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Features of the clinical presentation and course of community-acquired pneumonia against the background of type 2 diabetes mellitus

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ABSTRACT

Community-acquired pneumonia remains the leading infectious cause of death around the world. Many factors influence the prognosis and outcome of this disease. Compared with healthy individuals, patients with diabetes mellitus are at increased risk of respiratory tract infections, such as community-acquired pneumonia. Diabetes mellitus contributes to the development of pulmonary thrombotic microangiopathy, changing the functional state of the lungs.

In numerous studies involving patients with diabetes mellitus, data on the state of the lungs were obtained by instrumental tests, such as spirometry, ventilation / perfusion scintigraphy, perfusion computed tomography, and diffusing capacity of the lungs for carbon monoxide. In patients with community-acquired pneumonia, diabetes mellitus causes vague clinical symptoms, leads to a severe course of the disease, and contributes to development of complications. Diagnosing the functional state of the lungs in patients with community-acquired pneumonia against the background of diabetes mellitus has not been studied.

Keywords: community-acquired pneumonia, diabetes mellitus

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Особенности клиники и течения внебольничной пневмонии на фоне сахарного диабета 2-го типа

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РЕЗЮМЕ

Внебольничная пневмония остается ведущей инфекционной причиной смерти в мире. Многие факторы влияют на прогноз и исход данного заболевания. По сравнению со здоровыми лицами пациенты с сахарным диабетом подвергаются повышенному риску инфекций дыхательных путей, таких как внебольничная

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пневмония. Сахарный диабет способствует развитию микроангиопатии легких, изменяя функциональное состояние данного органа.

В многочисленных исследованиях пациентов с сахарным диабетом получены данные о состоянии легких инструментальными методами, такими как спирография, вентиляционная сцинтиграфия, перфузионная компьютерная томография и метод определения диффузионной способности легких по монооксиду углерода. У пациентов с внебольничной пневмонией сахарный диабет вызывает стертую клиническую симптоматику, приводит к тяжелому течению внебольничной пневмонии и способствует развитию осложнений. Диагностика же функционального состояния легких у пациентов с внебольничной пневмонией на фоне сахарного диабета не изучена.

Ключевые слова: внебольничная пневмония, сахарный диабет

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INTRODUCTION

Community-acquired pneumonia is one of the key causes of emergency hospitalization in inpatient facilities of internal medicine and a potentially lethal disease [1, 2]. The World Health Organization (WHO) reports that lower respiratory tract infections, including pneumonia, are still the deadliest infectious diseases. In 2015, 3.2 million people died of these diseases in the world. The severity and outcome of community-acquired pneumonia also depend on such comorbidities as chronic obstructive pulmonary disease (COPD), heart failure, cerebrovascular and kidney diseases, metabolic syndrome, and diabetes mellitus (DM) [2–9].

Many scientific publications thoroughly describe combinations of cardiovascular pathologies and DM as mutually exacerbating conditions. Only few studies analyze the course of community-acquired pneumonia against the background of DM. Most existing publications show data on the incidence of community-acquired pneumonia and analyze the risk of a lethal outcome due to community-acquired pneumonia and concurrent DM.

Currently, DM is one of the most common chronic diseases in the world. In 2016, WHO called it one of the most dangerous noncommunicable epidemics of the XXI century along with cardiovascular diseases, COPD, and cancers [10]. The number of DM patients increases at a rate exceeding the forecasts of

experts from the International Diabetes Federation. The research findings from NATION, a large epidemiological study conducted in 2013–2015 and aimed at detecting type 2 diabetes mellitus (T2DM) among the Russian population, show that the prevalence of this disease is 5.4% (about 6.5 million people). DM is characterized by macrovascular and microvascular complications. The latter include complications affecting the kidneys, eye retina, and nervous system [12, 13].

Now there is an increasing amount of data confirming that the lungs are one of the target organs for diabetic microangiopathy. An extensive network of pulmonary capillaries is involved in respiratory metabolism through alveoli that form a single membrane. Hyperglycemia causes structural changes in the pulmonary capillary walls, and these changes have a negative impact on the blood-air barrier, lead to microangiopathy in the DM-affected lungs, and change the functional state of the entire organ [14]. It is proved that DM patients are characterized by pulmonary function impairment, regardless of the duration of the disease [15, 16]. W.A. Davis et al. showed that in the group of DM patients without pulmonary diseases and with 4% reduction in the lung function parameters, the all-cause mortality rate increased by 12% [17].

