Journal of Medical Case Reports

https://doi.org/10.1186/s13256-026-06003-0

Article in Press

Coronary thrombosis without underlying atherosclerotic plaque in patients with Omicron variant of coronavirus disease 2019: a case series

Received: 14 Oct 2023

Accepted: 19 Feb 2026

Published online: 14 May 2026

Cite this article as: Gong, M., Mao, Y. Coronary thrombosis without underlying atherosclerotic plaque in patients with Omicron variant of coronavirus disease 2019: a case series. *J Med Case Reports* (2026). https://doi.org/10.1186/s13256-026-06003-0

Ming-Lian Gong & Yi Mao

Coronary thrombosis without underlying atherosclerotic plaque in patients with Omicron variant of COVID-19: a case series

Minglian Gong, MD, PhD, Yi Mao, MD, PhD

Title Page

Ming-Lian Gong, MD, PhD [a], Yi Mao, MD, PhD [b*]

[a] Department of Cardiology, Dalian University Affiliated Zhongshan Hospital.

[b] Department of Cardiology, Fuwai Hospital, Chinese Academy of Medical Science, Peking Union Medical College, National Center for Cardiovascular Disease, China.

Ming-Lian Gong, MD, PhD, Department of Cardiology, Dalian University Affiliated Zhongshan Hospital. Institutional address: No. 6 Jiefang Road, Zhongshan District, Dalian 116001, Liaoning, China. E-mail: gongml777@126.com.

[*] Corresponding author:

Yi Mao, Department of Cardiology, Fuwai Hospital, Chinese Academy of Medical Science, Peking Union Medical College, National Center for Cardiovascular Disease, China. Institutional address: No.167 Beilishi Road, Xicheng District, Beijing 100037, China, (E-mail: 13693305832@139.com).

Conflict of interest

The authors have no conflicts of interest to disclose.

Authors' contributions

Study conception and design: YM.

Acquisition of data: M-LG.

Analysis and interpretation of data: M-LG.

Writing, review, and/or revision of the manuscript: M-LG, YM.

Study supervision: YM.

Disclosures

This study was not supported by any foundation or institution.

Abstract

Background

Coronavirus disease 2019 (COVID-19) is associated with a hypercoagulable state that predisposes patients to thrombotic events, including acute myocardial infarction (AMI). In patients with COVID-19 presenting with ST-segment elevation myocardial infarction (STEMI), coronary artery angiography (CAG) often reveals significant thrombosis, which may occur in the presence or absence of underlying obstructive atherosclerotic plaques.

Case presentations

We report a case series of three Chinese patients with Omicron variant of COVID-19 infection who presented with acute myocardial infarction due to coronary thrombosis without underlying atherosclerotic plaque on

coronary angiography. The first case involved a 33-year-old man with Covid-19 who had coronary thrombosis without underlying atherosclerotic plaque on coronary angiography. The second case was a 76-year-old man who developed infarct related ventricular septal defect (VSD) and cardiogenic shock (CS) following acute myocardial infarction (AMI). The third case was a 76-year-old woman with AMI who was also found to have venous thromboembolism (VTE). All patients were discharged and continued to improve symptomatically on follow-up phone conversations.

Conclusions

This case series highlights that infection with the Omicron variant of COVID-19 virus (especially its critical and severe type) can increase thrombogenicity and contribute to coronary thrombosis without underlying atherosclerotic plaque. Prompt diagnosis and appropriate treatment such as primary percutaneous coronary intervention (PCI) are essential. Effective anticoagulant and antiplatelet drug therapy before, during and after reperfusion therapy is a key principle. The utilization of a "Heart Team" approach ensures systematic management, discussion of therapeutic options, hemodynamic stability, and durable outcomes.

