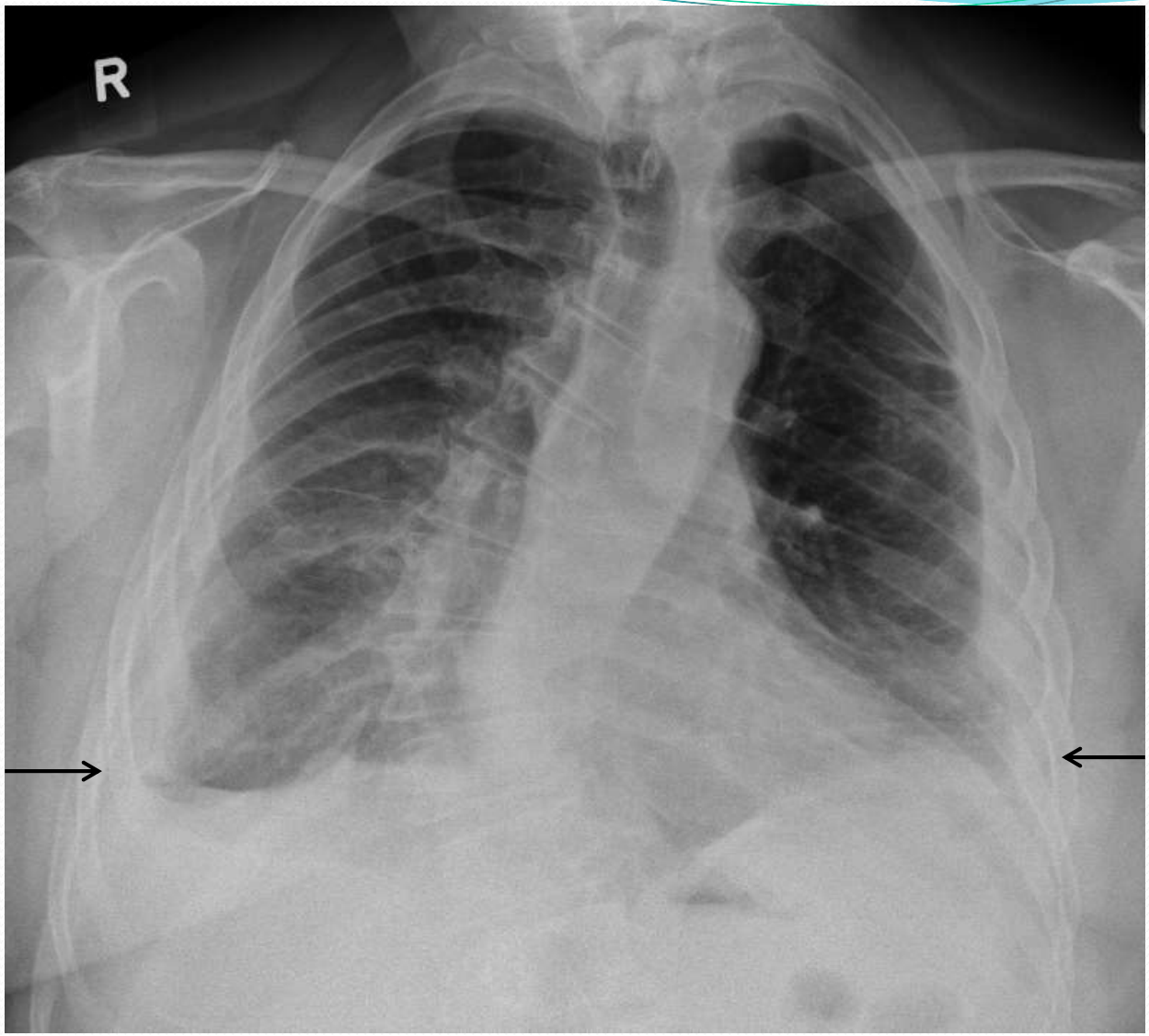
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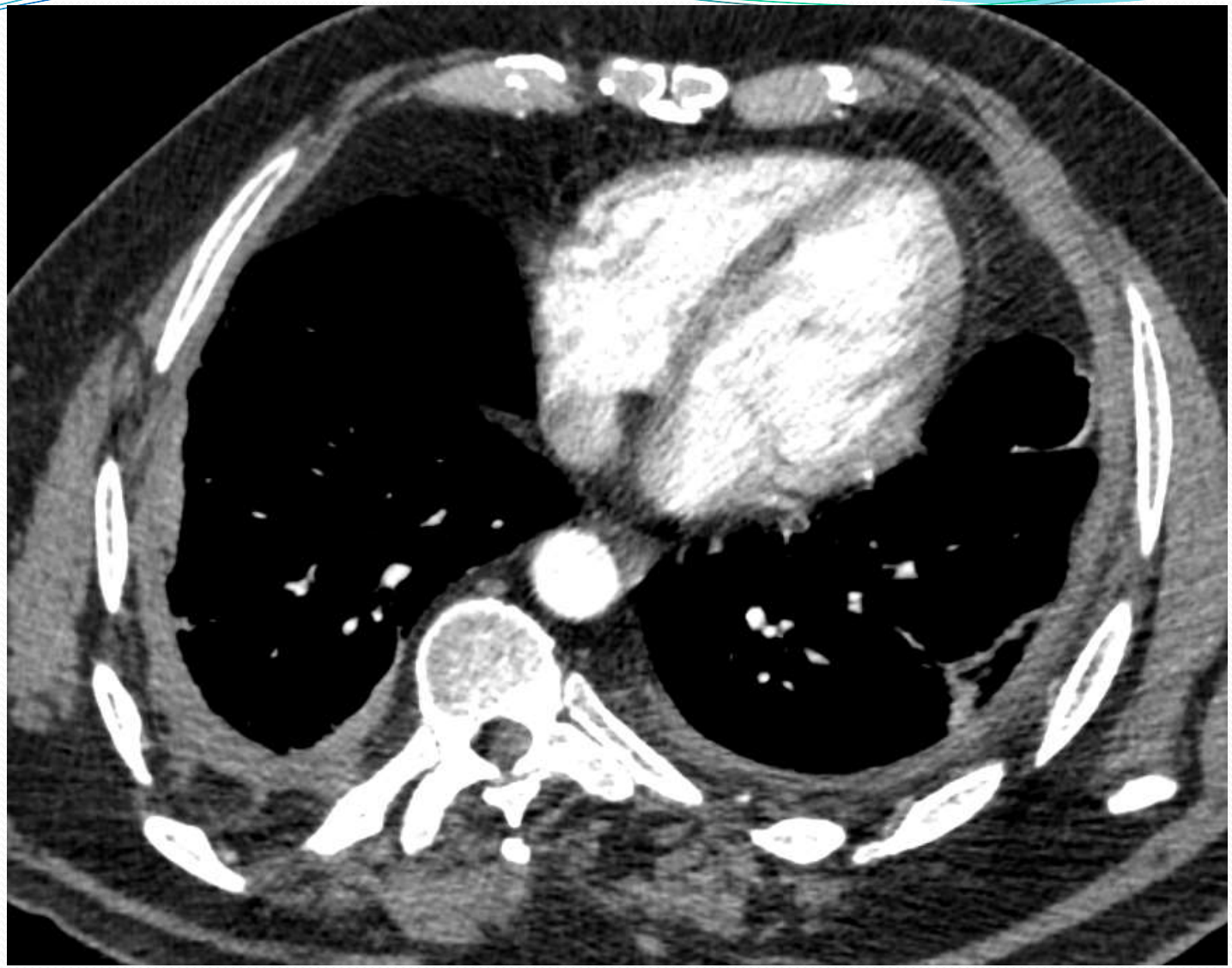
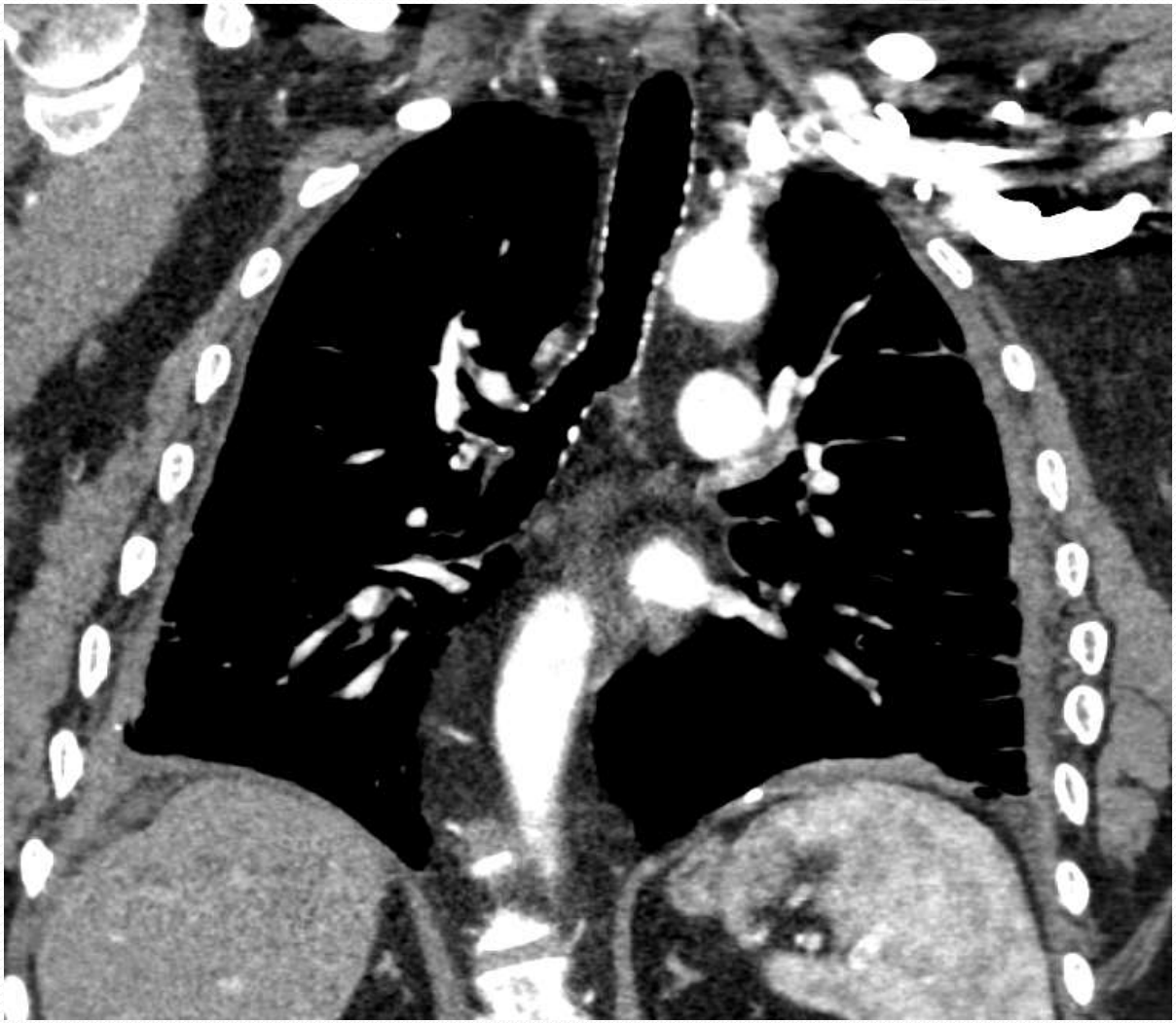
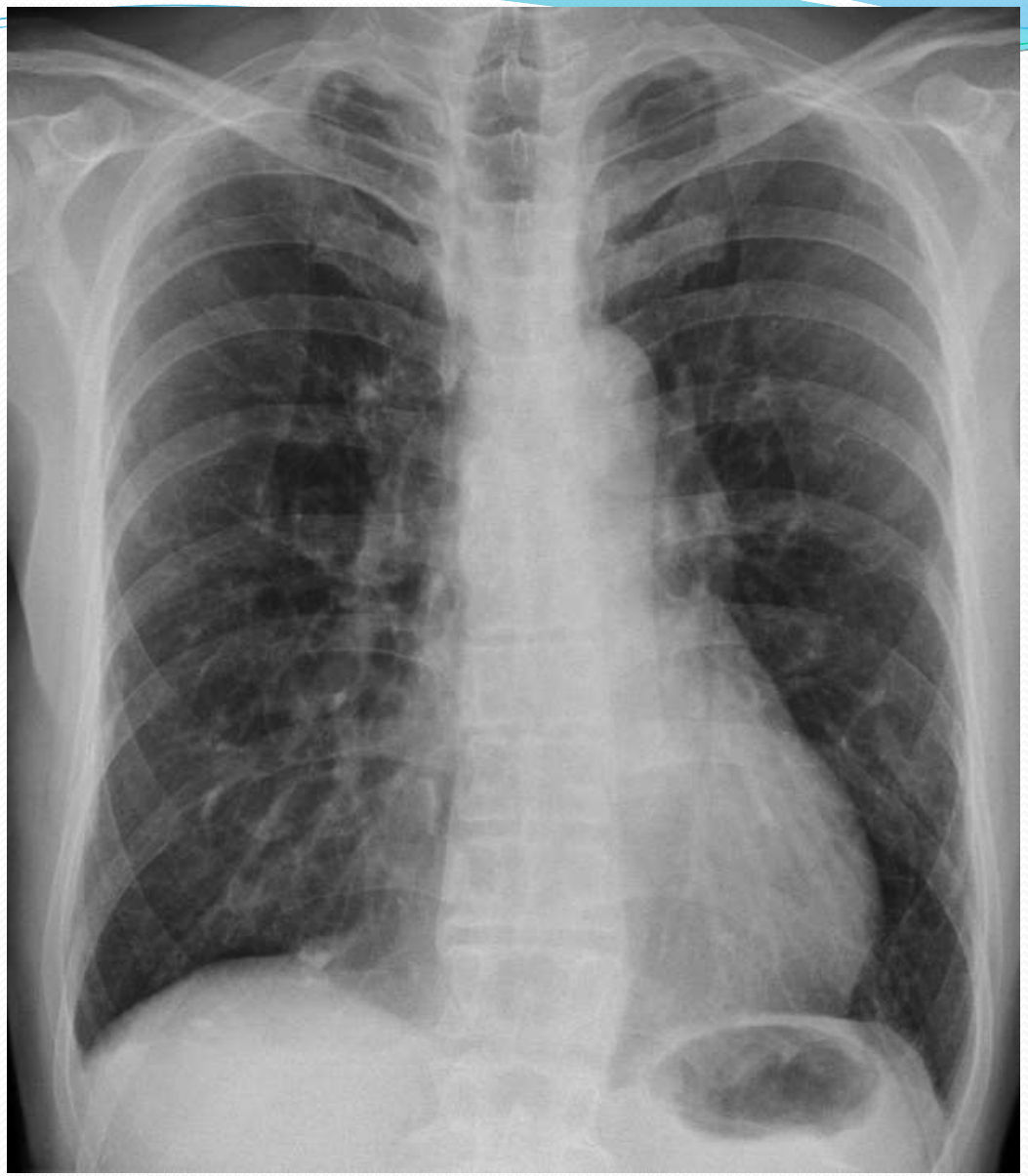
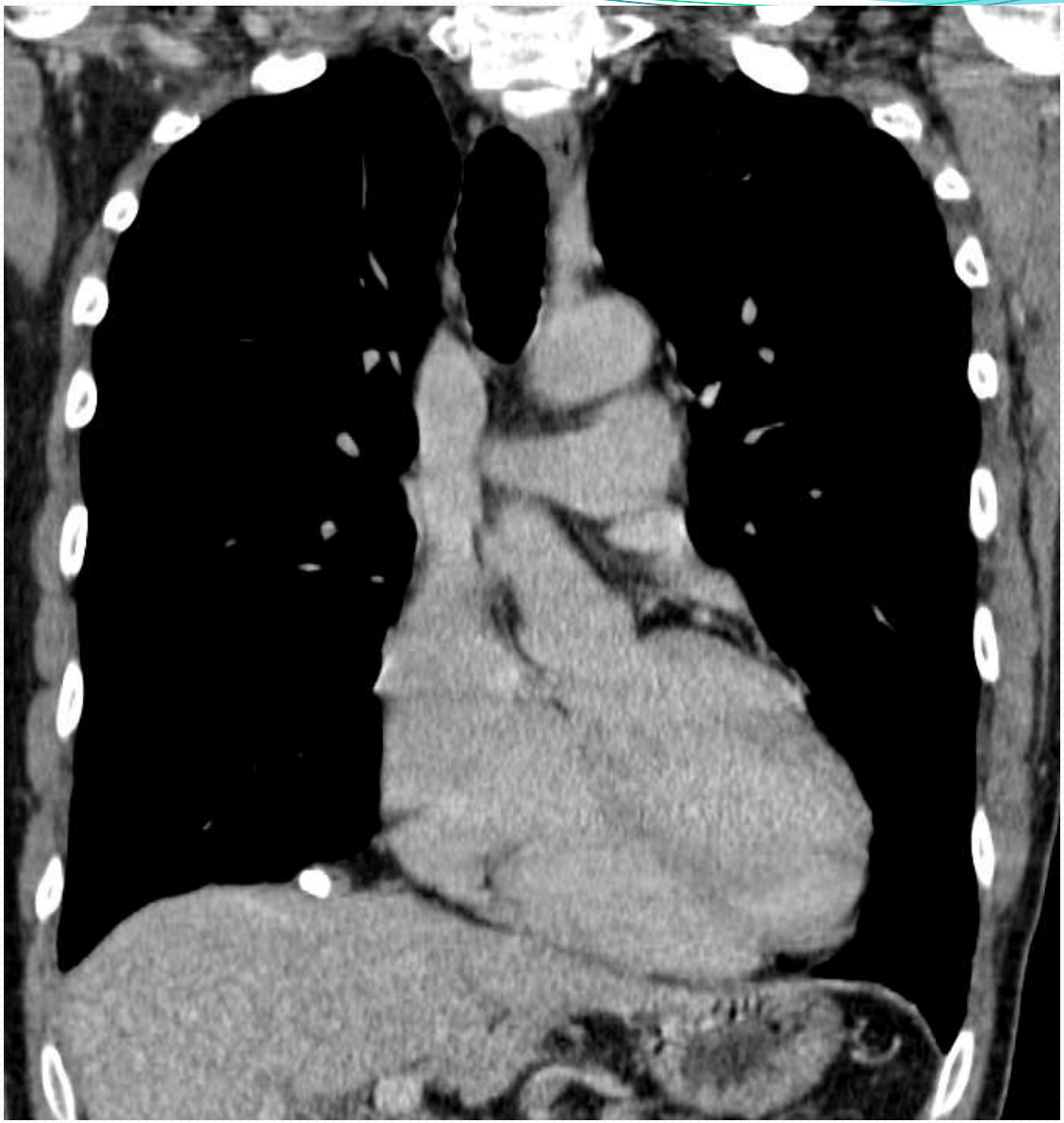


