

Bronchogenic Carcinoma

Etiology:

The exact cause and pathogenesis of bronchogenic carcinoma is unknown but certain factors have been incriminated in its causation:

Cigarette smoking: It is estimated that 90% of deaths from lung cancer are due to cigarette smoking and the risk of developing the disease varies directly with the number of cigarettes smoked per day and may be increased by 30 times in heavy cigarette smokers.

Pipe and Cigar smoking appears to carry less risk but this is still not established.

Besides the number of cigarettes, the smoking habit appears to affect the incidence of the disease. Those who inhale the smoke or smoke down to the end of cigarette or relight half smoked cigarettes are more at risk.

Cigarette smoke is formed of over 500 different chemicals both in the gaseous and the particulate phases.

Of these the most important are **nicotine, Tar and CO**. Tobacco “tar” is the name given to particulate matters that exist in the cigarette smoke after subtracting Nicotine and moisture and are mainly products of combustion of the cigarette paper. It is composed of poly cyclic aromatic hydrocarbons especially 3,4 Benzpyrine and methyl cholanthrene.

Tar is an important carcinogenic substance in cigarette smoke and it has been recently found that the membrane of the lymphocytic cells of

Patients with bronchial cancer has increased levels of the enzyme aryl hydrocarbon hydroxylase which converts the tar hydrocarbons into active carcinogenic substances.

Nicotine is a very active pharmacologic agent which is readily absorbed from the oral and respiratory tract mucosa.

Nicotine is indirect carcinogenic, through inhibition of endogenous endorphins which are tumor suppressor genes.

CO is present in variable amounts in Tobacco smoke and in heavy smokers the carboxyhemoglobin levels in blood may reach 10%, although harmless under normal circumstances, may be critical in patients with ischemic heart disease.

The risk of developing lung cancer diminishes with cessation of smoking and after 10 years of abstinence from smoking the risk of lung cancer approaches that of normal individuals. Certain histologic types e.g. the squamous cell carcinoma occurs almost exclusively in cigarette smokers.

Air pollution:

The incidence of bronchogenic carcinoma is more in urban than in rural areas and is increased in industries associated with the inhalation of radioactive gases e.g. Uranium.

Pulmonary fibrosis:

The incidence of bronchogenic carcinoma is increased in patients with old pulmonary fibrosis e.g. TB or fibrosing alveolitis.

Pathology:

Bronchogenic carcinoma usually arises from the segmental bronchi and less commonly from the main or lobar bronchi.

In about 50% of cases it is centrally located, near the pulmonary hilum and in 50% it arises peripherally. The upper lobe especially the anterior segments are more commonly involved and the right lung more than the left.

There are 4 main pathological types:

1) Squamous cell carcinoma (50%):

It is characterized by keratin formation, bridging between the cells and by the formation of large islands of cancer cells. It is nearly always associated with cigarette smoking and especially affects males. It is usually slowly growing and has a tendency for direct spread to the surrounding structures and may undergo cavitation and abscess formation. It is rarely accompanied by the paraneoplastic syndrome except hypercalcemia due to ectopic parathyroid hormone production.

2) Adeno carcinoma (17%):

It is the commonest lung cancer in women and characteristically has a peripheral location where it involves the pleura and presents with malignant pleural effusion or a coin shadow in the X-ray.

It is not associated with cigarette smoking. It usually spreads by blood stream but also has a tendency to seedle along the bronchi invading the other parts of the lungs through this route.

Microscopically; it shows a glandular architecture and may produce mucus.

Of the paraneoplastic syndromes, it is most commonly associated with thrombophlebitis migrans and deep venous thrombosis (mucine is a thromboplastic material).

3) Oat cell carcinoma (16%):

This type has a tendency to affect younger patients and is highly malignant. It may be central or peripheral in location and characteristically metastasizes early to the

Mediastinal lymph nodes presenting with manifestations of mediastinal compression. It is also the commonest bronchial carcinoma associated with Endocrine paraneoplastic syndromes.

4) Large cell carcinoma (17%):

This is made of large cancer cells which are different from both the squamous and the adenocarcinomatous cells.

Radiological characteristics by cell type

Squamous cell carcinoma

Squamous cell carcinoma are more often centrally located within the lung and may grow much larger than 4 cm in diameter. Cavitation is seen in up to 82%. They commonly cause segmental or lobar lung collapse due to their central location and relative frequency

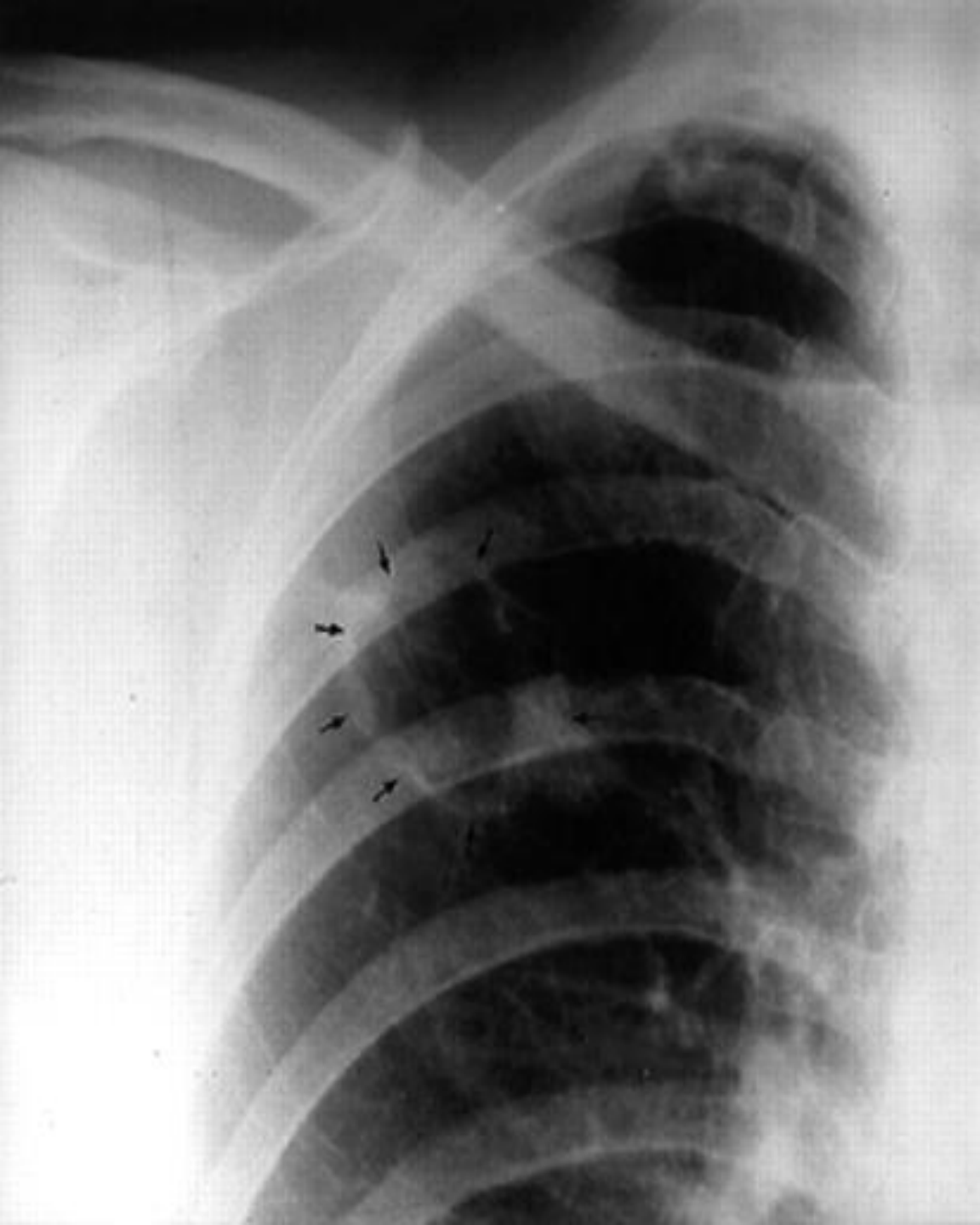


Fig. 1.— A 50-yr-old female with irregular cavitating squamous cell carcinoma in the right upper lobe (arrows).

Adenocarcinoma

Adenocarcinoma are typically peripherally located and measure <4 cm in diameter; only 4% show cavitation. Hila or hila and mediastinal involvement is seen in 51% of cases on chest radiography and a recent study describes two characteristic appearances on CT: either a localized ground glass opacity which grows slowly (doubling time >1 yr) or a solid mass which grows more rapidly (doubling time <1 yr).

Bronchoalveolar carcinoma

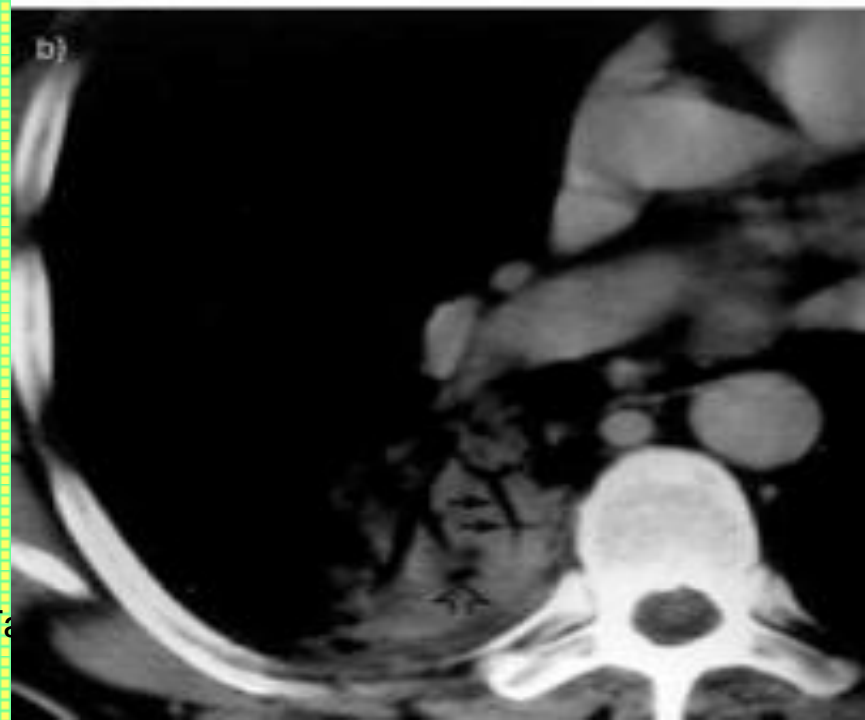
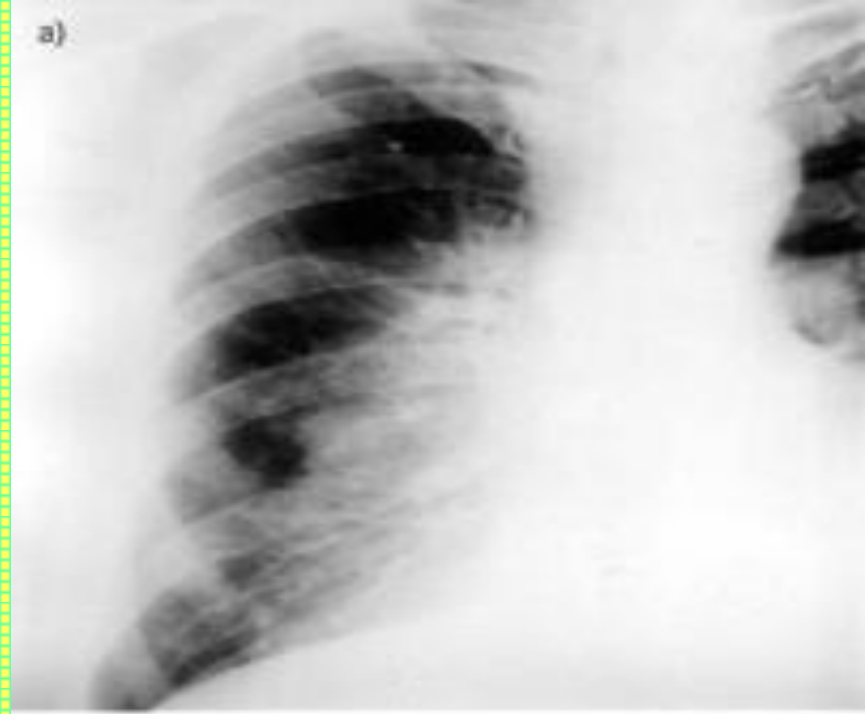
This is regarded as a subtype of adenocarcinoma and represents 2–10% of all primary lung cancers. There are three characteristic presentations: most common is a single pulmonary nodule or mass in 41%; in 36% there may be multicentric or diffuse disease; finally, in 22% there is a localized area of parenchymal consolidation.

Bronchoalveolar carcinoma

Bubble-like areas of low attenuation within the mass are a characteristic finding on CT. Hilar and mediastinal lymphadenopathy is uncommon. Persistent peripheral consolidation with associated nodules in the same lobe or in other lobes should raise the possibility of bronchoalveolar carcinoma.

Fig. 2.— a) Diffuse alveolar shadowing in the right lower lobe of a 58-yr-old male presenting as an unresolving pneumonia.

b) Air bronchograms (black arrows) and low attenuation lucencies (open arrow) in apical "consolidation", later confirmed as bronchoalveolar carcinoma.



Adenosquamous carcinoma

Adenosquamous carcinoma represents 2% of all lung cancers. This cell type is typically identified as a solitary, peripheral nodule. Over one-half are 1–3 cm in size and cavitation is seen in 13%. Evidence of parenchymal scars or fibrosis in or next to the tumour is seen in 50%.

Small cell lung cancer

Small cell lung cancer (SCLC) often present with bulky hila and mediastinal lymph node masses. A noncontiguous parenchymal mass can be identified in up to 41% at CT that very rarely cavitates. They form the malignant end of a spectrum of neuroendocrine lung carcinomas with typical carcinoid tumours being at the more benign end. A mass in or adjacent to the hilum is characteristic of SCLC and the tumour may well show mediastinal invasion.

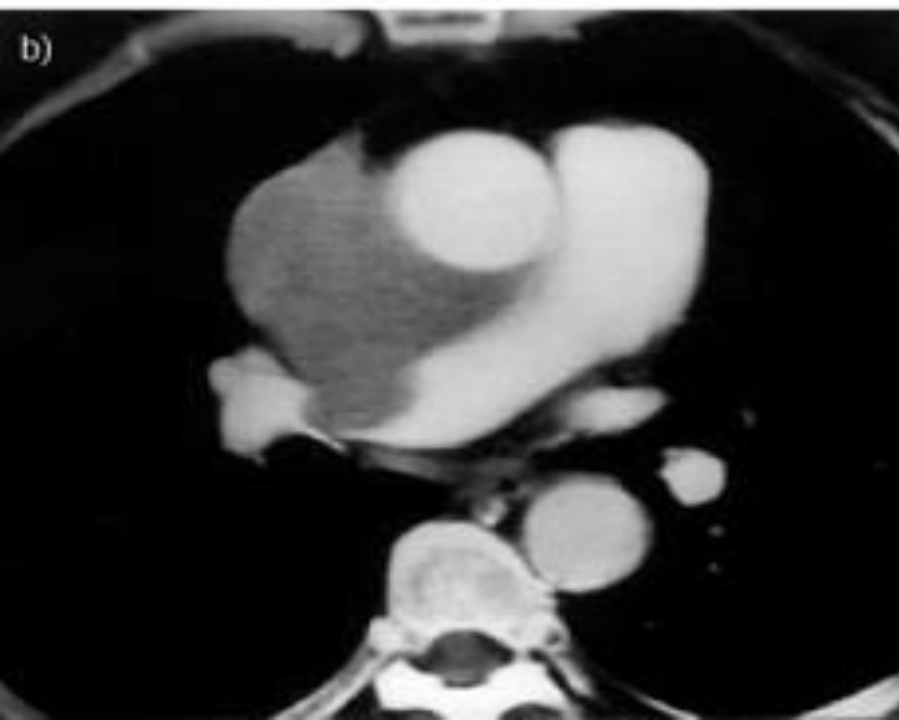


Fig. 3.— a) A 55-yr-old dyspnoeic female. Chest radiograph demonstrating widened mediastinum particularly on the right with reduced vascularity of the right lung.