Keywords Acute myocardial infarction, Coronary thrombosis, Without underlying atherosclerotic plaque, Omicron variant of COVID-19 virus

Introduction

From December 2022, following the abolition of China's "dynamic zero-

COVID policy," the Omicron variant spread rapidly across the country. COVID-19 infection triggers a hyper-inflammatory response and increases procoagulants and platelet activation, predisposing individuals to an acute myocardial infarction (AMI)(1-3). Coronary artery angiography (CAG) in patients with COVID-19 and ST-segment elevation myocardial infarction (STEMI) frequently demonstrates substantial thrombosis with or without obstructive atherosclerotic plaques(4). This case series describes three Chinese patients with Omicron variant COVID-19 infection who presented with AMI and were found to have coronary thrombosis without underlying atherosclerotic plaque on CAG.

Case presentations

Case 1

A 33-year-old Chinese man with no significant past medical history presented on Day 0 (initial presentation) with mild chest discomfort lasting 2 hours. He had tested positive for COVID-19 (Omicron variant) via PCR two days prior (Day -2) and was diagnosed with AMI at a local hospital on Day 0. Physical examination revealed blood pressure of 120/80 mmHg, heart rate of 80 bpm, respiratory rate of 16 breaths/min, and oxygen saturation of 98% on room air. CAG on Day 0 showed an acute massive thrombus of left anterior descending (LAD) coronary artery with TIMI 2 flow (Fig. 1A). He received the optimal pharmacological therapies including anticoagulant (low-molecular-weight heparin) and antiplatelet drug therapy (DAPT: aspirin 100 mg daily and ticagrelor 90 mg twice daily)

due to his refusal of interventional therapy and thrombolytic therapy.

He was transferred to our hospital on Day 15 for further evaluation. On admission (Day 15), an electrocardiogram in our hospital showed resolution of the ST-segment elevation and residual T-wave inversion in V1–V4 (Fig. 1B). Bedside echocardiogram demonstrated antero-apical wall hypokinesis. The echocardiography with bubble study had been done to rule out paradoxical embolism. A repeat CAG on Day 16 revealed no obstructive atheroma or thrombus in LAD with TIMI 3 flow (Fig. 1C). The patient was subsequently discharged on antiplatelet drug therapy, including aspirin, and ticagrelor and showed symptomatic improvement on follow-up.

Case 2

On 28th of January 2023, a 76-year-old Chinese man with a 1-month history of fever, cough, dyspnea and diagnosis of COVID-19 interstitial pneumonia was transferred from another hospital to our institution. He was diagnosed AMI, ventricular septal defects (VSD) and maintained on an intra-aortic balloon pump (IABP) without interventional therapy and thrombolytic therapy by Local hospital 1 month ago due to high risk (December 25 2022). Pharmacotherapeutic agents was used in accordance with the guidelines in managing patients presenting with COVID-19 pneumonia, AMI and heart failure in local hospital. On admission, physical examination on January 28 showed blood pressure of 130/55 mmHg(supported by IABP), heart rate of 92 bpm, body temperature of

36.5° C, oxygen saturation (SpO2) of 85% while breathing ambient air, respiratory rate of 18 breath/min. The electrocardiogram (on January 28) in our hospital showed resolution of the ST-segment elevation and residual T-wave inversion in V1–V4 (Fig. 2A). Echocardiography confirmed antero-apical wall hypokinesis and apical VSD. The hemodynamic was supported by an intra-aortic balloon pump and the respiration was supported by continuous positive airway pressure machine.

A coronary angiography was performed under the support of IABP in our hospital (on February 2), revealed no coronary atherosclerosis in the left main, LAD, circumflex, or right coronary artery with no obstructive atheroma or thrombus in LAD and TIMI 3 flow (Fig. 2B). Surgical VSD repair was performed on February 9, 2023. He was subsequently discharged home with dual antiplatelet drug therapy.

