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MODESTUM

Case Report **OPEN ACCESS**

A case of acute respiratory distress syndrome: An unusual presentation of post-COVID-19

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ABSTRACT

Background: COVID-19 is an infectious condition caused by SARS-CoV-2. This virus does not only cause acute infection but also can cause long-term sequelae such as post-COVID-19, which involves multisystem especially respiratory system. However, it is still unclear about the pathophysiology, manifestation, risk factors, and treatment of post-COVID-19 and its sequelae.

Case Report: This case report highlights an unusual clinical case in a healthy young smoker, 25 days after a mild COVID-19 infection. He presented with severe pulmonary symptoms which fulfilled the criteria of acute respiratory distress syndrome without evidence of infective cause.

Conclusion: This case suggests smokers are susceptible to develop post-acute lung sequelae without prior severe COVID-19 infection or presence of comorbidities. This instance emphasizes the need for more studies looking into the relationship between post-infectious pulmonary sequelae and smoking. Hence, help further management of post-COVID-19 in smokers.

Keywords: post-COVID-19, acute respiratory distress syndrome, smoking

INTRODUCTION

Post-COVID-19 condition is sequelae of post-COVID-19 infections. The condition involves multisystem including the pulmonary system and it has different severity. There are many studies about COVID-19 infection. However, there is a limited report or literature about pulmonary sequelae post-acute COVID-19 such as acute respiratory distress syndrome (ARDS) especially in patients without a history of severe COVID-19 infections [1].

Moreover, the are many studies that discussed COVID-19 infection in the smoker, but limited studies discussed smoking effects on post-COVID-19 patients [2]. The pathogenesis of post-COVID-19 still remains unclear, but risk factors were identified for a patient with COVID-19 infection to develop post-COVID-19 sequalae such as the severity of COVID-19 infection, presence of comorbidities and history of hospitalization [1, 3]. This case illustrates how a young smoker without comorbidities with a history of mild COVID-19 infections and no hospitalization developed severe post-acute COVID-19 problems

CASE REPORT

A 32-year-old man who had no medical illness but was an active smoker (10 pack-years) presented to our center with high-grade fever for three days. It was associated with a productive cough and shortness of breath. Besides that, he also had a few episodes of vomiting and passing loose stool which was watery in consistency. He denied any recent traveling, jungle tracking, or water activity. No other family member had a similar presentation. He was infected with COVID-19 infection category 2, 25 days prior to current presentation with mild upper respiratory symptoms (cough and runny nose) and was self-quarantined at home. He had completed his COVID-19 vaccination before.

On initial examination, the patient was alert and conscious. However, he looked lethargic and dehydrated. His temperature was 38°C and he was tachypneic with a respiratory rate of 27 breaths per minute. His blood pressure was 115/70 mmHg and his pulse rate of 127 beats per minute. His saturation was 97 % under room air. His lung examination finding revealed crepitation bilateral lower zone.

Other examinations are unremarkable. Initial investigation full blood count showed infective feature with white cell count was 15.22×109/L, hemoglobin level was 15 g/dl, platelet count 284×109/L. A chest radiograph was done as shown in **Figure 1**.

The patient was then admitted to the medical ward with the impression of post-acute COVID-19 with superimposed bacterial pneumonia. In the ward, his condition deteriorated rapidly as hemodynamically nonresponsive to fluid therapy and needing for inotropic support. Further investigation results revealed acute kidney injury and transaminitis as shown in **Table 1**.

Table 1. Investigation results

Investigation	At emergency department	Day 1	Day 10	Day 15	Day of discharge	One month after discharge
Haemoglobin (g/dl)	15	14.1	11.3	10.1	9.27	
WBC ($\times10^9$/L)	15.22	28.84	21.8	14.7	9.8	
Platelet ($\times10^9$/L)	284	249	487	674	687	
CRP (mg/dl)		>200	30	71	31	
Creatinine (µmol/L)	106	121	71	63	59	62
Urea (mmol/L)	3.8	6.2	9.8	7.4	5.2	2.3
Sodium (mmol/L)	134	139	145	140	134	139
Potassium(mmol/L)	3.5	3.9	3.5	4.7	4.2	4.0
AST (IU/L)		109	120	112	70	18
ALT (IU/L)		258	227	193	190	21
ALP (IU/L)		218	265	266	263	126
Albumin (g/L)		35	30	30	30	
PT (seconds)		16.10	15.10	14	13.80	
APTT (seconds)		45	30.70	34	31.70	
INR		1.19	1.12	1.03	1.02	

Note. APTT: Activated partial thromboplastin time; ALP: Alkaline phosphatase; ALT: Alkaline transaminase; AST: Aspartate transaminase; CRP: C-reactive protein; INR: International normalized ratio; LDH: Lactate dehydrogenase; LFT;: Liver function test; PT: prothrombin time; RP: renal profile; & WBC: White blood count