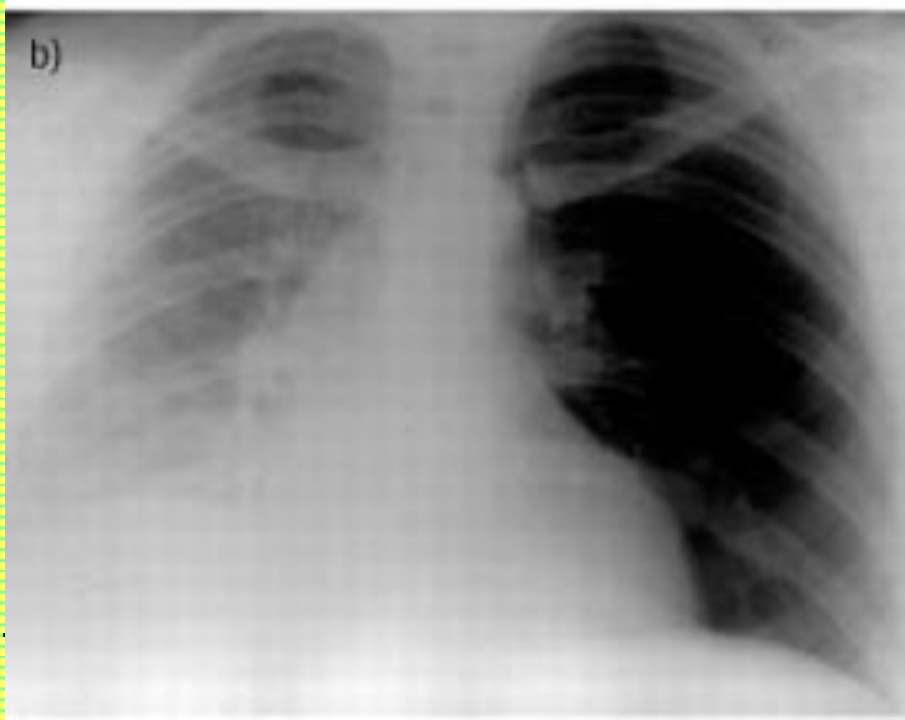
b) Contrast enhanced computed tomography showing central mediastinal mass invading the right pulmonary artery. Small cell carcinoma was confirmed on percutaneous biopsy.

Carcinoid tumour

Carcinoid tumour represents 1% of all lung cancers. Atypical carcinoid tumours tend to be larger (typically >2.5 cm at CT) while typical carcinoid tumours being more often associated with endobronchial growth and obstructive pneumonia. Carcinoids tend to be centrally rather than peripherally located and calcification is seen in 26–33%. The 5-yr survival for typical carcinoids is 95% against 57–66% for atypical carcinoids

Fig. 4.— a) Inspiratory film with asymmetrical vascularity.

b) Expiratory film confirming air trapping due to carcinoid tumour in the left main bronchus.



Large cell carcinoma

Large or giant cell carcinoma is a poorly differentiated nonsmall cell carcinoma (NSCLC) and is diagnosed histologically after exclusion of adenocarcinomatous or squamous differentiation. It may grow extremely rapidly to a large size but metastasizes early to the mediastinum and brain

Clinical manifestations:

Bronchogenic carcinoma is asymptomatic and detected only on routine X-ray chest films in 5% of patients. In the remaining 95% however various symptoms related to the respiratory tract, to distant metastases or to the presence of paraneoplastic syndromes can usually be detected.

I- Respiratory manifestations:

These are the mode of presentation in at least 85% of patients. The various symptoms depend upon the location and the histology of the tumour.

A: Hilar tumours

a) **Early manifestations:** consist of cough, usually productive of mucoid sputum (72%). It is accompanied by hemoptysis in 50% of cases usually in the form of regular daily staining of the sputum. Massive hemoptysis is rare.

I- Respiratory manifestations:

Chest pain occurs in 30% of patients, the most common variety is a poorly localised pain that is especially worse at night. Pleuretic pain occurs when there is complicating chest infection

b) **Late manifestations:** The presence of bronchial tumour usually leads to bronchial obstruction associated with manifestations of lung collapse, pneumonia, lung abscess or bronchiectasis.

I- Respiratory manifestations:

Any adult male presenting with unresolved pneumonia or with lung abscess which is not confined to the classic aspiration lung segments or which has an abnormal thick wall, should be bronchoscoped to exclude bronchogenic carcinoma. Stridor and localized wheez are late manifestations.

B: Peripheral tumours

These are more commonly due to adenocarcinoma.

I- Respiratory manifestations:

They may present as coin lesion or more commonly as a massive hemorrhagic pleural effusion. The latter mode of presentation is especially common in women and the pleural effusion although massive, may not be associated with shift of the mediastinum to the opposite side due to the presence of underlying pulmonary collapse.

I- Respiratory manifestations:

C: Apical tumours

These characteristically leads to the Pancost syndrome due to the infiltration of the lower cord of the brachial plexus, the sympathetic trunk and the posterior part of the upper ribs. This leads to characteristic manifestations which include the following:

- Pain in the shoulder, scapula and the inner arm.

I- Respiratory manifestations:

- Atrophy of the small muscles of the hand on the same side.
- Horner's syndrome on the same side.
- Erosion of the upper ribs posteriorly.
- Dull Kronig's isthmus.
- The deep supra-clavicular lymph nodes are usually enlarged and palpable on the same side.

I- Respiratory manifestations:

D: Superior mediastinal syndrome

The manifestations consists of venous engorgement of the upper part of the body including facial congestion, oedema of the conjunctiva (chemosis) with headache and non-pulsating external jugular veins on the front of the chest and upper limb (which indicates obstruction below the entrance of the vena azygos vein into the superior vena cava).

I- Respiratory manifestations:

D: Superior mediastinal syndrome

Paralysis of the left recurrent laryngeal nerve leading to hoarsness of voice, but it should be noted that laryngeal oedema due to S.V.C. obstruction can cause hoarsness without laryngeal paralysis.

Phrenic paralysis leads to high copula of the diaphragm which show paradoxical movements during fluoroscopy.

II- Metastatic manifestations:

About 12% of patients with bronchogenic carcinoma present with symptoms arising from metastases of the tumour, These include:

1. Lymph nodes:

The tracheo-bronchial and paratracheal lymph nodes are early involved and this can be detected radiologically. Clinically, the supraclavicular lymph nodes on the same side become involved and palpable early in the disease.

II- Metastatic manifestations:

1. Lymph nodes:

Rarely spread occurs retrogradely via para-aortic lymph glands to the inguinal lymph nodes.

Lymphangitis carcinomatosa

It occurs when there is diffuse spread of carcinoma through the lymphatic channels of the lung and occurs especially with adenocarcinoma.

II- Metastatic manifestations:

Lymphangitis carcinomatosa

It is characterized by the presence of very severe dyspnea and the syndrome of diffuse interstitial pulmonary fibrosis.

The syndrome also complicates adenocarcinomata of other organs but the radiological picture is then usually symmetrical and is not associated with unilateral hilar mass.

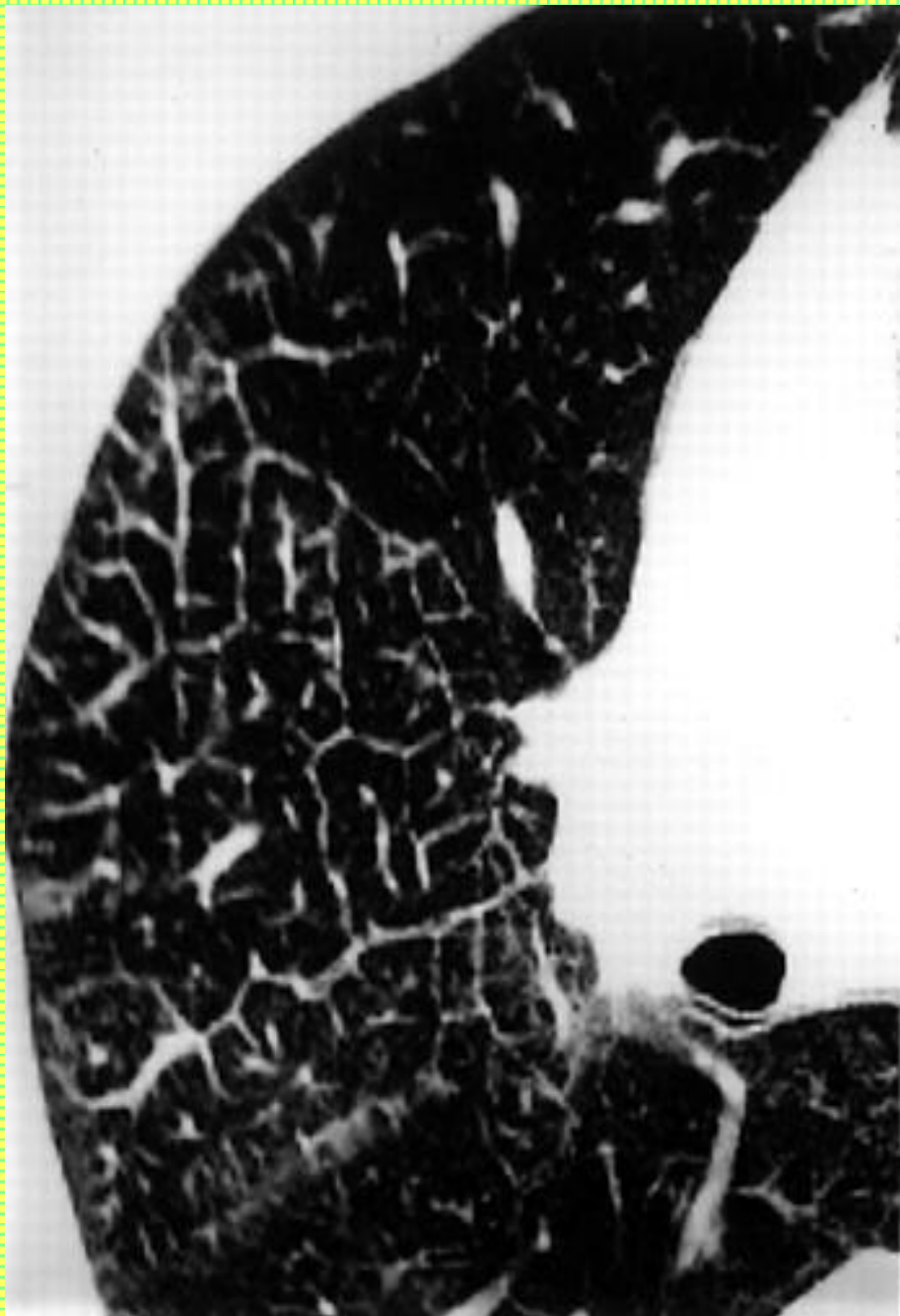


Fig. 5.—
Characteristic
septal nodular
thickening on high-
resolution scans
typical of
lymphangitis
carcinomatosa.

II- Metastatic manifestations:

2) Liver

This is involved pathologically in 40% of cases. Clinically, there is an irregular enlargement of the liver, sometimes associated with jaundice. It is typically accompanied by high levels of alkaline phosphatase but the Alpha-pheto protein is usually absent. (D.D. from hepatoma). Ascitis may occur.



Fig. 6.— Massive left adrenal (open arrow) and hepatic metastases (arrows).
M1 disease, stage IV.

II- Metastatic manifestations:

3) Brain

Occurs in 20% of cases and may present with by convulsions, hemiplegia and signs of increased intracranial tension.

Neurological manifestations of brain metastases should be differentiated from those due to metabolic encephalopathy associated with hyponatremia (increased ADH), hypercalcemia and hypoglycemia.

II- Metastatic manifestations:

3) Brain

Brain secondaries are much commoner than primary brain tumours and for this reason any patient presenting with manifestations of brain tumour should have an X-ray chest done to exclude primary in the lung.

4) Bone metastases

Occur in 15% of cases. The most commonly affected bones are the ribs.

Fig. 7.—
Computed
tomography scan
of enhancing
cerebral
metastasis with
marked oedema
and mass effect.





Fig. 8.— Vertebral body metastasis.

II- Metastatic manifestations:

5) Suprarenal gland

Metastases occur in 18% of cases but very rarely present clinically by adrenal insufficiency.

N.B.

It should be noted that 25% of patients with bronchogenic carcinoma die before the appearance of distant metastases and never present with manifestations outside the chest.

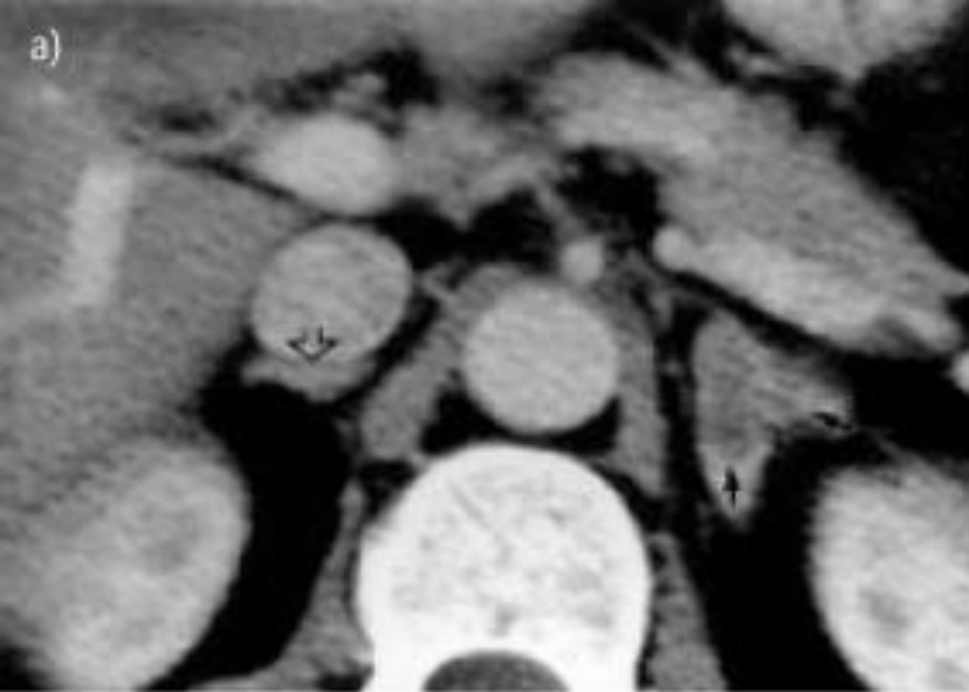


Fig. 9.— a) Low attenuation adrenal mass (arrows) with normal right adrenal (open arrow) which at biopsy, b) confirmed metastatic deposits.

III- Paraneoplastic syndromes:

About 10% of patients with bronchogenic carcinoma present with extra-thoracic non-metastatic manifestations. They are all rare except clubbing and pulmonary osteoarthropathy. Their main importance is that their detection in a patient with a suspicious pulmonary lesion points towards malignancy and they sometimes occur along before radiologic evidence of lung tumour appear.

III- Paraneoplastic syndromes:

These syndromes include the following:

1) Metabolic syndromes:

Loss of weight, anorexia, cachexia and fever are thought to be due to the secretion of antimetabolite poly-peptide.

2) Endocrinal syndromes

i) ACTH-like hormone secretion:

Usually occurs with Oat-cell carcinoma.

III- Paraneoplastic syndromes:

And produces the clinical picture of Cushing syndrome. It differs from the classic Cushing by the presence of more severe potassium depletion leading to polyuria, thirst and severe muscle weakness. There is also increased skin pigmentation due to secretion of M.S.H. like substance by the tumour.

ii) Inappropriate vasopressine or A.D.H like material:

This is secreted mainly by Oat-cell carcinoma.

III- Paraneoplastic syndromes:

ii) Inappropriate vasopressine or A.D.H like material:

It leads to the appearance of symptoms of cerebral oedema with headache, drowsiness, disorientation and convulsions. The serum sodium is nearly always below 120 mEq/L. and characteristically, the urine osmolarity is above that of plasma (above 275 ml Osmol/Kg BW).

III- Paraneoplastic syndromes:

iii) Hypercalcemia:

This is due to the secretion of parathyroid hormone-like substance which contains only 20 amino acids (as opposed to the 80 amino acids of the ordinary parathyroid hormone). It is mainly secreted by squamous cell carcinoma and leads to the various manifestations of hypercalcemia such as thirst, polyuria, anorexia, constipation, disorientation, generalized hyporeflexia, myoclonus and rarely metastatic calcification.