Case 3

A 76-year-old Chinese female with a history of atrial fibrillation presented to the emergency department with shortness of breath starting four days prior. She tested positive for COVID-19. Physical examination revealed blood pressure of 110/70 mmHg, irregular heart rate of 120 bpm, respiratory rate of 22 breaths/min, and oxygen saturation of 92% on room air. Auscultation showed irregular heart sounds without murmurs. The electrocardiogram showed rapid atrial fibrillation without ST-segment elevation (Fig. 3A). Troponin-T was 1.1 ng/mL (normal <0.4ng/mL) and transthoracic echocardiography showed marked inferolateral wall motion

abnormality. Emergent CAG revealed an acute massive thrombus in proximal segment of right coronary artery (RCA) and posterior lateral with TIMI 2 flow (Fig. 3B). No thrombus aspiration or stenting was performed due to thrombus burden and risk of embolization. Medical management included 24-hour tirofiban infusion, followed by low-molecular-weight heparin for 6 days, plus DAPT (aspirin and clopidogrel). The echocardiography with bubble study had been done to rule out paradoxical embolism. One week later, a repeat coronary angiography showed a non-occlusive thrombus without underlying atherosclerotic plaque (Fig. 3C). The presence of deep vein thrombosis was confirmed by ultrasonography due to the continuously increasing D-dimer levels, but pulmonary embolism was not found. She was discharged on rivaroxaban anticoagulation and clopidogrel antiplatelet therapy. Follow-up at 3 months showed no recurrent VTE or thrombosis.

All patients were discharged on follow-up phone conversations, continues to improve symptomatically.

Discussion

Coronary artery angiography (CAG) in patients with COVID-19 and ST-segment elevation myocardial infarction (STEMI) frequently demonstrates substantial thrombosis with or without obstructive atherosclerotic plaques (4). This case series describes three patients who developed acute myocardial infarction in the context of COVID-19 infection and were

found to have huge thrombosis without underlying atherosclerotic plaque. The main pathology of STEMI, is due to severe systemic inflammation in patients with COVID-19, contributing to thrombosis with the release of inflammatory cytokines such as tumor necrosis factor-alpha (TNF-a), interferon-gamma (IFN-g), and interleukin 1 (IL-1), directly and through sympathetic stimulation. On the other hand, Hypercoagulation, due to severe inflammation, is the main pathology of spontaneous thrombosis contributing to acute myocardial infarction and venous thromboembolic diseases (1-3,5). Recent literature suggests two possible pathophysiologic mechanisms related to COVID-19 coagulopathy(6). First, the virus affects the endothelium, the natural "barrier" between the circulating blood and vessel wall elements, resulting in thromboembolic events(7). Endothelial damage creates an inflammatory environment favoring platelet activation and the increase of cytokines IL-1β and IL-8. The second mechanism suggests a more indirect viral involvement through the induction of cytokine storms(8). The main cardiovascular complications of COVID-19 include acute cardiac injury, AMI, myocarditis, arrhythmia, heart failure, shock, and venous thromboembolism (VTE)/pulmonary embolism (PE)(9). A greater predisposition to thrombosis, both arterial and venous during COVID-19 has been established(10). During SARS-CoV-2 infection, cytokine storm occurs 5 to 10 days after the beginning of symptoms, resulting in endothelial damage, activation of the platelets, and

coagulation cascade(11). The third patient who had acute myocardial infarction and was also found to have VTE. No significant venous thromboembolic had been observed in the third patient with treatment of rivaroxaban and clopidogrel at 3- month follow-up.

Although, repeat coronary artery angiography in three patients showed no thrombosis, which benefited from anticoagulation therapy in addition to the double antiplatelet therapy, percutaneous coronary intervention with aspiration thrombectomy should remain the primary and preferred strategy for these patients based on superior outcomes (12). In patients with typical symptoms and ST-segment elevation in the ECG, emergent CAG is essential. CMR helps differentiate myocardial infarction from myocarditis in patients with elevated hs TnI levels and unobstructed coronary arteries(13). Revascularization is the first-line therapy in all patients with ACS and obstructed coronary arteries(14). Primary PCI is indicated in all COVID-19 patients presenting with STEMI. Depending on the angiographic findings, balloons, drug-coated balloons, drug-eluting stents and thrombus aspiration techniques can be implemented(15). In addition, optimal medical therapy with antiplatelets, b-blockers, ACE-i or AT-II antagonists and statins is recommended(15).