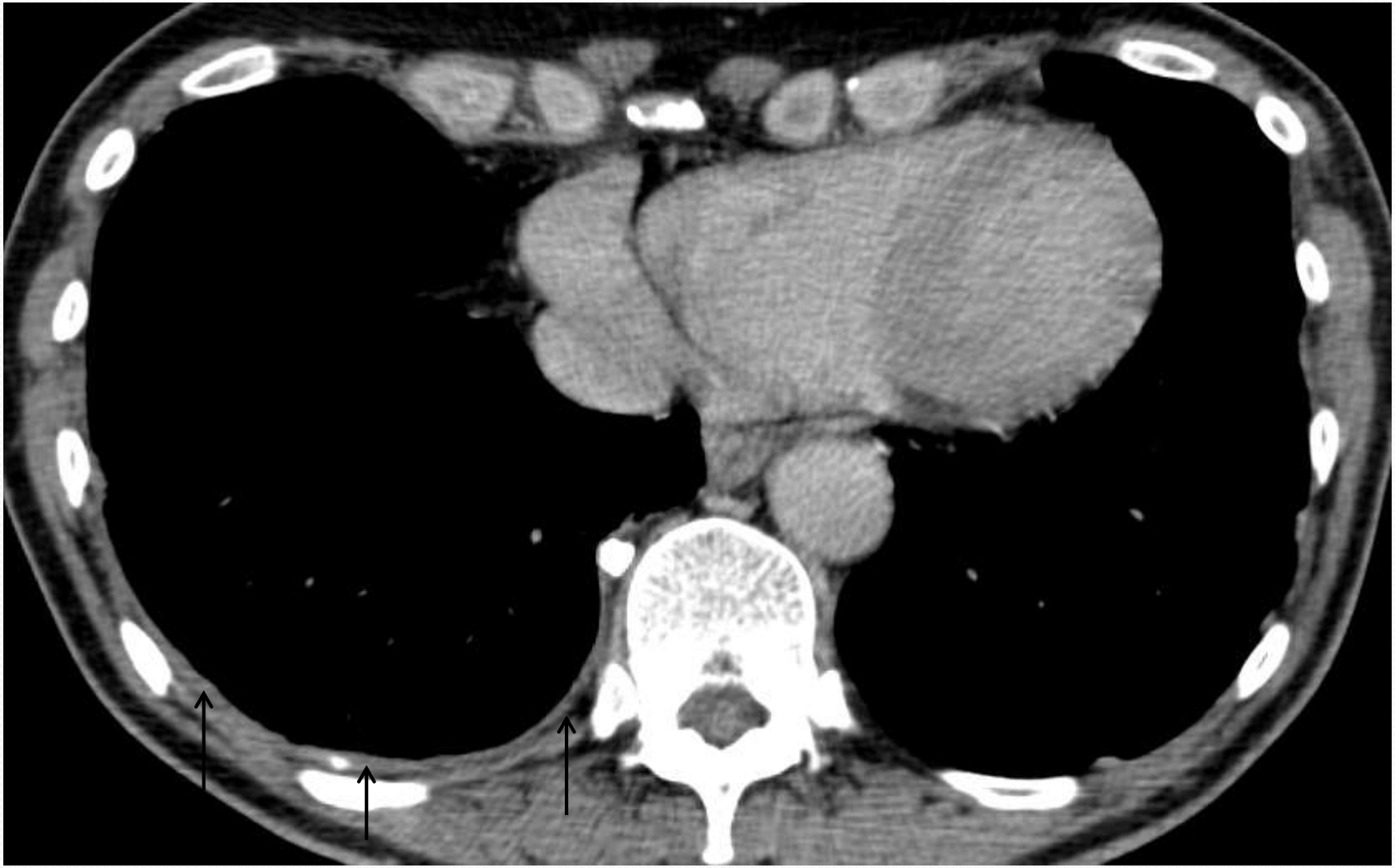


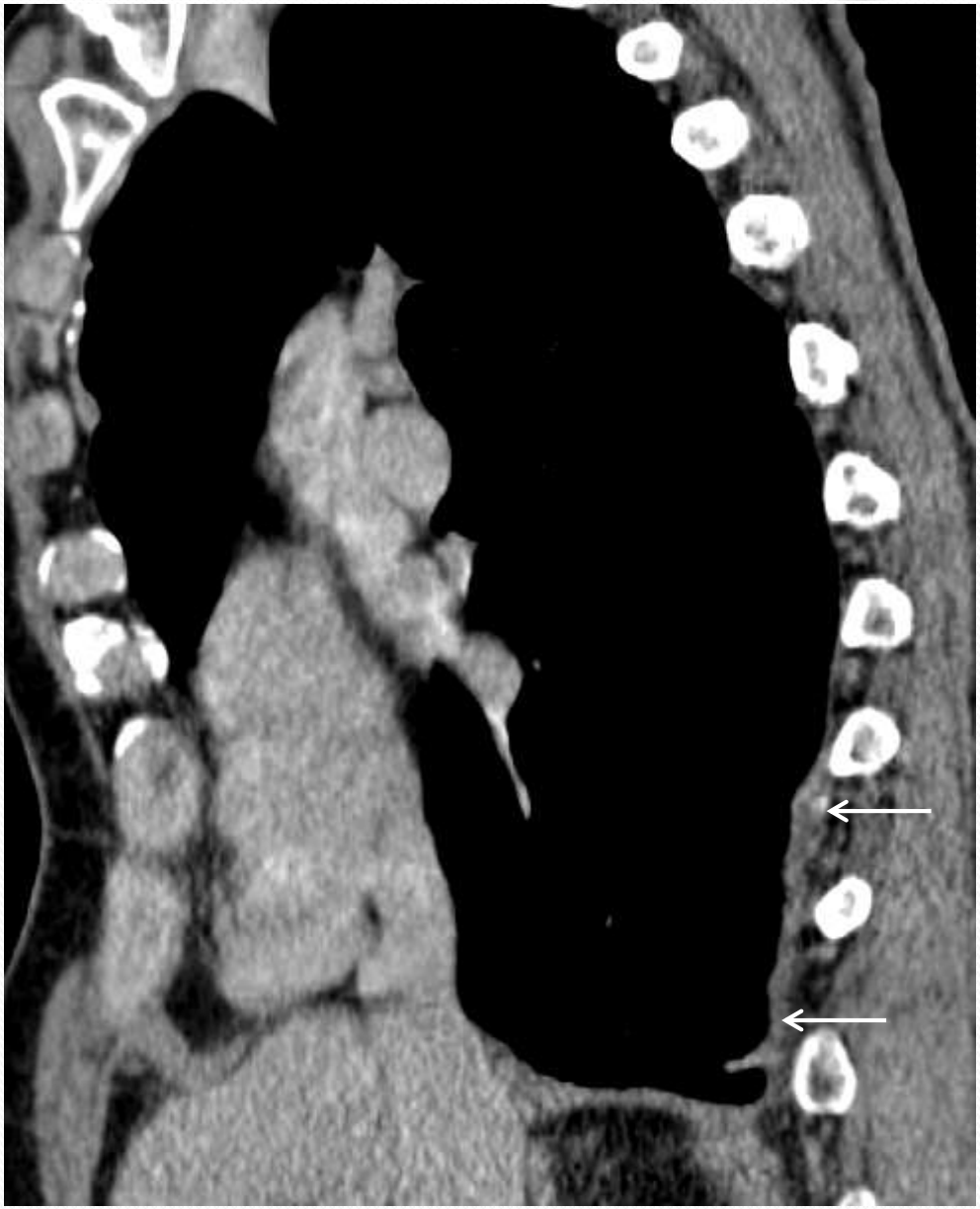
Diffuse Pleural Thickening (DPT)

- On chest radiographs, the absence of the ILO criteria for making the diagnosis of DPT, *does not* rule out the diagnosis.
- CT may demonstrate DPT which is located posteromedially and obliterates the posterior costophrenic recess, but spares the lateral costophrenic recess.







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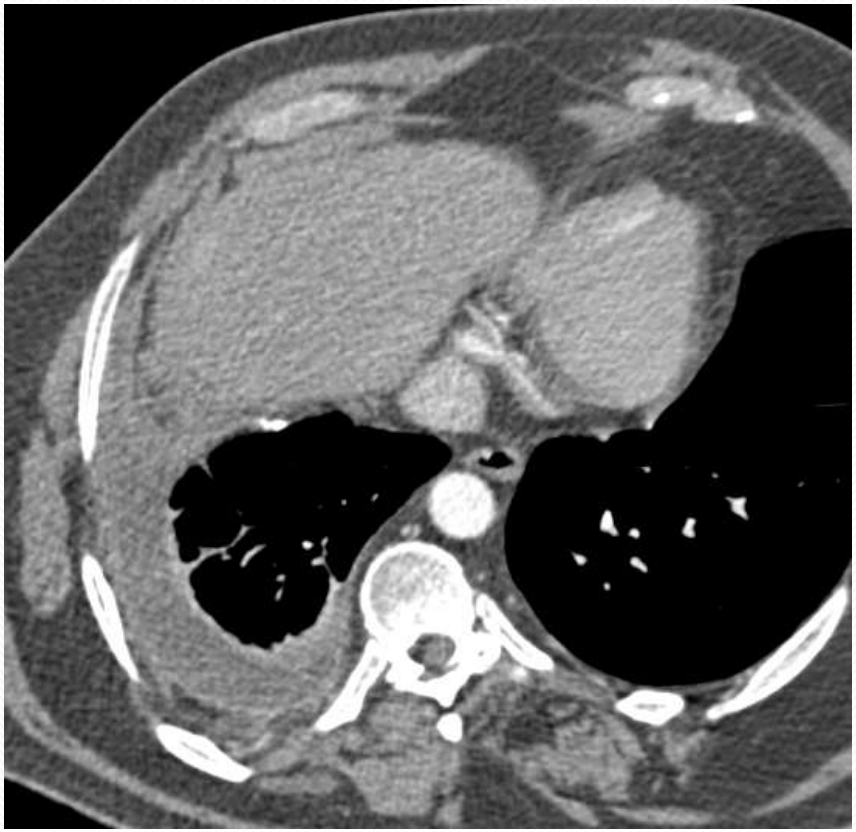
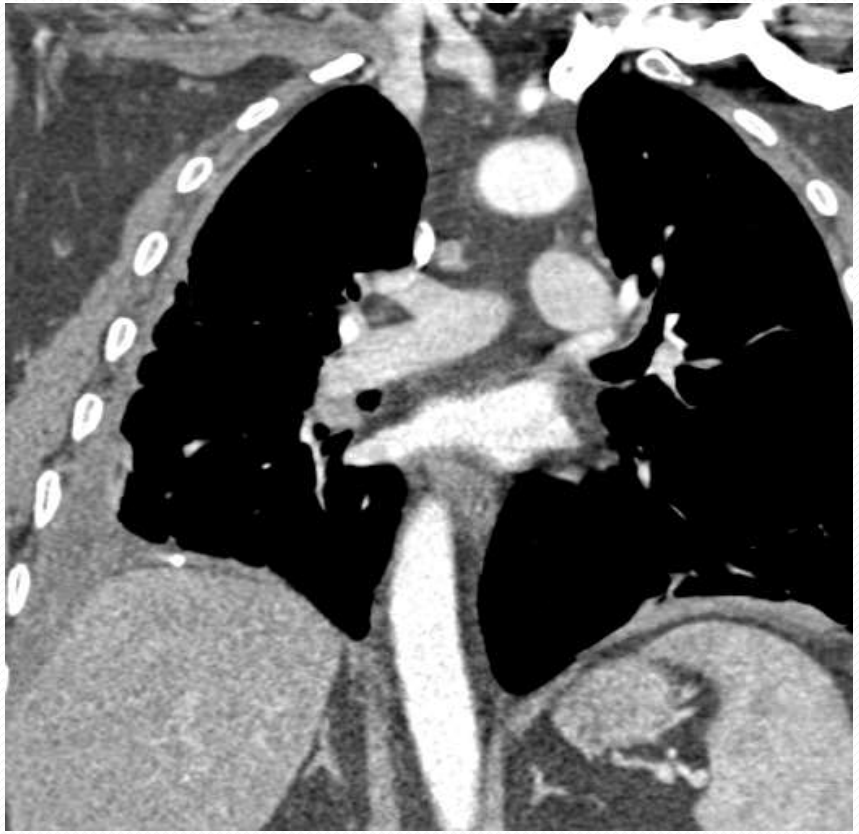
Benign Asbestos Pleural Effusion

- Exudative effusions.
- Usually quite small (<500 mL).
- May be unilateral or bilateral.
- Often transient and asymptomatic, and therfore not easy to diagnose.
- Most common abnormality seen with 10 years of exposure to asbestos.
- Used to be thought that they were a rare finding after a latent period of 20 years or so. This is now known *not* to be the case, with latent periods well in excess of 30 years being seen.

Benign Asbestos Pleural Effusion





Benign Asbestos Pleural Effusion

- Diagnosis of exclusion:

  1) A history of asbestos exposure.

  2) No other cause for the effusion.

  3) No evidence of malignancy within 3 years of detecting the effusion.

- When there is a large effusion with a long latent period, the clinician should be less willing to accept the diagnosis without thorough investigation and follow up.

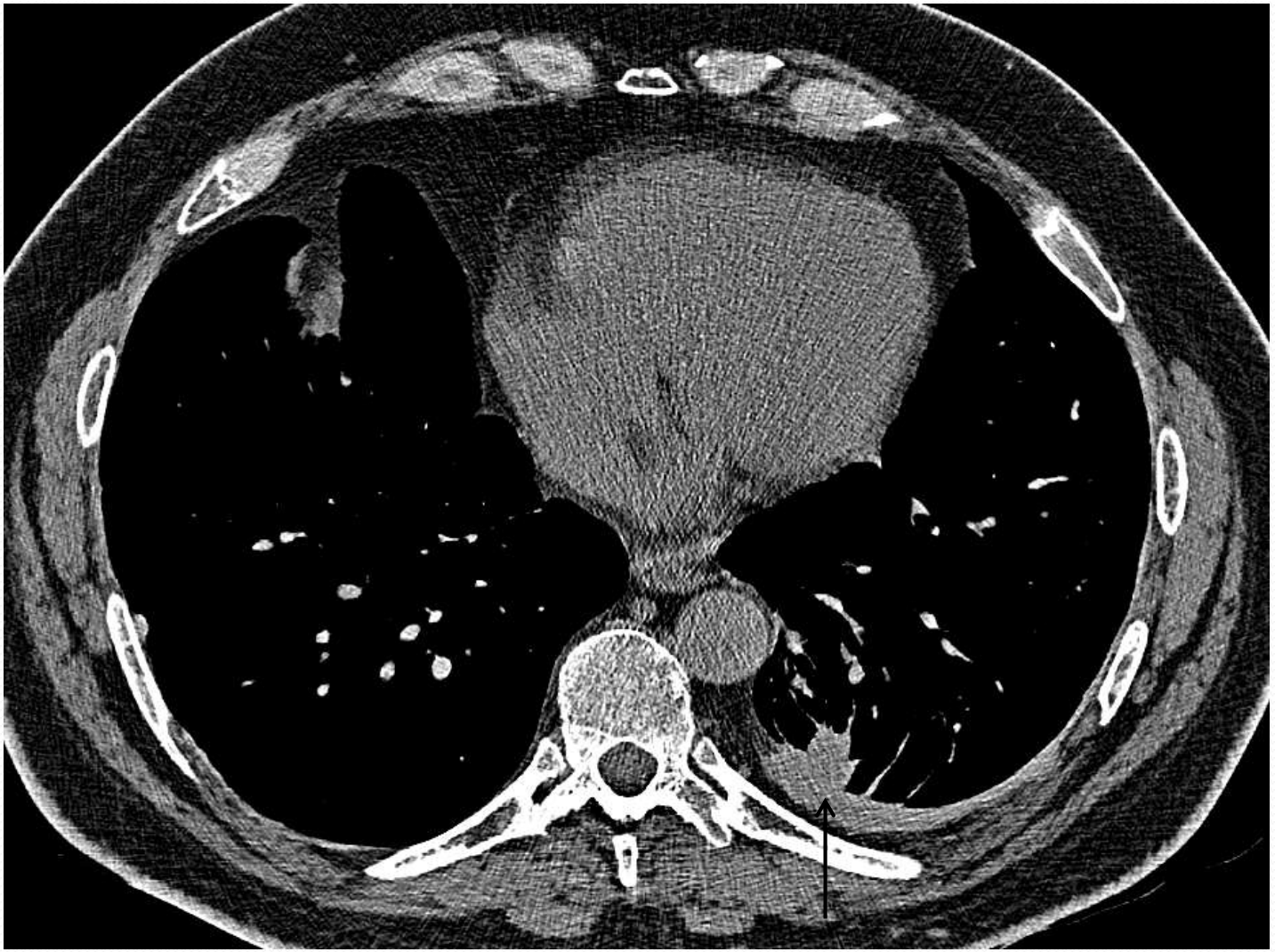
Rounded Atelectasis and “Crow’s feet”

