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A Patient with 38 Years of Smoking History and Severe Shortness of Breath

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Aim: to present a clinical case of post-COVID bronchiolitis in an adult.
Key points. A 54-year-old female patient with a long history of smoking was hospitalized in the pulmonology department of the Clinic with a leading complaint of progressive shortness of breath after a COVID-19 infection. The diagnosis was established: bilateral bronchiolitis associated with a new coronavirus infection (SARS-CoV-2 polymerase chain reaction — positive); chronic obstructive pulmonary disease stage II according to GOLD (Global Initiative for Chronic Obstructive Lung Disease), exacerbation. Against the background of the therapy, there was a significant positive dynamics in well-being and in CT-picture of bronchiolitis. The patient was discharged for outpatient follow-up treatment.
One of the most common symptoms of post-COVID syndrome is shortness of breath. Diagnosis of bronchiolitis, in which this symptom may be the only one, is difficult, especially in patients with a long history of smoking and the presence of comorbid pulmonary pathology.
Conclusion. A clinical case of bilateral bronchiolitis in the framework of the post-COVID syndrome is presented. The key to successful diagnosis of bronchiolitis is a thorough differential analysis of the totality of anamnestic and clinical laboratory data, as well as a characteristic CT pattern.
Keywords: post-COVID syndrome, bronchiolitis, computed tomography, steroid therapy
Conflict of interest: the authors declare that there is no conflict of interest.

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Пациентка со стажем курения 38 пачка-лет и прогрессирующей одышкой

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Цель: представить клиническое наблюдение постковидного бронхиолита у взрослого.
Основные положения. Пациентка в возрасте 54 лет с длительным анамнезом табакокурения госпитализирована в пульмонологическое отделение Клиники с ведущей жалобой на прогрессирующую одышку после перенесенной инфекции COVID-19. Установлен диагноз: двусторонний бронхиолит, ассоциированный с перенесенной новой коронавирусной инфекцией (ПЦР SARS-CoV-2 — положительная); хроническая обструктивная болезнь легких II стадии по GOLD (Global Initiative for Chronic Obstructive Lung Disease), обострение. На фоне проводимой терапии отмечена значительная положительная динамика в самочувствии и на снимках компьютерной томографии. Пациентка выписана на амбулаторное долечивание.
Одним из самых распространенных симптомов постковидного синдрома служит одышка. Диагностика бронхиолита, при котором данный симптом может быть единственным, затруднена, особенно у пациентов с длительным анамнезом курения и наличием коморбидной легочной патологии.
Заключение. Представлен клинический случай двустороннего бронхиолита в рамках постковидного синдрома. Залогом успешной диагностики бронхиолита служит тщательный дифференциальный анализ совокупности анамнестических и клинико-лабораторных данных, а также характерный КТ-паттерн.
Ключевые слова: постковидный синдром, бронхиолит, компьютерная томография, стероидная терапия
Конфликт интересов: авторы заявляют об отсутствии конфликта интересов.

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On March 11, 2020, the World Health Organization (WHO) announced the beginning of a pandemic of a new coronavirus infection COVID-19 [1]. Despite the fact that on May 5, 2023, the completion of its emergency phase was reported, the fight against the causative agent SARS-CoV-2 continues [2, 3]. According to official WHO statistics, 2,3061,960 confirmed cases of COVID-19 with 400,102 deaths were registered in the Russian Federation from January 3, 2020 to October 18, 2023 [4].

During the pandemic, the Clinic became one of the first university structures repurposed for the treatment of a new coronavirus infection. Under the leadership of Academician V.T. Ivashkin, key features of COVID-19-associated pneumonia were identified, a combination of steroid therapy with anticoagulants was used for the first time, which made it possible to achieve positive dynamics of CT on days 8–10 and a tendency to decrease hospital mortality [5]. The staff of the Clinic and the department continue to analyze the accumulated experience of managing such patients with a variety of organ and system lesions. Currently, special attention is paid to the understanding of clinical cases with multifaceted manifestations of post-COVID syndrome, one of which, in our opinion, is bronchiolitis. We are presenting a clinical case.

Patient B., female, 54 years old, was admitted to the Clinic with complaints of progressive shortness of breath during exercise, paroxysmal unproductive cough and pronounced general weakness. The severity of the leading complaint — dyspnea — according to the mMRC scale (Modified Medical Research Council Dyspnea Scale) corresponded to grade II [6]. The patient had had a long history of tobacco smoking (38 years of 20 cigarettes a day): the index of a smoking person — 240, the index of a pack — 38 years, which corresponds to a high risk of chronic obstructive pulmonary disease (COPD).

