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Coronavirus disease 19 (covid-19) affects the occurrence of acute heart attacks:case report

I Made Bayu Puradipa[1], Cyndiana Widia Dewi Sinardja[2]

[1]Faculty of Medicine, Udayana University, Bali, Indonesia

[2]Department of Cardiology and Vascular Medicine Prima Medika Hospital, Denpasar, Bali

Abstract

Global Pandemic of COVID-19 is a viral infection caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) that primary target in respiratory system and spread rapidly around the world through droplets and direct contact. On the other side, COVID-19 affects to damage the myocardial causing acute heart attacks including Unstable Angina (UAP), Non-ST Elevation Myocardial Infraction (NSTEMI) or ST Elevation Myocardial Infarction (STEMI) and still represents a major cause of morbidity and mortality worldwide that affects about 15.5 million in the United States. The common risk factors of ACS are male sex, family history of early myocardial infarction (55 years old), hypertension, diabetes, hyperlipidemia, smoking, sedentary lifestyle, and obesity. However, in this case report the viral infection is the only main cause of ACS

Case Description: A male 65 years old came to ER complained shortness of breath with no comobid. The ECG showed sinus rhythm, right bundle branch block, lateral ischemia. The COVID-19 RT-PCR resulted was positive with elevation of cardiac enzymes.

Discussion: ACE2 mediated SARS-CoV-2 enters into the lung alveolar epithelial cell and host cell. ACE2 is also spread widely in human heart, and vessels. Infection of endothelial and pericytes could lead to microvascular and microvascular dysfunction. Cytokine storm, increasing interleukin (IL)-6, IL-7, IL-22, and CXCL10 may leading to atherosclerosis plaque instability or rupture and contributing to development of acute coronary events.

Conclusion**:** COVID-19 can lead to NSTEMI. Although cardiac biomarker was high, NSTEMI need further examination including coronary angiography.

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*Corresponding Author
Name: I Made Bayu Puradipa
Email: puradipa@gmail.com

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Introduction

Corona Virus Disease 2019 (Covid19) is a disease that infected respiration system caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV2). Covid 19 was found in Wuhan, China in December 2019 and speared rapidly around the world. Clinical manifestations of patient who exposed with this SARS-CoV2 are fever, cough, and shortness of breath that appear within 2 to 14 days after exposure, and then usually become pneumonia. Although respiratory symptom is the most frequent clinical manifestation of Covid-19, but many patients also have cardiovascular disease (CVD), while other patients with underlying CVD might have an increased risk of death [1,2,3,4].

Acute coronary syndromes (ACS) are a pathological condition associates with myocardial infarct or ischemia because of reduction in coronary blood flow. The ACS clinical spectrum include myocardial infarction with ST-Elevation (STEMI), which reflect an acute total occlusion of coronary artery, infarction without ST-Elevation (NSTEMI), and unstable angina pectoris (UAP). Type 1 myocardial infarction is the myocardial infarction due to primary coronary event such as plaque erosion and/or rupture, fissuring, dissection. Meanwhile, the other conditions related to the mismatch between oxygen supply and demand, such as respiratory failure (predominantly hypoxemia) and infectious disease (particularly sepsis), may induce or lead to myocardial injury or to type 2 MI [1,5].

Angiotensin-converting enzyme 2 (ACE2) has a role in Renin Angiotensin System (RAS) by converting angiotensin (Ang) II to Ang-(1–7), and Ang I to Ang-(1–9). However, ACE2 also has a role as the coronavirus receptor in COVID-19. SARS-CoV-2 binding ACE2 to mediate entry into lung alveolar epithelial cells. Other than in the lungs, ACE2 is also spread widely in human heart, and vessels, is the main key of RAS that important in pathophysiology of CVD [3,6].

Case Illustration

A male 65 years old came to emergency room (ER) with chief complain of shortness of breath for two days before came to ER. The shortness of breath appears continuously and does not improve with position changing. Patient also complained chest pain for one day ago. The chest pain felt like squeezing and radiating to shoulder Patient also felt dizzy, nausea, and cough. Patient also had fever in the past 1 week before came to ER. History of cardiovascular disease, hypertension, type 2 diabetes melitus (T2DM), kidney disease was dined.

