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Primary and secondary tuberculosis

CLINICAL FORMS

- **TB of respiratory organs**

  Primary tuberculous complex.

  Disseminated lung tuberculosis.

  Nidus lung tuberculosis.

  Infiltrative lung tuberculosis.

  Caseous pneumonia.

  Lung tuberculoma.

  Lung fibrous-cavernous tuberculosis.

  Lung cirrhotic tuberculosis.

CLINICAL FORMS

- **TB of exstrarespiratory organs**

TB of bronchi, trachea and upper respiratory tract.
TB of intrathoracic lymphatic nodes.
TB pleurisy (including empyema).
TB of brain tunics and the central nervous system.
TB of bones and joints.
TB of urinary and sexual organs.
TB of intestine, peritoneum, mesenteric lymphatic nodes.
Miliary tuberculosis.
TB of other organs and systems.

CHARACTERISTIC OF TUBERCULOUS PROCESS

- **Localization and spreading:** Localization of defects in lungs according to the numbers (names) of segments, names of lung sections, and in other organs and systems - according to anatomical names of localization of a wound.
- **Presence of destruction:** Destr+ Destr-
- **Facultative it is necessary to specify phase of process:**

  - infiltration, decay, sowing;
  - suction, condensation, scarring, calcination

ETIOLOGIC METHOD OF CONFIRMATION:

- **(MBT +) – it is confirmed by results of bacteriological analysis (cipher code A 15), in this case to specify:**
- **M+ positive result of sputum analysis on acid-resisting bacteria (ARB)**
- **C0 – cultural analysis wasn't done**
- **C- negative result of cultural analysis**
- **C+ positive result of cultural analysis, in this case to specify:**

- **(Resist 0)** MBT resistance to preparations of I line wasn't analyzed;
- **(Resist -)** resistance to preparations of I line hasn't been established;
- **(Resist +)** (abbreviation of antitubercular preparations of I line) resistance to preparations of I line has been established (in brackets to list all the preparations of I line to which resistance has been determined)
- **(Resist II-)** resistance to preparations of II line hasn't been established;
- **(Resist II+)** resistance to preparations of II line has been established (in brackets to list all the preparations of I line to which resistance has been determined)

Types of TB cases

- ***New case of TB*** – A patient who has never been treated for TB or has taken anti-TB drugs for less than one month.
- ***Previously treated case of TB*** – A patient who has been treated for one month or more with anti-TB drugs in the past. Retreatment cases are further classified by the outcome of their most recent course of treatment into four categories.

Previously treated case of TB

- 1. ***Relapse*** patients have previously been treated for TB, were declared cured or treatment completed at the end of their most recent course of treatment, and are now diagnosed with a recurrent episode of TB (either a true relapse or a new episode of TB caused by reinfection).
- 2. ***Treatment after failure*** patients have previously been treated for TB and their most recent course of treatment failed i.e. they had a positive sputum smear or culture result at month 5 or later during treatment.
- 3. ***Treatment after loss to follow-up*** patients have previously been treated for TB and were declared 'lost to follow-up' at the end of their most recent course of treatment.
- 4. ***Other previously treated patients*** are those who have previously been treated for TB but whose outcome after their most recent course of treatment is unknown or undocumented.

- Case of ***multidrug-resistant TB (MDR-TB)*** – TB that is resistant to two first-line drugs: isoniazid and rifampicin.
- Case of ***rifampicin-resistant TB (RifTB)*** – A patient with TB that is resistant to rifampicin detected using phenotypic or genotypic methods, with or without resistance to other anti-TB drugs. It includes any resistance to rifampicin, whether mono-resistance, multidrug resistance, polydrug resistance or extensive drug resistance.
- Case of ***extremaly drug-resistant TB (XDR-TB)*** – TB, that is resistant to isoniazid, rifampicin, at least one fluoroquinolone and aminoglycosid.
- Case of **risk of MDR (RMDR)** – TB in cases, while patient has contact with MDR patient, but hasn't results of bacteriological investigation yet, or has negative bacteriological result.

Clinical forms of pulmonary tuberculosis

- There such clinical forms of pulmonary TB, as milliary, disseminated, focal, infiltrative, tuberculoma, caseous pneumonia, fibrous-cavernous, cirrhotic tuberculosis, primary tuberculosis complex.

Clinical forms of extra-pulmonary tuberculosis

- It depends on the affected organ. Miliary tuberculosis, tuberculosis of intrathoracic lymph nodes, bronchial TB, pleural effusion considers as pulmonary process in lung lesions cases.

Phases of TB

- There are such TB process phases: infiltration, decay (corresponding Destruction +), contamination, resorption, seals, scarring and calcification. Infiltration, decay and contamination characterize tubercular activity changes in patients. Resorption, seals, scarring and calcification (calcination) means dicreasing of active tuberculosis process in dynamics with a tendency to stabilization.

Diagnosis examples

1) New case of TB (01.02.2016) upper lobe of right lung (infiltrative), contamination phase, Destr +, MBT+, M+, MG+, Rif-, C+, Resist-, Hist0, Cat 1, Coh 1 (2016).
2) Relapse of TB (01.04.2016) lungs (disseminative), infiltration phase, Destr-, MBT+, M-, MG+, Rif-, C0, Resist0, Hist0, Cat 2, Coh 2 (2016).
3) MDR-TB (05.12.2015) left lung (caseous pneumonia), contamination phase, Destr+, MBT+, M+, MG0, Rif0, C+, Resist+ (HRES), Resist 2+ (EtOfx), infiltrative TB of B1B2B6 (bronchi) of right lungs with 2 stage of B2B6 stenosis, Hist0, Cat 4 (New case of TB), Coh 4 (2015).

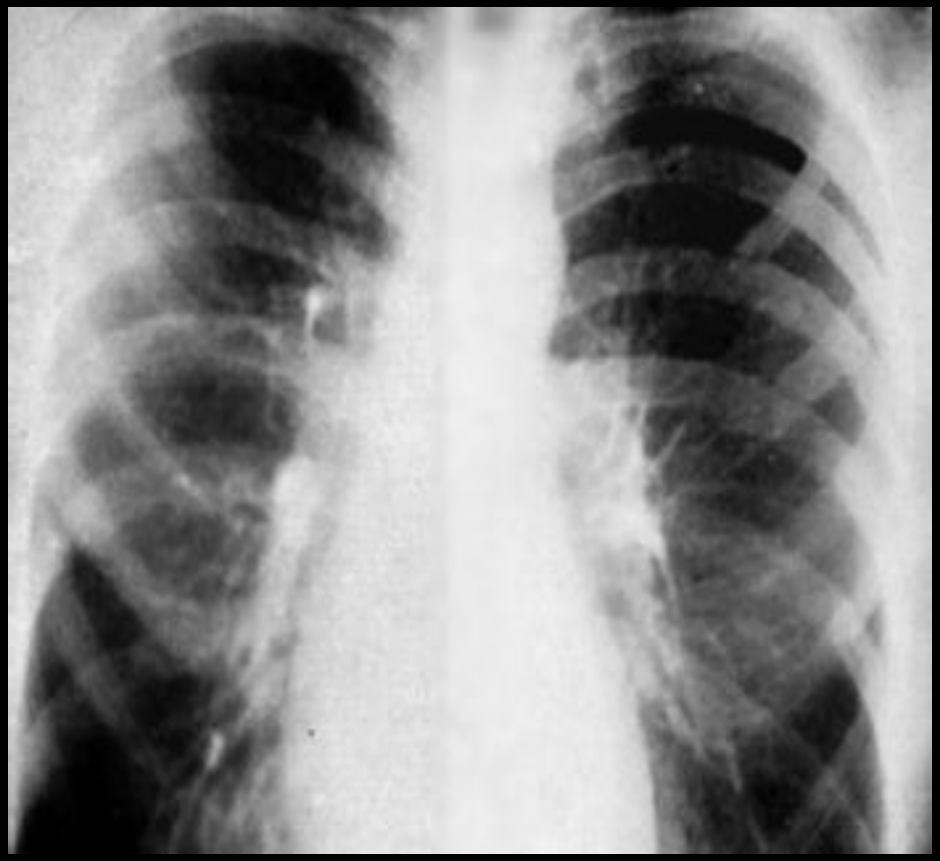
RADIOLOGICAL SYNDROMS

- To explain radiological features of tuberculosis clinical form we must understand radiological syndroms. There are 10 syndroms: abnormal pulmonary pattern, lung roots pathology, focal shadow, infiltrative shadow, disseminative syndrome, rounded shadow, ring-like shadow, increased enlightenment of the lung fields, mediastinal pathology and free fluid in the pleural cavity.

Abnormal pulmonary pattern syndrome

- ***Increased and enriching the lung picture*** (at inflammatory processes, collagenous diseases, tumor, pneumoconiosis, sarcoidosis, vascular lesions with symptoms of congestion and interstitial edema);
- ***deformation of lung pattern*** (at formation of inflammatory infiltrates, peri-bronchial inflammation, cicatricial due to wrinkling certain segments, the interparticle pathology and partial internal connective tissues, lung`s fibrosis with chronic venous stasis, the appearance of a thin mesh picture at hemosiderosis, the formation of numerous small ring shadows at scleroderma);

Abnormal pulmonary pattern syndrome

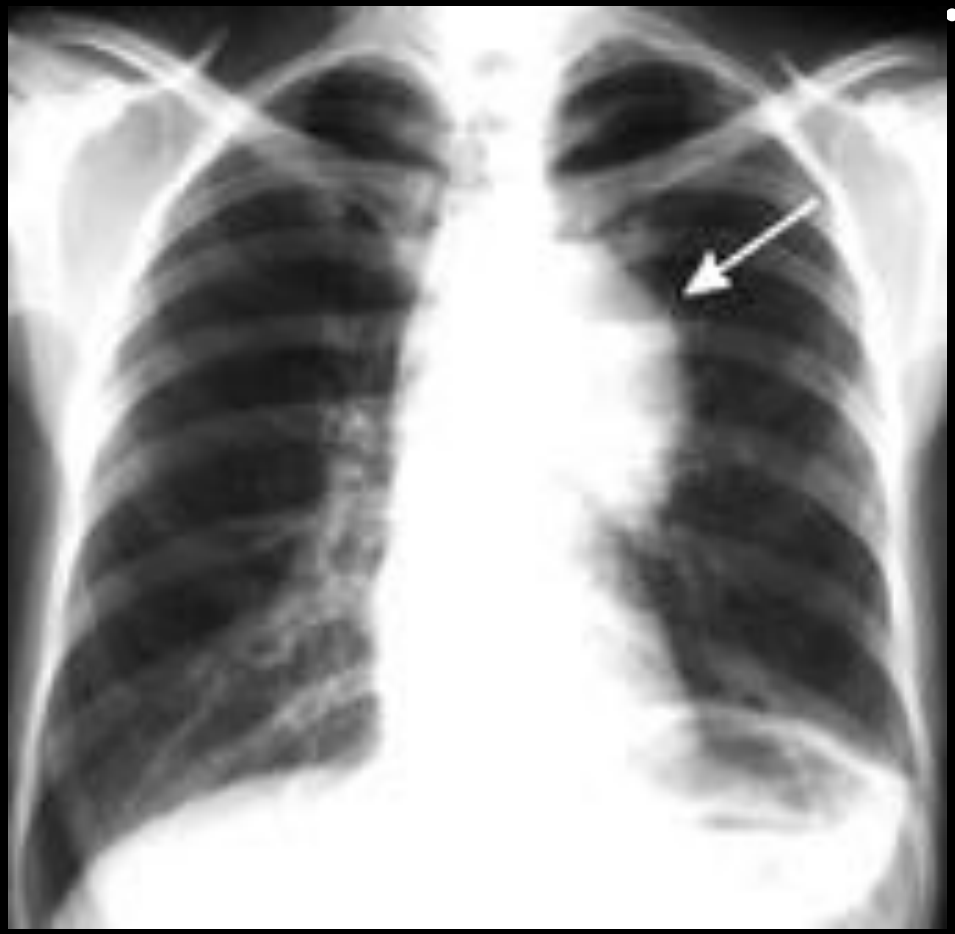


- ***weakening of lung`s pattern*** (in diffuse pulmonary dissemination, development of numerous small cavities);

Abnormal pulmonary pattern syndrome

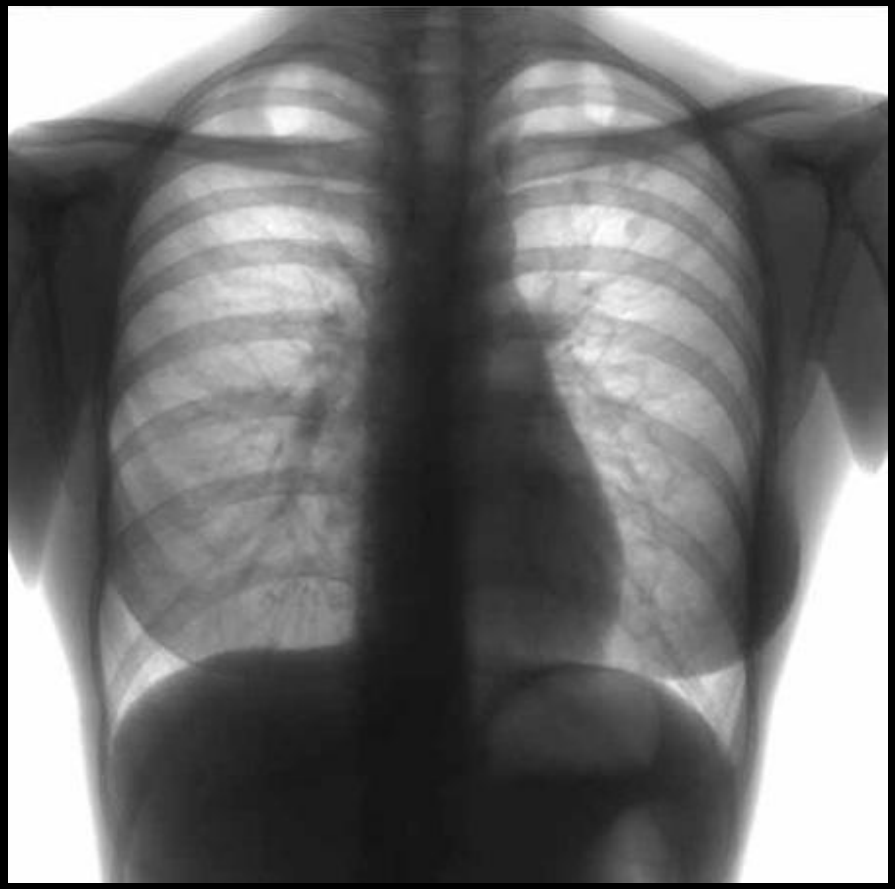
- ***depletion of the picture*** (at inflating the lungs, lung arterial nets hypoplasia);
- ***unusual elements of the picture***

Lung`s roots pathology



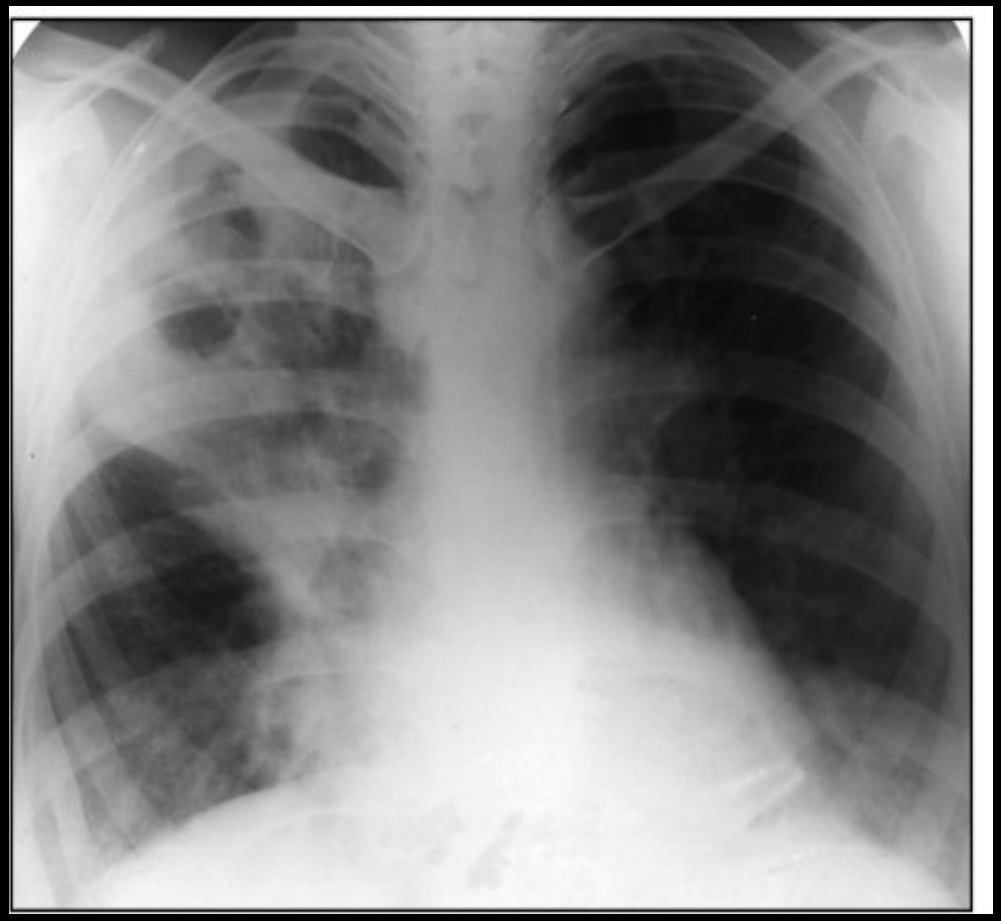
- Manifested with increase, deformation, increase the intensity and root of the lung shade structures violation, associated with vascular or bronchial lymph nodes disorders. Changes root of the lung occur when tuberculosis internal thoracic lymph nodes, sarcoidosis, lymphosarcoma, central cancer, lymphogranulomatosis, nonspecific inflammation (basal pneumonia), aortic aneurysm, expanding the trunk of pulmonary blood vessels in heart diseases with hypertension in the pulmonary circulation (mitral stenosis) and at primary pulmonary hypertension, benign tumors (thymoma), retrosternal goiter, acute childhood diseases (measles, scarlet fever) and others.