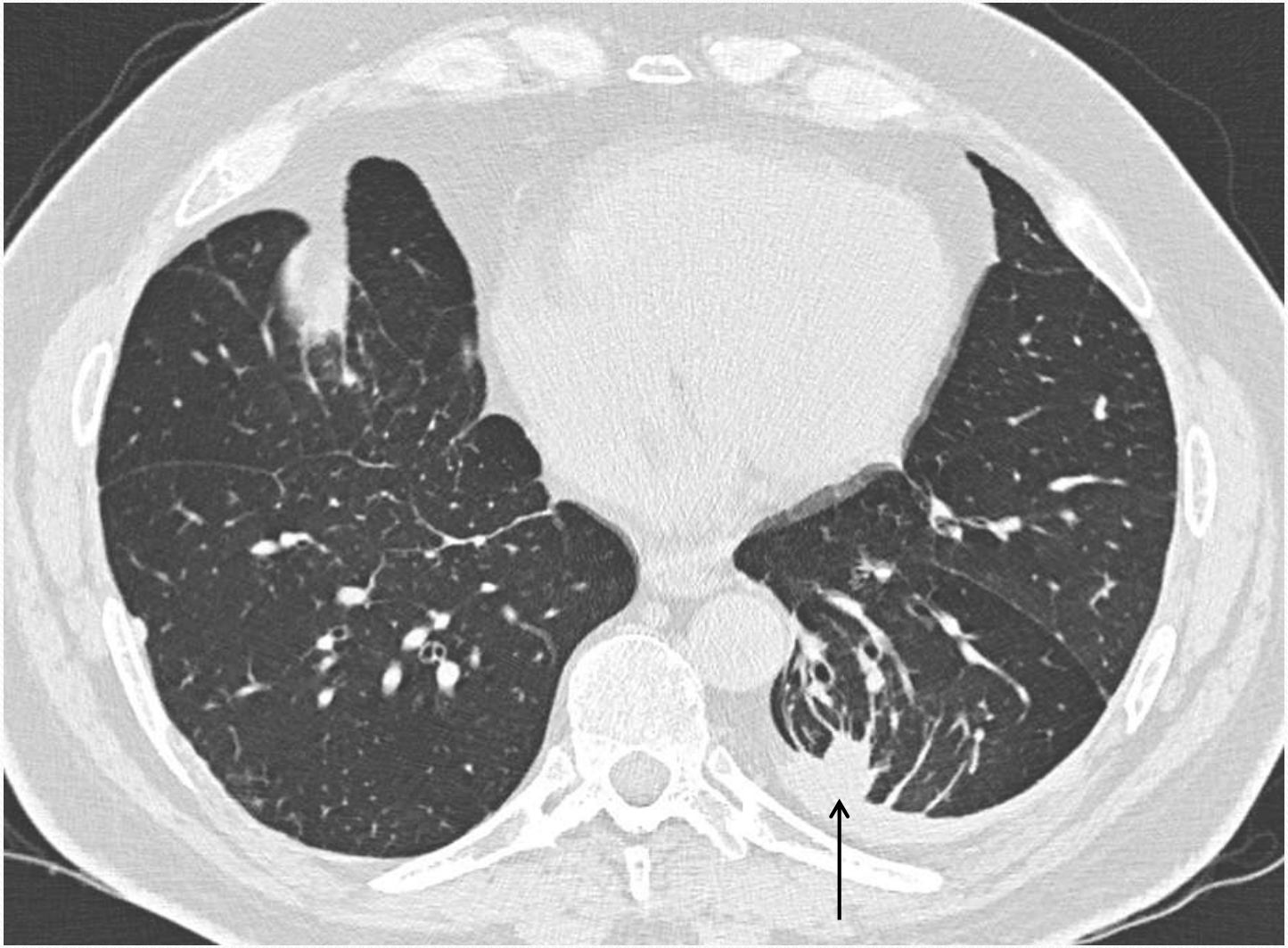
- With all causes of exudative effusions and fibrothoraces, the adjacent lung parenchyma is frequently abnormal.
- Rounded atelectasis/ folded lung:

  2-7cm intrapulmonary mass.

  Contiguous with underlying diffuse pleural thickening.

  Associated with volume loss (tethering of bronchovascular structures gives rise to a curved “comet tail” appearance).





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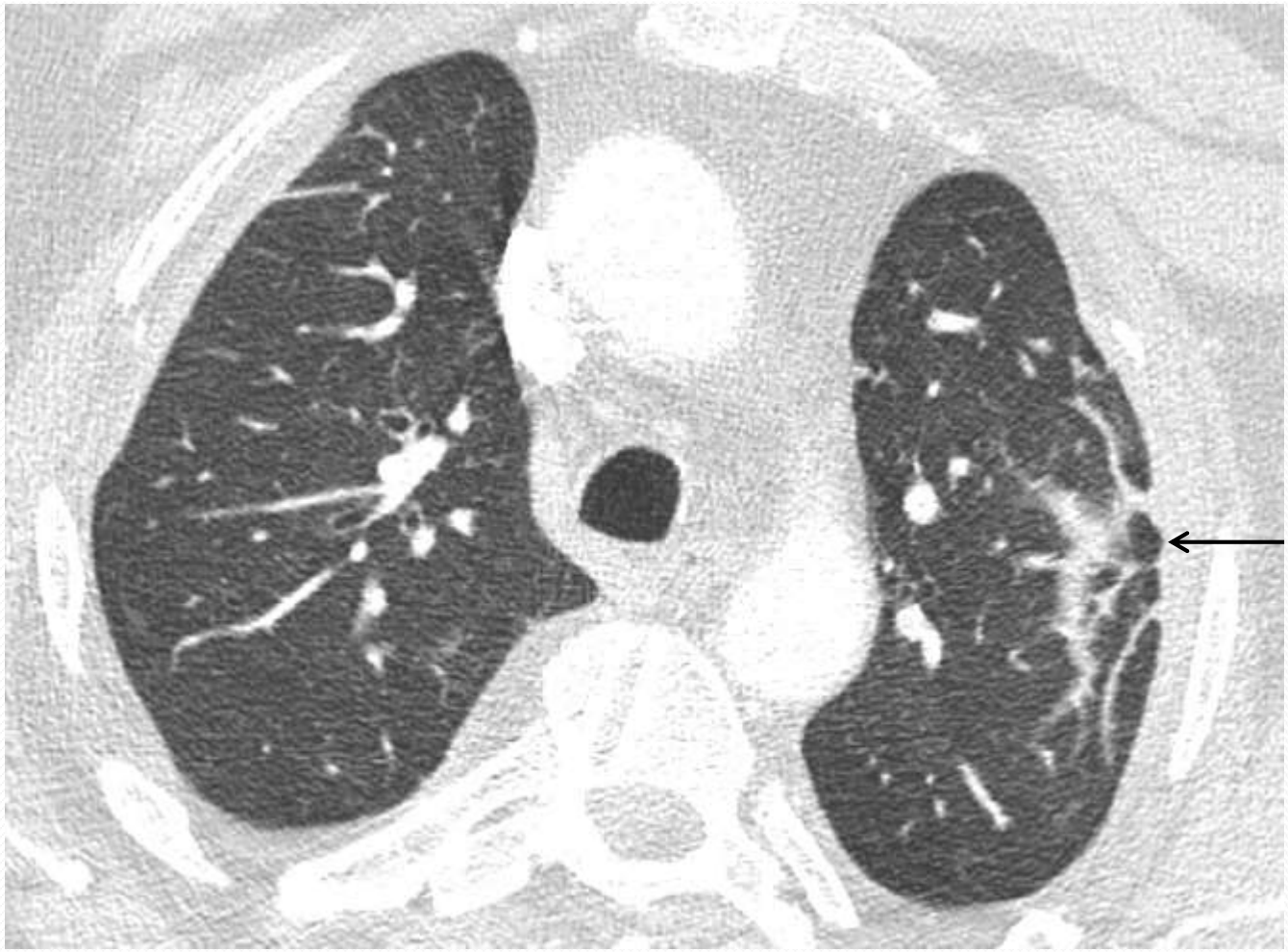
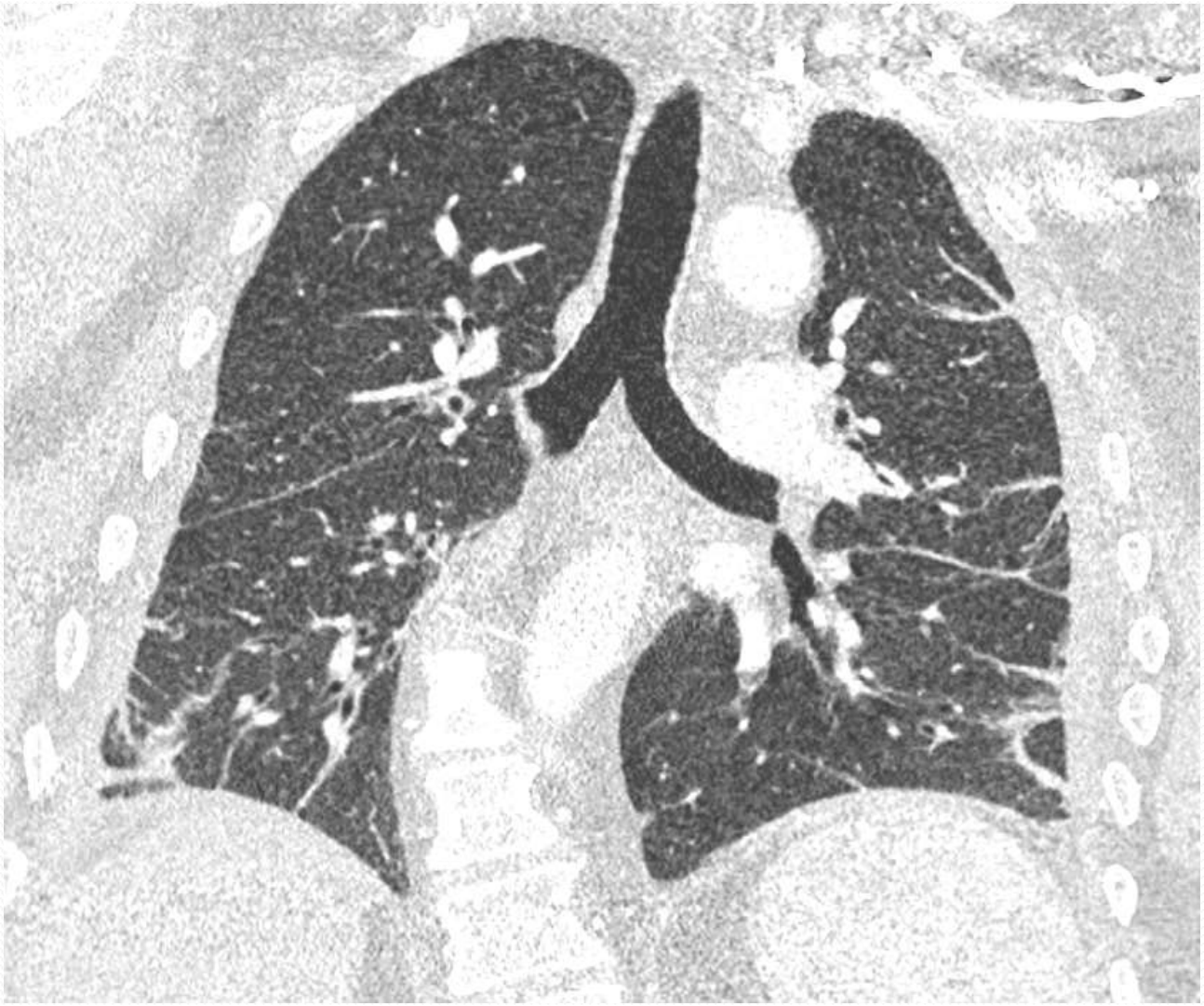
  Contiguous with underlying diffuse pleural thickening.

  Associated with volume loss (thethering of bronchovascular structures gives rise to a curved “comet tail” appearance).
- **Linear atelectasis:**

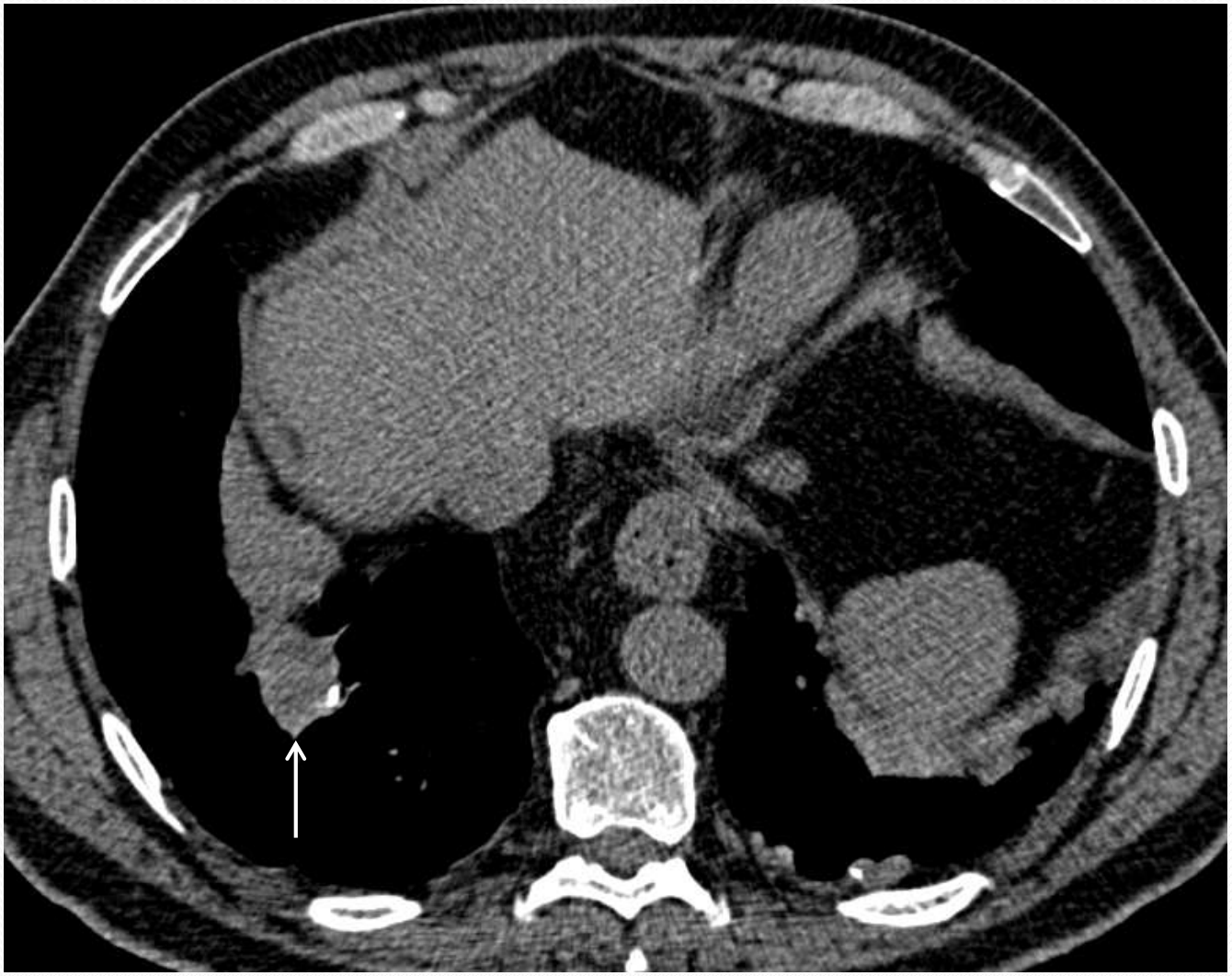
  Coarse parenchymal band shadows (“crow’s feet).

