

Case Report of STEMI Due to Early Stage Multiple Stent Thrombosis Accompanied By Covid-19 Infection

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Abstract

The outbreak of the novel coronavirus disease in 2019 (COVID-19) has a major threat to human health, especially on patients with underlying heart diseases. Stent thrombosis (ST) is a rare event often associated with large myocardial infarction (MI) or death. Here, we report a case of a patient with COVID-19 infection who developed ST-elevation myocardial infarction (STEMI) with multiple stent thrombosis under triple antithrombotic therapy in the early period after coronary stenting.

Introduction

ST is a rare but most feared complication of coronary artery stenting, usually presenting as acute STEMI and has a 30-day mortality rate of %20 to %45⁽¹⁾. It may be classified as early (within 30 days after stent implantation), late (between 1 month and 1 year after implantation), or very late (>1-year post-implantation)⁽²⁾. Despite advances in stent implantation technology and more effective antiplatelet therapy options, stent thrombosis develops at a rate of %1-4⁽³⁾. A higher susceptibility to both arterial and venous thrombosis has been identified during COVID-19 infection⁽⁴⁾. Inflammatory pathophysiological mechanisms leading to plaque rupture and the establishment of a prothrombotic environment can potentially increase the risk of local micro thromboembolism, impair reperfusion, and increase the risk of intra-coronary



stent thrombosis⁽⁵⁾. We present a COVID-19 PCR test positive patient who developed STEMI due to early stent thrombosis under triple antithrombotic therapy.

Case Report

A 73-year-old male patient was referred to our center for rescue percutaneous transluminal coronary angioplasty (PTCA) after fibrinolytic therapy (Tenecteplase) due to inferior ST elevation MI on 13 February 2021. The patient was taken from the emergency department directly to the coronary angiography unit due to chest pain and hypotensive and bradycardic course. After coronary angiography, total stenosis of the right coronary artery (RCA) and 70% stenosis of the left coronary artery (LAD) were detected. In the same session, percutaneous coronary intervention (PCI) was applied to the stenosis in the RCA. Two days later, PCI was applied to the stenosis in the LAD under elective conditions. The patient was followed up in the coronary intensive care unit (CICU) after the procedure. Medical cardioversion (with amiodarone) was applied to the patient who developed AF rhythm during CICU follow-up. Sinus rhythm was achieved. The patient was transferred to the cardiology ward on the 4th day of his CICU admission. On the 7th day of his hospitalization, he was discharged with Clopidogrel 75 mg 1x1, ASA 100 mg 1x1, Warfarin 5 mg 1x1, Atorvastatin 40 mg 1x1, Ramipril 2.5 mg 1x1, Metoprolol 25 mg 1x1, and Pantoprazole 40 mg 1x1. Three weeks after discharge, the patient was admitted to emergency room of another health care center with shortness of breath and was intubated due to respiratory arrest. He was referred to the cardiology ward of our center because of ST elevation in the anterior and inferior leads of his ECG (Figure 1). The hematological parameters of the patient measured before his referral to the ward and prior to respiratory arrest were as follows. INR: 2.3, PT: 28.5 sec, APTT: 31 sec, D-DIMER: 4 ug/ml.

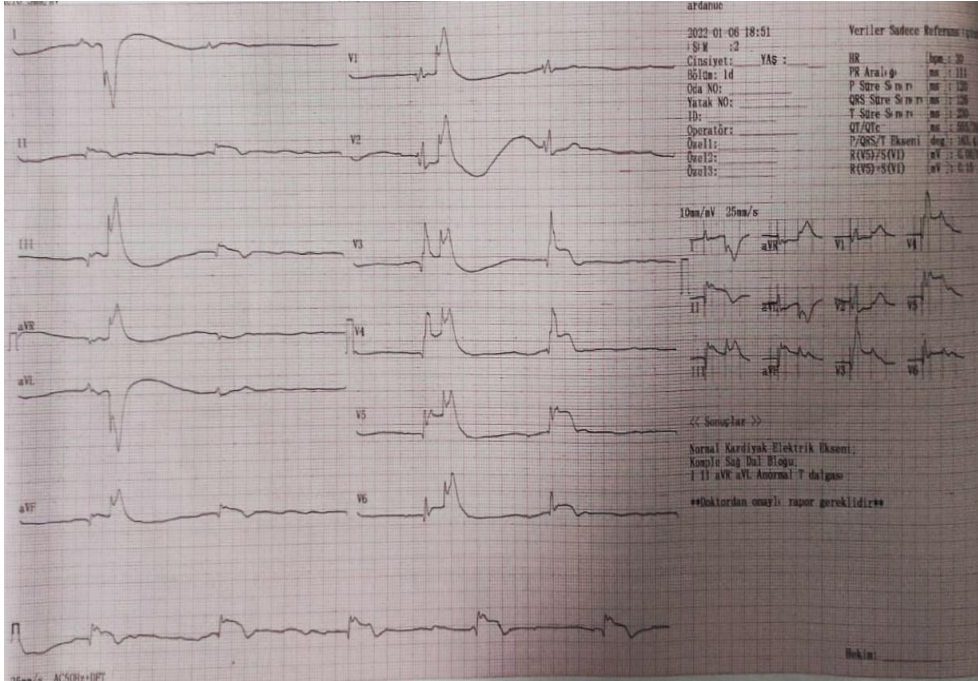


Figure 1: The patient’s ECG shows ST elevation at anterior and inferior leads

The patient was taken to the coronary angiography laboratory. Coronary angiography detected RCA and LAD intra-stent %100 thrombosed lesions (Figure 2). PCI was performed for RCA proximal and LAD proximal total lesions. TIMI 3 flow was provided. The patient was taken to the CICU after the procedure. The Covid-19 PCR test was positive for the patient, whose thorax CT images taken during his emergency admission were compatible with COVID-19 (Figure 3) . The patient was transferred to “Covid intensive care unit”.