In 2017, the patient first sought medical help at her place of residence with a complaint of an unproductive cough and was diagnosed with chronic bronchitis. Treatment with protected ampicillin and mucolytics with a positive effect was carried out. In June 2020, she noted the appearance of shortness of breath and an unproductive cough. According to computed tomography of the chest organs, bullous emphysema was detected on an outpatient basis. Deterioration occurred in June 2021 in the form of an increase in previous symptoms. According to spirometry, bronchial obstruction was detected: forced vital capacity of the lungs (FVC) — 90 %, volume of forced exhalation per 1 second (FEV1) — 65 %, FEV1/VC (Tiffno index) — 69 %. Taking into account a long history of smoking, a clinical diagnosis was made: COPD, stage II according to GOLD (moderate course: Tiffno index < 70 %, FEV1 from the proper 50 % ≤ FEV1 < 80 %), emphysema. Against the background of the prescribed basic therapy (M-holinoblocker, beta2-agonist, topical glucocorticosteroid (GCS), as well as nebulizer — combined bronchodilator, mucolytic), well-being improved, shortness of breath decreased.

In October 2021, the patient noted an increase in body temperature to 38 °C, loss of sense of smell, increased shortness of breath, cough, pronounced general weakness. The RNA of the SARS-CoV-2 virus was isolated by PCR, and a new coronavirus infection was diagnosed. She was receiving antibiotics (macrolide + respiratory fluoroquinolone) on an outpatient basis for 7 days, symptomatic therapy, against which the body temperature returned to normal, shortness of breath, cough and general weakness decreased.

Deterioration of health occurred in November 2021, when she noted an increase in shortness of breath, cough, and general weakness. According to CT scans of the chest organs, areas of mosaic density of the pulmonary parenchyma were noted. A diagnosis was made: COPD, emphysematous variant, bronchiolitis, and steroid therapy was recommended, which the patient did not take. Due to the persistence of the above complaints, she applied for a consultation at the Clinic in January 2022 and was hospitalized in the Pulmonology Department.

On objective examination: general condition of moderate severity, clear consciousness, body temperature 36.8 °C, body mass index (BMI) — 27.7 kg/m^2. The skin and visible mucous membranes were pale pink, moderately moist, clean. The cervical veins were not swollen. There was no peripheral edema. Lymph nodes were not palpated. The frequency of respiratory movements — 22 per minute, saturation when breathing atmospheric air — 96 %. The thorax was conical in shape. With percussion, the lungs emitted a box sound. With auscultation of the lungs, breathing was hard, scattered dry wheezing. The boundaries of relative cardiac dullness: right — at the right edge of the sternum, left — in the V intercostal space along the mid-clavicular line, upper — at the level of third rib. The heart tones were rhythmic. Heart rate (HR) and pulse — 90 beats per minute. Blood pressure — 140/90 mmHg. The abdomen was soft, painless in all departments. The liver did not protrude from under the edge of the costal arch. The spleen was not enlarged. There were no symptoms of irritation of the peritoneum. The symptom of pounding

was negative on both sides. Urination was free, painless. The stool was regular, decorated. Based on complaints, anamnesis data, objective examination, a preliminary diagnosis was formulated: post-COVID syndrome: bilateral bronchiolitis associated with a new coronavirus infection (October 2021, PCR SARS-CoV-2 positive), COPD of stage II by GOLD, moderate severity, exacerbation, respiratory failure of I degree, emphysema of the lungs. Taking into account the patient's main complaint of shortness of breath during exercise, the circle of differential diagnosis included congestive chronic heart failure, COPD, bronchial asthma, interstitial lung diseases, bronchiolitis, pneumonia, thromboembolism of the branches of the pulmonary artery.

In the general blood test, an increase in ESR to 24 mm/hour was noted, eosinophils were within the normal range; in the biochemical blood test, moderate cytolysis (the patient has a history of chronic hepatitis B), dyslipidemia, the level of C-reactive protein — at the upper limit of the norm; coagulogram (including D-dimer), the level of immunoglobulin E — within normal values. In the clinical analysis of sputum, leukocytes were 10–25 in the visual field, there were many alveolar macrophages, Kurshman spirals — 1–2 in the visual field. Electrocardiography recorded sinus tachycardia with a heart rate of 100 beats per minute. According to the study of the function of external respiration (FER), a disorder of an obstructive type was detected (FVC — 82 %, FEV1 — 62 %, Tiffno index — 67 %), the bronchodilation test was negative. Echocardiography revealed a compaction of the walls of the aorta, the chambers of the heart were not dilated, the ejection fraction was 64 %, the systolic pressure in the pulmonary artery — 20 mmHg. Thus, congestive chronic heart failure, pulmonary embolism, bronchial asthma were excluded from the circle of differential diagnosis.

According to CT scans of the chest organs, a pattern was determined that testified in favor of bronchiolitis (Fig. 1).

