

Acute Extensive Anterior Wall Myocardial Infarction Secondary to Novel Coronavirus Infection in a Young Patient: A Case Report

Liu Y, Wu H, Chen H, Dong F, Wang J and Yang C*

Department of Cardiology, Wuxi No.2 People's Hospital of Jiangnan University Medical Center, Wuxi, Jiangsu Province, 214000, China

*Corresponding author:

Chengjian Yang,
Department of Cardiology, Wuxi No.2 People's
Hospital of Jiangnan University Medical Center,
Wuxi, Jiangsu Province, 214000, China

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1. Abstract

This report reviews a young man who experienced a sudden, acute myocardial infarction without any other underlying health issues, and it discusses the connection between a novel coronavirus infection and endothelial damage and thrombosis in the coronary artery.

2. Case Report

A 22-year-old male was admitted to the emergency department with a chief complaint of "sudden chest pain for 4 hours." He started to have chest tightness on his way to work 4 hours before admission, which was located in the middle of the sternum, accompanied by sweating, soreness, and swelling of both upper limbs that were persisting and not relieved. Apart from smoking for two years at a rate of roughly 2-3 cigarettes per day, he denied having hypertension, diabetes, hyperlipidemia, varicose veins in the lower limbs, atrial fibrillation, and alcohol misuse. Ten days prior to admission, he had a new coronavirus infection, and following admission, the COVID-19 antigen was negative. Initial evaluation revealed a temperature of 36.8°C, 150/80 blood pressure, 80 heartbeats per minute, and a BMI of 28.6 kg/m² with no abnormal signals of a body check. The ECG showed sinus rhythm and ST segment elevations in I, avL, and V1-V6 leads (Figure 1). Investigators revealed a white blood cell count of 10.92*10⁹/L, hemoglobin 179g/L, platelet count of 304*10⁹/L, CRP<0.2mg/L, CK 95U/L, CK-MB 18U/L, troponin T 43.5ng/L, troponin I <0.03ng/ml and D-dimer 0.11ug/ml. The patient was taken to the cardiac catheterization lab

immediately, where he was found to have an occlusive anterior descending artery (LAD) thrombus (Figure 2C) for which he underwent manual thrombectomy and percutaneous balloon coronary dilatation, resulting in reconstitution of anterior descending artery flow (Figure 2D). As is shown in (Figure 2), no significant stenosis can be seen in the left main (LM), left circumflex branch (LCX), or right coronary artery (RCA). The patient's chest pain was relieved and he returned to CCU afterwards. As a result, he was diagnosed with acute extensive anterior myocardial infarction, Killip class I. The patient had postoperative nausea and vomiting of gastric contents that resembled coffee as well as a positive occult blood test result from the vomitus, which was diagnosed as a stress ulcer. Ticagrelor 90mg bis in die (BID), Cilostazol 100mg BID, Atorvastatin 20mg QN, Metoprolol 11.875mg QD and Esomeprazole sodium 40mg Q8H IV drip were started. The following day, the levels of CK, CK-MB, troponin T, and troponin I increased to 2437.3 U/L, 189.2 U/L, 3766.0 ng/L, and 7.94 ng/ml, respectively. The D-dimer obtained the next day was 0.06µg/mL. His blood test showed NT-pro BNP levels of 1017 pg/ml, triglycerides levels of 1.56 mmol/l, cholesterol levels of 4.8mmol/l, HDL levels of 1.16 mmol/l, and LDL levels of 2.83 mmol/l. Glycated hemoglobin, anti-O antibody, rheumatoid factor, CRP, creatinine, uric acid, routine fasting glucose, immune anti-nuclear antibodies, SM antibody, SSA antibody, SSB antibody, mitochondrial antibody, ribonucleoprotein antibody, SCL-70 antibody, nRNP antibody, double-stranded DNA antibody, and histone antibody were within

normal limits. His novel coronavirus nucleic acid also tested negative. Repetitive ECG revealed a reduction of ST elevation in the anterior-lateral leads (Figure 1B). Cardiac ultrasound on the fifth day showed a normal-sized heart with a good ventricular systolic function of 58% and slightly reduced left ventricular apical motion at rest. The coronary angiography was performed one week following surgery (Figure 3), and neither of these coronary arteries exhibited significant stenosis (Figure 3 A-B). In addition, an IVUS

catheter was delivered to the distal LAD segment, which revealed a minor residual thrombus in the distal segment (Figure 3D) and a smooth and undamaged intima of the LAD (Figure 3C). Since then, Ticagrelor and Cilostazol have been replaced with rivaroxaban 15 mg QD, and metoprolol has been increased by 100%. After the patient's health stabilized, he was discharged from the hospital and instructed to attend the outpatient clinic for follow-up care.

