Case Report

A 32-Year-Old Coronary Thrombus Case Found during the COVID-19 Pandemic: Could It Be Caused By Sinovac Inactivated Vaccine Or COVID-19 Late-Term Complication?

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Abstract

Coronavirus disease 2019 (COVID-19) infection is a public health problem, which globally is considered a pandemic, causing a significant level of morbidity and mortality. It has been defined as a viral infection, the etiology of which is unknown, causing mostly respiratory tract infections. It is surely beyond doubt that vaccine development also plays an important role in ending such pandemics as well as hygiene rules and personal protective equipment. It is becoming gradually more apparent that cardiovascular complications associated with COVID-19, such as myocardial damage, arrhythmia, acute heart failure, and venous thromboembolism, occur more frequently than what was thought before. The precise pathophysiological mechanisms of myocardial damage remain unclear but are thought to be the main pathway, with microthrombi, cytokine storm, and plaque rupture due to hypoxic damage, coronary spasm, direct endothelial, or vascular injury. In this study, we wanted to present a 32-year-old coronary thrombus case with no medical history and no risk factors, which we think may develop due to the Sinovac inactivated COVID-19 vaccine or as a late complication of COVID-19 disease. While cardiac side effects associated with inactive COVID-19 vaccines are still limited in the literature, this issue can be clarified if there are more case reports.

Keywords: Coronary thrombus, COVID-19, myocardial infarction, Sinovac inactivated vaccine

Introduction

Coronavirus is an enveloped RNA virus belonging to the *Coronaviridae* family.[1] COVID-19 virus, also known as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), was first discovered in Hubei province of Wuhan city in China in December 2019. It has been defined as a viral infection, the etiology of which is unknown, causing mostly respiratory tract infections.[2] The virus first spread quickly within the city and then worldwide. It has become a serious public health problem with its increasing rate of spread and was confirmed as a "Pandemic" by the World Health Organization in 2020. It is known to frequently cause symptoms such as fever, shortness of breath, cough, muscle pain, myalgia, and weakness. In addition, there are studies showing that a number of thrombotic events related to COVID-19 infection have increased.[3,4] It has been reported that it also causes pulmonary embolism as well as frequently causing venous thromboembolism and cerebral infarctions.[5-8] Even though it is less common, it may lead to renal artery thrombosis, myocardial damage, and mesenteric ischemia.[9-12] Although they are observed less frequently, thrombotic complications lead to a more serious and destructive course of the disease. While clinical evidence of thrombosis events associated

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with COVID-19 infection has been demonstrated, its pathophysiology is still an active field of research.[13] In pathology studies conducted on COVID-19 patients, it has been shown to cause diffuse microthrombi. These effects occur through complement-mediated microvascular damage in the lung and skin.[14,15] It is known that SARS-CoV-2 infects the angiotensin-converting enzyme-2 (ACE-2) receptor found in endothelial cells at an early stage. According to the researches on the molecular pathogenesis of the disease defined as endotheliitis, it causes the accumulation of inflammatory cells and microcirculatory disorder.[16] It has been shown in recent studies that COVID-19 infection significantly increases procoagulants associated with acute phase reactants, leading the authors to hypothesize that the systemic inflammatory response is a significant contributor to thrombogenesis.[17] In a study of early-stage conducted in Wuhan, China, a 12% myocardial injury rate was reported in patients with COVID-19 infection based on biomarker elevation.[18]

In this study, we wanted to present a case of coronary thrombus, which we think may develop due to the Sinovac inactivated COVID-19 vaccine or as a late complication of COVID-19 disease.