III- Paraneoplastic syndromes:

iv) Bilateral gynecomastia:

Secondary to the secretion of prolactin-like substance.

v) Carcinoid syndrome:

Secondary to the secretion of 5-hydroxy tryptamine (serotonin).

vi) Secondary Polycythemia:

Due to secretion of erythropoietin-like substance

III- Paraneoplastic syndromes:

3) Neuromyopathic syndromes:

i) Encephalopathies:

Dementia, psychotic changes. It may be metabolic encephalopathy (secondary to hypercalcemia, increased A.D.H., Causing syndrome, anemia, hypoglycemia or infection.

ii) Subacute cerebellar degeneration:

It has a characteristic clinical picture with subacute bilateral symmetrical cerebellar affection

III- Paraneoplastic syndromes:

ii) Subacute cerebellar degeneration:

Equal in the arms and legs and developing over 3 – 6 weeks period.

Severe dysarthria is present but nystagmus is absent, it is usually accompanied by long tract affection especially of the pyramidal tracts. The disease usually becomes stable after 6-8 weeks and does not progress and the C.S.F. may contain excess lymphocytes and proteins but is sometimes normal.

III- Paraneoplastic syndromes:

iii) Myelopathic manifestations:

It is said that 10-15% of cases of amyotrophic lateral sclerosis are associated with malignant disease especially bronchogenic carcinoma.

iv) Neuropathic manifestations:

Include 3 syndromes; ganglioradiculitis, peripheral neuropathy and acute polyneuropathy of the Guillian-Barre type.

III- Paraneoplastic syndromes:

iv) Neuropathic manifestations:

Ganglioradiculitis always suggest the presence of malignant neoplasm in the body. It is characterized by subacute distal sensory loss especially of the deep sensation usually with radicular pattern but the motor functions are relatively preserved. The disease always precedes the appearance of the cancer and progresses over few months and then stabilizes but leaves the patient with severe sensory loss and sensory ataxia, The spinal fluid protein is usually elevated.

III- Paraneoplastic syndromes:

v) Myopathic manifestations:

It includes the following:

a) Polymyositis: which especially affects the pelvic and shoulder girdles leading to weakness in climbing stairs and rising from low stools associated with hyporeflexia.

The finding of proximal muscle weakness in men after the middle age should always arouse the suspicion of bronchogenic carcinoma.

III- Paraneoplastic syndromes:

b) Dermatomyositis: especially occurs with adenocarcinoma and is associated with typical violet skin rash on the cheeks and knuckles usually with facial oedema.

c) The myasthenic syndrome (Eaton-Lambert syndrome): especially occurs with Oat cell carcinoma and other undifferentiated carcinomata. The patient complains of weakness of the proximal muscles especially of the pelvic girdle and thighs associated with

III- Paraneoplastic syndromes:

c) The myasthenic syndrome :

Dryness of the mouth and peripheral parathesia. The weakness is not associated with wasting but has the characteristic tendency to decrease with repeated muscular contraction. This is best demonstrated by EMG which shows that repeated nerve stimulation causes progressive increase in the size of the muscle action potential (exactly opposite to what occurs with ordinary myasthenia). The disease does not respond to anti-cholinestrases but respond to guanidine hydrochloride in the dose of 20-50 mg/Kg/day.

III- Paraneoplastic syndromes:

4) Vascular syndromes:

i) Thrombophlebitis migrans:

Or deep vein thrombosis especially when they affect the upper limbs in old people are very suspicious of malignancy in general and of mucin-secreting adenocarcinomata in particular, especially those of pancreas and lung. The mucin acts as thromboplastic material encouraging incidence of thrombosis.

III- Paraneoplastic syndromes:

4) Vascular syndromes:

ii) Venous gangrene.

iii) Non bacterial thrombotic endocarditis:

May also complicate adenocarcinoma of the bronchus.

5) Cutaneous manifestations:

Pemphygoid, Herpes-Zoster, dermatomyositis, Acanthosis Nigricans, Erythema multiforme.

III- Paraneoplastic syndromes:

5) Cutaneous manifestations:

Erythema multiforme may occur especially with adenocarcinomata. Pachydermatoperiostitis may occur leading to thickening of the skin and to an acromegaly-like appearance. It usually accompanies pulmonary osteoarthropathy.

III- Paraneoplastic syndromes:

6) Skeletal manifestations

Clubbing:

This is the most common paraneoplastic syndrome and occurs in 10-30% of patients. It may be associated with pulmonary osteoarthropathy which is characterized by the presence of seronegative bilateral and symmetrical arthropathy very similar to rheumatoid arthritis but without deformities.

III- Paraneoplastic syndromes:

Clubbing:

Or subcutaneous nodules. It is usually associated with sub-periosteal new bone formation especially affecting the radius, ulna and tibia. The overlying tissues become edematous and red and radiologically there is an onion-skin appearance.

Unilateral gynecomastia associated with excessive flushing of the hands and feet may also occur

III- Paraneoplastic syndromes:

Clubbing:

Although clubbing occurs in many conditions, the presence of the complete syndrome (clubbing. Sub-periosteal bone formation, arthropathy and gynecomastia suggest the presence of bronchogenic carcinoma in 90% of cases.

III- Paraneoplastic syndromes:

Clubbing:

The clubbing and pulmonary osteoarthropathy is more manifest on the tumour side and neurogenic reflex is thought to be considered since vagotomy below the level of the recurrent laryngeal nerve is associated with immediate relief of the osteoarthropathy. It also respond to resection of the tumour.

III- Paraneoplastic syndromes:

7) Hematologic manifestations

Anemia, Erythroleukemia, leukomoid blood picture and polycythemia may be present.

Disseminated intravascular thrombosis may occur in terminal cases especially in mucin-producing adenocarcinoma.

Rarely, hemolytic anemia or pure red cell aplasia are present.

Investigations

Noninvasive modalities

1- Chest Radiography

- Lesions are invisible until they are 7-10 mm in diameter
- Pleural effusion
- Elevation of hemi diaphragm
- Rib destruction

Benign nodules

Chest radiography

A number of findings enable a nodule to be classed as benign on the basis of chest radiographical findings. 1) Age <35 yrs, no history of cigarette smoking and no history of extrathoracic malignancy. 2) Comparison with old films and establishment of no growth over at least a 2-yr period. 3) If the nodule contains fat density or a benign pattern of calcification such as central nidus-type, popcorn, laminated or diffuse.

Note should be made that eccentric or stippled calcification is seen in 10% of lung cancers. An appropriate history such as fever or chest pain may promote the likelihood of a benign process such as focal pneumonia or an infarct.

A repeat radiograph should be performed at 2–6 weeks to assess resolution

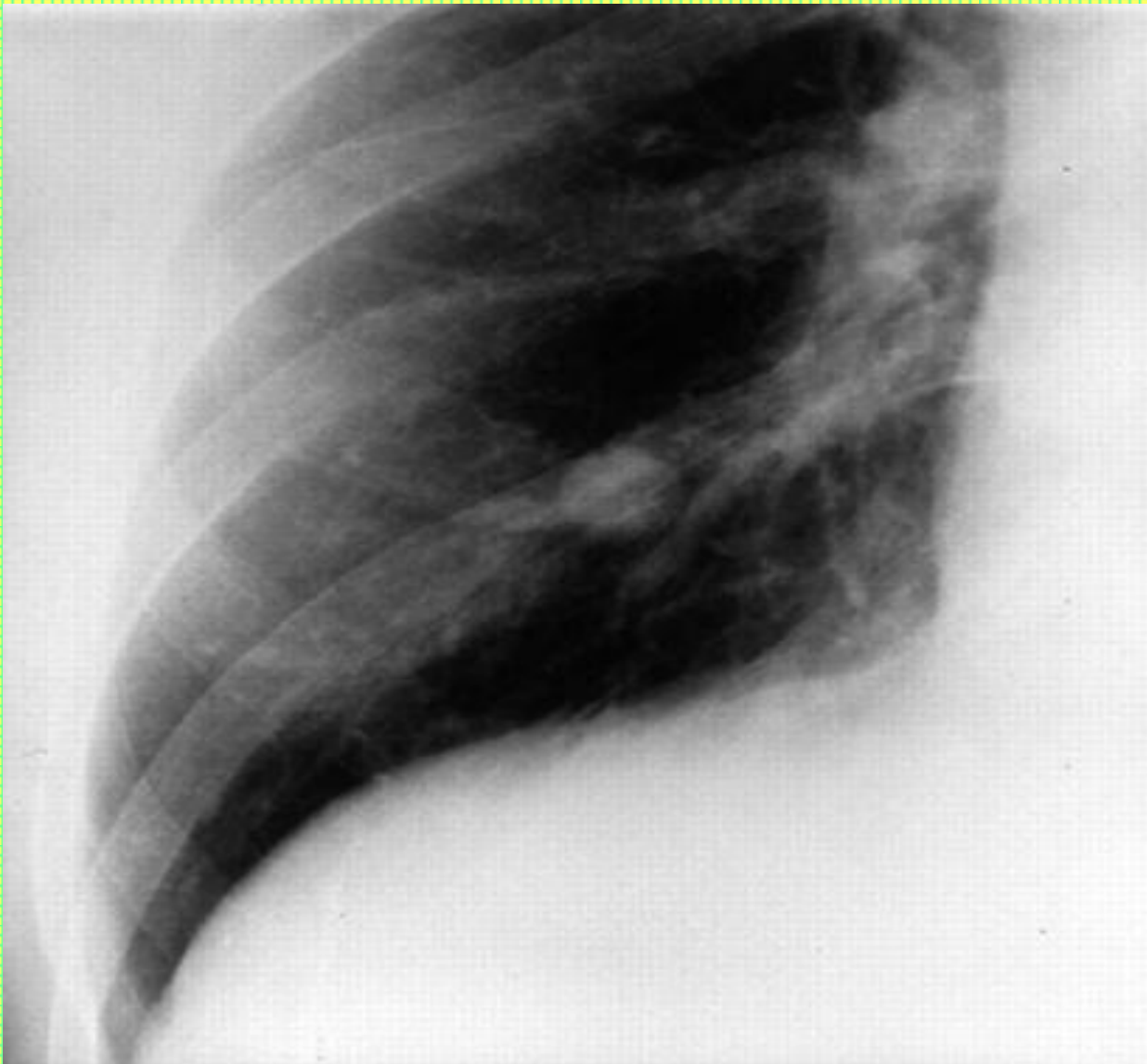


Fig. 10.— Diffusely calcified, well-defined nodule typical of a hamartoma.

2- Sputum Cytology

- Sample collected by saline nebulization or a 3-day morning sputum
- Overall sensitivity :
 - 71% for central lesions
 - 49% for peripheral lesions

3- High resolution CT

- Precise size measurement
- Demonstrating signs of malignancy
- Invasion of contiguous structures
- Details about remaining lung parenchyma
- Upper abdomen
- Distant metastasis
- Assessment of mediastinal lymph nodes:
sensitivity 57% and specificity 82%

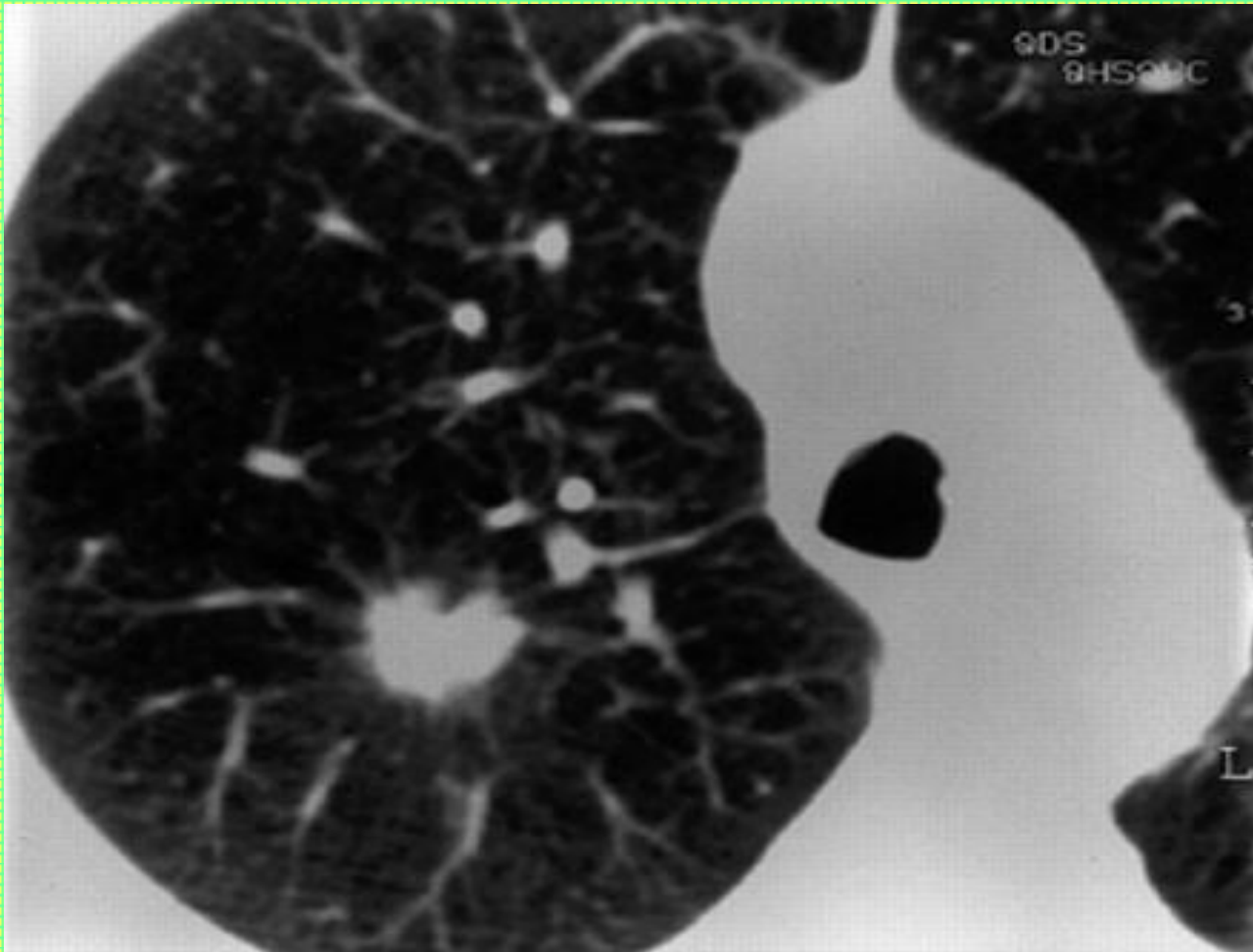


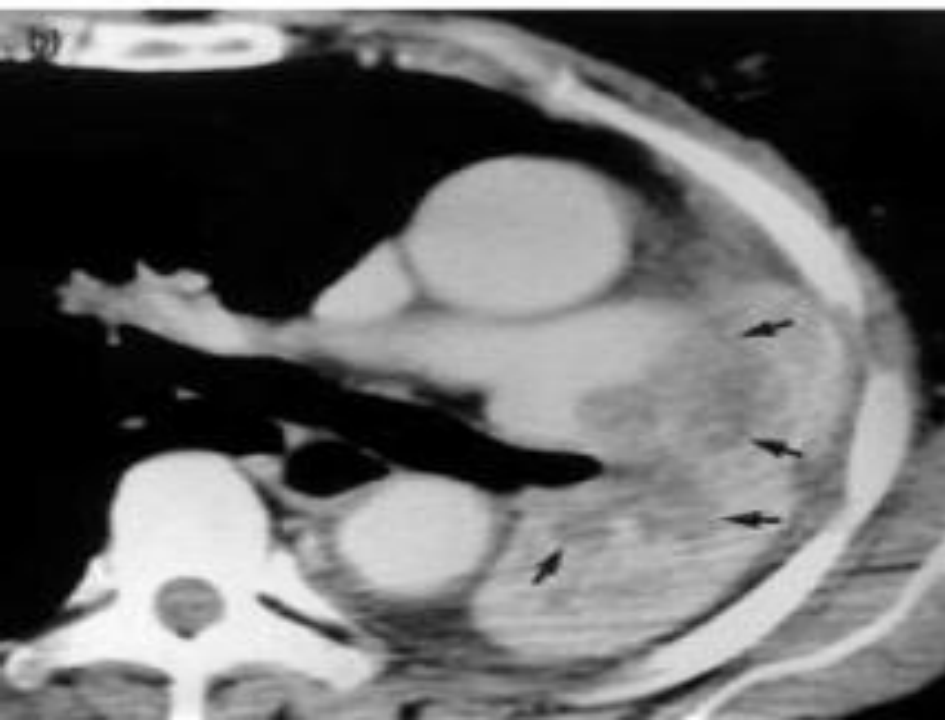
Fig. 11.— Spiculated mass typical of a carcinoma.

