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Review Article

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Etiopathogenesis and Clinical Picture of Acute Lung Abscesses

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ABSTRACT

Treatment of purulent diseases of the lungs and pleura invariably remains an urgent problem. This pathology, painful for the patient, time-consuming for the doctor and expensive for the medical institution, combines intoxication due to a purulent-destructive process and impaired respiratory function. The occurrence of infectious destruction of the lungs and pleura is facilitated by immunodeficiency and immunosuppression, which are characteristic of patients suffering from haematological diseases, increased tendency to thrombosis, diabetes mellitus, alcoholism, drug addiction, HIV infection, who have undergone extensive surgical interventions, organ and tissue transplantation. Despite the widest use of antibacterial drugs, it is not possible to avoid purulent-destructive and gangrenous complications of pneumonia. This review scientific article is devoted to the problems of acute lung abscesses, namely the aetiology, pathogenesis and clinical picture of this disease.

Keywords: *acute lung abscesses, aetiology, pathogenesis, clinical picture*

The main pathogenetic factors that provoke the development of acute lung abscesses are already known in the form of a triad of interrelated and mutually aggravating links that determine the choice of therapeutic measures: these are an acute infectious inflammatory process in the pulmonary parenchyma, impaired bronchial permeability, and impaired blood flow leading to necrosis of lung tissue [1].

The presence of an acute infectious inflammatory process in the pulmonary parenchyma is considered the first step in the occurrence of purulent destruction of this organ. Most clinicians agree that non-spore-forming anaerobic flora plays a leading role in the occurrence of acute lung abscesses. Although such information was reflected in scientific publications in the middle of the last century, they were deprived of close attention and were cited only as a statement of fact and the results of the study. The frequency of registration of facultative anaerobes in patients with acute lung abscesses ranged from 42.9% to 44.8%. Conclusions were made about the insignificant role of fusospirillosis symbiotic infection. It was considered a secondary infection, which only aggra-

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vated the purulent inflammatory process in the lungs [2-6]

The reason for this conclusion was the high polymorphism of the microflora, which was revealed by the results of studies in patients with lung abscesses. Supporters of the theory of polymorphism of microflora in acute lung abscesses were S.N. Spasokukotsky and M.S. Grigoriev [7]

Meanwhile, information appeared about the importance of a certain type of microorganism in the occurrence of acute lung abscesses. As a result of the work carried out, several foreign researchers have obtained evidence of the high role of gram-negative bacteria in the occurrence of acute lung abscesses [8-16].

Only at the end of the 20th century, thanks to the improvement of the methods of modern clinical bacteriology, including the development and implementation of an arsenal of studies based on anaerobic techniques, did scientists allow scientists to establish the leading role of non-clostridial anaerobes in the development of acute lung abscesses. The same results were obtained by scientists from Uzbekistan, in particular, V.V. Vakhidov, Sh.I. Karimov, E.I. Islambekov, N.F. Krotov and others back in the 80s of the last centuries.

According to Rolston K.V. and Nesher L., about 70% of S. aureus isolates in patients with lung abscesses were resistant to methicillin, and about 40-60% of VGS isolates were not susceptible to penicillins. This was due to the large number of gram-negative bacteria, such as E. coli, which is also relatively common in this environment. Carbapenem-resistant Enterobacteriaceae were less common, but when they were present, there were usually problems with the choice of methods of exposure. Less common were such pathogens as P. aeruginosa, Stenotrophomonas maltophilia, and Acinetobacter spp. [17]

C.T. Stock et al. [18] found that in more than 90% of cases, polymicrobial bacteria can be found in the focus of lung abscess destruction. Of the anaerobic bacteria in lung abscesses, Gram-negative isolates of Bacteroides Fragilis, Fusobacterium Capsulatum and Necrophorum predominate. Gram-positive anaerobic peptostreptostreptococcus and microaerophilic streptococci.

Many authors present publications with different results on the significant role of microflora species in lung abscesses. Staphylococcus aureus is the predominant isolates in lung abscesses, including methicillin-resistant Staphylococcus aureus, Streptococcus pyogenes [19], Klebsiella [20], Pseudomonas aeruginosa, Haemophilus influenzae type B, Acinetobacter spp, Escherichia Coli, and Legionella [21].