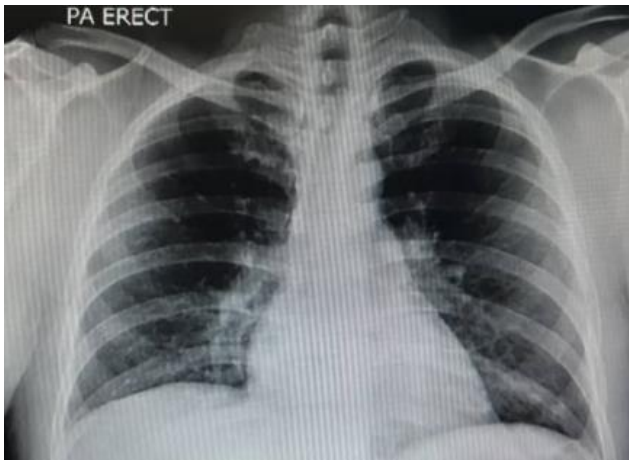

Figure 1. Chest radiograph showing bilateral lower zone haziness

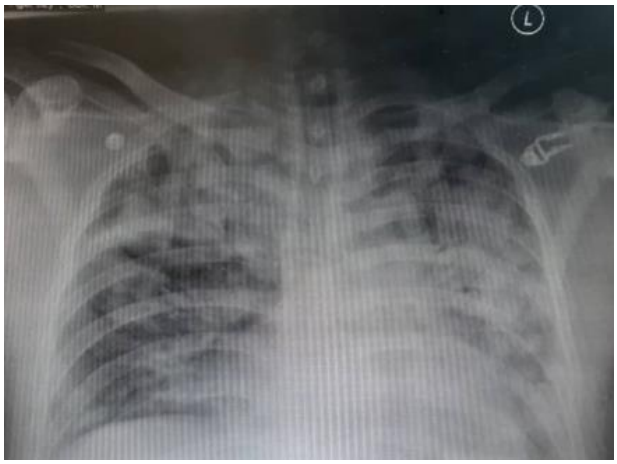

Figure 2. Chest radiograph on day 2 of admission showing bilateral diffuse infiltrate

The patient was treated as post-acute COVID-19 with septicemic shock secondary to bacterial pneumonia with multiorgan failure. The patient was given Ceftriaxone at first, but due to the severity of the illness, the antibiotic was changed to Meropenem. The patient also started with glucocorticoid therapy (hydrocortisone 100 mg TDS) as a treatment for post-COVID-19 with severe RDS. All initial infective workup was negative including sputum and blood culture and sensitivity and other infection screening such as leptospira serology, hepatitis B (HBsAg) and hepatitis C (anti HCV), blood film, and parasite for malaria, mycobacterium culture and sensitivity, fungal culture, and sensitivity. Besides all that, the COVID-19 PCR test was also sent to rule out COVID-19 re-infection; however, the result was also negative.

His respiratory condition deteriorated over time as his oxygen saturation did not improve despite high flow mask oxygen therapy. Arterial blood gas showed type 1 respiratory failure. Initially, he was started on a non-invasive ventilator (NIV) with a FiO2 of 0.6; however, unable to achieve good saturation, the patient then was intubated with a ventilator setting of FiO2 0.7 with PEEP 10. Repeated chest radiograph showed worsening as in **Figure 2**.

The patient also had a computed tomography pulmonary angiogram (CTPA) to rule out pulmonary embolism as he persistently had tachycardia with a pulse rate ranging from 120-to 140 beats per minute. Fortunately, his CTPA showed no evidence of pulmonary embolism. He then had high resolution computed tomography (HRCT), which showed typical features of ARDS as in **Figure 3**.

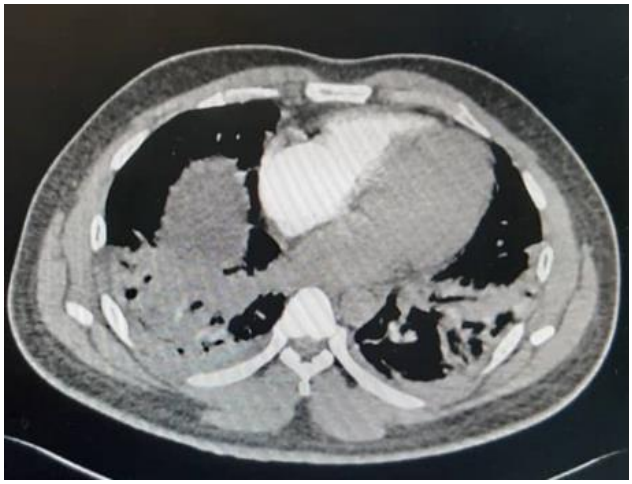

Figure 3. HRCT showing ground-glass appearance periphery in bilateral lung parenchyma with superimposed infection