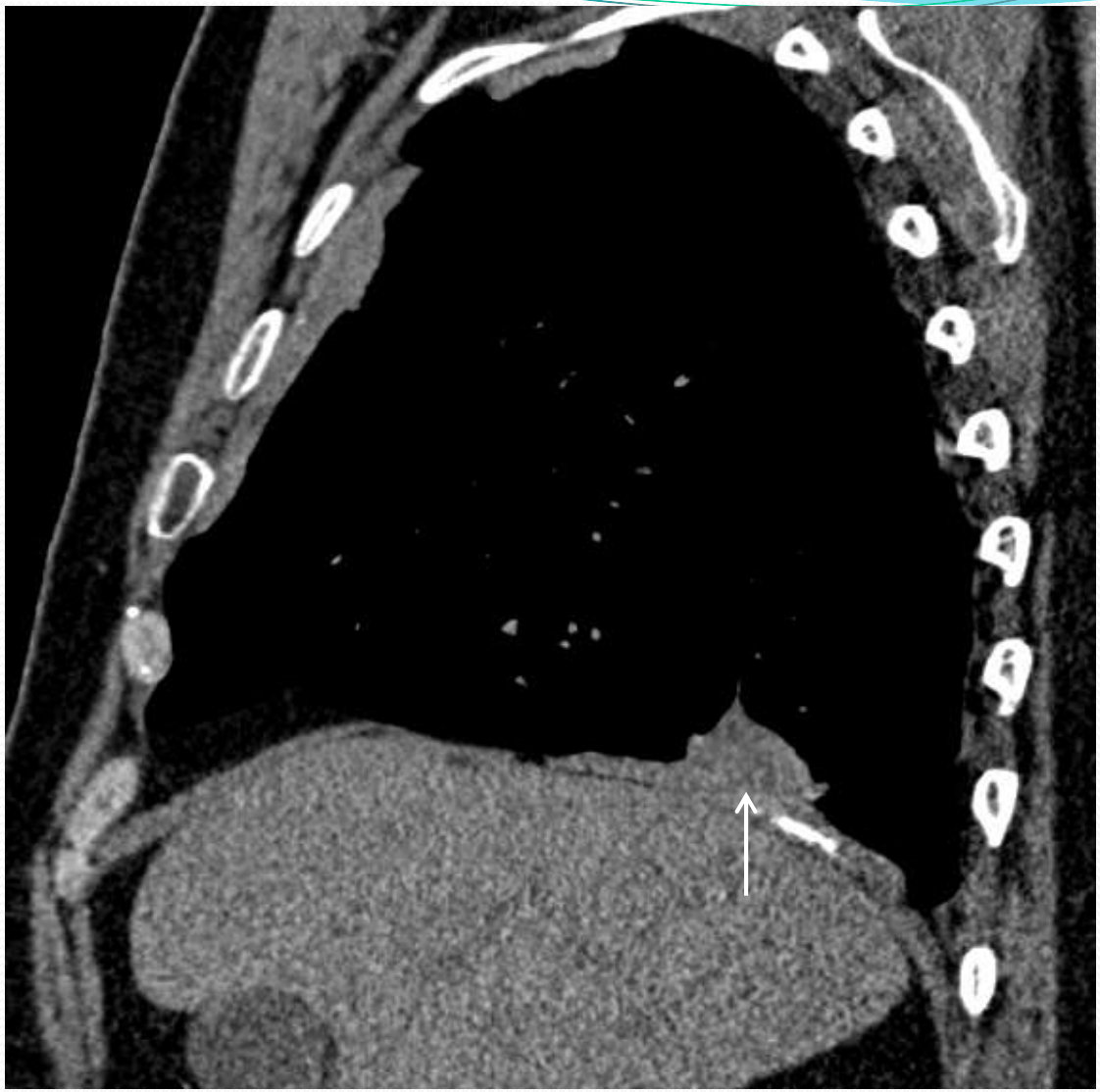


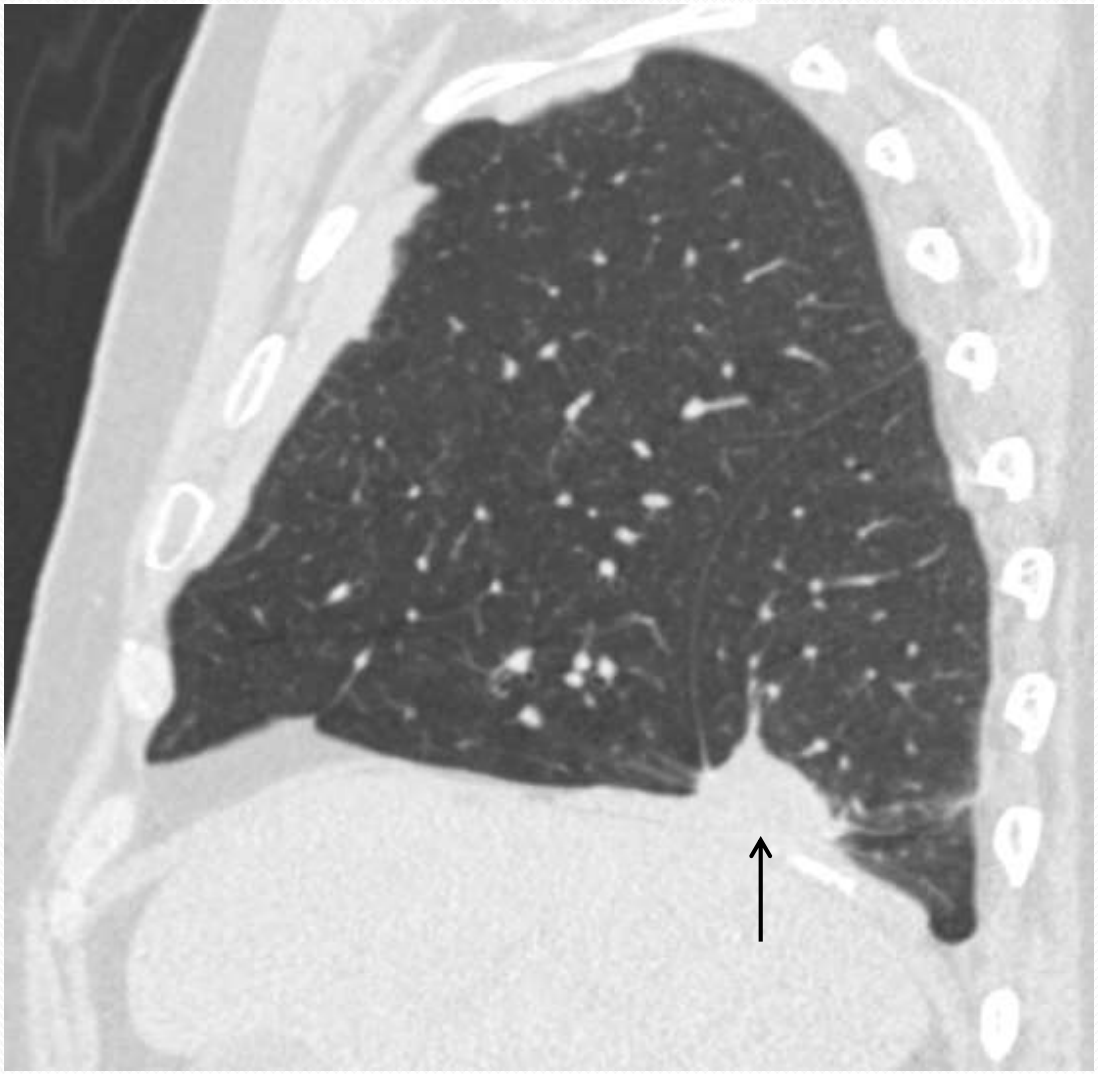


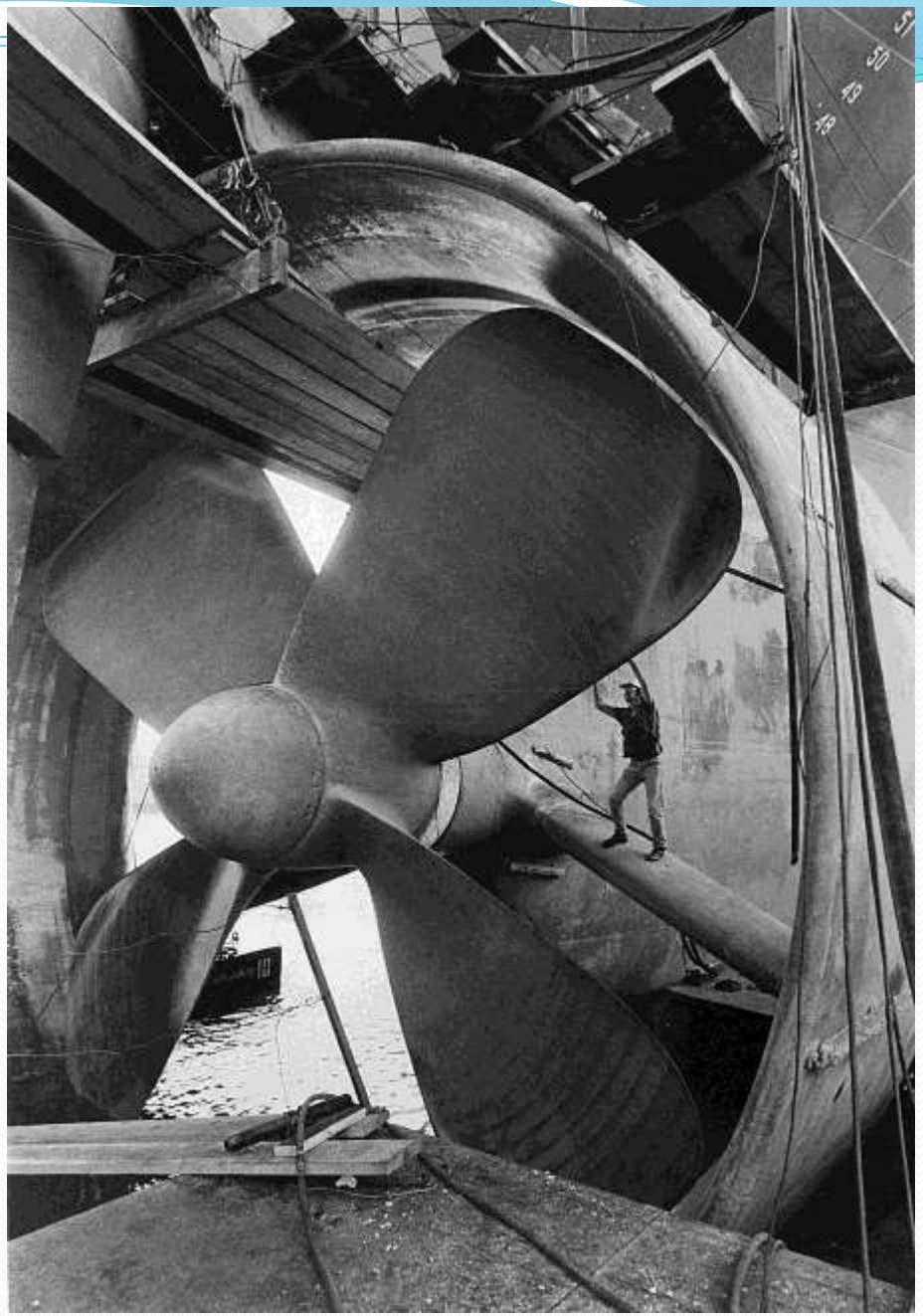
Focal Visceral Pleural Fibrosis

- ??? Focal areas of pleural thickening that do not meet the criteria of diffuse pleural thickening and do not have the appearance of simple pleural plaques???.
- When asbestos fibres reach the pleura, it seems that probable that host factors play a vital role in determining *what type* of reaction occurs.
- Most commonly parietal pleural plaques result. Occasionally an exudative effusion will be induced which may evolve into diffuse pleural thickening. Sometimes it seems likely that there is a focal area of visceral fibrosis without an effusion. This leads to a focal area of pleural thickening which isn't well circumscribed, and is associated with underlying lung parenchymal abnormalities and volume loss. These patients, like those with diffuse pleural thickening, may have restrictive pulmonary function defects.

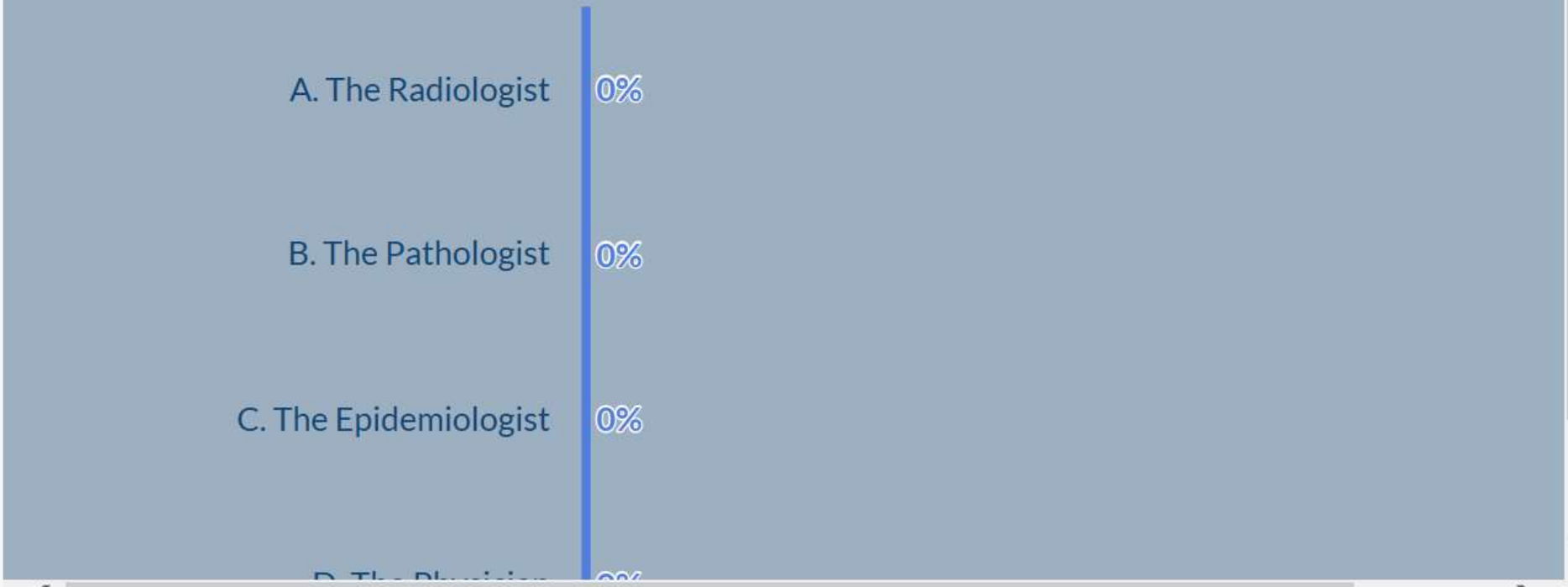






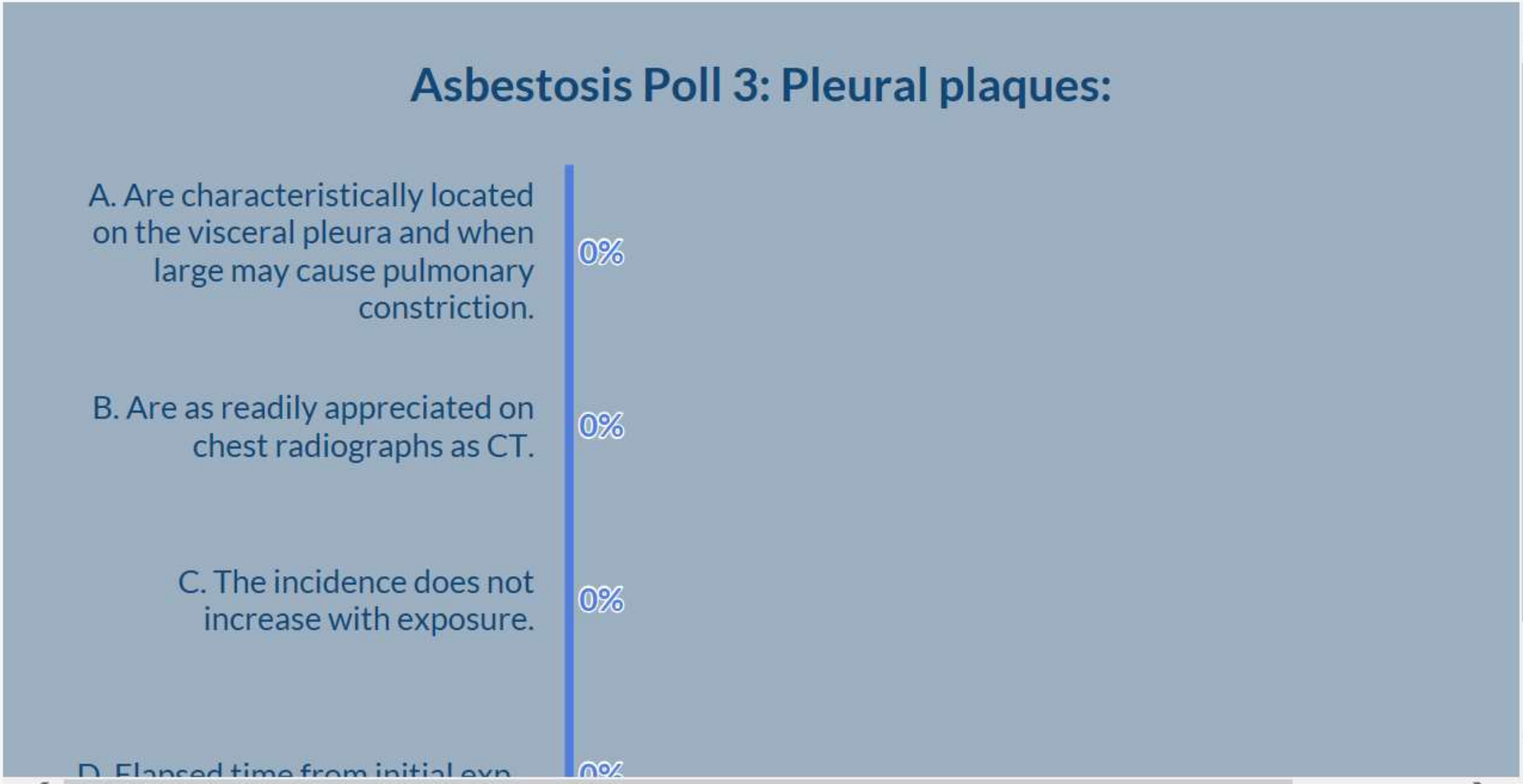


Asbestosis Poll 2: When litigating occupational asbestos exposure cases, the most important expert witness is:



Source: https://api.cvent.com/polling/v1/api/polls/spdad90z

Asbestosis Poll 3: Pleural plaques:

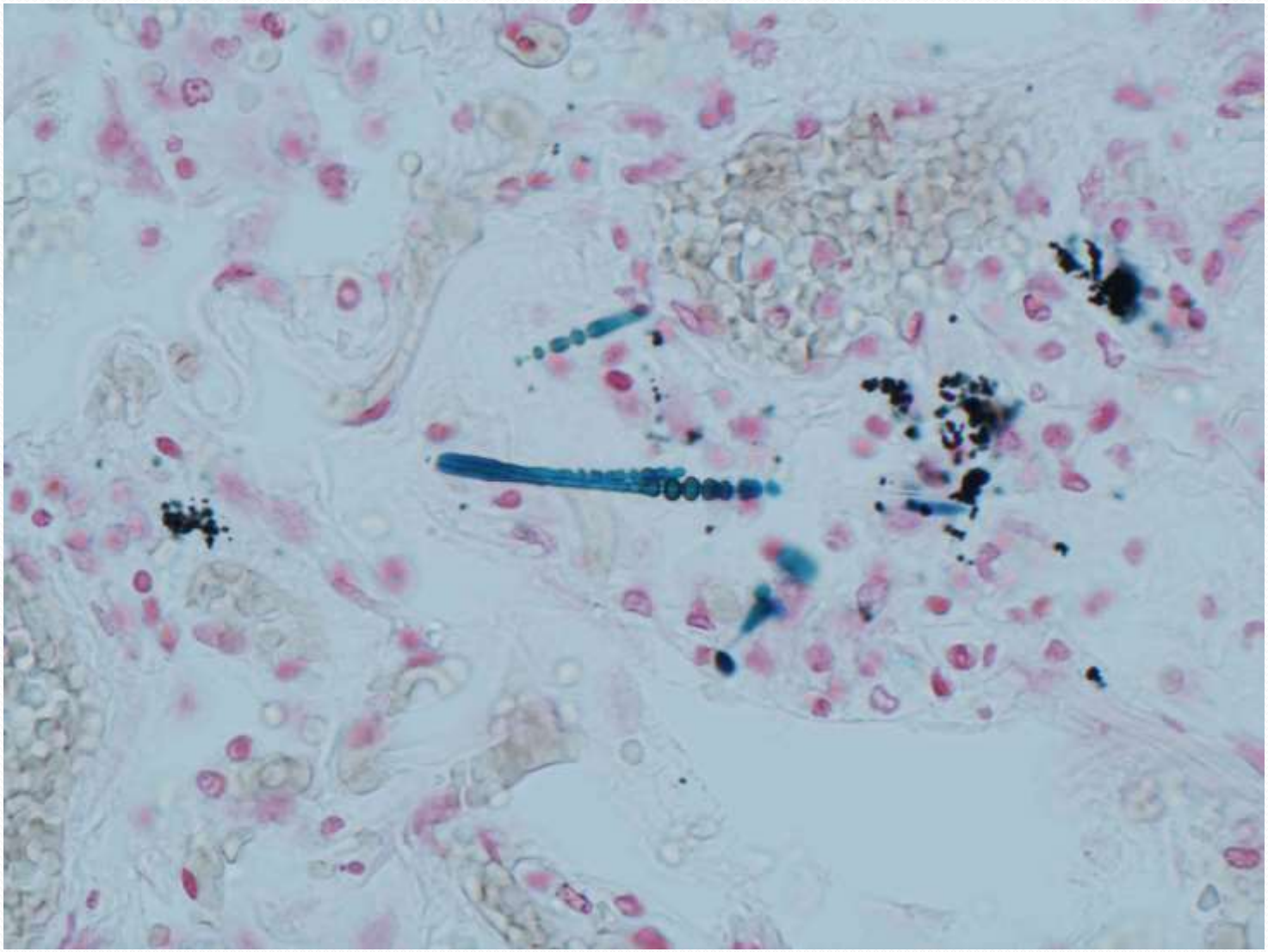


Source: https://api.cvent.com/polling/v1/api/polls/sp-jsfetg

Edit

Asbestosis

- Diffuse parenchymal fibrosis secondary to inhalation of asbestos fibres.
- Related to cumulative dust exposure.
- Usually associated with prolonged and heavy exposures. The sheer volume of fibres overwhelms the host clearing mechanisms, fibres enter the interstitium and provoke an inflammatory and fibrotic reaction.
- Consequently there is usually a large number of asbestos bodies and high fibre counts in tissues samples.



