E-ISSN:2320-8686
P-ISSN:2321-127X
Short Communication
COVID-19

International Journal of Medical Research and Review

2021 Volume 9 Number 5 September-October

Publisher
MEDRESEARCH
www.medresearch.in

Acute myocardial infarction in post COVID-19 patients

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DOI: https://doi.org/10.17511/ijmrr.2021.i05.02

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The SARS-CoV-2 infection has caused mortality in different populations; in addition, morbidity with immunological, hematological and/or cardiovascular compromises has been reported, with acute myocardial infarction (AMI) standing out. A literature search was conducted in databases to determine existing evidence regarding AMI in post-COVID-19 patients. It was found that initially AMI was described in some patients during the disease; however, a recently published case series showed that it could occur in recovered patients, secondary to systemic and procoagulant inflammation over time, suggesting further research in this area.

Keywords: AMI, COVID-19, Cardiovascular disease, Acute myocardial infarction

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How to Cite this Article

Moisés Andrés Lombana Salas, María Camila Miranda Jiménez, Michelle Bedoya Coronel, Wendy Yulieth Herazo Madera, Enrique Arturo Lombana Salas, Acute myocardial infarction in post COVID-19 patients. Int J Med Res Rev. 2021;9(5):286-288.
Available From
https://ijmrr.medresearch.in/index.php/ijmrr/article/view/1319

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Manuscript Received	Review Round 1	Review Round 2	Review Round 3	Accepted
2021-08-26	2021-08-28	2021-09-08	2021-09-20	2021-09-30

Conflict of Interest	Funding	Ethical Approval	Plagiarism X-checker	Note
No	Nil	Yes	5%	

Introduction

COVID-19 has left multiple health sequelae around the world, generating the need for continuous care in survivors of SARS-CoV-2 infection who consult for different systemic conditions, among which stand out: Guillain-Barré syndrome, rheumatoid arthritis, pediatric multisystemic inflammatory syndromes such as Kawasaki disease, hematologic and cardiovascular system dysfunction [1]; the latter system is known to be predisposed to be affected by SARS-CoV-2 directly by virus damage itself or indirectly through inflammation, endothelial activation and microvascular thrombosis. Approximately 25% of hospitalized patients present myocardial injury, associated with increased mortality risk [2]. Little has been written in the medical literature on post-COVID-19 acute myocardial infarction (AMI). Based on the above, this short review aims to describe the mechanisms that cause AMI in the context of COVID-19, based on the pathophysiological bases that have already been described and to analyze the possible causes that determine the appearance of cases of post-COVID-19 AMI based on what the literature suggests, using a recently published case series as a starting point.

Methodology

A search was performed in recognized scientific databases: PubMed, WorldWideScience, SciELO, LILACS, using the descriptors: "acute myocardial infarction" AND "post COVID19", "Myocardial infarction" AND "post COVID19", "cardiovascular dysfunction" AND "post COVID19". Case series, meta-analysis, review articles were selected for this review.

Pathophysiological mechanisms involved in AMI due to COVID-19

Myocardial involvement in COVID-19 has different pathophysiological mechanisms, which are:

01. a destroyed atherosclerotic plaque in type I myocardial infarction.[1,3].
02. A dysfunction between oxygen supply and demand as in type II AMI. Within this type of infarction, the following pathophysiological mechanisms are included: [1,3].

2.a. The presence of an atheroma generates myocardial perfusion limitation.[1,3].

2.b. Coronary microcirculation with endothelial failure.[1].

2.c. Elevated levels of Angiotensin II as a cause of severe arterial hypertension.[1].

02. d. "Intense arteriolar vasoconstriction and hypoxemia as a result of acute respiratory distress syndrome or pulmonary vascular thrombosis in situ." [1]

03. Sepsis.[1].

04. Lung tissue damage.[1]

05. Respiratory failure, related to severe physiologic stress that may be associated with elevations of stress biomarkers and myocardial injury". [1].

The dysfunctions described above are supported by a set of molecular factors such as the expression of angiotensin-converting enzyme II receptors in the myocardium, generating susceptibility in cardiomyocytes to attack by SARS-CoV-2.[4]. In addition, the presence of cytokines such as IL-6 and inflammatory mediators such as TNF-α and nitric oxide add much more myocardial damage, predisposing to the development of AMI.[1].

Current evidence

In the literature review, we found a single case series published in May 2021, described by Shiun Woei Wong et al., which mentions the presentation of AMI in 3 patients post COVID-19 with more than 80 days of recovery, all-male, with no previous cardiovascular history, standard features were elevated levels of von Willebrand factor antigen, factor VIII and D-dimer, ST-segment elevation and ultrasound findings of a fibrotic plaque with the minimal necrotic centre, with a hypercoagulable state determined by ultrasound analysis of the clot. [5].

Discussion

The myocardial damage resulting from SARS-CoV-2 infection determines the possibility of developing AMI during hospitalization, even in patients without known cardiovascular disease.[6]. The presentation of post-COVID-19 AMI is not frequently described in the medical literature, it is only known from the previously mentioned case series, and it is this report that shows the possibility of suffering AMI after recovery from COVID-19, based on the chance that systemic inflammatory and procoagulant activity is maintained over time.[4].

On the other hand, pneumonia-type pulmonary infections probably predispose to AMI; this is based on meta-analysis and long-term follow-up studies of patients who have suffered from community-acquired pneumonia, which showed an increased risk of cardiovascular disease without the presence of previous cardiovascular disease [4,7]. something similar happened with people who suffered from severe acute respiratory syndrome (SARS) who continued to present cardiovascular problems after 12 years of follow-up.[8].

This is complemented by the case series cited above, demonstrating "the strong thrombogenic nature of COVID-19 that persisted among patients who recovered from infection".[5].

Conclusion

The most significant described precedent so far regarding AMI in post-COVID-19 patients is the report of a small number of cases; however, taking into account the pathophysiology of direct and indirect myocardial and endothelial damage that can occur in a SARS-COV-2 infection, it would be exciting and with significant impact on the medical community to develop a longitudinal observational study to follow up patients who achieved resolution of their COVID-19 condition from a focus on the cardiovascular aspect, to obtain guidelines to propose protective measures and avoid conditions such as AMI.

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