SAINT PETERSBURG STATE PEDIATRIC MEDICAL UNIVERSITY
DEPARTMENT OF PROPEDEUTICS OF INTERNAL DISEASES

LUNG TISSUE LOBAR AND FOCAL CONSOLIDATION SYNDROME.

LUNG CAVITY SYNDROME

(tutorial for medical students)

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The tutorial presents the basic points of etiology, pathogenesis, clinical presentations, results of physical examination and the data of instrumental and laboratory examinations in case of lung tissue consolidation – focal and lobar pneumonia, as well as in case of lung cavity (illustrated by lung abscess).

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LUNG TISSUE CONSOLIDATION SYNDROME

A lung tissue consolidation is considered an area in the lung parenchyma containing no air. The areas can vary in size, and they can be both inflammatory and non-inflammatory in nature. Their size may range from a few acini, or lobules affected (a focal consolidation of the lung tissue) or they may involve the whole lobe (a lobar consolidation of the pulmonary tissue). A typical example of the focal inflammatory process in the lungs is focal pneumonia, which is not uncommon in the clinical practice. In some cases foci tend to join, this is called confluent pneumonia. Consolidation foci are sometimes so significant that they involve 2 segments or the whole lobe of the lung. This is a lobar pneumonia (croupous pneumonia).

Pathogenesis: a lung tissue consolidation may develop due to the following pathogenetic mechanisms:

inflammatory infiltration – alveoli are filled with exudate or fibrin (in pneumonia, tuberculosis inclined to caseous breakup);

alveoli are filled with blood in lung infarction associated with thromboembolism or local vascular thrombosis;

spread of connective tissue through a lung lobe in pneumo-sclerosis, carnification;

invasion of tumor tissue into a lung lobe in lung cancer;

atelectasis (obturational or compressive one) and hypoventilation;

congestive heart failure (hypostatic pneumonia).

Patients complain of the following signs: inspiratory dyspnoe\ difficulty in breathing (feeling lack of air) due to respiratory failure (insufficiency); dry cough at the 1st stage of lung abscess, lobar (croupous) pneumonia and in lung tumor. Productive cough in focal pneumonia and lobar pneumonia at the 2nd and 3rd stages, in lung abscess at the 2nd stage; hemoptysis in lung tumor and tuberculosis, pulmonary artery thromboembolism and bronchiectatic disease; chest pain in focal or lobar pneumonia in cases of subpleural location of the inflammation focus. General symptoms, like fever, intoxication, appear depending on the particular disorder, its causative agent and the patient's body state.

Physical examination reveals: chest movement asymmetry (the affected chest part lags behind) in respiration; local chest bulging is possible if the focus of consolidation is big and located superficially, or there may be a retracted chest area in large obturational atelectasis. **Palpation** can determine increased vocal fremitus in the projection of the consolidation and, in the same place: duller or dull **percussion** sound (depending on the size of the damage). **Auscultation** reveals weakened (diminished) vesicular, harsh or bronchial breathing depending on the size of the damage and on the patency of the draining bronchus, apart from that, positive

bronchophony may be heard. Crepitation can be found at the 1st and 3rd stage of lobar pneumonia, as well as fine bubbling rales. Pleural friction rub can be detected in lobar and focal pneumonia located under the pleura, and in case of lung infarction.

Physical findings depending on the size of pulmonary tissue consolidation

Pneumonia	Involve-ment size	Percussion findings	Findings defined		
			Vocal fremitus, broncho phony	Respiration	Collateral\side souffle on respiration\respirat ory souffle
Focal, with fine foci	Several fine foci	Unchanged	±	Vesicular or harsh	Fine bubbling rales
Focal	Within the limits of the segment	Slight dullness	+	Harsh	Fine bubbling rales
Focal, with a large focus	1/4-1/2 of a lobe	Slight dullness	++	Harsh	Fine bubbling rales
Confluent	Over 1/2 of a lobe	Slight dullness	+++	Bronchial	Fine bubbling rales
Lobar	Several segments, a lobe	Dullness	++++	Bronchial	Pleural friction rub

PNEUMONIA

By pneumonia one means a group of acute infectious diseases differing in their etiology, pathogenesis and morphological characteristics, but all typically involving respiratory parts of the lungs and characterized by intra-alveolar exudation.

The term “pneumonitis” is applied to inflammatory processes in the lungs that are of non-infectious nature (like allergic, toxic, eosinophilic, radial one etc).

Pneumonia is one of the commonest diseases in a doctor’s general practice.

At present there is an **accepted pneumonia classification** taking into account the circumstances under which it has developed, peculiarities of the pulmonary tissue infection and the state of the patient’s immunologic reactivity. In accordance with these conditions one can more definitely suppose etiology of pneumonia in the case. These are necessary to keep in mind when administering an anti-bacterial therapy. The following kinds of pneumonia are distinguished.

1. Community-acquired pneumonia (home or ambulatory-acquired)
2. Hospital-acquired pneumonia (Nosocomial pneumonia)
3. Aspiration pneumonia (in epilepsy, alcoholism, impaired swallowing, vomiting etc.)
4. Pneumonia in people with severe immunity deficiencies or immune-compromised ones) – in people with congenital immunity deficiency, in HIV infected people at the stage of AIDS, in drug addiction, alcoholism, oncological diseases, agranulocytosis, in immune-suppressive therapy administered. Patients having lung cancer not uncommonly develop secondary lung infiltration around the tumor (paracancerous pneumonia).

Distinguishing between community-acquired and nosocomial pneumonia is due to essential difference in their etiology, the disease course and prognosis. Community-acquired pneumonia most often develops without any severe somatic disorder. Its course is favorable, it occurs in the period of an epidemics (with influenza, upper viral respiratory infection), in the overcrowded environment, when exposed to animals, birds, etc. Within this community-acquired group one describes some special types like atypical pneumonia caused by intracellular agents such as mycoplasma, Chlamidia, Legionella.

Nosocomial pneumonia develops two or more days after the patient’s admission to the hospital. It is characterized by a severe course, rapidly progressing complications (in about 20%). Hospital-acquired pneumonia occurs frequently in newborns and in elderly patients associated with concomitant diseases (urologic, hematologic, cardio-vascular disorders and immunodeficiency). Sources of infection may include medical staff, transfused solutions, catheters, endoscopes and other medical equipment. Gram-negative microorganisms resistant to many antibiotics play an important part in these infectious etiologies (*Pseudomonas aeruginosa, Klebsiella pneumonia, Escherichia coli*). Besides that, the share of *Staphylococcus aureus, Streptococcus pneumoniae* is big. So high level of lethal outcomes of hospital pneumonia is due to all these causes, at the same time this is the reason of distinguishing this disease form as a special group.

Comparison of Community-acquired and Nosocomial Pneumonia

Characteristics	Community-acquired pneumonia	Nosocomial pneumonia
Cconditions of appearance	Found out before admission to hospital or during two days following admission	Found out 48 hours after admission to hospital and during the whole hospital stay
Original body state	Patients without any severe concomitant disorders	Patients with a severe concomitant pathology (in the postoperative period, after a stroke, with combined traumas, resuscitation unit patients, especially those on ALV – artificial lung ventilation)
Specific features of microorganism	Ordinary microflora, bacteria having – standard culture qualities, resistant to antibiotics and typical of the given area	Bacteria circulating in this hospital and resistant to antibiotic action
Intensity of clinical presentation	Usually bright clinical presentation: marked intoxication, fever	Often obliterated\effaced presentation, which makes it difficult to diagnose the disease on time
Specific features of therapy and outcome of the disorder	Anti-bacterial drugs are sufficiently effective. Vast majority of cases recover.	Antibiotics of reserve are required, not uncommonly in combination. Nosocomial pneumonia is the leading immediate cause of death of resuscitation unit patients

Classification of pneumonia offered by N.S. Molchanov in 1962 (with some supplement and changes) still remains relevant.