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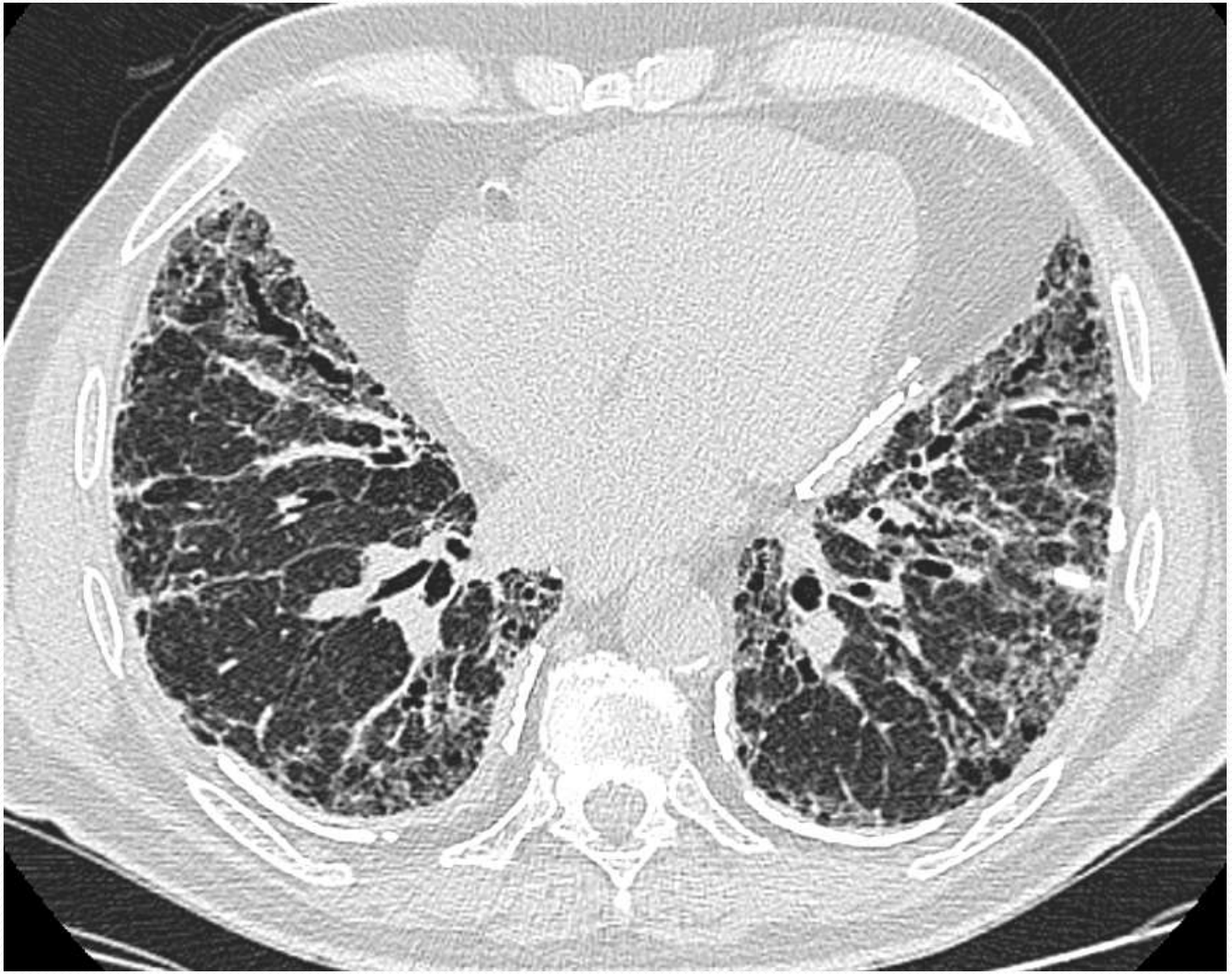
- Consequently there is usually a large number of asbestos bodies and high fibre counts in tissues samples.
- **Because it is the cumulative dose that is crucial, the time interval between initial exposure and evidence of asbestosis is variable. 20-30 years is usual, but intense exposures can cause asbestosis in just 3 years.**

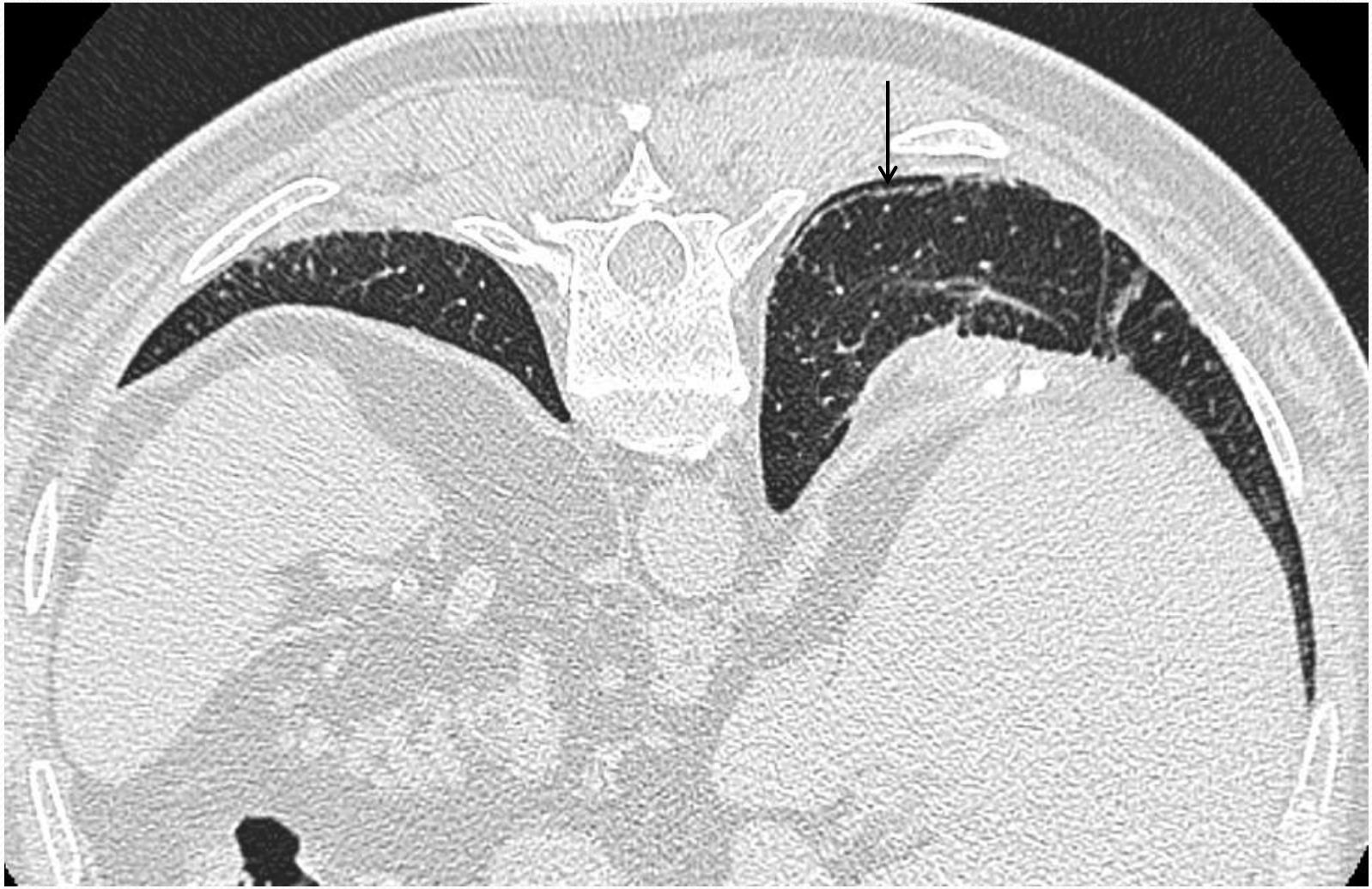
Asbestosis

- Fibrosis is most prominent in the subpleural regions in the lower zones, and can range from mild reticulation to obvious honeycomb formation and architectural distortion.



Asbestosis and Idiopathic Pulmonary Fibrosis

- There are no radiographic *features of fibrosis* that reliably distinguish asbestosis from idiopathic pulmonary fibrosis, although there are some features which favour one or other.
- Upper zone involvement, honeycomb formation and ground glass opacity favour IPF but does not exclude asbestosis.
- Subpleural crescents and parenchymal bands favour asbestosis but do not exclude IPF.
- In the presence of *pleural plaques*, lower zone fibrosis is usually taken to represent asbestosis.



Asbestosis and Idiopathic Pulmonary Fibrosis

- Pleural plaques and IPF are both common conditions, so clearly there is no reason why a patient cannot have IPF with coincidental plaques.
- Clinical, physiological and radiographic evidence of rapid progression more typical of IPF.
- What is rapid progression? Demonstrable progression of asbestosis over a fairly short timescale of 2-3 years is occasionally seen.

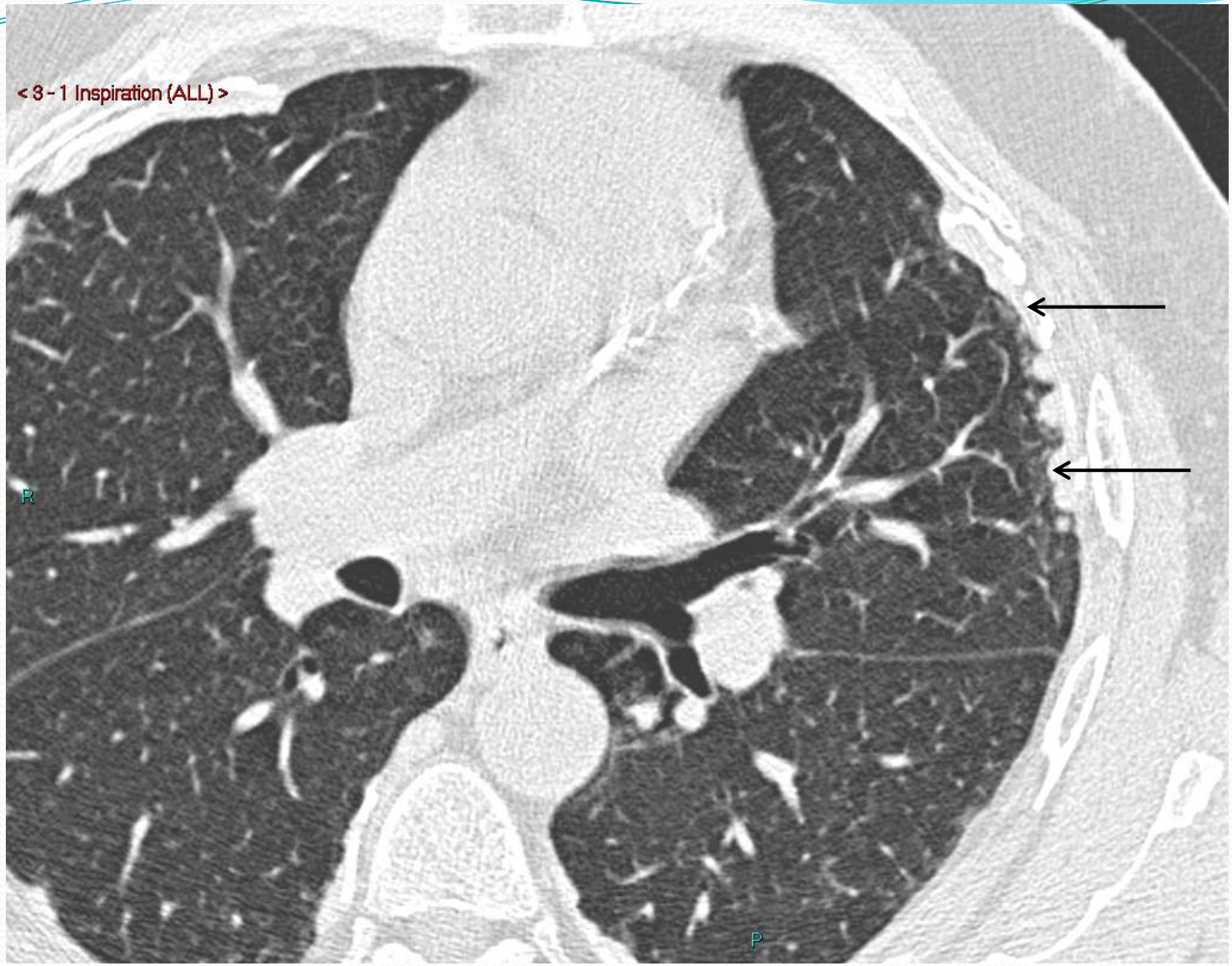
Predominantly Upper Zone Fibrosis

- Is it safe to exclude asbestosis when there is predominantly upper zone fibrosis?
- Hillerdal variant.
- (In all but one case, the fibrosis wasn't confined to the upper zones. There was also pleural and parenchymal fibrosis affecting all zones.)

Minor/Subtle Fibrotic Features

- “Hairy plaque”:

  Fine dot-like and linear opacities in the parenchyma adjacent to plaques. Due to localised fibrosis and atelectasis resulting from friction caused by the lung moving over the projecting plaque. Also seen where spinal osteophytes project into the lung parenchyma.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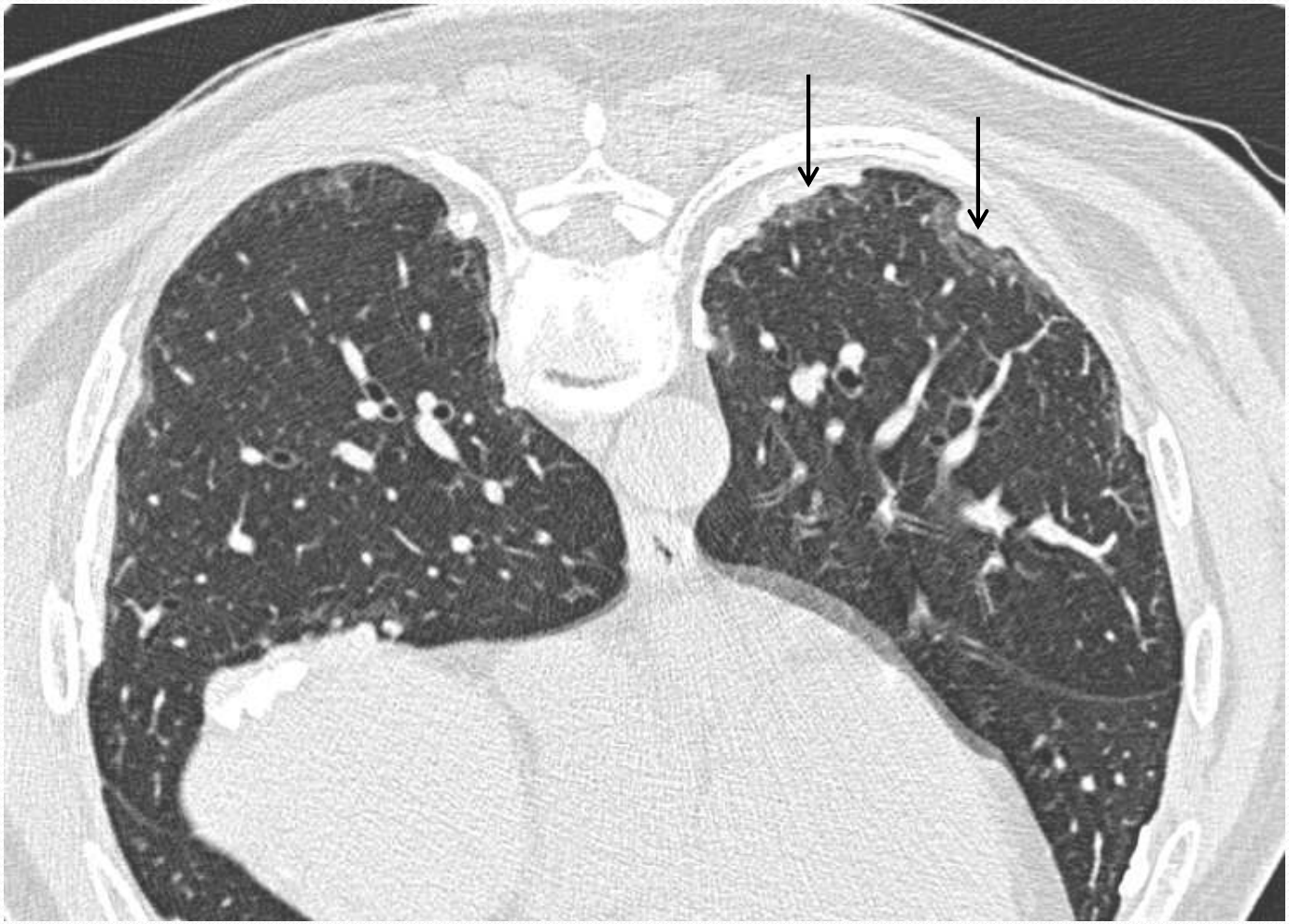
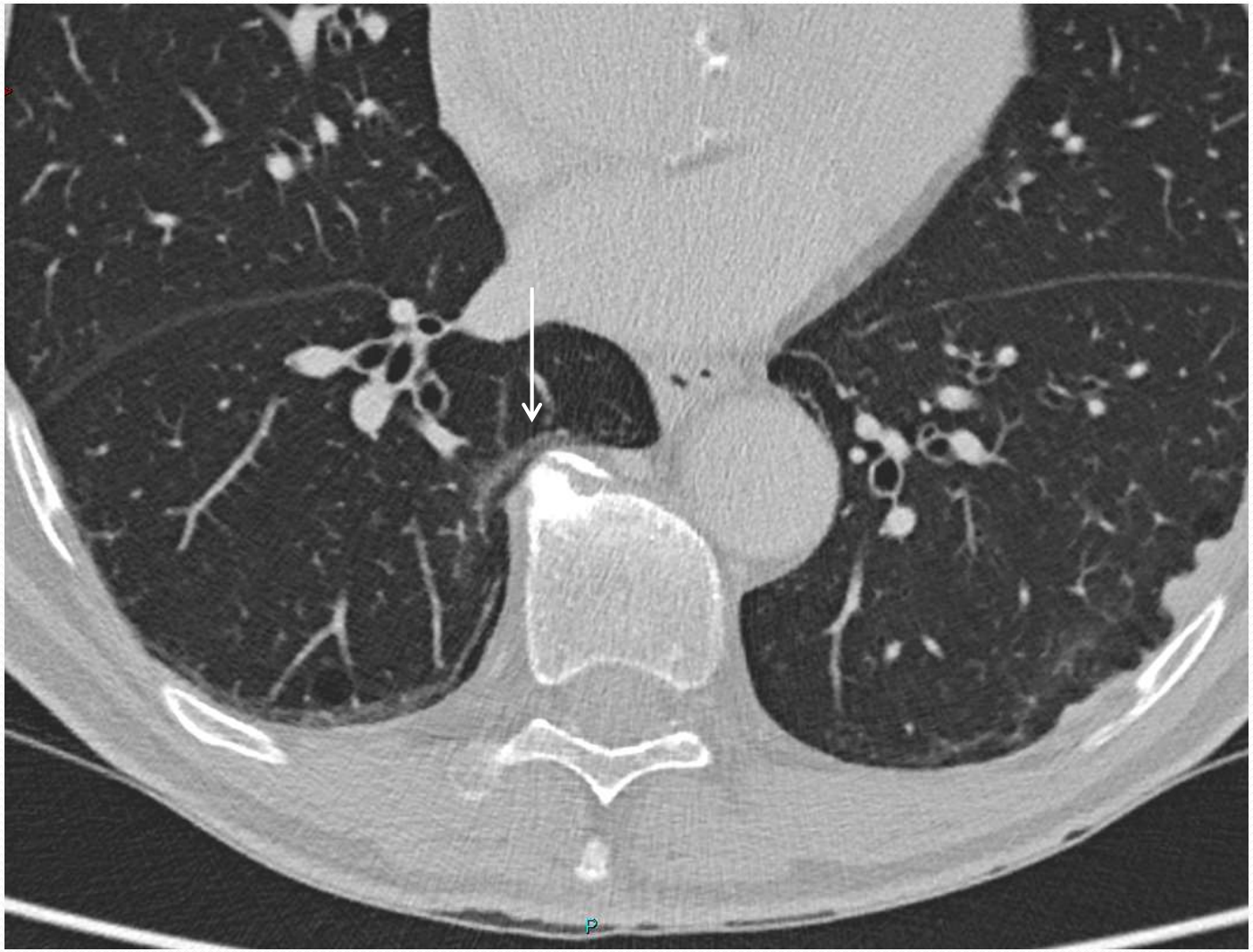