Taking into account the clinical picture, the results of laboratory and instrumental studies, the patient was diagnosed with: Combined diseases: 1. Post-COVID syndrome: bilateral bronchiolitis associated with a new coronavirus infection. Background disease: New coronavirus infection in October 2021 (positive SARS-CoV-2 PCR). 2. COPD, stage II by GOLD, moderate severity, exacerbation. Complication: Respiratory failure of stage 1. Pulmonary emphysema. Concomitant diseases: Chronic hepatitis B. Hypertension, degree 1. The patient was prescribed: intravenous drip of the GCS (dexamethasone); bronchodilator eufillin; nebulizer therapy (combined bronchodilator berodual, mucolytic ambroxol and topical GCS budesonide); oxygen, as well as proton pump inhibitor omeprazole, taking into account the steroid therapy. Against the background of the therapy, the state improved significantly, the degree of shortness of breath corresponded to grade 0–I on the mMRC

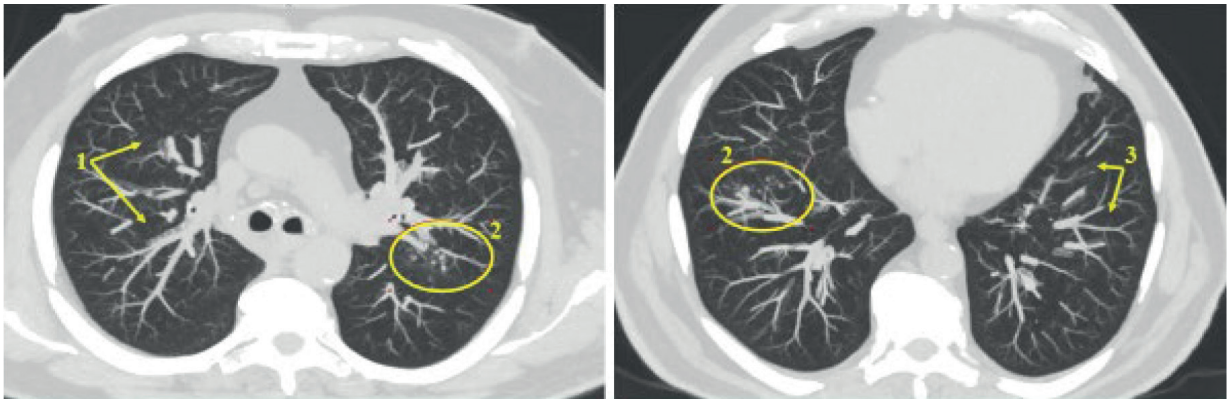

Figure 1. CT scan of the chest organs of Patient B., 54 years old. Multiple centrilobular foci of the "ground glass" type are identified (a slight increase in the density of lung tissue as a result of interstitial inflammation while maintaining the visibility of the walls of blood vessels and bronchi in the area of changes) with unclear contours (1), Y-structures with small nodules at the ends (2), uneven "mosaic" pneumatization of the lungs (a combination of areas of increased and decreased density of lung tissue with obstruction of the small airways) (3)

Рисунок 1. КТ органов грудной клетки пациентки Б., 54 лет. Определяются множественные центрилобулярные очаги по типу «матового стекла» (незначительное повышение плотности легочной ткани в результате интерстициального воспаления с сохранением видимости стенок сосудов и бронхов в зоне изменений) с нечеткими контурами (1), Y-структуры с мелкими узелками на концах (2), неравномерная «мозаичная» пневматизация легких (сочетание участков повышенной и пониженной плотности легочной ткани при обструкции малых дыхательных путей) (3)

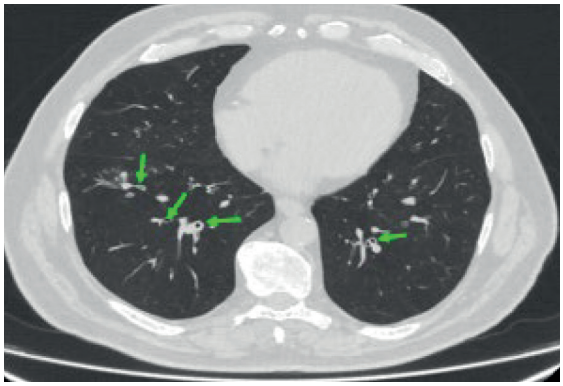

Figure 2. CT image of the chest organs of Patient B., 54 years old, in dynamics. There is a disappearance of "ground glass" areas and an almost complete disappearance of the CT pattern of bronchiolitis. Green arrows indicate thickened bronchial walls

Рисунок 2. КТ-картина органов грудной клетки пациентки Б., 54 лет, в динамике. Отмечается исчезновение участков «матового стекла», практически полное исчезновение КТ-паттерна бронхиолита. Зеscale. A month later, positive dynamics was noted in the CT picture (Fig. 2). The patient was given recommendations (to give up smoking, take basic therapy with a combination of olodaterol and tiotropium bromide; continue dynamic monitoring).

Discussion of the features of clinical case

The pathological effects of the SARS-CoV-2 virus on the human body continue even after acute infection, regardless of its severity, affecting the ability to work and quality of life. According to the WHO proposal, post-COVID syndrome (PCS) is a condition that occurs in individuals after infection with SARS-CoV-2, usually three months after the onset of COVID-19, or in individuals with suspected new coronavirus infection, with symptoms that last at least two months and cannot be explained by an alternative diagnosis [7]. Currently, there are few large clinical studies that help determine the diagnostic criteria and management strategy for patients with PCS [8].