Prof. Amgad A. Farhat

3/27/2016



Fig. 12.— a) Collapse of the left lung with mediastinal shift and a right middle zone nodule (arrow).



b) Perihilar low attenuation adenocarcinoma (arrows) with distal enhancing collapsed lung in same patient.

Fig. 13.—
Central mass
with Golden "S"
sign of proximal
tumour (arrows)
and distal
collapse.



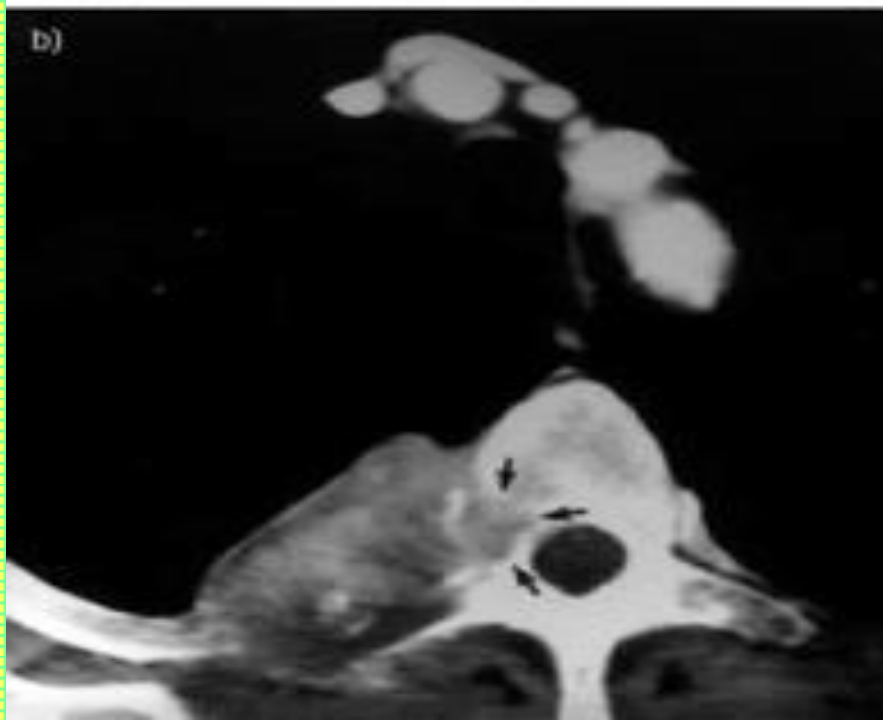
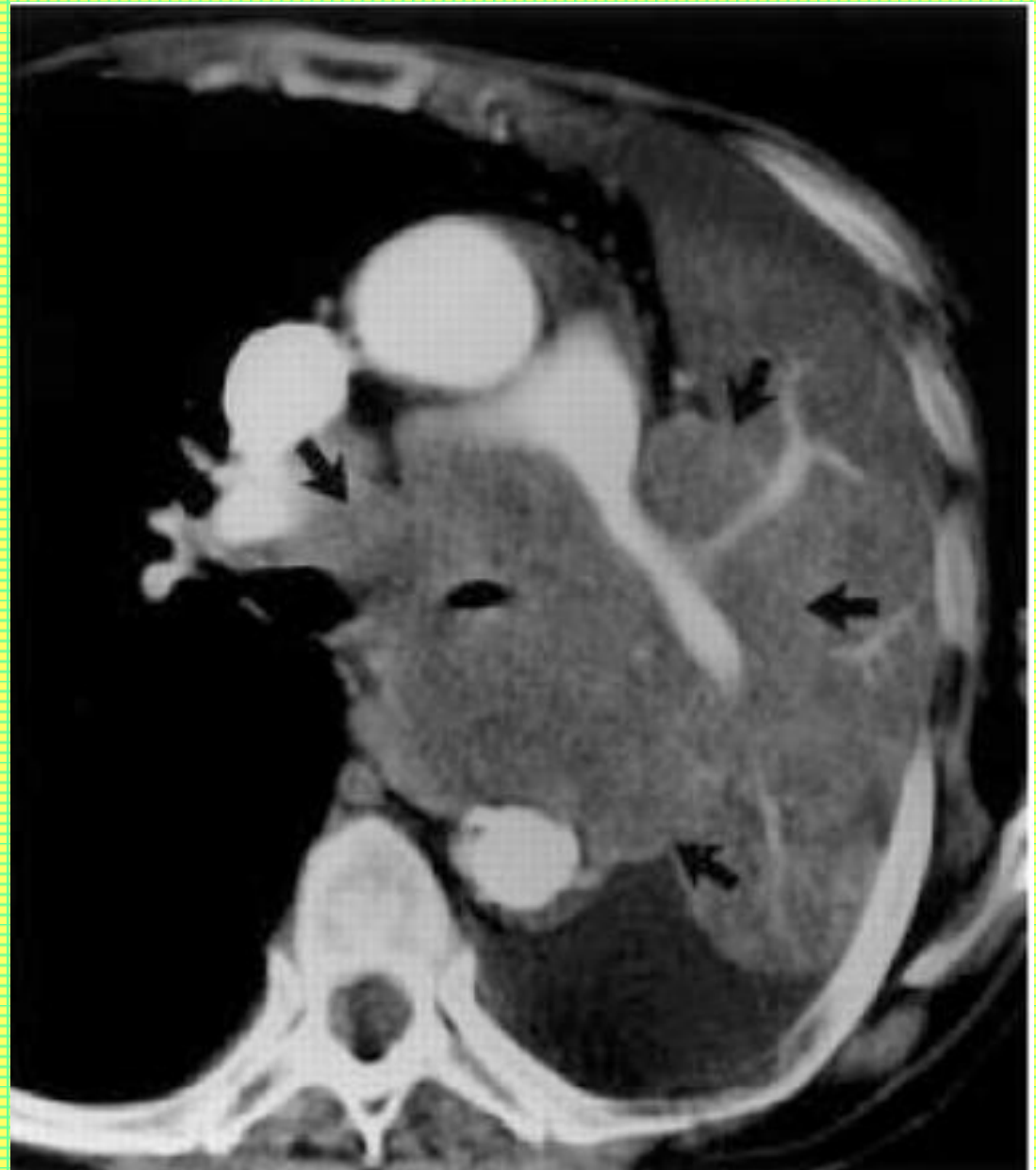


Fig. 14.— a) Rib erosion (large arrow) due to peripheral tumour (small arrows) suggesting at least T3 disease.

b) Corresponding computed tomography showing mass eroding rib and vertebral body (arrows) confirming T4 status and inoperability.

Fig. 15.— Large central mass (arrows) narrowing left main bronchus and encasing left pulmonary artery, indicating T4 status. A pleural effusion is noted.



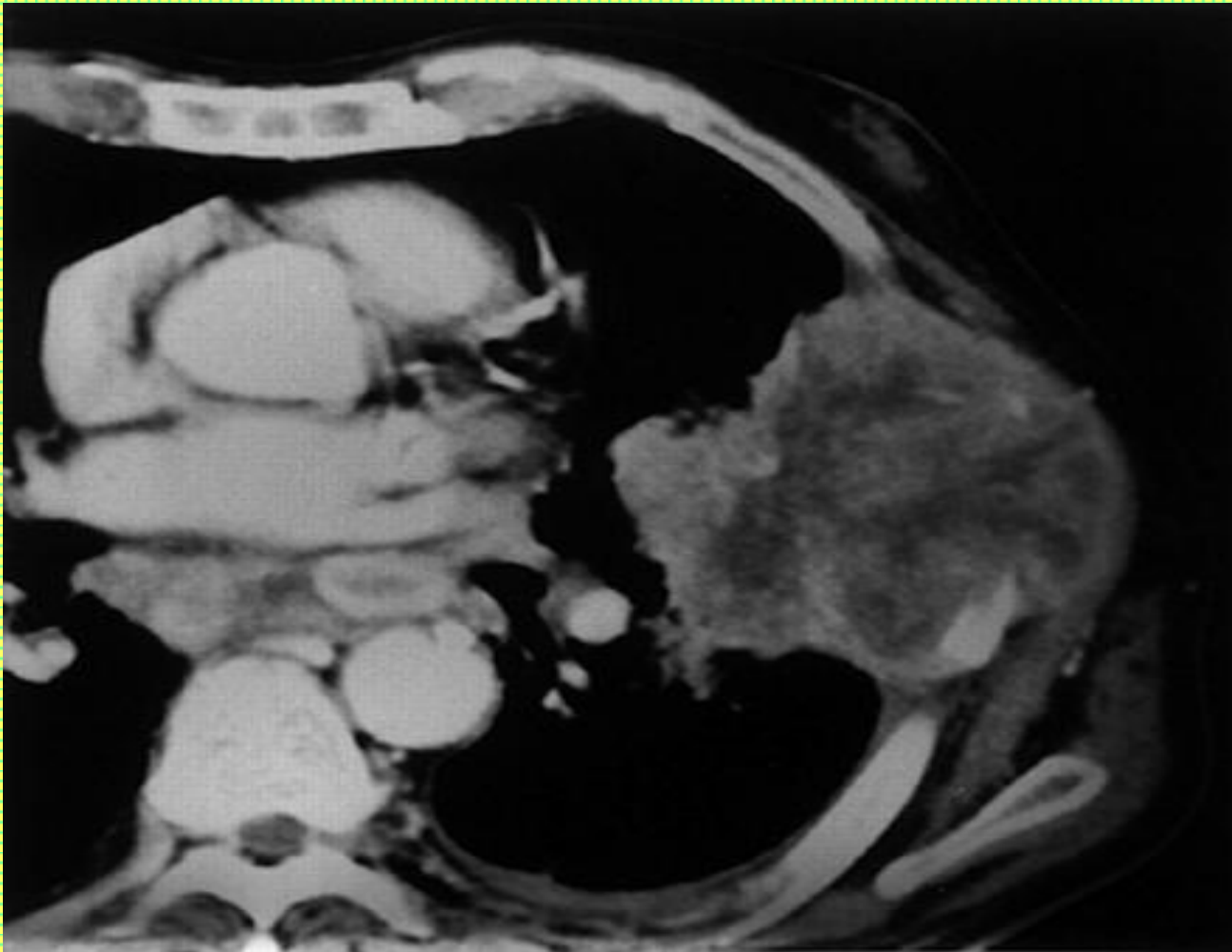
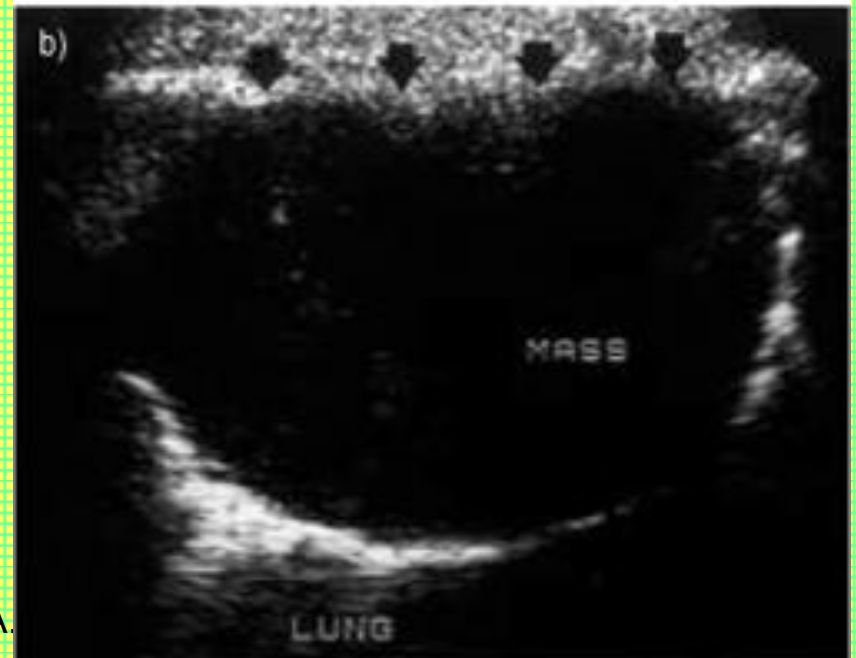
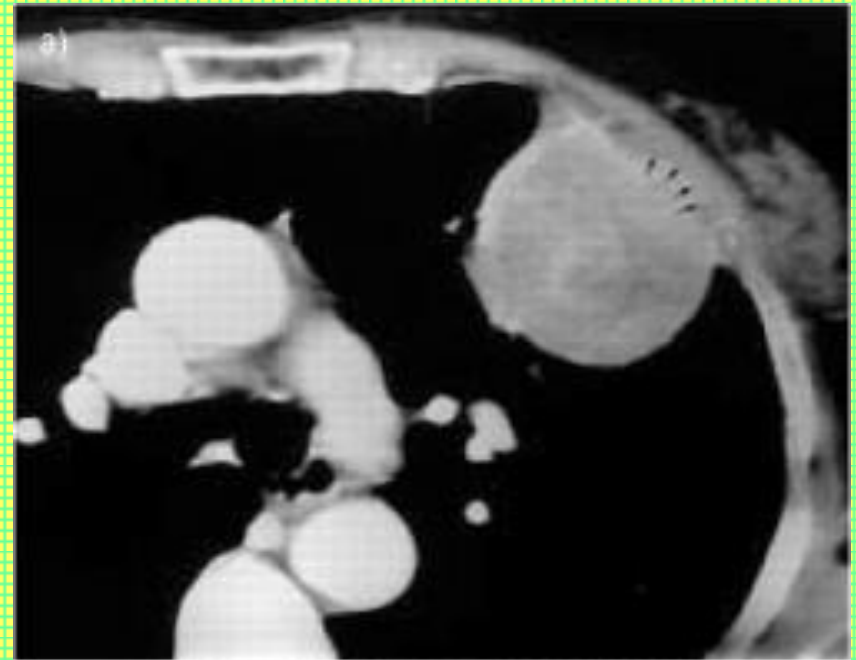


Fig. 16.— Frank chest wall invasion by large peripheral tumour.

Fig. 17.— a)
Computed
tomography scan
suggesting
infiltration of pleural
fat (arrows).

b) Lack of
movement relative
to chest wall
(arrows) confirms
invasion.



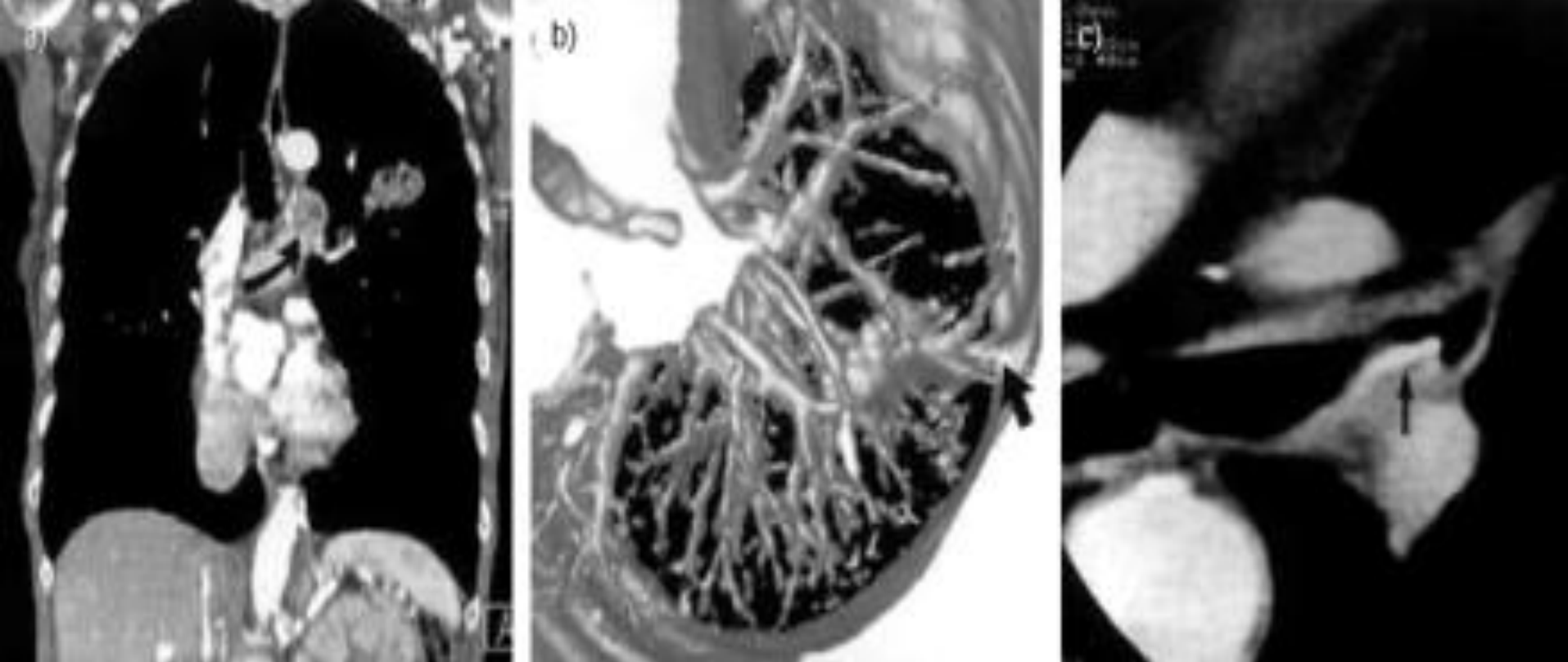


Fig. 18.— a) Coronal reformat from multislice computed tomography (CT) demonstrating mediastinal lymph nodes (arrow) and a necrotic tumour mass within the lung. b) Three-dimensional-reconstruction of a lung tumour with pleural tag (arrow) c) Thin slice reconstruction in the axial plane from spiral CT data permits the correct identification of an inhaled fish bone (arrow), in a different patient, presumed to be a tumour at bronchoscopy.