Focal shadow



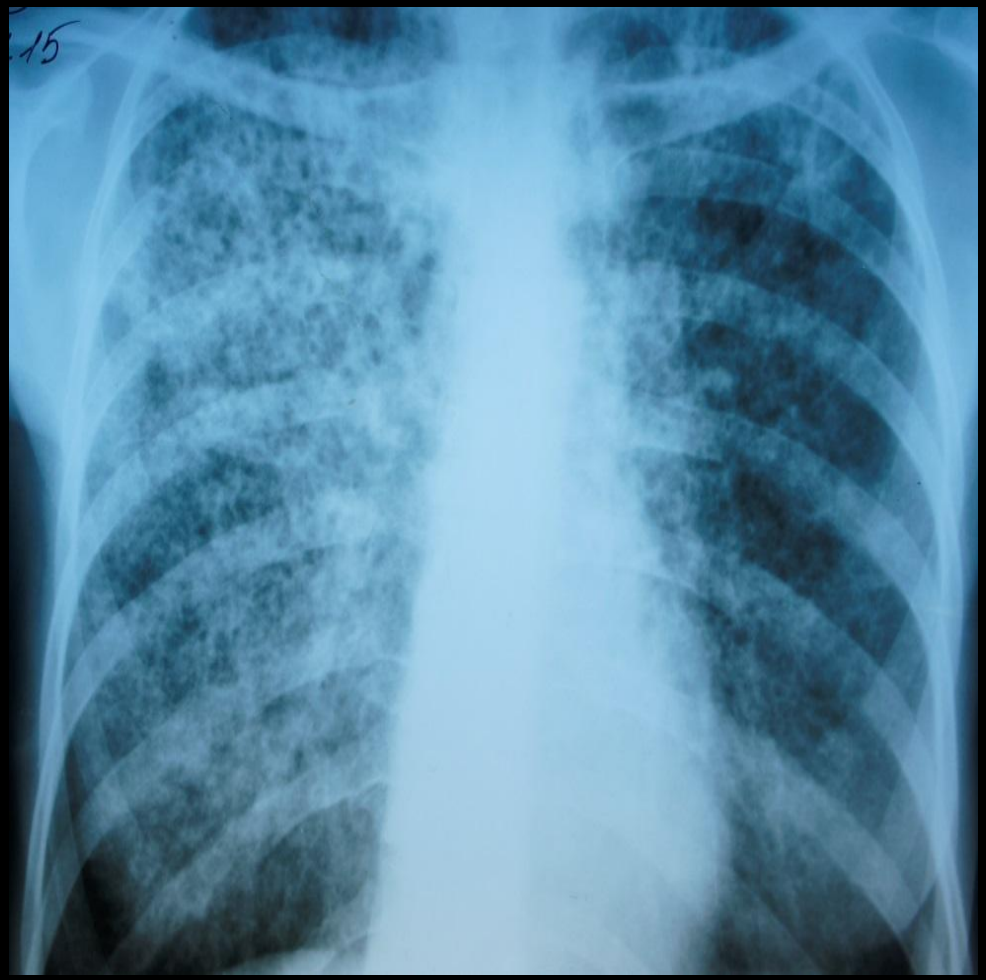
- Characterized by one or more shades (up to 10), round or irregular shapes, up to 1 cm in diameter, which can have a different intensity and are usually placed in a limited space in one or both lungs. Symptom “focal shadow” is a manifestation of many diseases, occurring with lesions of the lung parenchyma. Inflammation (bacterial and viral acute pneumonia, tuberculosis, fungal lesions), benign and malignant tumors, vascular disorders, collagen diseases, blood diseases, reticular and lymphoid tissues are the main pathological processes that accompanied the emergence of focal shadows, the formation of which results in the local disappearance of air from the

Infiltrative shadow



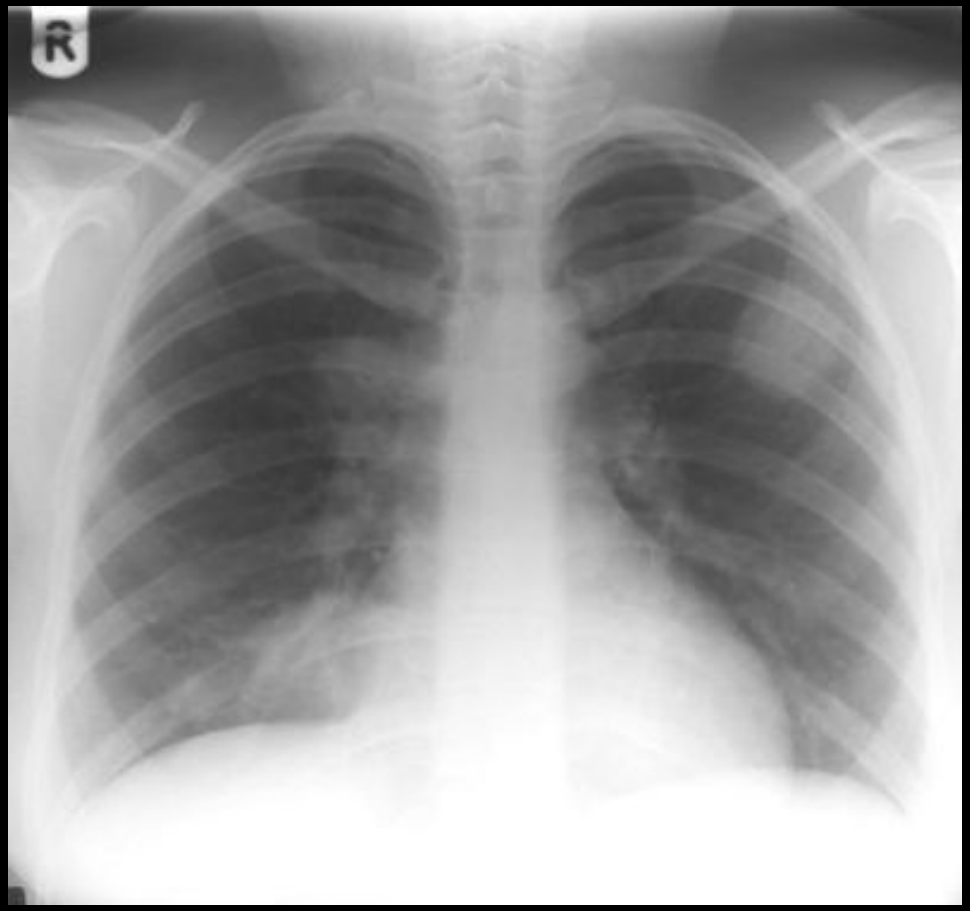
- This syndrome characterized by shadow areas of more than 1 cm, round or irregular shape, which has no clear contours. Depending on the severity there are syndrome “limited infiltrative shadow” in size from lobules up to lobe, and the syndrome of “total infiltrative syndrome”, which is characterized by the size of the shadow over 1 share for total blackout of all lung fields. Infiltrative changes in the lungs are the most widespread (50 %) among other pulmonary diseases. Causes of this syndrome may be inflammation, tumor process, atelectasis, pulmonary infarction, hematoma, accompanied by hypoventilation. This syndrome can develop at congenital defects – lobe hypoplasia and aplasia.

Disseminative syndrome



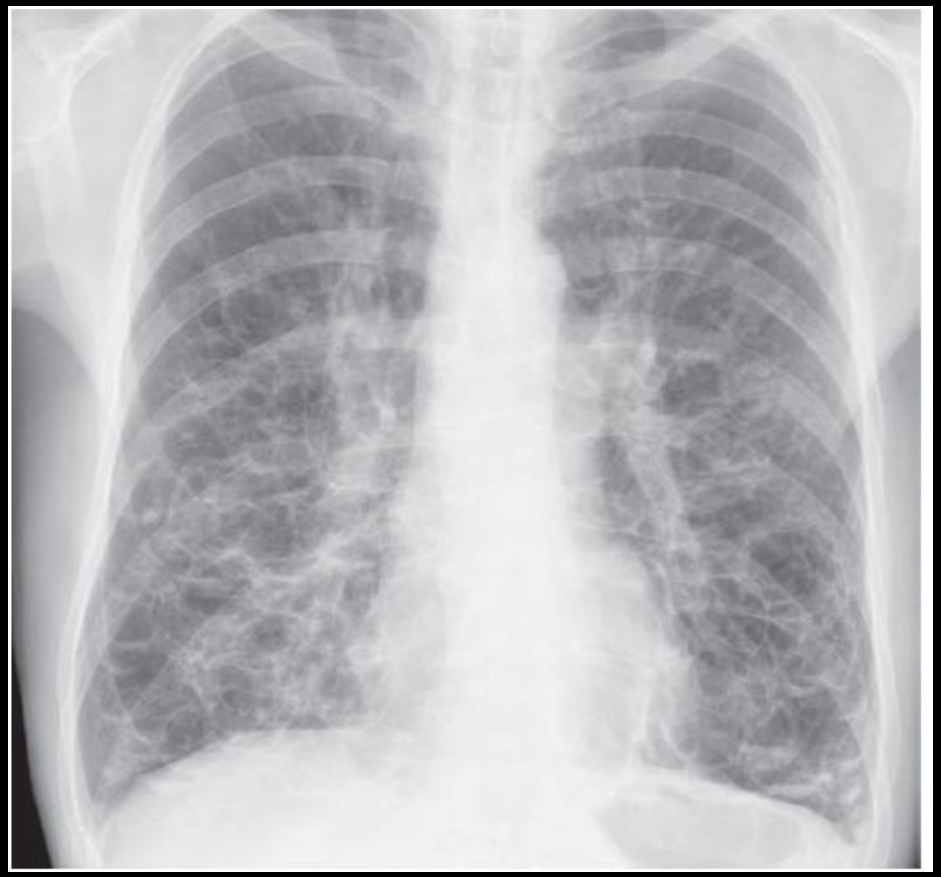
- Characterized by multiple focal and retinal shadows of varying intensity to 1 cm in diameter, that are placed on a large lung`s length the and are usually bilateral. There are more than 200 diseases of different aetiology and genesis, accompanied by disseminative syndrome in the lungs. Depending on the etiology and pathogenesis all diseases are divided into: 1) infectious-inflammatory (bacterial, viral, mycobacterial, fungal), 2) the tumor; 3) parasitic; 4) pneumoconiosis; 5) allergic; 6) collagenoses; 7) an inhaled and aspiration; 8) congenital constitutional; 9) metabolic-toxic; 10) reticulo-endothelial and hematopoietic; 11) cardiovascular; 12) traumatic; 13) unknown etiology.

Rounded shadow



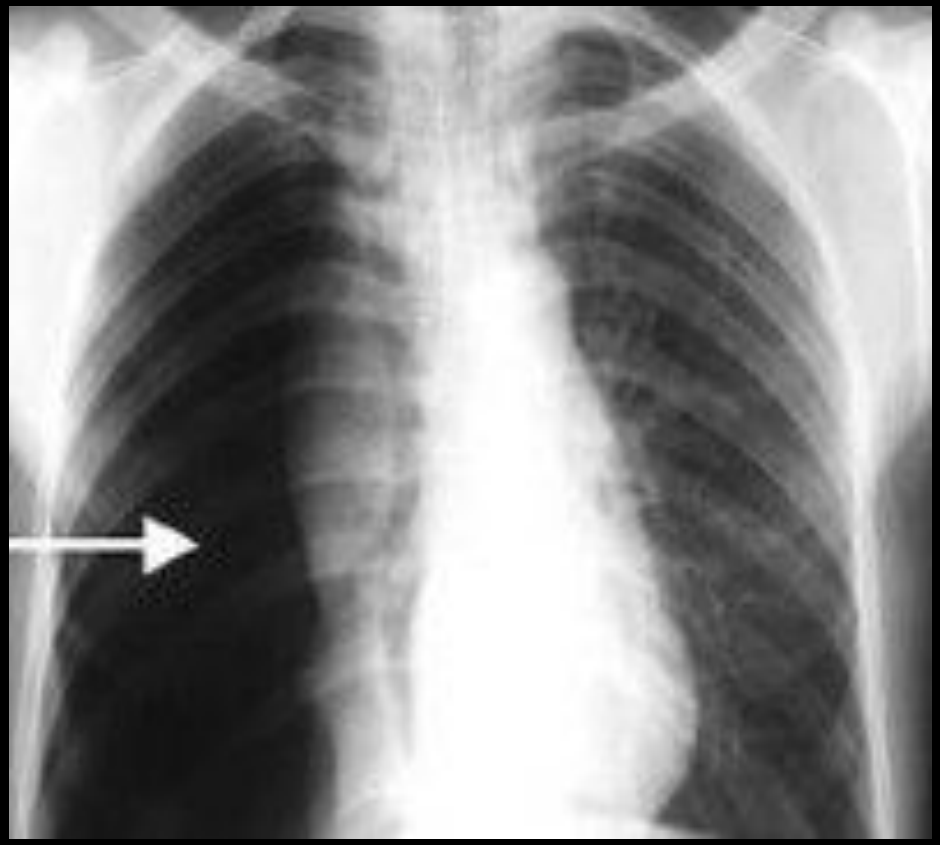
- Characterized by volume spherical or oval formation of correct, incorrect or polycyclic forms with clear or blurred contours more than 1 cm in diameter. It may be tuberculoma, nonspecific round pneumonia, eosinophilic rounded infiltrate, cancer, benign tumors (neurinoma, hemangioma, arteriovenous aneurysm, adenoma, veins varicose), tumors of bronchi, asperhiloma, retention cyst, Echinococcus

Ring-like shadow



- It characterized by rounded enlightenment, which is surrounded by a ring-liked shadow. Enlightenment in the lung may be due to lack of lung tissue and replacement of air with restriction tissue defect from the surrounding areas of wall or capsule. The cavity of the lungs may have primary (congenital cysts air, emphysematous bullae, bronchiectasis) or secondary (decay or inflammatory infiltrate tumors, cleaning parasitic cysts, tumors traumatic penetration in the interstitial tissue) native.