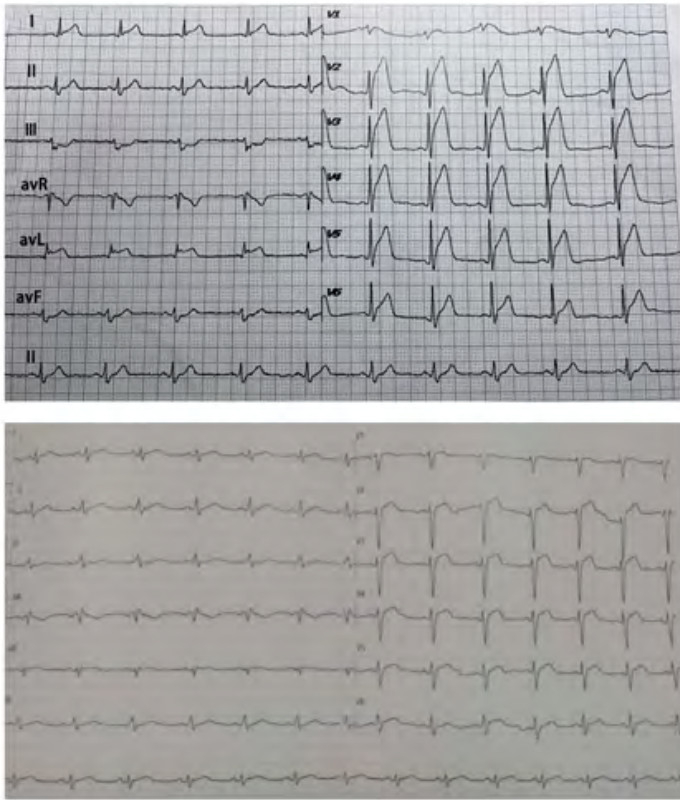


Figure 1: (A) 12-lead electrocardiogram (ECG) showing ST-elevation in the I, aVL, and V1-V6 leads. (B) ECG after the first angiography (B).

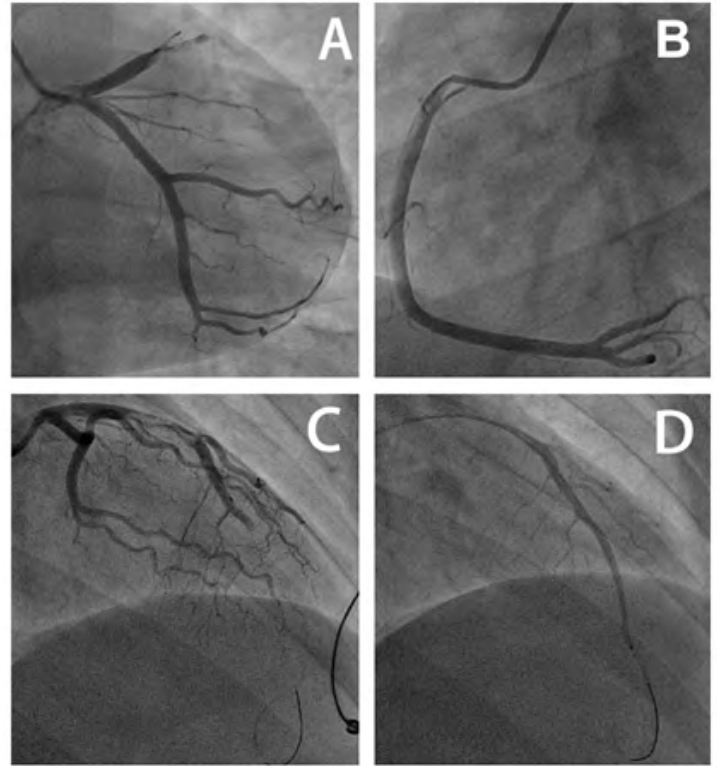


Figure 2: (A) According to the first angiography. No significant stenosis can be seen in the left main (LM), left circumflex branch (LCX). (B) No significant stenosis can be seen in the right coronary artery, with TIMI grade 3 flow. (C) Occlusion of the lumen of the proximal to the mid-ante-riodescending artery (LAD), TIMI grade 0 flow (D). After PTCA in LAD.