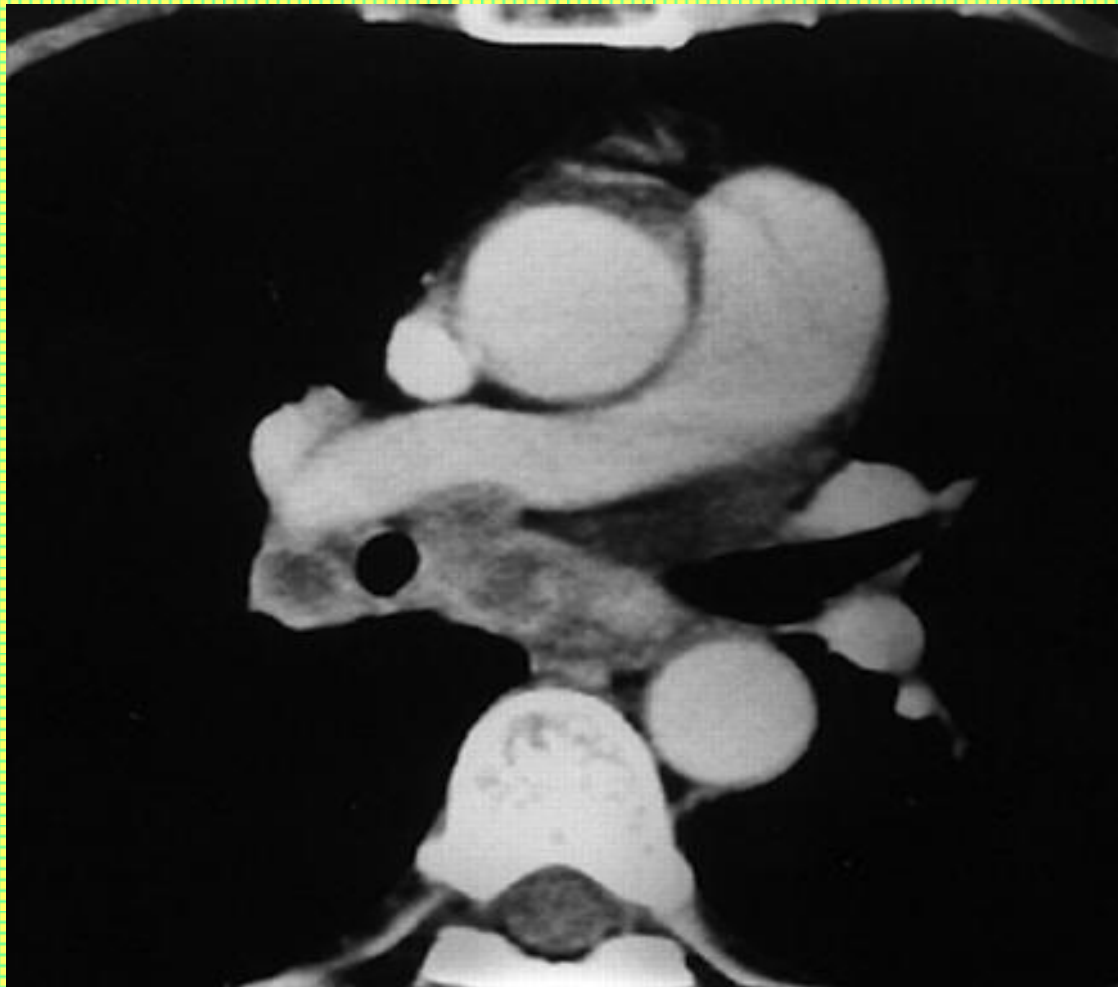


Fig. 19.— Necrotic mediastinal lymph nodes with irregular enhancing rims (arrows).

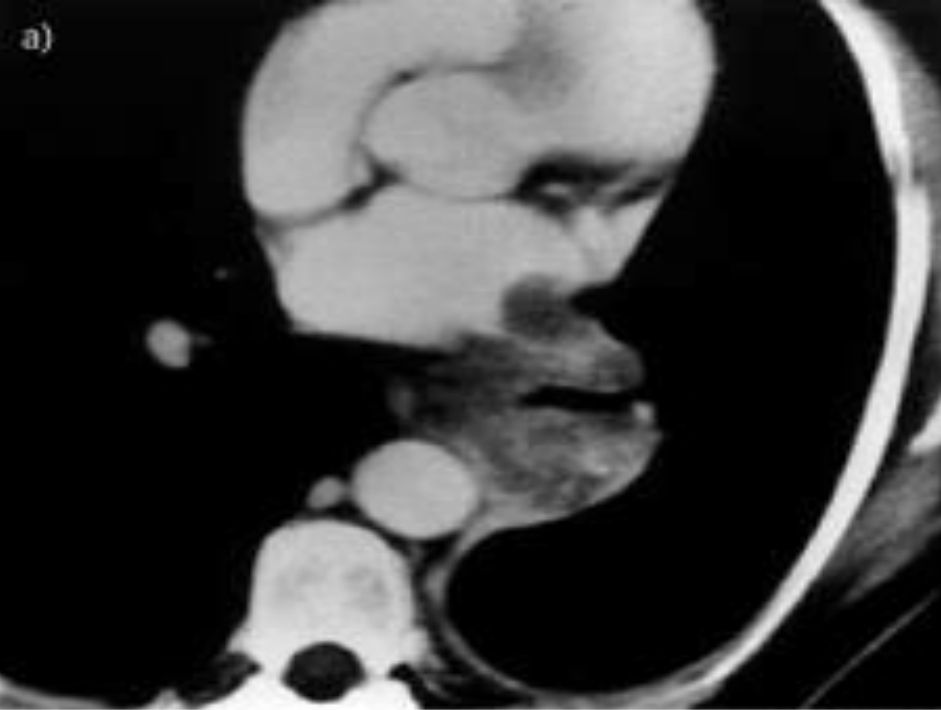
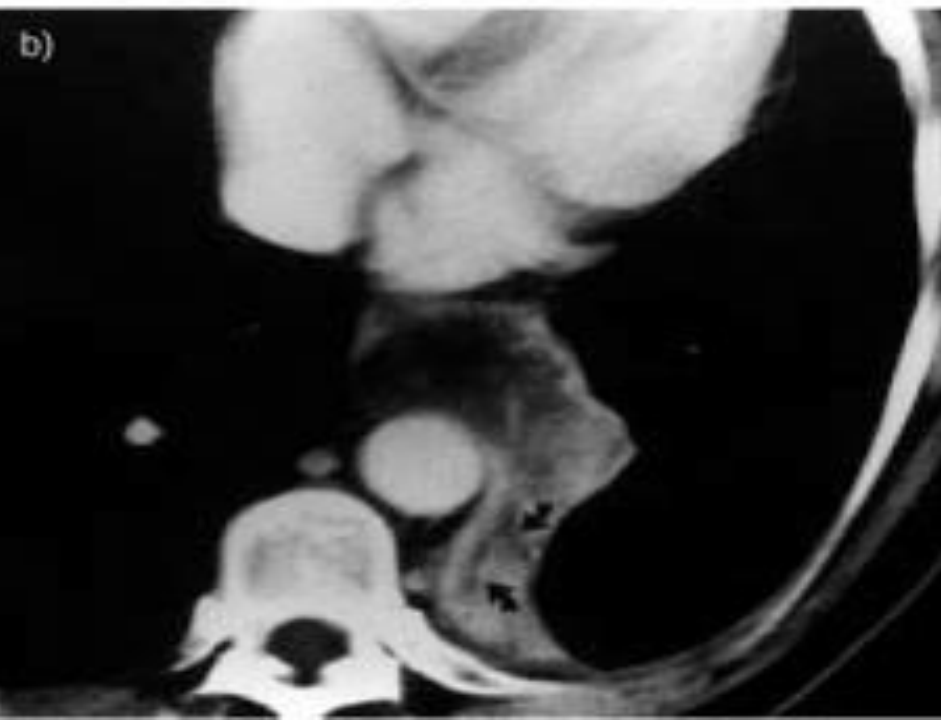


Fig. 20.— a)
Mediastinal mass
narrowing left lower
lobe bronchus and
invading left atrium.



b) Distal fluid-filled
bronchi (arrows) are
seen in the collapsed
lower lobe due to the
proximal tumour.

4- Positron Emission Tomography PET

- There is preferential accumulation of FDG within the primary tumor and potential metastatic sites
- Currently lower limit of resolution is 1 cm
- Detection of mediastinal lymph nodes
sensitivity 84% and specificity 89%
- Recently PET-CT

Fig. 21.— Avid uptake of ^{18}F deoxy -d-glucose in left apical tumour (arrow).



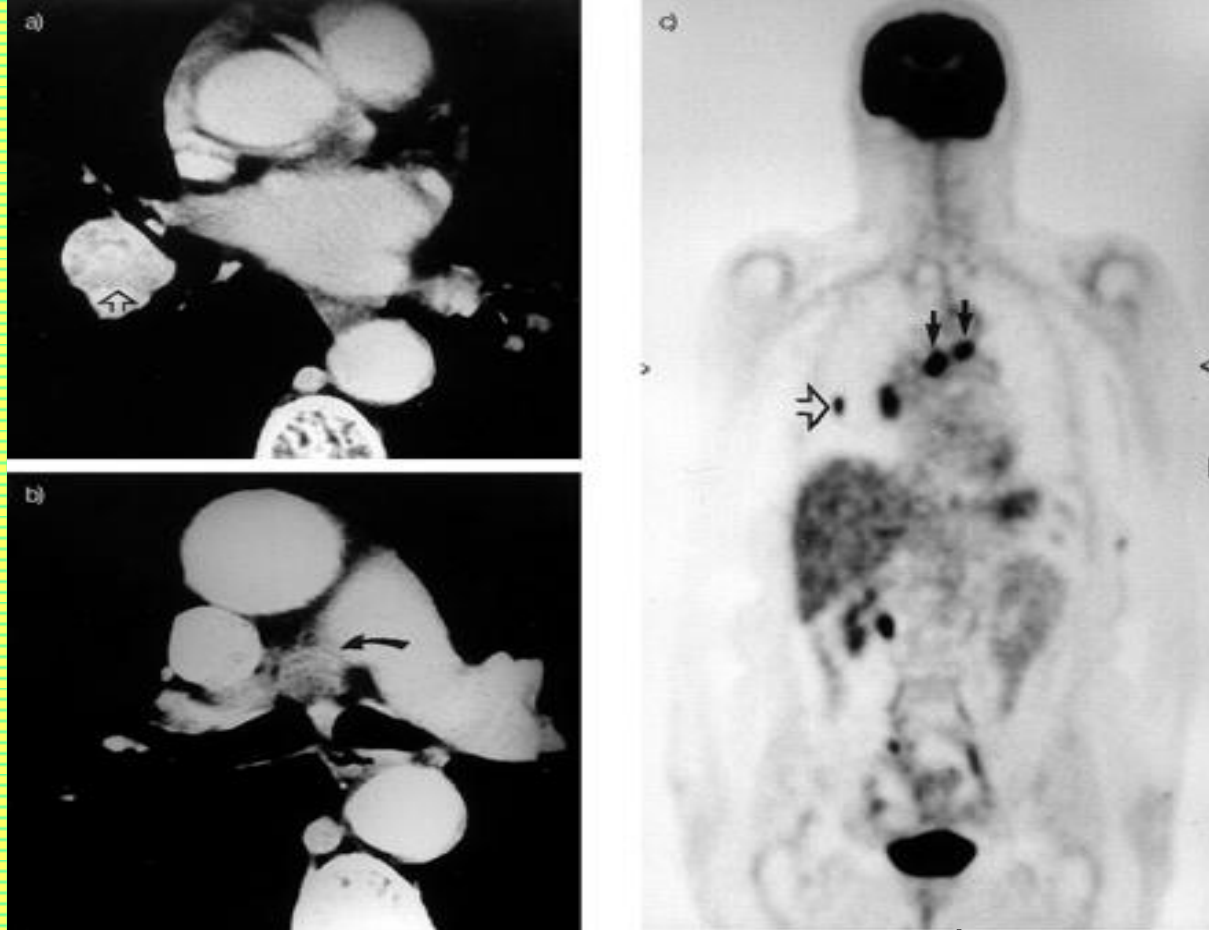


Fig. 22.— Middle-aged-female with a) right hilar mass (arrow) and b) equivocal precarinal lymph node (arrow). c) Positron emission tomography (PET) scan shows increased uptake in mediastinal nodes (arrows) and small peripheral nodule (open arrow). Biopsy of hilar mass confirmed nonsmall cell lung cancer.

5- Bone Scan

- Using technetium-99m methylene diphosphate

sensitivity 87% and specificity 67%

6- Magnetic Resonance Imaging MRI

- When tumors are adjacent to vertebral body and spinal canal
- Superior sulcus tumor, relationship to major vessels and brachial plexus

Fig. 23.— Coronal magnetic resonance imaging showing an adenocarcinoma in a young male infiltrating the aortopulmonary window. There is loss of the fat plane against the aorta (arrows) and invasion of the main pulmonary artery (arrowhead).



