

CASE REPORT

COPD exacerbation caused by SARS-CoV-2: A Case Report from the Louisville COVID-19 Surveillance Program

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Abstract

A 53-year-old male with a history of chronic obstructive pulmonary disease (COPD) on home oxygen presented to the hospital with worsening shortness of breath plus cough. He was admitted to the intensive care unit for COPD exacerbation and respiratory failure. A routine evaluation was performed including a nasopharyngeal swab for a respiratory viral panel, which was negative. His symptoms improved over 48 hours at which time a surveillance test for SARS-CoV-2 returned as positive. After clinical improvement, he was discharged to home isolation.

Introduction

An important factor of the current COVID-19 pandemic is the large number of healthcare workers (HCWs) who acquire the infection. Initial reports from China indicated that approximately 30% of hospitalized patients with COVID-19 pneumonia were HCWs. [1] In an attempt to achieve early identification of hospitalized patients with COVID-19, we implemented a surveillance program in all hospitals in the Louisville area. The goal of the surveillance was to protect HCWs by identifying patients with no risk factors for COVID-19 who may be hospitalized with the disease without being placed into isolation. [2] Here we present a case of a patient hospitalized with an acute exacerbation of COPD who was identified as having COVID-19 due to our active surveillance program.

Case Report

A 53-year-old male with underlying chronic obstructive pulmonary disease (COPD) and chronic respiratory failure on home oxygen per nasal cannula during the day and non-invasive positive pressure ventilation (NIPPV) at night was referred to the emergency department (ED) from the pulmonology clinic for worsening dyspnea and hypoxia. The patient was recently treated as an outpatient with oral azithromycin and prednisone for a suspected COPD exacerbation five days prior to admission. The patient had symptoms of purulent phlegm with an oxygen saturation in the 70s at home. In the ED, the patient had a temperature of 97.7°F with a prolonged expiratory phase of breathing and wheezing as well as diminished breath sounds. He was noted to have acute on chronic hypoxic/hypercarbic respiratory failure and was admitted to the ICU for further management. His arterial blood gas showed a pH of 7.27, a partial pressure of carbon dioxide (pCO₂) of 73, a partial pressure of oxygen (pO₂) of 185, a bicarbonate of 32.6, a base excess of 3.2 on 60% fraction of inspired oxygen (FiO₂) revealing respiratory acidosis. A rapid influenza screen was negative. His chest x-ray revealed COPD changes and no acute infiltrate. (Figure 1)

After admission to the ICU, the patient was placed on NIPPV with improvement. Azithromycin and steroids were again initiated for an acute exacerbation of COPD. The patient was also given oseltamivir due to a history of exposure to a contact with influenza virus. He was stabilized in the ICU and was subsequently transferred to the medical floor within 24 hours of hospitalization. A respiratory viral/bacterial panel was nega-

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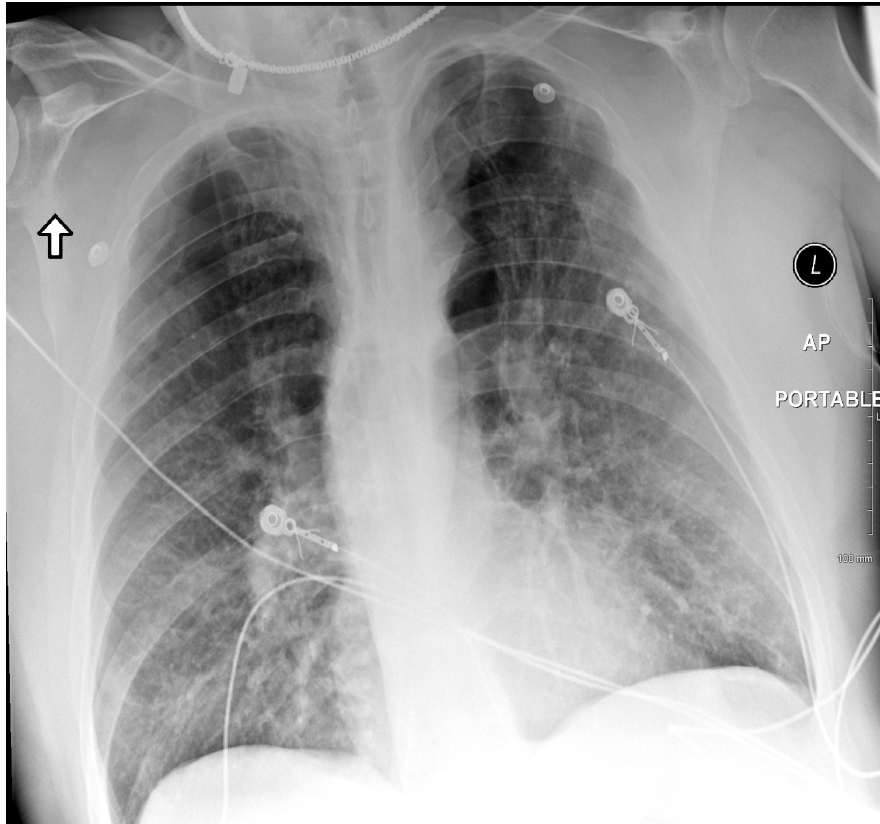