In most cases, shortness of breath is considered within the framework of the PCS as a symptom of a decrease in FER against the background of residual phenomena of organizing pneumonia or the development of fibrosis [9, 10]. However, as the experience of clinicians shows, the respiratory target for SARS-CoV-2 is not only the alveolus, but also the bronchiole [11]. Interestingly, the first description of bronchiolitis associated with coronavirus dates back to 2004, when Dutch researchers isolated a new HCoV-NL63 coronavirus (Human coronavirus NL63) from a seven-month-old child with bronchiolitis [12]. In 2005, scientists at the University of Hong Kong isolated a new human coronavirus HCoV-HKU1 (Human coronavirus HKU1) from a 71-year-old patient with acute respiratory disease. The majority of patients with HCoV-NL63 and HCoV-HKU1 viruses had symptoms of bronchiolitis and pneumonia [13–15].

F. Bianchi et al. described a case of bronchiolitis was described in a 61-year-old obese Italian woman (BMI — 41.66 kg/m^2) hospitalized with acute respiratory failure in the emergency department of the University Hospital of Siena in January 2020 with a 3-week history of shortness of breath and dry cough. The clinical picture of bronchiolitis was confirmed by CT of the chest organs, a coronavirus was detected by PCR, and the HKU1 genotype (HCoV-HKU1) was identified during subsequent typing [16].

In October 2022, the results of the analysis of 54 studies and two databases of medical records were published (n = 1.2 million people from 22 countries, including Russia, who had symptoms of SARS-CoV-2 infection). Three months after the illness, the main three groups of post-COVID complaints continued to be registered: respiratory (shortness of breath, cough, etc.) — 3.7 %, constant fatigue with bodily pain or mood swings — 3.2 %, cognitive problems — 2.2 % [17].

The prevalence of bronchiolitis within the framework of PCS is unknown. The long-established fact of the tropicity of respiratory viruses to ciliated cells and Clara cells suggests the involvement of bronchioles in the multicomponent pathogenesis of respiratory manifestations of a new coronavirus infection [18]. The share of small airways (terminal and respiratory bronchioles), as is known, accounts for only 20 % of the total resistance of airways [19], which may explain the asymptomatic course of bronchiolitis in the early stages and the duration of clinical manifestations within the framework of PCS. The suspected pathogenesis of bronchiolitis associated with COVID-19 infection is shown in Figure 3.

The structural features of bronchioles (small airways with a diameter of less than 2 mm) consist in the absence of cartilaginous rings and mucous glands. The epithelial lining has a smaller thickness than in cartilaginous bronchi, consists of cylindrical ciliated cells and secretory Clara cells, characterized by high metabolic activity. Under the epithelium lies a thin layer of its own plate of the mucous membrane, muscle and connective tissue membrane, performing a supporting function. There are a large number of thin-walled vessels in the walls of bronchioles, forming a capillary network at the level of respiratory bronchioles. The entrance gate