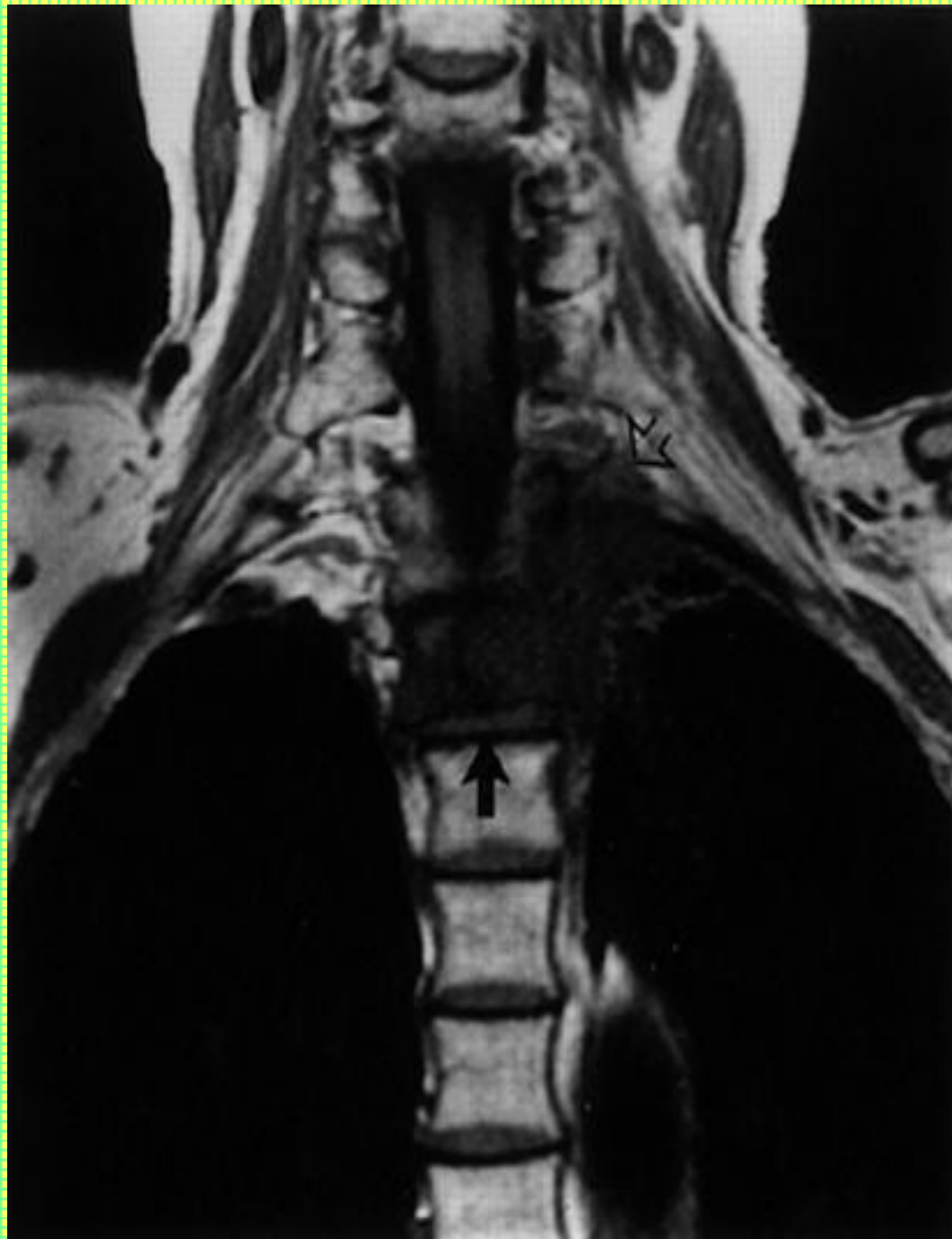


Fig. 24.— Coronal T1-weighted magnetic resonance imaging showing subtle Pancoast tumour (open arrow) with extension into the superior sulcus and erosion of the adjacent vertebral body (arrow).

Invasive Modalities

1- Bronchoscopy

- Rigid or flexible bronchoscopy with conventional white light
- Diagnosis, staging, resectability, visualization of bronchial tree
- Direct biopsy, brushing, saline lavage, transbronchial needle aspiration
- sensitivity 50% and specificity 96%

2- Autofluorescence bronchoscopy

- Normal mucosa : green
- Premalignant or malignant : brown-red

3- Percutaneous Transthoracic needle Biopsy

- CT guided is the best
- Central or peripheral lesions
- Relative contraindications:

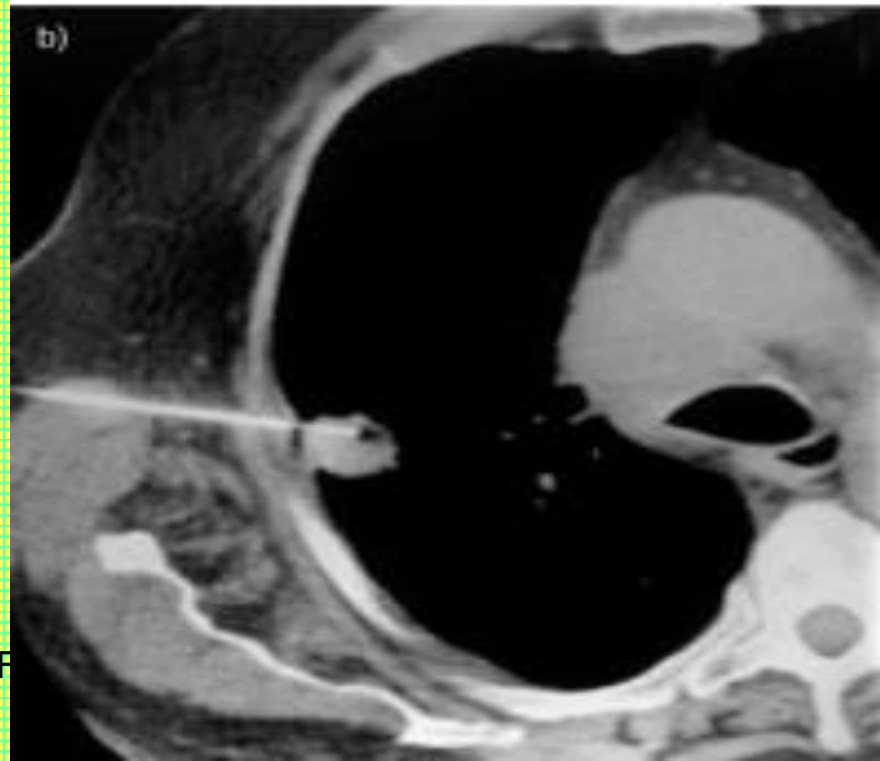
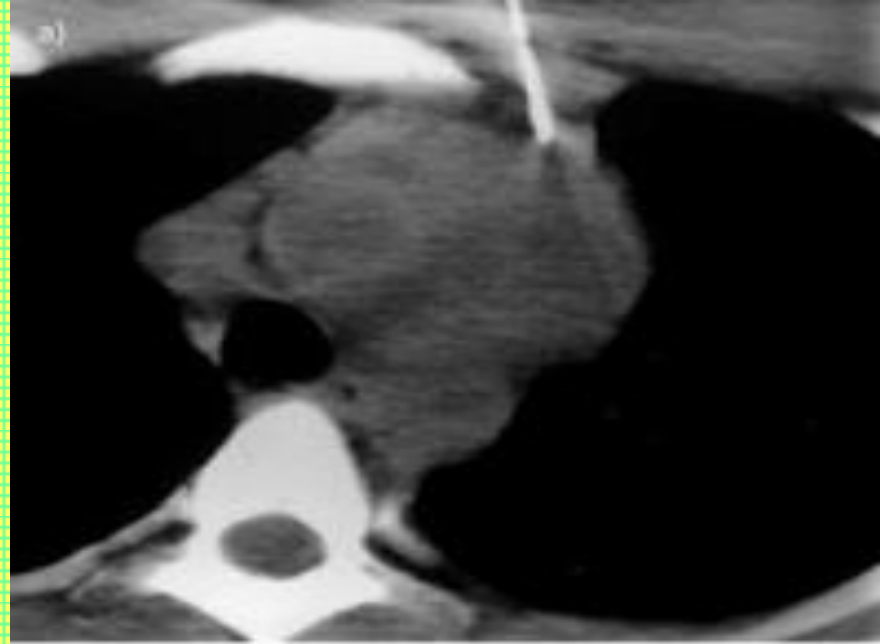
Severe COPD

Bleeding disorders

Contralateral pneumonectomy

Pulmonary hypertension

**Fig. 2.5— Versatility
of transthoracic
needle biopsy with
needle tip in a)
mediastinal mass
(note safe approach)
and b) peripheral
solitary nodule.**



4- Cervical Mediastinoscopy

- Most accurate prethoracotomy method of staging
- Access to superior mediastinum through avascular pretracheal space
- Sensitivity 81% and specificity 100%
- Is mediastinoscopy routine or not ??

5- Extended Cervical Mediastinoscop and Left Anterior Mediastinotomy

- For left upper lobe lung cancer

6- Scalene Node Biopsy

- To assess suspicious nodes in supraclavicular fossa
- To rule out N3 disease in patient with proven N2 disease
- Formal excision of the scalene fat pad may be performed

7- VATS

- Biopsy from both lung nodules and mediastinal lymph nodes
- Sensitivity and specificity 100%

8- Thoracotomy

- More than 95% of tumors can be characterized without thoracotomy

STAGING

TNM CLASSIFICATION

T (primary tumor)

- **Tx** Tumor that cannot be assessed, or malignant cells in sputum or bronchial washings but not visualized by imaging or bronchoscopy
- **Tis** Carcinoma in situ
- **T0** No evidence of primary tumor
- **T1** Tumor ≤ 3 cm, in a lobar bronchus or distal airways, and surrounded by lung or visceral pleura

- **T2** tumor that is either > 3 cm, involving the main bronchus (≥ 2 cm from the carina), invading the visceral pleura, or with atelectasis or obstructive pneumonitis that extends to the hilar region but does not involve the entire lung

- **T3** Tumor of any size that invade the chest wall (including superior sulcus tumors), diaphragm, mediastinal pleura, parietal pericardium; or tumor in the main bronchus < 2 cm from (but not involving) the carina; or associated atelectasis or obstructive pneumonitis of the entire lung

- **T4** tumor of any size that invades the mediastinum, heart, great vessels, trachea, esophagus, vertebral body, carina; or presence of malignant pleural / pericardial effusion; or satellite tumor nodules within ipsilateral primary-tumor lobe of the lung

N (Lymph Node)

- **Nx** regional LN cannot be assessed
- **N0** no regional LN metastasis
- **N1** metastasis to ipsilateral prebronchial and/or ipsilateral hilar LNs, and intrapulmonary LNs by direct extension
- **N2** metastasis to ipsilateral mediastinal and/or subcarinal LNs
- **N3** metastasis to contralateral mediastinal, contralateral hilar, scalene or supraclavicular LNs

M (distant metastasis)

- **Mx** distant metastasis cannot be assessed
- **M0** no distant metastasis
- **M1** distant metastasis present

STAGING

Stage 0: Tis N0 M0

Stage I A: T1 N0 M0

I B: T2 N0 M0

Stage II A: T1 N1 M0

II B: T2 N1 M0

T3 N0 M0

Stage III A: T3 N1 M0

T1-3 N2 M0

III B: T4 N0-2 M0

any T N3 M0

Stage IV : any T any N M1

Treatment options

Occult lung cancer

- Tumor not evident on radiological imaging but discovered either incidentally at bronchoscopy or sputum cytology
- If localized: Surgical resection
5-years survival 100%
- If not localized: photodynamic therapy

Stage I (T1 N0, T2N0)

- Surgical resection: Lobectomy, pneumonectomy, sleeve resection if central lesion
- No adjuvant treatment following complete resection
- 5-year survival 70%

Stage II (T1 N1, T2N1)

- Surgical resection: Lobectomy, pneumonectomy, sleeve resection if central lesion
- Post resection adjuvant treatment is controversial
- 5-year survival 40%

Stage II (T3 N0)

- Surgical resection + adjuvant chemo and radiotherapy
- Tumors invading chest wall: en bloc resection and chest wall reconstruction
 - Complete resection: 4-year survival 35%
 - Incomplete resection: 5-year survival 0%
- Superior sulcus tumors : the same
 - Complete resection: 5-year survival 40%
 - Incomplete resection: 5-year survival 10%

- **Tumors proximal to Carina:
Sleeve resection with
5-year survival 35%**

- **Tumors invading the mediastinum:
Partial resection + implantation of
isotopes + external radiotherapy
5-year survival 9%**

Stage IIIA (T3 N1)

- Complete resection + chemo and radiotherapy
5-year survival < 20 %

Stage IIIA (T3 N2)

- The most controversial stage between all authors

Stage III B (T4 or N3)

- Considered inoperable
- Some authors encourage primary chemo and radiotherapy then reevaluation

Stage IV (M1)

- Brain metastasis:
Resection of the primary tumor and the solitary metastasis

If both are detected simultaneously , which one done first ??

SMALL CELL LUNG CANCER

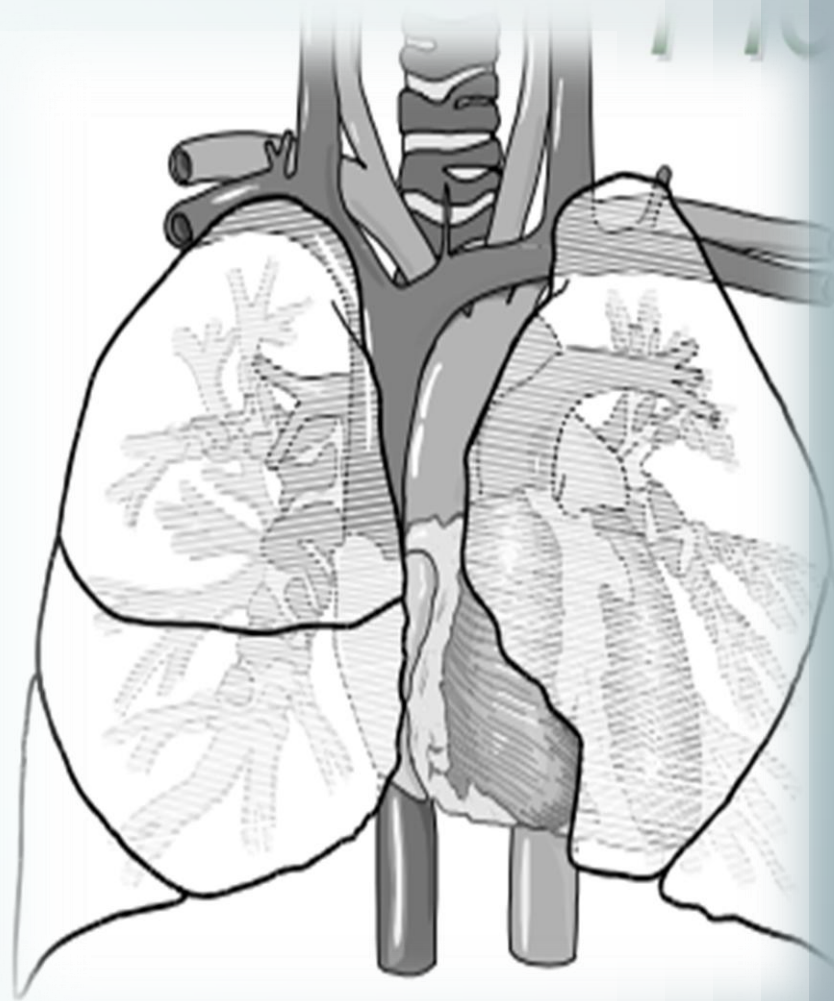
- Mean survival 10 months
- 5-year survival 5 %
- Standard therapy is chemotherapy
- Resection only if solitary nodule + multiple courses of chemotherapy + thoracic irradiation + prophylactic cranial irradiation

Palliative Resections

- Lung abscess
- Massive hemoptysis
- Chest wall invasion

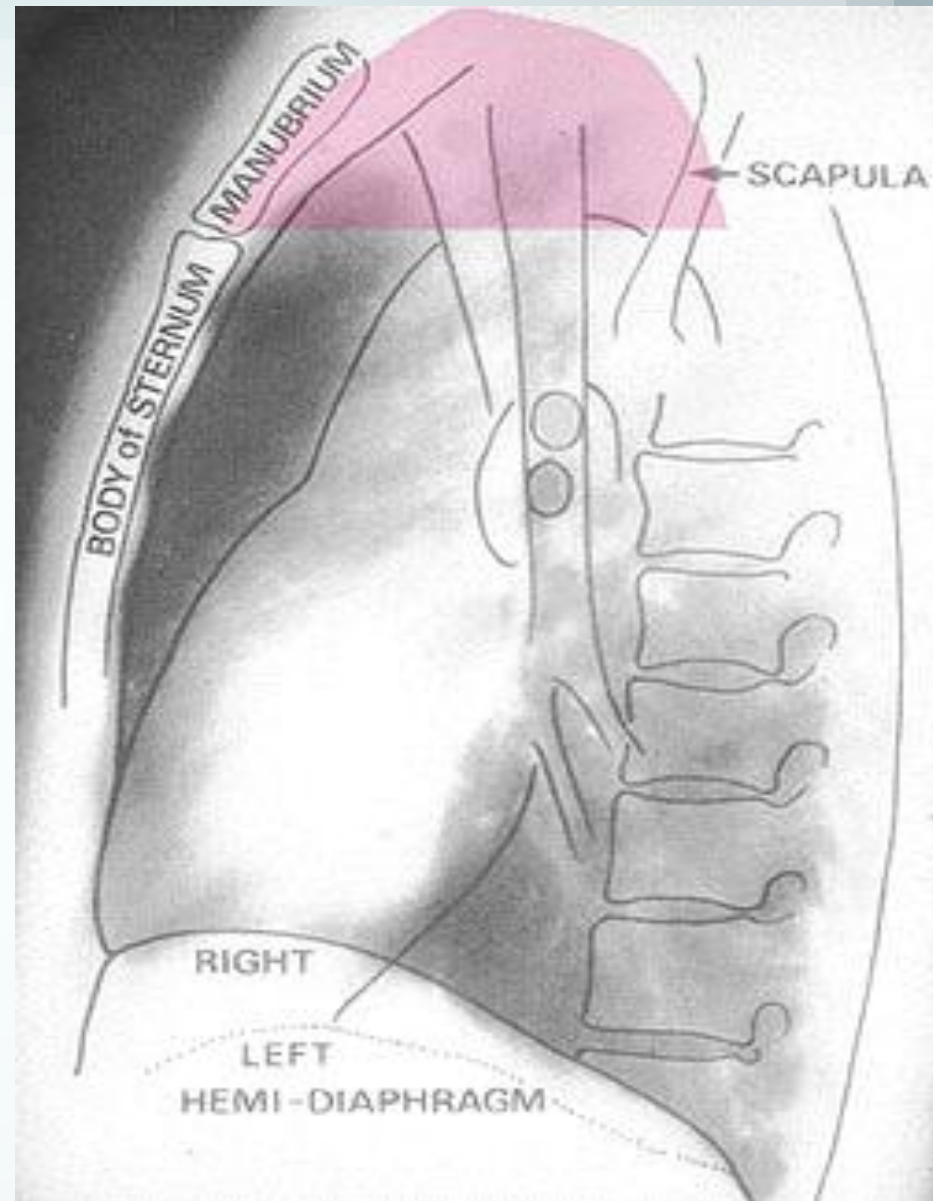
Mediastinal syndrome

The mediastinum is a space which lies between the **pleura** in and near the median sagittal plane of the chest. It extends from the sternum in front to the vertebral column behind, and contains all the thoracic viscera except the **lungs**.



