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Case Report

Single-Vessel Massive Coronary Thrombosis in a Patient with COVID-19 Infection: A Case Report

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Abstract

Introduction: The ratio of coronary thrombosis in patients with ST-segment elevation acute myocardial infarction and COVID-19 disease showed a high number, both for obstructive and non-obstructive atherosclerotic patterns. According to the detection of acute coronary syndrome in patients with COVID-19, it may be related to atherosclerosis and plaque rupture or formation of spontaneous thrombosis, or the combination of these two phenomena.

Clinical case: 36-year-old male with no cardiovascular risk factors that arrived to the emergency department with atypical chest pain who developed acute myocardial infarction with ST-segment elevation secondary to acute thromboembolism due to previous SARS-CoV2 infection. PCI was performed and single-vessel grade 4 thrombus was found in the proximal segment of the left anterior descending artery and was treated with thrombus aspiration with Stemi Cath 6 Fr catheter and a drug-eluting stent placement due to visualization of unstable plaque.

Keywords: Single vessel coronary thrombosis; Acute coronary syndrome; COVID-19; Hyperinflammatory state; Unstable plaque

Case Presentation

A 36-year-old male with no active cardiovascular risk factors (stopped smoking 8 years prior) with previous SARS-COV2 infection (13 days prior) who came to the emergency department for presenting abdominal pain of 4 hours of evolution and irradiation to the anterior chest with VAS 5/10, with no other symptoms and he self-medicated with bismuth subsalicylate, omeprazole and calcium carbonate without improvement. Physical examination showed vital signs within normal parameters, cardiac and respiratory examination without evident alterations, abdominal examination showed pain on superficial and deep palpation in the upper abdominal quadrant regions, with no evidence of peritoneal irritation. As part of his initial investigation, a 12-lead electrocardiogram at rest was taken, in which it was in sinus rhythm, with symmetrical and acuminate T waves in V2-V4 and ST-segment elevation in V3 and V4 (Figure 1). Laboratory studies were requested with blood panel with leukocytes of 13,300 mm^3, CPK 47 U/L, CPK-MB 20 U/L, negative qualitative Troponin I, D-Dimer 1130 ng/ml. The patient was admitted for observation, where he presented one hour later with typical anginal symptoms, accompanied by diaphoresis, facial pallor and imminent death feeling, so another EKG was taken showing sinus rhythm, with flattening of T waves in the high lateral face, and ST segment elevation in V2-V5 (Figure 2). It was decided to initiate oral therapy with acetylsalicylic acid 300 mg, clopidogrel 600 mg and atorvastatin 80 mg in single doses. With the diagnosis of acute myocardial infarction with ST-segment elevation, classified at the time as Killip 1, TIMI score 2, GRACE score 88 points, it was decided to admit the patient for Primary Interventional Therapy (PCI). During the study, a grade 4 thrombus was found in the proximal segment of the Left Anterior Descending artery (LAD), which occluded up to 80% with a TIMI I post-injury flow; the rest of the coronary arteries were without significant angiographic lesions (Figure 3). Thrombus aspiration was performed, using a StemiCath 6 Fr catheter, finally it was decided to directly place a BioMime 4.0x13 mm drug-eluting stent at 18 ATM for 15 seconds secondary to visualization of an unstable atheromatous plaque. After the procedure, intravenous infusion with tirofiban was started due to the thrombotic load and embolization of the distal segment of the LAD, starting with a bolus dose of 0.4 g/kg/min for 30 minutes and subsequent maintenance dose of 0. 1 g/kg/min as well as concomitant use of low molecular weight heparin (enoxaparin 60 mg every 12 hours), acetylsalicylic acid 100 mg, clopidrogrel 75 mg, atorvastatin 40 mg every 24 hours and metoprolol 50 mg every 12 hours. A transthoracic echocardiogram was performed 24 hours after the presentation of the infarction, which reported akinesia of the distal two thirds of the anterior interventricular septum and the entire tip, the other segments were normal.Type I/III diastolic dysfunction, absence of intraventricular thrombus, morphologically normal valves, absence of abnormalities of the proximal ascending aorta and left ventricular ejection fraction of 60% by modified Simpson's method. Subsequently, no associated factors suggestive of thrombophilia were found, as well as a previous family history of thromboembolism; however, a complete study for thrombophilia was not performed due to the high risk of false positives in this acute phase, particularly in the context of infection and acute inflammation. No further laboratory tests were performed due to the patient request voluntary discharge.

Discussion

Multiple theories have been postulated to date about the

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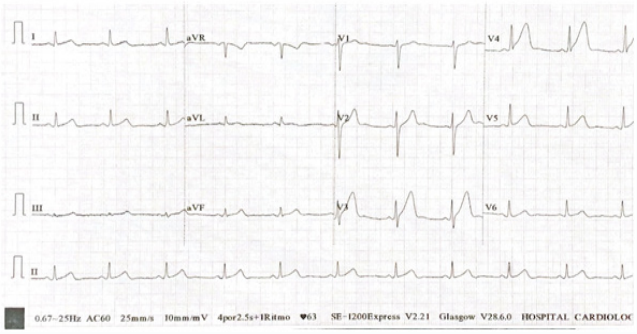

Figure 1: First 12-lead electrocardiogram at rest in sinus rhythm, shows ST-segment elevation in V3-V4, asymmetric and acuminate T wave in V2-V4.

