International Journal of Science and Research (IJSR)
ISSN: 2319-7064
ResearchGate Impact Factor (2018): 0.28 | SJIF (2019): 7.583

Severe Clinical Manifestation of COVID-19 with Acute Exacerbation of COPD: A Case Report

I Wayan Angga Suamerta Putra[1], Wayan Wahyu Semara Putra[2], Ni Made Dwita Yaniswari[3]

[1]General Practicioner, Wangaya General Hospital, Denpasar, Bali

[2, 3]Pulmonologist, Wangaya General Hospital Denpasar, Bali

Abstract: ***A 48 years-old Indonesian male with history of COPD and daily inhaler presented to the emergency room with shortness of breath, productive cough, mucous thickening with white to yellow color, fever, and nausea. COVID-19 RT-PCR test was performed on this patient through surveillance testing and not as diagnostic purpose, the result was positive. Routine evaluation, medication and nasopharyngeal swab test was performed. The patient got discharged on day ten since he showed improvement, but the positive result of COVID-19 RT-PCR still remained and we suggested the patient to do self isolation for fourteen days after discharge. The patient finally got negative result after self isolation when he controlled himself to the hospital.***

Keywords: COVID-19, Chronic Obstructive Pulmonary Disease (COPD)

1. Introduction

COVID-19 pandemic has infected over 25,4 million people around the world and caused more than 851.134 deaths as of early August 2020 [1]. Several cases showed, COVID-19 is a respiratory and systemic illness that can lead to severe hypoxemia that need some ventilatory support in 15-20% of suspected and confirmed case [2].

Chronic Obstructive Pulmonary Disease (COPD) is a common, persistent and preventable dysfunction of the lung associated with limitation in airflow. COPD is a complex disease associated with abnormalities of the airway and or alveoli which is predominantly caused by exposure to noxious gases and particulates over a long period [3]. While before the pandemic happened, exacerbations of COPD is a major cause of morbidity, mortality, and poor health status. It is estimated that 70-80% of COPD exacerbations are triggered by viral or bacterial respiratory infections [4]. As we know what COVID-19 can cause to the respiratory function, this needs to be a cautions for patients with underlying disease of COPD [5].

Here we present a case report of 48 years old Indonesian male with acute exacerbation of COPD with hypoxemic respiratory failure which confirmed as COVID 19 patient due to our surveillance programme.

2. Case Report

48 years-old Indonesian male with history of COPD got referred from another hospital and came to the emergency room of Wangaya General Hospital Denpasar, Bali in June 2020. He came with shortness of breath that worsening since two days before admission. There were productive cough, mucous thickening with white to yellow color, fever, and nausea present since two days before admission. He had history of hypertension and smoking cigarrate since 20 years ago. He usually used fluticason and salmeterol for daily inhaler, but since the pandemic of COVID-19 outbreak, he didn't take the medicine for almost 3 months, and didn't see his pulmonologist to got himself checked because he felt fear to visit hospital. He got amlodipine 10 mg/day for his fhypertension, but he didn't take it routinely. The vital sign showed 39 ^{o}C temperature with 26 times/minute respiratory rate, heart rate 60 times/minutes, and 88% of SpO_2. Expirational wheeze occurred on superior region of chest with low pitch rhonchi presented. Blood examinations showed leukopenia and trombositopenia (WBC 3,81 $10^3/\pi L$ (neutrophyl 68.8%, lymphosyte 24.5%, monosyte 5.1%, eosinophyl 0.4%, basophyl 0.1%), hemoglobin 13.4 g/dl, platelet 127.000, hematocrit 41.8%). Blood gas analysis showed partially compensated metabolic acidosis with severe hypoxemia (pH 7.28, PO_2 36 mmHg, PCO_2 18 mmHg, HCO_3 8, SO_2 67%, actual base excess -18), the blood gas analysis performed every two days. The electrolyte test showed hyperkalemia (Kalium 6.2 mmol/L) Since there was hyperkalemia occurred on this patient, we did the renal function test which the result was Acute Renal Failure (serum creatinine 5.67 mg/dl and blood urea nitrogen 70.5 mmol/L) with no anemia presented. We also had performed coagulation test and the tes was normal (PT 11,7 second, INR 1.10 second, APTT 25,5), but presented high d-dimer test which was 3.793 ng/mL FEU. ECG was performed and there was sinus bradicardia and RVR pattern on lead III. Chest x-ray showed left and right hillar thickening with infiltrate presented on left and right parahiller area. The patient undergone the antibody SARS-COV2 test and the result was non-reactive in IgM and IgG. Since the patient lived at the red-zone area of COVID-19 and had respiratory sign, patient was entered to isolation room to undergone nasopharyngeal swab test for RT-PCR COVID-19 sample. The result was positive and he confirmed as COVID-19 patient.

Oxygen mask with reservoir bag 6 lpm was given since the patient entered the emergency room and showed 96 % SpO_2 after the oxygen therapy performed. Injection of terbutaline and metilprednisolone was given as initial therapy at emergency room since the patient presented exacerbation of COPD. After the initial therapy, the wheezing didn't disappear, we added aminophylline as initial therapy, followed by the maintenance. Levofloxacin was given too to treat the exacerbation. N-acetylsistein was given for mucolytic.