Minor/Subtle Fibrotic Features

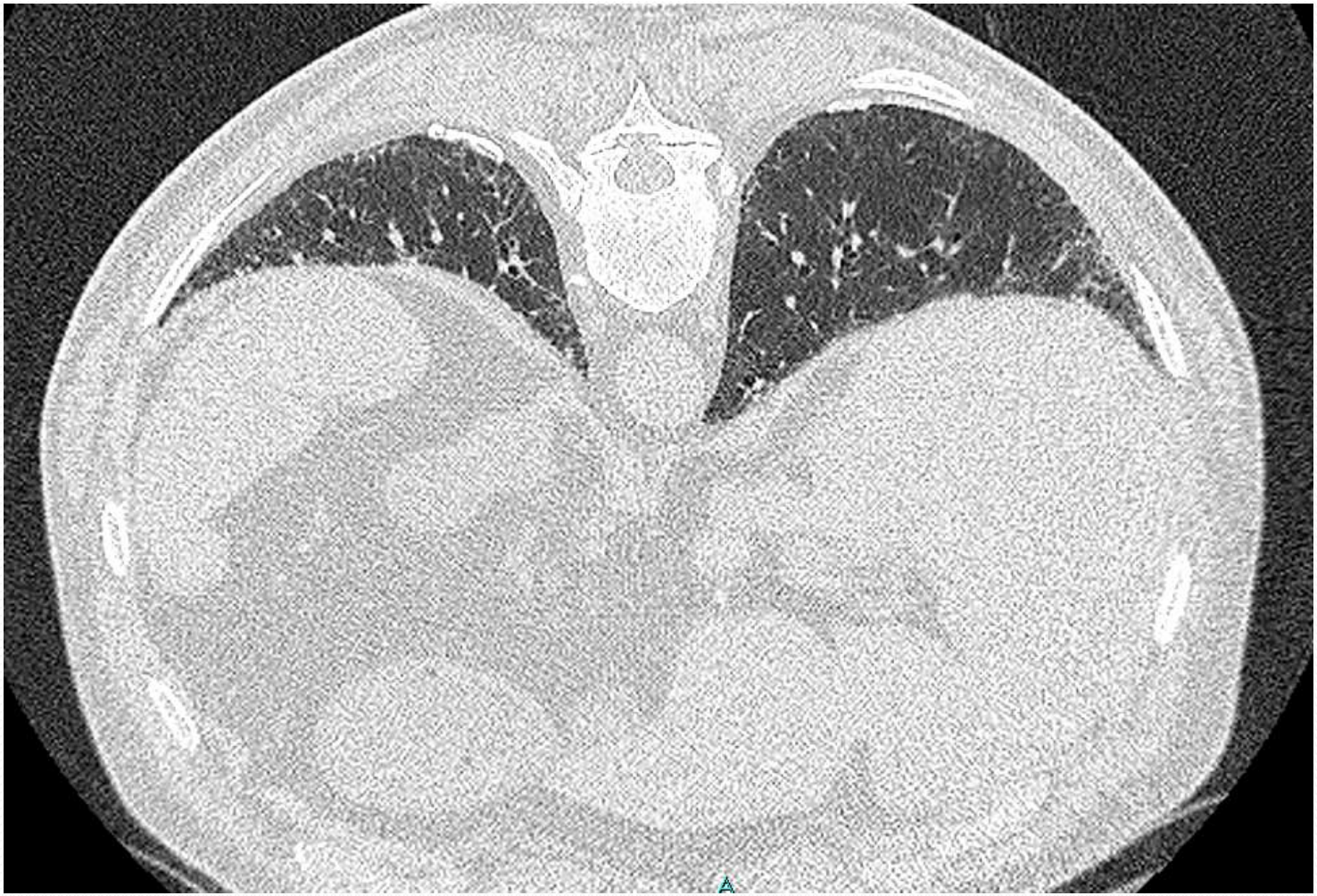
- Differential diagnosis of subtle, subpleural lower zone parenchumal opacities:

  Mild asbestosis

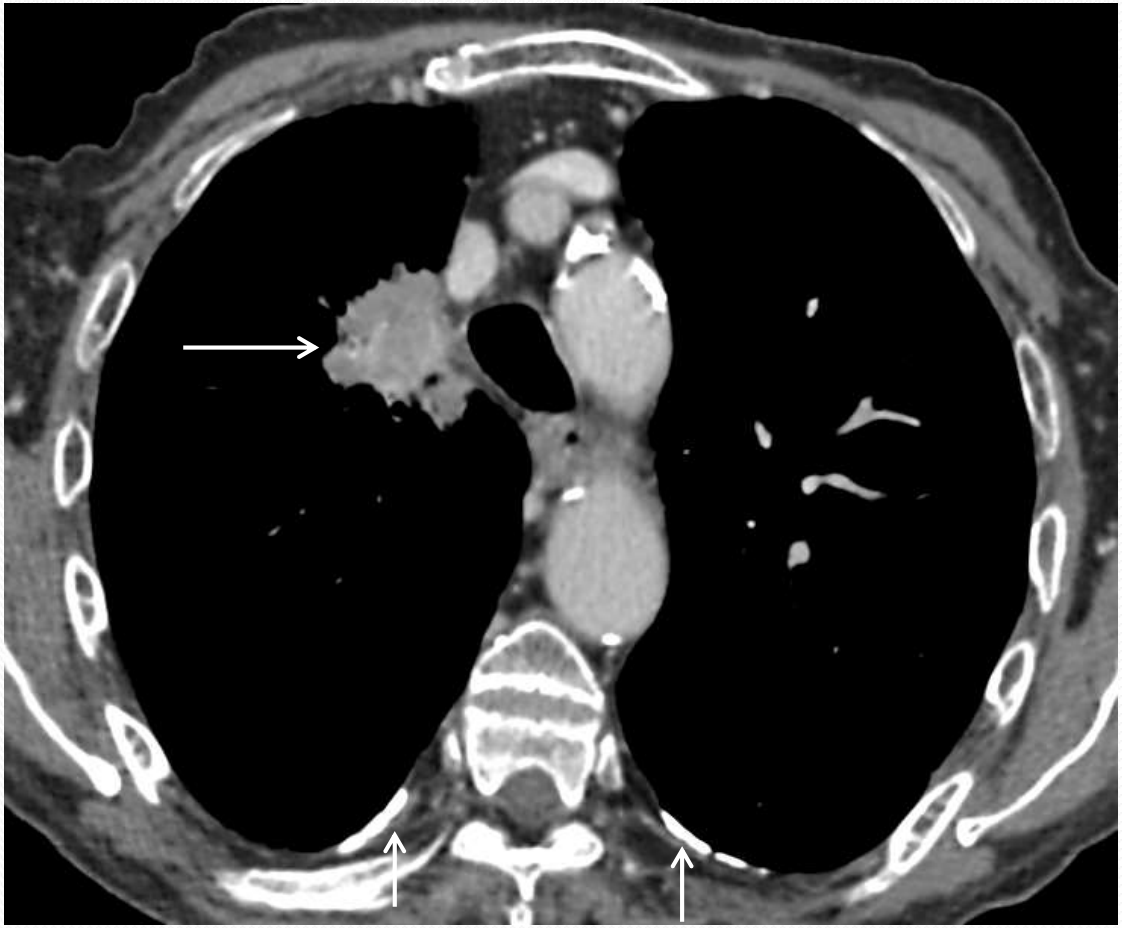
  Mild fibrosis due to another aetiology

  Senescent lung

  Bilateral post inflammatory scarring



Lung Cancer



Lung Cancer

- There is no doubt about the relationship between asbestos exposure and bronchial carcinoma, especially in smokers.
- The exact relationship between plaques and bronchial carcinoma is difficult to pin down because two parallel processes are under comparison.
- *Occupationally* exposed individuals with plaques have a higher than expected incidence of bronchial carcinoma (even after accounting for all confounding variables). The relationship of bronchial carcinoma and plaques in *environmentally* exposed individuals is less clear.

Mesothelioma

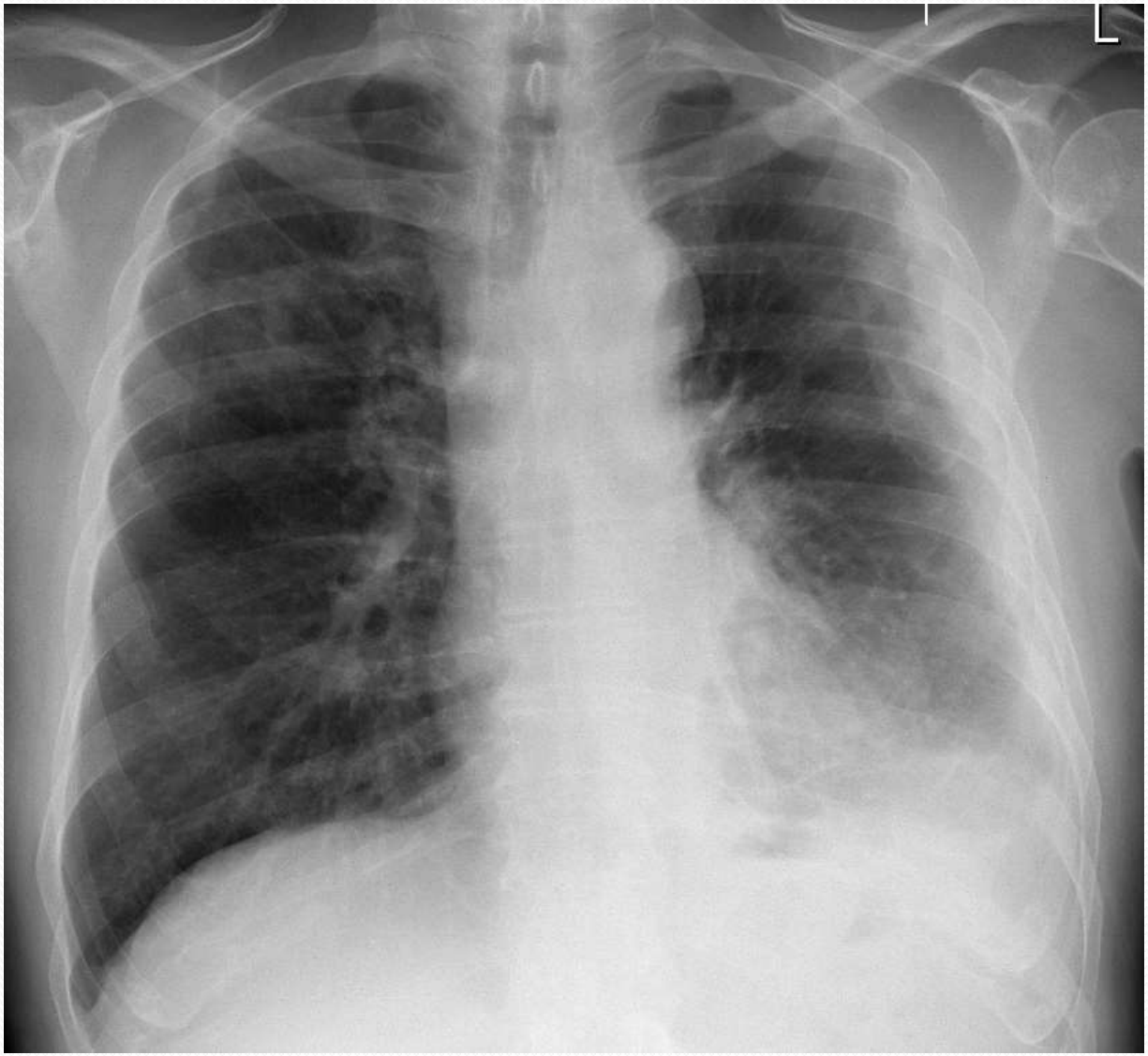
- Again, the relationship with plaques is difficult because two parallel processes are taking place.

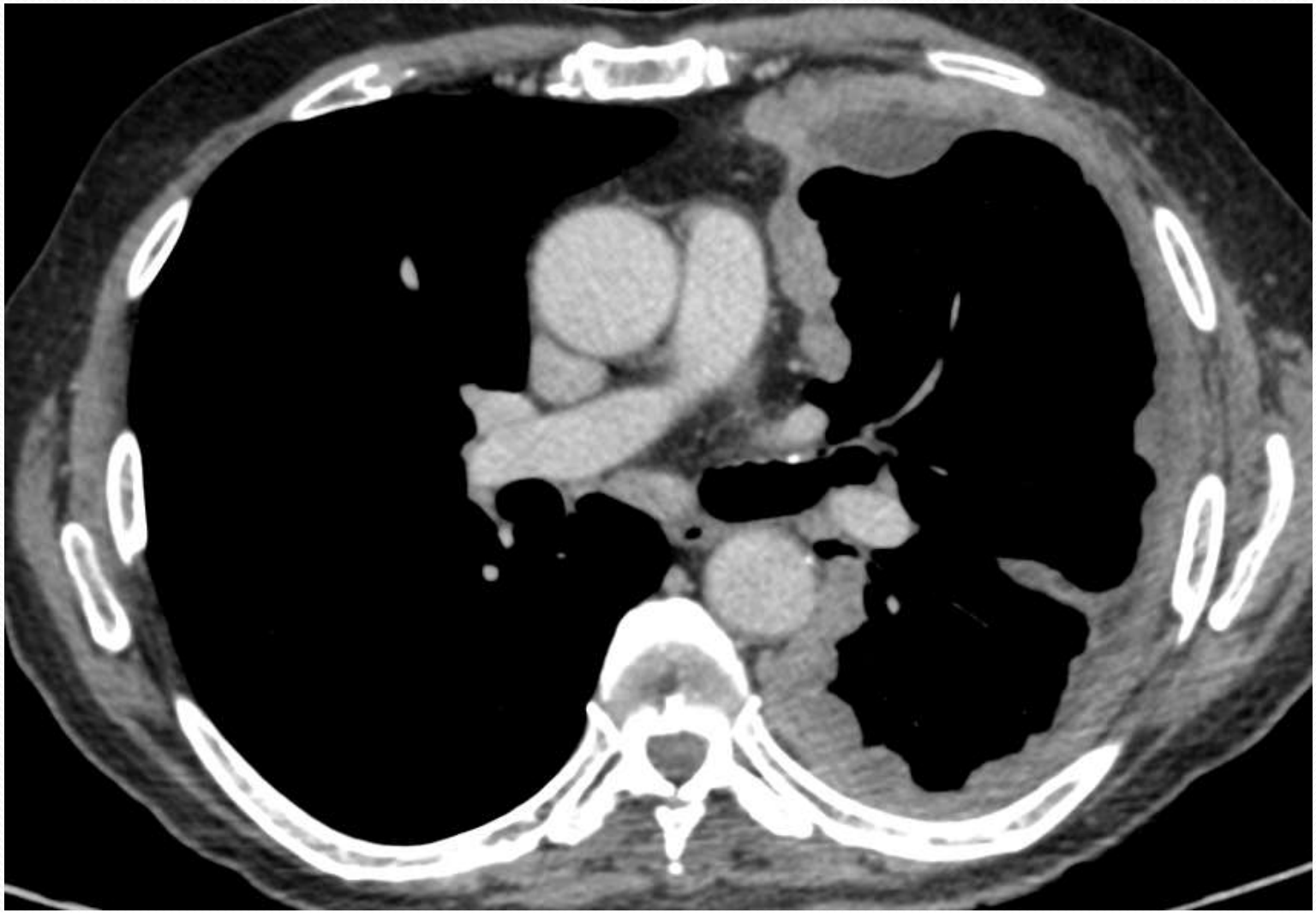
  Plaques do not degenerate into mesothelioma.

