Ministry of Health of the Republic of Belarus
Educational Institution
“Gomel State Medical University”

Department of Pathological Anatomy

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METHODICAL GUIDELINES

for conducting a practical class
in the academic discipline “Pathological Anatomy”
for students
of the 3rd year, Faculty of International Students (instruction in English),
enrolled in the specialty
7-07-0911-01 “General Medicine”

Topic: “Respiratory system diseases: acute bronchitis and pneumonias”

Duration: 3 hours

Approved at the meeting of the Department of Pathological Anatomy
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2025

EDUCATIONAL AND INSTRUCTIONAL GOALS, OBJECTIVES, AND MOTIVATION FOR MASTERING THE TOPIC

Educational Goal:

- To study the definition, classification, etiology, pathogenesis, morphology, complications, and outcomes of acute pneumonia. To analyze the differences between community-acquired and hospital-acquired (nosocomial) pneumonias. To examine the morphological features of various types of acute pneumonia: lobar (fibrinous) pneumonia, bronchopneumonia (lobular), interstitial pneumonia, and aspiration pneumonia. To describe the classic stages of lobar pneumonia (congestion, red hepatization, grey hepatization, resolution) and their microscopic correlates. To discuss the role of microbial agents (Streptococcus pneumoniae, Staphylococcus aureus, Klebsiella pneumoniae, Haemophilus influenzae, Legionella, Mycoplasma, viruses, fungi) and host factors (age, immunocompromised status, underlying chronic diseases). To analyze complications such as lung abscess, empyema, organization with carnification, respiratory failure, sepsis, and death. To emphasize the importance of morphological diagnosis in guiding antibiotic therapy and predicting outcomes.

Instructional Goal:

- Within the framework of the educational process for this academic discipline, the student must acquire not only theoretical knowledge, practical skills and abilities in the specialty but also develop their personal potential. They should cultivate qualities of responsibility and patriotism, be prepared to actively participate in the economic, socio-cultural, and public life of the country, recognize the social significance of their future professional activity, understand the norms of medical ethics and deontology, and learn to observe academic and labor discipline. In the course of studying the educational material, students should realize the importance of maintaining a healthy lifestyle and, as an example in the future, when performing professional duties, set an example for those around them and their patients.

Objectives: Upon completion of the practical session, the student must:

***Know*:**

- The definition and classification of acute pneumonia;
- The etiology and pathogenesis of lobar pneumonia and bronchopneumonia;
- The macroscopic and microscopic features of the stages of lobar pneumonia;
- The morphological features of bronchopneumonia;
- The complications and outcomes of acute pneumonia;
- The causes of death in patients with acute pneumonia.

Be able to:

- Distinguish lobar pneumonia from bronchopneumonia based on macroscopic and microscopic findings;
- Identify major general pathological processes and diseases using histological specimens under light microscopy;

- Diagnose pathological processes and diseases based on the description of macroscopic and microscopic changes in organs and tissues of the body.

Master:

- Basic techniques for working with a microscope;
- Skills in clinical-anatomical analysis;
- The fundamentals of synthetic generalization of morphological diagnostic signs of diseases and their correct interpretation in cause-and-effect relationships.

Motivation for Mastering the Topic:

Acute pneumonia remains one of the leading causes of morbidity and mortality worldwide, particularly in elderly patients, young children, and immunocompromised individuals. Accurate morphological diagnosis is essential for appropriate antimicrobial therapy and management of complications. Understanding the pathological features of different types of pneumonia is critical for clinicians in internal medicine, pulmonology, intensive care, infectious diseases, and general practice, as well as for pathologists who provide diagnostic guidance.

QUESTIONS

1. Acute bronchitis. Definition. Etiology. Pathogenesis. Morphology. Complications and outcomes.
2. Bronchopneumonia. Definition. Etiology. Pathogenesis. Morphology. Complications and outcomes.
3. Lobar pneumonia. Definition. Etiology. Pathogenesis. Classification. Morphology. Complications and outcomes.
4. Interstitial pneumonia. Definition. Etiology. Pathogenesis. Morphology. Complications and outcomes.
5. Acute respiratory distress syndrome (ARDS). Definition. Etiology. Pathogenesis. Morphology. Outcomes.
6. Aspiration pneumonia. Definition. Etiology. Pathogenesis. Morphology. Complications and outcomes.
7. Hypostatic pneumonia. Definition. Etiology. Pathogenesis. Morphology. Complications and outcomes.