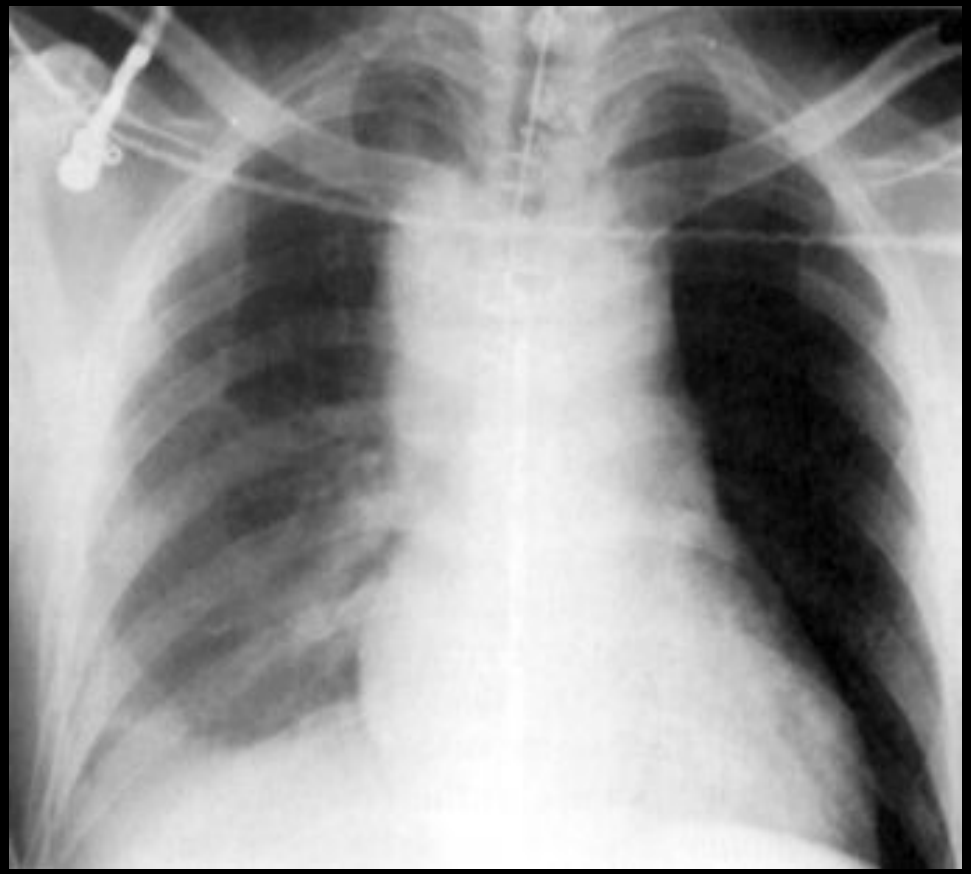
Increased enlightenment of the lung fields



- It includes varying prevalence of enlightenment, not limited by ring-like shadow and is located in the lungs or in the pleural cavity. Symptom can be caused by: defects in lung tissue (pneumothorax);

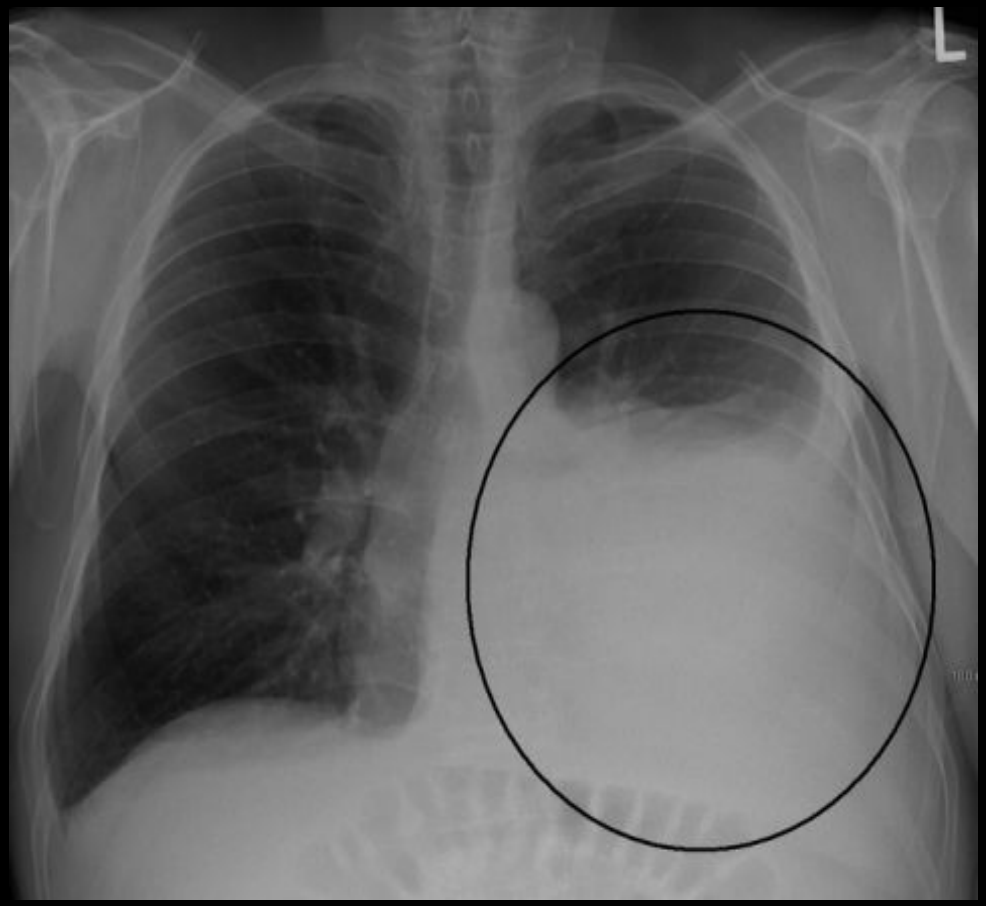
degenerative-dystrophic changes of intrapulmonary bronchial artery branches with presence of capillary or venous stasis; violation of bronchial patency as a result of chronic inflammation in them, increased viscosity of bronchial secretions, bronchial compression for inflammatory, neoplastic and sclerotic processes in the lung parenchyma; congenital bronchial pathology; adaptive reactions after lung

Mediastinal pathology syndrome



- It manifested by changing the mediastinum form or position. This syndrome may be present at fibrosis, cirrhosis, after lung`s resection, lung`s agenesia, lung atelectasis, in the presence of air or fluid in the pleural cavity, at diaphragmatic hernia, sometimes in large lung tumors or giant lungs cyst cases, at the bronchial tumors and enthetic bodies. Mediastinum forms changing (extension) may be at mediastinal cysts and tumors, inflammatory processes (acute, chronic, mediastinit encysted abscess of the mediastinum).

Free fluid in the pleural cavity



- It characterized by a one- or two-way shadow areas of different sizes, with the predominant placement in the lower divisions, with oblique upper limit. Depending on the position of the body section blackout can change the location. Pleural effusions are divided into exudate and transudate. Transudate resulting in increase of capillary pressure, or in decrease of oncotic pressure of blood plasma. The transudate nature pleural fluid has in congestive heart failure, liver cirrhosis, hydrothorax, glomerulonephritis, myxedema cases.

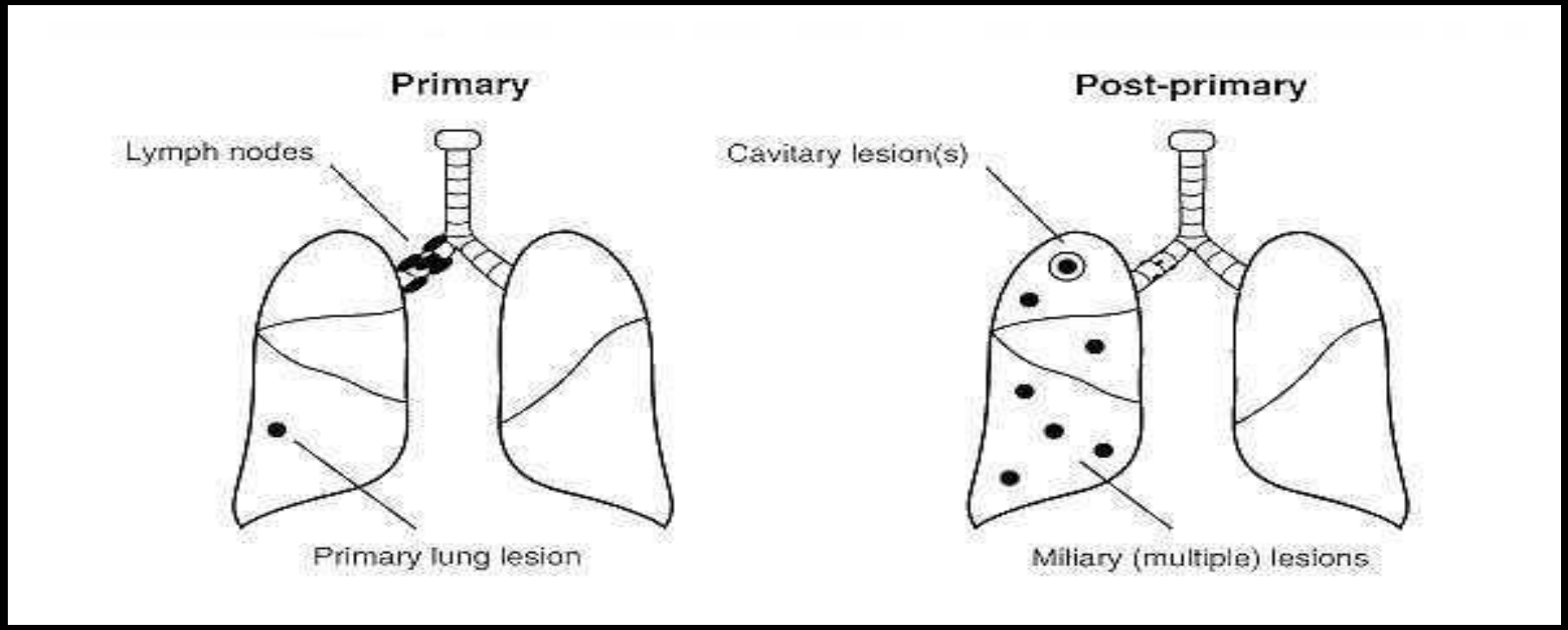
Free fluid in the pleural cavity

- The most frequent cause of exudative pleurisy of different etiology is increased permeability of the pleural surfaces for protein and decreased oncotic pressure gradient. The second reason is the lymphatic outflow reduction from the pleural cavity. A third reason could be a pressure reduction in the pleural cavity.
- Pleural effusion may develop in lung cancer, breast cancer, lymphoma, lymphogranulematosis, benign and malignant pleural mesothelioma, bacterial pneumonia, tuberculosis, fungal infections (aspergillosis, cryptococcosis, actinomycosis) and parasitic diseases (amebiasis, echinococcosis) at viral, infections, pulmonary embolism, pancreatitis, hepatic and subdiaphragmatic abscess, at collagen diseases (rheumatism, systemic lupus erythematosus, Wegener's granulomatosis), rupture of thoracic lymphatic duct.

Depending on pathogenesis the tuberculosis is divided primary and secondary.

Primary tuberculosis develops after the first contact of macroorganism with MBT.

Secondary forms of a tuberculosis arise at people which have been earlier infected, and after primary infestation should pass not less than year. It passes some years more often.



PRIMARY TUBERCULOSIS

***Primary** is considered tuberculosis that develops in firstly infected persons.*

The period from the moment of the intensity of tuberculin reaction during one year without signs of intoxication is called *the period of early tuberculous infection*

Para-specific reactions (tuberculosis “masks”)

- In primary tuberculosis there are situations where the disease occurs more on the type of therapeutic, hematological, rheumatologic disease. This is due to the fact that the body is infected TB patient responsible development of vasculitis and allergic reactions.

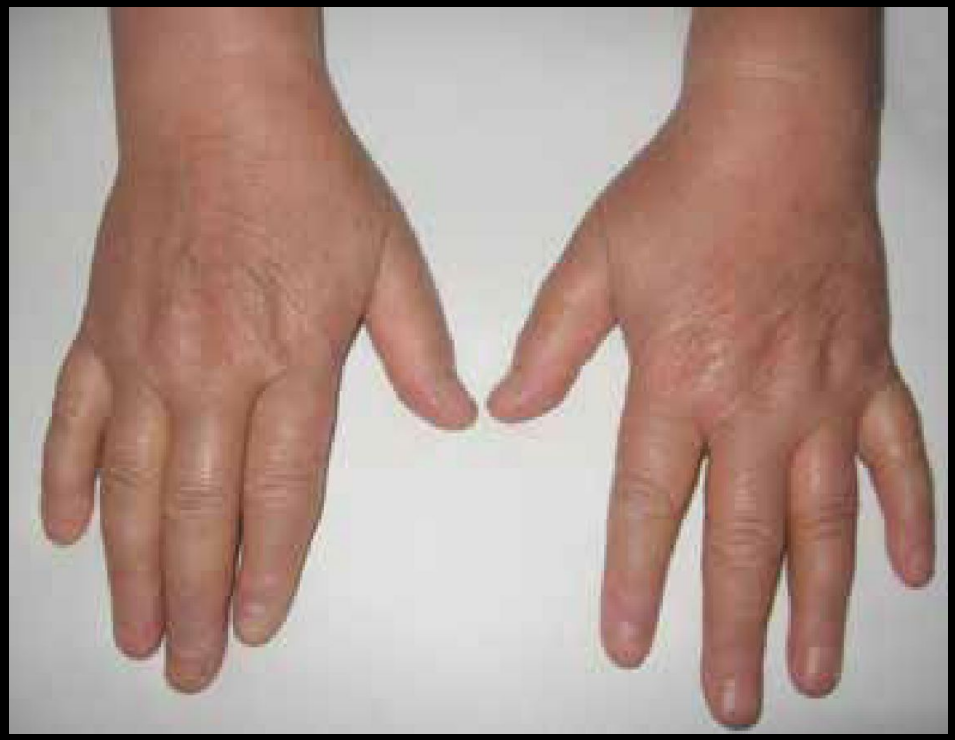
“Flu-like” TB mask

- The most frequently tuberculosis in active phase occurs in such frequent, long, unusual flu-like illness without clearly expressed inflammation of the upper respiratory tract and causes the patient's family outbreaks of influenza states – its a “flu-like” tuberculosis mask

“pneumonic” mask

The second frequency is “pneumonic” mask. This is repeated recurrent pneumonia, especially in the same lungs place with torpent course, having atypical clinic and course, difficult to treated, slowly resolved with the formation of small focal and fibrotic changes.

Poncet`s disease

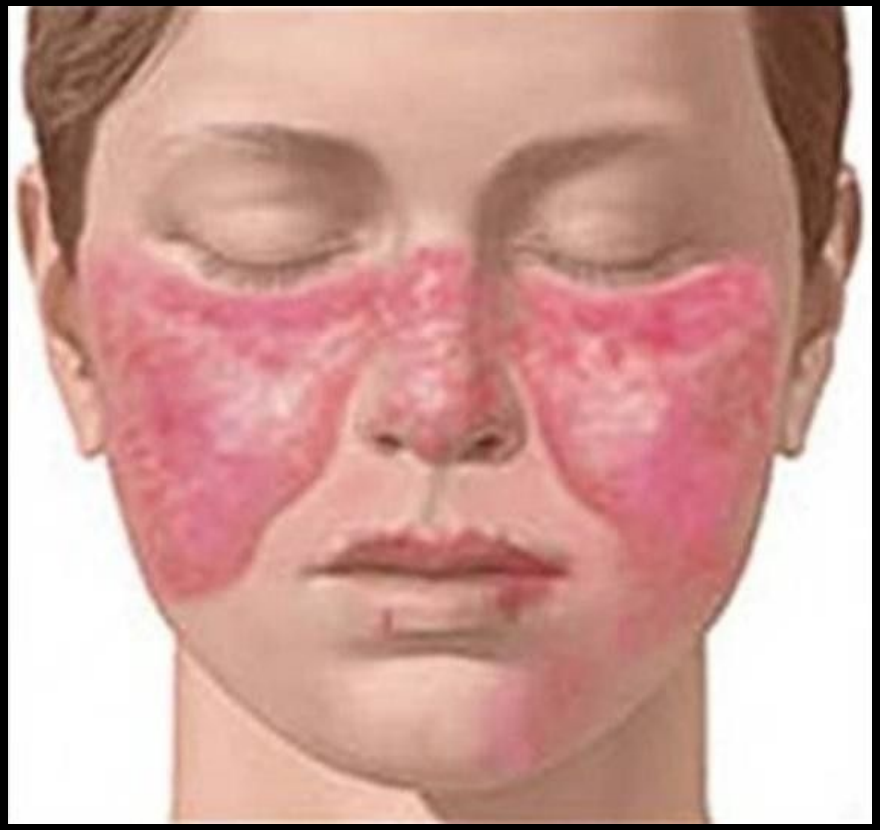


Tuberculosis can begin on type “rheumatic” mask, called “Poncet`s disease”. It manifested a long course articular syndrome with pain, swelling, breach of mobility in the joints with deformation, ankylosis. When X-rays there are typical signs of rheumatoid arthritis. There no efficiency after antirheumatic therapy in “Poncet`s disease” cases, no complications such as endocarditis. Only TB positive tuberculin tests, specific X-ray changes and the effect of specific therapy allows to confirm the diagnosis of tuberculosis.