Different kinds of pneumonia are distinguished by their etiology. There are:

- bacterial ones;
- viral ones;
- myco-plasmic ones;
- chlamydia trachomatis ones;

- rickettsial ones;
- mycotic ones;
- those of unknown etiology.

They may be distinguished by their pathogenesis as:

- primary ones;
- secondary ones.

They are distinguished by their clinical morphological signs as:

- pleuropneumonia (croupous\lobar one);
- bronchopneumonia (focal one).

They can also be divided by their localization into:

- unilateral (left- or right=sided) one
  a) subsegmental one,
  b) segmental one,
  c) lobar one;
- bilateral (with its extent indicated) one.

They may differ by the degree of their severity as:

- mild ones;
- those of an average severity;
- severe ones.

They may be discerned by presence of complications such as:

- purulent inflammatory lung diseases (lung abscess and gangrene)
- infectious toxic shock;
- exudative pleuritis;
- pericarditis;
- distress syndrome;
- infectious destruction;
- acute respiratory failure.

An approach to pneumonia management tactics divides the disease into two kinds. One includes pneumonia developing in a healthy person having no disorders of other organs or systems which could be complicated by pneumonia and contribute to its appearance. The other kind covers those pneumonia cases which develop in the presence of another chronic broncho-pulmonary disease, or complicating another disease (such as sepsis, leucosis, trauma etc). This factor changes the spectrum of the disease causative agents, which is significant for the medication administered. Pneumonia can take a prolonged course, with its length exceeding four weeks.

Pneumonia Etiology and Risk Factors

The proportion of the disease causative agents differs depending on the conditions of pneumonia development. There may be community-acquired and hospital-acquired pneumonia, primary and secondary ones, aspiration pneumonia and pneumonia in immune-compromised patients.

Currently pneumococcus plays lesser part in causing community-acquired pneumonia than it did before (it decreased from 70-80% of cases to 30-40%). Incidence of atypical causative agents (such as mycoplasma, legionella, clamydia) increased. Viruses (influenza, parainfluenza, adenovirus, respiratory-syncytial virus) still remain important in pneumonia etiology. In one third of pneumonia cases etiology fails to be defined.

The etiological structure of community-acquired pneumonia cases is the following:

- Str. pneumoniae
- M. pneumoniae
- H. influenzae
- Staphylococcus aureus
- Influenza A virus
- SARS-CoV-2
- C. pneumoniae
- L. pneumophila
- gram-negative flora
- unknown causative agent up to 30%

Causative agents of nosocomial pneumonia are most commonly gram-negative bacteria: *Pseudomonas aeruginosa*, Klebsiella *(Klebsiella pneumoniae*), colon bacillus (*E. coli*), Proteus (*Proteus vulgaris*) and staphylococcus, anaerobes, fungi.

In people of the group at risk having chronic obstructive lung disease, heart failure, diabetes mellitus, liver cirrhosis, chronic alcoholism, it is gram-negative bacteria (Klebsiella, Haemophilus influenzae) that play the leading part in the etiology of pneumonia, as well as pneumococcus, staphylococcus and microorganisms associations.

In **aspiration** pneumonia an anaerobic infection is usually revealed: (*Bacteroides fragilis, Prevotella melaninogenica, Fusobacterium nucleatum, Pentococcus*), rarer one can find *Staphylococcus aureus*, *Enterobacteriacea*, not uncommonly one can come across associations of gram-positive and gram-negative bacteria with anaerobic microbes.

In patients with immunodeficiency, (in HIV-infected people) the main causative agent of pneumonia is *Pneumocystis* and *Cytomegalovirus* (60%), as well as fungi and gram-negative bacteria.

The following are **risk factors** for pneumonia development:

- age (children and elderly people);
- smoking:
- chronic diseases of lungs, heart, kidneys and gastro-intestinal tract;
- immuno-deficient state;
- exposure to birds, rodents and other animals;
- travelling (trains, planes, railway stations, hotels);
- exposure to cold.

One of the aggressive risk factors triggering lung disorders development is smoking due to the action of tobacco smoke decreasing respiratory organs defensive mechanism ability. Atypical pneumonia results when a person is exposed to infectious sources like birds, rodents, or while travelling (water in a hotel, air conditioning systems may be inhabited by legionella. Overexposure to cold contributes to impairment of mucus barriers of the respiratory tract.

Risk factors for nosocomial pneumonia development include: the patient's stay in resuscitation or intensive care units, his\her septic state, postoperative period, artificial ventilation of lungs, bronchoscopy, etc.

Patients with primary and secondary immunodeficiency are at high risk of pneumonia.

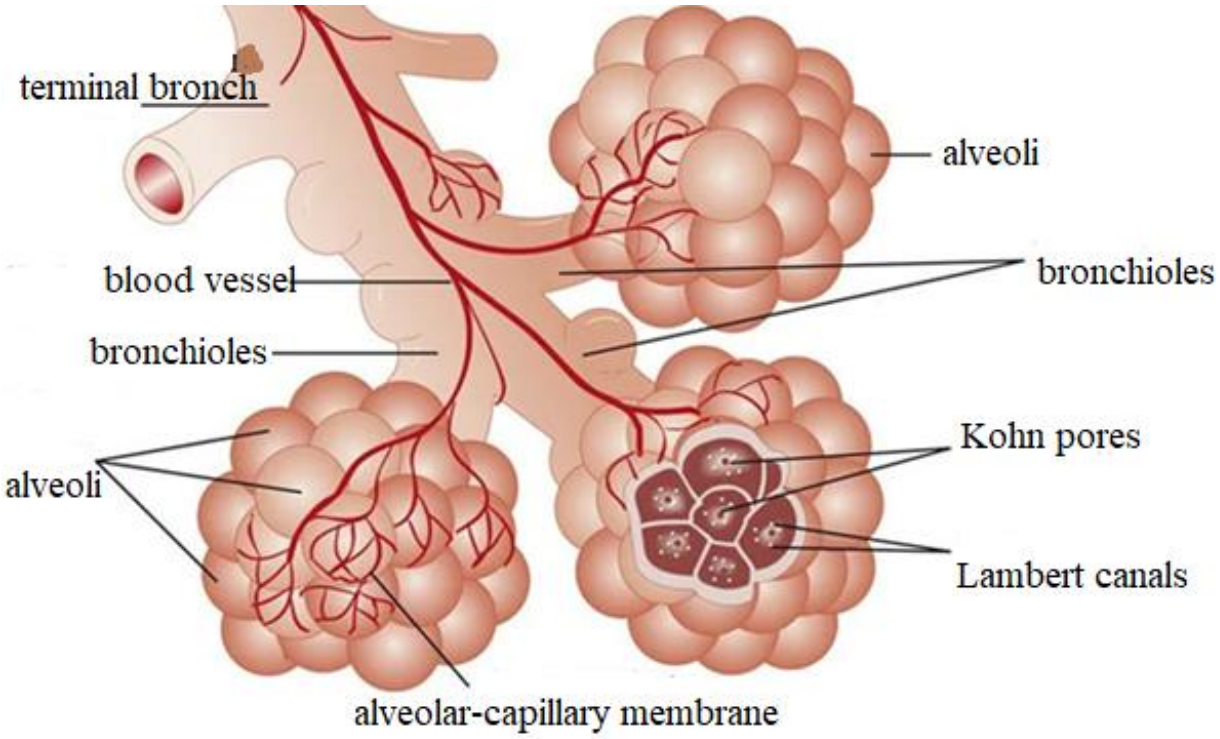
Pneumonia Pathogenesis

Lung respiratory departments are most frequently invaded by microbes through bronchi in micro-aspiration, i.e. while nasopharyngeal secretion is inhaled. In case of primary infection hematogenic or lymphogenic spread of infection are of no significance. They tend to be observed in sepsis and chest injuries. Reduced protection on the part of the respiratory tract mucous membrane mechanisms, impaired ciliated epithelium function, lowered mucociliary clearance, weakened microbe elimination, bronchial tree epithelium impaired by viruses or chemical substances, impaired draining function of bronchi, lowered cough reflex, diminished alveolar macrophags and neutrofile activity etc – these factors contribute to infection invasion and spread by micro-aspiration.

