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Case Report

Coronary Thrombosis Case in Female Patient with COVID-19 Confirmed Case: Coincidence or Complication?

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ABSTRACT

The worldwide health impact of the COVID-19 epidemic has been felt all across the world, mostly due to respiratory issues, but many additional signs such as cardiac manifestations and different thromboembolisms have also been observed. **Case Summary**. We provide you with a 48-year-old female patient with COVID-19 pneumonia following a severe infection, who developed Unstable Angina due to coronary thrombosis. She has a history of hypertension and obese posture. No diabetes, hypercholesterolemia, or smoking habit had been recorded. And she still has regular menstrual cycle. Around a month prior to being referred to our hospital, this patient has been admitted to ICU of satellite hospital. She was treated with ventilator and discharged around a week before she was admitted to our Emergency Department. She had typical chest pain with ST-segment depression ECG at antero-septal area. PCI was performed and a large thrombosis was found at LAD artery. **Discussion:** Many studies have showed that COVID-19, through various ways, has developed thromboembolism both in arteries and venous system. The actual process of thrombus development in the coronary arteries is unknown, however, it is thought that pro-inflammatory cytokinesis released by the body, which stimulates the coagulation cascade and prevent fibrinolysis, are the major cause. This condition that leads to hypercoagulable state which eventually makes an increased risk of thromboembolism.

Introduction

The worldwide health impact of the COVID-19 epidemic has been felt all across the world, mostly due to respiratory issues, but many additional signs, such as cardiac manifestations and different thromboembolisms, have been observed. COVID-19 has a high rate of thrombotic consequences and coagulopathy, our case shows the thrombosis formed in coronary artery as a complication of the severe COVID-19 infection, manifesting as ACS Unstable Angina.

Case Presentation

We provide you with a 48-year-old female patient with COVID-19 pneumonia following a severe infection, who developed Unstable Angina due to

coronary thrombosis. She has a history of hypertension and obese posture.

No diabetes, hypercholesterolemia, or smoking habit had been recorded. And she still has reguler menstrual cycle. Around a month prior to being reffered to our hospital, this patient had been admitted to ICU of satelite hospital. She was treated with ventilator and discharged around a week before admitted to our Emergency Department. She had typical chest pain with ST-segment depression ECG at antero-septal] area. During hospitalization, the patient received enoxaparin, aspirin, clopidogrel, beta-blocker, atorvastatin, and angiotensin receptor blocker. From echocardiography we found no significant abnormality detected. Invasive coronary angiography was performed and a large thrombus was found in middle segment of Left Anterior Descending artery with TIMI 3 flow and other coronary arteries were found with no abnormality. After discharged, the patient was planned for further diagnostic procedures.

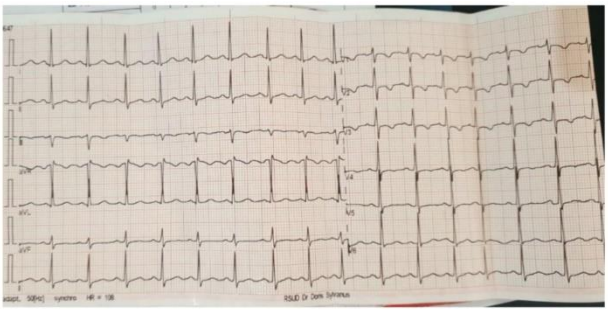

Figure 1. ECG on admission

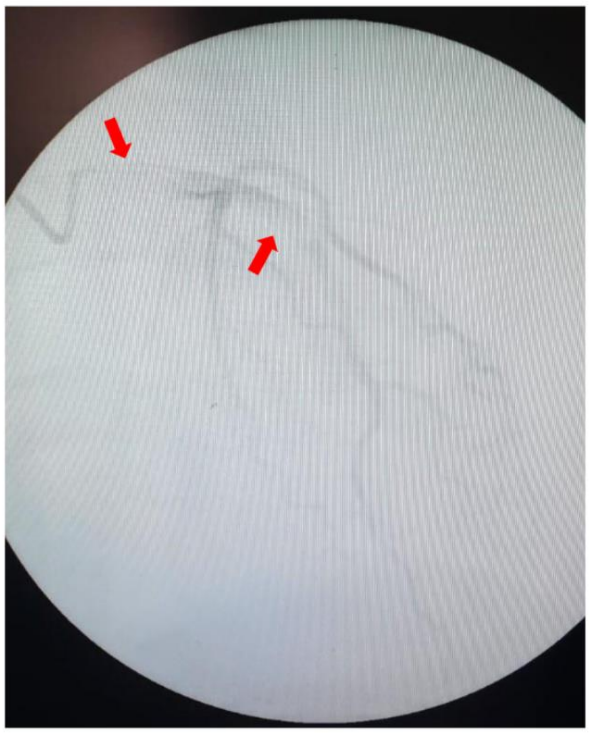

Figure 2. Invasive coronary angiography founding

Discussion

COVID-19 infection is characterized by respiratory symptoms, however it also seems to have comparable cardiac manifestations. Our patient had two established coronary artery disease risk factors and quite unlikely to develop one because of her ongaing menstrual cycle. We consider that COVID-19 might have played bid role in increasing risk of thromboembolism in this patient.

