New Day in Medicine
Новый День в Медицине
NDM

TIBBIYOTDA YANGI KUN

Ilmiy referativ, marifiy-ma'naviy jurnal

AVICENNA-MED.UZ

ISSN 2181-712X.
EiSSN 2181-2187

12 (74) 2024

ТИББИЁТДА ЯНГИ КУН
НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
NEW DAY IN MEDICINE

Илмий-рефератив, маънавий-маърифий журнал
Научно-реферативный,
духовно-просветительский журнал

УЧРЕДИТЕЛИ:

БУХАРСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ ИНСТИТУТ
ООО «ТИББИЁТДА ЯНГИ КУН»

Национальный медицинский исследовательский центр хирургии имени А.В. Вишневского является генеральным научно-практическим консультантом редакции

Журнал был включен в список журнальных изданий, рецензируемых Высшей Аттестационной Комиссией Республики Узбекистан (Протокол № 201/03 от 30.12.2013 г.)

12 (74)

2024

ноябрь

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Received: 20.10.2024, Accepted: 02.11.2024, Published: 10.11.2024

UDC 578.834.1

COPD AS A COMORBID CONDITION IN COVID-19

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✓ ***Resume***

Objective: To present current data on the problem of the comorbidity of chronic obstructive pulmonary disease (COPD) and the new coronavirus infection (COVID-19), according to the literature.

The review summarizes and systematizes modern ideas about the association of COPD and COVID-19 and highlights the most important aspects of this problem - epidemiological, pathogenetic, clinical. The results of meta-analyses on the impact of COPD on the course of COVID-19 infection are presented.

The need for further clinical studies on the problem of the comorbidity of COPD and COVID-19 has been shown, which will allow a detailed study of the mechanisms of mutual aggravation of the associated pathology, to find out 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 to improve the prognosis of patients with COPD who have undergone the new coronavirus infection -COVID-19

Key words: comorbid, COVID-19, chronic obstructive pulmonary disease

ХОБЛ – КАК КОМОРБИДНОЕ СОСТОЯНИЕ ПРИ COVID-19

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✓ ***Резюме***

Цель: на основании литературных источников представить современные данные о проблеме коморбидности хронической обструктивной болезни легких (ХОБЛ) и новой коронавирусной инфекции COVID-19.

Обзор обобщает и систематизирует современные представления об ассоциации ХОБЛ и COVID-19 и освещает наиболее важные аспекты этой проблемы – эпидемиологические, патогенетические, клинические. Приводятся результаты метаанализов о влиянии ХОБЛ на течение инфекции COVID-19.

Показана необходимость дальнейших клинических исследований по проблеме коморбидности ХОБЛ и COVID-19, которые позволят детально изучить механизмы взаимоотягощения ассоциированной патологии, выяснить влияние SARS-CoV-2 на респираторную систему и течение ХОБЛ с учетом фенотипа заболевания, определить эффективные методы лечения и улучшить прогноз пациентов с ХОБЛ, перенесших новую коронавирусную инфекцию COVID-19

Ключевые слова: коморбидность, COVID-19, хроническая обструктивная болезнь легких

O‘SOK - COVID-19DA KOMORBID HOLAT SIFATIDA KELIShI

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✓ ***Rezyume***

Maqsad: ilmiy manbalarga asoslanib, surunkali obstruktiv o'pka kasalligi (O`SOK) va yangi COVID-19 koronavirus infektsiyasi muammosi bo'yicha dolzarb ma'lumotlarni taqdim etish.

Sharh O`SOK va COVID-19 assotsiatsiyasi haqidagi zamonaviy g'oyalarni umumlashtiradi va tizimlashtiradi va ushbu muammoning eng muhim jihatlari - epidemiologik, patogenetik, klinik jihatlari yoritilgan. O`SOKning COVID-19 infektsiyasi kechish jarayoniga ta'siri bo'yicha meta-tahlil natijalari taqdim etilgan.

O`SOK va COVID-19ning birgalikdagi kelishi muammosi bo'yicha keyingi klinik tadqiqotlarga ehtiyoj borligi, bu komorbid patologiyaning o'zaro ta`sir mexanizmlarini batafsil o'rganish, SARS-CoV-2 va O`SOKning komorbid holatda nafas olish tizimiga ta'sirini kechishi, yangi COVID-19 koronavirus infektsiyasiga chalingan O`SOK bilan og'rigan bemorlarni samarali davolash usullarini aniqlash va bashoratlashda kasallikning fenotipini hisobga olish zarurligi ko'rsatildi

Kalit so'zlar: komorbidlilik, COVID-19, surunkali obstruktiv o'pka kasalligi

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) worldwide was 108.15 million, with deaths reaching 2 million.According to WHO data, the largest number of fatal outcomes occurs in the USA, Brazil, and India [1].

Coronaviruses are a large family of single-stranded enveloped RNA viruses, including 43 species as of May 2020, named for the crown-like projections on their shells that extend in different directions.

Coronaviruses cause a wide range of pathological processes in various representatives of the animal world, including humans. A specific sign of coronavirus infections is damage to the upper respiratory tract, less often - the gastrointestinal tract. The three strains that can cause severe respiratory syndrome are currently considered the most dangerous for humans: SARS-CoV (caused an epidemic of acute respiratory syndrome in late 2002), MERS-CoV (caused an outbreak of severe respiratory infection called Middle East respiratory syndrome in 2012), and SARS-CoV-2 (causes interstitial pneumonia and acute respiratory distress syndrome) [14].

