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COVID-19-Associated ST Elevated Myocardial Infarction with Atrial Fibrillation: A Case Report

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ABSTRACT

Coronavirus disease 2019 (COVID-19) displays various clinical manifestations, and mounting evidence suggests that COVID-19 has extrapulmonary and cardiovascular involvement. Although cardiac effects are less prevalent in COVID-19 than pulmonary manifestations, understanding potential cardiac issues with COVID-19 is critical for risk stratification and improving outcomes. We report a male patient aged 56 years who presented with sudden onset of chest pain and dyspnea. The patient has a history of both hypertension and stroke. He has never traveled abroad or had previous contact with a COVID-19-positive patient. Physical examination demonstrates tachypnoea, desaturation, increased JVP, bronchial breath sounds, and rhonchi on the right basal lung. Inflammatory markers, liver function, and CKMB levels were all elevated in the laboratories. . Infiltrates and cardiomegaly were observed on a chest X-ray. Sinus tachycardia, irregular rhythm, and ST elevation on lead V1-V5 were detected on the electrocardiogram. Our patient was diagnosed with COVID-19 critical degree with STEMI and atrial fibrillation. Increased inflammatory cytokines caused by COVID-19 and a history of cardiovascular disease may play a crucial role in cardiac injury. The patient did not undergo reperfusion therapy, considering the late arrival to the hospital, and was managed with anticoagulant therapy. The patient responded well to therapy. After 15 days of treatment in an isolation room and two weeks in a non-isolation room, the patient exhibited clinical improvement and could be treated as an outpatient case. Patients with preexisting cardiovascular conditions are more likely to contract COVID-19, which significantly impacts the disease's course, treatment, and prognosis.

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Introduction

In December 2019, Wuhan, Hubei Province, China reported the first SARS-CoV-2-related pneumonia case. The WHO declared a COVID-19 pandemic within three months due to the virus's global spread (1). Typical manifestations of a COVID-19 infection consist of acute respiratory disorders, namely pyrexia, coughing, and dyspnea. On average, the incubation period lasts for 5-6 days, while the maximum is observed to be 14 days. Severe symptoms of COVID-19 have been associated with the development of pneumonia, acute respiratory distress syndrome, renal insufficiency, and mortality (2).

Although China is the first country to spread COVID-19, the disease has spread almost worldwide. That is why several prospective and retrospective studies have been directed to describe COVID-19 and its complications. Cardiovascular diseases have been identified as a risk factor that exacerbates the morbidity and mortality rates of individuals infected with COVID-19. Furthermore, infected patients who exhibit cardiovascular risk factors are correlated with an unfavorable prognosis. Infection with SARS-CoV-2 is associated with a heightened inflammatory response that can cause cardiac arrhythmias, myocarditis, and inflammation in the vascular system that can cause heart damage (3,4).

Coronary heart disease (CHD) contributes as a cause of death in 1/3 of patients aged ≥ 35 years, although the incidence has decreased in the last four decades, which is due to good treatment in CHD patients. Myocardial infarction (MI) patients present with either ST-elevation myocardial infarction (STEMI) or non-ST elevation acute coronary syndrome (ACS). According to several studies conducted in China, patients with history of cardiovascular diseases become more vulnerable to fatal infections of SARS-CoV-2. It has been reported that SARS-CoV-2 may interfere with the heart, causing diverse clinical manifestations, including infarction of the myocardium, myocarditis, ACS, arrhythmia, heart failure, and cardiogenic shock. A study conducted in China revealed a 17% occurrence of cardiac arrhythmias among treated patients. Individuals diagnosed with COVID-19 in the intensive care unit (ICU) demonstrated a higher rate of arrhythmia (44%). Nevertheless, the type and consequences of arrhythmias in this population have not been fully understood (5).

Furthermore, Guo et al. reported that 7% of hospitalized patients in China experienced ventricular tachycardia (VT)/ventricular fibrillation (VF) during hospitalization in another observational study involving 187 hospitalized patients. Other research studies from Italy and New York indicate a connection of COVID-19 to increased heart attacks outside hospitals. These results strengthen the conclusion that infection with SARS-CoV2 and secondary cardiac damage could raise the likelihood of arrhythmia (6). The following report details the clinical presentation and management of a male patient, aged 56 years, who recovered from a critical COVID-19 infection accompanied by ST-elevation myocardial infarction (STEMI) and atrial fibrillation.