In addition, some studies show that blood glucose-lowering drugs ensure a 10% increase in lung function parameters (forced expiratory volume in

one second (FEV₁), vital capacity (VC)) compared with the baseline values in T2DM patients [18].

A longitudinal, observational study conducted in the USA in 2005 including 1,443 male patients aged 21–88 years analyzed lung function parameters and blood glucose values and detected statistically low FEV₁ and VC in patients several years before the emergence of DM compared with same aged patients without DM [19].

N. Guvener et al., using the gaseous diffusion method, demonstrated decreased alveolar capillary membrane permeability in DM patients compared with the control group ($p = 0.037$) [20]. At the same time, some studies provide exactly opposite data. In the study by K. Ozşahin, the gaseous diffusion method did not show differences in alveolar capillary membrane permeability between DM patients and healthy people, while it was significantly reduced in the group of DM patients ($p = 0.01$) when ventilation / perfusion scintigraphy was performed [21]. These findings confirm that impaired pulmonary function in DM patients causes subclinical changes in the lung structure that may aggravate such an acute infectious process as community-acquired pneumonia and promote complications.

A review studying patients who are susceptible to invasive pneumococcal infections shows that the risk of such infections, including community-acquired pneumonia, increases in case of DM [22, 23]. Most authors believe that DM contributes to an increase in hospital stay and acts as a predictor of higher mortality in patients with community-acquired pneumonia [24, 25]. For instance, the studies conducted by J.B. Kornum et al. in Denmark in 2007–2008 showed that patients with HbA1c $\geq 9\%$ were statistically often characterized by more severe community-acquired pneumonia and a 60% increase in the risk of hospitalization in relation to pneumonia [26, 27].

The findings of CAPNETZ, a multicenter, prospective cohort study, show that in 2007–2014, the mortality rate within the first month of hospitalization for community-acquired pneumonia amounted to 12.1% in DM patients vs. 3.8% in patients without DM ($p = 0.001$) [28]. The study carried out in Japan in 2005–2011 revealed that the mortality rate within the first month among DM patients hospitalized for community-acquired pneumonia was significantly associated with the degree of hyperglycemia

at the moment of hospitalization ($p < 0.0001$) [29]. S. Yende et al. demonstrated that hospitalization for community-acquired pneumonia was a significant risk factor for a lethal outcome within the first year after inpatient treatment for DM patients (hazard ratio (HR) = 1.87) [23]. M. Falcone et al. detected significantly higher annual mortality rates in DM patients hospitalized for community-acquired pneumonia vs. patients hospitalized for other reasons (30.3 vs. 16.8%, $p < 0.001$) [9].

The risk factors for mortality in patients with community-acquired pneumonia against the background of T2DM are bacteremia, septic shock, and comorbidities [30, 31]. It is proved that age has a strong impact on mortality rates in patients with community-acquired pneumonia and DM.

The findings from the NHANES III study (USA) and the analysis of 3,770 death certificates of people aged 65+ years allow to conclude that every year of life increases the risk of death due to community-acquired pneumonia by 16%. It was shown that an increased risk of death due to community-acquired pneumonia is typical of not only DM patients (34.1 per 10,000 person – years), but also of patients with impaired glucose tolerance (16.9 per 10,000 person – years) [23, 32].

The findings from a prospective cohort study (6 years) conducted in Finland (2014) showed that DM and postprandial hyperglycemia newly diagnosed in non-diabetic patients with community-acquired pneumonia were associated with a higher risk of late mortality within several years after experienced community-acquired pneumonia. The mortality rate at the end of the follow-up amounted to 54, 37, and 10% in DM patients, non-diabetic patients with diagnosed postprandial hyperglycemia, and patients without DM and postprandial hyperglycemia, respectively ($p < 0.001$). In addition, hyperglycemia has a prognostic value as a severity criterion for patients with severe community-acquired pneumonia [33]. Only single publications show practically similar mortality data. However, in the authors' opinion, the reason for that was that the studied patients were older and experienced a more severe condition at the moment of admission, so DM had an insignificant impact on their mortality rates [12, 34, 35].