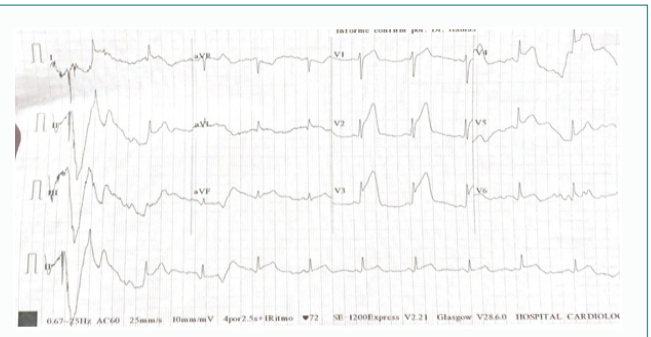

Figure 2: Second 12-lead electrocardiogram (one hour after arrival) in sinus rhythm and ST segment elevation in V2-V5, asymmetric and acuminate T wave in V2-V4.

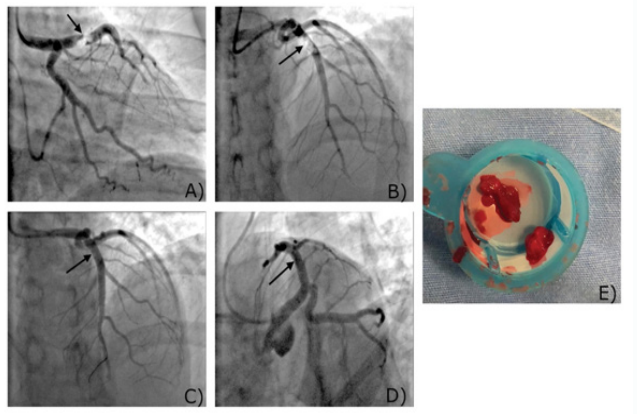

Figure 3: A) Right-caudal projection shows Cx without lesions and same image of thrombus in proximal LAD [arrow] B) AP-cranial projection with evidence of grade 4 thrombotic lesion in proximal third of the LAD immediately prior to the first septal branch of 80% [arrow]. C) AP-Cranial projection after the placement of a DES. D) Spider-projection after placement of a DES in the proximal segment of the LAD. E) Thrombus removed from the procedure.

possible triggers of hypercoagulability in patients with COVID-19 disease. One of these theories claims that patients may experience a hyperinflammatory state with a cytokine storm response. Qin et al. [1] reported that hyperinflammation is mediated by both IL-1, TNF-α and IL-6 leading to increased plasma concentrations of fibrinogen, lactate dehydrogenase (DHL), plasminogen activator inhibitor-1 (PAI-1) and alterations in the neutrophil-lymphocyte ratio (mostly due to CD4+ T lymphocyte depletion). The interaction between IL-6, IL-8 and TNF-α contributes to a pro-coagulant state which also contributes to platelet activation, as well as endothelial cells and tissue factor expression. In turn, there is also a decrease in the production of natural anticoagulants such as antithrombin III, tissue factor inhibitor and protein C, which promotes a prothrombotic state. Acute coronary ischemic syndrome can be triggered by respiratory tract infections, as reported by Kwong et al. [2] in 2018, where an increased incidence of up to 6-fold compared to a period control for one year before and one year after influenza infection is found, however there is no accurate and reliable data that has reported the prevalence of cardiovascular events, especially in acute coronary ischemic syndrome patients with COVID-19. Cardiovascular damage related to COVID-19 infected patients reported so far includes: myocarditis, arrhythmias, cerebral vascular events and thromboembolic diseases. In Acute Coronary Ischemic Syndrome (ACS) related to SARS-COV2 infection, it has been shown that there is a high association of thrombosis in both obstructive and non-obstructive atherosclerotic patterns, with severe systemic inflammation contributing to the rupture of atherosclerotic plaques and the consequent thrombosis with the release of cytokines such as TNF-α, IFN-γ and IL-1 directly and through sympathetic stimulation. The role of endothelial cells of the pulmonary vasculature is to maintain vascular tone by releasing endothelin, endoperoxidases, arachidonic acid derivatives, superoxide radicals, thromboxane A2 and its counterpart, nitric oxide. Virus-related damage to these cells leads to microvascular dysfunction, thereby shifting the vascular balance in favor of a pro-coagulant state [3]. Those patients with cardiovascular disease and dyslipidemia have higher concentrations of Asymmetric Di-Methyl-Arginine (ADMA) and L-arginine analog that inhibits the activity of the enzyme nitric oxide synthase 3 (NOS-3), leading to a decrease in nitric oxide levels, which would explain why endothelial dysfunction and procoagulant states are more severe in these patients [4,5]. The ratio of coronary thrombosis in patients with ST-Segment Elevation Acute Myocardial Infarction (STEMI) and COVID-19 disease showed a high number, both for obstructive and non-obstructive atherosclerotic patterns. According to the detection of STEMI in patients with COVID-19, it may be related to atherosclerosis and plaque rupture or formation of spontaneous thrombosis, or the combination of these two phenomena. In the case presented by Rey et al. [6] in which thrombotic involvement was found in two arteries (Right Coronary [RCA] and LAD) in a known patient with type 2 diabetes mellitus and systemic arterial hypertension, the presence of atherosclerotic plaques could not be defined by imaging studies, however their hypothesis, due to the presence of cardiovascular risk factors, was directed to the rupture of an atherosclerotic plaque. Another publication by Dominguez P et al. [7] reported the case of a 64-year-old patient with no previous cardiovascular factors with COVID-19 pneumonia, 7 days prior to admission for STEMI, angiography was performed where a critical thrombotic lesion of the RCA was found as well as a non-occlusive lesion with filling defect compatible with thrombus in the LAD. Thrombus aspiration was performed in the RCA but underlying stenosis was found, so a stent was placed and the LAD condition was managed with anticoagulation with low molecular weight heparin in adhesion with dual platelet antiplatelet therapy.

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