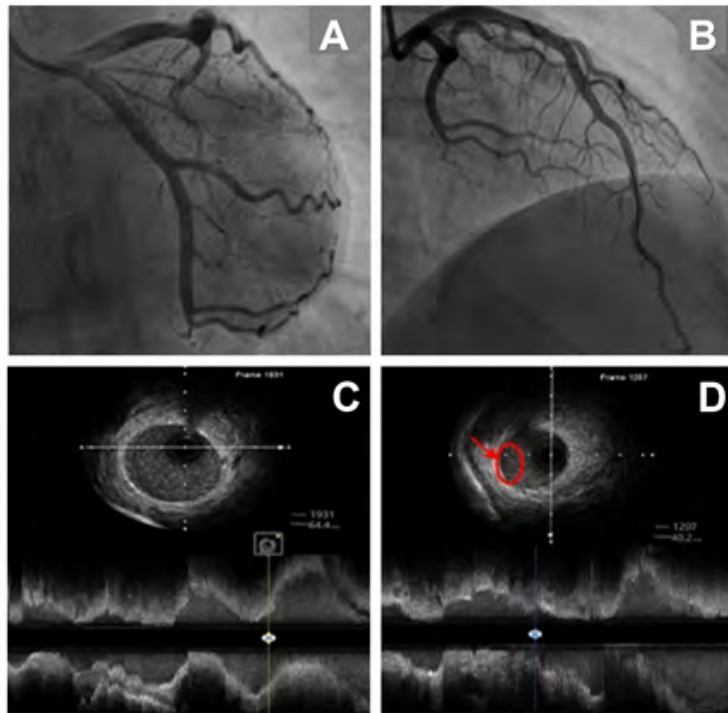


Figure 3: (A)According to the second coronary angiography, no obvious stenosis observed in LCX. (B) No obvious stenosis observed in LAD. (C) Mid-LAD under IVUS. (D)Distal-LAD under IVUS,the red arrow points to the thrombus shadow.

3. Discussion

In this case, the young man has no history of chronic disease other than a coronavirus infection 10 days prior to the onset. The patient was diagnosed with acute massive anterior wall myocardial infarction after emergency coronary angiography revealed acute thrombosis in the middle segment of the LAD but complete normality in the LCX and RCA. After treatment, the coronary angiography and IVUS examination showed that there was a little residual thrombus in the LAD, but the intima of the culprit vessel was intact.

Type I myocardial infarction is mostly caused by coronary atherosclerosis with plaque rupture or plaque erosion related to thrombosis, using the 2018 fourth edition of the worldwide definition of acute myocardial infarction [1]. Owing to an extreme imbalance between oxygen supply and demand, type II myocardial infarction is characterized by (i) decreased myocardial perfusion: coronary artery spasm, coronary microvascular dysfunction, coronary embolism, and coronary artery dissection, among others. (ii) oxygen deprivation: severe bradyarrhythmia, respiratory failure with severe anemia, shock, etc. (iii) increased oxygen consumption, including prolonged tachyarrhythmia and other conditions. In this instance, type I myocardial infarction may be ruled out because IVUS evaluation of the culprit vessel revealed no plaque and the intima was smooth. The patient had a clear culprit vessel with acute thrombotic event formation and was considered to have a possible coronary embolism (CE) due to ectopic thrombosis. Shibata [2] et al. demonstrated that atrial fibrillation (73%) was the leading cause of emboli in CE, followed by cardiomyopathy (25%) and valvular heart disease (15%). With no history of atrial fibrillation,

no structural abnormalities on cardiac ultrasound, no history of varicose veins in the lower limbs, and low D-dimers during the perioperative period, the possibility of an ectopic venous thrombosis was not considered; thus, the CE hypothesis could not explain the acute coronary thrombosis. Although the smooth intima observed on IVUS of the LAD arteries, the presence of a thrombus suggested endothelial cell damage. We can speculate that the infection of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) caused coronary vascular endothelial cell damage and activated platelets, resulting in thrombosis, because there was no evidence of autoimmune or tumor-related diseases and the patient was infected with a novel coronavirus 10 days earlier.