Anaerobic bacteria have been the most dominant type of bacteria in lung abscesses for decades. A. Nicolini et al. [22] showed that these were mainly streptococci (Streptococcus pneumonia serotype and Streptococcus anginosus complex).

According to N. Takayanagi et al. [23], At the turn of the 21st century, the most isolated bacterium for lung abscess, especially in Taiwan, was Klebsiella's pneumonia. In this regard, recommendations have been created for the use of special antibiotic therapy for this type of bacteria.

Studies by O. Yildiz and M. Doganay [24] have proved that the etiological pathogen in acute lung abscess can also be Mycobacterium spp., Aspergillus, Cryptococcus, Histoplasma, Blastomyces, Coccidoides, Entamoeba histolytica, and Paragominus westermani. Actinomycoses and Nocardia are known as important etiological pathogens of acute lung abscesses and require longer (6 months) administration of antibiotics.

According to I. Brook [25] and Patradoon-Ho P. [26], Staphylococcus aureus is the most common isolated etiological pathogen of lung abscess in children. This is because the lung tissue itself can serve as a good nutrient substrate for staphylococcus. The latter, in turn, can produce the enzyme lecithinase, which creates a nutrient medium for microorganisms. The growth of staphylococci in such conditions occurs due to the utilisation of lipoproteins, fatty acids and carbohydrates - the main components of lung surfactants. The production of highly toxic microbes by staphylococci was proved by D.A. Egorkina [27]. It has been proven that staphylococci can produce strictly defined toxins and enzymes and have practically no differences in cytotoxic action.

Pseudomonas aeruginosa, also producing enzymes such as hemolysin, lecithinase and phosphatase, uses the protein fraction of surfactant. At the same time, by destroying the superficial part of the surfactant of the lung, Pseudomonas aeruginosa can cause haemorrhages, atelectasis, and destruction of the structural elements of the lung tissue.

The topical location of lung abscesses is characterised by their location in the apical segments of the lower lobe, to a greater extent than the left lung. This is due to the gravitational dependence of the accumulation of sputum in the bronchial tract. In the second place in terms of frequency, the localisation of lung abscesses can be in the lateral part of the posterior segment of the right upper lobe (the so-called axillary subsegments) and the middle

lobe in the case of aspiration of vomit in a horizontal position of the patient, which is usually found in alcoholic intoxication. Nevertheless, in 75% of cases, a lung abscess is localised in the posterior segment of the right upper lobe or the apical part of the lower lobe of both lungs [28].

Lung abscesses, the etiological causes of which were associated with infection of the oropharyngeal region, are often also located in the posterior segments of the lungs. Clinical practice has not revealed any patterns for the hematologic spread of lung abscesses.

Initial hypersecretion of aspiration is usually localised in the distal areas of the bronchi. Accumulation of sputum in these areas provokes the development of local pneumonitis [29, 30]. In the next 24-48 hours, necrotic tissues accumulate in the focus of stagnation and the area of inflammation increases. Invasive bacterial toxins, vasculitis, venous thrombosis, and proteolytic enzymes from neutrophil granulocytes form a colliquation necrotic focus [31].

Subsequently, in cases of lesions of the visceral pleura, there may be a variant of the development of pleural complications in the form of pyopneumothorax or pleural empyema. In the case of adequate antibiotic therapy and with a satisfactory immunological status of the patient, a chronic inflammatory reaction will limit the process. In case of inadequate or delayed antibiotic therapy or poor general condition of the patient, sepsis may occur. If there is a connection with the bronchus, necrotising detritus will empty the abscess cavity, and radiographic signs of the level of air fluid will occur. In case of a favourable outcome, the necrotic tissue will be eliminated by lysis and phagocytosis, and the abscess cavity will be filled with granulation tissue and obliterated. In case of an unfavourable outcome, the infectious process can spread to the entire lung tissue, or various fistulas can be organised. The completion of the chronic process is possible by reabsorption of the abscess contents with the formation of fibrosis and calcification of the cavity.

Early signs and symptoms of a lung abscess may not be different from pneumonia and include fever with chills, cough, night sweats, dyspnea, weight loss and fatigue, chest pain, and sometimes anaemia. In the beginning, the cough is non-productive, but when there is communication with the bronchus, a productive cough is a typical sign of a lung abscess [32, 33].