COURSE OF THE SESSION

Acute bronchitis presents as a sudden **inflammation of the bronchi**, which can occur either as an **independent condition** or as a **symptom** of various underlying diseases such as **pneumonia** or acute **uremic bronchitis** associated with renal failure. Viruses, notably respiratory syncytial virus (**RS-virus**), are the primary culprits behind bronchitis, although exposure to **chemical agents** like cigarette smoke, sulfur dioxide, chlorine vapor, and nitrogen oxides, as well as **physical agents** such as dry or cold air and radiation, can also trigger it. Additionally, high concentrations of **household or industrial dust** and, less commonly, **bacteria** like Haemophilus influenzae and Streptococcus pneumoniae can contribute to its onset.

Pathologically, acute bronchitis manifests as **hyperemia and swelling of the bronchial mucous membrane**, with the potential for **small hemorrhages** and **ulcerations**. The bronchial passages become congested with mucus, fostering various forms of catarrhal inflammation characterized by the accumulation of serous, mucous, purulent, or mixed exudate, occasionally leading to ulceration. Productive coughing in acute bronchitis can result in the **thickening of bronchial walls** due to infiltration by inflammatory cells.

Within the bronchioles, acute inflammation, known as **bronchiolitis**, can manifest in three primary types:

1. **Primary Bronchiolitis**: This is an uncommon infection of the respiratory tract primarily caused by viruses, particularly respiratory syncytial virus (RSV). It predominantly affects children under the age of 2. While most cases of primary bronchiolitis resolve within a few days, there is a risk of progression to bronchopneumonia in some instances.
2. **Follicular Bronchiolitis**: This form of bronchiolitis is associated with rheumatic diseases and is characterized by the development of lymphoid infiltrates containing germinal centers within the walls of bronchioles. This infiltration leads to a narrowing of the airway lumen.
3. **Bronchiolitis Obliterans**: Characterized by the accumulation of polypoid masses formed from granulation tissue and organizing inflammatory exudate, bronchiolitis obliterans spreads from the alveoli into the bronchi. This type of bronchiolitis can occur in various scenarios, including respiratory syncytial infections, exposure to toxic substances, allergic alveolitis, pulmonary fibrosis, and certain vasculitis conditions.

Pneumonia is characterized by the **acute inflammation of the lung parenchyma beyond the terminal bronchioles**, encompassing structures such as the respiratory bronchiole, alveolar ducts, alveolar sacs, and alveoli.

The terms "**pneumonia**" and "**pneumonitis**" are frequently used interchangeably to denote inflammation of the lungs, while "**consolidation**," indicating solidification, describes the macroscopic and radiologic appearance of the lungs affected by pneumonia.

Pathogenesis involves microorganisms accessing the lungs through one of **four primary routes**:

1. **Inhalation** of microbes present in the air.
2. **Aspiration** of organisms from the nasopharynx or oropharynx.
3. **Hematogenous spread** from a distant focus of infection.
4. **Direct spread** from an adjacent site of infection.

On the basis of the anatomic part of the lung parenchyma involved, pneumonias are traditionally classified into **3 main types**:

1. **Lobar pneumonia** (or croupous)
2. **Bronchopneumonia** (or lobular pneumonia)
3. **Interstitial pneumonia**.

Bacterial pneumonia has **two patterns** of anatomic distribution: lobular bronchopneumonia and lobar (croupous) pneumonia.

Patchy consolidation of the lung is the dominant characteristic of bronchopneumonia, while consolidation of a large portion of a lobe or of an entire lobe defines lobar pneumonia.

These anatomic categorizations may be difficult to apply in individual cases because patterns overlap. The **patchy involvement may become confluent**, producing lobar consolidation. Moreover, the same organisms may produce either pattern depending on **patient susceptibility**.

Most important from the clinical standpoint are **identification of the causative agent** and determination of the extent of disease.