The COVID-19 pandemic increased mechanical complications of AMI. The outcomes for these complications are poor without a prompt recognition and a systematic approach to management(16). The

mechanical complication of (VSD) in the second patient result from delayed presentation. Effective afterload reduction to decrease the left-to-right shunt is essential: intra-aortic balloon pumps (IABP) or impella with pharmacotherapy are used in more than 80% of emergencies and 65% of urgent repairs(17). The second patient ultimately underwent surgical repair successfully with the IABP support.

Patients with ACS and COVID-19 present a significantly increased risk of short and 1-year mortality compared to ACS patients without COVID-19 due to advanced age, comorbidities, higher inflammatory milieu, prolonged time to PCI, or even no access to PCI(18,19). Saad et al. (20) also compared in hospital STEMI patients with COVID-19 to in hospital STEMI patients without COVID-19 from prior years and reported a strikingly higher mortality rate for the STEMI and COVID-19 subset.

Limitations

This case series has limitations. 1)Intracoronary imaging (IVUS or OCT) was not used in any case, so the absence of underlying obstructive CAD cannot be confirmed with 100% accuracy; subtle plaques may have been missed. Endovascular imaging techniques OCT or IVUS may also be helpful in diagnosing underlying atherosclerotic plaques(11). Coronary angiography alone can reliably detect significant obstructive

atherosclerosis or large thrombi, but it may miss subtle or minimal underlying plaques, plaque erosion, or minor irregularities that could contribute to thrombosis. IVUS and OCT provide higher-resolution cross-sectional views of the vessel wall, allowing better characterization of plaque burden, composition, and the presence of even non-obstructive atherosclerosis. The use of intracoronary imaging may prolong the procedure time unavoidably, so we were concerned that prolonged operation may increase potential occupational hazard to staff members and cross infection during the pandemic. Full personal protective equipment was worn by all health care professionals involved in each of the procedures. Intracoronary imaging (IVUS or OCT) was unavailable for Patients with COVID-19 due to preventing cross infection during the epidemic. 2) The timing of angiography and beginning of symptoms is also quite distant and vague which could have altered the angiographic images. Delays in presentation, absence of adequate COVID-19 testing, potential occupational hazard to staff members, prolonged evaluation times in the emergency department resulted in longer door to balloon times. 3) No cardiac MRI was performed to rule out alternative diagnoses like myocarditis. Cardiac MRI was unavailable to prevent potential cross infection during the pandemic.

Conclusions

With this case series, we have highlighted Omicron variant COVID-19

infection with increased thrombogenicity can play a role in the development of Coronary thrombosis without underlying atherosclerotic plaque. Prompt diagnosis and appropriate treatment such as primary PCI are demanded. Effective anticoagulant and antiplatelet drug therapy before, during and after reperfusion therapy is also a prominent principle in proceeding with the treatment process. The "Heart Team" approach facilitates systematic management and discussion of options for hemodynamic stability and durable outcomes.

Acknowledgements

Not applicable.

Author contributions

MLG drafted the manuscript. MLG, YM reviewed the manuscript. YM is the corresponding author. All authors have approved the final version of the manuscript.

Funding

None.

Availability of data and materials

Not applicable.

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the ethical standards of the institutional review board (IRB) of Tehran University of Medical Sciences

(TUMS) and the 1964 Helsinki Declaration. Ethical approval was waived by the IRB due to the retrospective nature of the study and anonymous data reporting. Written informed consent was obtained from the patient for participation in this case study.

Consent for publication

Written informed consent was obtained from the patient's legal guardians and from the patients (as stated above) for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

The authors declare no conflicts of interest in association with the present study.