Case Report

A 56-year-old smoker came with seven days of shortness of breath that worsened two days prior to admission to the Emergency Room (ER) at Ulin Hospital Banjarmasin, accompanied by typical chest discomfort. One day prior, the patient complained of fever and vomiting. The patient had a history of stroke four years ago, resulting in left limb weakness and hypertension, for which he takes amlodipine 10 mg daily. He has never traveled abroad or had any previous contact with COVID-19-positive patients. The patient was conscious and had a blood pressure of 120/70 mmHg, an irregular heart rate of 120 beats per minute, and a breathing rate of 26 breaths per minute with a saturation level of oxygen of 92% room air, which raised to 98% with non-rebreathing mask (NRM) oxygen supplementation of 10 liters per minute, and a temperature of 36.6^{o}C. Increased jugular venous pressure (JVP) 5+4 cmH2O was identified with normal heart sounds. Bronchial breath sounds, and rhonchi were heard on the right basal lung.

The initial laboratory data were as follows: increased white blood cells of 18,500/µL (high neutrophils at 82% and low lymphocytes at 7%), neutrophil-lymphocyte

ratio (NLR) of 10.71, and absolute lymphocyte count (ALC) of 1400; increased lactate dehydrogenase (LDH) 3453 U/L and C-reactive protein (CRP) 48 mg/dl; abnormalities in liver function with SGOT of 386 U/L and SGPT 68 U/L and elevated level Creatine kinase-MB (CKMB) of 153.82 U/L; and low potassium level of 2.73 meq/L. Blood gas analysis demonstrated pH 7.547, pCO2 19.5, pO2 77, HCO3 17.2, BE -6.0, SpO2 98%, PaO2/FiO2 ratio 208 with oxygen supplementation 4 liters/minute. Figure 1A exhibits infiltrates in the peripheral region of the right pulmonary basal with a CTR ≥ 55%. Electrocardiography (Figure 2A) showed sinus tachycardia, an irregular rhythm, and ST elevation on leads V1-V5. The diagnosis of COVID-19 was further verified by a nasopharyngeal swab real-time reverse transcriptase-polymerase chain reaction (RT-PCR) assay. Patients were administered a 320 mg loading dose of acetylsalicylic acid, subsequent to 80 mg once daily, and 300 mg of clopidogrel, continued by 75 mg once daily, enoxaparin 0.6cc per 24 hours subcutaneously, isosorbide dinitrate (ISDN) 5 mg three times a day, bisoprolol 5 mg and atorvastatin 40 mg once daily, potassium chloride 600 mg three times daily, remdesivir as an antiviral, steroid, antibiotic, hepatoprotection, and multivitamin. The patient's clinical condition deteriorated on day 6 (12 days after the onset of symptoms), with worsening dyspnea, bilateral air space opacity on radiology (Figure 1B), and increased CRP to 58 mg/dL. Antibiotics were escalated to meropenem, then the patient improved gradually, and his oxygen requirement decreased. On day 15 of hospitalization (23 days after the onset of symptoms), the RT-PCR evaluation went negative. The patient was discharged from the isolation room to a non-isolation room for continuous treatment. Due to limited resources, a bedside transthoracic echocardiography can be performed later in a non-isolation room (Figure 3). It revealed dilatation of the left ventricle (LV) with markedly impaired systolic function and a predicted left ventricular ejection fraction (LVEF) of 27%. After almost two weeks in the non-isolation room, the patient was no longer hypoxic, with a saturation of 97% on room air and improvement on follow-up of Chest X-ray (Figures 1C and 1D) and ECG (Figure 2B). The patient was discharged home.

Discussion

Respiratory symptoms dominate most of COVID-19 cases, but cardiovascular diseases and their complications are associated with higher patient mortality and morbidity and are frequently occured following COVID-19 infections. Patients with COVID-19 who have a previous diagnosis of cardiovascular disease are at higher risk for morbidity and mortality from myocardial damage, myocarditis, congestive heart failure (CHF), thromboembolism, and arrhythmia (7,8). Acute myocardial infarction occurs in advanced, moderate to critical stages in COVID-19 patients (shown in Figure 4) and is associated with a worse prognosis (9).