Bronchopneumonia (Lobular) Pneumonia

Bronchopneumonia is characterized by **patchy consolidation of lung tissue**, typically affecting **multiple small areas or lobules**.

It commonly arises from **aspiration** of **oropharyngeal secretions** or **inhalation** of bacteria, including Streptococcus pneumoniae, Haemophilus influenzae, and Staphylococcus aureus.

Bronchopneumonia often presents with symptoms such as **cough, sputum production, fever, and shortness of breath.**

Diagnosis may involve chest X-rays showing **diffuse infiltrates or consolidation in the lower lung fields**.

Treatment usually includes **antibiotics** tailored to the likely causative organisms.

Bronchopneumonia, also known as lobular pneumonia, is characterized by an infection originating in the **terminal bronchioles that spreads into the adjacent alveoli**, leading to the formation of **patchy areas** of lung consolidation.

This condition commonly occurs as a terminal event in **chronic debilitating diseases** and frequently arises as a **secondary infection** following viral respiratory illnesses such as influenza and measles.

Etiologically, bronchopneumonia is typically caused by a variety of pathogens including staphylococci, streptococci, pneumococci, Klebsiella

pneumoniae, Haemophilus influenzae, and various gram-negative bacilli such as Pseudomonas and coliform bacteria.

Pathologically, bronchopneumonia is identifiable by gross features such as **patchy areas of red or grey consolidation** affecting one or more lobes, often bilaterally and **predominantly in the lower lung zones** due to the gravitational pooling of secretions.

Upon sectioning, these consolidated lesions appear **dry, granular, firm, and exhibit a red or grey coloration**, typically measuring 3 to 4 centimeters in diameter.

These patches are often **slightly elevated over the surface** and are frequently centered around a bronchiole.

Palpation of the cut surface aids in identifying these patchy areas by their characteristic texture and appearance.

Histologically, bronchopneumonia exhibits the following features:

1. **Acute bronchiolitis** characterized by inflammation of the bronchioles.
2. Presence of **suppurative exudate**, primarily composed of neutrophils, within the **peribronchiolar alveoli**.
3. **Thickening of the alveolar septa** due to congested capillaries and infiltration by leukocytes.
4. **Alveoli less affected** by inflammation may contain edematous fluid.

Complications of bronchopneumonia mirror those of lobar pneumonia; however, complete resolution is less common. Bronchopneumonia often results in some degree of **bronchiolar destruction**, leading to areas of **bronchiolar fibrosis**. Over time, this fibrosis can progress to **bronchiectasis**, a condition characterized by abnormal dilation of the bronchioles

Lobar (Croupous) Pneumonia

Lobar pneumonia affects **entire lobes or large portions thereof**.

It typically results from infection by **bacteria** like Streptococcus pneumoniae, but can also be caused by other pathogens such as Klebsiella pneumoniae.

This type of pneumonia often presents with abrupt onset of **high fever, chills, productive cough, and chest pain**.

On auscultation, there may be characteristic findings such as **bronchial breath sounds and crackles** over the affected lobe.

Lobar pneumonia can **lead to significant consolidation** of lung tissue and may require hospitalization for management with antibiotics and supportive care.

Lobar pneumonia, characterized by its involvement of **entire lobes** or **multiple lobes** of the lungs, presents a significant burden on public health globally.

Etiologically, it is predominantly caused by various bacterial pathogens:

1. **Pneumococcal pneumonia**: Streptococcus pneumoniae, also known as pneumococcus, is the leading cause of lobar pneumonia worldwide. It primarily affects individuals in the community setting, with increased susceptibility among the elderly, young children, and immunocompromised individuals. Pneumococcal vaccination has proven effective in reducing the incidence of this type of pneumonia.

2. **Staphylococcal pneumonia**: Staphylococcus aureus, including methicillin-resistant strains (MRSA), can cause severe and often necrotizing pneumonia. This type of pneumonia may complicate influenza infections or arise as a consequence of hematogenous spread from other sites of infection, such as skin abscesses or infected prosthetic devices.