1. Shi S, Qin M, Shen B et al. Association of Cardiac Injury With Mortality in Hospitalized Patients With COVID-19 in Wuhan, China. JAMA Cardiol 2020;5:802-810.
2. Warren-Gash C, Hayward AC, Hemingway H et al. Influenza infection and risk of acute myocardial infarction in England and Wales: a CALIBER self-controlled case series study. J Infect Dis 2012;206:1652-9.
3. Claeys MJ, Coenen S, Colpaert C et al. Environmental triggers of acute myocardial infarction: results of a nationwide multiple-factorial population study. Acta Cardiol 2015;70:693-701.
4. Bangalore S, Sharma A, Slotwiner A et al. ST-Segment Elevation in Patients with Covid-19 - A Case Series. N Engl J Med 2020;382:2478-2480.
5. Setia G, Tyler J, Kwan A et al. High thrombus burden despite thrombolytic therapy in ST-elevation myocardial infarction in a patient with COVID-19. Rev Cardiovasc Med 2020;21:289-295.
6. Nicosia RF, Ligresti G, Caporarello N, Akilesh S, Ribatti D. COVID-19 Vasculopathy: Mounting Evidence for an Indirect Mechanism of Endothelial Injury. The American journal of pathology 2021;191:1374-1384.
7. Yamada S, Asakura H. Coagulopathy and Fibrinolytic Pathophysiology in COVID-19 and SARS-CoV-2 Vaccination. International journal of molecular sciences 2022;23.

8. Jiang Y, Rubin L, Peng T et al. Cytokine storm in COVID-19: from viral infection to immune responses, diagnosis and therapy. International journal of biological sciences 2022;18:459-472.
9. Dou Q, Wei X, Zhou K, Yang S, Jia P. Cardiovascular Manifestations and Mechanisms in Patients with COVID-19. Trends Endocrinol Metab 2020;31:893-904.
10. Lodigiani C, Iapichino G, Carenzo L et al. Venous and arterial thromboembolic complications in COVID-19 patients admitted to an academic hospital in Milan, Italy. Thrombosis research 2020;191:9-14.
11. Skorupski WJ, Grygier M, Lesiak M, Kałużna-Oleksy M. Coronary Stent Thrombosis in COVID-19 Patients: A Systematic Review of Cases Reported Worldwide. Viruses 2022;14.
12. Temporary Emergency Guidance to STEMI Systems of Care During the COVID-19 Pandemic: AHA's Mission: Lifeline. Circulation 2020;142:199-202.
13. European Society of Cardiology guidance for the diagnosis and management of cardiovascular disease during the COVID-19 pandemic: part 1-epidemiology, pathophysiology, and diagnosis. European heart journal 2022;43:1033-1058.
14. Kunadian V, Chieffo A, Camici PG et al. An EAPCI Expert Consensus Document on Ischaemia with Non-Obstructive Coronary Arteries in Collaboration with European Society of Cardiology Working Group on Coronary Pathophysiology & Microcirculation Endorsed by Coronary Vasomotor Disorders International Study Group. EuroIntervention : journal of EuroPCR in collaboration with the Working Group on Interventional Cardiology of the European Society of Cardiology 2021;16:1049-1069.
15. Triantafyllis AS, Sfantou D, Karapedi E et al. Coronary Implications of COVID-19. Medical principles and practice : international journal of the Kuwait University, Health Science Centre 2025;34:1-12.
16. Damluji AA, Gangasani NR, Grines CL. Mechanical Complication of Acute Myocardial Infarction Secondary to COVID-19 Disease. Cardiol Clin 2022;40:365-373.
17. Alsidawi S, Campbell A, Tamene A, Garcia S. Ventricular Septal Rupture Complicating Delayed Acute Myocardial Infarction Presentation During the COVID-19 Pandemic. JACC Case Rep 2020;2:1595-1598.
18. Çınar T, Şaylık F, Akbulut T et al. One-year outcomes of invasively managed acute coronary syndrome patients with COVID-19. Heart & lung : the journal of critical care 2022;52:159-164.
19. Case BC, Yerasi C, Forrestal BJ et al. Comparison of Characteristics and Outcomes of Patients With Acute Myocardial Infarction With Versus Without Coronarvirus-19. The American journal of cardiology 2021;144:8-12.
20. Saad M, Kennedy KF, Imran H et al. Association Between COVID-19 Diagnosis and In-Hospital Mortality in Patients Hospitalized With ST-Segment Elevation Myocardial Infarction. Jama 2021;326:1940-1952.