Figure 1. The chest radiograph of a patient with an exacerbation of chronic obstructive pulmonary disease who tested positive for SARS-CoV-2.

tive. The patient remained afebrile during his hospitalization. Surveillance testing for COVID-19, caused by the SARS-CoV-2, collected on the day of admission (two days earlier), and performed from left over specimen used to process a respiratory viral panel, was positive. To that point (early March 2020), no COVID-19 patients had been identified in the hospital, and he was never considered a ‘Centers for Disease Control and Prevention (CDC) person under investigation’ according to the definition at the time (which included international travel or exposure to a known case). Upon further questioning, the only place he had been around people other than his spouse was at a large indoor event a week prior to symptoms starting. He subsequently decided to leave against medical advice to home isolation.

Discussion

In an effort to protect patients and healthcare workers, as well as to know for whom PPE should actually be used, a surveillance study was performed in Louisville, KY. As one of 13 biosafety laboratories in the country, the University of Louisville was among the first to receive a sample of the virus from the CDC. With the virus, a real time PCR (Tecan EVO 100 with multichannel arm, Männedorf, Switzerland) was tested, and validated allowing Emergency Use Authorization to the laboratory by the US Food and Drug Administration (FDA). This report highlights that COPD exacerbations may be caused by SARS-CoV-2.

Approximately 30 % of patients with COPD exacerbations are due to viruses; influenza, parainfluenza, endemic coronavirus, rhinovirus, adenovirus and respiratory syncytial virus. [3] This report’s patient history identified one specific social activity, which permitted him to be exposed to COVID-19 implying that there was unknown community spread at the time. Pathophysiologically, his chronic lung disease made him more likely to be susceptible to COVID-19 than an average immunocompetent person like his wife who had the same exposure. Furthermore, COVID-19 is estimated to have an R0 between two and three [4], while influenza is approximately 1.3, reflecting the higher transmission of COVID-19. The combination of these factors supports his exacerbation of COPD by COVID-19. His improvement may have stemmed from the azithromycin he was given as it has been shown to be significantly associated with viral load reduction in COVID-19 patients. [5]

Some studies have explored a relationship between COVID-19 and COPD exacerbation. First, a study of 197 patients in China during the early period of the pandemic included 55 deaths (28%) – a proportion much higher than the overall pandemic mortality rate. [6] A total of 3% of the patients had COPD. Only 1% of those who survived had it, while 24% of those who died had it; ($P < 0.0001$). Surprisingly, a univariate analysis did not find COPD to be a significant risk factor for death in hospitalized patients.

Second, a meta-analysis of eight studies, including over 46,000 patients, found respiratory system disease to be one of the four most common comorbidities in COVID-19 patients; (2 ± 0 , 95% CI 1-3%). [7] Compared to the other comorbidities, it had the least heterogeneity within and between studies compared to hypertension, diabetes and cardiovascular diseases; meaning the studies in the meta-analysis were relatively similar regarding the frequency of severe versus non-severe patients with COPD.

Finally, a study of 788 patients with COVID-19 comparing epidemiological characteristics between older and younger patients (\geq or $<$ 60 years) found that only 2% of elderly patients had COPD while none of the younger patients had it. COPD was significantly higher among older adults compared to younger adults with COVID-19, but overall, it was not common as it ranged from 2-3%. [8]

Conclusion

In the present case report, a patient had a COPD exacerbation due to COVID-19. The test was performed for surveillance and not diagnostic purposes, emphasizing the ease of transmissibility, the relevance of surveillance testing, and the importance of understanding the epidemiology of this novel pathogen.

Appendix: Louisville COVID-19 Study Group

Available upon request.

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