Due to ciliary epithelium dysfunction, increased amount and viscosity of bronchial secretion, adhesion of microorganisms to the bronchial tree epithelial cells

surface takes place. Then the microbes colonize the epithelial cells and are inhaled into the lung respiratory departments (so aspiration occurs).

In lobar pneumonia pneumococci located around the edema (along its perimeter) spread to the neighboring regions by way of contact through Kohn pores and Lambert channels connecting lung lobules provide collateral ventilation in physiological conditions.



Acinus structure and routes of collateral ventilation of the secondary lung lobule

Inflammatory process first affects alveoli, then occupies more space – the whole lobe or a few lobes. Thus, pneumonia becomes a lobar pneumonia. Fibrinous exudate appears in the alveoli and fibrinous layers cover the pleura. The lung lobe and the pleura covering it are affected at the same time, because of which lobar (croupous) pneumonia is sometimes called pleuropneumonia.

Focal pneumonia (bronchopneumonia) often develops with an edema, as well as infiltration of bronchi and bronchioles mucous membrane in the background. It is a consequence of these organs being affected. Primary process starts from bronchioles spreading to the surrounding adjacent pulmonary tissue. Pneumonic foci usually arise in the posterior or posterior-lower portions of the lungs. There is exudate collected in the alveoli, with admixture of mucus containing a large number

of neutrophils, macrophages, erythrocytes, alveolar epithelium sloughed off. Inter-alveolar septa are pierced with cellular infiltration.

Focal pneumonia may have a number of specific features depending on its causative agent. *Staphylococci, streptococci* and *Pseudomonas aeruginosa*, due to their producing exotoxin and their high virulence, cause purulent dissolution, fusion, necrosis of the pulmonary tissue and abscess formation. Freelander pneumonia caused by Klebsiela results in clotting fine vessels and forming extensive infarction-like pulmonary tissue necrosis. Because of the highly marked exudation bacterial edema invades healthy areas of lung tissues through clefts in alveolar septa, thus giving pneumonia its confluent character.

LOBAR (CRUPOUS) PNEUMONIA

(The 2nd stage corresponds to lobar consolidation of the lung tissue)

Inflammatory infiltration of pulmonary tissue progresses changing the character and degree of the airiness of the region affected. Owing to this fact, main clinical manifestations, as well as physical examination findings, differ at different stages of disease. This clear division into stages is especially inherent to lobar pneumonia. One can notice 3 stages of the disease course in patients with lobar pneumonia. They are: congestion (flash), hepatization, resolution.

At the stage of **congestion** (**flash**) pulmonary tissue edema and infiltration take place. However, at this time the lobe still preserves airiness, a lot of microbes can be found in the edematous fluid. This stage lasts 1 – 3 days.

At the height of the disease course (red or grey hepatization) the lung lobe becomes solid and airless. In cases when hepatization is red fibrinous exudate in the alveoli contains a large number of erythrocytes, when it is grey leucocytes prevail. The region affected changes its color into red or greyish-yellow and grows solid, airless, with the effect of graininess at the cut. It gets drowned in the water. The stage lasts up to 9 days.

At the **resolutio**n stage exudate and its phagocytes become thinner, dissolve and partly get out with phlegm due to the proteolytic enzyme emerging as a result of leucocytes destruction. After that a part of the affected region gains airiness again, though a part of it still contains exudate. With time, the whole affected lung recovers its airiness in a favorable outcome.

Clinical Manifestations of Lobar Pneumonia

Lobar pneumonia is characterized by acute onset with typical **complaints** of shivering chill, fast (within 2-3 hours) increase of body temperature from normal one to 39-40° C, appearance of dry cough, and shortness of breath by the end of the

first stage. The cough becomes productive at the second stage, there are ***rusty (rust-colored)*** sputum and pain on the affected part, intoxication and respiratory failure are marked at their maximum. At the third stage the cough is characterized by abundant purulent sputum discharge.

Physical examination during the first stage makes it possible to reveal cyanosis, *Herpes labialis et nasalis* (lat.), mental confusion, sometimes delirium and hallucinations. A forced body position can attract attention when the patient is lying on the back, or he\she may have a lateral body position on the affected side. In case of the sitting position the patient presses the affected chest area by his\her hand to limit its excursion. The affected chest part is slower in the process of breathing, the cheeks are hyperemic, the lateral sides of nose become swollen, there is a herpetic eruption around the mouth angles. Cyanosis is observed at the first stage of the disease. Reduced mobility of pulmonary edge is determined by **topographical percussion**, and dullness of percussion sound tone with tympanic resonance (**duller-tympanic sound**) is found out by **comparative percussion** above the affected lobe. **Auscultation** of the same place reveals weakened (diminished) vesicular breathing changing to a harsh one (during the process of pulmonary tissue consolidation increase and improving sound conductivity), soundless crepitation (*crepitation indux*). Strengthening of vocal fremitus and positive bronchophony appear by the end of the 1st stage.

Intoxication and respiratory failure are clinically intense at the second stage. **Dull percussion** sound is revealed above the affected lobe, and **auscultation** reveals bronchial breathing and pleural friction rub. Rales and crepitation above the affected lobe are not heard at the second stage due to complete filling of alveoli by exudate and absence of ventilation of pulmonary tissue. Vocal fremitus is reinforced, and bronchophony is positive.

At the resolution stage, with the patient health improvement, the mobility of pulmonary edge is gradually increasing, according to **topographical percussion** examinations. On **comparative percussion** the pulmonary sound again becomes **duller-tympanic** (due to airiness restoration), **auscultation** reveals harsh breathing and sonorous fine bubbling rales. Later the breathing becomes vesicular weakened one, crepitation (crepitation redux) returns. The number of fine-bubbling rales is gradually decreasing. Vocal fremitus changes from its intense form to normal, symmetric, correlating with percussion and auscultation phenomena. This indicates decomposition of the lung tissue infiltration, and bronchophony becomes negative.

FOCAL PNEUMONIA

Focal pneumonia manifestations (in the form of intoxication and intensity of fever) are significantly associated with the character of causative agent, initial condition of the body and size of the pulmonary tissue involvement due to the infection spread to pulmonary tissue from bronchioles and small bronchi. The intensity of respiratory failure and physical data depend on the size of focal involvement and depth of its localization.

Cough which quickly changes from dry to productive one, accompanied by purulent sputum discharge, moderate or marked shortness of breath, sometimes lateral chest pains (in case of pleura involvement) are characteristic **complaints** for such patients.

During **examination** one can notice feverish blush of the patient's cheeks with a cyanotic tint (in case of considerable involvement of lung tissue - for example, in confluent pneumonia), delay of the corresponding chest part in breathing process (in case of the focus localization in the lower lobe). During **topographic percussion** the limitation of mobility of the lower lung border is identified at the same place. On **comparative percussion** it is possible to determine a limited area of duller-tympanic sound (partially preserved airiness), and in case of confluent pneumonia – a focus of dull sound.

Vocal fremitus above the consolidation focus can be increased, and bronchophony becomes positive.

Harsh breath is identified during **auscultation**; and in case of confluent pneumonia – bronchial breath. Harsh breath is heard due to the fact that in case of focal consolidation there are conditions to perform bronchial breathing, but surrounding pulmonary tissue changes it in such a manner that it is heard as harsh one. The most pathognomonic feature of focal pneumonia is sonorous fine bubbling moist rales limited by the center of inflammation focus (local). They are sonorous because the compressed pulmonary tissue resounds and strengthens the sound. In case of subpleural localization of the focus, pleural friction rub is heard.