3. **Streptococcal pneumonia**: While Streptococcus pyogenes (group A streptococcus) is a common cause of pharyngitis and skin infections, it can also lead to lobar pneumonia, particularly in individuals with underlying chronic illnesses or compromised immune systems. Post-influenza pneumonia caused by Streptococcus pneumoniae or Staphylococcus aureus is a notable concern, particularly during influenza outbreaks.

4. **Pneumonia caused by gram-negative aerobic bacteria**: Gram-negative organisms, such as Haemophilus influenzae, Klebsiella pneumoniae, Pseudomonas aeruginosa, Proteus species, and Escherichia coli, are less frequent causes of lobar pneumonia but can lead to severe infections, especially in hospitalized or immunocompromised patients. Klebsiella pneumoniae, for instance, is associated with a distinctive form of **necrotizing pneumonia**, often accompanied by cavitation and abscess formation.

Appropriate antibiotic therapy should be guided by local resistance patterns and patient-specific factors.

Lobar pneumonia, as described by *Rene Theophile Hyacinthe Laënnec*, historically unfolds in **four sequential stages** of the inflammatory response:

1. **Stage of Congestion** (Initial Phase): Characterized by vascular engorgement and leakage of fluid (exudate) into the alveolar spaces, leading to consolidation of lung tissue.

2. **Red Hepatization** (Early Consolidation): During this phase, the lungs appear red and firm due to the accumulation of red blood cells, fibrin, and inflammatory cells within the alveoli, resulting in further consolidation.

3. **Grey Hepatization** (Late Consolidation): In this stage, the red cells disintegrate, and the lung tissue becomes greyish-brown as neutrophils and macrophages continue to infiltrate the alveoli, leading to further consolidation.

4. **Resolution**: In the final stage, resolution occurs as the inflammatory process subsides, and the lung tissue returns to its normal state, with restoration of normal lung architecture and function.

However, with the widespread use of antibiotics and advancements in medical care, these classic stages are less frequently observed in untreated cases of lobar pneumonia.

In lobar pneumonia, characterized by its involvement of part or all of a lobe, or even two lobes, the lower lobes are most commonly affected, often bilaterally. The pathologic changes associated with bacterial infection include the aforementioned stages of inflammatory response, reflecting the progression of the disease within the lungs.

Lobar pneumonia: congestion

- Typically lasts for **1 to 2 days**.
- Grossly the lung appears **heavy**, **boggy**, and exhibits a **reddish hue**.
- Histological features include:

1. **Vascular Engorgement**: The pulmonary vasculature becomes congested with blood, leading to dilation and engorgement of capillaries and venules within the affected lung tissue. This increased blood flow contributes to the characteristic redness observed grossly.

2. **Intra-alveolar Edema**: Edema fluid accumulates within the alveolar spaces, resulting in the lung tissue becoming heavy and boggy. This fluid contains a limited number of neutrophils, reflecting the early stage of the inflammatory response.

3. **Bacterial Presence**: Bacteria, primarily the causative organism of the infection, can be observed within the alveoli. While bacterial infiltration may vary in intensity, their presence is a hallmark feature of bacterial pneumonia. Gram staining and culture techniques can be utilized to identify and characterize the specific bacterial species involved.

Lobar pneumonia: red hepatization

This phase typically lasts for **2 to 4 days** and is characterized by the affected lobe exhibiting a **liver-like consistency upon cut section**, hence the term "hepatization."

Gross examination reveals that the affected lobe appears **red**, **firm**, and **consolidated**. Upon sectioning, the involved lobe displays an **airless**, **red-pink**, **dry**, granular appearance with a liver-like consistency.

Additionally, this stage is often accompanied by **serous-fibrinous pleuritis**.

Histologically, massive confluent exudation occurs, with **neutrophils, red blood cells, and fibrin filling the alveolar spaces**. This extensive exudate contributes to the consolidation of lung tissue observed grossly.

Lobar pneumonia: grey hepatization

This phase typically spans from **4 to 8 days** in duration.

Gross examination reveals that the affected lobe remains **firm** and **heavy**. Upon sectioning, the cut surface appears **dry**, **granular**, and takes on a **greyish hue** with a **liver-like consistency**. Importantly, the change in color from red to grey typically initiates at the hilum and progresses outward towards the periphery of the lobe.