It may be divided for purposes of description into two parts:

- **Upper portion:** above the upper level of the pericardium, which is named superior mediastinum with its inferior limit at the plane from the sternal angle to the disc of T4-T5.



•**lower portion :**

below the upper level of the pericardium.

This lower portion is again subdivided into three parts:

- 1. In front of the pericardium, the anterior mediastinum.**
- 2. One containing the pericardium and its contents, the middle mediastinum.**
- 3. That behind the pericardium, the posterior mediastinum**

Superior mediastinum

It **contains** the

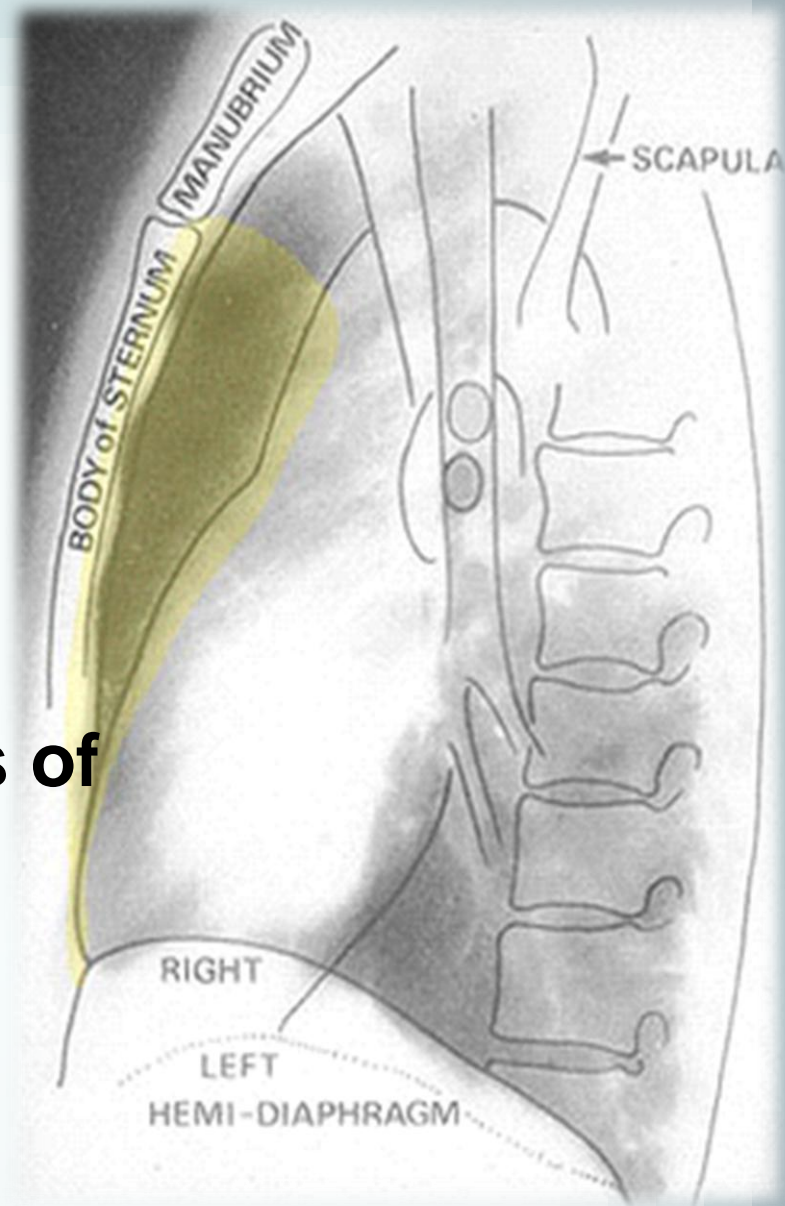
- **Origins of the Sternohyoidei and Sternothyreoidei**
- **lower ends of the Longi colli;**
- **Aortic arch; the innominate artery and the thoracic portions of the left common carotid and the left subclavian arteries;**

- **Innominate veins and the upper half of the superior vena cava**
- **Vagus , cardiac, phrenic, and left recurrent nerves;**
- **Trachea**
- **Esophagus**
- **Thoracic duct;**
- **Remains of the thymus,**
- **some lymph glands.**

Anterior mediastinum.

It contains:

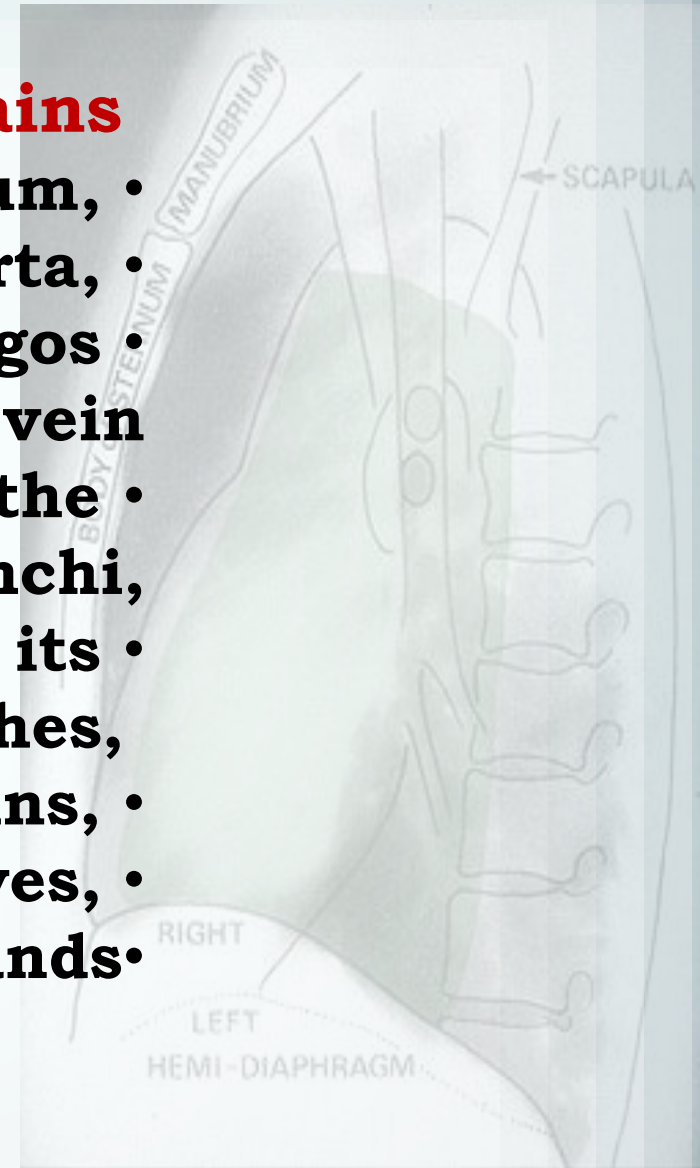
- Quantity of loose areolar tissue,
- Some lymphatic vessels
- Small mediastinal branches of internal mammary artery.



Middle mediastinum

It contains

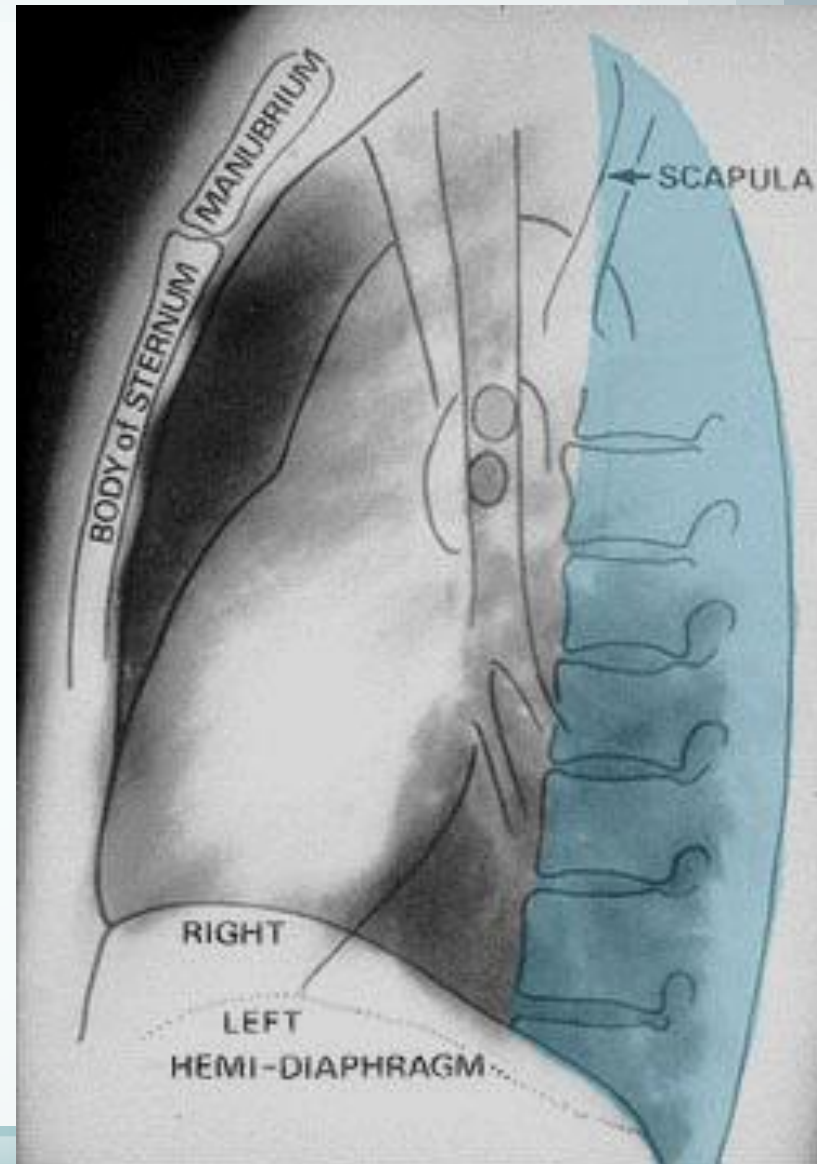
- Heart enclosed in the pericardium, •**
- Ascending aorta, •**
- Lower half of SVC with the azygos •**
vein
- Bifurcation of the trachea and the •**
two bronchi,
- Pulmonary artery dividing into its •**
two branches,
- Right and left pulmonary veins, •**
- Phrenic nerves, •**
- Some bronchial lymph glands•**