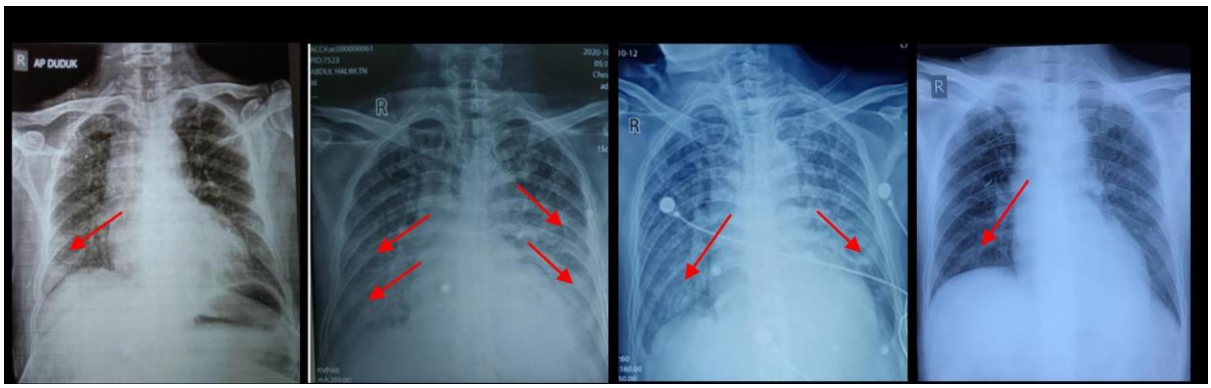

Figure 1. Chest X Rays **(A)** First admission: infiltrate (arrows) in the peripheral area of the right pulmonary basal with CTR > 55%, **(B)** Infiltrate (double arrows) bilateral in both lungs 6th days hospitalization, and **(C)** and **(D)** infiltrate (arrow) begin disappeared on 13th and 21st days hospitalization.

Our case showed a patient with previous diagnoses of hypertension and stroke who experienced pneumonia with cardiac manifestations, including shortness of breath, chest pain, fever, and vomiting. This patient's clinical classification of COVID-19 is critical degree based on his clinical symptoms and signs. Diagnosis of anterior STEMI was made based on a classical picture of chest pain which patient described as a feeling like being crushed by a heavy object and lasted approximately 20 minutes with the elevation of the ST segment > 2mm in the V1-V5 (anterior STEMI) with stratification of the risk of Killip 1 (no sign of heart failure). This patient had thrombolysis in myocardial infarction (TIMI) with a score of 5, which showed a high risk of cardiovascular events of 41%. TIMI has been validated as a predictor of death within 30 days and one year on a broad spectrum of acute coronary syndrome, including STEMI. The patient also had a high CKMB of 153.82 U/L. CKMB is a sign of necrosis of the heart myocytes and is a marker for diagnosing myocardial infarction besides troponin I/T (we cannot examine troponin due to limited resources).

The incidence of STEMI in these patients can be caused by atherosclerosis and the COVID-19 infection itself. Patients with coronary artery disease are susceptible to coronary rupture of plaque due to viral-induced systemic inflammation. However, it is known from the anamnesis that the patient only started experiencing chest pain and dyspnea one day prior to visit the emergency room. The patient exhibited atherosclerosis risk factors, including smoking, hypertension, and a history of a stroke four years ago. Patients with existing cardiovascular disease are particularly vulnerable to contracting COVID-19 and suffer from more severe diseases and poorer medical outcomes, according to recent COVID-19 research (10). Angiotensin-converting enzyme 2 (ACE2) may play an integral part in the mechanism of acute myocardial injury caused by COVID-19. SARS-CoV-2 penetrates the cells by adhering to the ACE2 receptor found in heart, lung, endothelial, and immune cells. Recent research has shown that ACE2 and various SARS-CoV-2 entry mediators, such as cathepsin B and cathepsin L, are particularly abundant in cardiomyocytes, which may

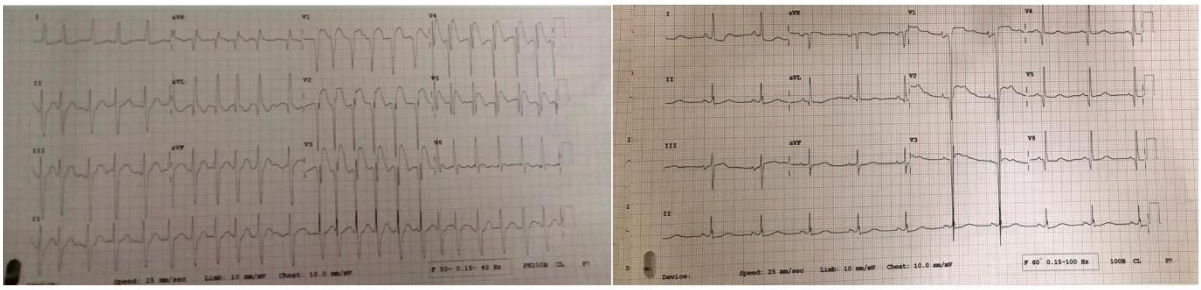

Figure 2. Electrocardiogram (ECG) (**A**) Sinus tachycardia, irregular rhythm, ST elevation on lead V1-V5 on first admission, (**B**) sinus rhythm, regular and no ST elevation in 21 days hospitalization**.**

