¹ Instituto de Cardiologia, Porto Alegre, RS, Brazil.

Coronary thrombosis after COVID-19 infection in a young patient with no comorbidities

Trombose coronariana em paciente jovem sem comorbidades após infecção pela COVID-19

Mathias Silvestre de Brida^{1iD}, Sergio Ferreira de Ferreira Filho^{1iD}, Bruno Schaaf Finkler^{1iD}, Raphael Boesche Guimarães^{1iD}

DOI: 10.31160/JOTCI202129A20210018

ABSTRACT – COVID-19 emerged as a viral infection causing severe acute respiratory syndrome, taking global proportions in 2020, with significant impact on public health. The scenario has become alarming since the infection is more severe in patients with cardiovascular diseases, mortality being up to four-fold greater in these patients, as compared to the general population. As a probable contributor, the prothrombotic inflammatory state has been subject of discussion among scientists, with no consolidated treatments in the short and long term. We describe the case of a young patient, with no cardiovascular risk factors, with ST-segment elevation myocardial infarction caused by coronary thrombosis, in the late period after COVID-19 infection, and reviewed the most recent recommendations for its treatment.

Keywords: Acute coronary syndrome; ST elevation myocardial infarction; COVID-19; Coronary thrombosis; Angioplasty

RESUMO – A COVID-19 emergiu como uma infecção viral causadora da síndrome respiratória aguda grave, tomando proporções globais no ano de 2020, com impactos significativos na saúde pública. A maior severidade da infecção em portadores de doenças cardiovasculares tem tornado o cenário alarmante, com mortalidade até quatro vezes maior em relação à população geral. Como provável contribuinte, o estado inflamatório pró-trombótico vem sendo alvo de discussão no meio científico, ainda sem terapêuticas consolidadas a curto e longo prazo. Descrevemos o caso de um paciente jovem, sem fatores de risco cardiovasculares, com infarto agudo do miocárdio com supradesnível do segmento ST causado por trombose coronariana no período tardio pós-infecção pela COVID-19, revisando as mais recentes recomendações para seu tratamento.

Descritores: Síndrome coronariana aguda; Infarto do miocárdio com supradesnível do segmento ST; COVID-19; Trombose coronária; Angioplastia

How to cite this article:

Brida MS, Ferreira SF, Finkler BS, Guimarães RB. Coronary thrombosis after COVID-19 infection in a young patient with no comorbidities. J Transcat Intervent. 2021;29:eA20210018. <https://doi.org/10.31160/JOTCI202129A20210018>

Corresponding author:

Mathias Silvestre de Brida
Avenida Princesa Isabel, 395 – Santana
Zip code: 90040-371 – Porto Alegre, RS, Brazil
E-mail: mathias-sb@hotmail.com

Received on:

May 14, 2021

Accepted on:

August 24, 2021

INTRODUCTION

Coronavirus disease 2019 (COVID-19) has been weakening global public health since 2020, when it was declared pandemic.¹ The scenario has become alarming for the infection is more severe in patients with cardiovascular diseases, and mortality being up to four-fold greater in this group of patients as compared to the general population.² We describe the case of a young patient, with no cardiovascular risk factors, with ST-segment elevation myocardial infarction (STEMI) caused by coronary thrombosis in the late period after COVID-19 infection, and reviewed the most recent recommendations for its treatment.

The Internal Review Board of the Instituto de Cardiologia do RS/Fundação Universitária de Cardiologia evaluated and approved this study (opinion number 4.682.382, CAAE: 45636121.5.0000.5333) on April 30th, 2021.

CASE REPORT

A 27-year-old male patient presented to the emergency department for pressure-type retrosternal pain that had initiated 8 hours before, irradiating to both

This content is licensed under a Creative Commons Attribution 4.0 International License.

shoulders and associated with nausea. He was unaware of comorbidities or family history of cardiovascular diseases, and reported only oligosymptomatic COVID-19 infection 25 days before the visit, confirmed by reverse transcriptase polymerase chain reaction (RT-PCR). He denied the use of illicit substances or hormone or nutritional protein supplements, as well as smoking or alcohol drinking habits. Physical examination with no significant abnormalities, stable vital signs, symmetrical pulses, and absence of heart murmur.

Electrocardiogram (ECG) upon arrival showed sinus rhythm and ST-segment elevation in DII, DIII and aVF leads, as well as V3R and V4R (Figure 1), and an inferior wall and right ventricle STEMI was diagnosed. A loading dose of dual antiplatelet therapy (DAP; acetylsalicylic acid – ASA – 300mg and clopidogrel 600mg) and anticoagulation with unfractionated heparin were given. The patient was referred for primary percutaneous coronary intervention (PCI) in due time (less than 120 minutes).