There are various pathophysiology and mechanism of this disease in connection to the cardiovascular system. The virus binds to the Angiotensin Converting Enzyme (ACE2) receptor, which mediates cell entrance, as one of the mechanisms. As we know, Human heart, endothelial cells, and pericytes also express ACE2. Endothelial cell or

pericyte infections are particularly dangerous because they can induce severe microvascular and macrovascular dysfunction, especially in coronary arteries. Angiotensin-converting enzyme 2 (ACE2) converts angiotensin II to angiotensin 1-7, which increases the production of nitric oxide by endothelial cells in a healthy state (NO). NO aids in the dilation of blood arteries and inhibits platelet aggregation. SARS-CoV-2 binds to ACE2 in COVID-19, causing a rise in angiotensin II levels and reduced blood flow. Von Willebrand factor (VWF) held in the Welbel Palade body is released into the circulation, increasing clot formation.

The increase of inflammatory cytokines is another prevalent theory for coronary thrombus development in COVID-19. COVID-19-related coagulation alterations signal the existence of a hypercoagulable condition, which might raise the risk of thromboembolic consequences. The coagulation cascade is activated by pro-inflammatory cytokines, which impede fibrinolysis and can lead to thrombus formation. This coagulopathy state alone is not completely understood and has many hypothetical mechanisms. The pathophysiology of COVID-19-associated coagulopathy (CAC) is complicated, and it is likely to differ in significant ways from the conventional thrombosis mechanisms observed in critically sick patients. CAC shares some characteristics with Sepsis-induced coagulopathy/Disseminated intravascular coagulation (SIC/DIC), Hemophagocytic syndrome/Hemophagocytic lymphohistiocytosis (HPS/HLH), Antiphospholipid syndrome (APS), and Thrombotic microangiopathy (TMA) including Thrombotic thrombocytopenic purpura (TTP) and Hemolytic uremic syndrome (HUS) in some ways, but it also has distinct characteristics that might lead to it being classified as a new coagulopathy category.

An elevated D-dimer concentration, a small reduction in platelet count, and a lengthening of the prothrombin time are the most common laboratory results in individuals with COVID-19 and coagulopathy. Increased levels of proinflammatory cytokines such as tumor necrosis factor-a (TNF-a) and interleukins (IL), especially IL-1 and IL-6, are also linked to severe COVID-19. Tissue factor expression is induced by IL-6 in mononuclear cells, which leads to coagulation activation and thrombin production. TNF-a and IL-1 are the major mediators that cause endogenous anticoagulant pathways to be suppressed, resulting in a procoagulant state in COVID-19. This procoagulant condition can result in leukocyte migration and adhesion, platelet activation and adhesion, and endothelial dysfunction, all of which can contribute to the development of thrombus.

The closest mechanisms are DIC and TMA coagulopathies. The presence of thrombocytopenia, a prolonged prothrombin time, and an elevated D-dimer suggests DIC, however the pattern differs from that found in sepsis.

Thrombocytopenia is more common in sepsis, and D-dimer concentrations do not approach the high levels reported in COVID-19 patients. In reality, most COVID-19 patients would not be classed as having DIC by the International Society on Thrombosis and Hemostasis' DIC score. Increased lactate dehydrogenase (LDH) and, in some cases, dramatically elevated ferritin concentrations, similar to those seen in thrombotic microangiopathy, are further laboratory abnormalities in COVID-19 that might be related to the coagulopathy. Patients with COVID-19 have microvascular platelet-rich thrombotic depositions in tiny arteries of the lungs and other organs, according to post-mortem observations. But this mechanism also differs, since the blood film shows no evidence of hemolysis or schistocytes, and the platelet count is larger than predicted in thrombotic microangiopathy. However, because CAC is caused by a combination of variables, a better knowledge of the underlying pathophysiology is required for effective intervention.

According to the ESC, the same principles apply to diagnosing and treating cardiovascular problems during the COVID 19 pandemic as they did previously, with the emphasis on the utilization of the correct Personal protective equipment (PPE). In ACS patients, especially ST-segment slevation| myocardial infarction (STEMI) and very high-risk Non-ST-segment elevation myocardial infarction (NSTE)-ACS, the managements are still the same, incorporating a triage system that recognizes symptoms, fast laboratory investigation such as an ECG, cardiac markers, and Primary PCI, regardless the nasopharyngeal swab result.

Other types of ACS patients should have a nasopharyngeal swab done right away after being admitted. Patients must be separated in a monitored environment while waiting for the swab result. Coronary angiography and PCI may be done if two negative findings are obtained within 48 hours. In many hospitals, this will be quite a struggle.

During the invasive procedure, this patient was found having thrombus in LAD artery, but with TIMI 3 flow. According to MINOCA (MI with Non-Obstructive Coronary Artery) management, this patient should be planned to have further diagnostic investigations with Contrast Cardiac MRI and/or Intravascular Imaging

Conclusion

COVID-19 patients can present with acute coronary syndrome which might be in coronary thrombosis form. Many similar cases throughout different countries had been reported, several case even reported in much younger patients, which remind us to always be alert in COVID-19 various complications, predominantly in thromboembolism.

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