Additionally, **fibrinous pleurisy** is often prominent, further contributing to the pathological presentation.

Histologically, grey hepatization is characterized by the progressive **disintegration of red blood cells** and the persistence of a **suppurative exudate with fibrin** within the alveolar spaces. This process results in a color change from red to greyish-brown, corresponding to the observed gross appearance.

Lobar pneumonia: resolution

The resolution stage of pneumonia typically **commences around the 8th to 9th day** and spans a **period of 1 to 3 weeks**, although antibiotic therapy can expedite this process, often achieving resolution by the 3rd day. Resolution unfolds progressively, marking the gradual restoration of normal lung function.

Gross examination reveals significant changes within the affected lobe. The previously solid **fibrinous exudate undergoes liquefaction** due to enzymatic action, facilitating the restoration of normal lung aeration. This process of **softening initiates centrally and spreads peripherally**. The cut surface exhibits a **grey-red or dirty brown** appearance, with the presence of **frothy**, **yellow**, **creamy** fluid upon pressure. Moreover, pleural reactions may also exhibit resolution, although in some cases, organization may occur, leading to **fibrous obliteration** of the pleural cavity.

Histologically, the exudate within the alveolar spaces undergoes enzymatic digestion, resulting in the production of granular, semifluid debris. This debris is **subsequently resorbed**, ingested by **macrophages**, expectorated, or **organized by fibroblasts** infiltrating the exudate.

Complications of lobar pneumonia

Since the advent of **antibiotics**, serious complications of lobar pneumonia are **uncommon**.

1. **Organization**. In about 3% of cases, resolution of the exudate does not occur but instead it is organized. There is ingrowth of fibroblasts from the alveolar septa resulting in fibrosed, tough, airless leathery lung tissue. This type of post-pneumonic fibrosis is called **carnification**.

2. **Pleural effusion**. About 5% of treated cases of lobar pneumonia develop inflammation of the pleura with effusion. Sometimes it may undergo organization with **fibrous adhesions** between visceral and parietal pleura.

3. **Empyema**. Less than 1% of treated cases of lobar pneumonia develop **encysted pus** in the pleural cavity termed empyema.

4. **Lung abscess**. A rare complication of lobar pneumonia is formation of lung abscess, especially when there is secondary infection by other organisms.

5. **Metastatic infection**. Occasionally, infection in the lungs and pleural cavity in lobar pneumonia may extend into the pericardium and the heart causing purulent pericarditis, bacterial endocarditis and myocarditis. Rare forms are otitis media, mastoiditis, meningitis, brain abscess and purulent arthritis.

Lung abscess is a localized area of necrosis of lung tissue with suppuration.

1) **Primary** lung abscess that develops in an otherwise **normal lung**. The commonest cause is aspiration of infected material.

2) **Secondary** lung abscess that develops as a **complication** of some other disease of the lung or from another site.

Grossly, abscesses may be of variable size from a few millimeters to large cavities, 5 to 6 cm in diameter. The cavity often contains exudate. An acute lung abscess is initially surrounded by acute pneumonia and has poorly-defined ragged wall. With passage of time, the abscess becomes chronic and develops fibrous wall.

Histologically, the characteristic feature is the destruction of lung parenchyma with suppurative exudate in the lung cavity. The cavity is initially surrounded by acute inflammation in the wall but later there is replacement by exudate of lymphocytes, plasma cells and macrophages.

Etiopathogenesis of lung abscess

1. **Aspiration** of infected foreign material. A number of foreign materials such as food, decaying teeth, gastric contents, severely infected gingivae and teeth, and necrotic tissue from lesions in the mouth, upper respiratory tract or nasopharynx may be aspirated.

2. **Preceding bacterial infection**. Preceding bronchopneumonia in a debilitated patient may develop into lung abscess. Tuberculosis, bronchiectasis and mycotic infections may occasionally result in formation of lung abscess.

3. **Bronchial obstruction**. An abscess may form distal to an obstructed bronchus such as from bronchial tumour or from impacted foreign body.

4. **Septic embolism**. Infected emboli originating from pyemia, thrombophlebitis or from vegetative bacterial endocarditis may be disseminated in the venous circulation and reach the right side of the heart from where they are lodged in the lung and result in multiple abscesses.