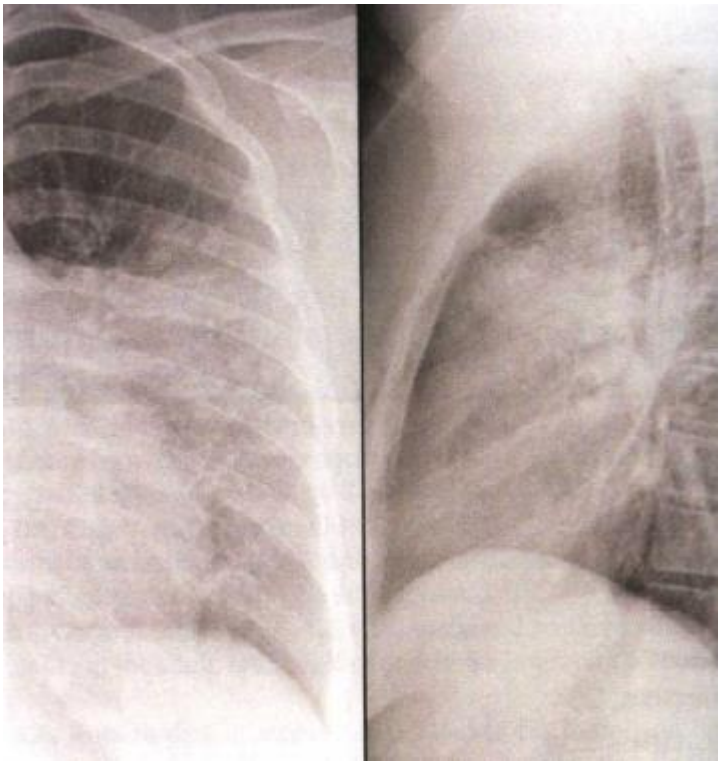
Instrumental and Laboratory Diagnosis of Pneumonia

The basic method to confirm the diagnosis of pneumonia is two-dimensional (in two projections) radiographic examination of the lungs (Chest X-ray). The description includes areas of shadow with recognizing their dimensions and intensity, localization, number, pleura reaction (in case of pleuropneumonia) and lymph nodes of the lung roots, as well as intensified lung pattern (in case of bronchopneumonia).

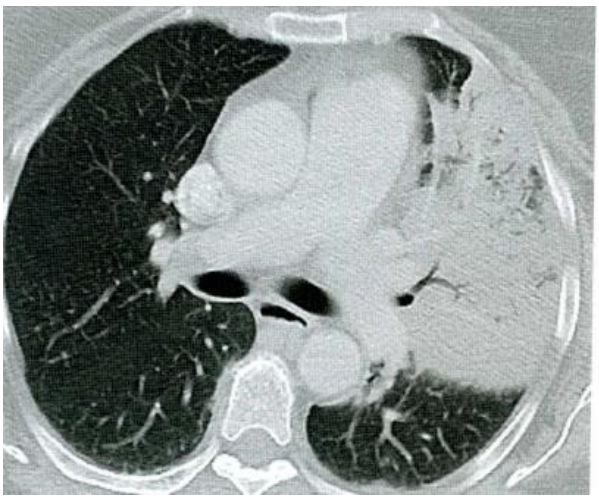
Radial methods of examination identify 3 types of pneumonia infiltration:

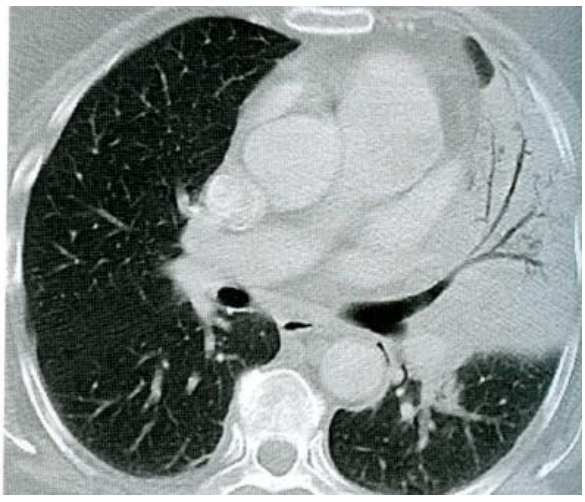
Pleuropneumonia (alveolar) type is characteristic of the syndrome of lobar consolidation of pulmonary tissue. This type of infiltration involves the largest part

of the lobe or entire lobe, as the pleura is a natural border of distribution of the causative agent in pores of Kohn and Lambert canals. It is characterized by the shadow of pulmonary tissue of various intensity within anatomic borders of the lung. Computer tomographic image demonstrates manifestation of consolidation (induration) of pulmonary tissue with a necessary condition to preserve patency of bronchial tubes (the symptom “airiness of bronchogram”). The most intensive infiltration density is noticed in subpleural parts where there is the largest number of alveoli, the interlobar pleura is bent towards the involvement space. Towards the lung root, the infiltration intensity is usually decreased according to the reduction of pulmonary parenchyma amount and prevalence of bronchial vascular fascicle.



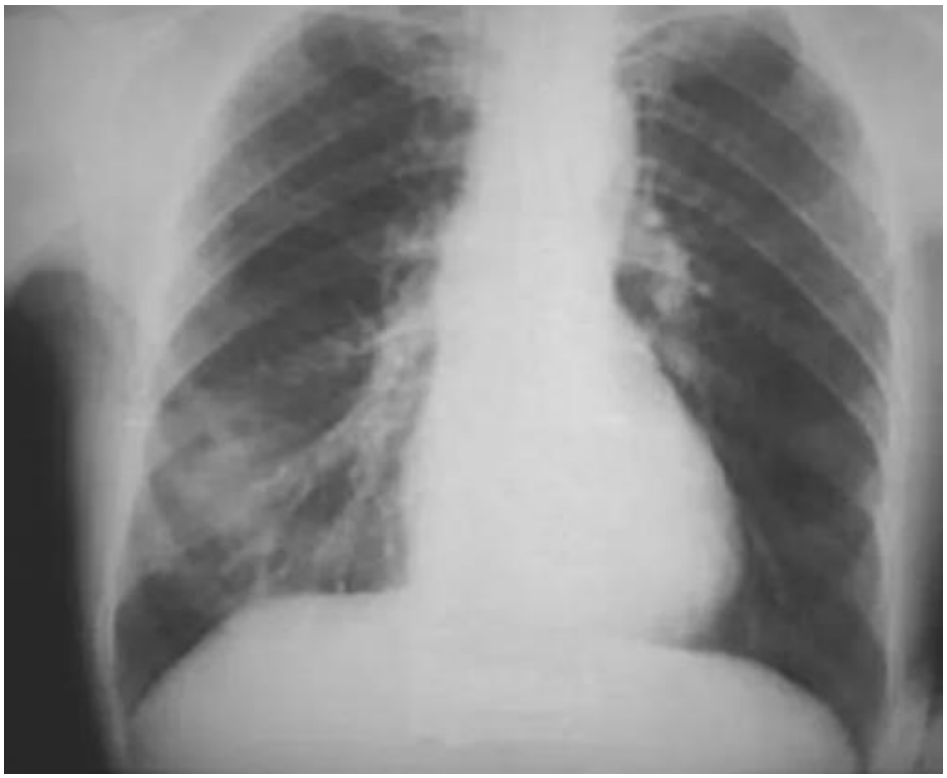
Radiographic image. Pleuropneumonia type of infiltration. Shadow of the lower lobe of the left lung