“Neurological” TB mask

“Neurological” TB mask manifests as long, persistent neuralgia, which can not be usually treated, especially for intercostal and sciatic nerves, but without signs of compression (osteochondrosis) or inflammatory lesions nerve (a radiculitis).

“Lupus-like” TB mask

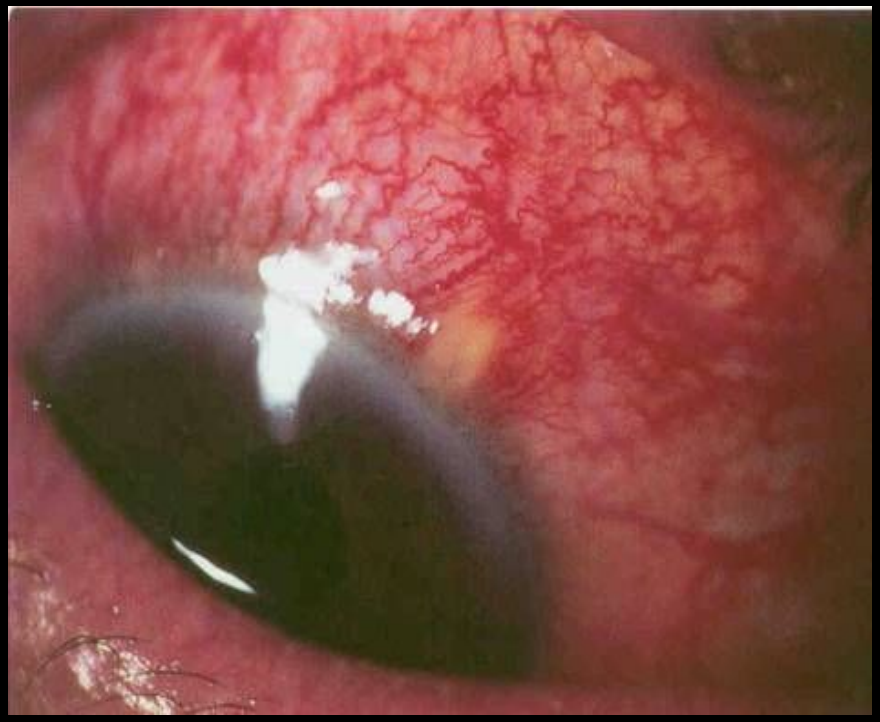


“Lupus-like” mask manifests typical erythema on the face in the form of "butterfly", trophic disorders, arthralgia, leukopenia, sharply increased ESR, sometimes specific blood cells and antibodies to DNA are finding.

“Hematological” mask

“Hematological” mask of tuberculosis occurs with bone marrow hypoplasia, leukopenia, anemia, thrombocytopenia, sometimes with leukemoid reactions. Often manifests lymphadenopathy, splenomegaly, B12-deficiency anemia and hypoplastic anemia.

Keratoconjunctivitis phlyctenular



- Keratoconjunctivitis phlyctenular. Most often its tubercular-allergic process in children with broncho-adenitis and tuberculosis of the lymph nodes, and other allergic reactions. On the bulbar conjunctiva and cornea near the limbus there are single or multiple inflammatory nodules of yellowish-pink color with a bunch of the blood vessels that are often completely resolve, but sometimes disintegrate with the formation of ulcers followed by replacement with connective tissue

CHARACTERISTIC SIGNS OF PRIMARY TUBERCULOSIS:

the intensity of tuberculin reactions

organism hypersensibilization to MBT

injury of lymphatic system (lymphatic nodes) with the susceptibility to caseous necrosis

susceptibility to lymphogenous and haematogenous dissemination, possibility of spontaneous recovery

availability of paraspecific reactions

The main forms of primary tubercular process:

1. Tubercular intoxication at children and teenagers.
2. Primary - tubercular complex.
3. Tuberculosis of intrathoracic lymph nodes.

PRIMARY TUBERCULOUS COMPLEX

1. PATHOGENESIS

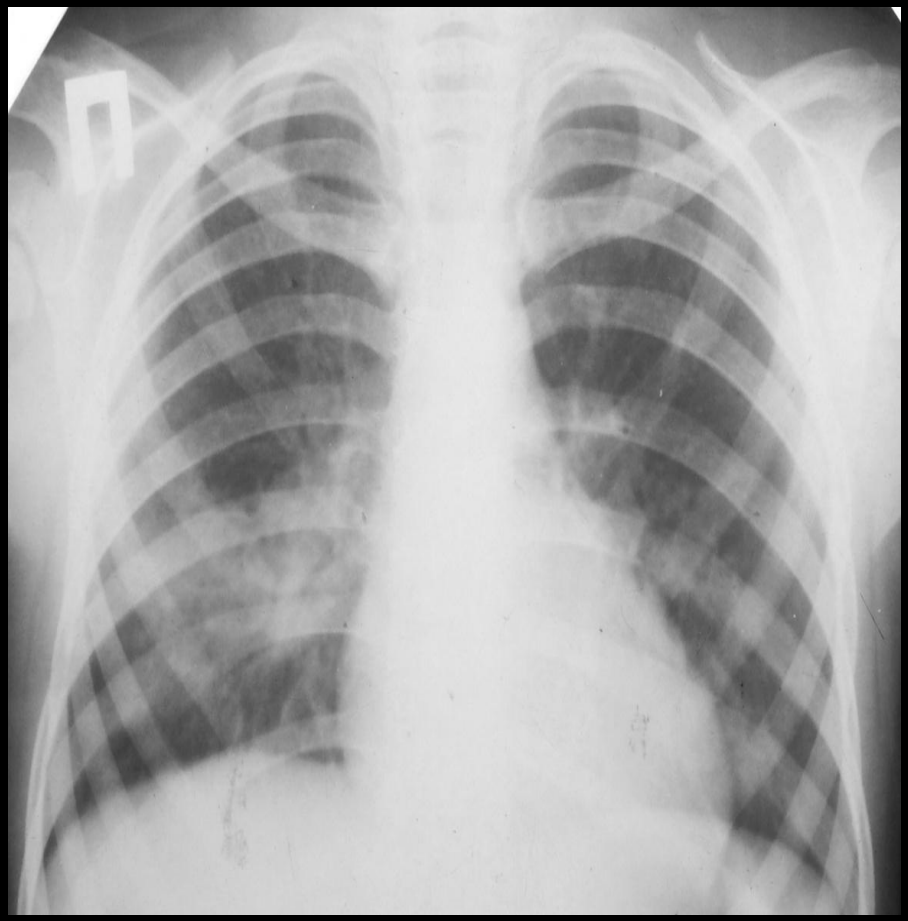
After the penetration of MBT into the lungs, primary lesion (primary affect), of the size from a millet grain to a section of a lung, is predominantly localized subpleurally in the II, III, VIII, IX segments. From the primary affect the infection spreads along lymphatic vessels to intrathoracic lymphatic nodes.

2. PATHOLOGICAL ANATOMY OF THE PRIMARY TUBERCULOSIS

In the primary lung focus, alveolitis develops, which is quickly replaced by the typical development of caseosis necrosis. In the centre of primary focus, caseosis forms but in the periphery – elements of non specific inflammation occur.

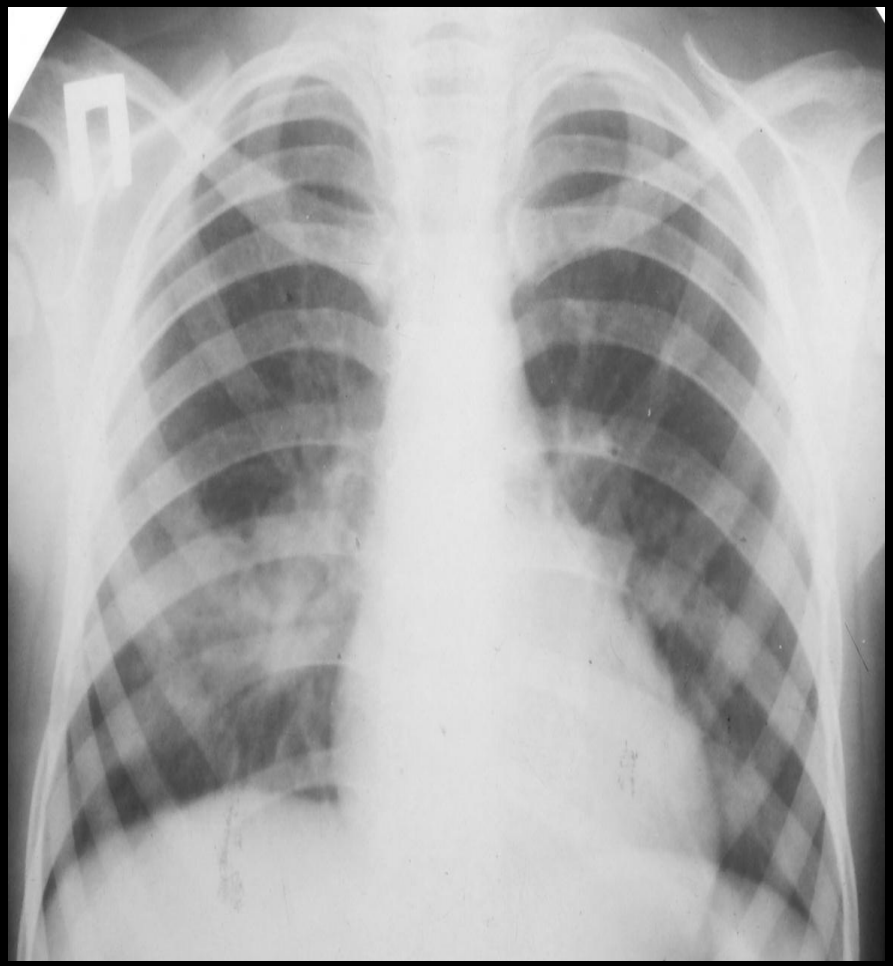
The primary lung affect localizes more often just under pleura, therefore frequently pleura is involved in the inflammation process. The lymphatic vessels expand, their walls becoming infiltrated and tubercles appear. In the regional lymphatic nodes, there are elements of inflammations converting into specific caseous changes with necrosis

PRIMARY TUBERCULOUS COMPLEX



- The dynamic study of primary pulmonary processes among children has allowed to allot 4 phases of the primary tuberculosis:

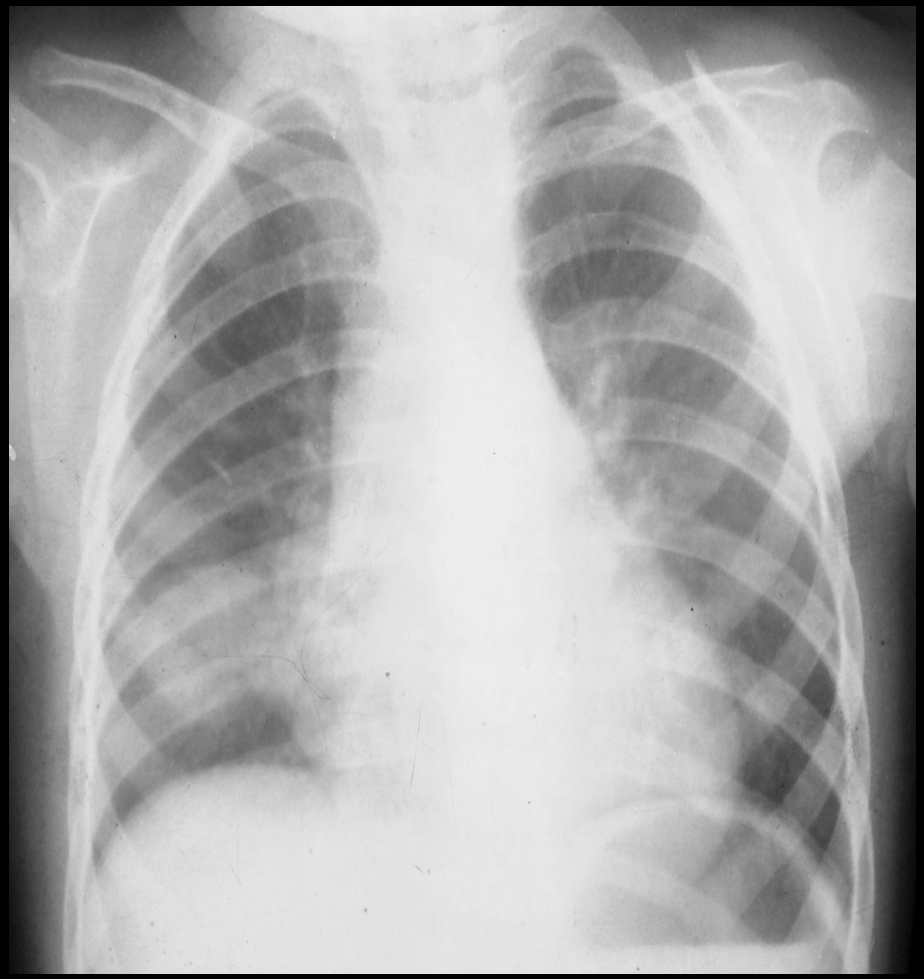
PRIMARY TUBERCULOUS COMPLEX



1) PNEUMONIC;

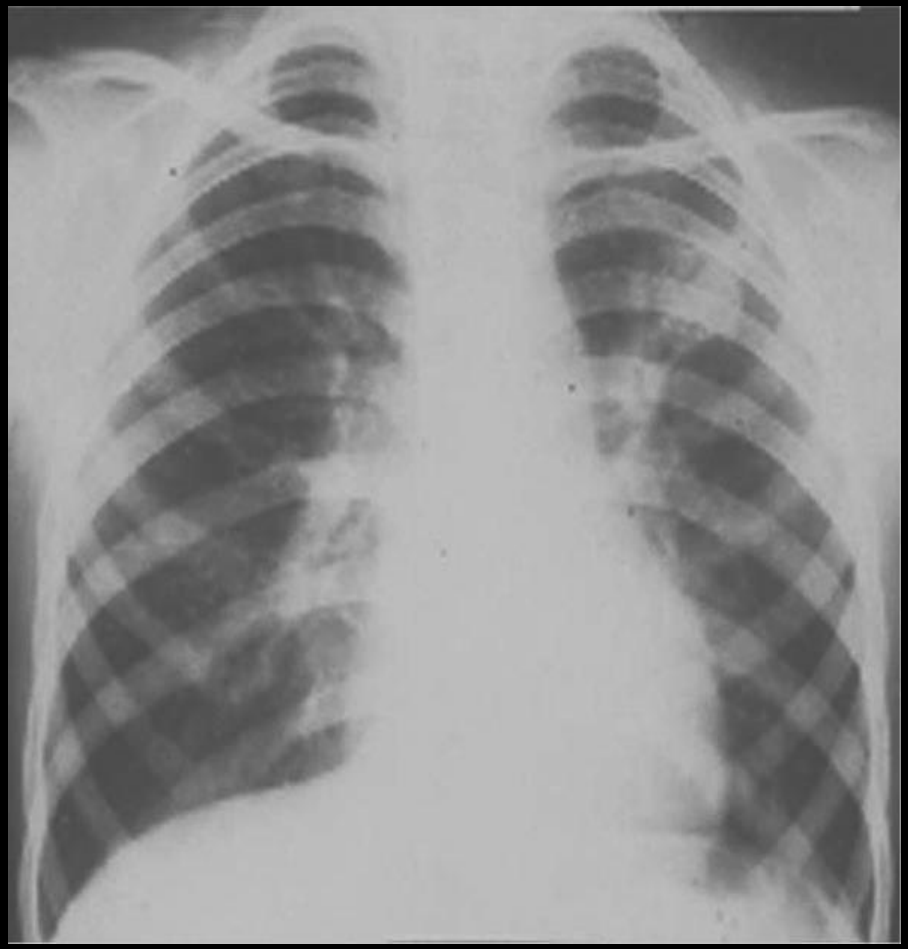
In the first phase (pneumatic) the focus of broncho-lobular pneumonia (3) is determined with a size of 1,5-2 till 5 cm.
The form of the lung focus (3) is round or irregular, with heterogenous character and dim contours.
Enlarged regional lymphatic nodes (1) are determined simultaneously (the picture of infiltrative bronchoadenitis) and there is an amplification of bronchial vessels picture – lymphangitis (2) between the focus and the lung root.