Figure 1. 12-lead electrocardiogram showing ST-segment elevation in the inferior wall and right leads.

He was admitted to a reference tertiary care hospital, with persistence of chest pain and ECG abnormalities, and was referred for coronary angiography, which demonstrated absence of obstructions in the left coronary system, and right coronary artery (RCA) occluded in the proximal third by acute thrombosis (Figure 2). Percutaneous coronary intervention was carried out implanting two drug-eluting stents, with complete recovery of the vessel lumen, Thrombolysis in Myocardial Infarction (TIMI) flow III, with no signs of dissection (Figure 3). The patient was subsequently referred to the coronary unit for monitoring and post-acute coronary syndrome care.

ADA: anterior descending artery; LCx: left circumflex artery; RCA: right coronary artery.

Figure 2. Coronary angiogram showing left coronary system in right anterior oblique caudal view (A), no obstructions. The right coronary system is viewed in oblique anterior left view (B), with acute thrombotic obstruction in the proximal third of the right coronary artery.

Figure 3. Right coronary angiography after angioplasty with drug-eluting stent, with recovery of coronary flow.

The patient progressed with no complications. Laboratory tests demonstrated elevation of ultrasensitive troponin T with a peak of 1,794.00pg/mL (reference value <14.00pg/mL), non-reactive viral serologies, negative inflammatory and autoimmune tests, and normal glycemic and lipid tests. Considering the need for early anticoagulation and the state of acute thrombosis, clinical conditions that may interfere in the investigation of thrombophilia, the decision was for additional investigation on an outpatient basis. Transthoracic echocardiogram showed preserved ejection fraction, with no associated mechanical complications. The treatment was optimized with angiotensin-converting enzyme inhibitor, statin, beta-blocker, DAP with ASA and clopidogrel, and anticoagulation with unfractionated heparin.

After 5 days at the hospital, he was discharged in good general conditions, with no clinical signs of heart failure or chest pain. It was decided to maintain full anticoagulation with rivaroxaban for 3 months, associated with DAP, due to the high thrombotic load and the probable correlation between the clinical event and the prothrombotic state induced by COVID-19.

DISCUSSION

COVID-19 emerged as a viral infection causing severe acute respiratory syndrome, taking global proportions in 2020, with significant impact on public health.¹ High morbidity and mortality have been observed in patients with preexisting cardiovascular diseases, being up to four times more lethal in this population as compared to the general population.²

Cardiovascular involvement in the clinical presentation of COVID-19 is commonly reported, with increased markers of myocardial injury (troponin) in up to 12% of cases, which is related to higher mortality.^{2,3} As for the mechanism of myocardial injury, the possible causes are direct toxicity of the virus in cardiomyocytes, microvascular dysfunction secondary to endothelial injury, imbalance between oxygen supply and demand, rupture of a preexisting atherosclerotic plaque, and formation of thrombi in the microvasculature secondary to disseminated intravascular coagulation.⁴

The prothrombotic state is being proven as the reports of these arterial or venous thrombotic events increase in the infected people, and may manifest in patients with no respiratory symptoms, or even weeks after the acute condition.^{5,6} A multifactorial model is proposed as an explanation for this phenomenon, related to macrophage activation of the complement system, cytokine storm, hyperactivation of the renin-angiotensin system, among others.⁷

In this report, the early clinical presentation of the coronary event, along with the absence of cardiovascular risk factors, strengthens the hypothesis of COVID-19 infection as the etiology of thrombogenesis, even in a late phase. In a retrospective study recently published with 28 patients with COVID-19 undergoing angiography for STEMI, Stefanini et al. demonstrated in 85.7% of them, infarction was the first manifestation of the viral disease. In 60.7% of those, there was obstructive lesion requiring revascularization.⁸ However, other scenarios that predispose to vascular thrombosis in this age group must be investigated, such as smoking, alcohol consumption, thrombophilia, and the use of illicit drugs or ergogenic hormones, nutritional protein supplements, and stimulants.