Physical examination found compos mentis with blood pressure 129/62 mmHg, pulse rate 95 beats/minutes, body temperature 36°C, respiration rate 24 times/minutes with 98% of oxygen saturation via room air. Jugular vein pressure (JVP) PR±2cm. From thorax auscultation was S1S2 single, regular, no murmur, no wheezing but rhonchi on the right lung. Warm and no edema.

Laboratory test found erythrocyte (RBC): $3{,}73.10^6$/uL, hemoglobin (HGB): 11,8g/dL, hematocrit (HCT): 31,7%, platelet (PLT): 276.10^3/uL, leucocyte (WBC): $16{,}24.10^3$/uL, neutrophil: $12{,}51.10^3$/uL, lymphocyte: $2{,}36.10^3$/uL with neutrophil and lymphocyte ratio (NLR): 5,32. Blood chemistry analyzed troponin I: 0,22ug/L, creatinine (SC): 1,02mg/dL, SGOT: 21U/L, SGPT: 19U/L, anti-SARS-CoV-2 IgG reactive. Thorax photo (Thorax PA) showed infiltrate in the left and right pericardial with the conclusion was pneumonia bilateral (Figure 1). ECG showed sinus rhythm, 83 beats/minutes, right bundle branch block, lateral ischemia (Figure 2).

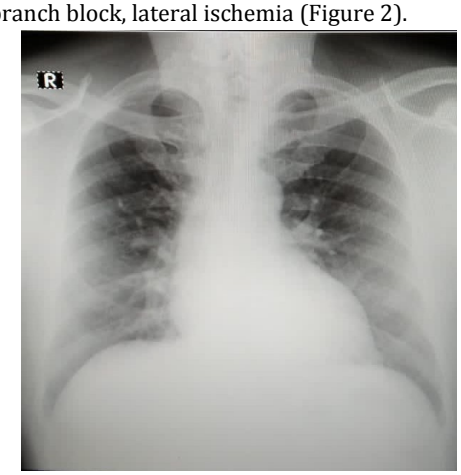

Figure 1 Thorax X-Ray

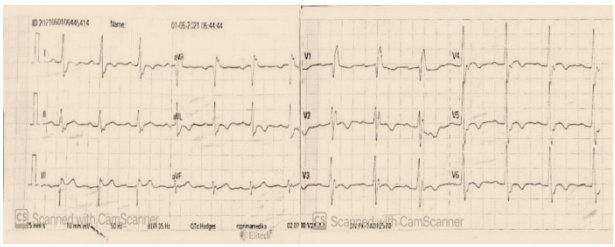

Figure 2. ECG

Patient diagnosed with Pneumonia Bilateral ec. Suspect Covid-19 and Non-ST Elevation Myocardial Infarction (NSTEMI). Patient treated with NaCl 0,9% 16dpm, O_2 3lpm via nasal canul, Aspilet loading 160mg followed by 80mg once daily orally, Clopidogrel loading 160mg followed by 80mg once daily orally, Fondaparinux subcutaneous injection 2,5cc once daily, Atorvastatin 40mg once daily orally, Nitroglycerin 2,5mg twice daily orally, Levofloxacin intravenous injection 750mg once daily, Azithromycin 500mg once daily orally, Lansoprazole 30mg twice daily orally, and Sucralfate syrup 15cc three times daily orally.

After one day of treatment, the RT-PCR result was positive COVID-19. Then, the patient was diagnosed with Confirmed COVID-19 with Moderate Symptom (Pneumonia Bilateral) and NSTEMI. Patient was given additional therapy, Acetylcysteine 600mg once daily orally, Vitamins D3 1000IU twice daily orally. After three days of being threated in isolation ward, the D-Dimmer result in this patient is 100ng/mL. After nine days of treatment in the isolation ward, the chest pain resolved and the shortness of breath has gone. Patient was discharged after negative SARS CoV-2 RT PCR.