PRIMARY TUBERCULOUS COMPLEX



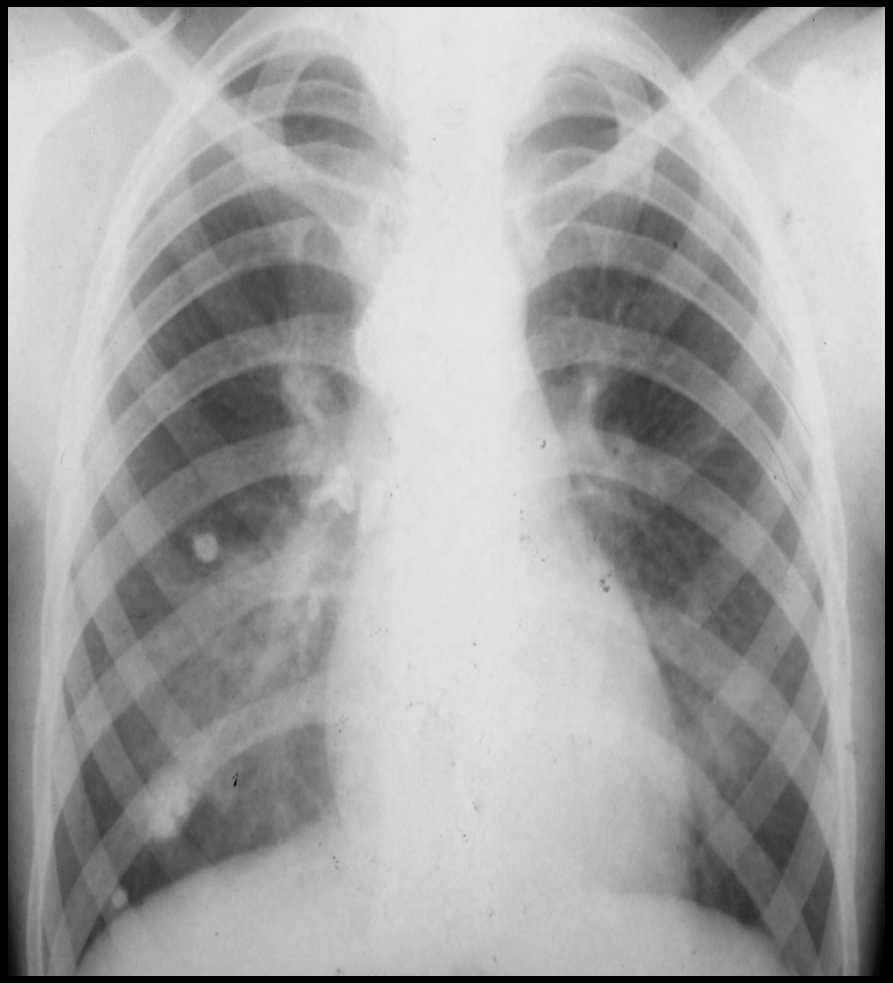
- 2) PHASE OF DISSOLVING;
  In the second phase of dissolving (bipolarity) the reduction of the perifocal zone of inflammation (3) is observed.
  The centrally located caseous focus becomes more prominent. The signs of inflammation in regional lymphatic nodes (1) and in the zone of bronchopulmonary vessels are decreaseding (2).

PRIMARY TUBERCULOUS COMPLEX



- 3) PHASE OF CONDENSATION;
  In the third phase, the phase of condensation: the primary focus is well outlined (3), its contours are cleared, on periphery of the focus there is the beginning of calcification as fine pieces; at peripheral regions of lung bronchial lymphatic nodes calcification is also present (1).

PRIMARY TUBERCULOUS COMPLEX



4) FORMATION OF GOHN'S FOCUS.

- In the fourth phase, in the place of broncho-lobular pneumonia (3) calcification become compact, the focus is round with regular precise contours, its size does not exceed 3-5 mm. This formation is called Gohn's focus.

TUBERCULOSIS OF INTRATHORACIC LYMPHATIC NODES

Bronchoadenitis is a disease of the lymph nodes of the lungs roots and the mediastinum. In this form of primary tuberculosis, intrathoracic lymph nodes are mainly involved in the process of inflammation

Pathogenesis. Infestation generally takes place by the droplet-dust way, through the mucous membrane of tonsils and bronchi MBT penetrate into lymphatic vessels, nodes, where a specific process develops. Depending on the state of micro- and macroorganism, infiltrative-inflammatory or necrotic changes in lymphatic nodes prevail.

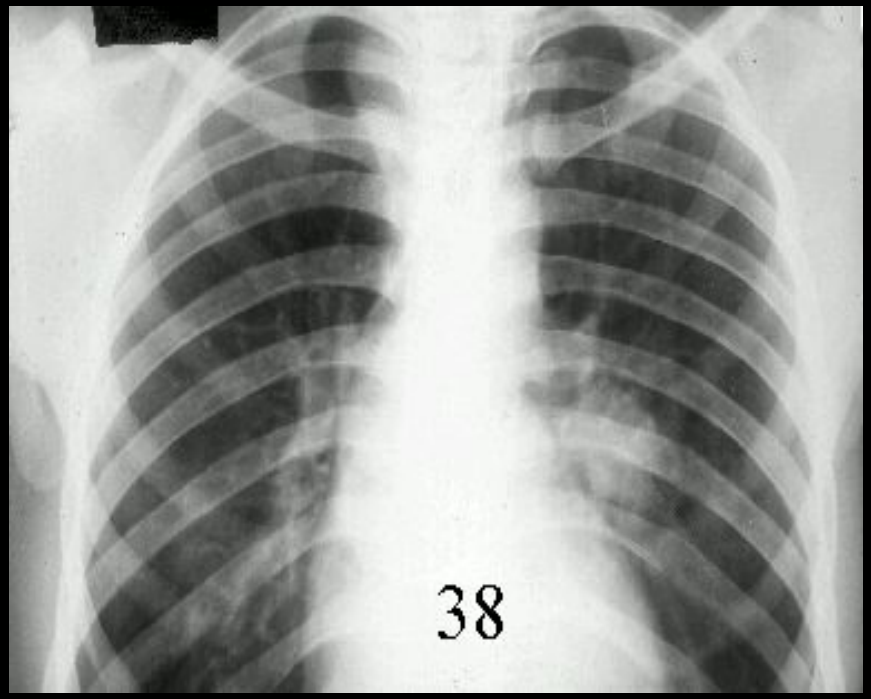
Pathomorphism. One or some groups of lymphatic nodes may be in jured at tuberculosis. Paratracheal, tracheobronchial, bronchopulmonary, bifurcating and other lymphatic nodes are hurt. The process may be uni- or bilateral, predominantly asymmetric.

Clinical pattern of tuberculous bronchoadenitis

- acute intoxication
- specific clinical symptoms: subfebrile temperature, deterioration of general condition, loss of appetite, weight loss, adynamia or excitation of nervous system
- in progression, and especially in small children, appears bitonal cough
- among adults, attacks of dry, «hoarse» tickling cough take place. It is caused by irritation of the mucous of bronchi or formation of the broncho-pulmonary fistula
- Blood analyses are without any features
- Detection of MBT. In gastric lavage, it is possible to find out MBT, especially often it is discovered in the sputum and in bronchial lavage during the rupture of the caseous mass into the bronchus.

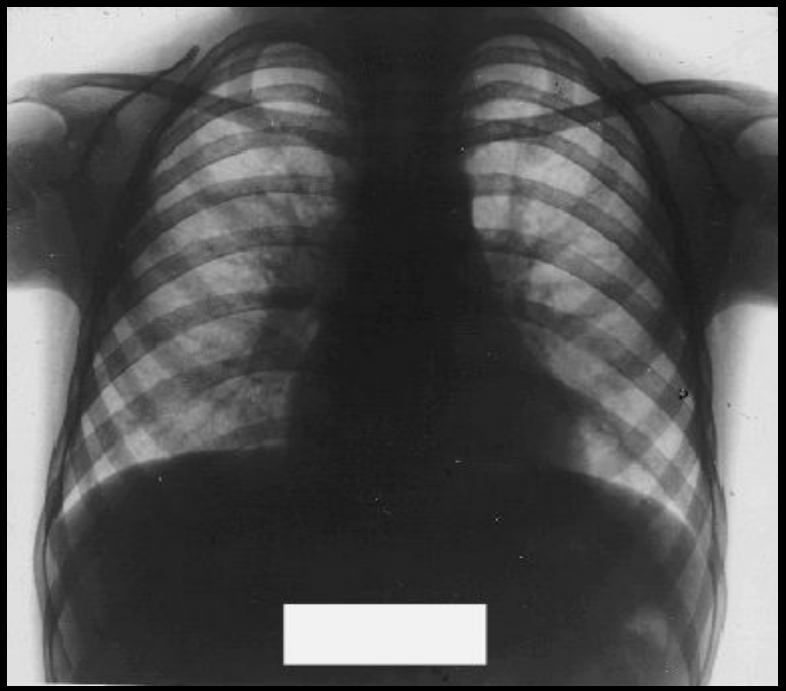
Clinico-roentgenologically variants of intrathoracic lymphatic nodes TB

tumour like (tumoursimilar) form



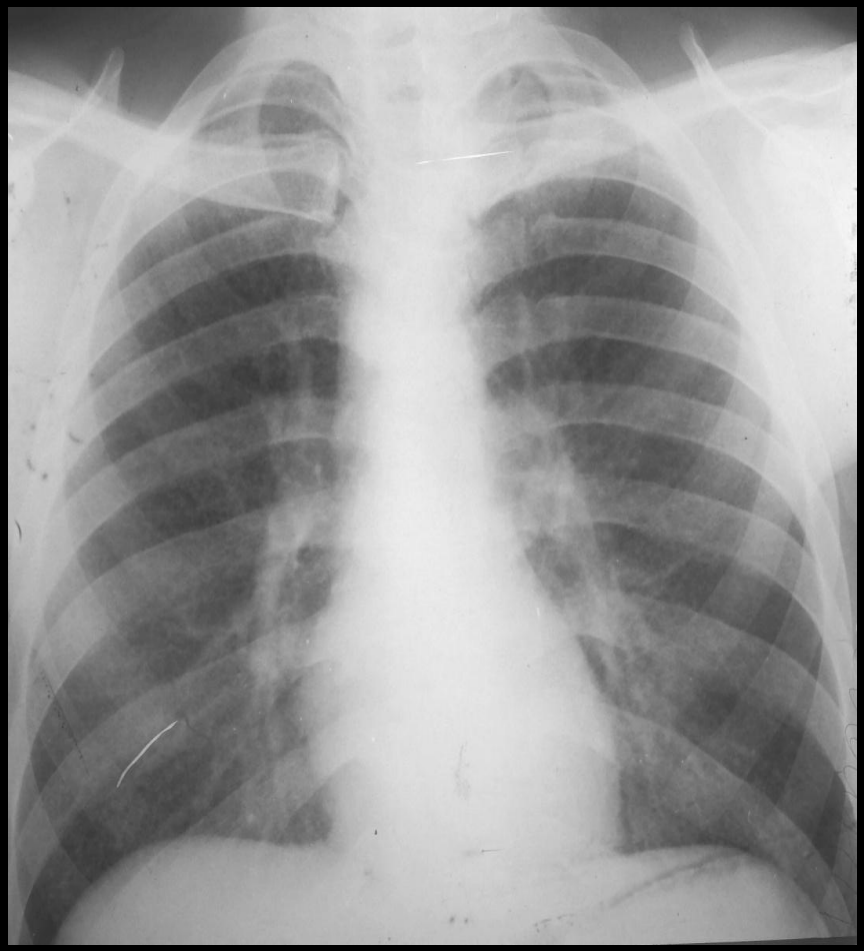
Left side tumorous lymphadenitis. Massive enlargement of broncho-pulmonic lymphatic nodes

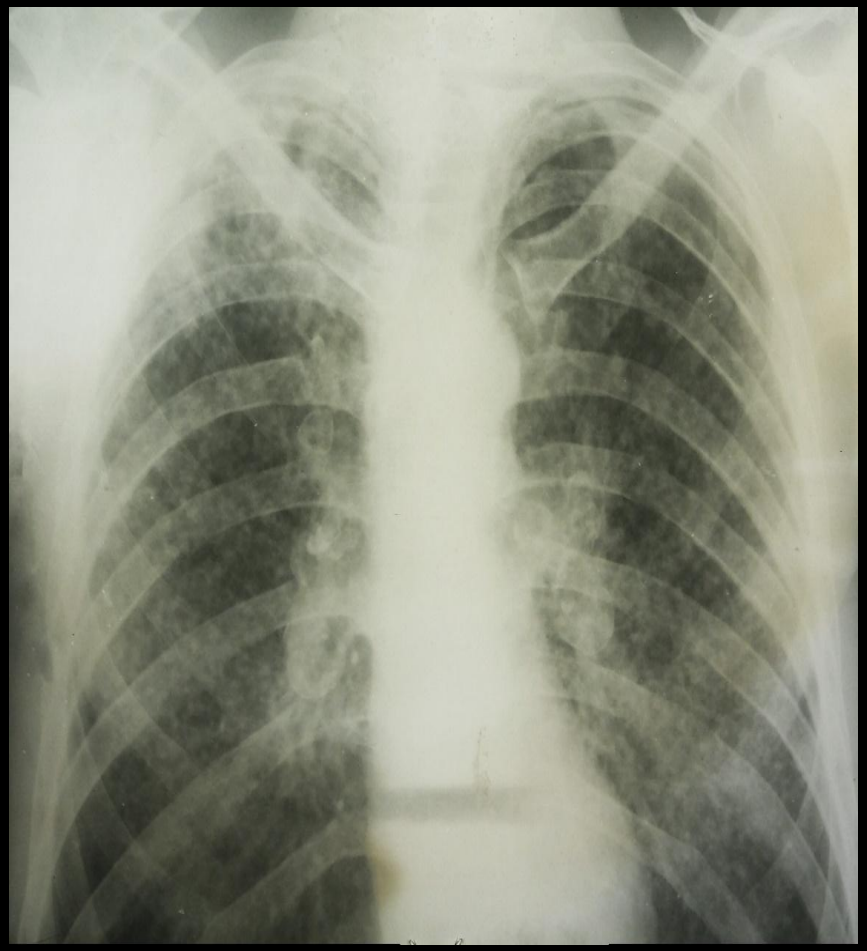
infiltrative form



On chest x-ray the shadow of the right lung root is extended the outside contour is dim, the structure is blurred and intensity is increased.