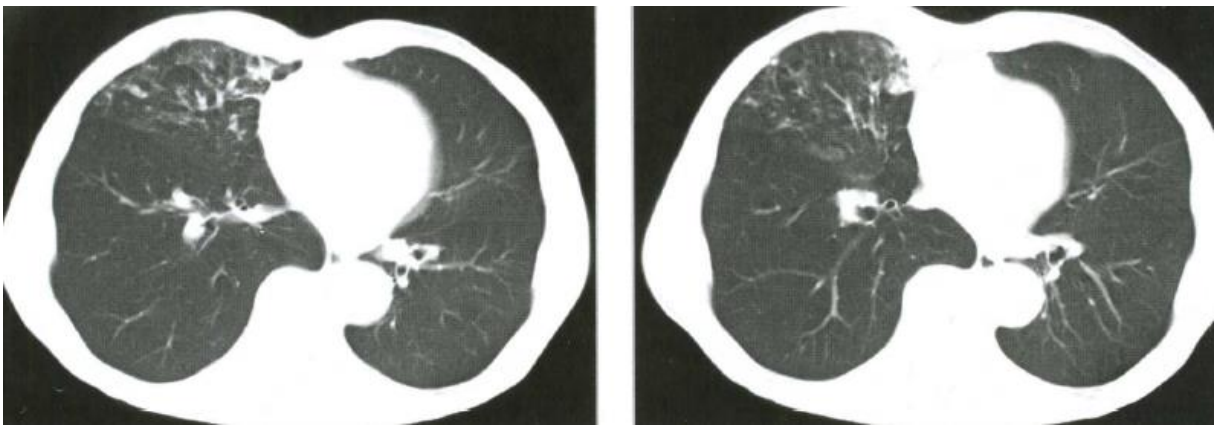


Computer tomographic image of the lungs. Pleuropneumonia type of infiltration. Consolidation of the lower lobe of the left lung (consolidation, the symptom “airiness bronchogram”)

Bronchopneumonia type is characteristic of the syndrome of focal consolidation of pulmonary tissue. It is characterized by the presence of areas of the consolidation initially located in one or two bronchopulmonary segments (transition of inflammatory process from a bronchial tube wall to pulmonary tissue). In case of progression this type of infiltration provides numerous bilateral areas with indistinct contour. According to computer tomographic image the process usually begins with exudate broncholith (an image “tree in kidneys”) and it is mainly localized in the nuclear parts of the lungs having the biggest number of vascular fascicle.



Radiographic image of the lungs. Bronchopneumonia type of infiltration. Shadow of consolidations of pulmonary tissue in the lower lobe of the right lung

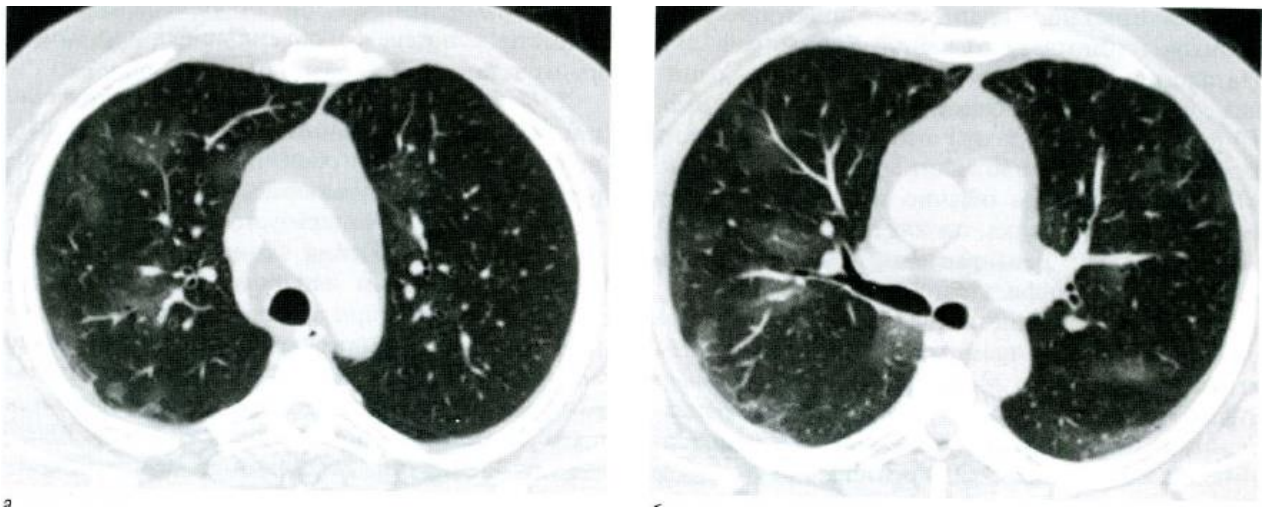


Computer tomographic image of the lungs. Bronchopneumonia type of infiltration. Focal shadow\opacity of pulmonary tissue in the middle lobe of the right lung

Despite the existing practice of empirical antibacterial therapy for pneumonia based on suspected agents and their sensitivity to antibacterial medicine, the following necessary points can be used to diagnose and find the proper treatment for the patient:

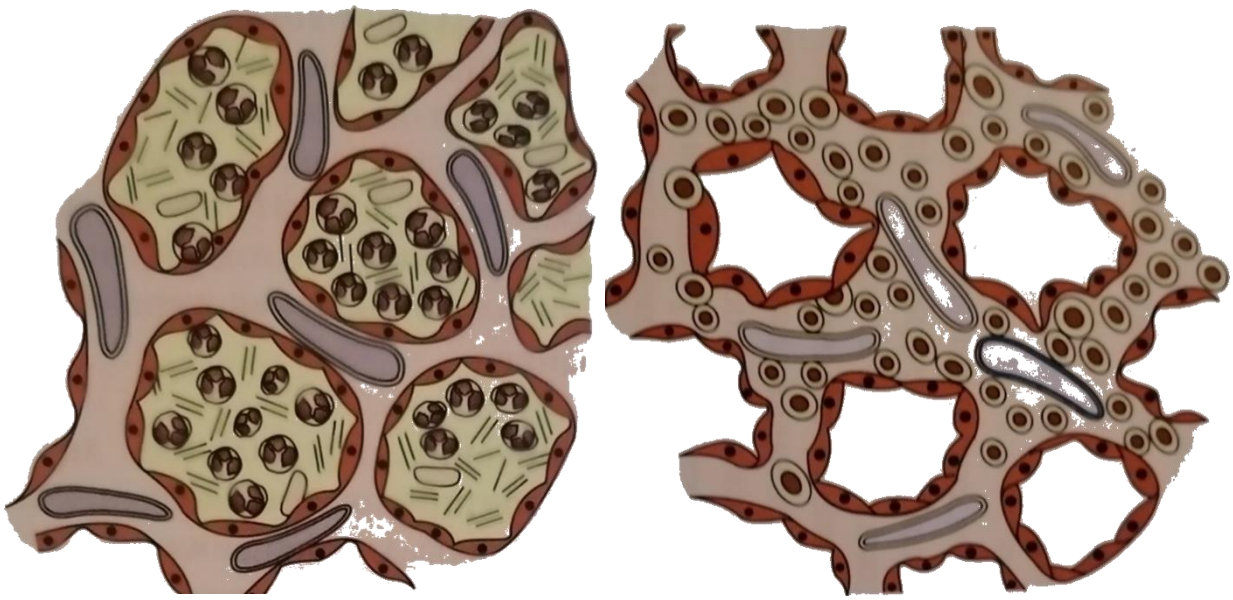
- clinical (common) blood test which includes leukocytosis and stab neutrophil shift of leukogram;
- biochemical blood test: indicators of inflammatory process activity (proteinogram, fibrinogen, C-reactive protein);
- microscopic sputum investigation by Gram-staining;
- general sputum test, including examination for BK (Koch's bacteria – Mycobacterium tuberculosis) with Ziehl-Neelsen staining to reveal acid-resisting flora;
- sputum culture with quantitative identification UQI/ml and sensitivity to antibiotics.