5. Miscellaneous: infection in **pulmonary infarcts**; **amoebic** abscesses; **trauma** to the lungs; **direct extension** from a suppurative focus in the mediastinum, esophagus, subphrenic area or spine.

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Pneumocystis carinii pneumonia

Pneumocystis carinii, a protozoan organism ubiquitous in the environment, can lead to pneumonia when inhaled, particularly in neonates and individuals with compromised immune systems. This opportunistic infection is notably prevalent among individuals with **AIDS**, where it may manifest as Pneumocystis carinii pneumonia (**PCP**).

In addition to AIDS patients, other **immunocompromised populations** are also at risk, including those undergoing chemotherapy for organ transplantation or

cancer, individuals with malnutrition, and those with conditions such as agammaglobulinemia.

Microscopic examination reveals the presence of **foamy exudate within the alveolar spaces**. This exudate consists of a mixture of inflammatory cells, proteinaceous material, and Pneumocystis organisms.

Legionella pneumonia

Legionnaire's disease, caused by the gram-negative bacillus Legionella pneumophila, is an epidemic illness predominantly found in aquatic environments. It gained recognition after a high mortality rate was observed among attendees of the American Legion Convention in Philadelphia in July 1976.

This epidemic tends to occur **during the summer months** and is often associated with the spread of Legionella organisms through **contaminated drinking water** or **air-conditioning cooling towers**. Host factors such as immunodeficiency, corticosteroid therapy, advanced age, and smoking are significant predisposing factors to infection.

In terms of morphology, Legionella pneumonia typically presents with **widespread bronchopneumonia** involving multiple lung lobes. Gross examination reveals changes consistent with **extensive lung involvement**, often culminating in consolidation of the entire lung. Additionally, **pleural effusion** is frequently observed as a concurrent finding.

Aspiration pneumonia results from inhaling different agents into the lungs. These substances include **food**, **gastric contents**, **foreign body** and **infected material from oral cavity**.

A number of factors **predispose** to aspiration pneumonia which include: unconsciousness, drunkenness, neurological disorders affecting swallowing, drowning, necrotic oropharyngeal tumours, in premature infants and congenital tracheoesophageal fistula.

Some patients die immediately from asphyxiation or laryngospasm without developing pneumonia.

Pathologic changes **vary depending upon the particulate substance aspirated** but in general right lung is affected more often due to direct path from the main bronchus.

Hypostatic pneumonia is the term used for the collection of **edema fluid** and secretions in the dependent parts of the lungs in severely debilitated, bed-ridden patients. The accumulated fluid in the **basal zone and posterior part of lungs** gets infected by bacteria from the upper respiratory tract and sets in bacterial pneumonia. Hypostatic pneumonia is a common terminal event in the old, feeble, comatose patients.

Lipoid pneumonia

1. **Exogenous lipoid pneumonia**. This is caused by aspiration of a variety of oily materials. These are: inhalation of oily nasal drops, regurgitation of oily

medicines from stomach (e.g. liquid paraffin), administration of oily vitamin preparation to reluctant children or to debilitated old patients.

2. **Endogenous lipoid pneumonia**. Endogenous origin of lipids causing pneumonic consolidation is more common. The sources of origin are tissue breakdown following obstruction to airways e.g. obstruction by bronchogenic cancer, tuberculosis and bronchiectasis.

Fungal infections of lung

These infections in healthy individuals are rarely serious but in **immunosuppressed individuals** may prove fatal.

1. **Histoplasmosis**. The condition may remain asymptomatic or may produce lesions similar to tuberculous nodule.

2. **Coccidiodomycosis**. The infection in human beings is acquired by close contact with infected dogs. The lesions consist of peripheral parenchymal granuloma in the lung.

3. **Cryptococcosis**. The infection occurs from infection by inhalation of pigeon droppings.

4. **Blastomycosis**. It is an uncommon condition which result from inhalation of spores from the ground.

5. **Aspergillosis** is the most common fungal infection of the lung caused by Aspergillus fumigatus. The fungus exists as thin septate hyphae with dichotomous branching and grows best in cool, wet climate. Immunocompromised persons develop more serious manifestations.