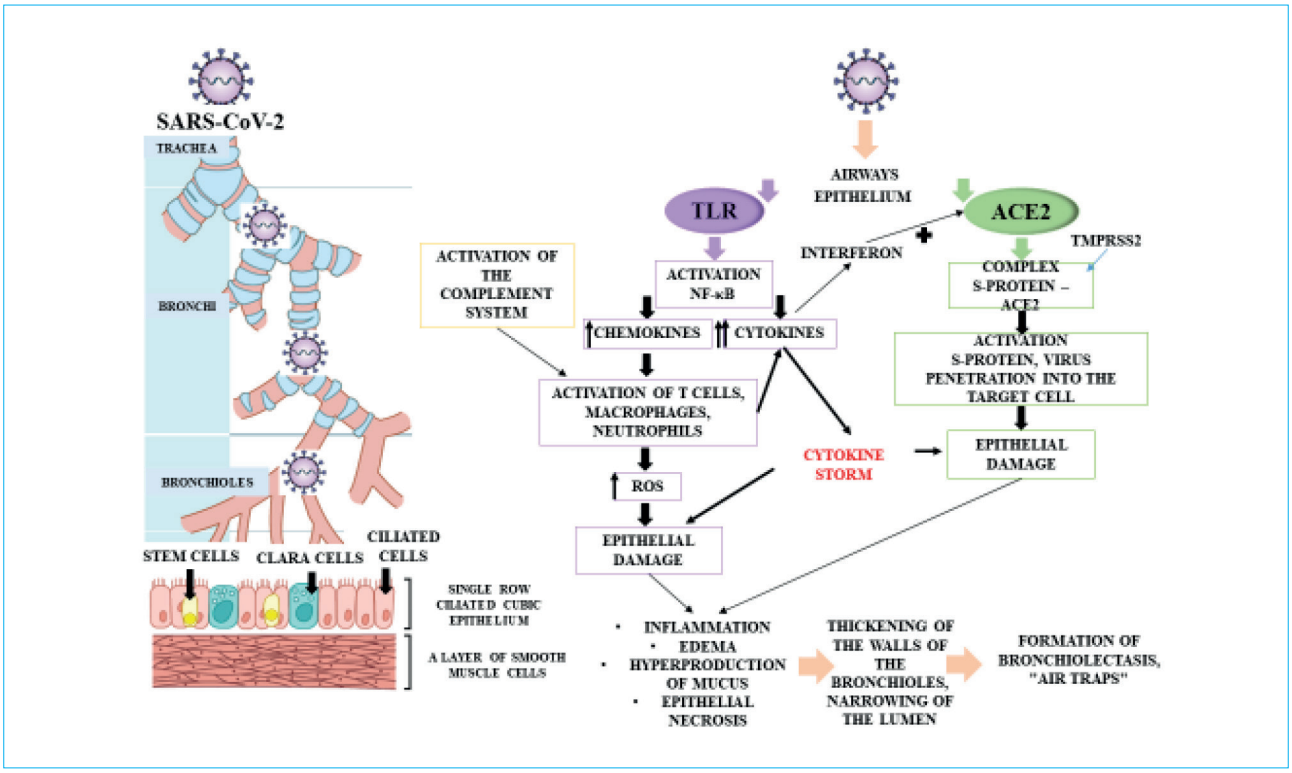

Figure 3. Scheme of the structure of bronchioles and the expected pathogenesis of bronchiolitis during a new coronavirus infection (according to [20, 21] with modifications). SARS-CoV-2 — severe acute respiratory syndrome coronavirus-2; TLR — Toll-like receptors; NF-κB — nuclear factor kappa bi; ROS — reactive oxygen species; ACE2 receptor — angiotensin-converting enzyme 2 receptor; TMPRSS2 — transmembrane serine protease 2; S-protein — spike protein

Рисунок 3. Схема строения бронхиол и предполагаемый патогенез бронхиолита при новой коронавирусной инфекции (по [20, 21] с изменениями). SARS-CoV-2 (severe acute respiratory syndrome-related coronavirus 2) — коронавирус тяжелого острого респираторного синдрома-2; TLR (Toll-like receptor) — Толл-подобные рецепторы; NF-κB (nuclear factor kappa B) — ядерный фактор каппа би; ROS (reactive oxigen species) — активные формы кислорода; ACE2 receptor (angiotensin-converting enzyme 2) — рецептор ангиотензинпревращающего фермента 2; TMPRSS2 (transmembrane serine protease 2) — трансмембранная сериновая протеаза 2; S-protein — спайковый белок

for SARS-CoV-2 is, first of all, the epithelium of airways. On the one hand, the virus interacts with specialized TLR that recognize SARS-CoV-2 and trigger the mechanism of nonspecific protection of the body through the activation of transcription NF-kB. Activated epithelial cells begin to synthesize cytokines, including chemokines, which have a chemoattracting effect. As a result, macrophages and polymorphonuclear leukocytes (neutrophils, etc.) are attracted to the site of virus introduction, which activate, phagocytize, secrete pro-inflammatory mediators, lead to an increase in the formation of ROS, thus triggering the mechanism of damage to epithelial cells. On the other hand, the coronavirus spike protein binds to the ACE2 receptor, which is highly expressed on epithelial cells of airways. This complex undergoes the proteolysis of TMPRSS2, which leads to the cleavage of ACE2 and activation of the spike protein, contributing to the penetration of the virus into the target cell, damaging it. Interferon stimulates the ACE2 gene, thereby opening up opportunities for additional damaging effects of the virus [20]. The complement system is also assumed to be involved in the pathogenesis of the inflammatory response in a new coronavirus infection: the epithelium and smooth muscle cells of airways contain receptors for anaphylotoxins C3a and C5a, which have a pro-inflammatory effect, activating neutrophils, monocytes, macrophages and eosinophils [22]. Thus, hyperproduction of cytokines leads to the development of a cytokine storm, taking into account damage to the alveoli and the development of endothelial dysfunction, causing the severity of the course of the disease. Bronchiolitis is characterized by inflammation and edema of small airways, hyperproduction of mucus and necrosis of the epithelium. As a result, there is a thickening of the walls of the bronchioles and a narrowing of the lumen, which leads to bronchiolar obstruction.

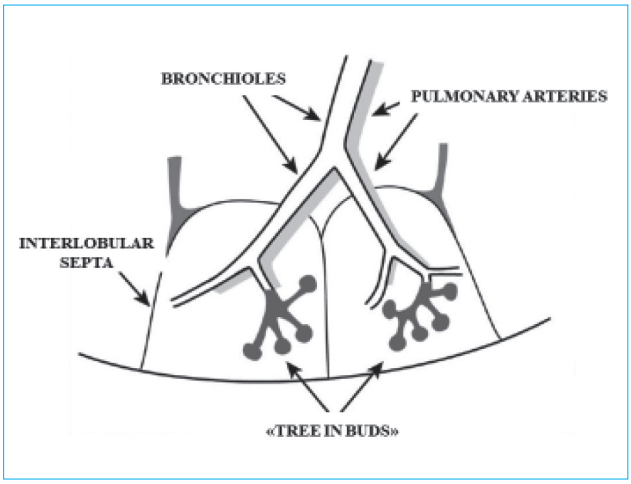

Figure 4. Scheme of the "tree-in-buds" CT pattern (according to [23], with modifications). The name of the symptom is associated with the external similarity of the CT image to a branch of a flowering tree. Centrilobular branching Y-structures with small nodules at the ends reflect the filling of intralobular bronchioles with inflammatory secretions, dilatation of bronchioles and thickening of their walls [24]