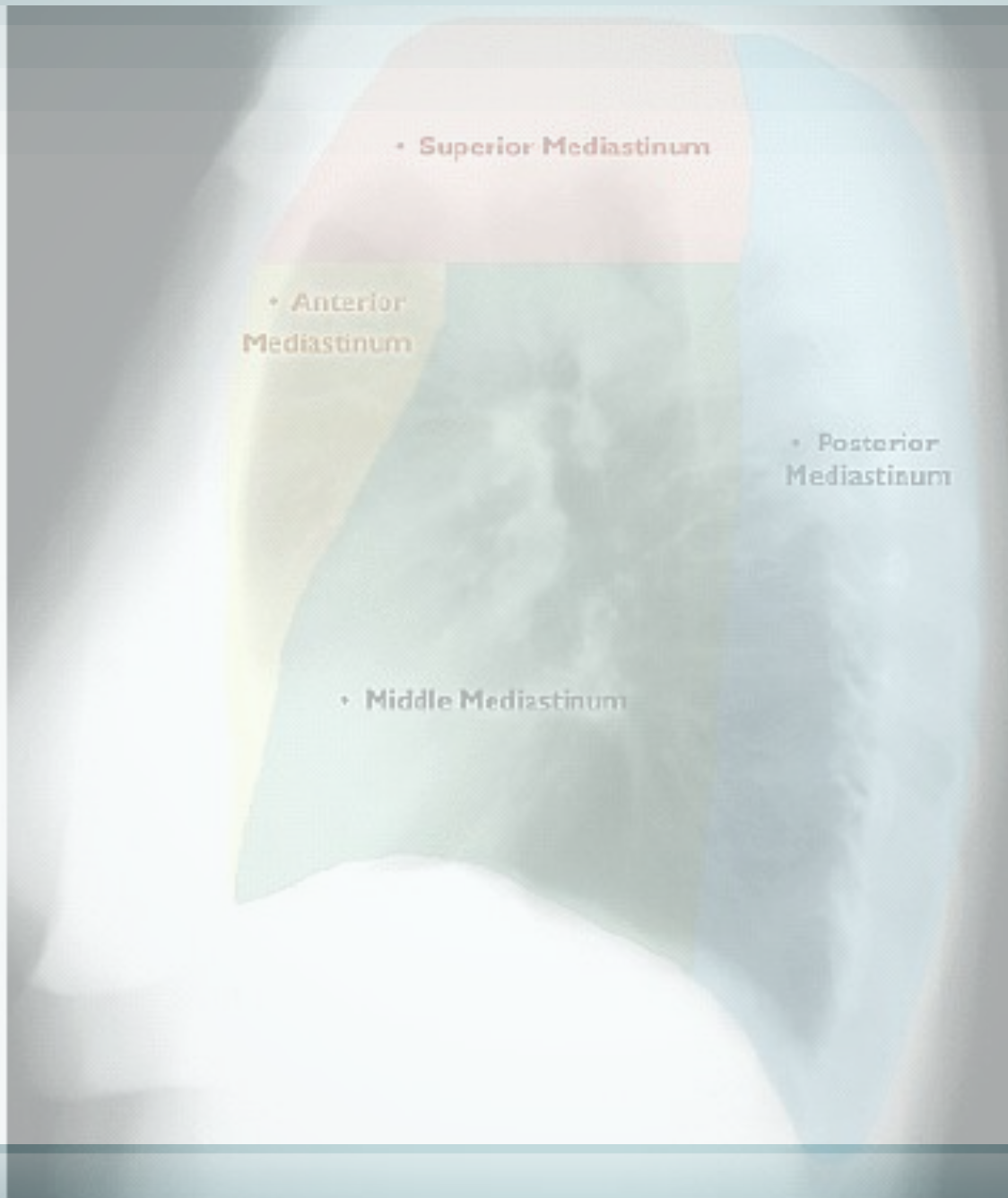


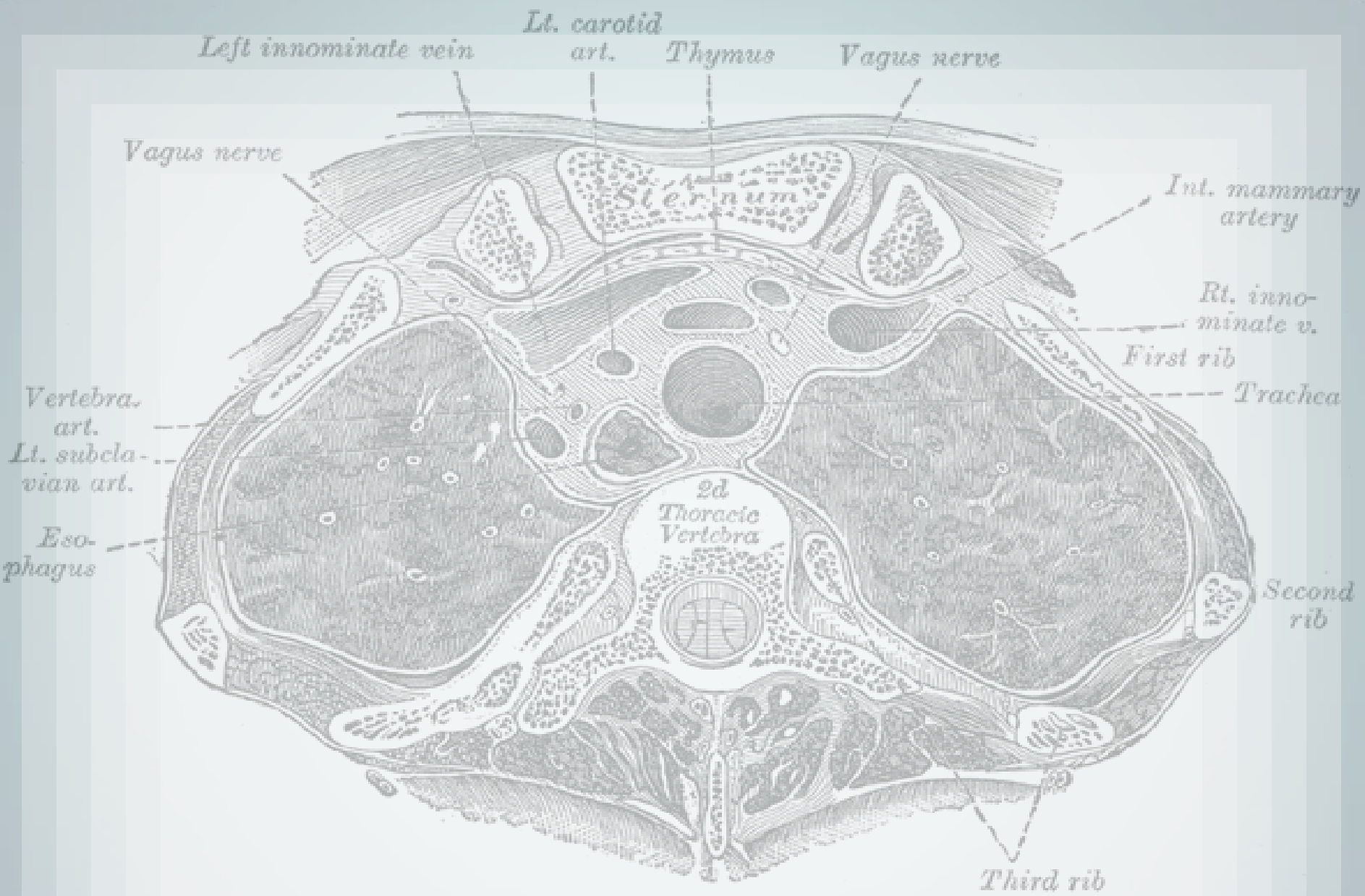
Posterior Mediastinum

It **contains**

- Thoracic part of the descending aorta,
- Azygos and the two hemiazygos veins,
- Vagus and splanchnic nerves,
- Esophagus
- Thoracic duct,
- Some lymph glands.







Transverse section through the upper margin of the second thoracic vertebra.

**Any space occupying lesion
in the mediastinum leads to
what is called**

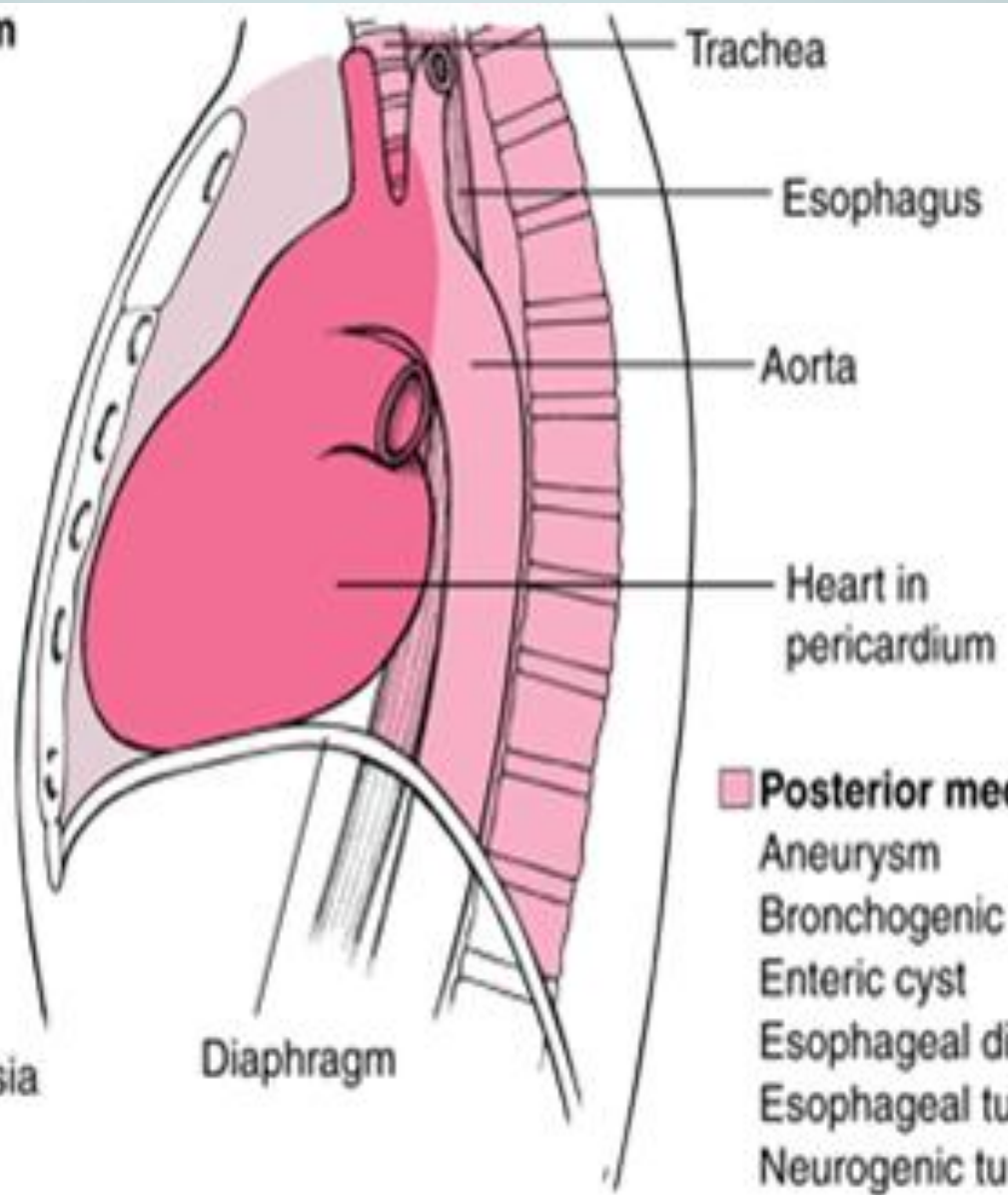
Mediastinal Syndrome

■ **Anterior mediastinum**

- Aneurysm
- Angiomatous tumor
- Goiter
- Lipoma
- Lymphoma
- Morgagni hernia
- Parathyroid tumor
- Pericardial cyst
- Teratoma
- Thymoma
- Thyroid tumor

■ **Middle mediastinum**

- Bronchogenic cyst
- Bronchogenic tumor
- Lymph node hyperplasia
- Lymphoma
- Pleuropericardial cyst
- Vascular masses



■ **Posterior mediastinum**

- Aneurysm
- Bronchogenic tumor
- Enteric cyst
- Esophageal diverticula
- Esophageal tumor
- Neurogenic tumor

Clinical picture:

A) Asymptomatic

B) Compression manifestations:

1-SVC:

**•Face Puffiness , headach, convulsions,
cough (tracheal oedema) and dilated non-
pulsating veins**

2- Trachea and bronchi:

- **Cough , wheez, dyspnea, pneumonia**

3-The oesophagus:

- **Dysphagia**

4-The nerves:

- **Horner's syndrome, arrhythmias , GIT symptoms, hoarsness of voice and diaphragmatic paralysis**

5-aortic arches :

- **pulse inequality or clubbing.**

6-Thoracic duct and pleura:

- **Chylouthorax**

Investigations

✿ According to the suspected cause:

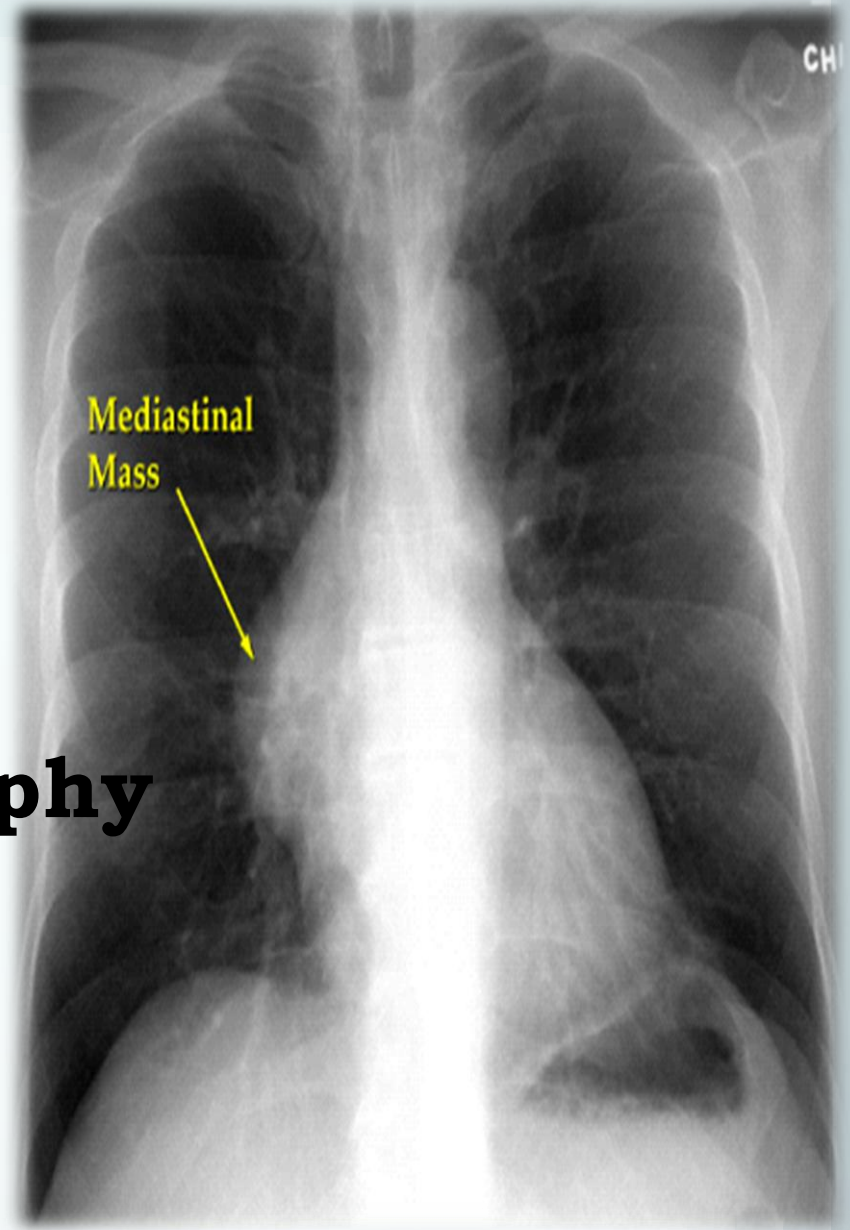
✿ Chest X- ray

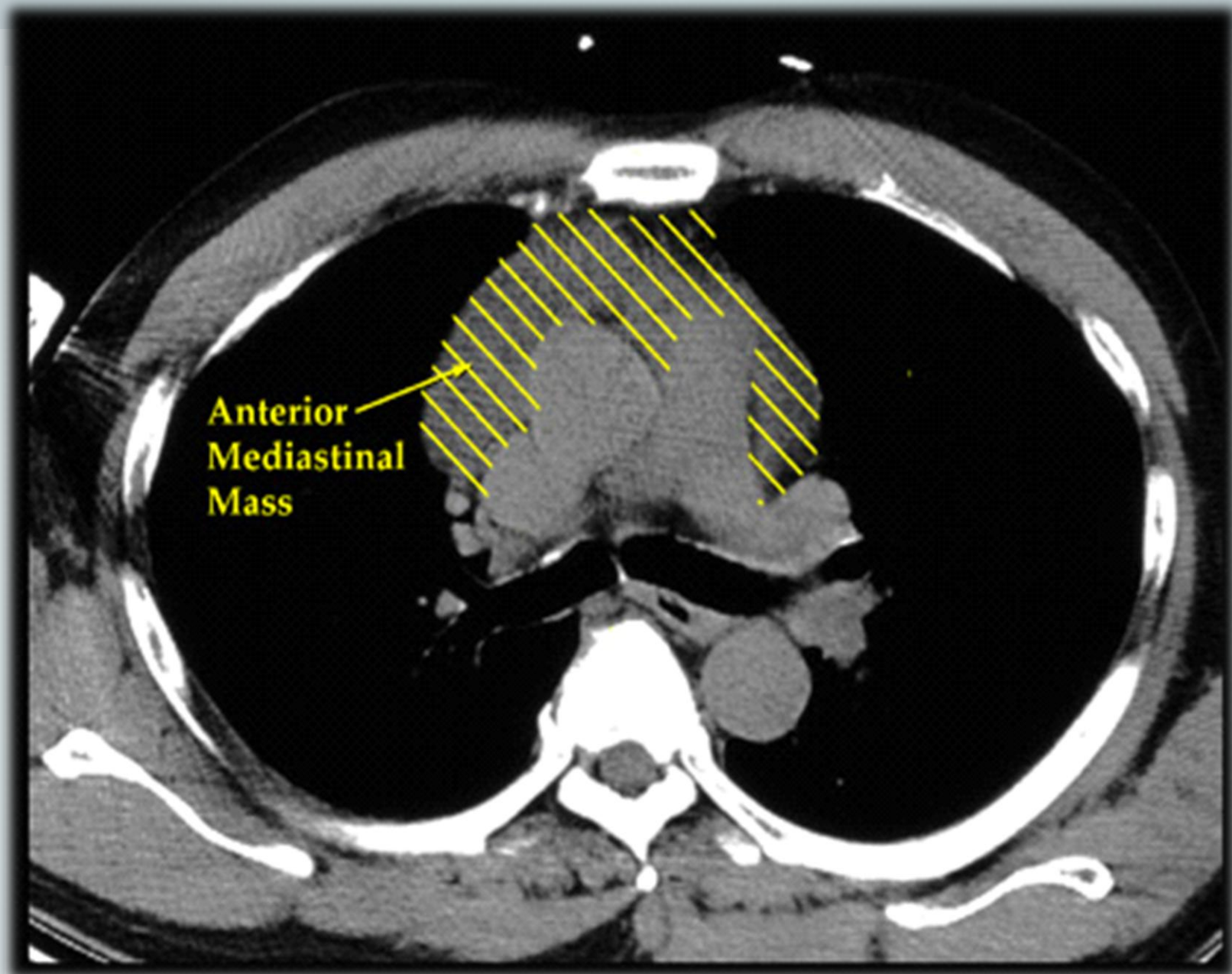
✿ CT chest

✿ Barium swallow

✿ Thoracic aortography

✿ MRI





Treatment

- **If asymptomatic with benign nature:
leave without treatment (unless
complicated)**

• **According the aetiology:**

- ✓ **Surgical removal or correction of anomaly**
- ✓ **Needle aspiration as for cystic lesions**
- ✓ **Oxygen**
- ✓ **+ve radioactive iodine isotop scan as for retrosternal thyroid**
- ✓ **Sclerotherapy as for lymphangioma**

Thank you