6. **Mucormycosis** or phycomycosis. The infection in the lung occurs in a similar way as in aspergillosis. The pulmonary lesions are especially common in patients of diabetic ketoacidosis.

7. **Candidiasis**. Candidiasis or moniliasis caused by Candida albicans is a normal commensal in oral cavity, gut and vagina but attains pathologic form in immunocompromised host. Angio-invasive growth of the organism may occur in the airways.

Interstitial pneumonia, also known as **atypical** or **viral pneumonia**, primarily affects the **interstitial spaces of the lung parenchyma** rather than the alveolar spaces.

It can be caused by **viruses** such as influenza, respiratory syncytial virus (RSV), adenovirus, or human metapneumovirus.

Unlike lobar and bronchopneumonia, interstitial pneumonia often presents with **milder symptoms, such as dry cough, low-grade fever, and fatigue**.

However, it can progress to severe **respiratory failure**, especially in vulnerable populations such as the elderly or immunocompromised individuals.

Diagnosis may involve chest imaging showing **diffuse interstitial infiltrates**.

Viral and mycoplasmal pneumonia is characterized by patchy inflammatory changes, largely confined to interstitial tissue of the lungs, without any alveolar exudate.

Other terms used for these respiratory tract infections are **interstitial pneumonitis**, reflecting the interstitial location of the inflammation, and 'primary atypical pneumonia', atypicality being the absence of alveolar exudate commonly present in other pneumonias.

Etiology. Interstitial pneumonitis is caused by a wide variety of agents, he most common being **respiratory syncytial virus** (RSV). Others are **Mycoplasma pneumoniae** and many **viruses** such as influenza and parainfluenza viruses, adenoviruses, rhinoviruses, coxsackieviruses and CMV.

In most cases, the infection remains **confined to the upper respiratory tract** presenting as common cold. Occasionally, it may extend lower down to involve the interstitium of the lungs. The circumstances favoring such extension of infection are malnutrition, chronic debilitating diseases and alcoholism.

Irrespective of the etiologic agent, the **pathologic changes** are similar in all cases. Grossly, depending upon the severity of infection, the involvement may be patchy to massive and widespread consolidation of one or both the lungs. The **lungs are heavy and congested**. Sectioned surface of the lung exudes small amount of bloody fluid. The pleural reaction is usually infrequent and mild.

The hallmark of viral pneumonias is the **interstitial inflammatory reaction**.

1) **Interstitial inflammation**: There is thickening of alveolar walls due to congestion, edema and inflammatory infiltrate comprised by lymphocytes, macrophages and some plasma cells.

2) **Necrotizing bronchiolitis**: This is characterized by foci of necrosis of the bronchiolar epithelium.

3) **Reactive changes**: The lining epithelial cells of the bronchioles and alveoli proliferate in the presence of virus and may form multinucleate giant cells and syncytia in the bronchiolar and alveolar walls.

4) **Alveolar changes**: In severe cases, the alveolar lumina may contain edema fluid, fibrin, scanty inflammatory exudate and coating of alveolar walls by pink, hyaline membrane similar to the one seen in respiratory distress syndrome. Alveolar changes are prominent if bacterial infection supervenes.

INFORMATIONAL AND METHODOLOGICAL PART

MAIN LITERATURE

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NORMATIVE LEGAL ACTS AND DOCUMENTS

1. Order of the Ministry of Health of the Republic of Belarus No. 111 of 06/01/1993 "On further improvement of the pathological service of the Republic of Belarus"

Access mode: http://patan.by/menyu/administrativnyie-proczeduryi.html - The date fccess: 20.03.2022.

ELECTRONIC DATABASES

1. Physician's consultant. Electronic Medical Library = Consultant of the doctor. Electronic medical library [Electronic resource] / Publishing group "GEOTAR-Media", LLC "IPUZ". – Access mode: http://www.rosmedlib.ru/ . – Access date: 03/15/2022.

2. Springer Link [Electronic resource] / Springer International Publishing AG. – Access mode: https:link.springer.com. – Date of access: 03/15/2022.