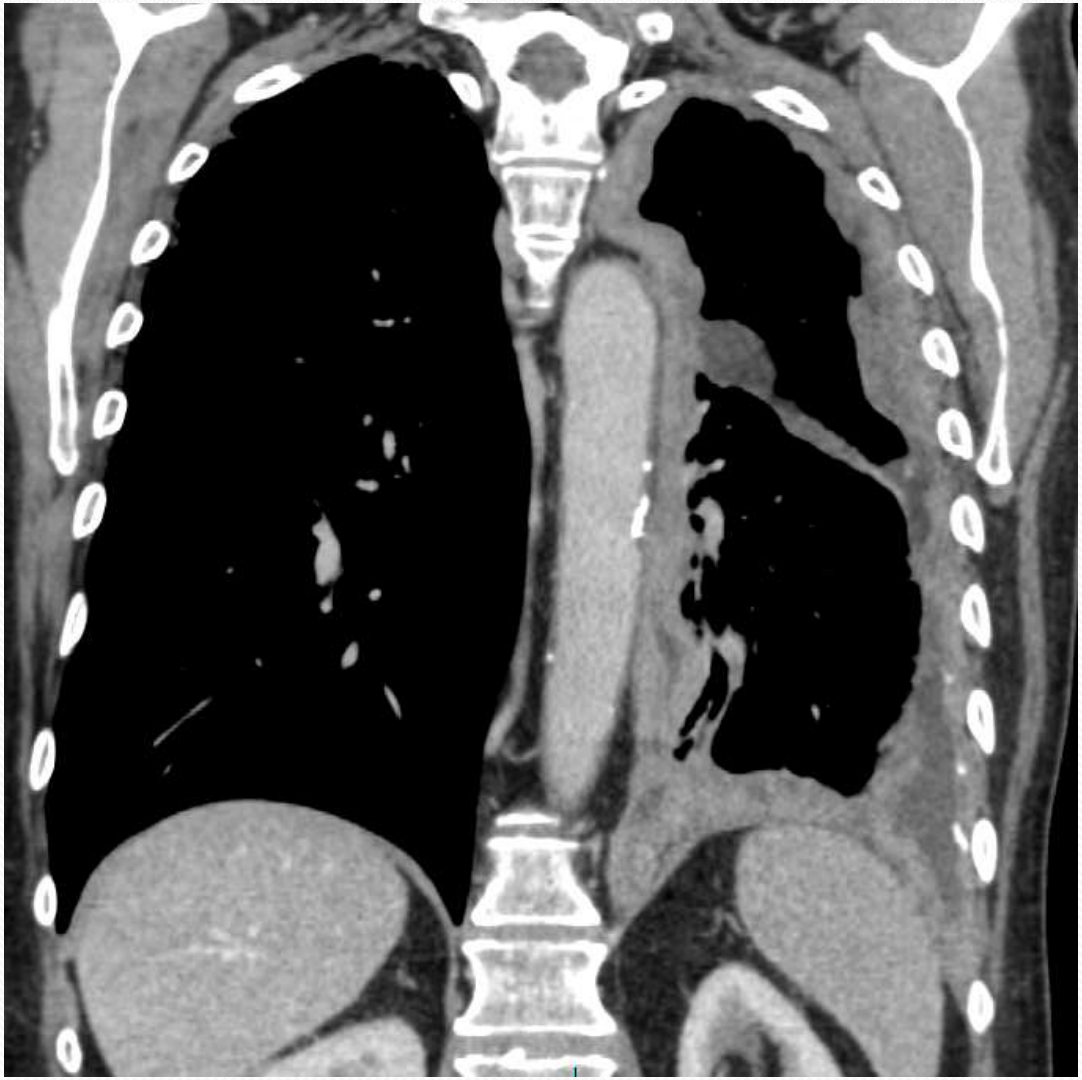
  Both in post mortem and CT studies, plaques are by no means invariably found in cases of mesothelioma (the prevalence of plaques is between 30-70%).
- Nonetheless, what is abundantly clear is that there is a close relationship between asbestos exposure and mesothelioma, which was first described in 1960.
- The asbestos exposure history is variable. In some the asbestos burden is very light, or overlaps with the general population.
- The interval between first exposure and diagnosis is in the order of 20-40 years (average > 40 years). Given the usage of asbestos in the 20th century it is not surprising that the worldwide incidence of mesothelioma in developed countries has steadily risen since 1960 (and is due to peak before 2030).

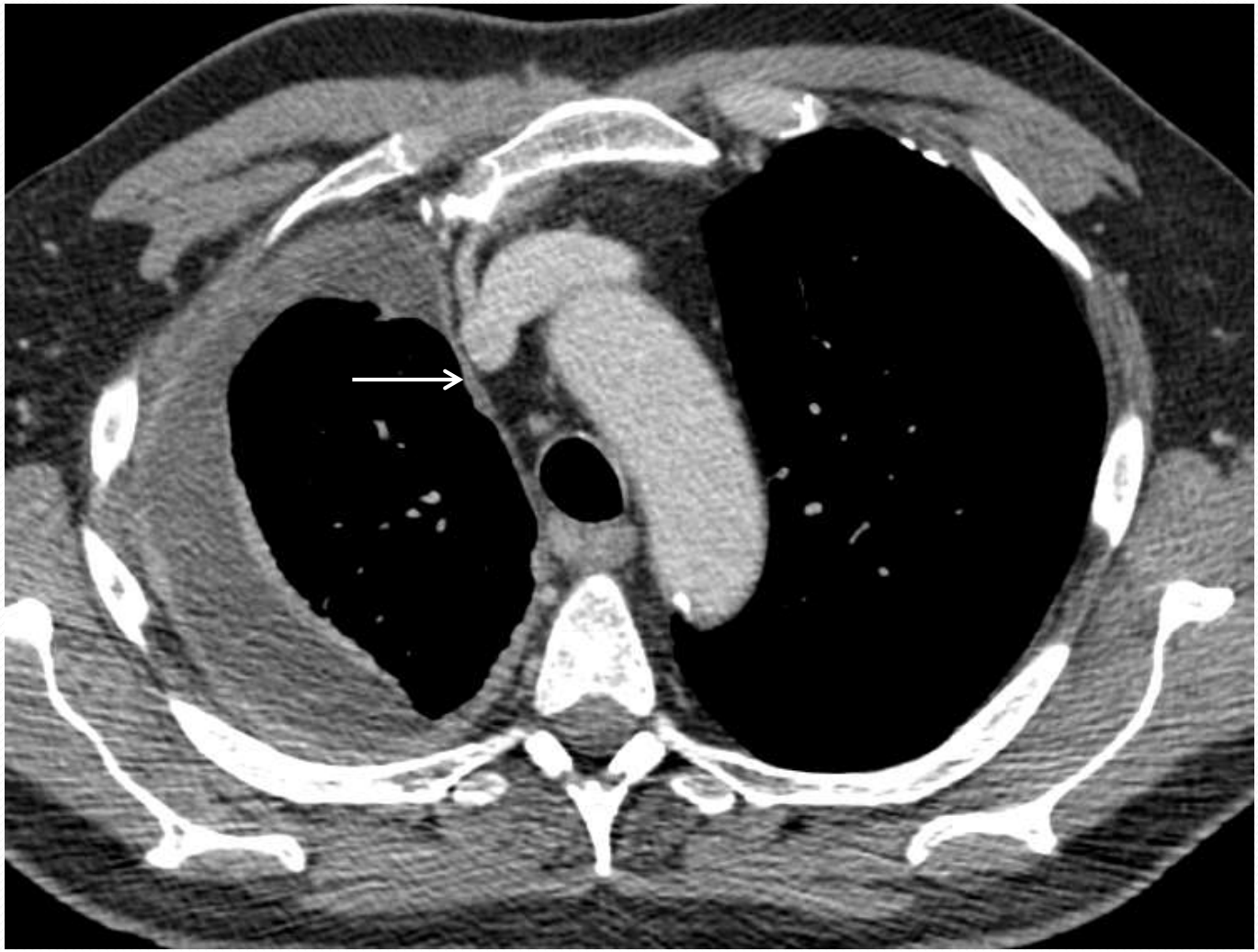
Diffuse Malignant Mesothelioma: Imaging Features

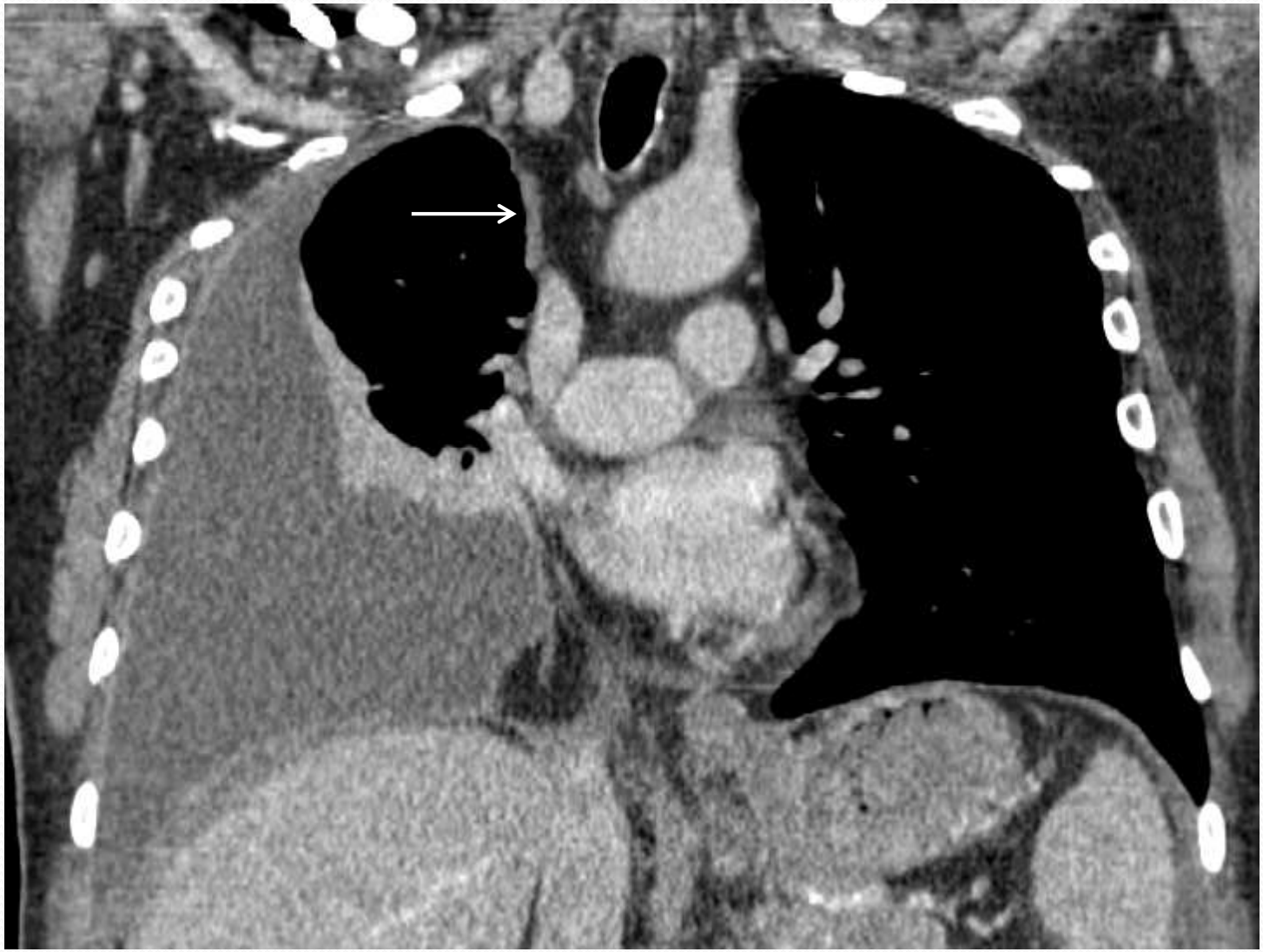
- Similar imaging features are seen on chest radiographs and CT, although CT is much more accurate in showing the full extent of the disease, and frequently allows a much more confident diagnosis to be made.
- Nodular pleural thickening, which may conglomerate to form a circumferential lobular sheet of tissue encasing the lung and extending into the fissures.
- Involvement of the mediastinal pleura.
- Pleural effusion, which may be large.
- Direct local invasion into the mediastinum and chest wall, and across the diaphragm into the upper abdomen.
- Metastases to the lung, mediastinal nodes and extrathoracic organs.