Case Report

The patient was a 32-year-old female patient who was admitted to the emergency room. She complained of chest pain, sweating, and palpitations spreading to her left arm for about 6 h. Fever was 36.7°, respiratory rate 22/min, blood pressure 110/70 mm/Hg, and SpO_2 97%. On physical examination, lung breathing sounds were normal; heart sounds were rhythmic and normal. The electrocardiogram (ECG) was in sinus rhythm and the heart rate was 97/min, with no pathology found [Figure 1]. The ejection fraction was evaluated within normal limits in the echocardiography image. No significant pathology was detected in the heart valves. In her medical history, she had a depot progesterone derivative. She did not use drugs, cigarettes, or alcohol. She was not pregnant. There was no history of chronic illness. She had a COVID-19 infection 3 months ago. She was not hospitalized while infected with COVID-19 disease. At that time, there were symptoms of widespread muscle pain, weakness, and loss of taste. She did not use any medications during the COVID infection. No pathology was found on thorax computed tomography taken in the emergency room. The Sinovac inactive 1st dose COVID vaccine was administered 50 days ago, and the Sinovac inactive 2nd dose COVID vaccine was administered 20 days ago. A progressive increase was observed in serial troponin levels, which were monitored at 3-h intervals in the emergency department (0 ng/mL–1.19 ng/mL–7.77 ng/mL, [determined reference range: 0–0.06 ng/mL]). No significant pathology was found in biochemical, hemogram parameters, and infection markers. All coagulation parameters and D-dimer obtained from the patient were within normal limits. The global registry of acute coronary events score was 58 points and the thrombolysis in myocardial infarction risk score was calculated as 2. Coronary angiography was decided because the patient's troponin values increased and his angina continued. The patient was taken to the catheter laboratory with a diagnosis of non-ST-elevation myocardial infarction. In the coronary angiography performed, an appearance compatible with thrombus in the distal left anterior descending artery was observed. No pathology was observed in circumflex and right coronary artery [Figure 2]. Two hundred microgram nitrate was administered intracoronary. However, abciximab treatment was planned because angina persisted, there was no significant atherosclerotic plaque, and the appearance consistent with thrombus continued. Abciximab (glycoprotein 2b/3a inhibitor) was given as an intracoronary bolus of 15 mg, followed by a systemic venous route for 12 h as 7.5 μg/min maintenance therapy. The procedure was finalized. Afterward, acetylsalicylic acid, clopidogrel, and enoxaparin treatments were ordered, and the patient was taken to the coronary

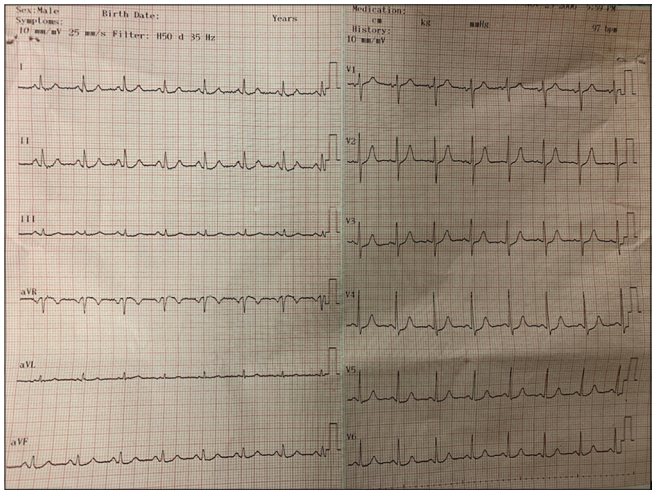

Figure 1: Electrocardiogram of the patient at the time of admission to the emergency department

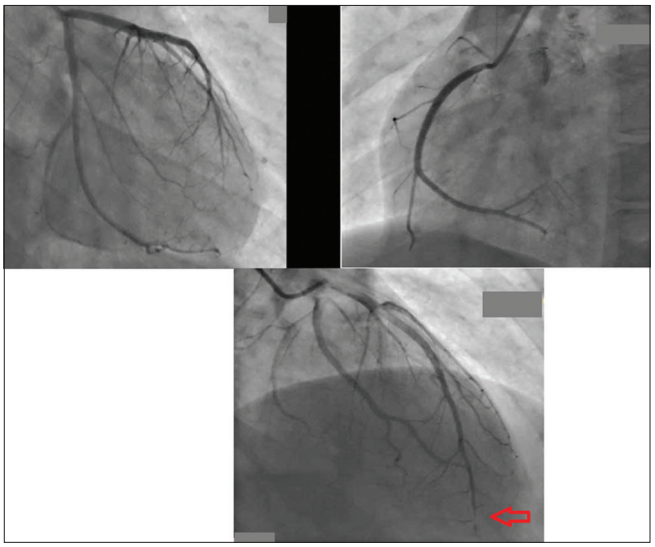

Figure 2: Coronary angiography images of the patient. Red Arrow: thrombus distal to the left anterior descending artery