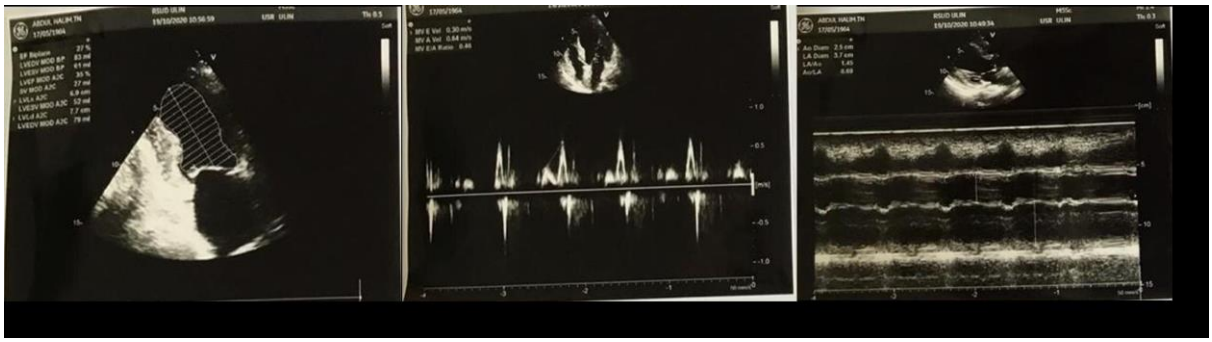

Figure 3. Transthoracic echocardiography after 20 days of hospitalization revealed Left Ventricle (LV) dilatation with severely reduced systolic function, with an estimated LVEF of 27%.

account for cardiac susceptibility to COVID-19. In addition, severe COVID-19 can cause a cytokine storm due to systemic inflammation, resulting in multi-organ failure, including cardiovascular failure (10,11). Hypoxemia, brought on by respiratory failure in COVID-19 patients, leads to a mismatch between supply and demand in cardiac cells, resulting in acute myocardial injury. Micro thrombotic embolism can also result in acute myocardial injury in COVID-19 due to SARS-CoV-2's ability to induce a prothrombotic state, contributing to microthrombi formation. Once the microthrombi have formed, they may be embolized, which would cause acute ischemia (12,13).

Based on the guidelines, acute coronary syndrome (ACS) patients are treated with anti-ischemia (beta blockers, nitrates, and calcium channel blockers are included), antiplatelets, anticoagulants, glycoprotein receptor inhibitors IIb/IIIa, a combination of antiplatelet and anticoagulants, ACE inhibitors and angiotensin receptor inhibitors, and statin. Reperfusion therapy is the primary treatment for ACS patients with STEMI. Reperfusion therapy is divided into primary catheterization interventions (PCI) and fibrinolytic. In hospitals capable of PCI, the desired target is a 'door-to-balloon' delay of < 60 minutes between patient arrival and the start of PCI (14). In our case, it took 48 hours from the start of clinical chest pain manifestations to the STEMI diagnosis. It indicates patient's postponement between the onset of symptoms and the initial medical consultation, so no reperfusion therapy was performed in this patient. Patients who do not get reperfusion therapy can be given anticoagulant therapy (non-UFH regimen) during hospitalization, up to a maximum of 8 days of administration.

In our case, the patient also experienced an arrhythmia. In COVID-19-confirmed patients treated in Wuhan, the prevalence of arrhythmias was 16.7% and increased 44.4% in intensive care patients. In a cohort study of 99 individuals who had COVID-19, 19% had atrial fibrillation (AF), and 36% had a history of cardiovascular disease. AF was a frequent complication among deceased patients (13,15). The SARS-CoV-2 mechanism can cause arrhythmias through several mechanisms, including (1) SARS-CoV-2 can directly cause cardiomyocyte injury, which can trigger arrhythmias by affecting potential action, (2) SARS-CoV-2 can trigger cell death, and (3) hypoxemia. Respiratory problems that result in hypoxic myocardial conditions are the cause of hypoxemia. Hypoxia can lengthen or change the action and repolarization of the heart, kill cardiomyocyte cells, and affect the ion canal, all of which can increase arrhythmogenesis (16–18).