At the same time, many authors are sure that higher mortality rates are typical of DM patients after inpatient treatment of community-acquired pneumo-

nia vs. patients hospitalized for noncommunicable pathologies [23, 34]. Few publications describe the clinical aspects of community-acquired pneumonia with concomitant DM, and these findings are contradictory.

The clinical features of community-acquired pneumonia are different in DM patients. Patients with community-acquired pneumonia against the background of DM experience less pronounced clinical symptoms, such as cough, chills, and acute onset of the disease. This may be the reason why patients with community-acquired pneumonia and DM score less on the CURB-65 and PSI / PORT scales at the moment of admission, however, the duration of hospital stay for patients with community-acquired pneumonia with concomitant DM is longer than for patients without DM [34, 36].

Fever, cough, shortness of breath, and tachycardia, i.e. the classical symptoms of community-acquired pneumonia, disappear, become silent or are absent at all in DM patients. These symptoms are often accompanied by radiographically detected multilobar lung infiltrates, pleural effusion, and empyema. M.A.Saibal reported significant differences ($p < 0.001$) following X-ray examinations. DM patients with community-acquired pneumonia are more often characterized by multisegmental pulmonary lesions than patients without DM [37]. Some other studies show no significant difference in the pulmonary infiltration area in community-acquired pneumonia patients with and without DM [25, 36].

Low symptom intensity makes the disease difficult to diagnose. Extrapulmonary manifestations, such as altered mental state, mental block, hypotonia, and tachypnea, are often prevailing or the only symptoms detected in patients with community-acquired pneumonia with DM during a physical examination. DM patients hospitalized for community-acquired pneumonia more often suffer from metabolic disorders and cardiovascular events [12, 37].

The authors of this study have not found literature data on the features of physical examinations in patients with community-acquired pneumonia and concomitant DM. Community-acquired pneumonia often cannot be diagnosed based only on clinical symptoms and laboratory findings, especially in senior patients and patients with comorbidities, such as DM [38–40]. Community-acquired pneumonia is definitely diagnosed only if infiltration signs are

detected using diagnostic radiology methods. At the same time, these instrumental procedures allow to obtain information only on structural changes in the lungs [38, 41, 42].

In some cases, in patients with community-acquired pneumonia and comorbidities, including DM, instrumental methods are not always capable of determining the contribution of pneumonia to the severity of patients' condition. To determine the functional state of the lungs, several methods are used, such as inspiratory and expiratory computed tomography and ventilation / perfusion scintigraphy [43, 21].

In 2006, K.Özşahin conducted a study on alveolar capillary membrane permeability in DM patients and healthy individuals using two methods: gaseous diffusion and ventilation / perfusion scintigraphy. The first method did not detect any difference between the groups. Ventilation / perfusion scintigraphy revealed a significant increase in the half-life of a radiopharmaceutical in DM patients vs. the control group ($T_{1/2} = 112.7\%$ and $T_{1/2} = 84.6\%$, $p = 0.01$) [21]. It was demonstrated that the walls of pulmonary arterioles became thicker due to the development of chronic inflammation [44], an increase in collagen and elastin levels, and fibroblast proliferation [45]. Therefore, ventilation / perfusion scintigraphy is a more sensitive method to determine the alveolar capillary membrane permeability.

In the study by K. Kuziemy et al., carried out in Poland in 2011, perfusion computed tomography detected upward quantitative changes in the blood flow volume, blood filling, and vascular wall permeability parameters in DM patients compared with the control group of healthy individuals ($p = 0.01$) [46]. The authors have not found any data on the sensitivity and specificity of diagnostic radiology methods in patients with community-acquired pneumonia and T2DM in the available literature.

CONCLUSION

Therefore, DM is characterized by lung damage accompanied by functional disorders in the lungs. Changes in the pulmonary microvasculature are more diffused than in the renal arteries or vessels in the eye. Therefore, they may remain compensated and have no clinical manifestations for a long time. However, there is much evidence of subclinical manifestations of diabetic lung associated with a

high risk of a lethal outcome in community-acquired pneumonia.

For instance, data on the information value of ventilation / perfusion scintigraphy and perfusion computed tomography in DM patients are available, but the capacities of these methods in patients with community-acquired pneumonia and DM have not been completely unveiled yet. Therefore, the sensitivity and specificity of such methods as inspiratory and expiratory computed tomography and ventilation / perfusion scintigraphy in patients with community-acquired pneumonia and T2DM should be further explored.

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