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MODERN CLINICAL AND EPIDEMIOLOGICAL CHARACTERISTICS OF VIRAL AND BACTERIAL INFECTIONS

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Abstract. This review aimed to summarize the literature data regarding the pathomorphology of lung lesions in COVID-19 and compare it with lung lesions in bacterial pneumonia and pneumonia caused by influenza virus. The analysis of scientific literature containing studies of domestic and foreign authors of different years related to morphology and anatomical pathology of lung injury was carried out. Special attention was paid to the data devoted to COVID-19 obtained between 2019 and 2021. Based on the study, the main aspects of lung lesions were identified and grouped into blocks depending on the etiology of the process. The review collects and summarizes information on etiology, pathogenesis and stages of disease development, outcomes and morphological picture during the autopsy of patients with bacterial pneumonia, influenza pneumonia and COVID-19 pneumonia. The common features and differences in the course, outcomes and typical morphological findings, most characteristics for each of the diseases were presented in the table. There is a great similarity of morphological findings in influenza pneumonia and COVID-19 pneumonia despite the background of the difference in their epidemiology. Most Uzbek and foreign authors agree that a key factor in the pathogenesis of the development of COVID-19.

Keywords: inflammation, thromboembolism, microembolism, interstitium, alveoli.

Bacterial pneumonia. Sources and pathogens. Nearly all pathogenic or opportunistic microorganisms that have entered the lower respiratory tract can become causative agents of bacterial pneumonia. Traditionally, bacterial agents of pneumonia are divided into “typical” and “atypical.” Typical pathogens include Streptococcus pneumoniae, Haemophilus influenzae, Staphylococcus aureus, group A streptococci, Moraxella catarrhalis, and anaerobic and aerobic gram-negative bacteria. According to data from international and Uzbek authors, obtained from the PubMed database and online archives of journals indexed by Scopus and the Higher Attestation Commission, S. pneumoniae is the most common cause of community-acquired pneumonia (CAP), which accounts for 30%–50% of the cases. The proportion of mixed infections usually does not exceed 10%; however, in some studies, this indicator reaches 27%, and 54% in patients aged >60 years [1–4].

The second most common causative agent of CAP as a monoflora is M. pneumoniae (11% and 22% of clinically and radiologically confirmed cases, respectively). Other bacteria are less significant in terms of pneumonia pathogens [6]. In most cases, the source of CAP is a sick person or an asymptomatic carrier of an infectious agent. Some pathogens, such as Legionella and Chlamydia, can be transmitted to humans through contaminated air-conditioning systems or from birds and certain animals [1]. For hospital-acquired pneumonia, the most typical causative agent is the combination of S. aureus (methicillin-resistant S. aureus) with Pseudomonas aeruginosa, Acinetobacter baumannii, and Escherichia coli. S. aureus and Klebsiella pneumoniae were identified most often as etiological factors in purulent destructive pneumonia [5]. Mechanism of damage and target tissue of the pathogen (e.g.,

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pneumococcal CAP as the most common). The development of pneumonia is associated with a disruption of the delicate balance between the microorganisms in the lower respiratory tract and local and general immunity. Disorders of the systemic immune response, mucociliary clearance, cough reflex, and drainage function of the bronchi are crucial to o the pathogenesis of pneumonia [7]. Pathomorphological characteristics. Clinical and morphological analyses of changes in the lungs of patients who died from CAP or hospital-acquired pneumonia in Uzbekistan and in other countries demonstrated similar patterns [5]. CAP is characterized by a more pronounced inflammatory reaction (in terms of the incidence of infiltrates and time of formation), and areas with alveolar edema and hemorrhages were adjacent to the foci of inflammation. Areas of organization were identified more often. Interstitial edema was rare and moderate. In hospital-acquired pneumonia, alveolar edema (which was of a total or subtotal nature) was predominant, as well as infiltration of interalveolar septa by segmented leukocytes (beyond the foci of inflammation. Mechanism of damage and target tissue of the pathogen in COVID-19.[8] The pathogen is characterized by the presence of a specific receptor related to the receptor for angiotensin-converting enzyme type 2 (ACE2). ACE2 receptors are expressed in the epithelium of the respiratory tract, pneumocytes, alveolar monocytes, vascular endothelium, epithelium of the gastrointestinal tract, urinary tract, macrophages, and other cells of organs and tissues, including the myocardium and some parts of the central nervous system.[9] ACE2 receptors serve as functional receptors for SARS-CoV-2 and provide CoV access to the epithelial cells of the upper respiratory tract, where it is capable of the most active replication . Yuki suggested that CoV spread through the infected lung epithelium into the pulmonary vascular endothelium, which also expresses the ACE2 receptor. Other authors considered the gender difference in the expression of ACE2 receptor as one of the factors of higher mortality in male patients (105.2 ± 9.1 RFU/μL/h2 in men versus 84.7 ± 6.9 RFU/μL/h in women, $p = 0.05$) . Zayratyants [10] compared the course and progression of COVID-19 with SARS and noted the similarity of the viral replication in the lower respiratory tract with the development of secondary viremia, immune disorders, and hypoxia. This situation leads to damage in many target organs such as the heart, kidneys, and gastrointestinal tract, which cells express the ACE2 receptor. Thus, clinical deterioration is observed at week 2 from disease onset. However, the author considered the development of microangiopathy in the form of destructive productive thrombovasculitis and hypercoagulable syndrome, as well as damage to the organs of the immune system, as the key difference between the new CoV infection and SARS. Moreover, a persistent inflammatory response acts as a trigger of the coagulation cascade, which, in combination with direct viral damage to the endothelium of the microvasculature, aggravates the severity of the clinical course and worsens the prognosis.

Conclusion

When comparing the autopsy results of patients with COVID-19 and the data on pneumonia of a different etiology, a high morphological similarity with viral pneumonia in influenza was revealed. A differential characteristic of SARS-CoV-2 is the presence of a specific pathway for entering cells through a tissue receptor that has an affinity for ACE2.

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