intensive care unit for follow-up. In addition, a maintenance dose of abciximab was given in the intensive care unit. The antiphospholipid antibody, antithrombin-3, protein-C, protein-S, factor V Leiden mutation levels in the blood after the procedure resulted in the normal reference range. In the control echocardiography examined at the bedside on the 2nd day after the procedure, again no wall motion defect was seen. Control coronary angiography was not planned due to a decrease in troponin follow-ups (4.58–2.27 ng/mL), no dynamic changes in ECG follow-ups, and improvement in the patient's symptoms. The patient was discharged 72 h after the procedure.

Discussion

Thrombotic complications from COVID-19 infection are well known, based on previous clinical studies and experience from in healthcare settings. In a study conducted during the COVID-19 outbreak, hospital admissions ascribed to acute coronary syndromes decreased by about 40%. This rate most likely reflects the patient's preference to remain at home despite having symptoms, considering the risk of virus transmission in health-care facilities.[19] The relationship between COVID-19 infection and cardiovascular disease has been well established. Studies show that patients infected with SARS-CoV-2 have an increased risk of severe disease and death if they have a medical history of cardiovascular disease.[20] Elevation of cardiac enzymes is associated with more severe cases of COVID-19 and higher mortality. It is becoming increasingly clear that cardiovascular complications associated with COVID-19, such as myocardial damage, arrhythmia, acute heart failure, and venous thromboembolism occur at higher frequencies than previously thought.[21] The mechanism of heart damage is thought to be multifactorial. SARS-CoV-2 infection occurs through receptor-mediated endocytosis triggered by the binding of the viral spike protein to the ACE-2 receptor on the host cell. The ACE-2 receptor is highly expressed in myocardial tissue, and this can function as a direct invasion pathway for the virus to disease-causing myocardial tissue.[22] The virus can also cause systemic inflammation that leads to cytokine storm, which can develop into multi-organ system failure, including the cardiovascular system.[18,20] Respiratory failure and the subsequent hypoxemia can lead to increased supply and demand mismatch and thus acute myocardial damage. SARS-CoV-2 is also thought to trigger a prothrombotic state that leads to the formation of microthrombi that can go on to form emboli. It leads to an acute ischemic event of the target end organ.[23] This mechanism may be what we think occurs in our young patient. The precise pathophysiological mechanisms of myocardial damage remain unclear, but as aforementioned, microthrombi, cytokine storm, and hypoxic damage are thought to be the main pathway, along with plaque rupture, coronary spasm, or direct endothelial or vascular injury. Our patient did not have an active COVID-19 infection. There were no risk factors for coronary artery disease, and there was no history of drug use that increased the risk of thrombus. We did not come across any significant pathology that increases the susceptibility to thrombosis in the thrombophilia panels. The fact that our patient had a history of COVID-19 as of few months ago and had an inactive COVID-19 vaccine in the recent past indicates that the coronary thrombus event that occurred-if there is no late complication of COVID-19 infection-may demonstrate such a complication after inactive COVID-19 vaccines. We wanted to discuss this issue. We require more case series and large-scale studies on this subject. To our knowledge, this is the first case report showing that coronary thrombus may develop after an inactive COVID-19 vaccine.

Conclusion

COVID-19 infection is a public health problem, which globally is considered a pandemic, causing a significant level of morbidity and mortality. Without a doubt, vaccine development has an important role in ending pandemics as well as hygiene protocol and personal protective equipment. Studies are continuing to demonstrate the effectiveness and reliability of the vaccines that receive emergency approval for use. In this case study, we wanted to report a rare case of coronary embolism where we could not find any underlying cause, which we believe that this situation may be a delayed complication of COVID-19 disease or a rare complication of inactive COVID-19 vaccination.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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