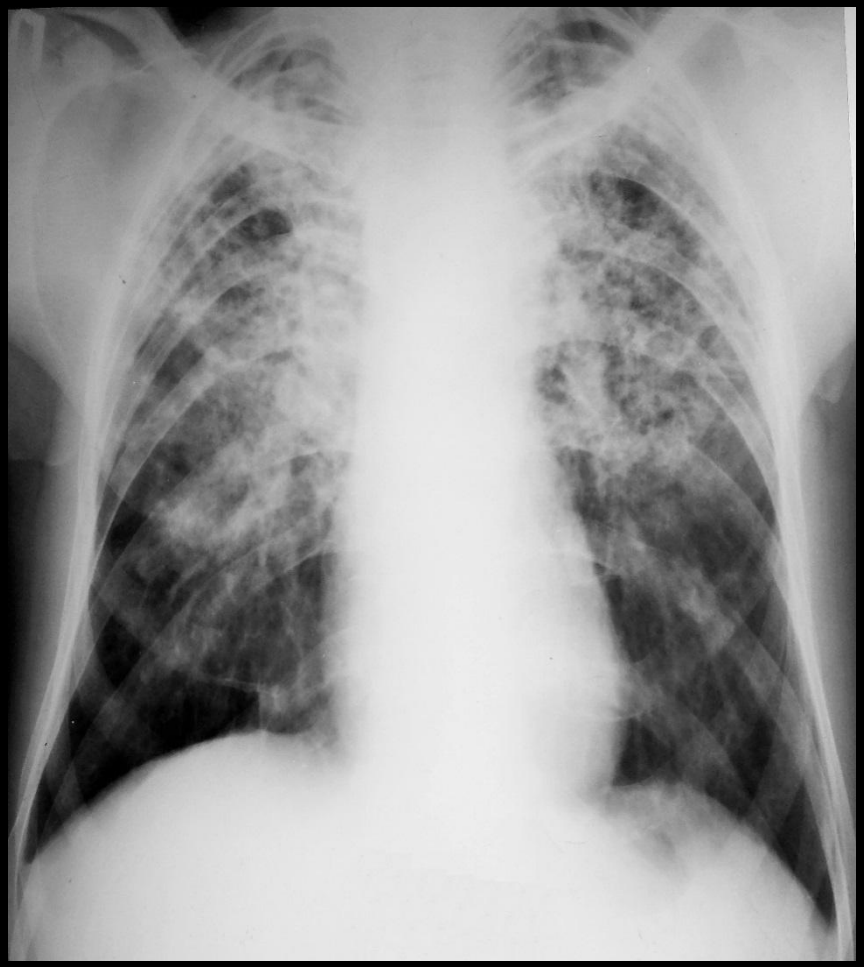
Miliary tuberculosis





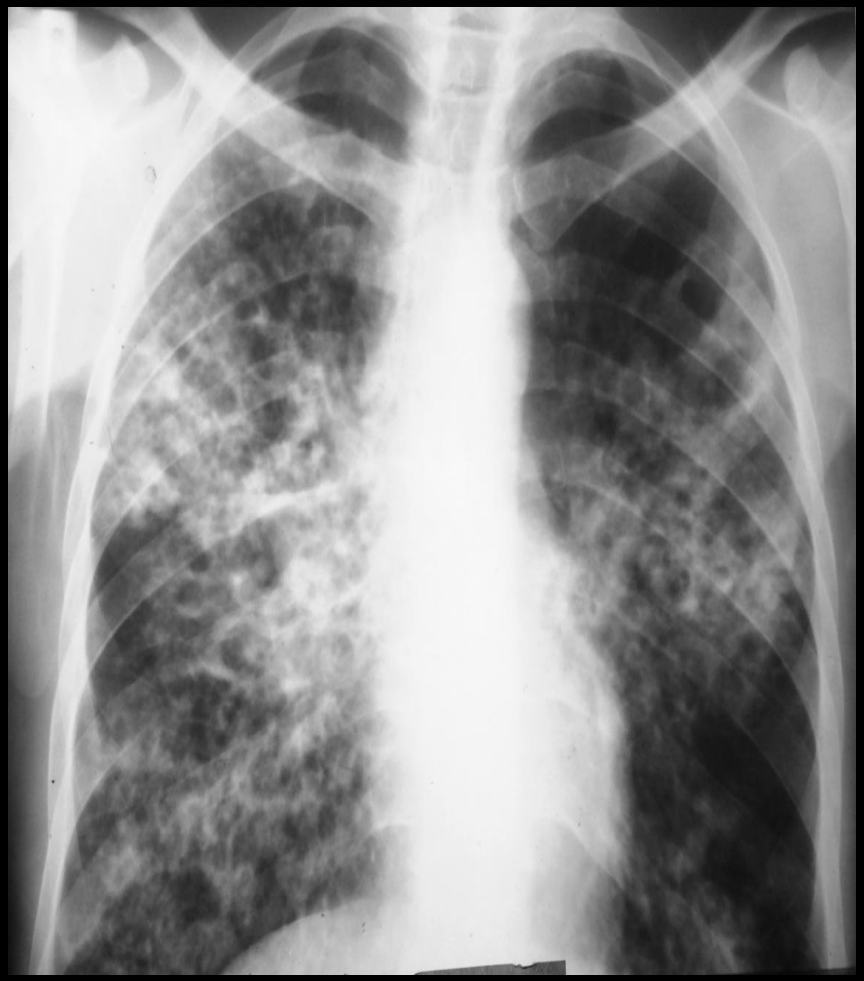
Generalized TB clinical form with the hematogenic type of dissemination and acute course

Sub-acute disseminated tuberculosis



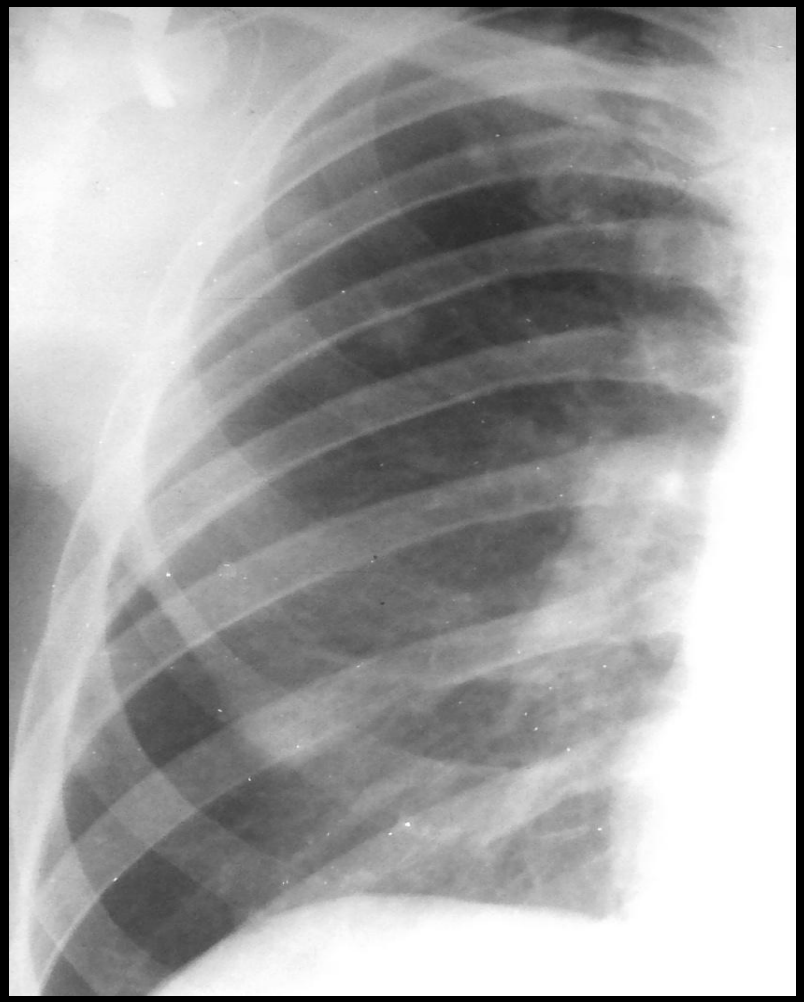
- Bilateral lung injury with predominant affecting upper lobes and trend for destructive progressive course

Chronic disseminated tuberculosis



- **Disseminated lung injury with the wavy course and progressive alveoli substitution by connective tissue**

Focal (nidus) TB

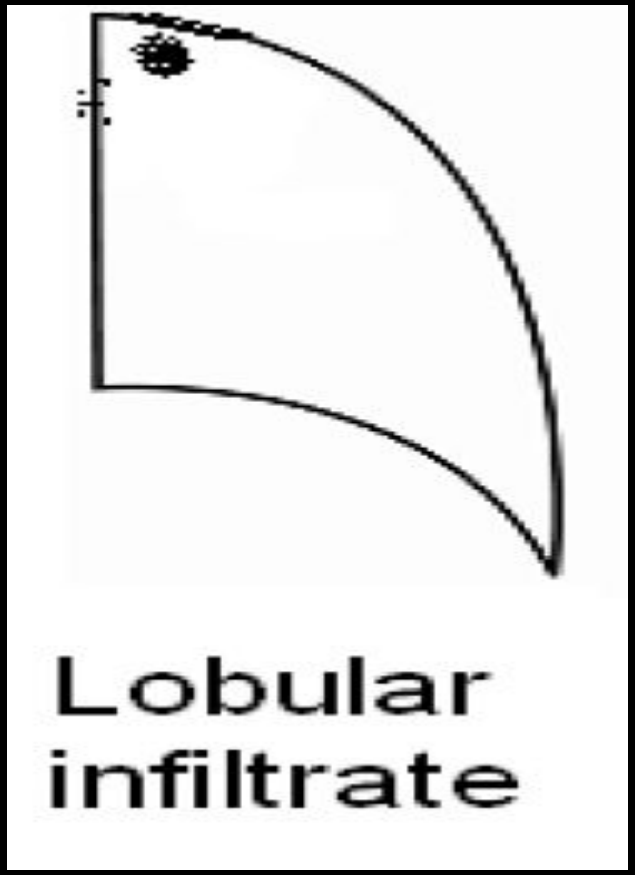


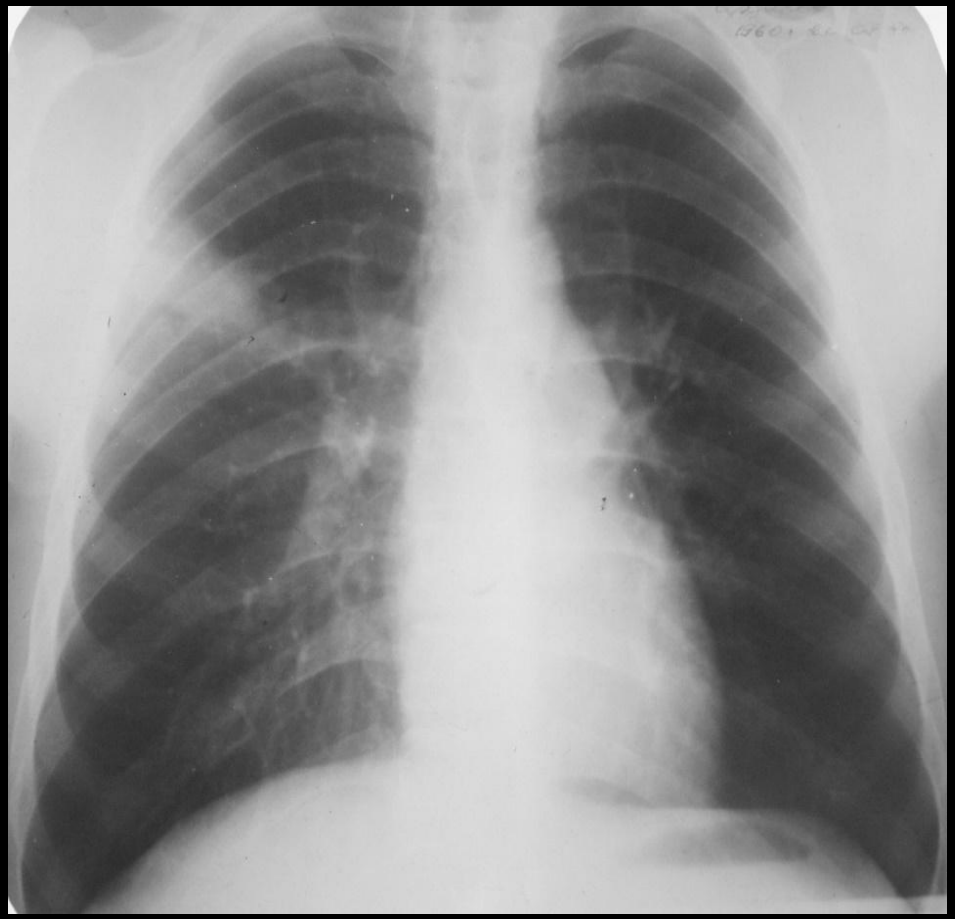
- The mildest limited lung TB clinical form which is characterized by the appearance of single or several nidi (opacity with the diameter up to 1 cm in diameter) in one or more segments of one or both lungs with predominantly productive type of the inflammation and slow torpid progression.

Infiltrative TB

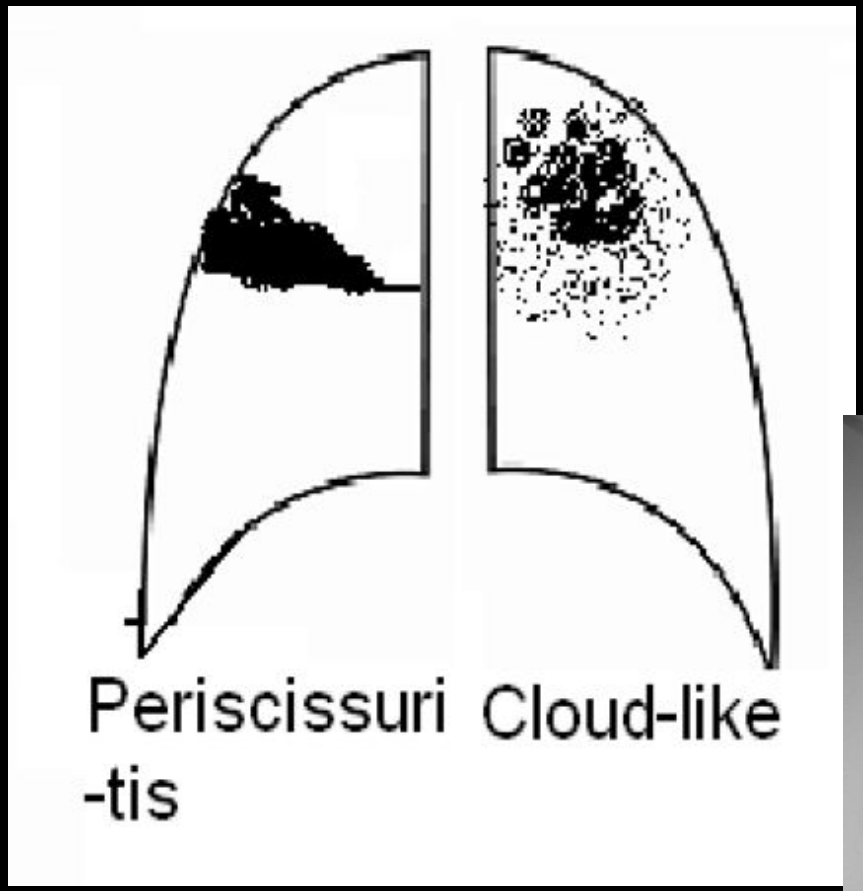
The expansive lung TB clinical form which is characterized by the appearance of different size and shape lesions in one or more segments of one or both lungs with predominantly exudative type of the inflammation and strong trend to the rapid progression and decay.

