Letter

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COVID 19 Infection as a Cause of COPD Exacerbation in a Vaccinated Patient

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Letter

Coronavirus disease 2019 (COVID-19) is an infectious disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [1]. It is initially identified in Wuhan, China, in December 2019, and has spread to more than 215 countries causing a global pandemic [2]. Most patients diagnosed with COVID-19 have mild symptoms, while about 20% had severe or critically severe disease, including pneumonia, respiratory failure, septic shock and multi-organ failure [3]. Individuals with chronic medical conditions such as diabetes mellitus, chronic lung disease, and cardiovascular disease have an increased risk for COVID-19 severe form of disease [4]. Nearly all deaths have occurred in patients with significant underlying chronic diseases, including COPD and cardiovascular diseases [3]. According to a largest systematic review, COPD is a significant risk factor for hospitalization, ICU admission and mortality in patients with COVID-19 [1]. These can be explained by reduced lung function and upregulated expression of ACE2 (angiotensin-converting enzyme 2) in COPD patients and current smokers [3]. The spike protein of the virus binds to ACE2 during viral attachment to host cells and that viral entry is facilitated by transmembrane protease serine 2 (TMPRSS2) [5]. While the upregulation of ACE-2 may be useful in protecting the host against acute lung injury, chronically, this may predispose individuals to an increased risk of coronavirus infections. This may in part explain the increased risk of viral respiratory tract infection in active smokers and virus-related exacerbations in those with COPD [3]. Conversely, it was reported reduction in hospital admissions for COPD exacerbation during the COVID-19 pandemic [5]. Explanation could be that patients wear facial coverings, social distancing and pollutant emissions is reduced with following air quality improvement due to countries lockdown and shut down of industrial activities. Coronaviruses are among the respiratory viruses that trigger COPD exacerbations. However, to date COVID-19infection have not been reported in COPD exacerbations. Nonetheless, in COPD patients presenting with respiratory symptoms requiring changes in their maintenance medications would fulfil the definition of an exacerbationRT-PCR testing should be conducted [5].

Despite of governmental and public health measures, COVID-19 infections continue to spread, suggesting that effective vaccines and antiviral drugs are required to curtail disease and end the pandemic [2]. According to British Thoracic Society patients with severe COPD are considered Clinically Extremely Vulnerable and should receive COVID-19 vaccine [6]. A Pfizer's BioNTech Covid-19 vaccine (BNT162b2) two-dose regimen showed 95% protection against COVID-19 in persons 16 years of age or older [7]. In adults ≥65 years old, the vaccine efficacy rate was 94% [8]. A single dose of vaccine is around 80% effective at preventing hospital admission in people aged over 80, three to four weeks after the first dose. Also a single dose of the Pfizer vaccine led to 85% reduction in deaths from COVID-19 in people aged 70 and over [9]. It is also estimated that a single dose of mRNA vaccine provokes in COVID-19 recovered individuals a level of immunity that is comparable to that seen in infection naïve persons following a double dose regimen [10].

We present case of COPD exacerbation after BNT162b2 vaccine which has not been described in the available literature so far.

A 70-year old female patient was admitted at Institute for pulmonary diseases of Vojvodina in high dependency unit on 23rd

February 2021 due to COPD exacerbation and decompensated hypercapnic respiratory failure. She is ex-smoker 40 pack/year and stopped six years ago. Diagnosis of COPD was made in 2014. On her last ambulatory control in January 2020 spirometry indicated very severe obstructive disorder: FEV1 0.6 L, FEV1% 27 and FEV1/FVC 47. Meanwhile she did not have exacerbations and has regulary taken prescribed therapy: LABA/LAMA inhaler, slow realese aminophylline per os and SABA/SAMA as a reliver. Her comorbidities are: arterial hypertension and one year before diagnosed rheumatoid arthritis, treated with methotrexate and pronisone. Imunosupressiv therapy was stopped in January 2021. She recived 15th January first dose, and second dose of Pfizer vaccine on 5th February. About ten days after revaccination she felt gradually increasing dyspnea and headache, without fever. On the day of admission, an ambulance was called due to a change in mental status. At moment of hospitalizastion she was bradypsychic, dyspnoic, normocardial, normotensive with no signs of heart failure. Blood gas analysis revealed decompensated hypercapnic respiratory failure: pO_2 8.56 kP, pCO_2 11.47 kP and pH 7.21. The rapid SARS-CoV2 antigen test was positive. Chest X-ray showed attenuated bronchovascular pattern, elevated left hemidiaphragm, indirect signs of hyperinflation, without lung parenchyma consolidation. In complete blood count leucocytes (10.7 x 10^3/µL), erythrocytes (5.2 x 10^{12}/L) and thrombocytes (295 x 10^3/µL) were in referent range, lymphocytes were reduced (0.82 x 10^3/µL) and neutrophiles were elevated (9.1 x 10^3/µL). Markers of inflammation and cardiac insufficiency were normal: procalcitonin 0.05 ng/mL, CRP 3 mg/L, fibrinogen 3.44 g/L and NT-proBNP 141 pg/mL. D-dimer was 409 ng/mL FEU. Liver enzymes and bicarbonates were slightly elevated. Therapy was started with non-invasive mechanical ventilation, parenteral and inhalator bronchodilatator therapy, parenteral corticosteroids, antihypertensives, proton pump inhibitor and low molecular weight heparin in prophylactic doses. She did not recive antibiotics because the markers of inflammation were in referent range and she was afebrile. Intravenous aminophylline was administrated with frequent controls of theophylline concentration in the blood. After 24h she has been successfully weaned from non-invasive ventilation on an oxygen venturi mask. Discontinuation of oxygen therapy was unsuccessful. Considering that the patient was highly motivated for treatment at home, she was discharged on 1st March with an oxygen concentrator and reccomendation for ambulatory CT thorax and echocardiography.

Discussion and Conclusion

The leading cause of COPD exacerbations are respiratory infections which have long-term deleterious effects on patients' lungs [5]. Efforts to reduce infection rates are paramount to reducing repeat hospitalizations, chronic bacterial colonization, morbidity, mortality, and health care utilization cost in patients with COPD [5]. COVID-19 patients with COPD demonstrate increased odds of being hospitalized, requiring ICU admission, and mortality compared to COVID-19 patients who do not have COPD[1]. This indicates that COPD patients should be prioritized for immunization. Vaccines such as Pfizer's BNT162b2 tend to prevent severe forms of COVID-19 [6], but they can also have side effects such as pain and swelling at the injection site, tiredness, headache, muscle aches, chills, joint pain, and fever [7]. In our case patient had severe form of COPD exacerbation after vaccination, which required hospitalization and the non-invasive ventilation but without intubation and radiology verified pneumonia. Main limitations of our case are that we did not perform additional microbiological testing to exclude other possible causes of COPD exacerbation. Also although there were no signs of pneumonia on chest X-ray we did not perform CT of the thorax to exclude parenchimal leasons due to COVID 19 infection. Neverthless, this case of fully Pfizer's BNT162b2 vaccinated COPD patient with severe COPD exacerbation and positive rapid SARS-CoV-2 antigen test suggests that while vaccination may decrease the risk of severe respiratory distress and mortality in COPD patients, these patients may still be susceptible to COPD exacerbations caused by SARS-CoV-2 infections.

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