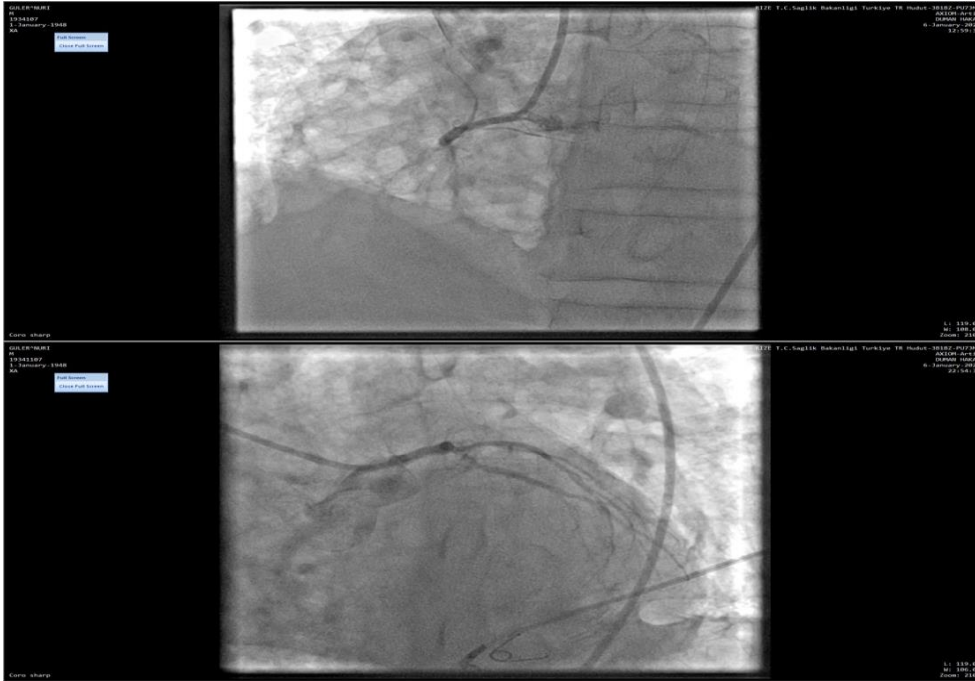


Figure 2: Coronary angiography detected RCA and LAD intra-stent %100 thrombosed lesions



Figure 3: The patient's thorax CT images taken during his emergency admission were compatible with COVID-19

Discussion:

ST is a dramatic complication of coronary stenting. Despite its rarity, stent thrombosis is therefore an important complication and research topic, given the high number of PCI performed each year. Immediate revascularization is essential to prevent long-term cardiac dysfunction or death⁽⁶⁾. This is even more important in patients with COVID-19 infection than in patients without comorbidities. Despite optimal antithrombotic medical therapy, there appears to be an increased risk of ST in patients infected with COVID-19, possibly due to excessive platelet aggregation. Intra-stent thrombosis may potentially be the result of greater thrombus load and specific mechanisms associated with plaque disruption in patients infected with COVID-19, leading to an increased risk of thrombus formation and coronary thrombotic complications⁽⁷⁻⁸⁾. The mechanisms underlying the formation of ST are multifactorial, which include patient-related factors (like diabetes mellitus, renal failure), procedural factors (like complex lesions, poor stent expansion and vessel wall adhesion), and post-procedural factors (like type and duration of antiplatelet therapy)⁽⁹⁾. During COVID-19 infection, and in severe cases, a cytokine storm occurs 5 to 10 days after the onset of symptoms, resulting in endothelial damage, activation of platelets, and triggering of the coagulation cascade. Zhang et al.⁽¹⁰⁾ describe the presence of antiphospholipid antibodies that can lead to thrombotic events in patients infected with COVID-19. In this context, the necessity of intensifying antiplatelet therapy or an established therapy in patients infected with COVID-19 with pre-existing coronary heart disease or a history of stenting, particularly recent stenting, remains the target of further investigation. In our case, the result of PCI performed at the first hospitalization was satisfactory with adequate stent expansion. The INR value of the patient discharged with triple antithrombotic therapy was found to be within the therapeutic range in the blood tests performed at the time of admission. From this point of view, it is apparent that stent thrombosis developing under optimal triple antithrombotic therapy is rare in the literature.

Conclusions

Given the increasing incidence of COVID-19 worldwide and the high prevalence of coronary artery disease, we may be faced with a significant number of patients with STEMI suffering from COVID-19 infection. The possibility of stent thrombosis should be considered in patients with active COVID-19 infection and symptoms of acute coronary syndrome after PCI.



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