Рисунок 4. Схема КТ-паттерна «дерево в почках» (по [23], с изменениями). Название симптома связано с внешней схожестью КТ-картины с веточкой цветущего дерева. Центрилобулярные ветвящиеся Y-структуры с мелкими узелками на концах отражают наполнение внутридольковых бронхиол воспалительным секретом, дилатацию бронхиол и утолщение их стенок [24]

Table. Differential diagnosis of bronchiolitis with the "tree-in-bud" CT pattern

Таблица. Дифференциальный диагноз бронхиолита с КТ-паттерном «дерево в почках»

Type of bronchiolitis ***Тип бронхиолита***	**CT features** ***КТ-особенности***	**Clinical features** ***Клинические особенности***
Aspiration *Аспирационный*	Often local or multifocal process. Broncho- and bronchiolectasis, fibrosis (indicate chronic aspiration) *Часто локальный или мультифокальный процесс. Бронхо- и бронхиолоэктазы, фиброз (свидетельствуют о хронической аспирации)*	Risk groups for aspiration: - neurological diseases (stroke, traumatic brain injury, multiple sclerosis); - laryngeal cancer - diseases of the esophagus (esophagitis with the formation of strictures, diverticulum, tracheoesophageal fistula, tumors, achalasia, large diaphragmatic hernia) Clinically — long-lasting cough with clear sputum, bronchospasm. Often, these symptoms are regarded as asthma or COPD that is refractory to therapy with steroids and bronchodilators, resulting in late diagnosis. *Группы риска развития аспирации:* *- неврологические заболевания (инсульт, черепно-мозговая травма, рассеянный склероз);* *- рак гортани;* *- заболевания пищевода (эзофагит с формированием стриктур, дивертикул, трахеопищеводный свищ, опухоли, ахалазия, большая диафрагмальная грыжа).* *Клинически — длительно продолжающийся кашель с прозрачной мокротой, бронхоспазм. Зачастую данные симптомы расцениваются как течение бронхиальной астмы или ХОБЛ, рефрактерных к терапии стероидами и бронходилататорами, вследствие чего характерна поздняя диагностика*
Infectious *Инфекционный*	Asymmetric focal or multifocal process. Inflammation of small airways → filling of bronchioles with pathological secretions → centrilobular nodules of varying density, "tree-in-buds" pattern, thickening of bronchial walls *Асимметричный фокальный или мультифокальный процесс. Воспаление малых ДП → заполнение патологическим секретом бронхиол → центрилобулярные узелки различной плотности, паттерн «дерево в почках», утолщение стенок бронхов*	Nonspecific signs of a respiratory viral infection (including recent ones) + progressive shortness of breath + dry cough. In the presence of cavitation and bronchiectasis, it is necessary to exclude mycobacteriosis *Неспецифические признаки респираторной вирусной инфекции (в том числе недавно перенесенной) + прогрессирующая одышка + сухой кашель.* *При наличии кавитаций и бронхоэктазов необходимо исключить микобактериоз*

Table continued. Differential diagnosis of bronchiolitis with the "tree-in-bud" CT pattern
Продолжение таблицы. Дифференциальный диагноз бронхиолита с КТ-паттерном «дерево в почках»

Diffuse panbronchiolitis *Диффузный панбронхиолит*	Centrilobular nodules, thickening of the bronchiole wall. Progression with the formation of bronchiectasis, cysts, bullae, "air traps" *Центрилобулярные узелки, утолщение стенки бронхиол. Прогрессирование с формированием бронхоэктазов, кист, булл, «воздушных ловушек»*	It occurs much less frequently than other forms of cellular bronchiolitis. A steadily progressing pathological process in the upper and lower respiratory tract of unknown etiology. Symptoms of chronic sinusitis in combination with cough and gradual increase in shortness of breath. Infection with *P. aeruginosa* is typical with a CT picture of organizing pneumonia *Встречается значительно реже остальных форм клеточного бронхиолита. Неуклонно прогрессирующий патологический процесс в верхних и нижних дыхательных путях неизвестной этиологии. Симптомы хронического синусита в сочетании с кашлем и постепенным нарастанием одышки. Свойственно инфицирование P. aeruginosa с КТ-картиной организующейся пневмонии*

The lesion of small airways makes a significant contribution to the severity of the course of the combined pulmonary disease, while not being clinically manifested for a long time due to the anatomical and physiological features of the structure of the bronchiole [19]. CT serves as a method of choice for confirming the diagnosis, allowing to assess the state of the structures of the secondary lobule involved in the pathological process. For postinfectious bronchiolitis, the characteristic CT pattern is a "tree in buds" (Fig. 4).

Taking into account the clinical manifestations (progressive dyspnea, tachypnea, tachycardia, dry wheezing) and the "tree-in-buds" CT pattern, we performed a differential diagnosis of bronchiolitis. As can be seen from Table 1, this CT imaging combines three types of cellular bronchiolitis: aspiration, infectious and diffuse panbronchiolitis [24, 25].