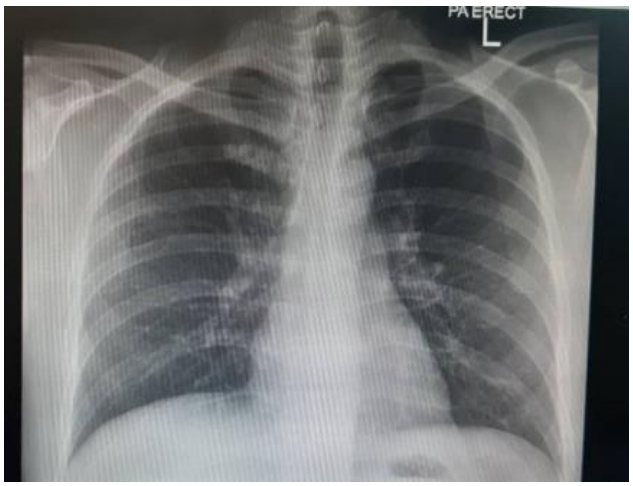

Figure 4. Chest radiograph one-month post-discharge from the ward showing clear lung field

His condition improved after 10 days of admission. Unfortunately, as prolong stayed in Hospital, he then developed Hospital-acquired pneumonia. Besides that, his condition was complicated with two episodes of upper gastrointestinal bleeding secondary to the gastric ulcer which had improved further with pantoprazole infusion. His condition had improved further after almost one month, and he was discharged well with a follow-up in a medical clinic in one month. Repeated chest radiograph after one month showed improvement as shown in **Figure 4**.

DISCUSSION

COVID-19 is caused by the SARS-CoV-2 virus. It does not only cause a wide spectrum of illnesses but also can result in long-term complications which are post-COVID-19 condition. There are a few terminologies to describe this condition such as post-acute COVID-19 syndrome, long COVID-19, chronic COVID-19 syndrome, or post-COVID-19 syndrome [3]. A post-acute COVID-19 syndrome is defined as a condition that lasts more than ranging three weeks to four weeks after the beginning of the first symptoms [4]. Post-acute COVID-19 syndrome involves multiple systems, including the respiratory system, and it affects roughly 10% of persons following COVID-19 infection [4]. The pathogenesis of post-COVID 19 is believed that immune dysregulation in response to disease progression and pathological inflammation after COVID-19 infection. It can potentially harm to a variety of organs, especially the respiratory system [5, 6].

The sequelae of COVID-19 infection can occur whether after mild, moderate, or severe COVID-19 infection. Some patients may experience mild post-acute COVID-19 symptoms such as cough, mild fever, and fatigue while others may have severe post-COVID-19 consequences such as thromboembolic condition and pulmonary complications [1, 4]. The pulmonary sequelae of post-acute COVID-19 are defined as persistent symptoms or delayed long-term caused by COVID-19 infection after four weeks from the onset of symptoms [1]. The majority of the patients with radiological abnormalities have coexisting symptoms such as shortness of breath with or without cough. On other hand, some patients have symptoms suggestive of pulmonary involvement without radiological abnormalities and some of them have imaging abnormalities without any symptoms. One radiological finding of post-acute COVID-19 of lung disease is ground glass appearance. It has been reported that pulmonary changes occurred as early as two weeks post-acute COVID-19 infection [7]. A similar chest radiograph presentation was seen in the patient.

ARDS is described as an acute onset of hypoxemia defined as the arterial partial pressure of oxygen to fraction of inspired oxygen (PaO2/FiO2) 200 mmHg with bilateral infiltrate on chest radiograph [7]. It causes direct injury to the lungs such as infection or indirect lung injury by the inflammatory process [7]. It is presumed that ARDS in this patient was caused by dysregulation of the inflammatory process post-COVID-19 since there is no evidence of infection found in this patient. Besides that, he responded well to steroid therapy indicates underlying inflammation of his condition.

There are a few factors that have been described to increase the severity of post-acute COVID-19 infection such as older age, longer hospital stays, need for mechanical ventilation, and presence of comorbidities [1, 3]. Although smoking is not one predictor cause of severe post-acute COVID-19, smoking increases the risk of lung injury, which causes chronic inflammation and functional abnormalities in smokers [8, 9]. Besides that, smoking also can cause lung fibrosis which then lead to lung sequalae of post-acute COVID-19 [10]. Hence, there is possibility of smoker to develop severe pulmonary sequalae. However, there is a limited study on this. Most of the studies emphasize on the effect of smoking on COVID-19 infection [2]. In this case, the patient has had only mild COVID-19 infection without significant risk factors to develop severe post lung sequelae such as ARDS as he is young without comorbidities, no history of intubation or hospital admission. There is no other explanation for his severe post-acute COVID-19 except the history of smoking.

As is known, many kinds of literature described smoking as an increased risk for the development of severe COVID-19 infection, increased risk of admission to ICU, and mortality in COVID-19 patients [11]. However, there are limited data that described the prevalence of smoking and lung sequelae post-acute COVID-19.

CONCLUSIONS

In conclusion, severe post-COVID-19 sequalae can occurs in a young healthy smoker after having mild COVID-19 infection and was only home quarantined. The possibility of having chronic inflammation because of smoking habit can be risk factor for patient with mild COVID-19 infections developed severe pulmonary sequelae post-COVID-19 symptoms. However, more studies are needed to evaluate the relationship between smoking and its effect on post-COVID-19 complications. Hence, it is important for medical doctor to advice and increase awareness to quit smoking among the public, especially smokers with a history of COVID-19 infections.

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Data sharing statement: Data supporting the findings and conclusions are available upon request from the corresponding author.

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