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**Resume.**The main task of clinicians when a patient with COPD seeks help is to distinguish an exacerbation of this disease from COVID-19, since there is a similarity in clinical symptoms: cough, fever, intoxication and shortness of breath.

The need for further clinical studies on the problem of comorbidity of COPD and COVID-19 is shown, which will allow a detailed study of the mechanisms of mutual aggravation of associated pathology, to clarify the effect of SARS-CoV-2 on the respiratory system and the course of COPD taking into account the phenotype of the disease, to determine effective treatment methods and improve the prognosis of patients with COPD who have had the new coronavirus infection COVID-19.

# Key words: comorbidity, COVID-19, chronic obstructive pulmonary disease.

**Relevance.** A new acute respiratory infection caused by the Betacoronavirus SARS-CoV-2 coronavirus (severe acute respiratory syndrome-related coronavirus 2) was first identified in late 2019 in Wuhan, China. The virus is highly contagious and continues to spread rapidly around the world. On March 11, 2020, the World Health Organization (WHO) declared a pandemic caused by this infection. As of February 15, 2021, the number of confirmed cases of COVID-19 (Coronavirus Disease-2019) in the world was 108.15 million, the number of deaths was 2 million. According to WHO, the largest number of fatal outcomes occurs in the United States, Brazil, and India [1].

A meta-analysis assessing the impact of COPD on COVID-19 mortality concluded that COPD, along with hypertension, CVD, DM, and age  $\geq$  65 years, is among the conditions associated with a high mortality risk (odds ratio - OR - 3.53; 95% confidence interval - CI - 1.79-6.96; p < 001) [16]. According to another meta-analysis, the risk of severe COVID-19 in patients with COPD increases by 4.38 times [15]. Therefore, COPD can be considered as a predictor of poor outcome in COVID-19.

A common risk factor for the development and progression of COPD and COVID-19 is tobacco smoking. Since the outbreak of diseases caused by the new coronavirus in China, many conflicting materials have appeared, some of which claim that smoking increases the risk of contracting SARS-CoV-2 and the likelihood of developing severe forms of COVID-19, while others say the opposite.

In both cases, doctors explained this by the fact that the lungs of smokers and non-smokers contain different numbers of ACE2 receptors [8, 17, 18, 19]. Subsequently, several more studies of this kind were published, the authors of which

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also came to conflicting conclusions. Doctors from the University of California (San Francisco, USA) tried to clarify this issue. To do this, they combined the results and observational data collected by the authors of 12 scientific papers (10 from China, 1 from Korea, and 1 from the USA) describing 9,025 patients with COVID-19 - 878 (9.7%) with severe disease and 495 with a history of smoking (5.5%). The meta-analysis showed a significant association between smoking and the progression of COVID-19 (relative risk - OR 2.25, 95% CI 1.49–3.39, p = 0.001). The study concluded that smoking is a risk factor for COVID-19 progression, with smokers having a higher risk of COVID-19 progression than those who have never smoked. The authors also recommended collecting smoking data as part of clinical management and adding smoking cessation to the list of methods for combating the COVID-19 pandemic [17]. The following factors may also contribute to the worsening of COVID-19 in COPD: impaired immunity, microbiome imbalance, increased mucus production, structural damage to the tracheobronchial tree tissue, and the use of inhaled glucocorticosteroids (IGCS).

**Target.**To study the clinical features of the course of the comorbid condition of COVID-19 and COPD.

**Materials and research methods.**We examined 80 patients, including 50 with covid pathology and 30 patients with a comorbid condition of COPD and covid. The study was conducted at the Bukhara Multidisciplinary Medical Center, in the pulmonology department. The study examined the clinical signs of monopathology and comorbid condition, their differences and similar signs.

**Own results.**The most striking clinical distinguishing feature between infectious exacerbation of COPD and COVID-19 is the difference in the types of fever. In COVID-19, approximately 90% of all patients have low-grade fever, and 20% have febrile fever. Febrile fever is not typical for exacerbation of COPD. Flulike symptoms with dyspnea can differentiate COVID-19 infection from dyspnea due to exacerbation of COPD. A detailed personalized patient survey was also conducted about the appearance of new clinical symptoms that go beyond the usual course of the exacerbation episode, such as myalgia, anorexia, and signs of gastrointestinal tract damage (Table 1). In general, exacerbation in patients with COPD is manifested by a rapid increase in respiratory symptoms, in the early stage of COVID-19, systemic symptoms such as fever and fatigue often predominate, and difficulty breathing can join in 6-7 days.

Table 1

		Taulo
Signs:	COVID-19	Exacerbation of COPD
	(n=50)	and COVID-19
		(n=30)
Fever	40 has subfebrile	Subfebrile
	10 have a febrile illness	
Shortness of breath	+	+

#### Asian journal of Pharmaceutical and biological research 2231-2218 http://www.ajpbr.org/ researchbib 8 Volume 13 Issue 3 SEPT.-DEC. 2024 Flu-like symptoms +-Myalgia ++Anorexia ++Gastrointestinal \_ +tract damage Fatigue ++Difficulty breathing ++In 10% of the subjects Telangiectasia \_

In addition, given the development of endothelial dysfunction in COPD, as an extrapulmonary complication resulting from the chronic inflammatory process, and the increased levels of procoagulant factors, these patients may be more susceptible to vascular damage and thrombosis during SARS-CoV-2 infection.

The risk assessment and stratification protocol proposed by R. Tal-Singer and JD Crapo recommends mandatory testing of all COPD patients for SARS-CoV-2 to avoid late diagnosis of COVID-19 [18]. There is evidence that some laboratory parameters (lactate dehydrogenase, D-dimer, C-reactive protein, fibrinogen, ferritin) can be used to detect early symptoms of coronavirus infection and predict its severity.

**Conclusion.** All of the above shows the relevance of the problem of comorbid COPD and COVID-19. Promising areas are: establishing a link between the frequency of exacerbations or severity of COPD with the outcomes or complications of COVID-19; determining the long-term negative effects of SARS-CoV-2 on the respiratory system and increasing the rate of COPD progression.

The physician should also conduct a detailed, personalized survey of the patient about the appearance of new clinical symptoms that go beyond the usual course of an exacerbation episode, such as myalgia, anorexia, and signs of gastrointestinal damage.

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