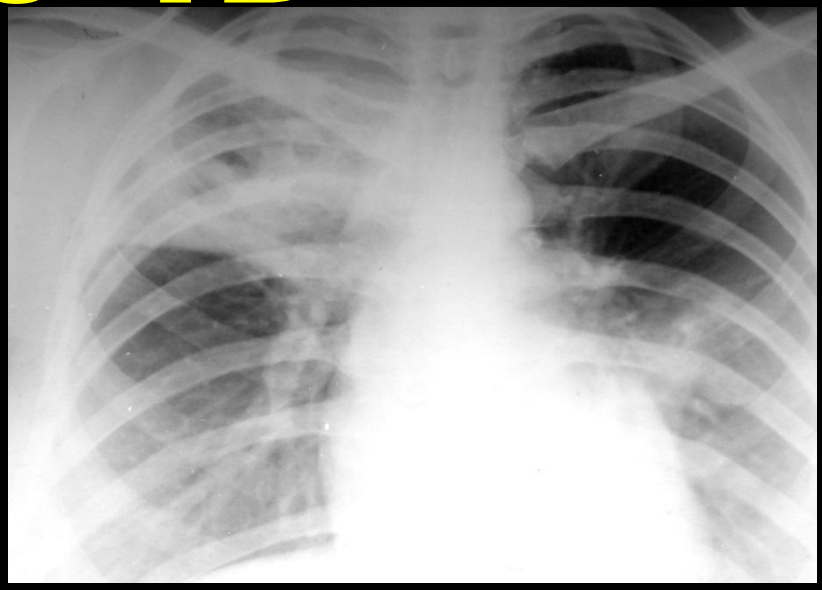
Infiltrative TB

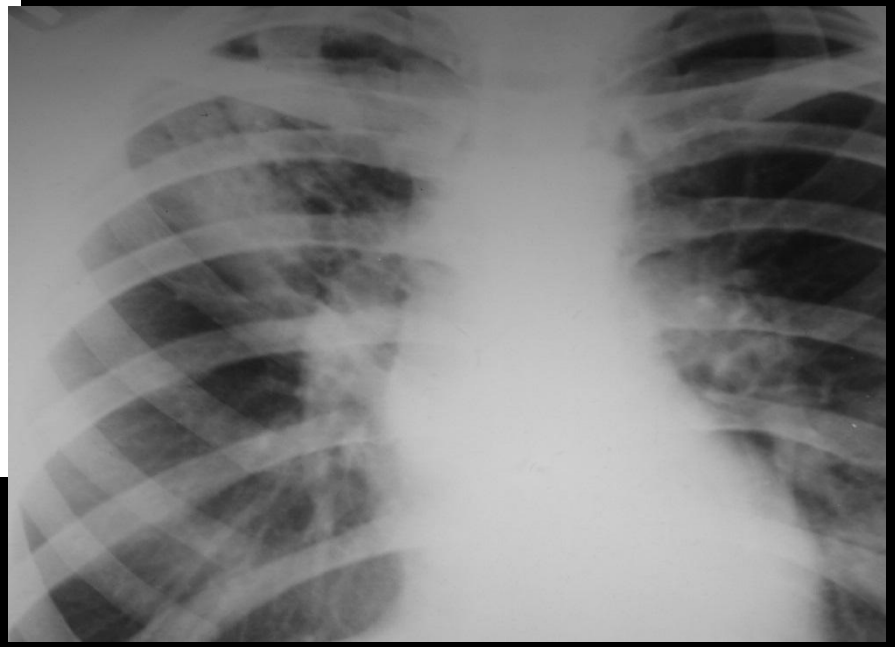




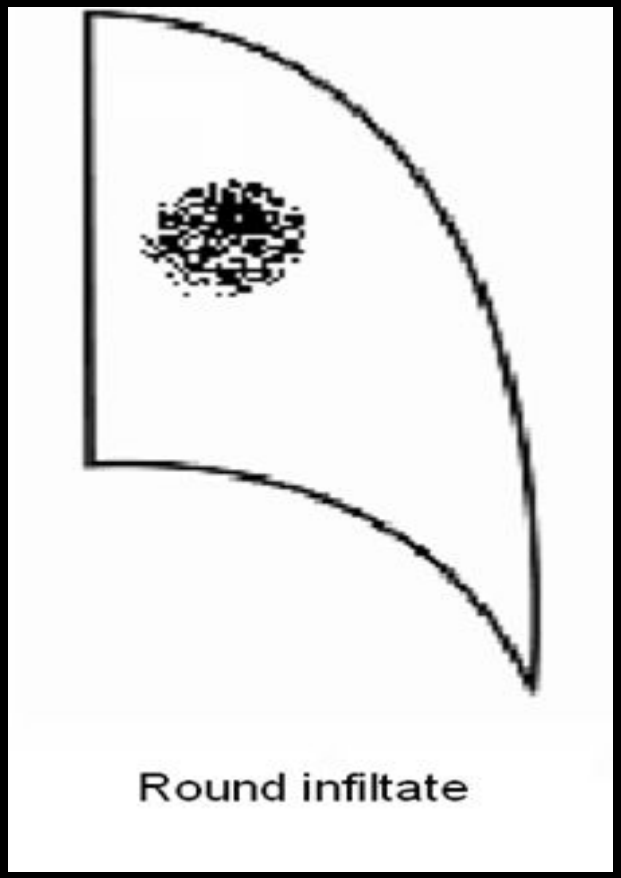
Infiltrative TB

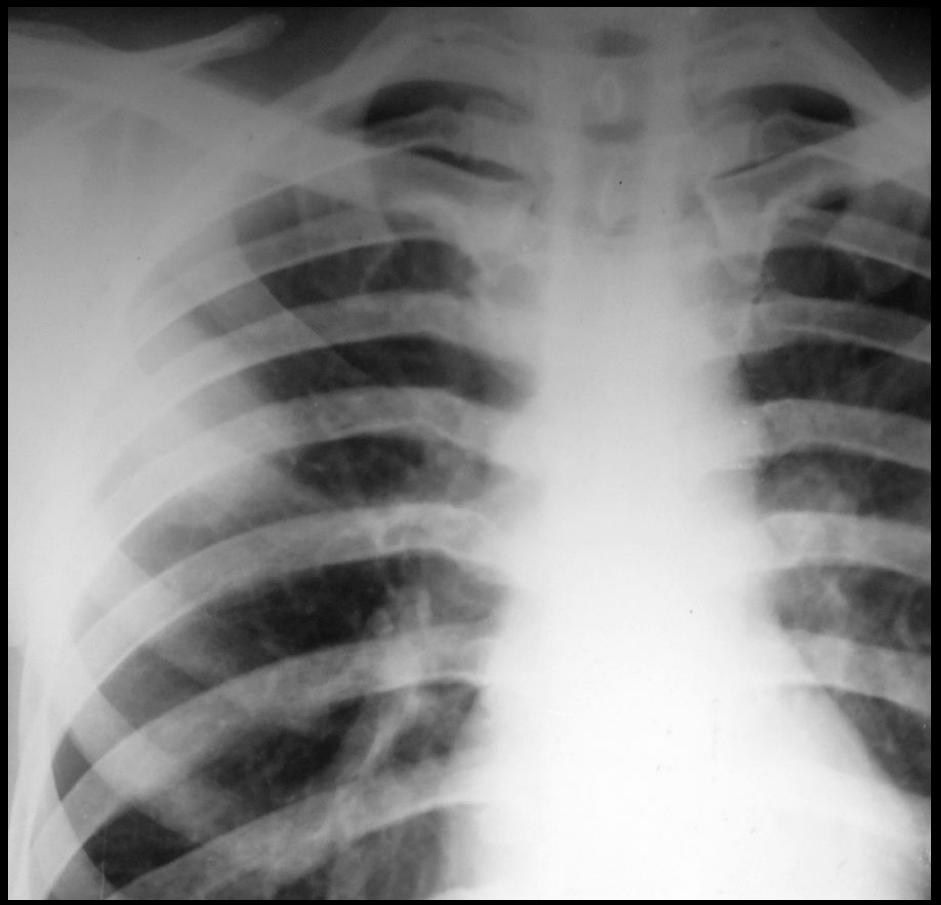




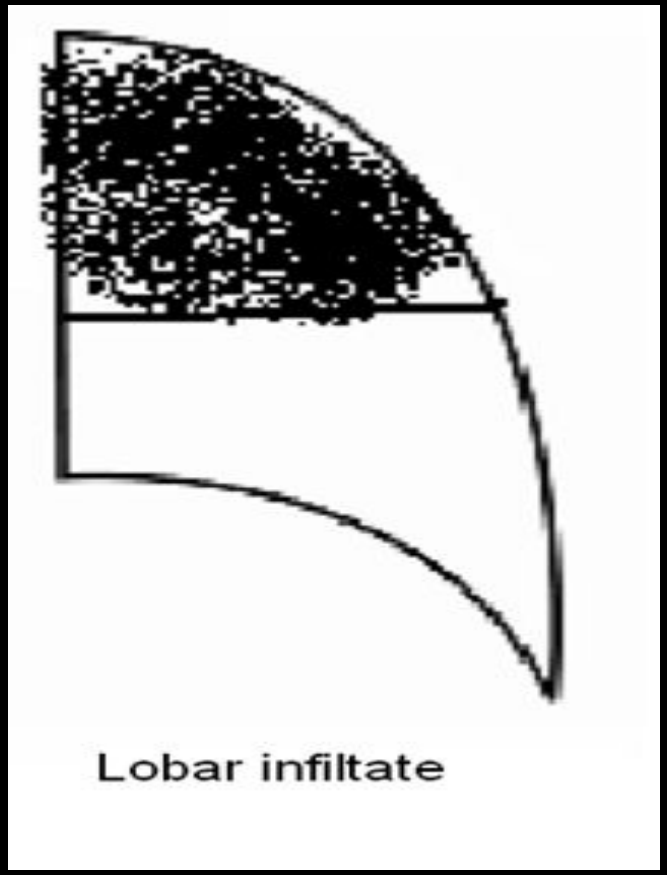


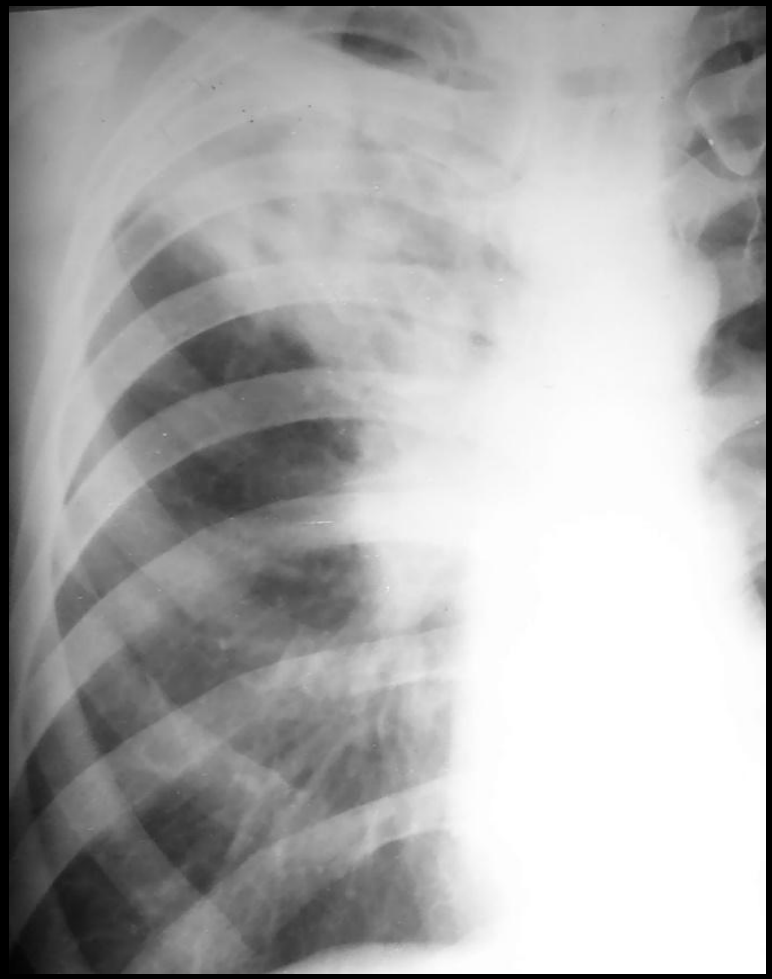
Infiltrative TB



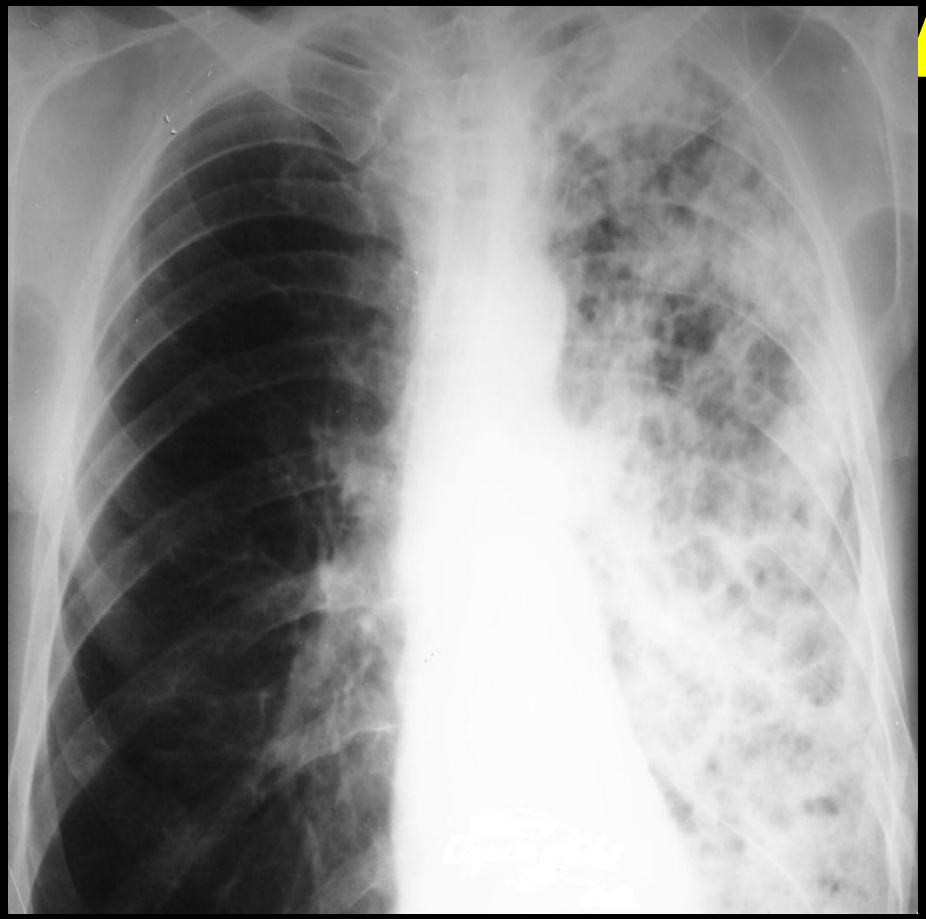


Infiltrative TB





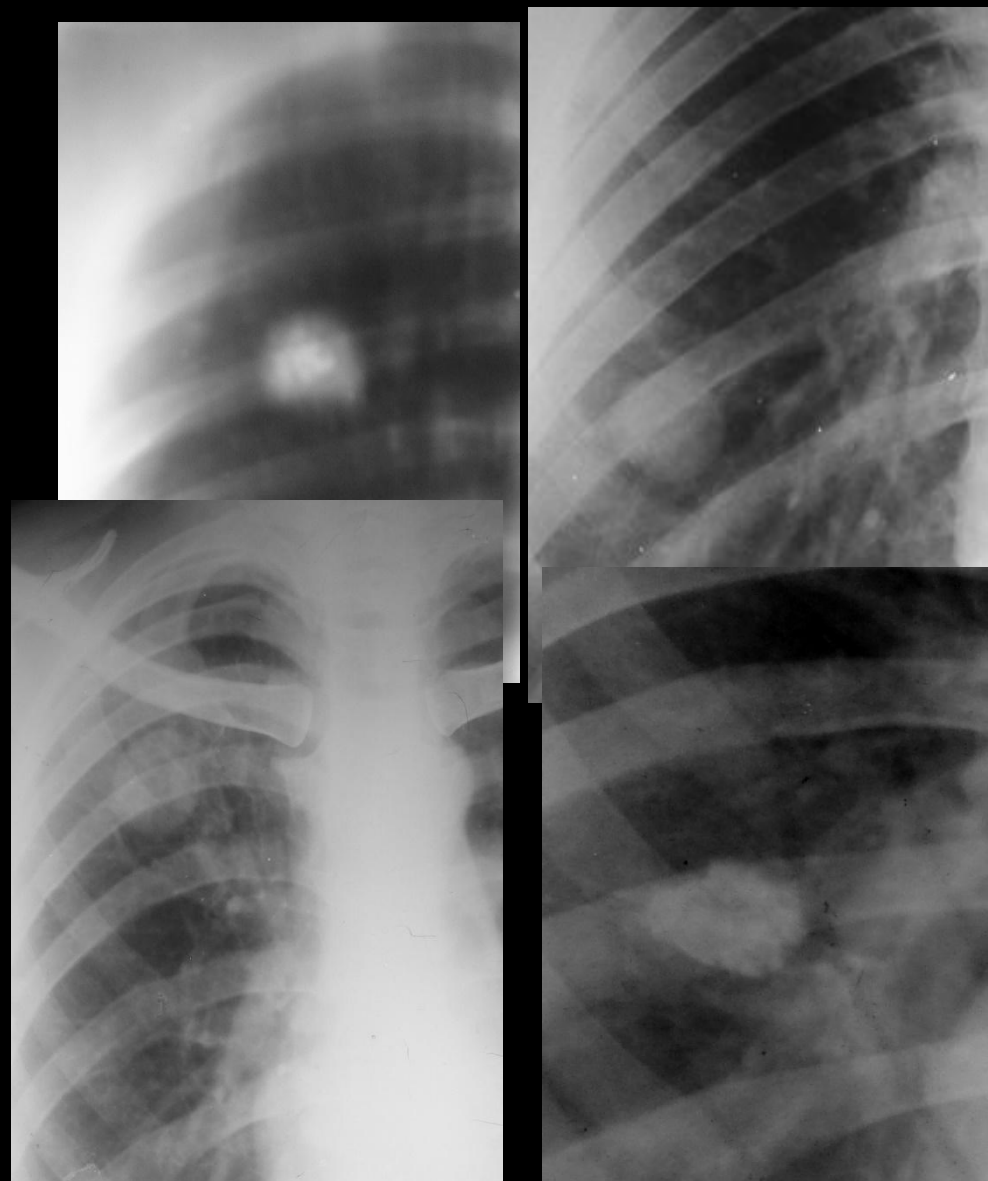
Caseous pneumonia



- An acute TB form with the decay predominance and severe progressive course

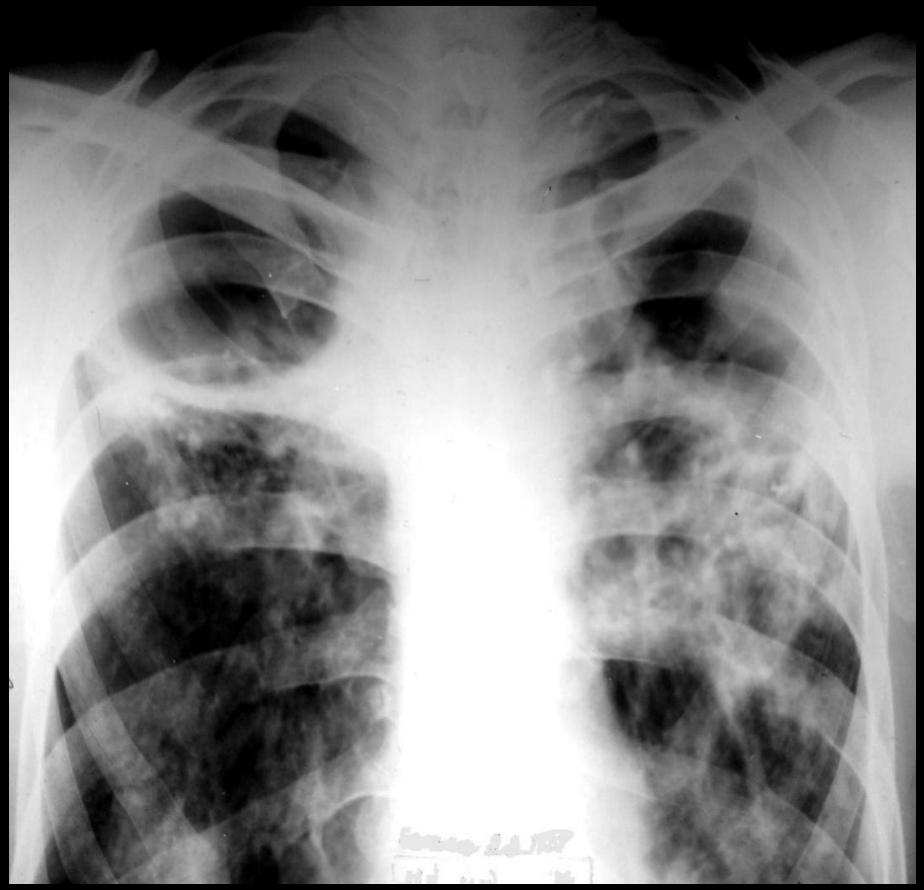
Tuberculoma

The inflammative focus limited by connective tissue capsule



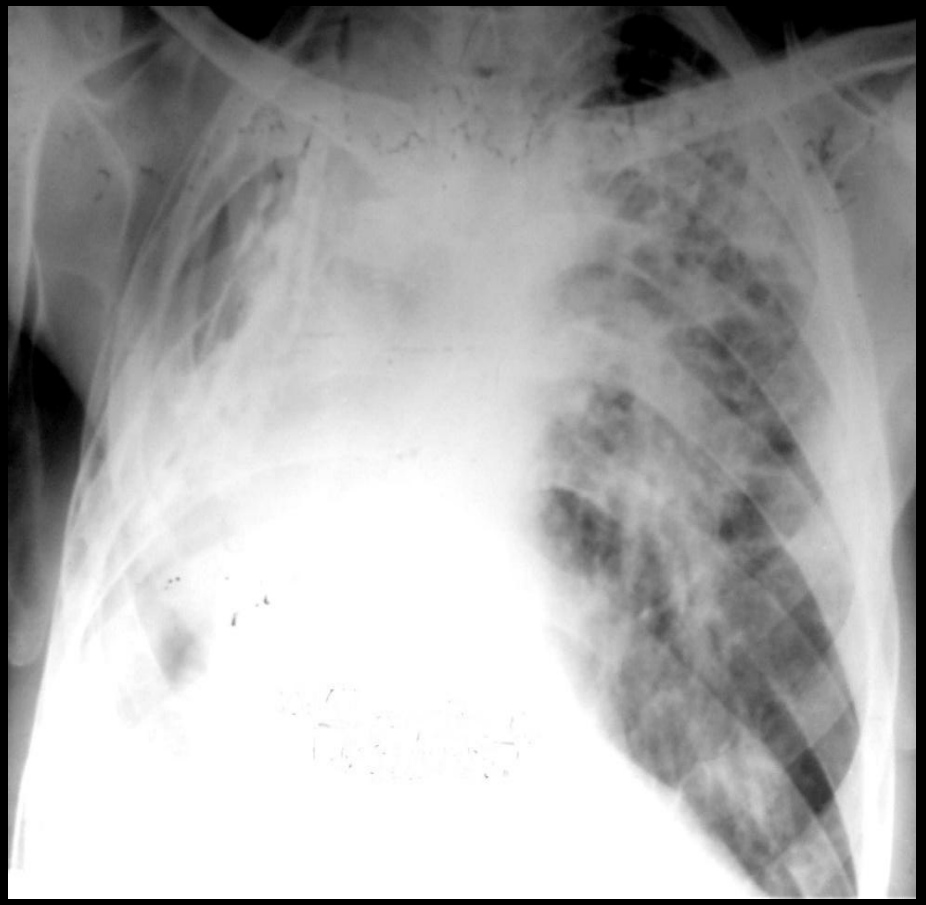
Fibrous-cavernous tuberculosis

The chronic destructive clinical TB form characterized by the presence of the old fibrous cavity (cavern) in the lung tissue surrounded by extended fibrosis and satellite nidi



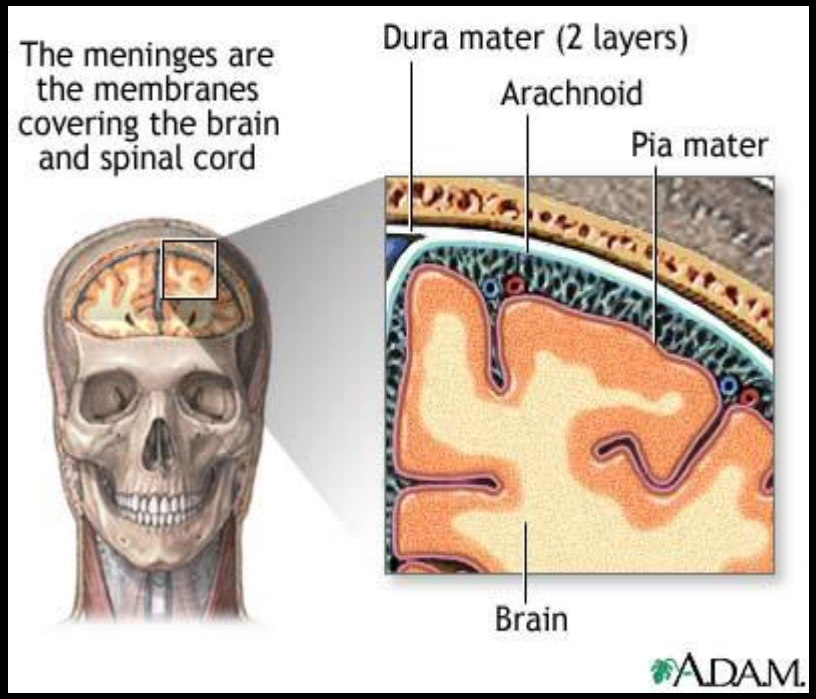
Cirrhotic lung tuberculosis

The chronic destructive clinical TB form characterized by the predominant cirrhosis in the lung tissue with the embedded cavitations inside



Tuberculous meningitis is the inflammation of the membranes of cerebrum and (or) spinal cord, caused by MBT.

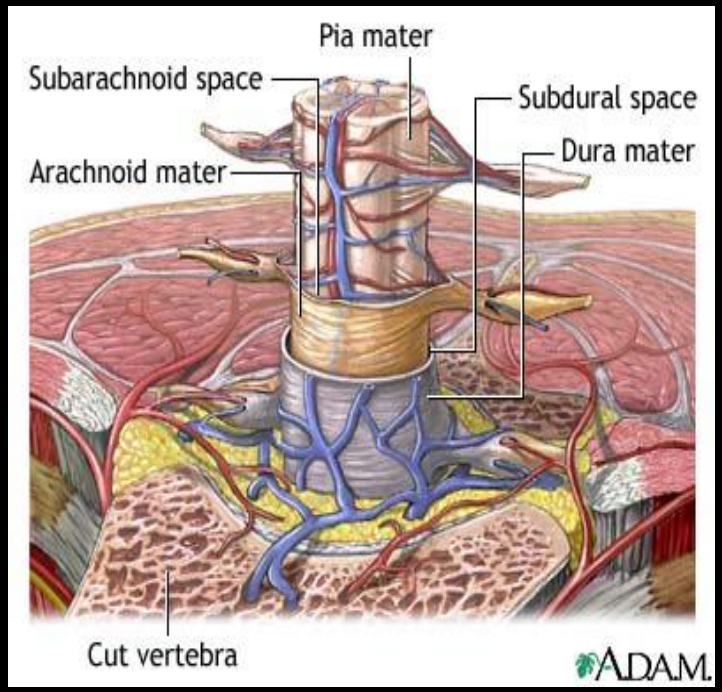
Specific lesion of cerebral membranes and substances is tuberculous meningoencephalitis.



Causes, incidence, and risk factors

- **Tuberculous meningitis is caused by the spread of *Mycobacterium tuberculosis* to the brain, from another site in the body. The symptoms usually begin gradually. Risk factors include a history of pulmonary tuberculosis, excessive alcohol use, AIDS, or other disorders that compromise the immune system.**

Pathogenesis. **Tuberculous meningitis may be primary (in 20 %) and secondary (in 80 %), as children and teenagers primary tuberculosis complication and predominantly disseminated lung tuberculosis in adults.**
MBT penetrate into submembranous space of cerebrum and (or) spinal cord by haematogenous, lymphogenous and rarely — by perineural way.



Pathological anatomy. **The specific process is predominantly localized in the soft membrane of cerebral base, in which connection cranial nerves, located here, are injured. The illness may progress as meningoencephalitis (64-70 %) - inflammation of cerebral membranes and substance, as basal meningitis (20-30 %) - inflammation of cerebral membranes and as spinal meningitis (4-6%) - inflammation of spinal cord membranes.**