Based on the electrolyte test, then the patient showed hyperkalemia and got corrected.

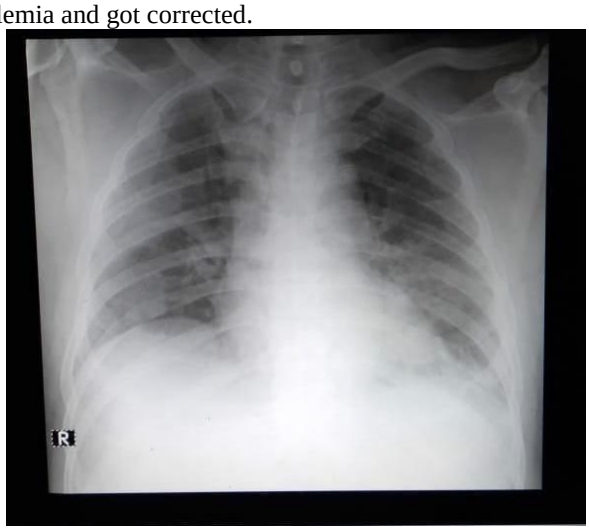

Figure 1: Chest x-ray with AP position showed bilateral bronchopneumonia

For the COVID-19, the patient was given osetalmivir, azithromycin, hydroxychloroquin orally and high dose of vitamin C. ECG patterns were monitored every two days to observed prolonged QT interval which is the side effect of hydroxychloroquin.

Sixth day after the patient got hospitalized, the oxygen was switched to nasal canule 4 lpm and the showed improvement of the oxygen saturation with 95% SpO_2. Another symptoms like fever, productive cough and nausea showed improvement. The patient got discharged on day ten and had 96% of oxygen saturation with no oxygen therapy needed. The RT-PCR after swab test of nasopharyngeal was still positive. But since the patient showed improvement, we adviced the patient to do self isolation for fourteen days after got discharged from hospital and perform nasopharyngeal swab test RT-PCR. He got negative result test of nasopharyngeal swab RT-PCR after he controlled himself to the hospital after discharged.

3. Discussion

COVID-19 that classified as SARS CoV-2 induced host immune response. SARS-CoV2-2 targets cells through the viral structural spike (S) protein that binds to the angiotensin-converting enzyme 2 (ACE-2) receptor. The serine protease type 2 transmembrane serine protease (TMPRSS2) in the host cell further promotes viral uptake by cleaving ACE2 and activating the SARS-CoV-2 S protein. In the early phase, the viral numbers can be detected high in the lower respiratory tract. Inflammatory signaling molecules are released by infected cells and alveolar macrophages in addition to recruited T lymphocytes, monocytes, and neutrophils. In the late stage, pulmonary edema can fill the alveolar spaces with hyaline membrane formation, compatible with early-phase acute respiratory distress syndrome [6].
In COPD patients may display the machinery required for SARS-CoV-2 cellular entry. SARS-CoV-2 contains an envelope spike protein that is used by the cellular serine protease TMPRSS2 to facilitate bonding of the virus with the cell's angiotensin-converting enzyme 2 (ACE-2) receptor and subsequent cell entry [7]. Leung et.al have recently demonstrated that in three separate cohorts with available gene expression profiles from bronchial epithelial cells, ACE-2 expression was significantly elevating in COPD patients compared to control subjects [8]. Current smokers also associated with higher ACE-2 expression compared with former and never smokers, an observation which has been validated by other groups in separate cohorts of lung tissue and airway epithelial samples [9] and supported by additional evidence linking ACE-2 expression with nicotine exposure [10]. ACE-2 expression alone hasn't been shown yet to influence the severity of disease. Leung et. al has concluded that COPD patients have increased risks of severe pneumonia because of increased expression of ACE-2 receptor in small airways that actually the target of SARS-CoV-2 to activate the SARS-CoV-2 S protein that facilitate the viral binding to host cell membrane [11].

The patient was given terbutaline and metilprednisolone as bronchodilator and anti-inflammation for exacerbation of COPD. Since the wheeze didn't disapearaminophylline was added as bronchodilator, followed by the maintenance. Levofloxacin was added as antibiotic for the exacerbation of COPD.For the COVID-19, the patient was given osetalmivir, azithromycin, hydroxychloroquin orally as antiviral and high dose of vitamin C as supportive treatment.