Materials and methods

The SARS-CoV-2 coronavirus, like SARS-CoV, is a member of the Beta-CoV coronavirus group and is a zoonotic disease. The pathognomonic clinical signs of COVID-19 are respiratory failure and respiratory distress syndrome [20]. Scientists have found that the new SARS-CoV-2 coronavirus penetrates the cell using the same “key” as the SARS-CoV (atypical pneumonia) virus: it binds to receptors for angiotensin-converting enzyme 2 (ACE2), which is expressed on the epithelium of the mucous membranes of the airways and some other organs, and is involved in the regulation of blood circulation. This connection serves as a “gate” for the virus to enter the cell. Penetration of the infection is facilitated by cellular proteases - transmembrane serine protease TMPRSS2, serine protease, furin and pH-sensitive endosomal protease CTSL [6]. It has been established that ACE2 receptors are contained in large quantities in the mucous membrane of the upper respiratory tract and lungs, small intestine, testicles, kidneys, heart, thyroid gland, and adipose tissue [4]. This is why people with higher expression of ACE2 in tissues are at risk for severe COVID-19: with diabetes mellitus (DM), cardiovascular diseases (CVD), chronic obstructive pulmonary disease (COPD). COPD patients infected with SARS-CoV-2 are a vulnerable group of people with a complicated course and often an unfavorable outcome of the disease due to age (> 40 years), comorbidities and smoking.

COPD is a progressive disease based on chronic inflammation in the respiratory tract as a result of exposure to harmful particles and gases, and characterized by persistent airflow limitation. The disease occurs with episodes of exacerbations and the development of extrapulmonary complications, which significantly contribute to the clinical picture and prognosis [11]. COPD is a highly prevalent disease: according to WHO, it ranks 3rd (4.8%) among causes of death worldwide.

Results and discussions

The leading etiologic cause of COPD exacerbation is respiratory infection. I. Satia et al., having analyzed the data of virological examination of 817,141 patients with COPD exacerbations who applied to the emergency department of one of the hospitals in Ontario (Canada) from 01.01.2003 to 31.12. 2013, of which 31.9% were hospitalized, found that a positive test for influenza A and B, respiratory syncytial virus, parainfluenza and adenovirus infection was in 39.2% of those hospitalized and in 41.1% of those visiting the emergency department daily [3]. The authors of the study noted that over the last 3 years of the study, the incidence of viral infection increased to 48.8% in hospitalized patients and to 52.5% in individuals who visited the emergency department daily, due to metapneumovirus, rhinovirus, and coronavirus. Viral predisposition may be due to a decrease in the production of α-interferon-1 [19] or a change in the cellular composition of the T-subpopulation [10, 20].

According to foreign studies, COPD is not a common comorbid pathology in COVID-19, but it aggravates the course and increases the risk of an unfavorable outcome of coronavirus infection, which, in turn, aggravates the condition of patients with COPD.

A nationwide analysis published in 2020 in China showed that the most common comorbid conditions were hypertension (17%), diabetes (8%), CVD (4%), COPD (2%), chronic kidney disease (1%), cerebrovascular disease (2%), and malignant neoplasms (1%). About 6.2% of patients in the overall cohort required emergency department admission, 3.1% required invasive ventilation, and 3.1% died. When all 3 indicators were taken together, the risk of achieving them in COPD patients increased by 2.7 times [9, 15]. In another study conducted in China, which involved 1,099 patients with laboratory-diagnosed COVID-19, COPD was found in 1.1% of patients [12].

Correlations between different non-coronavirus diagnoses and the severity of COVID-19 are presented in the first meta-analysis conducted by Chinese researchers [4]. The authors analyzed sixretrospective studies examining a total of 1,558 cases of the disease, proving that the comorbidity of COVID-19 and COPD increases the risk of severe infection by 5.97 times. Conclusions of the meta-analysis: COPD, along with arterial hypertension, diabetes, cardiovascular disease and cerebrovascular diseases, is recognized as an independent risk factor for severe course of the new coronavirus infection.

Another similar meta-analysis analyzed seven preprint studies and also identified COPD as one of the indicators of a poor outcome of COVID-19 [21]. After analyzing data from China, Italian scientists also concluded that the presence of COPD, as a comorbid pathology, increases the risk of severe COVID-19 by more than 5 times [15]. Patients with COVID-19 and concomitant COPD have a 2.3-fold higher rate of primary [13] and re-hospitalization after discharge.

A meta-analysis assessing the impact of COPD on COVID-19 mortality concluded that COPD, along with hypertension, CVD, DM, and age ≥ 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 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 severity 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) [15].

In addition, given the development of endothelial dysfunction in COPD, as an extrapulmonary complication resulting from the chronic inflammatory process, and the increased level of procoagulant factors [3], these patients may be more susceptible to vascular damage and thrombosis during SARS-CoV-2 infection [18].

The main task of clinicians when a patient with COPD seeks medical attention 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 most striking clinical distinguishing feature of an infectious exacerbation of COPD and COVID-19 is the difference in the types of fever. With COVID-19, approximately 90% of all patients have subfebrile fever, and 20% have febrile fever. Febrile fever is not typical for an exacerbation of COPD. Flu-like symptoms with shortness of breath can distinguish COVID-19 infection from shortness of breath due to an exacerbation of COPD. The doctor 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 [7]. In general, exacerbation in patients with COPD is characterized 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 may occur after 6-7 days.

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

Thus, 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; developing prognostic models of unfavorable prognosis of comorbid pathology and patient management protocols; studying the effect of long-term self-isolation on the natural course of COPD.

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Entered 20.10.2024