The cough remains productive and is sometimes followed by hemoptysis.

Differential diagnosis involves the exclusion of tuberculosis and mycosis of the lungs, but it is rare to see an X-ray sign of horizontal fluid levels. Pulmonary cystic lesions, such as intrapulmonary bronchial cysts, sequestration, or secondary infected emphysematous bullae, may be difficult to distinguish, but the location of the lesion and clinical signs may suggest an appropriate diagnosis. Localised pleural empyema can be distinguished by computed tomography or ultrasound [34].

Diagnostic features of bronchial carcinomas, such as squamous or small cell carcinoma, usually have a thicker and irregular wall compared to an infectious lung abscess [35]. The absence of fever, purulent sputum, and leukocytosis may indicate carcinoma rather than an infectious disease [36]. An X-ray sign of a horizontal fluid level can also be seen in a parasitic lung cyst [37, 38].

Diagnostic bronchoscopy is part of the diagnostic protocol for taking material for microbiological examination and confirmation of the intrabronchial cause of the abscess - a tumour or a foreign body. Sputum examination is useful for the detection of microbiological agents or confirmation of bronchial carcinoma [39].

Thus, despite the long history of studying the etiology and pathogenesis of acute lung abscesses, many aspects of this problem remain unresolved.

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O'PKANING O'TKIR XO'PPOZLARINING ETIOPATOGENEZI VA KLINIK KO'RINISHI

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ABSTRAKT

O'pka va plevra yiringli kasalliklarini davolash doimo dolzarb muammo bo'lib qolmoqda. Bemor uchun og'riqli, shifokor uchun vaqt talab qiladigan va tibbiy muassasa uchun qimmat bo'lgan ushbu patologiya, yiringli vayron qiluvchi jarayon va nafas olish funktsiyasining buzilishi tufayli intoksikatsiyani birlashtiradi. O'pka va plevra qismining yuqumli yo'q bo'lishining yuzaga kelishi gematologik kasalliklar bilan og'rigan bemorlarga xos immun tanqisligi va immunosupressiya yordam beradi, bu keng jarrohlik aralashuvlari, organ va to'qima transplantatsiyasi bo'lgan tromboz, qandli diabet, alkogolizm, giyohvandlik, OITS infeksiyasi bilan og'rigan bemorlarga xos bo'ladi. Antibakterial preparatlarni keng qo'llashiga qaramay, pnevmoniyaning yiringli vayron qiluvchi va gangrenöz asoratlarini oldini olish mumkin emas. Ushbu tarjima ilmiy maqola o'pkaning o'tkir xo'ppozlari muammolariga, ya'ni ushbu kasallikning etiologiyasi, patogenezi va klinik ko'rinishiga bag'ishlangan.

Kalit so'zlar: o'pkaning o'tkir absessiyalari, etiologiyasi, patogenezi, klinik ko'rinishi

ЭТИОПАТОГЕНЕЗ И КЛИНИЧЕСКАЯ КАРТИНА ОСТРЫХ АБСЦЕССОВ ЛЕГКИХ

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АБСТРАКТ

Лечение гнойных заболеваний легких и плевры неизменно остается актуальной проблемой. Эта патология, мучительная для пациента, трудоемкая для врача и дорогостоящая для лечебного учреждения, сочетает в себе интоксикацию вследствие гнойно-деструктивного процесса и нарушение дыхательной функции. Возникновению инфекционных деструкций легких и плевры способствует иммунодефицит и иммуносупрессия, характерные для пациентов, страдающих гематологическими заболеваниями, повышенной склонностью к тромбообразованию, сахарным диабетом, алкоголизмом, наркоманией, ВИЧ-инфекцией, подвергшихся обширным хирургическим вмешательствам, трансплантации органов и тканей. Несмотря на широчайшее использование антибактериальных препаратов, не удается избежать гнойно-деструктивных и гангренозных осложнений пневмоний. Данная обзорная научная статья посвящена проблемам острых абсцессов легких, а именно этиологии, патогенезу и клинической картине данного заболевания.

Ключевые слова: острые абсцессы легких, этиология, патогенез, клиническая картина