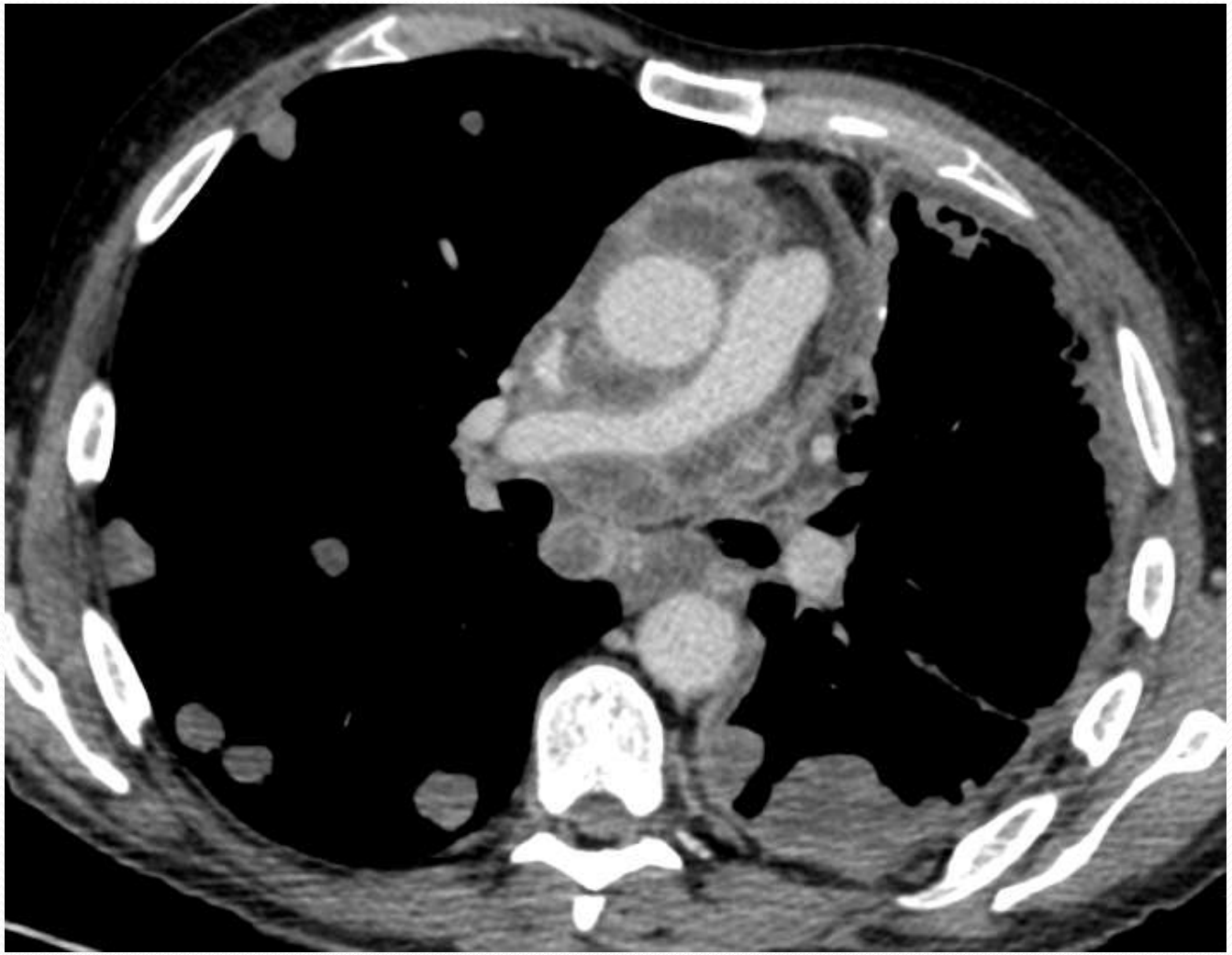










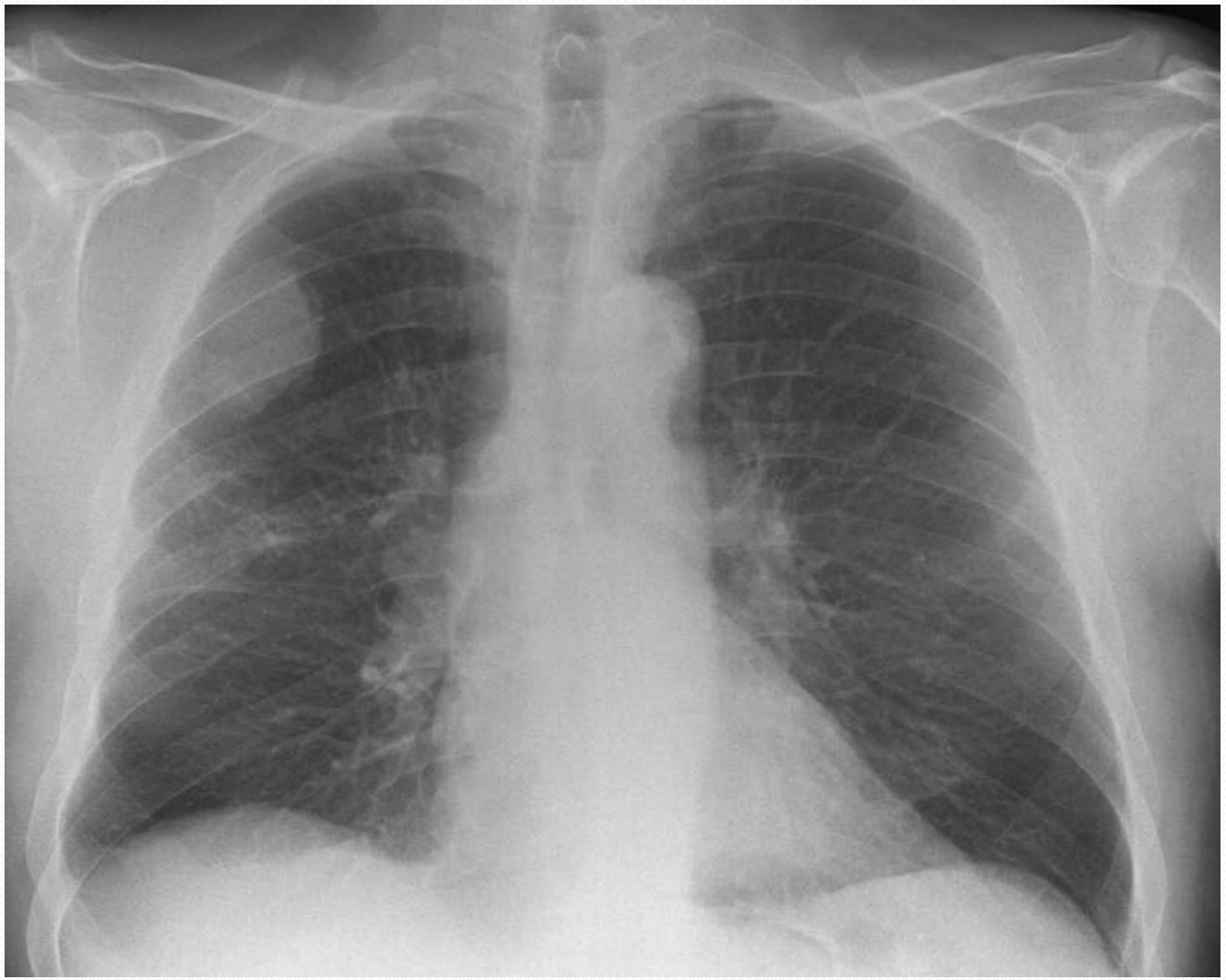


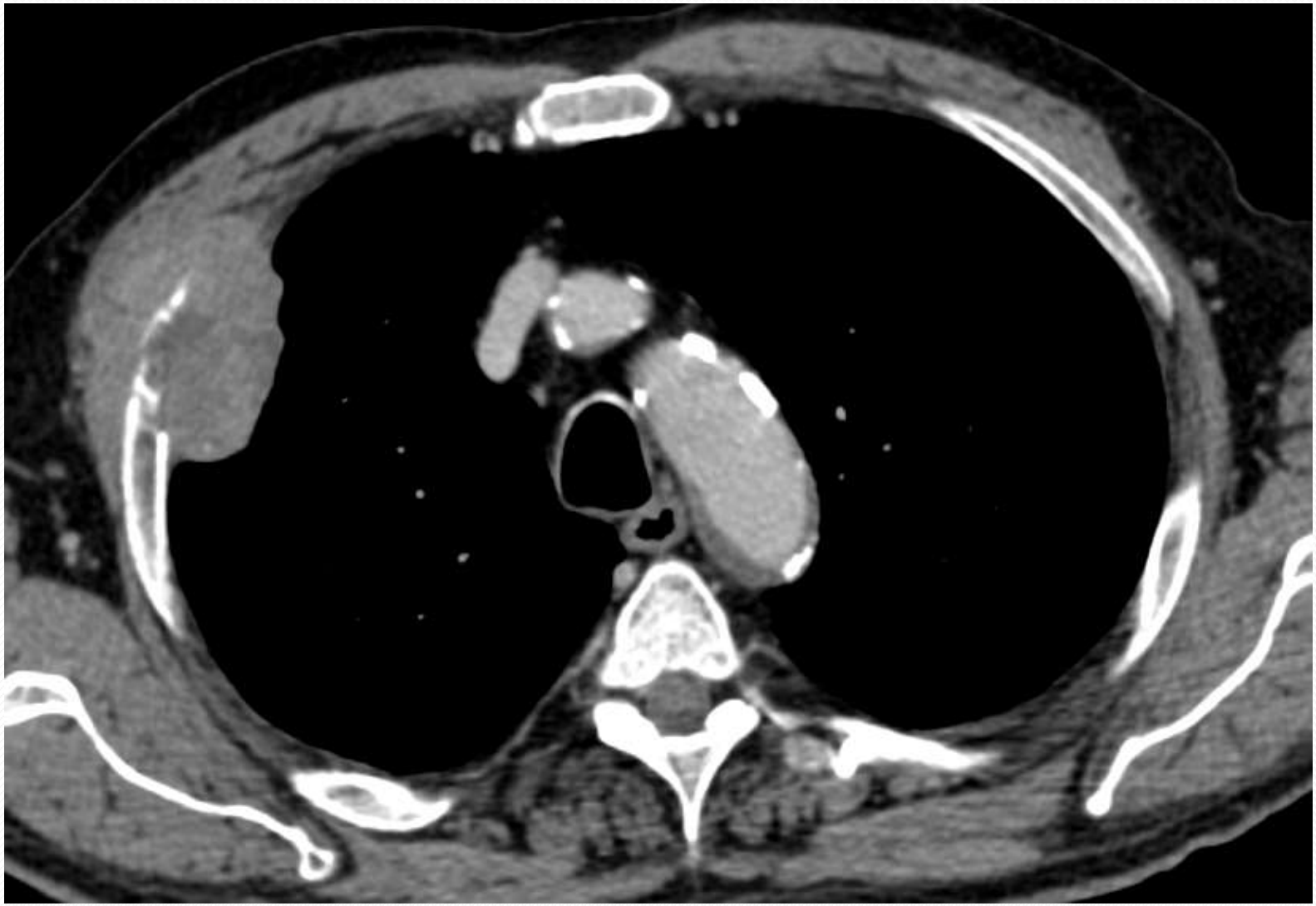
Solitary Malignant Mesothelioma

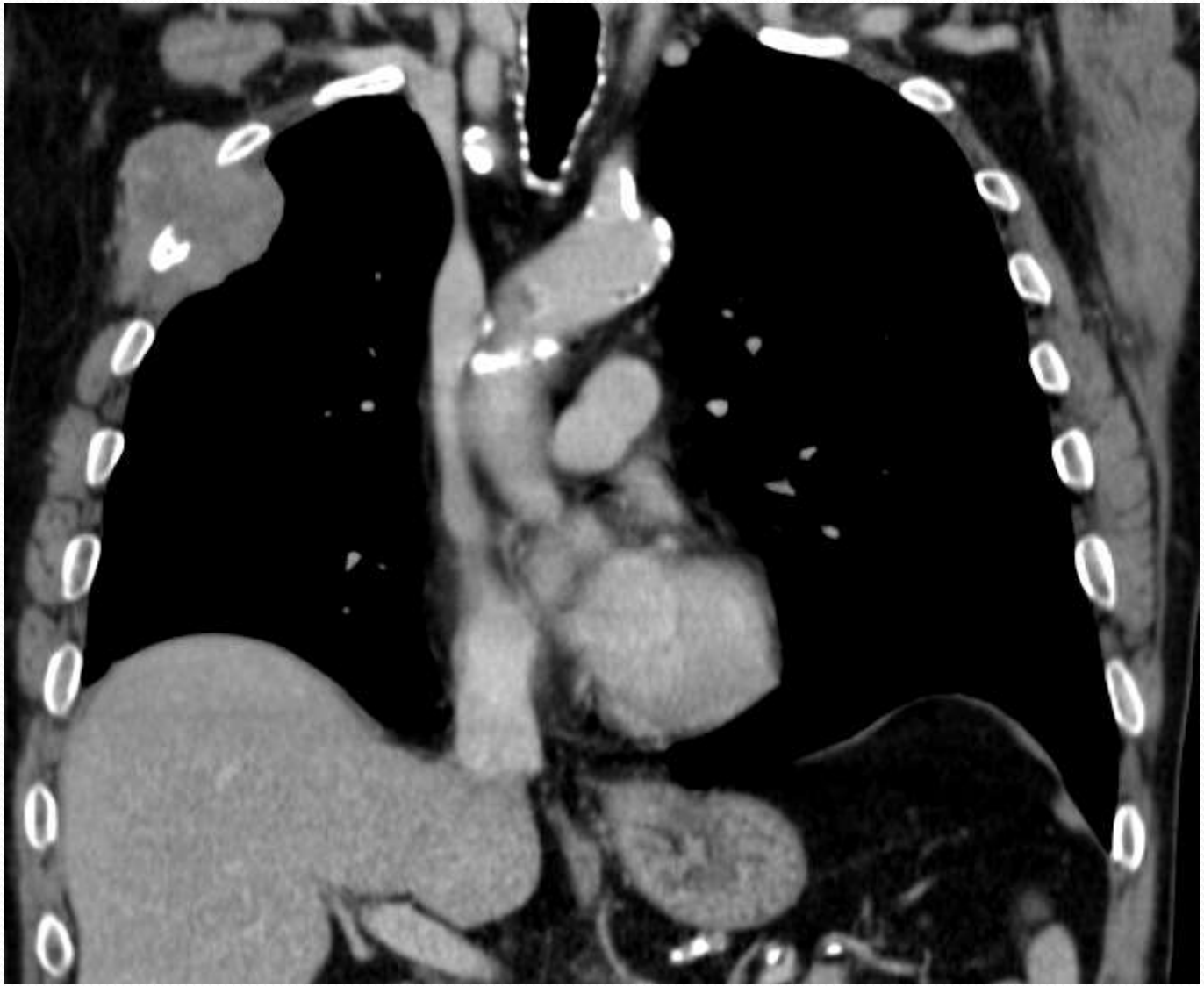
- (Not *diffuse* malignant mesothelioma with a dominant mass.)
- Solitary malignant mass, which on imaging appears as a locally invasive tumour of indeterminate origin, but in keeping with a sarcoma.
- Diagnostic criteria:

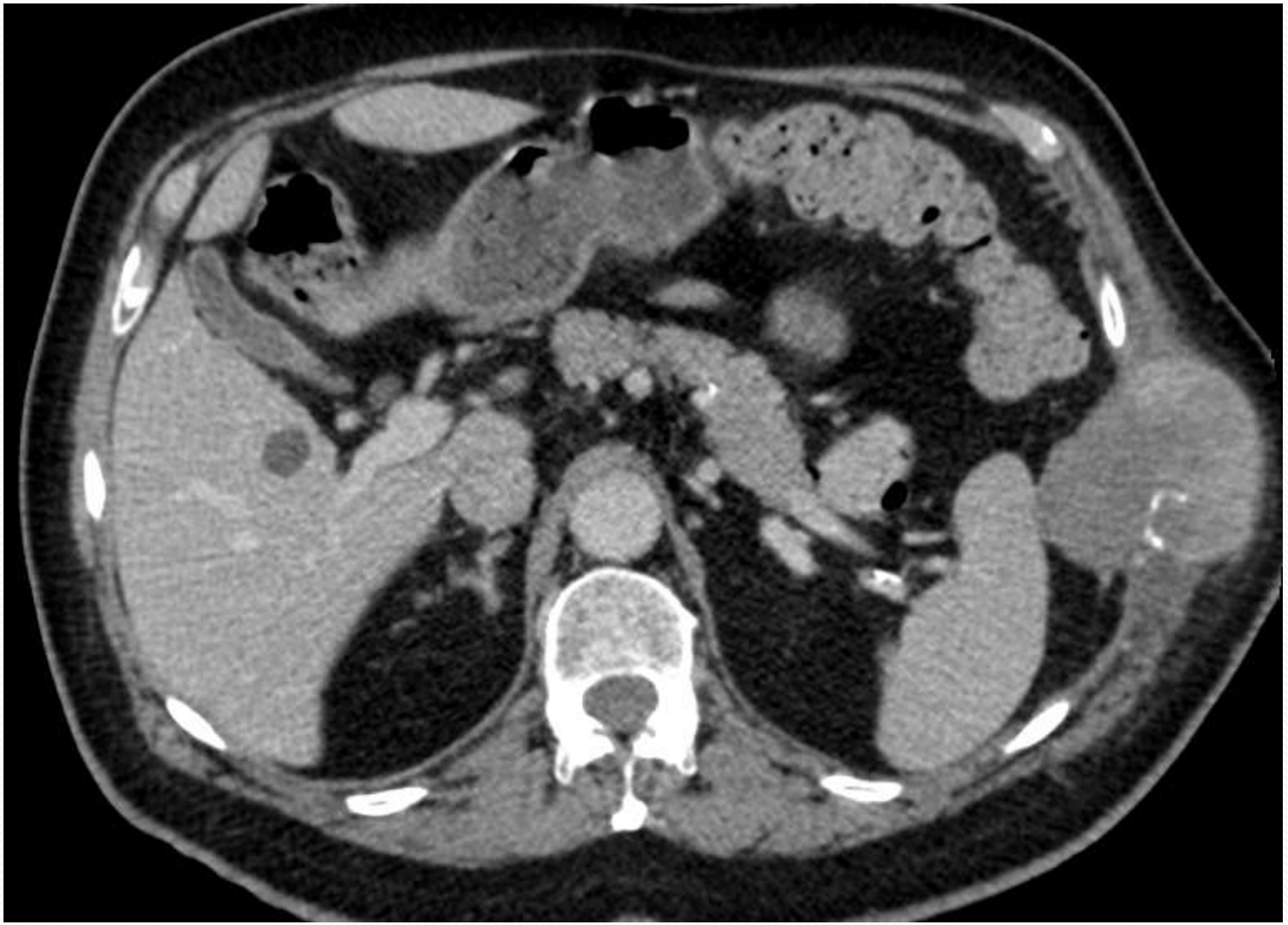
  Radiological, surgical or pathological evidence of a localised serosal/ subserosal (but not organ centred) tumour mass without evidence of diffuse serosal spread.

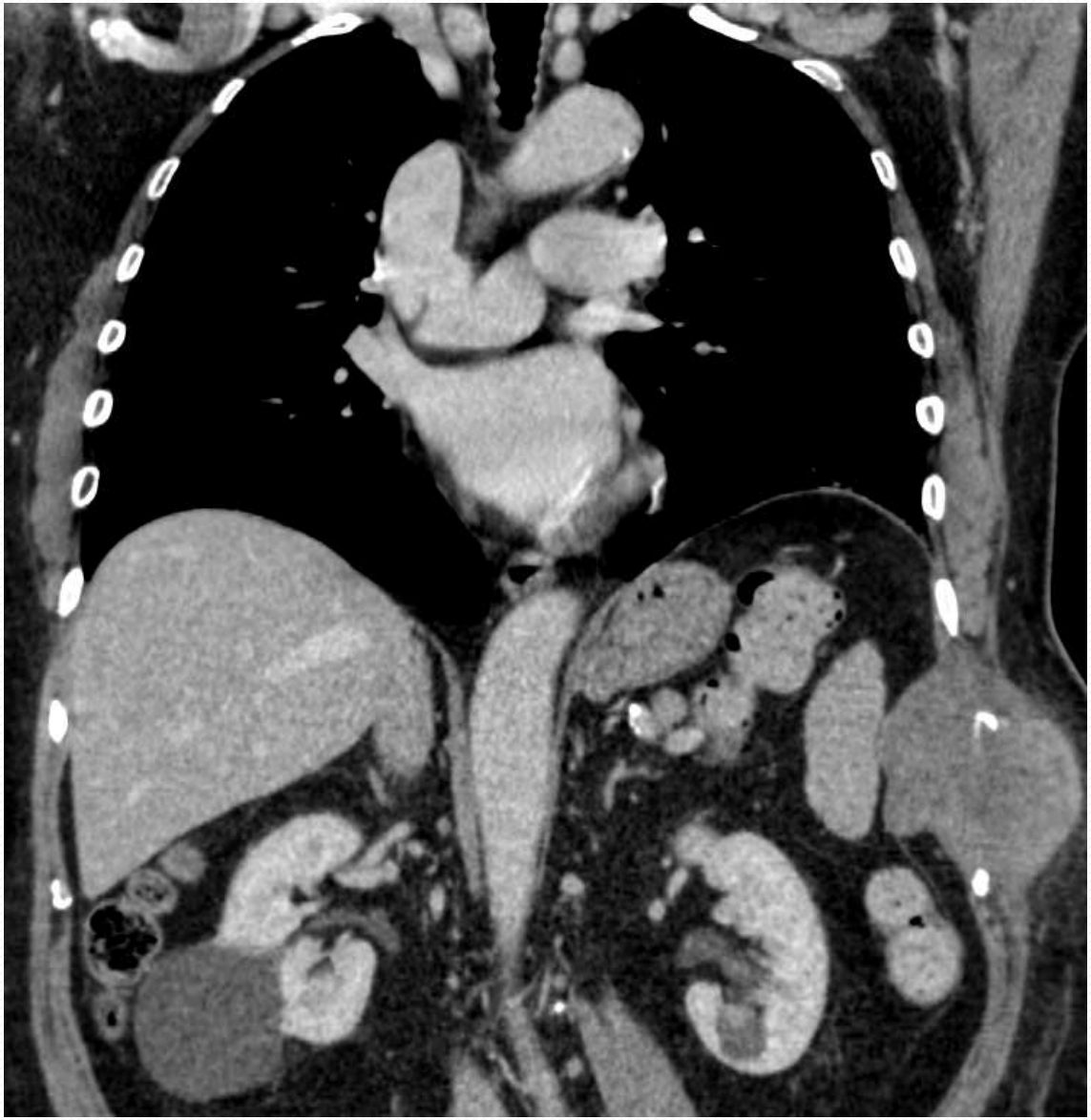
  A microscopic pattern identical to that found in diffuse malignant mesothelioma, and an immunocytochemical profile suggestive of this diagnosis.











Solitary Malignant Mesothelioma

- Imaging features and behaviour are similar to those of a sarcoma.
- May be treated with a complete resection.
- Carries a better prognosis than *diffuse* malignant mesothelioma.
- Like diffuse malignant mesothelioma, may have peritoneal or pleural origin.
- Very rare. ? < 100 cases reported world wide. Beware the "rare bird"!

Conclusions

- Depending on where you practice, asbestos related pleural and pulmonary disease is common.
- Despite the ban on the use of asbestos, and because of the long latent period, these conditions are going to remain prevalent for many years to come.
- There are a number of pleural and lung parenchymal conditions to consider, ranging from the trivial to the catastrophic.
- Sometimes the imaging features are virtually pathognomonic. On other occasions, asbestos related pleural and/or pulmonary disease is part of a differential diagnosis.