The intensity of anticoagulation in COVID-19 has been widely discussed. Recent guidelines recommend maintaining prophylactic anticoagulation in hospitalized patients with no evidence of thromboembolic event. However, in cases of high risk for thrombosis with low risk of bleeding, full anticoagulation may be considered.⁹ After discharge, for high-risk patients, rivaroxaban 10mg, for 31 to 39 days, has been approved as antithrombotic prophylaxis in non-COVID-19 patients and can be considered individually in infected patients.¹⁰

Due to the large number of thrombi on angiography, it was decided to maintain anticoagulation for a prolonged period (3 months), associated with DAP. There is no evidence so far in the literature regarding the duration of prothrombotic state after coronavirus infection, and the duration of the treatment should be individualized according to the risk of new events, thrombotic load, and risk of bleeding.

Myocardial infarction due to coronary thrombosis may present as a single manifestation of coronavirus infection, even weeks after the acute phase. There is no consensus in the scientific literature on prevention and treatment of these events. The period of prothrombotic state after infection is uncertain, and oral anticoagulation may be con-

sidered for a longer period after the thromboembolic event, especially in affected patients who do not have higher risk factors for bleeding related to anticoagulant therapy.

SOURCE OF FINANCING

None.

DECLARATION OF CONFLICTS OF INTEREST

The authors declare having no conflicts of interest.

CONTRIBUTION OF AUTHORS

Conception and design of the study: MSB, SFFF, BSF and RBG; data collection: MSB, SFFF, BSF and RBG; data interpretation: MSB, SFFF, BSF and RBG; text writing: MSB, SFFF, BSF and RBG; approval of the final version to be published: MSB, SFFF, BSF and RBG.

REFERENCES

1. Zheng YY, Ma YT, Zhang JY, Xie X. COVID-19 and the cardiovascular system. *Nat Rev Cardiol*. 2020;17(5):259-60. <https://doi.org/10.1038/s41569-020-0360-5>
2. Guo T, Fan Y, Chen M, Wu X, Zhang L, He T, et al. Cardiovascular implications of fatal outcomes of patients with coronavirus disease 2019 (COVID-19). *JAMA Cardiol*. 2020;5(7):811-8. <https://doi.org/10.1001/jamacardio.2020.1017>
3. Guan WJ, Liang WH, Zhao Y, Liang HR, Chen ZS, Li YM, et al. Comorbidity and its impact on 1590 patients with COVID-19 in China: a nationwide analysis. *Eur Respir J*. 2020;55(5):2000547. <https://doi.org/10.1183/13993003.00547-2020>
4. Atri D, Siddiqi HK, Lang JP, Nauffal V, Morrow DA, Bohula EA. COVID-19 for the cardiologist: basic virology, epidemiology, cardiac manifestations, and potential therapeutic strategies. *JACC Basic Transl Sci*. 2020;5(5):518-36. <https://doi.org/10.1016/j.jacbts.2020.04.002>
5. Klok FA, Kruip MJ, van der Meer NJ, Arbous MS, Gommers D, Kant KM, et al. Confirmation of the high cumulative incidence of thrombotic complications in critically ill ICU patients with COVID-19: An updated analysis. *Thromb Res*. 2020;191:148-50. <https://doi.org/10.1016/j.thromres.2020.04.041>
6. Giannis D, Ziogas IA, Gianni P. Coagulation disorders in coronavirus infected patients: COVID-19, SARS-CoV-1, MERS-CoV and lessons from the past. *J Clin Virol*. 2020;127:104362. <https://doi.org/10.1016/j.jcv.2020.104362>
7. Hanff TC, Mohareb AM, Giri J, Cohen JB, Chirinos JA. Thrombosis in COVID-19. *Am J Hematol*. 2020;95(12):1578-89. <https://doi.org/10.1002/ajh.25982>
8. Stefanini GG, Montorfano M, Trabattoni D, Andreini D, Ferrante G, Ancona M, et al. ST-Elevation myocardial infarction in patients with COVID-19: Clinical and angiographic outcomes. *Circulation*. 2020;141(25):2113-6. <https://doi.org/10.1161/CIRCULATIONAHA.120.047525>
9. Cuker A, Tseng EK, Nieuwlaar R, Angchaisuksiri P, Blair C, Dane K, et al. American Society of Hematology 2021 guidelines on the use of anticoagulation for thromboprophylaxis in patients with COVID-19. *Blood Adv*. 2021;5(3):872-88. <https://doi.org/10.1182/bloodadvances.2020003763>
10. National Institute of Health (NIH). Coronavirus disease 2019 (COVID-19). Treatment guidelines. NIH, 2021 [cited 2021 Jul 6]. Available from: <https://www.covid19treatmentguidelines.nih.gov/>