Some studies have explored a relationship between COVID-19 and COPD. COVID-19 was reported had a relationship with severity and mortality rates in COPD. Alqahtani et.al comparing former smoker, never smokers, and current smokers. And the result is current smokers were at greater risk of severe complications and higher mortality rate. Alqahtani et. al have concluded that COPD patients were at a higher risk (63%) to have more severe disease compared to patient without COPD (33,4%) [12]. An analysis of comorbidities study in 1590 COVID-19 patients in China that researched by Guan et.al found that COPD carried an odds ratio of 2.681 (95% CI 1.424–5.048; p=0.002) for ICU admission, mechanical ventilation or death, even after adjustment for age and smoking. 62,5% of COPD patients in thisresearch have severe cases, and have 25% death rate [13]. There's also a case report of COPD exacerbation which predicted caused by COVID-19 found by Arnold et.al [14]. A 53 years old male with history of COPD on home oxygen which come to the hospital with exacerbation sign. The patient was performed a surveillance COVID-19 test and the result was positive. Further research and investigation are needed to confirm that the relation between COVID-19 and COPD excacerbation.

There's a factor of this patient's exacerbation that he hadn't seen pulmonologist for 3 months already to maintain his COPD and didn't get his daily inhaler to maintain his COPD. In COPD patients, face to face visits or home visits with physicians are so important. Patients who may have presented exacerbation might choose to stay at home for fear of exposure during the COVID-19 pandemic. Vasilopoulou et. al have researched about tele-rehabilitation for COPD and the result for home based maintenance tele-rehabilitation is equally effective as hospital based to reduce the risk of acute exacerbations of COPD and lower the risk for emergency department visits [15]. But in Indonesia, it

can't be implemented yet, because the lack of internet and communication access for several areas in Indonesia.

4. Summary

This case report shows about the clinical manifestation between COPD exacerbation and COVID-19 coinfection of 48 years-old Indonesian male. The test was performed through surveillance testing and not as diagnostic purpose. COPD patients have a higher risk of severe clinical manifestation and mortality while there is COVID-19 infection. COPD patients should get themselves to be maintained for visiting or at least have a consultation with their physicians and continue their daily medication to lower the risk of acute exacerbation. Further research and investigation are needed to specifically prove the relation between COVID-19 and COPD exacerbation.

5. Author Contribution

All authors contributed equally.

6. Conflict of Interest

There is no conflict of interest in this case report.

7. Acknowledgement

The authors acknowledge and feeling thankful for the patient and patient's family, doctors, nurses, and all of the hospital superintendent.

References

[1] CoronavirusWorldometers.Date last accesed: August 27th 2020.
[2] Lippi G, Henry MB. Chronic Obstructive Pulmonary Disease is Associated with Severe Corona Virus Disease 2019 (COVID-19). Respiratory Medicine 167 (2020) 105941.
[3] Qureshi H, Sharafkhaneh A, Hanania N. Chronic Obstructive Pulmonary Disease Exacerbations : latest evidence and clinical implications. Ther Adv Chronic Dis. 2014, Vol 5(5) 212-227.
[4] Sethi S, Murphy T. 2008. Infection in the pathogenesis and course of chronic obstructive pulmonary disease. *N Engl J Med* 359: 2355–2365.
[5] Ejaz H, Alsrhani A, Zafar A. 2020. COVID-19 and comorbidities: Deleterious impact on infected patient. Journal of Infection and Public Health. JPH-1420.
[6] Wiersinga WJ, Rhodes A, Cheng A. Pathophysiology, Transmission, Diagnosis, and Treatment of Coronavirus Disease 2019 (COVID-19) A Review. JAMA, 2020, 324(8):782-793.
[7] Li W, Moore MJ, Vasilieva N, et al. Angiotensin-converting enzyme 2 is a functional receptor for the SARS coronavirus. Nature 2003; 426: 450–454.
[8] Leung JM, Yang CX, Tam A, et al. ACE-2 Expression in the small airway epithelia of smokers and COPD patients: implications for COVID-19. Eur Respir J 2020; 55: 2000688.
[9] Cai G, Bosse Y, Xiao F, et al. Tobacco smoking increases the lung gene expression of ACE2, the receptor of SARS-CoV-2. Am J Respir Crit Care Med 2020; 201: 1557–1559.
[10] Leung JM, Yang CX, Sin DD. COVID-19 and nicotine as a mediator of ACE-2. Eur Respir J 2020; 55: 2001261.
[11] Leung JM, Niikura M, et.al. COVID-19 and COPD. Eur Respir J 2020; 56: 2002108.
[12] Alqahtani J, Oyelade T, Aldhahir AM, et.al. Prevalence, Severity and Mortality associated with COPD and Smoking in patients with COVID-19: A Rapid Systematic Review and Meta-Analysis. Journal Pone.
[13] Guan WJ, Liang WH, Zhao Y, et al. Comorbidity and its impact on 1590 patients with Covid-19 in China: a nationwide analysis. Eur Respir J 2020; 55: 2000547.
[14] Arnold FW, Mahmood K. COPD exacerbation caused by SARS-CoV-2: A Case Report from the Louisville COVID-19 Surveillance Program. Louisville Journal of Respiratory Infections. doi :10.18297/jri/vol4/iss1/5
[15] Vasilopoulou M, Papaioannou A, Kaltsakas G. Home-based maintenance tele-rehabilitation reduces the risk for acute exacerbations of COPD, hospitalitations and emergency department visits. Eur Respir J 2017; 49: 1602129.