Given the new coronavirus infection, our patient is most likely to have an infectious nature of bronchiolitis. The peculiarity of this clinical case is the development of infectious bronchiolitis within the framework of PCS on an initially unfavorable background (the presence of chronic respiratory failure due to a long history of smoking and the development of COPD). It is noteworthy that the degree of respiratory failure is minimal, clinical manifestations and response to oxygen therapy indicate, on the one hand, in favor of the centralization of blood circulation, the predominance of violations of the ventilation-perfusion balance, on the other hand, a patient with an obstructive type of FER has no obvious signs of hypoxemia and hypercapnia.

The isolation of bronchiolitis in PCS is of particular importance in patients with a burdened bronchopulmonary history and determines the need for timely diagnosis and treatment, quality of life and prognosis. The appointment of steroid therapy allows you to stop the main symptom — shortness of breath — and achieve positive radiological dynamics. The main effect of GCS is a decrease in the level of circulation of proinflammatory cytokines, a decrease in immune-mediated damage to organs and systems in a new coronavirus infection [5]. However, there is currently no evidence base for the use of GCS in patients with bronchiolitis within the framework of PCS.

Conclusion

Thus, post-COVID syndrome is an urgent problem of the present time, the scale of which cannot be estimated yet. In real clinical practice, it is very difficult to suspect bronchiolitis in patients with a nonspecific complaint of progressive shortness of breath, the absence of hypoxemia with prolonged smoking, and the development of COPD. The persistence and increase of shortness of breath after a COVID-19 infection requires a thorough differential diagnosis. In the diagnosis of bronchiolitis, the identification of a characteristic CT pattern helps. It is necessary to further study the pathogenesis of COVID-19 infection and post-COVID syndrome to improve treatment and develop comprehensive rehabilitation programs.