Additional methods of investigation include: computer tomography of the lungs administered in case of inconsistency between the clinical data and the data received by traditional radiographic examination (a marked clinical presentation vs normal radiograph without any complaints or physical signs but with radiological changes present). It is particularly recommended in case of viral pulmonary disorders which are characterized by *interstitial type* of infiltration that is poorly visible at traditional radiological examination, it is manifested in the form of intensive pulmonary pattern. At computer tomography, infiltration of this type is characterized by the symptom of "ground-glass opacity", that is the appearance of the areas of lowered airiness of pulmonary tissue against the background of which there can be seen bronchial tubes and vessels.



Computer tomographic image of the lungs. Interstitial type of infiltration. The symptom of “ground-glass opacity” (COVID-19)

This type of infiltration is particularly characteristic of SARS-CoV-2 infection, the viral involvement of the lungs is not identified by physical methods as it has the features of morphological pattern, it is defined by radiation methods only.



Morphological pattern of alveolar and interstitial type of infiltration of pulmonary tissue

The indications for computer tomography include some aspects of differential diagnosis – suspicion of a tumor presence, an abscess formation, thromboembolism of pulmonary artery, decreased lobe volume, etc.

PULMONARY CAVITY PRESENCE SYNDROME

A pulmonary cavity is usually formed against the background of inflammatory infiltration. Thus, purulent dissolution, or necrosis is formed in the center of inflammatory infiltrate. Healthy pulmonary tissue surrounding the place of dissolution forms a capsule, isolating the cavity and interfering with distribution of purulent process. Some time later discharge of purulent secretion or necrosis mass through a bronchial tube takes place. So, air enters the area of the focus through this bronchial tube.

To be detected with the help of physical methods, the pulmonary cavity should have the following features:

1. The cavity diameter should not be less than 4-5 cm.
2. The cavity should be connected with its environment through the bronchial tube and contain air.
3. The cavity should have smooth walls.
4. The cavity should be closely adjacent to the chest surface.
5. The cavity should have a firm (solid) capsule, and the surrounding pulmonary tissue should be condensed.

In case of the absence of these criteria the pulmonary cavity can not be identified by physical methods ("inarticulate" cavity) and can be found out only by radiological methods of examination.

Pulmonary cavity presence syndrome (cavitary syndrome) is observed in case of pulmonary tuberculosis (tubercular caverns), lung abscess, bronchiectasic disease (in case of the presence of wide bronchiectasis).

LUNG ABSCESS

Lung abscess is one of the most frequent pulmonary complications in case of pneumonia.

Lung Abscess Pathogenesis

Lung destruction in the area of pneumonia infiltration is characteristic of the inflammatory processes caused by staphylococci and other Gram+ cocci – due to the influence of toxins the pulmonary tissue is destroyed. A firm connective tissue capsule of healthy pulmonary tissue is formed around the area of destruction isolating it (1st stage) from the areas of healthy lung. As a result of it, a closed space is formed, where lysis of pulmonary tissue continues, gases are produced due to vital activity of bacteria and the pressure in the cavity is gradually increasing. Later (within some days) the capsule becomes thinner and is damaged. Various variants of the abscess damage are possible.

The most frequent variant is **breakethrough** (2nd stage) into the pulmonary tissue – because it is penetrated by bronchial tubes and bronchioles, in this case purulent contents will come out by its natural way with cough, and the cavity will be gradually filled in with air. In case of peripheral localization of the abscess its damage and breakthrough into the pleural cavity is possible. In this case purulent pleurisy (pleural empyema) develops, and this considerably worsens the patient's condition. The most threatening condition is breakthrough into the mediastinum, filled by friable connective tissue, the pus impregnates it, causing dissolution of nerves there (of the vagus nervus and the diaphragmatic one). In such a variant of the damage the patient dies in some hours.

Clinical Features of Lung Abscess

In the process of management of a hospitalized patient with suspected pneumonia, change of the patient's **complaints** character – sudden reduction or disappearance of the sputum, cough becomes dry and unproductive – will be a threatening clinical signs connected with an abscess formation. At the same time the clinical symptoms in the form of intoxication and respiratory failure remain and even aggravate. During the second stage, in case of the abscess damage and breakthrough into the bronchial tube, the cough becoming productive - the patient expectorates purulent, liquid, black-grey sputum, with an acute and unpleasant smell (putrefactive one), frequently in big amounts (sputum discharge "fills the mouth"). Intensity of intoxication during the abscess drainage is usually decreased.

Physical examination can reveal cyanosis, the affected part of the chest is retarded in the breathing process.

Comparative percussion does not allow to verify abscess development – dull percussion sound will be identical in both cases: in pneumonia, and at the first stage of abscess (the space is filled by pus). Vocal fremitus is intensified in case of pneumonia, and at the first stage of abscess (before breakthrough of the abscess). It becomes weakened. Bronchophony, that is positive above the area of pneumonia infiltration, becomes negative with the appearance of the closed cavity filled with pus due to obturation and drainage of bronchial tube and deteriorated conductivity of sound. Auscultation above the abscess area at the first stage reveals abruptly weakened breath, the rales disappear.

Physical data **at the second stage** will depend on the proportion of pus to air in the abscess cavity, as well as on its localization in relation to the chest surface. Quite a big size of the cavity (over 5 cm), presence of the firm capsule conducting sound, make bronchophony positive and vocal fremitus more intense, as besides the compressed pulmonary tissue conducting sound, the air cavity produces good

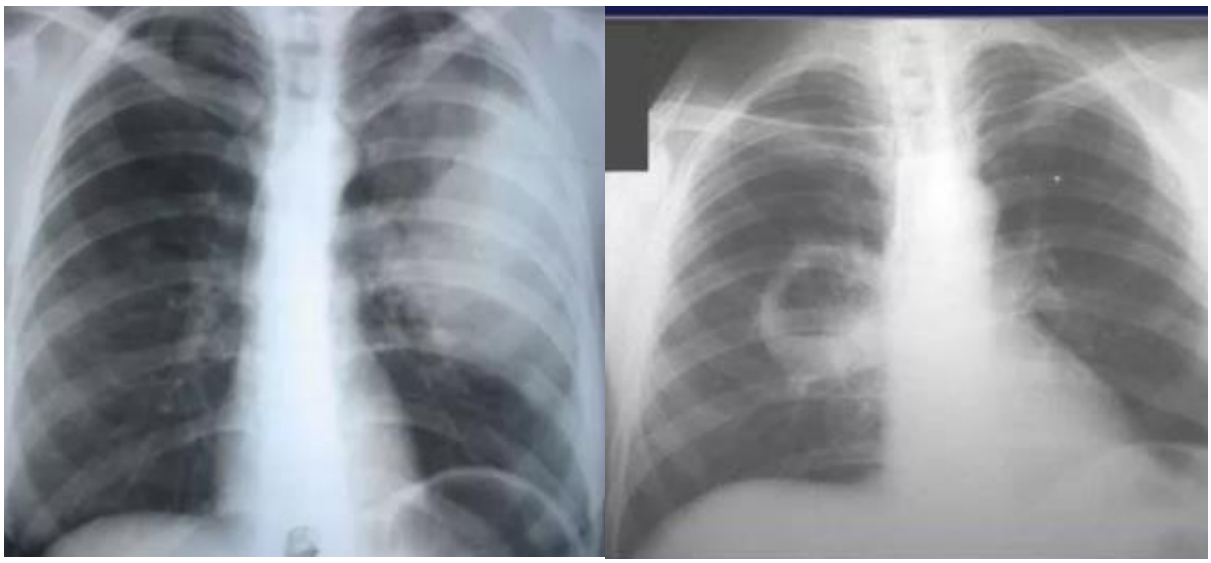
resonance that additionally strengthens the sounds conducted from vocal chords along the bronchial tree.

Percussion sound becomes tympanic, and sometimes in the presence of big smooth caverns it gets a metal tone. If the cavity is connected with the environment by a narrow and twisted bronchial tube it can lead to the appearance of the symptom "the cracked pot". It appears during percussion because the air leaves the cavity gradually in some portions, and this produces heterogenous percussion tone with a specific nuance.