Discussion

We report a case of male 65 years old with COVID-19 (Moderate Symptom) and NSTEMI without history of CVD, hypertension, T2DM, and kidney disease. Angiotensin-converting enzyme 2 (ACE2) has a role in Renin Angiotensin System (RAS) by converting angiotensin (Ang) II to Ang-(1–7), and Ang I to Ang-(1–9). However, ACE2 also has a role as the coronavirus receptor in COVID-19. SARS-CoV-2 binding ACE2 to mediate entry into lung alveolar epithelial cells [3,6]. In this case patient had fever, shortness of breath, and cough at the beginning with rhonchi on the right lung 5,32 of NLR, infiltrate in the right pericardial with the conclusion was pneumonia pericardial of right lung from thorax x-ray, and SARS-CoV-2 positive from RT-PCR. The ACE2 disrupted by SARS-CoV-2 is the pathogenic role in severe lung injury and respiratory failure in COVID-19.

Other than in the lungs, ACE2 is also spread widely in human heart, and vessels, is the main key of RAS that important in pathophysiology of CVD. COVID-19 is primarily a respiratory disease, but many patients also have CVD, including hypertension, acute cardiac injury and myocarditis. SARS-CoV-2 mediated by ACE2 to enter host cell including type-2 pneumocytes, macrophage, cardiac myocytes, endothelial cell, and pericytes. Infection of endothelial and pericytes could lead to microvascular and microvascular dysfunction. Cytokine storm, increasing interleukin (IL)-6, IL-7, IL-22, and CXCL10 may leading to atherosclerosis plaque instability or rupture in COVID-19. This condition contributing to development of acute coronary events (type 1 MI) [1,3,6]. The patient had a typical chest pain for one day before came to ER, without S-T segment elevation in ECG, but elevated cardiac biomarker Troponin I. Although the clinical manifestations of COVID-19 are dominated by respiratory symptoms, evidence of myocardial injury was recognized in early cases in China. Huang et al. first reported a prevalence of acute myocardial injury of 12% as a major complication among 41 hospitalized patients infected with SARS-CoV-2. Zhou et al., in a cohort retrospective report 33 (17%) of 191 patients admitted with SARS-Cov-2 pneumonia, diagnosed acute myocardial injury. Some of them may resemble the ones identified for other respiratory infectious agents, such a pro-inflammatory state and a cytokine storm (which could cause plaque instability), or a prothrombotic state which is type 1 MI and hypoxemia-related damage due to acute respiratory failure which is type 2 MI [1,5].

Venous thromboembolism (VTE) is the other concern in patients with COVID-19 hospitalized in medical wards even under thromboprophylaxis. Artifoni M, et al. D-dimer < 1,0ug/mL has an excellent negative predictive value for VTE, and thromboembolic event is strikingly high in patients with D-dimer > 3,0 ug/mL [7]. Choi J et al. divided into three levels of D-dimer that stratified patients into low-probability (< 1000 ng/mL), intermediate-probability (1000–7500 ng/mL), and high-probability groups (> 7500 ng/mL) [8]. In this patient the d-dimer result was 100ng/ml, it's mean this patient had a low-probability of VTE and had been treated with anticoagulant Fondaparinux subcutaneous injection 2,5cc once daily due to NSTEMI.

Important investigation after discharge and quarantine in 14 days are echocardiography and coronary angiography. Echocardiography to see the abnormalities of myocardium, ejection fraction (EF), and the other condition like valve disease. The Coronary angiography for evaluate the anatomy of coronary vessel.

Conclusion

ACE2 mediated SARS-CoV-2 enters into the lung alveolar epithelial cell and host cell. Other than in the lungs, ACE2 is speared widely in human heart, and vessels, is the main key of RAS that important in pathophysiology of CVD. Cytokine storm, increasing interleukin (IL)-6, IL-7, IL-22, and CXCL10 may leading to atherosclerosis plaque instability or rupture and contributing to development of acute coronary events (type 1 MI). Important investigation after discharge are echocardiography and coronary angiography.

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