References / Литература

1. *World Health Organization.* Novel coronavirus (2019-nCoV) situation report 5. 25 January 2020 URL: https://www.who.int/docs/default-source/coronaviruse/situation-reports/20200125-sitrep-5-2019-ncov.pdf?sfvrsn=429b143d_8
2. *Lenharo M.* WHO declares end to COVID-19's emergency phase. *Nature*. 2023 May 5. (Online ahead of print). DOI: 10.1038/d41586-023-01559-z
3. *Профилактика, диагностика и лечение новой коронавирусной инфекции (COVID-19): временные методические рекомендации.* Версия 17, утверждены Министерством здравоохранения РФ 14 декабря 2022 г. [*Prevention, diagnosis and treatment of new coronavirus infection (COVID-19): Temporary guidelines*. Version 17, approved by the Ministry of Health of the Russian Federation December 14 2022. (In Russ.)]. URL: rosminzdrav.ru/ministry/med_covid19
4. *World Health Organization.* Coronavirus (COVID-19) Dashboard. Russian Federation. URL: https://covid19.who.int/region/euro/country/ru
5. *Ивашкин В.Т., Зольникова О.Ю., Свистунов А.А., Кокина Н.И., Джахая Н.Л., Поцхверашвили Н.Д. и др.* Кортикостероиды в лечении пациентов с поражением легких, обусловленным инфекцией SARS-CoV-2. *Сеченовский вестник*. 2020;11(2):19–28. [*Ivaschkin V.T., Zolnikova O.Yu., Svistunov A.A., Kokina N.I., Dzhakhaya N.L., Potskhverashvili N.D., et al.* Corticosteroids in the treatment of SARS-CoV-2 related lung disease. *Sechenov Medical Journal*. 2020;11(2):19–28. (In Russ.)]. DOI: 10.47093/2218-7332.2020.11.2.19-28
6. *Глобальная стратегия диагностики, лечения и профилактики хронической обструктивной болезни легких (пересмотр 2014 г.).* М.: Российское респираторное общество, 2014. [*Global strategy for the diagnosis, treatment and prevention of chronic obstructive pulmonary disease (revision of 2014)*. Moscow: Russian Respiratory Society, 2014. (In Russ.)].
7. *World Health Organization.* A clinical case definition of post COVID-19 condition by a Delphi consensus. October 6, 2021. URL: https://www.who.int/publications-detail-redirect/WHO-2019-nCoV-Post_COVID-19_condition-Clinical_case_definition-2021.1
8. *Abdel-Gawad M., Zaghloul M.S., Abd-Elsalam S., Hashem M., Lashen S.A., Mahros A.M., et al.* Post-COVID-19 syndrome clinical manifestations: A systematic review. *Anti-inflamm Antiallergy Agents Med Chem*. 2022;21(2):115–20. DOI: 10.2174/1871523021666220328115818
9. *Torres-Castro R., Vasconcello-Castillo L., Alsina-Restoy X., Solis-Navarro L., Burgos F., Puppo H., et al.* Respiratory function in patients post-infection by COVID-19: A systematic review and meta-analysis. *Pulmonology*. 2021;27(4):328–37. DOI: 10.1016/j.pulmoe.2020.10.013
10. *Zhao Y.M., Shang Y.M., Song W.B., Li Q.Q., Xie H., Xu Q.F., et al.* Follow-up study of the pulmonary function and related physiological characteristics of COVID-19 survivors three months after recovery. *EClinicalMedicine*. 2020;25:100463. DOI: 10.1016/j.eclinm.2020.100463
11. *Sebina I., Phipps S.* The contribution of neutrophils to the pathogenesis of RSV bronchiolitis. *Viruses*. 2020;12(8):808. DOI: 10.3390/v12080808
12. *van der Hoek L., Pyrc K., Jebbink M., Vermeulen-Oost W., Berkhout R.J., Wolthers K.C., et al.* Identification of a new human coronavirus. *Nat Med*. 2004;10:368–73. DOI: 10.1038/nm1024
13. *Arden K.E., Nissen M.D., Sloots T.P., Mackay I.M.* New human coronavirus, HCoV-NL63, associated with severe lower respiratory tract disease in Australia. *J Med Virol*. 2005;75(3):455–62. DOI: 10.1002/jmv.20288
14. *Woo P.C., Lau S.K., Chu C.M., Chan K.H., Tsoi H.W., Huang Y., et al.* Characterization and complete genome sequence of a novel coronavirus, coronavirus HKU1, from patients with pneumonia. *J Virol*. 2005;79(2):884–95. DOI: 10.1128/JVI.79.2.884-895.2005
15. *Lau S.K., Woo P.C., Yip C.C., Tse H., Tsoi H.W., Cheng V.C., et al.* Coronavirus HKU1 and other coronavirus infections in Hong Kong. *J Clin Microbiol*. 2006;44(6):2063–71. DOI: 10.1128/JCM.02614-05
16. *Bianchi F., Bennett D., Alderighi L., Pieroni M., Refini R.M., Fossi A., et al.* Coronavirus HKU 1 infection with bronchiolitis, pericardial effusion and acute respiratory failure in obese adult female. *J Asthma*. 2021;58(8):1128–31. DOI: 10.1080/02770903.2020.1761981
17. *Global Burden of Disease Long COVID Collaborators; Wulf Hanson S., Abbafati C., Aerts J.G., Al-Aly Z., Ashbaugh C., Ballouz T., et. al.* Estimated global proportions of individuals with persistent fatigue, cognitive, and respiratory symptom clusters following symptomatic COVID-19 in 2020 and 2021. *JAMA*. 2022;328(16):1604–15. DOI: 10.1001/jama.2022.18931
18. *Florin T.A., Plint A.C., Zorc J.J.* Viral bronchiolitis. *Lancet*. 2017;389(10065):211–24. DOI: 10.1016/S0140-6736(16)30951-5
19. *Авдеев С.Н., Авдеева О.Е.* Бронхиолиты взрослых. Атмосфера. *Пульмонология и аллергология*. 2005;2:11–7. [Avdeev S.N., Avdeeva O.E. Bronchiolitis of adults. Atmosphere. *Pulmonology and Allergology*. 2005;2:11–7. (In Russ.)].
20. *Болевич С.Б., Болевич С.С.* Комплексный механизм развития COVID-19. *Сеченовский вестник*. 2020;11(2):50–61. [*Bolevich S.B., Bolevich S.S.* Complex mechanism of COVID-19 development. *Sechenov Medical Journal*. 2020;11(2):50–61. (In Russ.)]. DOI: 10.47093/2218-7332.2020.11.2.50-61
21. *Risitano A.M., Mastellos D.C., Huber-Lang M., Yancopoulou D., Garlanda C., Ciceri F., et al.* Complement as a target in COVID-19? *Nat Rev Immunol*. 2020;20(6):343–4. DOI: 10.1038/s41577-020-0320-7
22. *Carvelli J., Demaria O., Vély F., Batista L., Chouaki Benmansour N., Fares J., et al.; Explore COVID-19 IPH group; Explore COVID-19 Marseille Immunopole group.* Association of COVID-19 inflammation with activation of the C5a-C5aR1 axis. *Nature*. 2020;588(7836):146–50. DOI: 10.1038/s41586-020-2600-6
23. *Lee N.J., Do K.-H.* Many faces of the centrilobular nodules: What should radiologists consider? *ECR 2017*. Poster number C-1781. DOI: 10.1594/ecr2017/C-1781
24. *Карнаушкина М.А., Струтынская А.Д., Бабак С.Л., Горбунова М.В.* КТ-признаки инфекционного бронхиолита. Руководство пользователя для клинициста. *Архивъ внутренней медицины.* 2021;11(2):85-93. [*Karnaushkina M.A., Strutynskaya A.D., Babak S.L., Gorbunova M.V.* Characteristics radiologic signs of infectious bronchiolitis. A practical approach for the general doctors. *The Russian Archives of Internal Medicine*. 2021;11(2):85–93. (In Russ.)]. DOI: 10.20514/2226-6704-2021-11-2-85-93
25. *Berniker A.V., Henry T.S.* Imaging of small airway diseases. *Radiol Clin North Am*. 2016;54(6):1165–81. DOI: 10.1016/j.rcl.2016.05.009

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