Auscultation reveals bronchial breathing as the cavity is a kind of continuation of respiratory passages and conducts laryngotracheal breathing in case of the presence of a firm capsule. On the periphery this is called bronchia breathing. Besides, the firm capsule surrounding the cavity, produces additional resonance and vibrates being filled with air - the combination of bronchial breathing with additional overtone that appears due to the cavity fluctuation (that is named ***amphoric breathing***) can be heard. Above the cavity, closer to the lung root, there can be heard fine and average bubbling moist rales, i.e. the presence of liquid exudate in bronchial tubes of small and middle size causes, at air passage, bubbles that burst later, and the capsule increases these sounds. When the patient changes the position of his/her body it is possible to hear pathognomonic sign "phenomenon of a falling drop" - flowing of pus in the cavity.

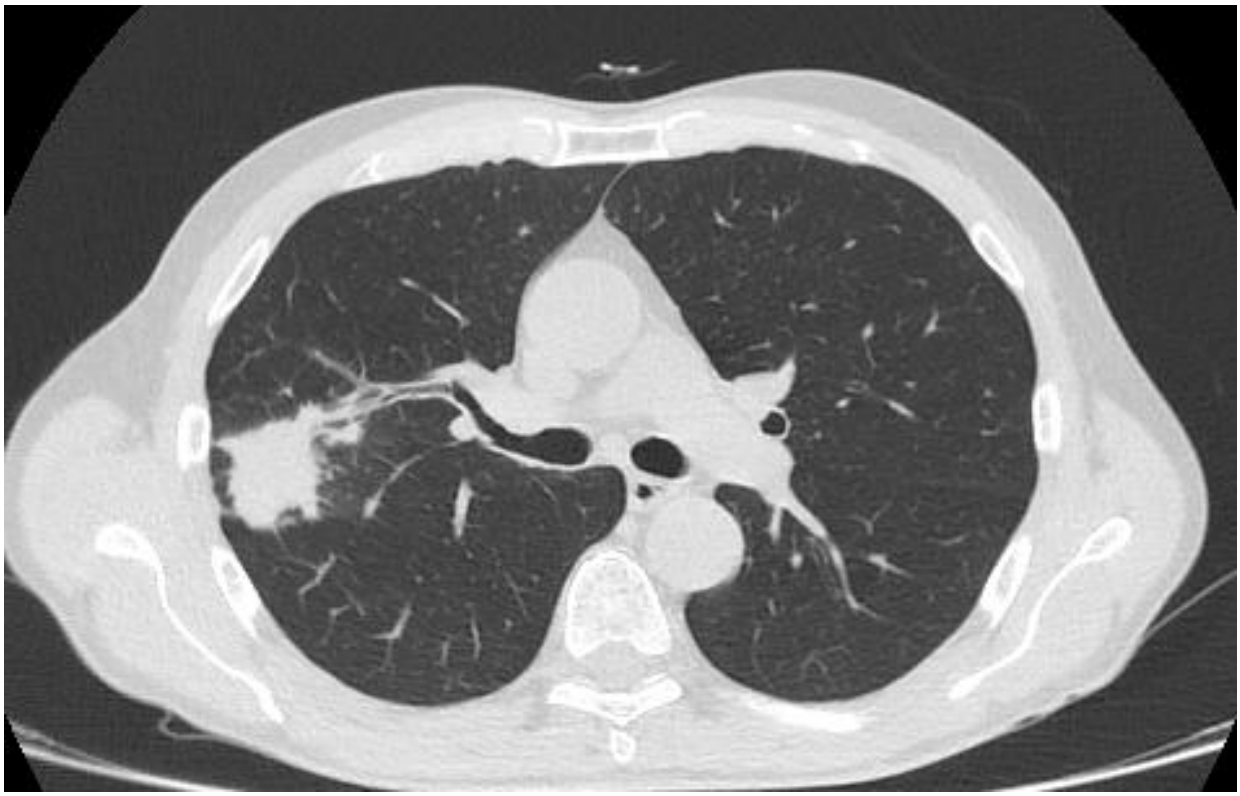
Instrumental and Laboratory Diagnosis of Lung Abscess

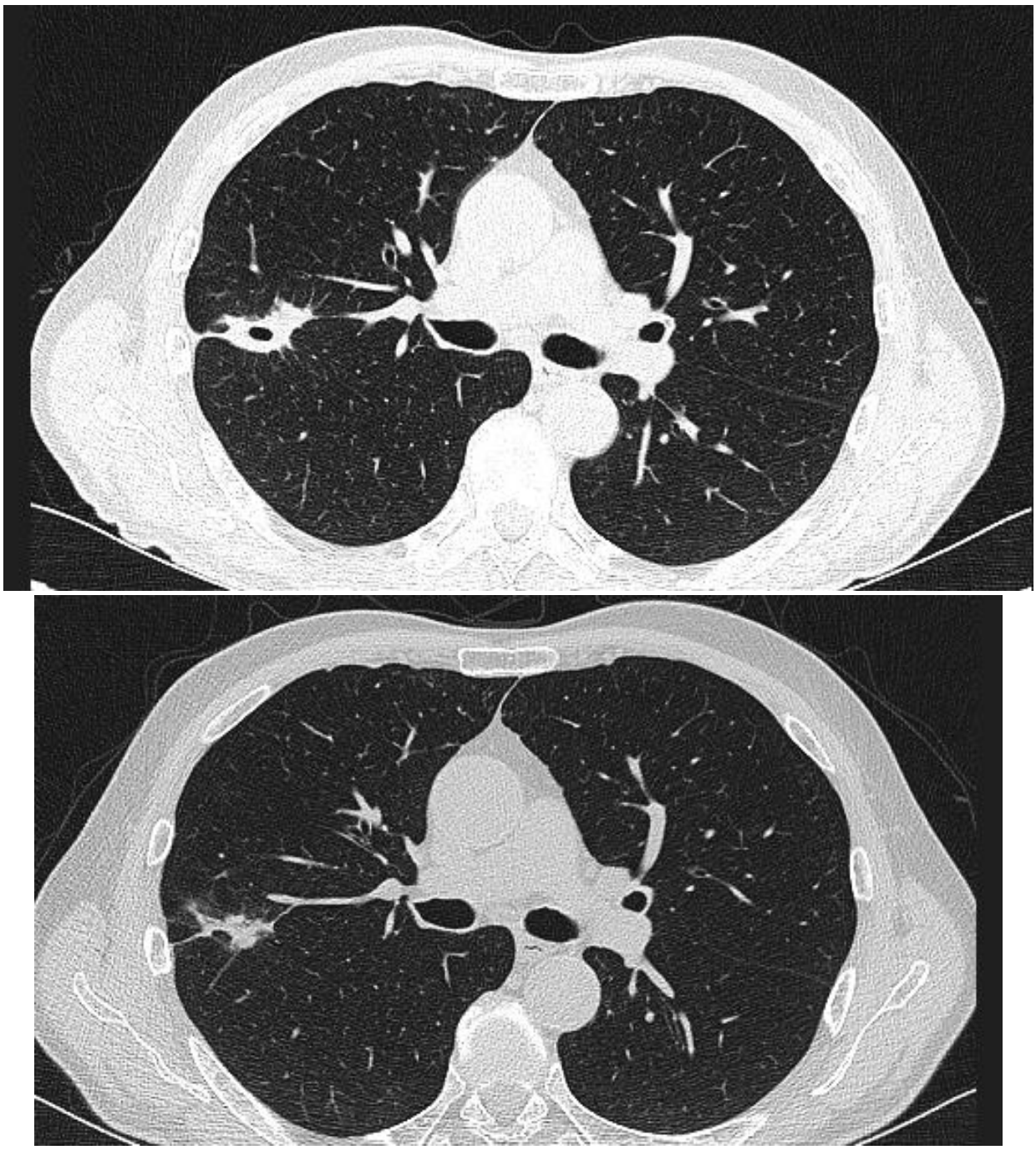
The most informative instrumental method to identify an abscess is raidiography of the lungs (chest X-ray). At the first stage one describes the appearance of roundish shadows with distinct borders and homogeneous contents, at the second one - levels of liquid in roundish cavities - with some radiolucency above and homogeneous shadow below. During the emptying of the abscess of liquid contents the proportion of radiolucency and shadow changes towards the increased area of radiolucency.