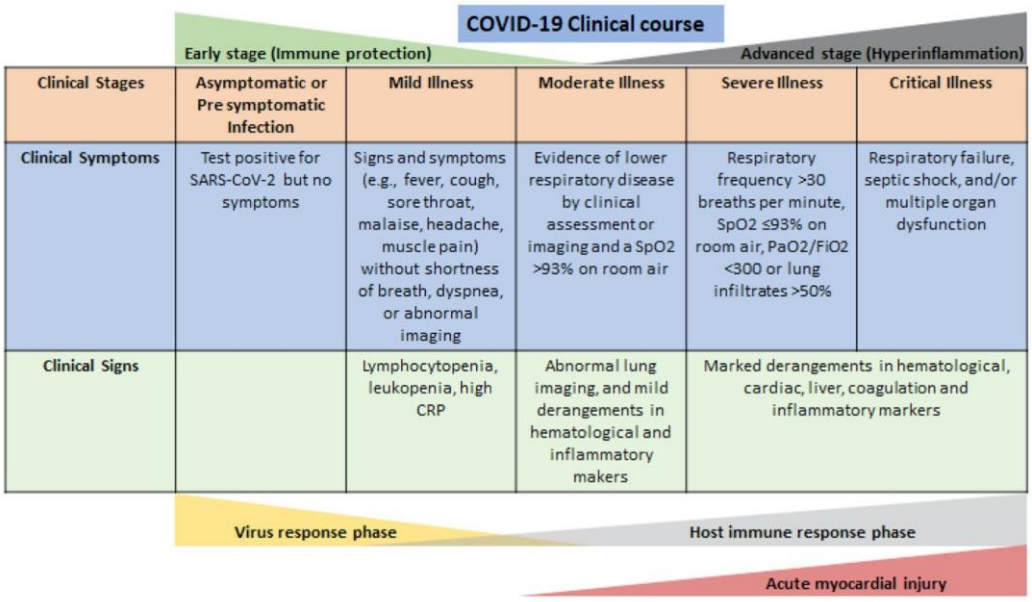

Figure 4. Clinical Manifestations of COVID-19 and Acute Myocardial Infarction (9).

Several factors can cause the incidence of atrial fibrillation itself. These factors include increased pressure or atrial resistance, infiltrative and inflammatory processes, infectious processes, endocrine disorders, neurogenic, atrial ischemic (myocardial infarction), drugs, and genetics. TNF, IL-1, and IL-6, which are inflammatory cytokines produced by the SARS-CoV-2 infection, have significant arrhythmogenic effects, resulting in cardiomyocyte damage and potential disruption of action. The significance of inflammatory cytokines in cardiac injury has been widely established. Certain COVID-19 patients have encountered arrhythmic events that threaten their lives, even without severe respiratory impairment, when there is a severe systemic inflammatory activation. In addition, SARS-CoV-2 infection is also suspected to cause direct injury to the heart by structural and electrical changes and causing thrombosis in the coronary microvascular as a result of inflammatory responses that cause myocardial infarction. SARS-CoV-2 infection also causes ARDS in patients, where hypoxia causes myocardial damage. So this event gives rise to a vicious cycle that can lead to atrial fibrillation (19).

In this patient, an ECG taken at the beginning showed an atrial effect of rapid ventricular fibrillation response with HR 140 beats/minute with stable hemodynamics. Beta-blockers and other rate-controlling drugs treat AF in the acute phase and new onset (20). In managing AF in this patient, oral beta blockers are given, namely bisoprolol 5 mg once daily. The patient finally recovered and was discharged from the hospital after being hospitalized for 15 days in the isolation room and two weeks in the non-isolation room.

Hence, we concluded that SARS-CoV-2 infection is linked to notable cardiovascular outcomes, such as sudden myocardial infarction and arrhythmia. Hypoxia, direct injury of SARS CoV-2 to cardiomyocytes, pericardium infection, stress-induced cardiomyopathy, cytokine storm, microvascular/thrombotic injury, and preexisting cardiovascular disease are the mechanisms for the emergence of acute COVID-19 cardiovascular syndrome. Individuals with previous cardiovascular conditions are more susceptible to contracting COVID-19, significantly impacting the disease's course, treatment, and prognosis.

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