Exudate, tubercles, tender fibrin threads appear on soft cerebral membrane at tuberculous meningitis. Pathologic changes are also observed on the vessel membrane of cerebral ventricles.

Clinic of Tuberculous meningitis

I. A prodromic period

- **the duration is from 1 to 4 weeks:** general weakness, irritability, sleeplessness, lability, unstable headache, often subfebrile body temperature

II. obvious clinical manifestations

- the body temperature rises to febrile, the headache of sharp intensity, joined by vomiting
- **meningeal syptoms develop gradually:** rigidity of cervical muscles, Kernig's and Brudzinsky's symptoms
- eye motional and drain cranial nerves are injured
- pareses according to the central type of the VII, IX, X, XII pairs of cranial nerves
- vegetovascular disturbances in the form of vasomotor reactions develop, stable red dermographism, Trusso spots, relative bradycardia, disturbance of sleep and appetite

III. The period of pareses and paralyses

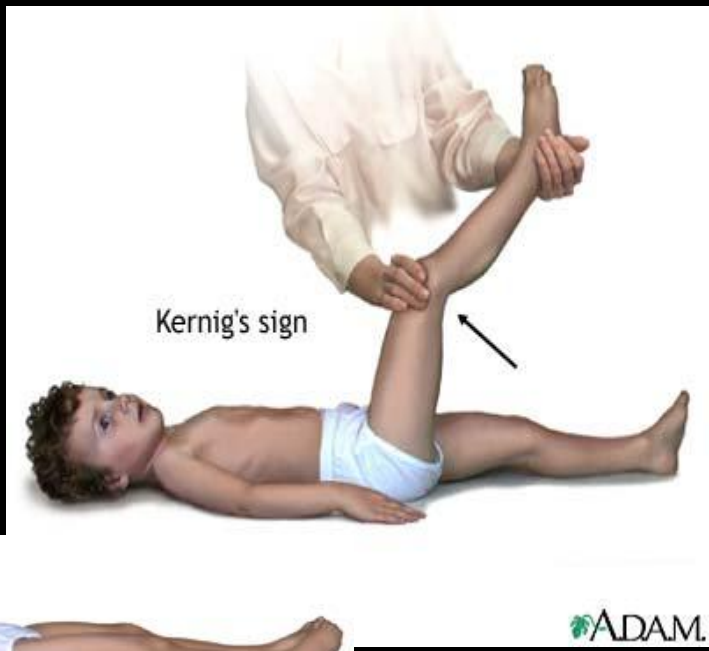
- expressed adynamy, apathy to all surrounding, later on soporose state develops, coma

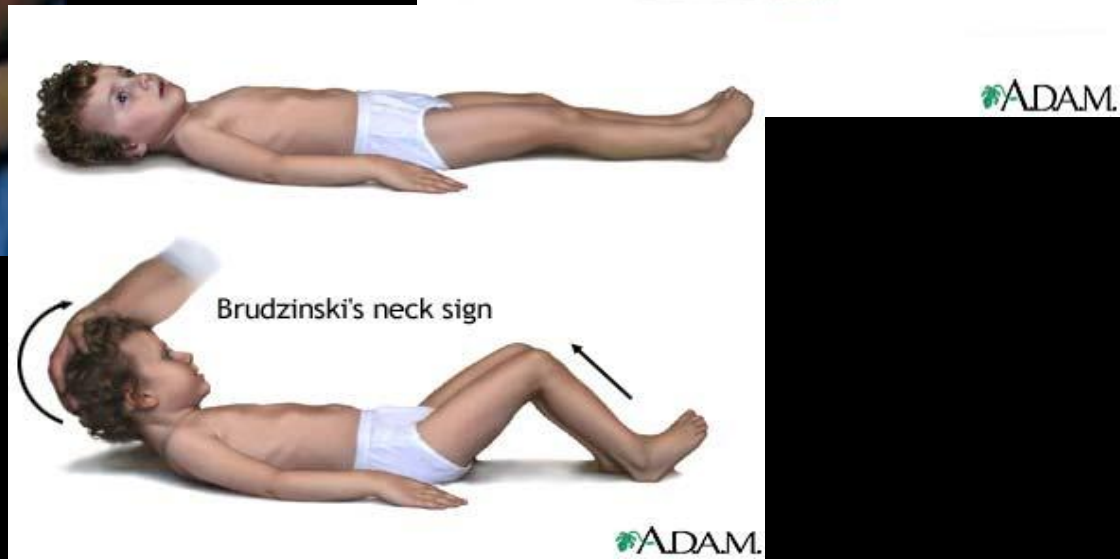
5 components (syndromes) are discriminated in the clinical picture of tuberculous meningitis:

- ***Intoxicating syndrome***
- ***Meningeal syndrome: headache, vomoting, hyperestasis***
- ***Symptoms of cranial nerves lesions* (III, VI, VII, XII) *and the spinal cord roots***
- ***Changes of spinal fluid***
- ***Symptoms of irritation and prolapse of functions owing to cerebral tissue lesion***

Meningeal symptoms

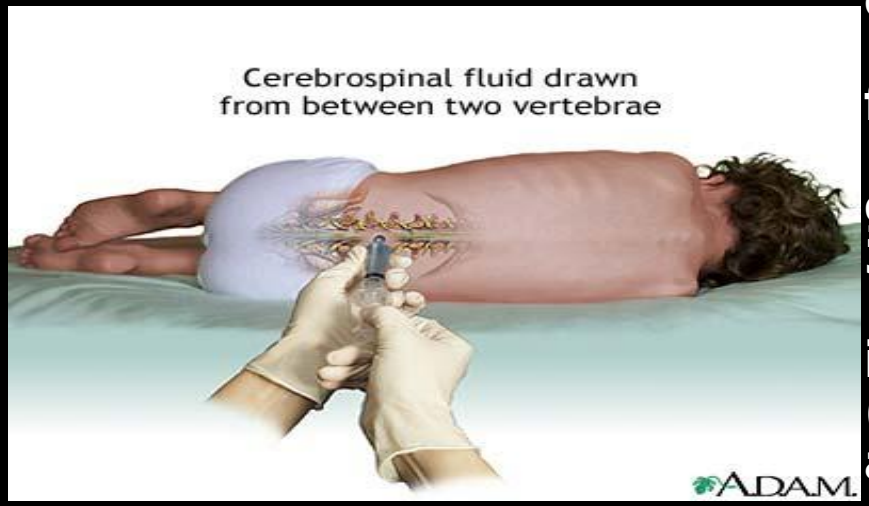
- rigidity cervical





Characteristics of spinal fluid

Liquor is usually transparent or opalescent



flows under increased pressure

greater number of cells (100 – 300 in 1 ml, at norm - up to 10)

increased protein content (0,6 –1,0 g/L and more, at norm – 0,2-0,4 g/L)

lowered sugar concentration 1,5 mmol/L and less, norm- 2,22-3,88 mmol/L) and chlorides concentration (110 mol/L and less, norm -120 -130 mmol/L)

Pandi and None-Apelta's positive reactions

in liquor a weblike film is formed in 24 hours, in which in 10-20 % of cases MBT are revealed