On the left – the 1st stage of lung abscess of the upper lobe of the left lung (a roundish shadow)
On the right – the 2nd stage of lung abscess of the middle lobe of the right lung (a roundish cavity containing air and the exudate – a horizontal level is visible)

Pulmonary tissue consolidation syndrome changing to the pulmonary cavity presence syndrome during the progress of lung abscess course is finished by the formation of local pulmonary fibrosis.





Lung abscess, the course progress. The 1st stage (at the top), the 2nd stage (in the middle), local pulmonary fibrosis (below).

After revealing the lung abscess the treatment should be performed at the department of thoracic therapy – i.e. cavity drainage, and filling it in with antibiotic solutions.

LUNG ATELECTASIS

Atelectasis is considered a noninflammatory consolidation of pulmonary tissue developing due to pulmonary collapse of an entire lung or a part of a lung.

Etiology and Pathogenesis of Lung Atelectasis

According to the development mechanism, one can identify obturation atelectasis (closing of bronchial tube lumen from inside by a tumor, a foreign particle or a mucoid plug) and compressive atelectasis (compression of pulmonary tissue during pathological processes in pleural cavity – due to accumulation of liquid or air, tumor processes or lymphadenopathy). Less often atelectasis develops in case of decreased respiratory muscles tone in weakened patients (distensive one), at traumas or chest surgeries due to bronchospasm and blood flash into capillaries (contractile one).

Partial preservation of airiness of pulmonary tissue and bronchial tube permeability mean incomplete atelectasis, and total disappearance of air and absence of bronchial tube permeability is a complete one.

Atelectasis development is accompanied by decreased intra-alveolar pressure during alveoli collapse, dilatation of blood and lymphatic vessels with congestion of blood and lymph, and also with reduction of intrapleural pressure.

Clinical Features of Lung Atelectasis

A characteristic **complaint** in case of atelectasis is shortness of breath associated with the decreased area of respiratory surface, the degree of which depends on the involvement space.

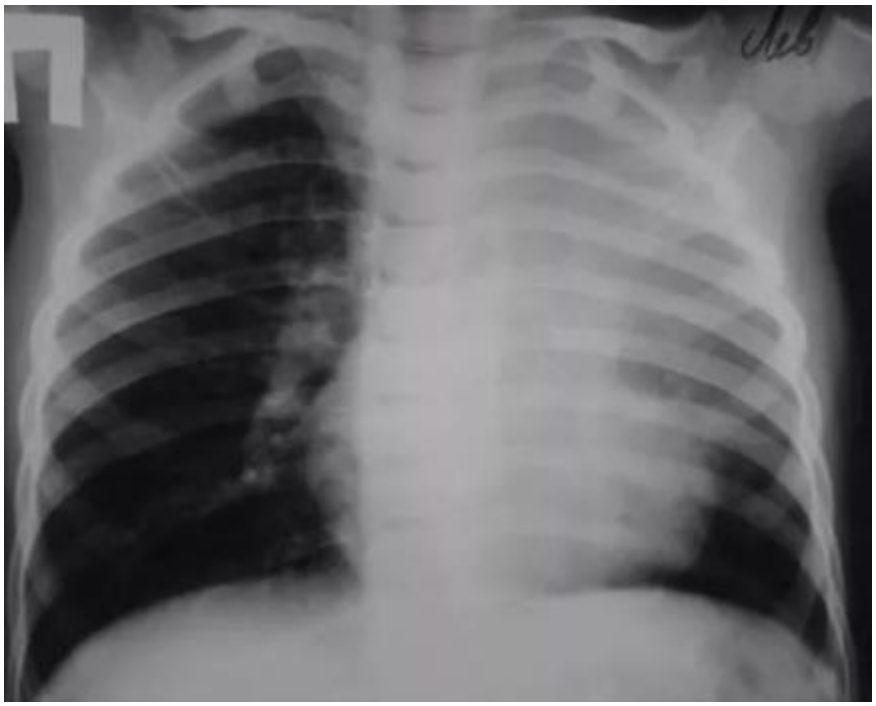
Physical examination reveals decreased size of the corresponding half of the chest, with this one being delayed in breathing process, intercostal retraction and narrowing.

According to **topographical percussion** there is a limited mobility of the lower pulmonary border. Comparative percussion above the atelectasis zone reveals dull percussion sound, and in case of incomplete atelectasis duller-tympanic sound is revealed. At **auscultation** the breathing is not heard or it is vesicular and very weak (***diminished vesicular breathing***). In case of compressive atelectasis (a pressed lung) there can be identified ***bronchial breathing*** due to intensity of the sound provided, by a noninflammatory consolidation of pulmonary tissue. Vocal fremitus is weakened or unidentified, bronchophony is negative. In case of compressive atelectasis one may notice intensity of vocal fremitus and a positive symptom of bronchophony.

Instrumental and Laboratory Diagnosis of Lung Atelectasis

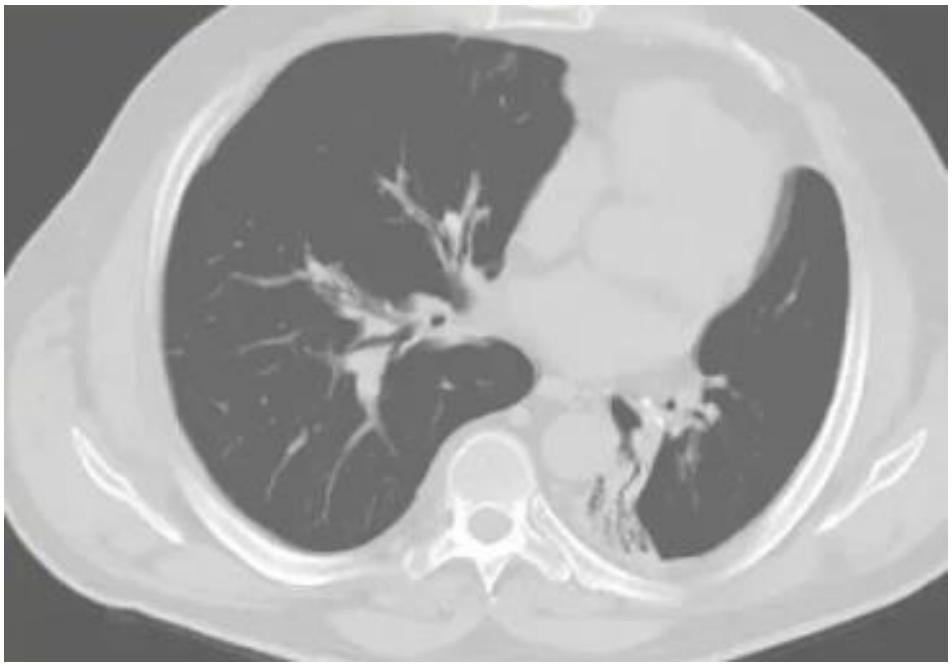
Radial methods of investigation (X-ray radiography, computer tomography) have a crucial importance in atelectasis diagnosis. The basic radiological symptom is the presence of homogeneous shadow within anatomic borders of the lung, a high

position of the diaphragm, narrowing of intercostal intervals, displacement of mediastinum toward the involvement area and compensatory increase of airiness of the healthy lung areas.



Atelectasis of the upper lobe of the left lung

Computer tomography scan of the atelectasis zone can partially demonstrate the deformed twisted bronchial tubes in the atelectasis zone.



Atelectasis of the left lung

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