SARS-CoV-2 can infect the host by binding to the alveolar epithelial angiotensin-converting enzyme 2 (ACE2) receptor [3] via the S protein. ACE2 receptors are abundantly expressed on endothelial cells and dispersed in numerous organs [4], including the heart, pulmonary arteries, mesenteric veins, and glomerular capillaries. Studies have found that SARS-CoV-2 can impair endothelial cell function and can cause apoptosis of endothelial cells by directly infecting the endothelium or by recruiting immune cells[4]. Under physiological conditions, vascular endothelial cells maintain anticoagulant, antithrombotic and fibrinolytic properties, but when stimulated by inflammatory and infectious agents, they can promote coagulation and thrombosis by[5,6] expressing tissue factor (TF), releasing von Willebrand Factor (vWF), and producing thromboxane and fibrinogen activator inhibitor-1 (PAI-1). Using blood samples from 115 patients, Younes [7] et al. discovered that SARS-CoV-2 RNA can act on platelets and cause platelet release

α and dense particles, and that activated platelets under flow circumstances could stick more effectively to collagen-coated surfaces at lower thrombin concentrations. This shows that SARS-CoV-2-induced endothelial cell injury and increased platelet activation could adequately explain coronary thrombus formation in this young patient. In addition, Younes [7] discovered that there was no association between D-dimer levels and platelet activation markers, which could potentially account for the absence of significantly enhanced D-dimers in this instance.

Activated platelets and endothelium can create factor Xa in the early stages of arterial thrombosis. Rivaroxaban [8] acts directly on factor Xa, blocking the common pathway of endogenous and exogenous activation pathways during the coagulation cascade reaction. Tobias [9] et al. discovered in 2020 that FXa is also a potent platelet agonist independent of thrombin, activating platelets via PAR-1 (protease-activated receptor 1), and that rivaroxaban inhibits this pathway, reducing platelet activation, aggregation, and thrombus formation via a dose-dependent mechanism. Thus, ticagrelor and cilostazol were discontinued and substituted with rivaroxaban following the second angiography. There have been numerous reports [10] of COVID-19 patients presenting with an acute coronary syndrome; however, these patients are older and have more severe underlying illnesses. Angiography of the coronary arteries reveals that there are varying degrees of stenosis in the blood vessels. In this case, the patient is only 22 years old and has no preexisting conditions, suggesting that SARS-CoV-2 may harm normal coronary vascular endothelial cells, activate platelets, cause thrombosis, and induce acute coronary syndrome.

References

1. Thygesen K, Alpert J S, Jaffe A S, et al. Fourth Universal Definition of Myocardial Infarction (2018). *Circulation*, 2018; 138(20): p. e618-e651.
2. Shibata T, Kawakami S, Noguchi T, et al. Prevalence, Clinical Features, and Prognosis of Acute Myocardial Infarction Attributable to Coronary Artery Embolism. *Circulation*. 2015; 132(4): p. 241-50.
3. Hoffmann M, Kleine-Weber H, Schroeder S, et al. SARS-CoV-2 Cell Entry Depends on ACE2 and TMPRSS2 and Is Blocked by a Clinically Proven Protease Inhibitor. *Cell*. 2020; 181(2): p. 271-280. e8.
4. Varga Z, Flammer A J, Steiger P, et al. Endothelial cell infection and endotheliitis in COVID-19. *The Lancet*. 2020; 395(10234): 1417-1418.
5. Esposito L, Cancro F P, Silverio A, et al. COVID-19 and Acute Coronary Syndromes: From Pathophysiology to Clinical Perspectives. *Oxid Med Cell Longev*. 2021; 4936571.
6. Yau J W, Teoh H, Verma S. Endothelial cell control of thrombosis[J]. *BMC Cardiovasc Disord*, 2015; 15: 130.

7. Zaid Y, Puhm F, Allaey I, et al. Platelets Can Associate with SARS-Cov-2 RNA and Are Hyperactivated in COVID-19. *Circ Res*. 2020; 127(11): 1404-18.
8. Weitz J I, Lensing A W A, Prins M H, et al. Rivaroxaban or Aspirin for Extended Treatment of Venous Thromboembolism[J]. *N Engl J Med*. 2017; 376(13): 1211-22.
9. Petzold T, Thienel M, Dannenberg L, et al. Rivaroxaban Reduces Arterial Thrombosis by Inhibition of FXa-Driven Platelet Activation via Protease Activated Receptor-1. *Circ Res*. 2020; 126(4): 486-500.
10. Hamadeh A, Aldujeli A, Briedis K, et al. Characteristics and Outcomes in Patients Presenting With COVID-19 and ST-Segment Elevation Myocardial Infarction. *Am J Cardiol*. 2020; 131: p. 1-6.